

City Research Online

City St George's, University of London

Citation: Barnard, N. A. S. (1999). A quantitative analysis of eye movement characteristics during the cover test. (Unpublished Doctoral thesis, City University London)

This is the submitted version of the paper.

This version of the publication may differ from the final published version. To cite this item please consult the publisher's version.

Permanent repository link: https://city-ref-test.eprints-hosting.org/id/eprint/12049/

Copyright and Reuse: Copyright and Moral Rights remain with the author(s) and/or copyright holders. Copies of full items can be used for personal research or study, educational, or not-for-profit purposes without prior permission or charge, unless otherwise indicated, provided that the authors, title and full bibliographic details are credited, a hyperlink and/or URL is given for the original metadata page and the content is not changed in any way. For full details of reuse please refer to City Research Online policy.

City Research Online: http://openaccess.city.ac.uk/ publications@citystgeorges.ac.uk

A quantitative analysis of eye movement characteristics during the cover test

Nigel Andrew Simon Barnard

Submitted for the degree of PhD

Department of Optometry & Visual Science

City University

London

Submitted July 1999

Table of Contents

Abstract 29

CHAPTER 1. INTRODUCTION 31

1.1 Definitions and fundamental laws 32

1.1.1 Positions of gaze 32

1.1.2 Torsion 32

1.1.3 Listing's Plane 33

1.1.4 Listing's Law 33

1.1.5 Donder's Law 34

1.1.6 Hering's Law of Equal Innervation 34

1.1.7 Sherrington's Law of Reciprocal Innervation 34

1.2 The Ocular Motor System 35

1.2.1 Anatomy 35

1.2.2 Physiology 36

1.2.3 Neurology 36

1.3 Eye Movements 42

1.3.1 Saccades 42

1.3.2 Vergence eye movements 52

1.4 Vergence anomalies 75

1.4.1 Heterophoria 76

1.4.2 Associated phoria 94

1.4.3 Fusional reserves 97

1.4.4 Assessment of convergence 97

1.4.5 Anomalies of vergence 98

1.5 Symptomatology of binocular vision anomalies.....107
1.5.1 Specific symptoms.....108
1.5.2 The relationship between binocular function and symptoms.....110
1.5.3 Grading systems for symptoms.....115

1.6 The treatment of binocular vision anomalies.....116
1.6.1 Prismatic correction.....116
1.6.2 Refractive correction.....117
1.6.3 Orthoptic exercises.....118
1.6.4 Surgery.....124

1.7 Summary.....124

CHAPTER 2. METHOD FOR ASSESSING EYE MOVEMENTS DURING THE COVER TEST.....125

2.1 Introduction.....125

2.2 Methods.....126
2.2.1 Automated cover test.....126

2.3 Data analysis.....138
2.3.1 Determination of an optimal cover/uncover protocol.....138
2.3.2 Experiment to assess the repeatability of eye movement characteristics during the cover test.
.....156
2.3.3 Variation of eye movement characteristics during the cover test over a 6 hour period.....157
2.3.4 Conclusions.....163

CHAPTER 3. EYE MOVEMENT CHARACTERISTICS DURING THE COVER TEST IN A POPULATION OF 100 NORMAL SUBJECTS 164

3.1 Subjects.....164

3.2 Results.....164

3.2.1 The cover phase 166

3.2.2 The recovery phase 189

CHAPTER 4. THE RELATIONSHIP BETWEEN THE RESULTS OF A RANGE OF CLINICAL BINOCULAR VISION TESTS AND EYE MOVEMENT CHARACTERISTICS DURING THE COVER TEST 213

4.1 Introduction.....213

4.2 Method.....214

4.3 Results.....217

4.3.1 Symptoms.....217

4.3.2 Associated phoria.....218

4.3.3 Near fusional reserves 218

4.3.4 Near point of convergence (NPC).....220

4.3.5 Conventional cover test, Maddox rod and Maddox wing.....220

4.3.6 AC/A ratio 224

4.3.7 Amplitudes of accommodation 224

4.3.8 Stereopsis 225

4.3.9 Binocularity.....225

4.4 Relationship between results of clinical tests and eye movement characteristics during the automated cover tests.....226

4.4.1 Near fusional reserves 226
4.4.2 Near point of convergence.....227
4.4.3 Associated phoria.....228
4.4.4 Comparison of the conventional cover test and the automated cover test phoria amplitudes .228
4.4.5 Comparison of automated cover test with Maddox Rod and Maddox Wing 231
4.4.6 Discussion of levels of agreement between automated and conventional cover tests, Maddox rod and Maddox Wing 233
4.4.7 Stereopsis 234

4.5 Summary of results.....235

CHAPTER 5. EYE MOVEMENT CHARACTERISTICS DURING THE COVER TEST IN A GROUP OF 30 SUBJECTS REFERRED FOR TREATMENT OF BINOCULAR VISION ANOMALIES..... 236

5.1 Introduction.....236

5.2 Subjects.....236

5.3 Methods 237

5.4 Results.....237
5.4.1 The cover phase 238
5.4.2 The recovery phase 247

5.5 Summary of results.....255

CHAPTER 6. THE RELATIONSHIP BETWEEN SYMPTOM SCORES AND OCULAR MOTOR CHARACTERISTICS 257

6.1 Symptom scores and recovery time 258

6.1.1 Average of right and left eye recovery times (recovery time^{RL}) 258

6.1.2 Maximum recovery time (recovery time^{max}) 260

6.2 Symptom scores and number of eye movements to achieve recovery 260

6.2.1 Number of eye movements to achieve recovery - average of right and left eye 261

6.2.2 Number of eye movements to achieve recovery – the greater of right or left eye 262

6.3 Symptom scores and NPC 263

6.4 Symptom scores and fusional reserves 264

6.5 Symptom scores and associated phoria 265

6.6 Summary 265

CHAPTER 7. THE EFFECT OF A REGIME OF ORTHOPTIC TREATMENT ON OCULAR MOTOR CHARACTERISTICS 267

7.1 Introduction 267

7.2 Methods 268

7.3 Subjects **269**
7.3.1 Anecdotal control group 269

7.4 Results **269**
7.4.1 Subjects diagnosed as CI 272
7.4.2 Discussion of individual case histories 283
7.4.3 Non-CI subjects 306
7.4.4 Anecdotal controls 318

7.5 Discussion **332**

CHAPTER 8. DISCUSSION **333**

8.1 A comparison between a 2 s, 10 s and alternate cover test **333**

8.2 Eye movement characteristics of 100 'normal' subjects during the cover test **334**

8.3 Comparison of the cover test with other tests of binocular function **338**

8.4 Eye movement characteristics during the cover test of 30 referred subjects **338**

8.5 Symptom scores **339**

8.6 The effect of orthoptic treatment on cover test eye movements **340**

8.7 Conclusion 342

Table of Figures

Figure 1.1. Illustrating false torsion. An eye movement from a primary to a tertiary position is always associated with a definite and unique tilt (false torsion) of the corneal vertical meridian with respect to the objective vertical (from Myers KJ, 1975). 33

Figure 1.2. Illustration showing Listing's Plane (from Myers KJ, 1975).....33

Figure 1.3. The Warwick schema for the oculomotor nucleus of the monkey (adapted from Wolf, 1968). 38

Figure 1.4. Origin of the oculomotor nerve in the midbrain. (Motor neurons are red; preganglionic parasympathetic neurons are blue) (from Barr & Kiernan, 1993).....39

Figure 1.5. Origin of the trochlear nerve in the midbrain (from Barr & Kiernan, 1993). 40

Figure 1.6. Origin of the abducens nerve in the mid- brain (from Barr ML & Kiernan JA, 1993).....41

Figure 1.7. Some of the pathways for the control of eye movements by the cerebral cortex and superior colliculus (from Barr ML & Kiernan, 1993). 43

Figure 1.8. Pathways involved in conjugate lateral movements of the eyes (from Barr ML & Kiernan, 1993).....44

Figure 1.9. Sampled-data saccadic control model (from Young LR, 1981).....46

Figure 1.10. Saccadic main sequence diagrams showing peak velocity, duration, and the first peak accelerations as functions of saccadic eye movements of 13 subjects with normal vision (from Bahill et al, 1981).....47

Figure 1.11. Main sequence disparity vergence responses for a variety of stimulus conditions in one subject. Filled symbols, Instrument space environment (that is, disparity stimulation only); open symbols, free-space environment (that is, disparity plus blur plus proximity; X disparity-only standard step stimulation in instrument space (from Hung GK, Ciuffreda KJ, Semmlow JL, Hornig JL, 1994). 57

Figure 1.12. Static model of accommodation and vergence. See text for details. (from Hung 1992).....65

Figures 1.13. Diagrams describing a dynamic model of disparity vergence only (from Hung et al 1986).....68

Figure 1.14. Eye movement traces showing time constants for relaxation of fusional vergence after stimulating convergence for 5 seconds (upper trace) and 60 seconds (lower trace). A = onset of stimulation; B = occlusion of one eye (from Schor C, 1980). 72

Figure 1.15. A simple schematic diagram illustrating the possible relationships between various categories of ocular motor imbalances and their possible sequelae. 76

Figure 1.16. Frequency distribution of 925 symptom-free heterophoric subjects (from Dowley D, 1990).....78

Figure 2.1. Photograph of a subject positioned on the dental bite with the occluders both in the uncovered position. 127

Figure 2.2. Diagram to illustrate the arrangement of an infrared limbal reflection system for recording eye movements.....128

Figure 2.3. Diagrams to illustrate areas of iris/sclera illuminated by infrared light by a pair of infrared emitting diodes. 128

Figure 2.4. Diagram illustrating the design of the fixation target used during the automated cover test. *129*

Figure 2.5. Schematic representation of the equipment used during the automated cover test. The subject is viewing the distance fixation target. The computer screen used to present the near fixation target could be seen slid to one side on the optical bench. *130*

Figure 2.7 Schematic representation of the pattern of eye movements during repeated fixation between a central target (0°) and a target 10° from the centre. *133*

Figure 2.8. Photograph showing the equipment used during the automated cover test. The set up shown is for distance fixation with the near display screen situated out of position. *135*

Figure 2.9. Diagram to illustrate periods of binocular fixation and unilateral occlusion during the 10 s cover test. *136*

Figure 2.10. Diagram to illustrate periods of binocular fixation and unilateral occlusion during the 2 s cover test. *136*

Figure 2.11. Diagram to illustrate periods of binocular fixation and unilateral occlusion during the alternate cover test *137*

Figure 2.14. Scatter plot showing mean phoria amplitude^{RL} for each cycle of the alternate cover test for distance. (Each datum point describes the average of 6 measurements; error bars denote ± 1 standard deviation). *142*

Figure 2.15. Scatter plot showing mean phoria amplitude^{RL} for each cycle of the alternate cover test for near. (Each datum point describes the average of 6 measurements; error bars denote ± 1 standard deviation). *143*

Figure 2.16. Change in mean latency as the distance 2s cover test cycle progresses. (Error bars denote ± 1 standard deviation). *148*

Figure 2.17.Change in mean latency as the near 2s cover test cycle progresses. (Error bars denote ± 1 standard deviation). *148*

Figure 2.18. Change in mean latency with progression of the distance cover test cycle (Error bars denote ± 1 standard deviation). *149*

Figure 2.19. Change in mean latency with progression of the near cover test cycle. (Error bars denote ± 1 standard deviation). *150*

Figure 2.20. Mean recovery times following the distance 2 s cover test. (Error bars denote ± 1 standard deviation). *151*

Figure 2.21. Mean recovery times following the near 2 s cover test. (Error bars denote ± 1 standard deviation). *152*

Figure 2.22. Change in mean recovery times with repeated distance alternate cover test. (Error bars denote ± 1 standard deviation). *153*

Figure 2.23. Change in mean recovery and latency times with repeated distance alternate cover test. *153*

Figure 2.24. Change in mean recovery times with repeated near alternate cover test. (Error bars denote ± 1 standard deviation). *154*

Figure 2.25. Change in mean recovery and latency times with repeated near alternate cover test. *154*

Figure 2.26. Scatter plot showing variation of distance phoria amplitude with time for 3 subjects. ...158

Figure 2.27. Scatter plot showing variation of near phoria amplitude with time for 3 subjects. 159

Figure 2.28. Scatter plot showing right and left eye latency times following the distance cover test for subjects C and M (RE = right eye; LE = left eye). 160

Figure 2.29. Scatter plot showing right and left eye latency times following the near cover test. 161

Figure 2.30. Scatter plot showing recovery time ^{RL} following the distance cover test. 161

Figure 2.31. Scatter plot showing recovery time ^{RL} following the near cover test. 162

Figure 3.1. Schematic diagram showing various parameters extracted from the eye movement traces (not to scale). The continuous blue line denotes an eye movement trace. 165

Figure 3.2. An example of an eye movement recording during the near cover test. 167

Figure 3.3. Expanded view of the right eye cover phase. 168

Figure 3.4. Eye movement trace showing a slow exophoric drift while each eye is occluded. 169

Figure 3.5. Eye movement trace for the right eye of the same subject as in Figure 3.4, showing an expanded view of the eye movement profile under the cover. 169

Figure 3.6. Eye movement trace showing a more rapid exophoric fusional decay while each eye is occluded. 170

Figure 3.7. Expanded view of Figure 3.6 showing eye movements of the left eye under the cover. 170

Figure 3.8. Eye movement trace to show how one eye completes the dissociation movement to reach the maximum amplitude more quickly than the other. 171

Figure 3.9. Frequency distributions of phoria amplitudes (degrees) of right and left eyes during the distance cover test (a) maximum phoria (b) after 2 seconds occlusion (c) after 10 seconds occlusion. 172

Figure 3.10. Diagram showing relationship between right and left eye phoria amplitudes (degrees) during the distance cover test in the normal group (a) maximum amplitudes (R^2 = 51%; n = 81), (b) after 2 seconds occlusion (R^2 = 43%; n = 79), and (c) after 10 seconds occlusion (R^2 = 42%, n = 81). All show a 1-Tail probability of < 0.001. 174

Figure 3.11. Relationship between the difference between right and left eye phoria amplitudes and their average for the distance cover test. (a) maximum phoria amplitudes, (b) 2 second amplitudes and (c) 10 second amplitudes. 175

Figure 3.12. Frequency distributions (%) of phoria amplitudes (degrees) of right and left eyes during the near cover test (a) maximum phoria (b) after 2 seconds occlusions, (c) after 10 seconds occlusion. 176

Figure 3.13. Diagram showing relationships between right and left eye phoria amplitudes (degrees) during the near cover test in the normal group (a) maximum amplitudes (Pearson R^2 = 73%, n= 91) (b) after 2 seconds occlusion (R^2 = 70%, n = 87) and (c) after 10 seconds occlusion (R^2 = 67%, n = 93). All show a 1-Tail probability of < 0.001. 178

Figure 3.14. Relationship between the difference between right and left eye phoria amplitudes and their average for the near cover test. (a) maximum phoria amplitudes, (b) 2 second amplitudes, and (c) 10 second amplitudes. 179

Figure 3.15. Box and whisker and dot plot illustrating the distribution of phoria amplitude^{RL} for distance and near. 180

Figure 3.16. Graph showing the relationship between the amplitude^{RL} of distance and near phorias. 181

Figure 3.17. Diagram showing the relationship between the difference between the 10 s and 2 s phoria amplitudes^{RL} and the average of 10 s and 2 s phoria amplitudes^{RL} for distance. 183

Figure 3.18. Diagram showing the relationship between the difference between the 10 s and 2 s phoria amplitudes^{RL} and the average of 10 s and 2 s phoria amplitudes^{RL} for near. 183

Figure 3.19. Frequency distributions of the time taken to reach the phoria amplitude measured following 10 s occlusion. 185

Figure 3.20. Diagram showing cumulative frequencies of time taken to reach 10 s distance phoria amplitude. The blue line shows the normal distribution curve. The 95% frequency line is shown. 186

Figure 3.21. Diagram showing cumulative frequencies of time taken to reach 10 s near phoria amplitude. The blue line shows the normal distribution curve. The 95% frequency line is shown. 187

Figure 3.22. Scatter diagram illustrating the relationship between 10 s distance phoria amplitude and the time taken to reach that amplitude. (a) exophoric eyes and (b) esophoric eyes.... 188

Figure 3.23. Scatter diagram illustrating the relationship between 10 s near phoria amplitude and the time taken to reach that amplitude. (a) esophoric eyes and (b) exophoric eyes. 189

Figure 3.24. Eye movement trace illustrating the latency for a recovery movement following removal of the cover..... 191

Figure 3.25. Eye movement trace illustrating an example of an 'average' latency. 191

Figure 3.26. Eye movement trace illustrating an example of a long latency. 192

Figure 3.27. Eye movement trace illustrating an example of a short latency. 193

Figure 3.28. Expanded view of the recovery phase of the right eye following removal of the cover illustrating the associated movements of the fixating eye. 194

Figure 3.29 Further expanded view of right eye during recovery phase. 195

Figure 3.30. Expanded view of the right eye of a subject with 3 degrees exophoria taking two movements to achieve recovery. 195

Figure 3.31. Expanded view of the left eye of a subject with a 4.5 degree exophoria. 196

Figure 3.32. Frequency distributions (%) for latency of recovery movement following removal of the occluder for all eyes in the normal group for distance and near. Two outlying near latencies (4100 ms and 6780 ms) are omitted (see text). 199

Figure 3.33. Frequency distributions (%) for latency of recovery eye movements during the distance cover test. 200

Figure 3.34. Frequency distribution (%) of latency of recovery eye movements during the near cover test. This excludes two esophoric eyes with saccadic latencies of 6780 ms and 4100 ms. 201

Figure 3.35. Frequency distributions of number of eye movements to achieve recovery following the automated cover test for distance and near fixation.....204
Figure 3.36. Frequency distributions of number of eye movements to achieve recovery following the distance automated cover test for exophores and esophores. 205
Figure 3.37. Frequency distributions of number of eye movements to achieve recovery following the near automated cover test for exophores and esophores. 206
Figure 3.38. Frequency distributions of right and left recovery times (seconds) following the distance cover test.....207
Figure 3.39. Frequency distributions of right and left recovery times (seconds) following the near cover tests in the normal group 207
Figure 3.40. Relationship between right and left recovery times (seconds) following cover test for (a) distance (R^2 = 14%; n= 56) and (b) near (R^2 = < 1%; n = 66).....208
Figure 4.1. Frequency distribution of near symptom scores. 218
Figure 4.2. Box and whisker plot illustrating the distribution of blur/break/recovery fusional reserve measurements.....219
Figure 4.3. Frequency distribution of near point of convergence (cm).....220
Figure 4.4. Distance phoria measurements by conventional cover test and Maddox rod (n = 39 subjects).....221
Figure 4.5. Difference between distance phoria amplitude measurements with the conventional cover test and with the Maddox rod plotted against the average of the results for the two test methods (n= 39 subjects).....222
Figure 4.6. Near phoria estimation by conventional cover test and Maddox wing (n = 39). 223
Figure 4.7. Difference between near phoria amplitude measurements with the conventional cover test and with the Maddox wing plotted against the average of the results for the two test methods (n = 39).....223
Figure 4.8. Frequency distribution of AC/A ratios. 224
Figure 4.9. Frequency distribution of stereo-acuity (n = 39).....225
Figure 4.10. Distance phoria amplitudes (prism dioptres) measured by the conventional and automated cover test with line of equality (n = 27).....229
Figure 4.11. Near phoria amplitudes (prism dioptres) measured by the conventional and automated cover test with line of equality (n = 34).....230
Figure 4.12. Difference between near phoria measurements with the conventional and automated cover tests plotted against the average. 230
Figure 4.13. Distance phoria amplitudes (prism dioptres) measured by the Maddox rod and automated cover test with line of equality (n = 28).....231
Figure 4.14. Difference between distance phoria measurements with the automated cover test and Maddox rod plotted against the average.....232
Figure 4.15. Near phoria amplitudes (prism dioptres) measured by the automated cover test and the Maddox wing with line of equality (n = 35).....232

Figure 4.16 Difference between near phoria measurements with the automated cover test and Maddox rod plotted against the average.....233

Figure 5.1. Frequency distributions (%) of phoria amplitudes (degrees) of right and left eyes during the distance cover test after 10 seconds occlusion.....238

Figure 5.2. Diagram showing relationships between right and left eye phoria amplitudes (degrees) during the distance cover test in the referred group after 10 seconds occlusion (Wilcoxon Signed Rank R^2 = 19%; n = 30; 1-Tail probability = 0.01).....239

Figure 5.3. Relationship between the difference between right and left eye 10 second phoria amplitude and their average for the distance cover test. 240

Figure 5.4. Frequency distributions (%) of phoria amplitudes (degrees) of right and left eyes during the near cover test after 10 seconds occlusion.....241

Figure 5.5. Diagram showing relationships between right and left eye phoria amplitudes (degrees) during the near cover test in the referred group after 10 seconds occlusion (R^2 = 60%; n = 30; 1-Tail probability = 0.00).....242

Figure 5.6. Relationship between the difference between right and left eye 10 s phoria amplitudes and their average for the near cover test.....242

Figure 5.8. Frequency distributions of the time taken to reach the phoria amplitude measured following 10 s occlusion.....245

Figure 5.9. Diagram showing cumulative frequencies of time taken to reach 10 s distance phoria amplitude during the distance cover test. The blue line shows the cumulative normal distribution curve. The 95% frequency line is shown. 245

Figure 5.10. Diagram showing cumulative frequencies of time taken to reach 10 s near phoria amplitude. The blue line shows the cumulative normal distribution curve. The 95% frequency line is shown.....246

Figure 5.11. Frequency distributions (%) for latency of recovery movement following removal of the occluder for all eyes in the referred group for distance and near. An outlying distance latency was omitted. 249

Figure 5.12. Frequency distributions of number of eye movements to achieve recovery following the automated cover test for distance and near fixation.....250

Figure 5.13. Frequency distributions of number of eye movements to achieve recovery following the near automated cover test for normal and referred groups. 252

Figure 5.14. Relationship between right and left recovery times (seconds) following the near cover test (Spearman Rank R^2 = 3%; n = 23).....254

Figure 6.1. Frequency distribution of symptom scores for the combined groups (n = 69 subjects).....257

Figure 6.2. Frequency distribution of recovery time^{RL} of the combined groups (two outliers omitted) (n= 44).....258

Figure 6.3. Scatter plot showing distribution of symptom scores as a function of recovery time. 259

Figure 6.4. Scatter plot showing relationship between symptom score and recovery time^{max}. 260

Figure 6.5. Frequency distribution the number of recovery movements^{RL} (n = 37 subjects). 261

Figure 6.6. Scatter plot showing the relationship between symptom scores and the number of eye movements to achieve recovery. Some points describe overlapped data. 261

Figure 6.7. Scatter plot showing the relationship between symptom score and the maximum (right or left eye) number of recovery eye movements. Some points describe overlapped data.262

Figure 6.8. Scatter plot showing the relationship between symptom score and NPC. 263

Figure 6.9 (a) Relationship between symptom score and blur (R^2 = 4 %); (b) Relationship between symptom score and break (R^2 = < 1%) and (c) Relationship between symptom score and recovery (R^2 = <1 %).....264

Figure 7.1. Scatter plot showing mean near recovery time (right and left eyes) for subjects within group α. 275

Figure 7.2. Scatter plot showing mean recovery time (right and left eyes) for subjects within group β. 276

Figure 7.3. Graph showing mean NPC before treatment commenced, after one month of treatment and after treatment had ceased for one month.....279

Figure 7.4. Graph showing mean fusional reserves (prism dioptres) on presentation, after 1 month of treatment and 1 month after cessation of exercises (standard deviations (SD) and the number of subjects (n) are shown).....280

Figure 7.5. Changes in symptom score and NPC over the treatment period for subject allyo. 283

Figure 7.6.Changes in symptom score and recovery time over the treatment period for subject allyo. 283

Figure 7.7. Changes in symptom score and fusional reserves over the treatment period for subject allyo.....284

Figure 7.8. Changes in symptom score and associated phoria over the treatment period for subject allyo.....284

Figure 7.9. Changes in symptom score and NPC over the treatment period for subject kurp.....286

Figure 7.10. Changes in symptom score and recovery time over the treatment period for subject kurp. 286

Figure 7.11. Changes in symptom score and fusional reserves over the treatment period for subject kurp. 286

Figure 7.12. Changes in symptom score and associated phoria over the treatment period for subject kurp. 286

Figure 7.13. Changes in symptom score and NPC over the treatment period for subject mdob.....287

Figure 7.14. Changes in symptom score and recovery time over the treatment period for subject mdob. 287

Figure 7.15. Changes in symptom score and fusional reserves over the treatment period for subject mdob. 288

Figure 7.16. Changes in symptom score and associated phoria over the treatment period for subject mdob. 288

Figure 7.17. Changes in symptom score and NPC over the treatment period for subject siwh. 290

Figure 7.18. Changes in symptom score and recovery time over the treatment period for subject siwh. 290

Figure 7.19. Changes in symptom score and fusional reserves over the treatment period for subject siwh....290

Figure 7.20. Changes in symptom score and associated phoria over the treatment period for subject siwh....290

Figure 7.21. Changes in symptom score and NPC over the treatment period for subject aisha.....292

Figure 7.22. Changes in symptom score and recovery time over the treatment period for subject aisha. 292

Figure7.23. Changes in symptom score and fusional reserves over the treatment period for subject aisha. 292

Figure 7.24. Changes in symptom score and associated phoria over the treatment period for subject aisha. 292

Figure 7.25. Changes in symptom score and NPC over the treatment period for subject fatma. 293

Figure 7.26. Changes in symptom score and recovery time over the treatment period for subject fatma. 293

Figure 7.27. Changes in symptom score and fusional reserves over the treatment period for subject fatma....294

Figure 7.28. Changes in symptom score and associated phoria over the treatment period for subject fatma....294

Figure 7.29. Changes in symptom score and NPC over the treatment period for subject gurao.....296

Figure 7.30. Changes in symptom score and recovery time over the treatment period for subject gurao. 296

Figure 7.31. Changes in symptom score and fusional reserves over the treatment period for subject gurao. 296

Figure 7.32. Changes in symptom score and associated phoria over the treatment period for subject gurao. 296

Figure 7.33. Changes in symptom score and NPC over the treatment period for subject jimh. 297

Figure 7.34. Changes in symptom score and recovery time over the treatment period for subject jimh. 297

Figure 7.35. Changes in symptom score and fusional reserves over the treatment period for subject jimh....298

Figure 7.36. Changes in symptom score and associated phoria over the treatment period for subject jimh....298

Figure 7.37. Changes in symptom score and NPC over the treatment period for subject kadep.....299

Figure 7.38. Changes in symptom score and recovery time over the treatment period for subject kadep. 299

Figure 7.39. Changes in symptom score and fusional reserves over the treatment period for subject kadep. 300

Figure 7.40. Changes in symptom score and associated phoria over the treatment period for subject kadep. 300
Figure 7.41. Changes in symptom score and NPC over the treatment period for subject neth. 301
Figure 7.42. Changes in symptom score and recovery time over the treatment period for subject neth. 301
Figure 7.43. Changes in symptom score and fusional reserves over the treatment period for subject neth. 302
Figure 7.44. Changes in symptom score and associated phoria over the treatment period for subject neth. 302
Figure 7.45. Changes in symptom score and NPC over the treatment period for subject vicox. 304
Figure 7.46. Changes in symptom score and recovery time over the treatment period for subject vicox. 304
Figure 7.47. Changes in symptom score and fusional reserves over the treatment period for subject vicox. 304
Figure 7.48. Changes in symptom score and associated phoria over the treatment period for subject vicox. 304
Figure 7.49. Scatter plot showing average recovery time (right and left eyes) for subjects within the non-CI group. 307
Figure 7.50. Changes in symptom score and NPC over the treatment period for subject airey. 312
Figure 7.51. Changes in symptom score and recovery time over the treatment period for subject airey. 312
Figure 7.52. Changes in symptom score and fusional reserves over the treatment period for subject airey. 313
Figure 7.53. Changes in symptom score and associated phoria over the treatment period for subject airey. 313
Figure 7.54. Changes in symptom score and NPC over the treatment period for subject arif. 314
Figure 7.55. Changes in symptom score and recovery time over the treatment period for subject arif. 314
Figure 7.56. Changes in symptom score and fusional reserves over the treatment period for subject arif. 315
Figure 7.57. Changes in symptom score and associated phoria over the treatment period for subject arif. 315
Figure 7.58. Changes in symptom score and NPC over the treatment period for subject shmeh. 316
Figure 7.59. Changes in symptom score and recovery time over the treatment period for subject shmeh. 316
Figure 7.60. Changes in symptom score and fusional reserves over the treatment period for subject shmeh. 317
Figure 7.61. Changes in symptom score and associated phoria over the treatment period for subject shmeh. 317
Figure 7.63. Changes in symptom score and NPC over the treatment period for subject jadoo. 319

Figure 7.64. Changes in symptom score and recovery time over the treatment period for subject jadoo. 319
Figure 7.65. Changes in symptom score and fusional reserves over the treatment period for subject jadoo. 319
Figure 7.66. Changes in symptom score and associated phoria over the treatment period for subject jadoo. 319
Figure 7.67. Changes in symptom score and NPC over the treatment period for subject mayli. 322
Figure 7.68. Changes in symptom score and recovery time over the treatment period for subject mayli. 322
Figure 7.69. Changes in symptom score and fusional reserves over the treatment period for subject mayli. 322
Figure 7.70. Changes in symptom score and associated phoria over the treatment period for subject mayli. 322
Figure 7.71. Changes in symptom score and NPC over the treatment period for subject nazmo. 325
Figure 7.72. Changes in symptom score and recovery time over the treatment period for subject nazmo. 325
Figure 7.73. Changes in symptom score and fusional reserves over the treatment period for subject nazmo 325
Figure 7.74. Changes in symptom score and associated phoria over the treatment period for subject nazmo 325
Figure 7.75. Changes in symptom score and NPC over the treatment period for subject sselb. 327
Figure 7.76. Changes in symptom score and recovery time over the treatment period for subject sselb. 327
Figure 7.77. Changes in symptom score and fusional reserves over the treatment period for subject sselb. 327
Figure 7.78. Changes in symptom score and associated phoria over the treatment period for subject sselb. 327
Figure 7.79. Changes in symptom score and NPC over the treatment period for subject vcurt. 329
Figure 7.80. Changes in symptom score and fusional reserves over the treatment period for subject vcurt 329
Figure 7.81. Changes in symptom score and associated phoria over the treatment period for subject vcurt 329
Figure 7.82. Changes in symptom score and NPC over the treatment period for subject vicup 331
Figure 7.83. Changes in symptom score and recovery time over the treatment period for subject vicup. 331
Figure 7.84. Changes in symptom score and fusional reserves over the treatment period for subject vicup. 331
Figure 7.85. Changes in symptom score and associated phoria over the treatment period for subject vicup. 331

List of Tables

Table 1.1. Actions of the extraocular muscles from the primary position. 35

Table 1.2. Factors that affect saccadic latency (adapted from Ciuffreda & Tannen, 1995). 49

Table 1.3. Categories of divergence insufficiency (Tait, 1951). 99

Table 1.4. Summary of selected studies of CI in normal clinical or school populations (adapted from Rouse et al, 1998). 102

Table 1.5. Prevalence of types of inadequate jump-convergence in 455 patients (from Pickwell & Hampshire, 1981). 103

Table 1.6. Criteria used by Rouse et al (1998) to categorise subjects. 104

Table 1.7. Prevalence of symptoms in a population of students manifesting binocular dysfunctions (from Porcar & Martinez-Palomera, 1997) 108

Table 2.1. Fixation accuracy of the eye movement recording system/subject combination. 134

Table 2.2. List of showing the 18 permutations of order of carrying out the three types of cover test. 140

Table 2.3 Comparison of 10s phoria amplitude^{RL}, 2 s amplitude$^{RL}{}_{ALL}$, and alternate cover test amplitude$^{RL}{}_{ALL}$ for distance. 144

Table 2.4 Comparison of 10s phoria amplitude^{RL}, 2 s amplitude$^{RL}{}_{ALL}$, and alternate cover test amplitude$^{RL}{}_{ALL}$ for near 144

Table 2.5. Comparison of 10 s distance amplitude^{RL} and first cycle 2 s and first cycle alternate distance cover test phoria amplitudes $^{RL}{}_{1st}$ 145

Table 2.6. Comparison of 10 s near amplitudes^{RL} and first cycle 2 s and first cycle alternate near cover test phoria amplitudes $^{RL}{}_{1st}$ 145

Table 2.7. Comparison of 10 s distance amplitude^{RL}, last cycle 2 s amplitude$^{RL}{}_{4th}$, and last cycle alternate distance cover test phoria amplitudes$^{RL}{}_{6th}$ 145

Table 2.8. Comparison of 10 s near amplitude^{RL}, last cycle 2 s amplitude$^{RL}{}_{4th}$, and last cycle alternate distance cover test phoria amplitudes$^{RL}{}_{6th}$ 146

Table 2.9 Mean latency times (ms) following 2 s cover test. 147

Table 2.10. Mean latency times (ms) following the alternate cover test 149

Table 2.11. Recovery times (s) following 2 s cover test. 151

Table 2.12. Recovery times (s) following the alternate cover test. 152

Table 2.13. Data for repeated measures of parameters for a single subject. 156

Table 3.1. Summary of mean maximum, 2 second and 10 second distance phoria amplitudes (degrees) for right and left eyes. 172

Table 3.2. Summary of mean maximum, 2 second and 10 second distance phoria amplitudes (degrees) for right and left eyes. 176

Table 3.3. Mean times for eyes to reach the 10 s phoria amplitude during occlusion. 186

Table 3.4. Relative frequencies of the type of eye movement initiating recovery. 197

Table 4.1. Summary of fusional reserve findings. 219

Table 4.2. Average values of horizontal fusional reserves for near (33 – 40 cm) (after Pickwell, 1965). 219

Table 4.3. Spearman Rank R^2 data for average latency time and fusional reserves. 226

Table 4.4. Relationships between the number of recovery movements and fusional reserves. 227

Table 5.1. Relative frequencies of type of eye movement initiating recovery in the referred group....247

Table 7.1. Data table showing clinical data of referred subjects at first presentation. Automated cover test phoria amplitudes^{RL} are shown to the nearest ½ Δ 271

Table 7.2. Table listing progression of treatment for those subjects who attended at least one follow-up appointment. 272

Table 7.3. Symptom profiles of the CI subjects over time.....273

Table7.4. Recovery times(s) of CI subjects following the automated near cover test over time.....274

Table 7.5. Mean number of eye movements made to achieve recovery. 277

Table 7.6. NPC over time for the CI group.....278

Table 7.7. Positive fusional reserves (blur/break/recovery) over time.....279

Table 7.8. Changes in associated phoria.....281

Table 7.9. Symptom profiles of the non-CI subjects over time.....306

Table7.10. Recovery times(s) of non-CI subjects following the automated near cover test over time..307

Table 7.11. Average number of eye movements made to achieve recovery 308

Table7.12. NPC measures (cm) of non-CI over time.....309

Table 7.13. Positive fusional reserves (blur/break/recovery) of non-CI subjects over time. 310

Table 7.14. Changes in associated phoria over time.....310

Dedication

To my family. To Mum and Dad to whom I am ever grateful. To Louise who supported me unselfishly. To Gabriella, Reuben and Boaz who unknowingly sacrificed their time to enable me to complete this thesis.

Acknowledgements

I would like to thank sincerely my friend and colleague Dr David Thomson whose help and encouragement has been unwavering throughout this study. His support and expertise has been invaluable.

I would also like to thank my dear lifelong friend, Andy Peace, who spent many an hour discussing statistics with me. Also to David Seidel for finding time to read the manuscript.

Finally, I wish to thank all my colleagues at the Department of Optometry & Visual Science for their general help and encouragement.

Author's statement

I, Nigel Andrew Simon Barnard, grant powers of discretion to the University Library to allow this thesis to be copied in whole or in part without further reference to me. This permission covers only single copies made for study purposes, subject to normal conditions of acknowledgement.

Abstract

The cover test is probably the most widely used clinical test of oculomotor status. It is surprising therefore, that there has been only one previous study which has attempted to provide a quantitative analysis of the eye movements during the test. There is also a dirth of information concerning the relationship between eye movements during the cover test and symptomatology and the correlation between cover test results and the outcome of other tests of binocular function.

For the investigations described in this thesis, apparatus was developed to provide precise measurements of eye movements during a computer-contolled cover test, with subjects fixating a distant (3.4m) and near (0.4m) target.

In the first study, this apparatus was employed to assess a group of asymptomatic emmetropes aged between 18 and 35 years (n = 100). The pattern of eye movements recorded was more complex than is often assumed. Eye movements during dissociation followed various patterns, with some subjects reaching a position of equilibrium within a few seconds while 20% had not reached a stable position at the end of the 10 s occlusion period. It was concluded that the 'standard' procedure of occluding an eye during the cover test for only about 2 s is not adequate. The mean phoria after 10 s occlusion was 0.00° for distance fixation and –1.38° (exo) for near fixation. The pattern of eye movements during the recovery phase consisted of a variety of saccadic and vergence movements. There was a statistical difference between exophores and esophores for frequencies of initiating saccades and vergence eye movements ($p < 0.001$) with esophores more commonly commencing recovery with a saccade and exophores with a vergence eye movement. Recovery movements were often associated with movements of the "fixating" eye. There was a poor correlation between phoria amplitude and recovery time.

In the second study, the relationship between the nature of eye movements during the cover test and the results of other common tests of binocular function was investigated. There was very little correlation between any

aspect of the eye movements and the results of the other tests, or indeed between any of the tests of binocular function.

In the third study, a group of symptomatic individuals were characterised using the automated cover test and a battery of other tests of binocular function.

In the fourth study, the association between eye movement characteristics during the cover test and symptomatology was investigated. Results gave some support to the long-held view that a slow multi-stage recovery tends to be associated with a symptomatic binocular vision problem.

In the fifth study, various aspects of binocular function were monitored throughout the course of orthoptic therapy. Not all subjects responded to treatment and none of the clinical tests assessed were found to be good discriminators of subjects who were likely to benefit. While several aspects of binocular function were found to parallel the amelioration of symptoms, the results were very variable.

Chapter 1. Introduction

The cover test is probably the most widely used test of ocular motor balance enabling the practitioner to determine the presence and measure the amplitude of heterophoria and strabismus. The cover test requires only an occluder and a trained observer/practitioner. It enables the practitioner to make a rapid assessment of the ocular motor balance with patients fixating at different distances and in different directions of gaze. Apart from the patient being required to maintain fixation on a target, there is usually no other subjective input by the patient. In this respect the cover test may be described as objective although it is the practitioner's subjective observations that are recorded.

For over a century there have been descriptions of the qualitative aspects of recovery eye movements during the cover test. These descriptions have been generally accepted as clinical 'wisdom' and have frequently been passed down almost as an oral tradition. Although the cover test is used by most eye care practitioners there have been surprisingly few studies to investigate its validity. This thesis describes a series of investigations of eye movements during the cover test and an analysis of these eye movements before and after a course of orthoptics/ vision training. The main aim of this thesis is to describe and quantify the true nature of eye movements during the cover test with the objective of providing clinicians with evidence that confirms or refutes the conventional wisdom that is imbued into their thoughts.

Whilst orthoptic treatment or vision training has been used for many years to treat vergence anomalies, the author is not aware of any studies that have investigated the effect of orthoptic exercises on eye movement characteristics during the cover test. A secondary aim of this thesis is to investigate any changes in these characteristics in a small group of subjects before and after they received a regime of orthoptic exercises with the objective of providing the practising clinician with a better understanding of the effects of such treatment.

1.1 Definitions and fundamental laws

There are a number of definitions and laws that are used in relation to eye movements and the ocular motor system. The most important of these are defined below.

1.1.1 Positions of gaze

Primary position of gaze is that position of the eyes in binocular vision when, with head erect, the object of regard is at infinity and lies at the intersection of the saggital plane of the head and a horizontal plane passing through the centres of rotation of the two eyeballs (Myers, 1975).

Secondary position of gaze is obtained by a rotation around either a horizontal or vertical axis.

Tertiary position of gaze is the position obtained by a simultaneous rotation around the horizontal and vertical axes.

1.1.2 *Torsion*

True torsion is a true cyclorotational movement of the eye about an anterior posterior axis such as the line-of-sight.

False torsion is the apparent cyclorotation of the eye associated with a change in direction of regard from the primary position to a tertiary position. False torsion is a consequence of the reference system used for specification of the eye position and orientation in space (Ciuffreda & Tannen, 1995) (see Figure 1).

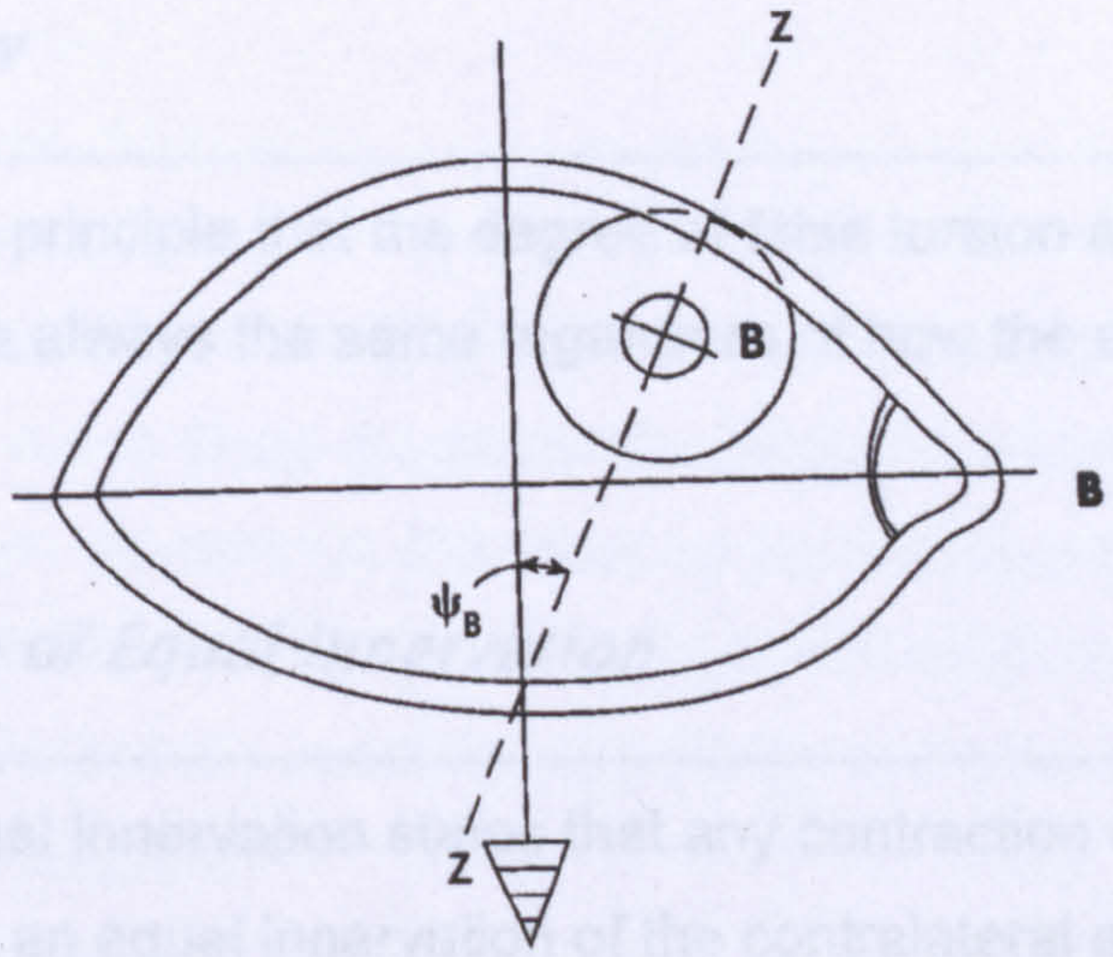


Figure 1.1. Illustrating false torsion. An eye movement from a primary to a tertiary position is always associated with a definite and unique tilt (false torsion) of the corneal vertical meridian with respect to the objective vertical (from Myers KJ, 1975).

1.1.3 Listing's Plane

Listing's Plane is the plane passing through the head and the centre of rotation of the eyes that is perpendicular to the line of sight when the eyes are in the primary position (see Figure 1.2).

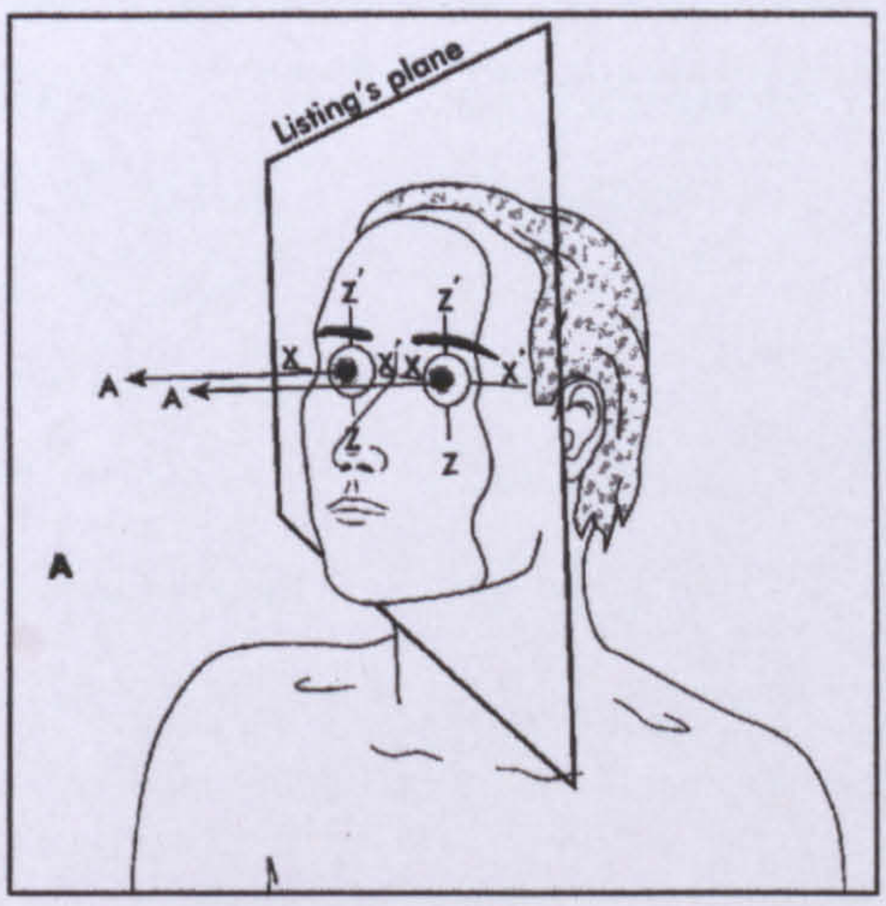


Figure 1.2. Illustration showing Listing's Plane (from Myers KJ, 1975).

1.1.4 Listing's Law

Listing's Law states that the movement of the eyes away from the primary position of gaze to any other position is equivalent to a single rotation about Listing's plane, and, is associated with a definite degree of false torsion.

1.1.5 Donder's Law

Donder's Law is the principle that the degree of false torsion at a given tertiary position of the eye is always the same regardless of how the eye reached that position.

1.1.6 Hering's Law of Equal Innervation

Hering's Law of Equal Innervation states that any contraction of an extraocular muscle will produce an equal innervation of the contralateral agonist.

1.1.7 Sherrington's Law of Reciprocal Innervation

Sherrington's Law of Reciprocal Innervation stands for the principle that when an agonist contracts during movement of the eye, there is a simultaneous and equal relaxation of the ipsilateral antagonist.

1.2 The Ocular Motor System

Whilst this thesis is primarily engaged in the study of those horizontal eye movements carried out to fixate targets in the primary position of gaze, particularly those eye movements produced by the lateral and medial recti, in order to study the characteristics of these eye movements it is necessary to have an understanding of the anatomy, physiology and neurology of all the extraocular rotatory muscles. [1]

1.2.1 Anatomy

Each eyeball is suspended in its bony orbit by the extraocular rotatory muscles ("EOM"), the fascia and the fibrous septa. Of the seven extrinsic muscles located outside the eye, six of these are the EOM – namely the lateral rectus, the medial rectus, the superior rectus, the inferior rectus, the superior oblique and the inferior oblique. Table 1.1 sets out the actions of the EOM from the primary position.

Apart from the inferior oblique muscle, all of the extraocular muscles take their origin in the Common Tendinous Annulus of Zinn.

Muscle	**Primary Action**	**Secondary action**	**Secondary action**
Lateral rectus	Abduction	-	-
Medial rectus	Adduction	-	-
Superior rectus	Elevation	Intorsion	Adduction
Inferior rectus	Depression	Extorsion	Adduction
Superior oblique	Intorsion	Depression	Abduction
Inferior oblique	Extorsion	Elevation	Abduction

Table 1.1. Actions of the extraocular muscles from the primary position.

---

[1] The author acknowledges the use of Adler's Physiology of the Eye (Eds. Moses & Hart, 1970) and Movements of the Eyes (Carpenter,1977). The reader is referred to these texts for a more comprehensive treatment of this subject

The extraocular muscles are striated and voluntary. They are approximately 40 mm long and 6-10 mm wide and they insert into the sclera approximately 5-7 mm posterior to the limbus (Bergmanson, 1995).

1.2.2 Physiology

There are two types of muscle fibres found in the striated muscle: (a) extrafusal fibres and (b) intrafusal fibres. The latter are located inside of the muscle spindle. Muscle spindles are afferent sensory organs and their role in the eye is not fully understood (Bergmanson, 1995).

Muscle fibres are multinucleated and are composed of fibrils. These in turn are composed of even smaller filaments that are the basic contractile structure in the striated muscle. There is a rich network of endoplasmic reticulum (called sarcoplasmic reticulum in muscle cells) which serves to initiate the contraction process by releasing its calcium stores as a response to depolarisation of the sarcolemma and t-tubule system (Bergmanson, 1995).

Muscle fibres may also be categorised into fast-"twitch" (or fibrillen struktur) and the slow-"twitch" fibres (feldern-struktur). The former has numerous mitochondria and is innervated by a single nerve fibre ("en plaque innervation"). In contrast, the latter has fewer mitochondria and is innervated by a cluster of nerve fibres ("en grappe innervation") (Bergmanson, 1995).

1.2.3 Neurology

Extraocular muscles of particular interest with regard to the current study are the lateral and medial recti. These muscles are exclusively involved in eye movements along the horizontal plane. The following is a brief overview of the innervation for all the extraocular muscles.

1.2.3.a. Cranial nerve III: The Oculomotor Nerve

Cranial nerve III innervates the:

- Medial rectus,

- Superior rectus,
- Inferior rectus,
- Inferior oblique,
- Levator Palberbra Superioris,
- Iris sphincter muscle,
- Iris dilator muscle,
- Ciliary muscle.

The oculomotor nucleus in humans is approximately 5-6 mm long and lies in the midbrain below the cerebral aqueduct in the upper portion of the cerebral peduncle at the level of the superior colliculus. The paired nuclei have a triangular outline in transverse section and are bounded by the medial longitudinal fasiculi. The cells for individual extraocular muscles (excluding the levator palpebra superioris) are localised in longitudinal groups (Barr & Kiernan, 1993). Warwick (1953) was the first to describe the structure of the oculomotor nucleus in monkey that comprised of three laterally disposed cell groups supplying the inferior rectus, inferior oblique and medial rectus muscles. Fibres from the Edinger-Westphal nucleus accompany other oculomotor fibres into the orbit where they terminate in the ciliary ganglion. Axons then pass through the short posterior ciliary nerves to supply the sphincter pupillae and the ciliary muscle. A reproduction of the Warwick schema is shown in Figure 1.3.

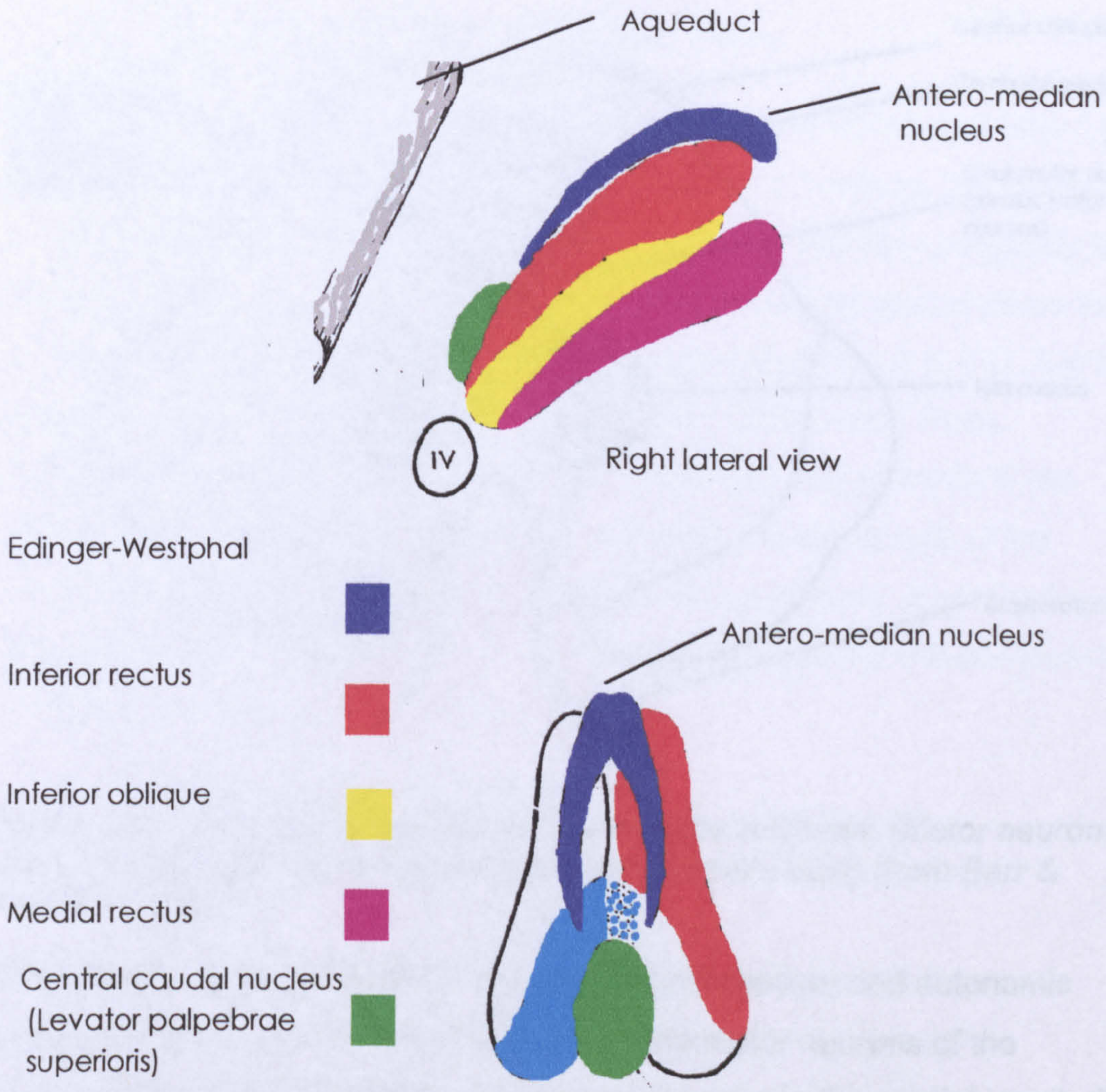


Figure 1.3. The Warwick schema for the oculomotor nucleus of the monkey (adapted from Wolf, 1968).

The oculomotor nerve consists of approximately 24,000 nerve fibres which leave the nuclear complex and pass ventrally through the red nucleus, exiting the brain stem through the medial portion of each cerebral peduncle to emerge in the inter-peduncular space (see Figure 1.4).

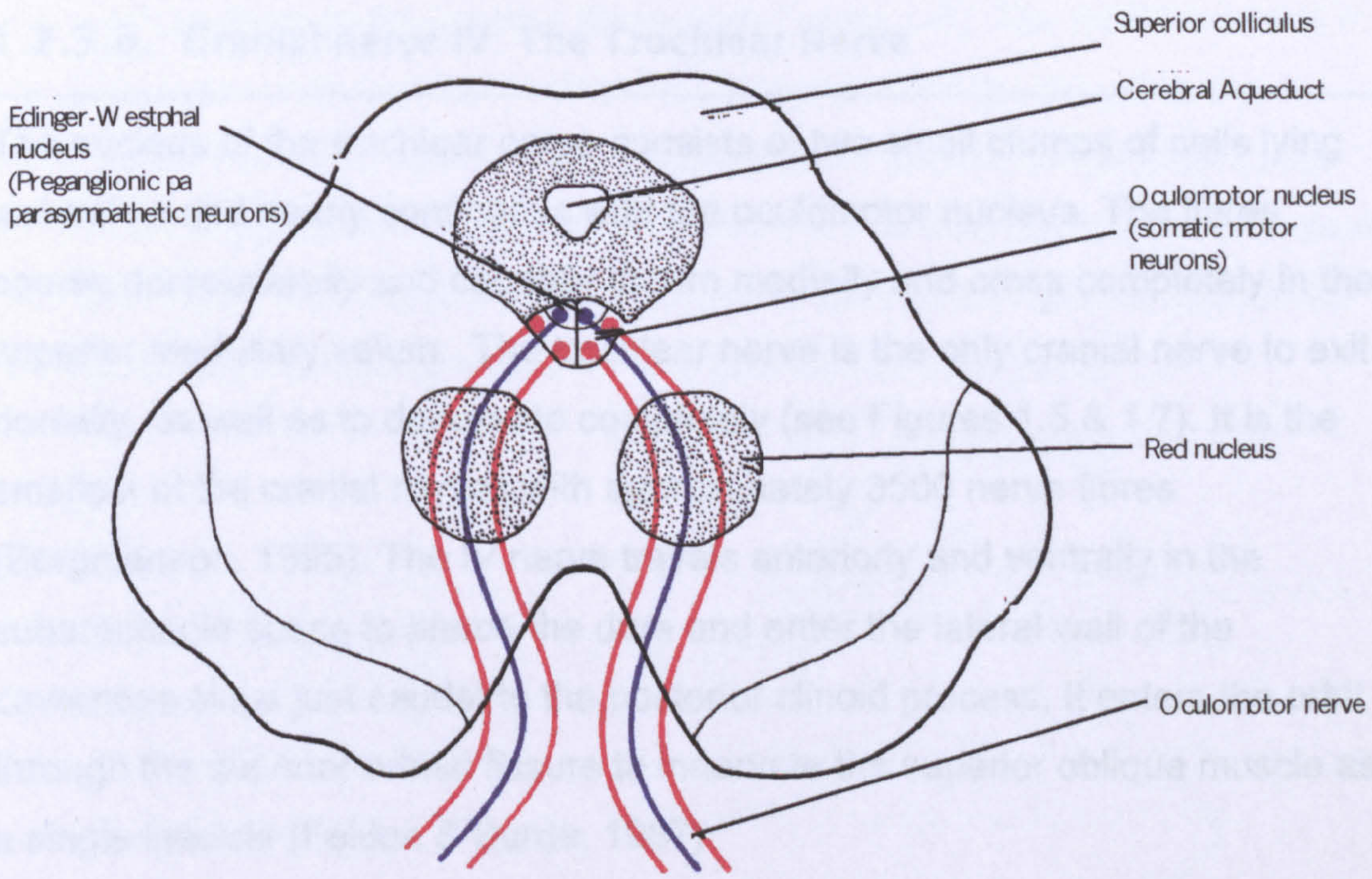


Figure 1.4. Origin of the oculomotor nerve in the midbrain. (Motor neurons are red; preganglionic parasympathetic neurons are blue) (from Barr & Kiernan, 1993).

The III nerve consists of motor, sensory (proprioceptive) and autonomic (parasympathetic) nerve fibres. Mixed with the motor neurons of the oculomotor nucleus are neurons whose axons pass in the medial longitudinal fasiculus to the trochlear (IV) and, especially, the ipsilateral and contralateral abducens (VI) nuclei. These internuclear neurons mediate inhibition of the antagonistic muscles whenever the eyes are moved.

The III nerve passes between the posterior cerebral and superior cerebellar arteries to travel lateral to, but parallel with, the posterior communicating artery of the circle of Willis.

The III nerve enters the cavernous sinus and the orbit through the superior orbital fissure and dividing into superior and inferior divisions that pass through the Annulus of Zinn. Individual nerve branches enter their respective extraocular muscle on the ocular side except the inferior oblique that is entered on its posterior side (Feldon & Burde, 1987; Bergmanson, 1995).

1.2.3.b. Cranial nerve IV: The Trochlear Nerve

The nucleus of the trochlear nerve consists of two small clumps of cells lying just below and nearly continuous with the oculomotor nucleus. The fibres course dorsolaterally and caudally to turn medially and cross completely in the superior medullary velum. The trochlear nerve is the only cranial nerve to exit dorsally, as well as to decussate completely (see Figures 1.5 & 1.7). It is the smallest of the cranial nerves with approximately 3500 nerve fibres (Bergmanson, 1995). The IV nerve travels anteriorly and ventrally in the subarachnoid space to pierce the dura and enter the lateral wall of the cavernous sinus just caudal to the posterior clinoid process. It enters the orbit through the superior orbital fissure to innervate the superior oblique muscle as a single fascicle (Feldon & Burde, 1987).

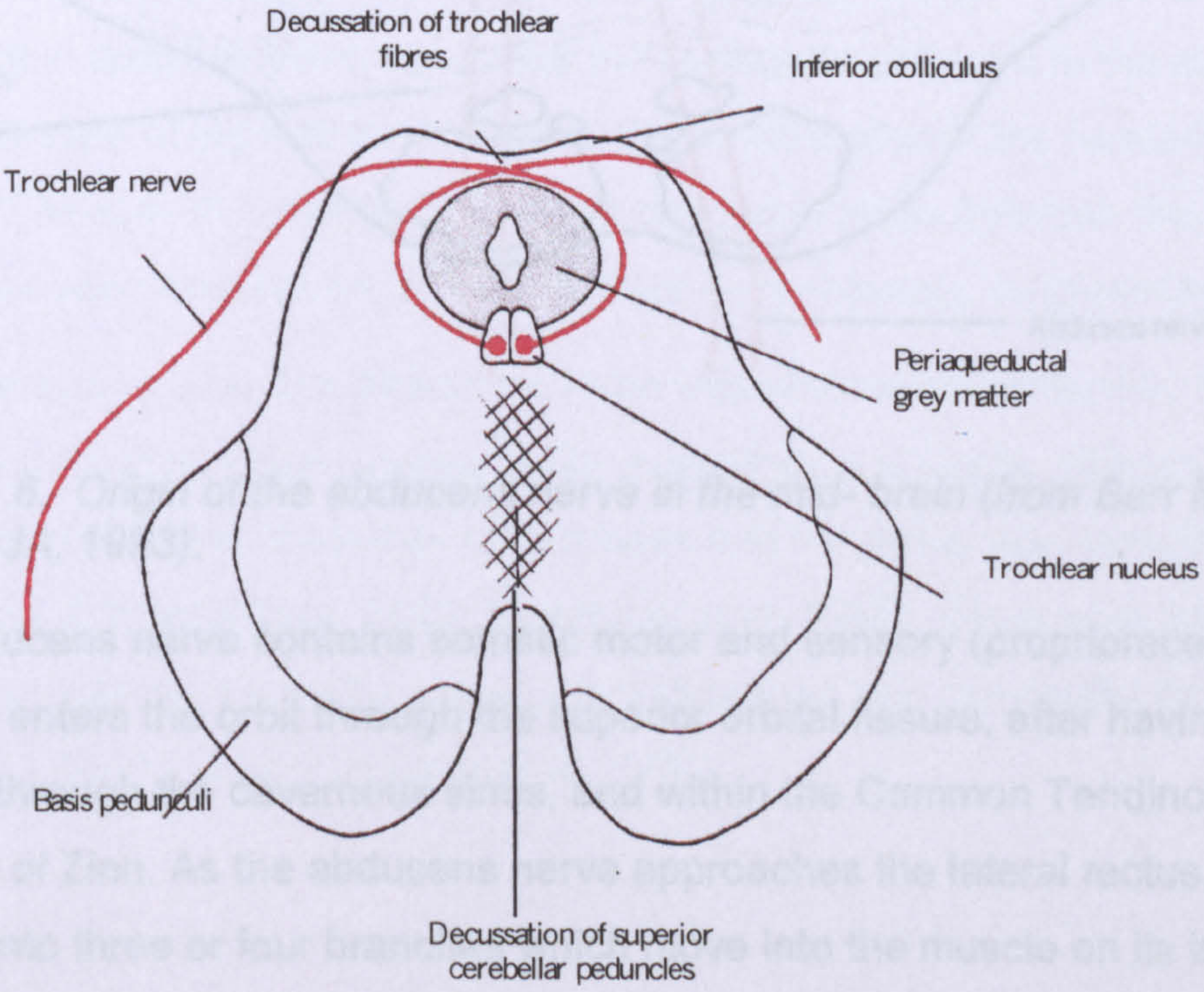


Figure 1.5. Origin of the trochlear nerve in the midbrain (from Barr & Kiernan, 1993).

1.2.3.c. Cranial nerve VI: The Abducens Nerve

The abducens consists of approximately 6,600 nerve fibres that innervate the lateral rectus muscle. It has a small nucleus that is found in the floor of the

4th ventricle close to the median plane (see Figure 1.6). The motor neurons in the abducens nucleus give rise to axons that pass through the pons in a ventrocaudal direction and emerge from the brain stem at the junction of the pons and the pyramid. The abducens nerve nucleus also contains internuclear neurons whose axons travel to the part of the oculomotor nucleus concerned with supplying the medial rectus muscle (Barr & Kiernan, 1993).

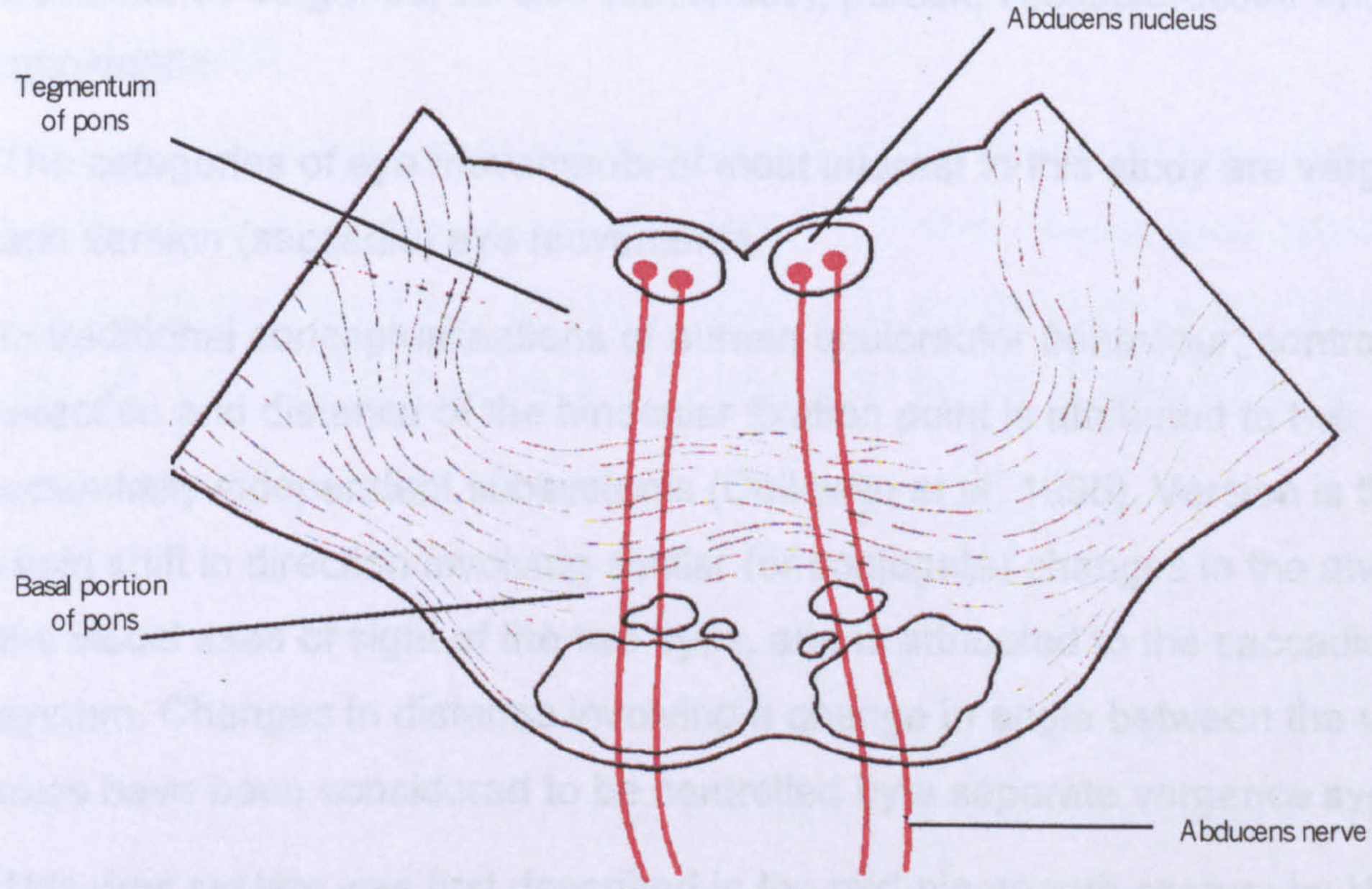


Figure 1.6. Origin of the abducens nerve in the mid- brain (from Barr ML & Kiernan JA, 1993).

The abducens nerve contains somatic motor and sensory (proprioreceptive) fibres. It enters the orbit through the superior orbital fissure, after having passed through the cavernous sinus, and within the Common Tendinous Annulus of Zinn. As the abducens nerve approaches the lateral rectus, it divides into three or four branches which move into the muscle on its internal side facing the eyeball (Bergmanson, 1995).

1.3 Eye Movements

In humans there are five types of eye movement used for the common purpose of bringing the image of an object of interest onto the fovea (foveation) or maintaining foveal fixation. Types of movement may be classified as vergence, version (saccades), pursuit, vestibulo-ocular and optokinetic.

The categories of eye movements of most interest to this study are vergence and version (saccadic) eye movements.

In traditional conceptualisations of human oculomotor behaviour, control of direction and distance of the binocular fixation point is attributed to two essentially independent subsystems (Collewijn et al, 1995). Version is the rapid shift in direction involving similar (or conjugate) changes in the angles of the visual axes of sight of the two eyes, and is attributed to the saccadic system. Changes in distance involving a change in angle between the visual axes have been considered to be controlled by a separate vergence system.

This dual system was first described in the mid-nineteenth century by Hering and the concept has been supported by experiments showing different characteristics in the properties of saccades and vergence eye movements (Rashbass & Westheimer, 1961; Semmlow et al, 1998). Other work has suggested that there may be closer interaction than was previously thought (Kenyon & Stark, 1983; Collewijn et al, 1995).

1.3.1 Saccades

The term for these rapid eye movements is derived from the French *saquer* meaning to "pull" and refers to the jerk of a horse's head when the reins are applied (Troost & Dell'Osso, 1979).

1.3.1.a. Saccades: Neural control

Saccades may occur reflexly or volitionally. They are generated in the frontal eye field (FEF areas 8, 6 and adjacent supplementary eye fields), located anterior to the motor cortex, in response to visual auditory, tactile and other exteroreceptive stimuli via cortical areas with visual associations. Saccades are centrally instigated when volitional, in darkness, or during sleep (REM) (Ruskell, 1998).

Figure 1.7 illustrates some of the pathways for the control of eye movements by the cerebral cortex and superior colliculus. There are separate pathways for horizontal and vertical eye movements with a central integrator governing the two. The pathways for horizontal saccades are illustrated in Figure 1.8.

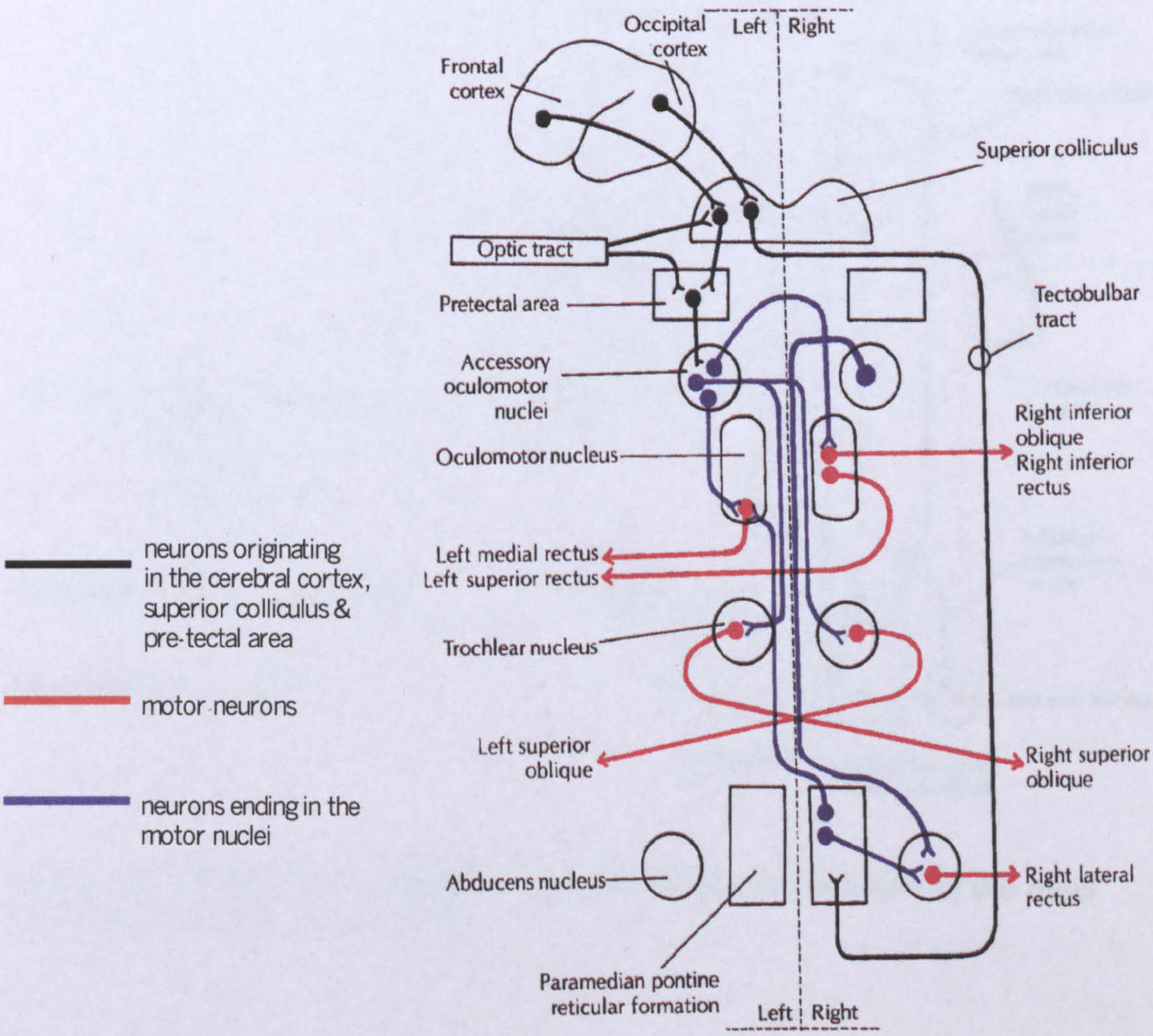


Figure 1.7. Some of the pathways for the control of eye movements by the cerebral cortex and superior colliculus (from Barr ML & Kiernan, 1993).

They start at area 8 and proceeds via the internal capsule to the superior colliculus and on to the paramedian pontine reticular formation (PPRF) which serves as a centre for lateral gaze. The PPRF sends fibres to the ipsilateral abducens nucleus and, through the media longitudinal fasiculus, to those cells of the contralateral oculomotor nucleus that supply the medial rectus muscle. The actions of the medial and lateral recti are thereby co-ordinated in horizontal movements of the eyes (Barr & Kiernan, 1993).

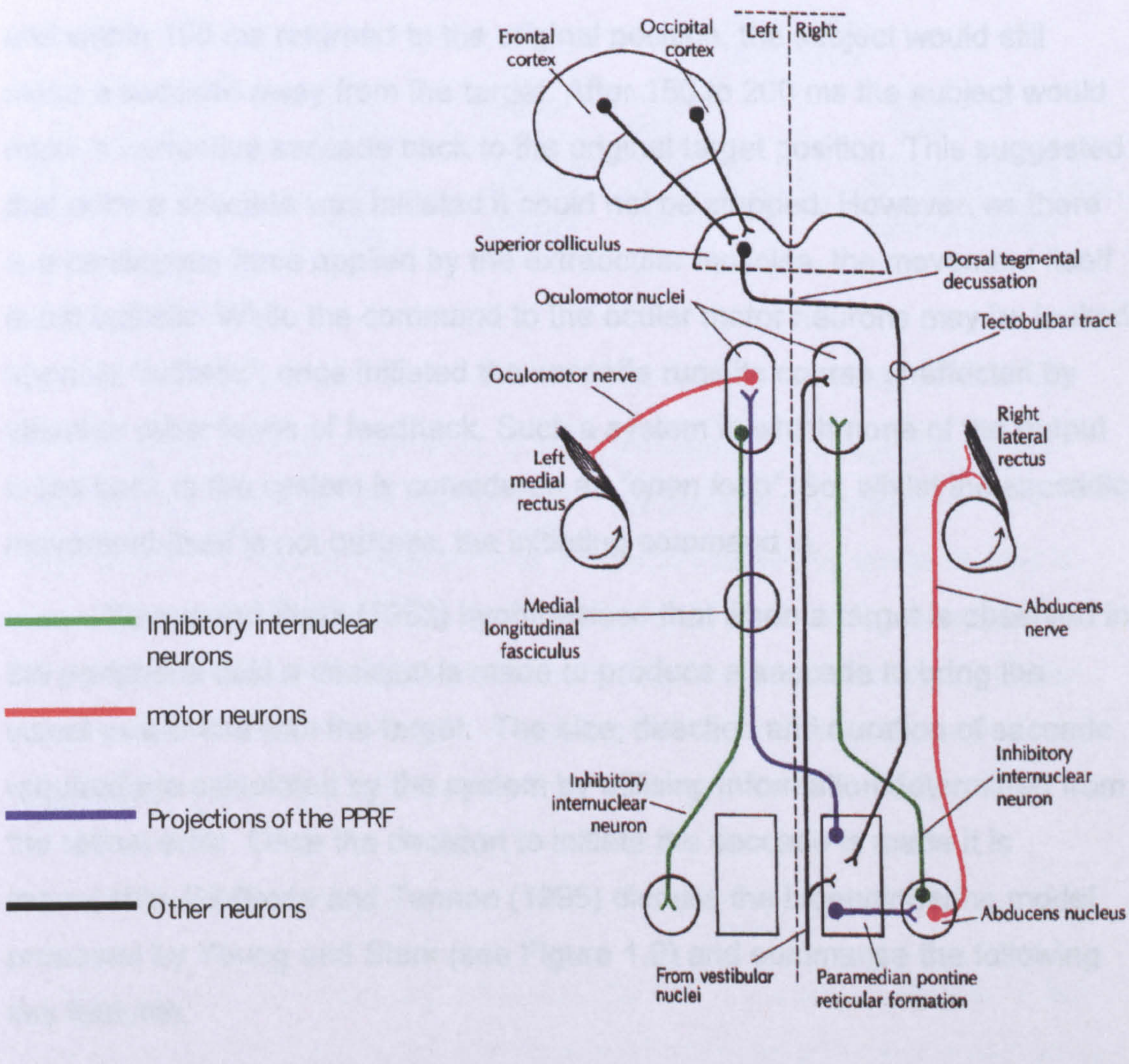


Figure 1.8. Pathways involved in conjugate lateral movements of the eyes (from Barr ML & Kiernan, 1993).

1.3.1.b. Saccades: Characteristics

Bahill & Stark (1979) describe saccades as accurate, high velocity, non-ballistic eye movements used to foveate objects of interest. This needs some explanation because it may also be argued that such movements are essentially ballistic in nature in that once a saccade has been launched it cannot be influenced by any visual feedback unless it is pathologically slow (Zee et al, 1976).

Westheimer (1954) reported that when a target was moved to a new position and within 100 ms returned to the original position, the subject would still make a saccade away from the target. After 150 to 200 ms the subject would make a corrective saccade back to the original target position. This suggested that once a saccade was initiated it could not be stopped. However, as there is a continuous force applied by the extraocular muscles, the movement itself is not ballistic. While the command to the ocular motor neurons may be looked upon as "ballistic", once initiated the saccade runs its course unaffected by visual or other forms of feedback. Such a system in which none of the output is fed back to the system is considered as *"open loop"*. So, whilst the saccadic movement itself is not ballistic, the initiating command is.

Young and Stark (1963) hypothesised that when a target is observed in the peripheral field a decision is made to produce a saccade to bring the visual axis in line with the target. The size, direction and duration of saccade required are calculated by the system by utilising information determined from the retinal error. Once the decision to initiate the saccade is made it is irrevocable. Ciuffreda and Tannen (1995) discuss the bioengineering model proposed by Young and Stark (see Figure 1.9) and summarise the following key features:

- the difference between desired gaze angle and actual eye angle (retinal error) is sampled by a motor impulse modulator at 200 ms intervals, an interval being the mean saccadic refractory period.
- the sampling is synchronised to occur with the onset of any target movement (assuming that no saccade occurred within the prior 200 ms interval) and as noted by Westheimer (1954), target changes

that occur between samples are not processed until the next sampling period.

- the information is then processed to generate a corrective saccade eye movement one latency period later, which brings the eye to a position sampled 200 ms earlier.
- position errors of less than 0.5 degrees are generally not corrected. This is because they lie within an effective "foveal dead zone" over which small position errors are tolerated.

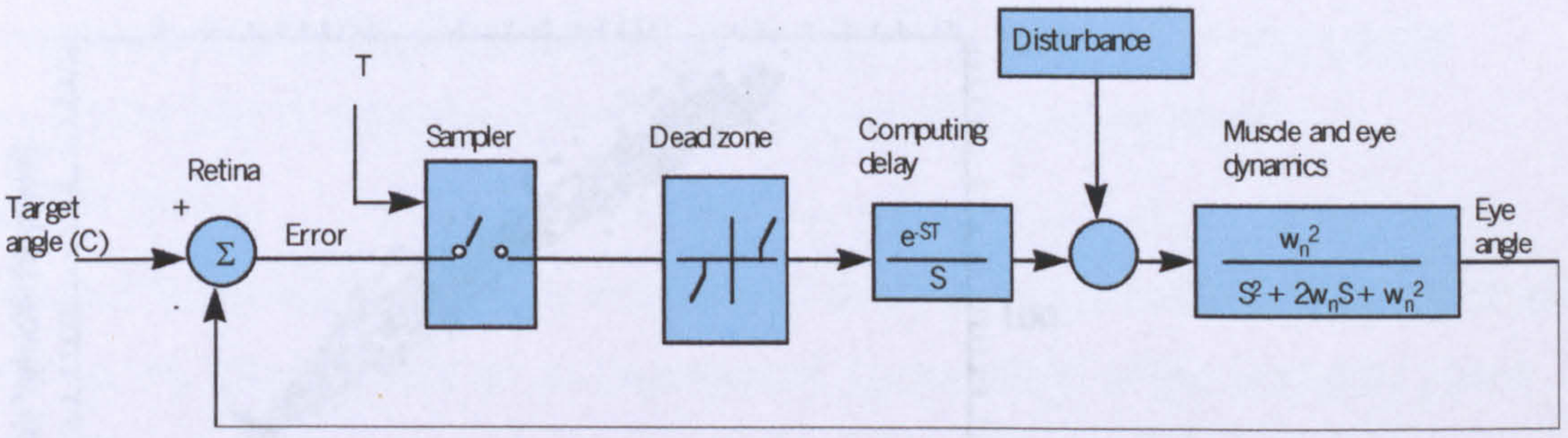


Figure 1.9. Sampled-data saccadic control model (from Young LR, 1981).

Saccades are neurally generated by the combination of a high-frequency pulse and a much lower-frequency step. The pulse is necessary for overcoming the viscous resistance of the globe and orbital contents and is responsible for moving the eye rapidly to the new position. The step is necessary for overcoming the elastic restoring forces of the eye and orbital contents and is responsible for maintaining the eye in this new position (Ciuffreda and Tannen, 1995).

1.3.1.c. Saccades: Velocity

Velocity reaches an upper limit of about 700 ° s^{-1} for a large saccade of 80° (Boghen et al, 1974) and thereafter any increase in amplitude is obtained by an increase in saccadic duration. There are differences in velocity for different directions of movement (Collewijn et al, 1988). Centripetal saccades are saccades from the periphery to the primary position and these show a higher peak velocity than centrifugal saccades. Abducting saccades show a higher peak velocity than adducting saccades. It is worth noting that recovery

saccadic eye movements following the cover test may be described as being centripetal.

Another characteristic of saccades, known as the *main sequence* (Bahill et al, 1975a) is that the peak velocity is proportional to the size of the eye movement and this can be extended to include saccade duration, and saccade peak acceleration and deceleration. Figure 1.10 illustrates these relationships. For young normal subjects, as saccade amplitude increases the correlated saccade duration, peak velocity and peak acceleration increase.

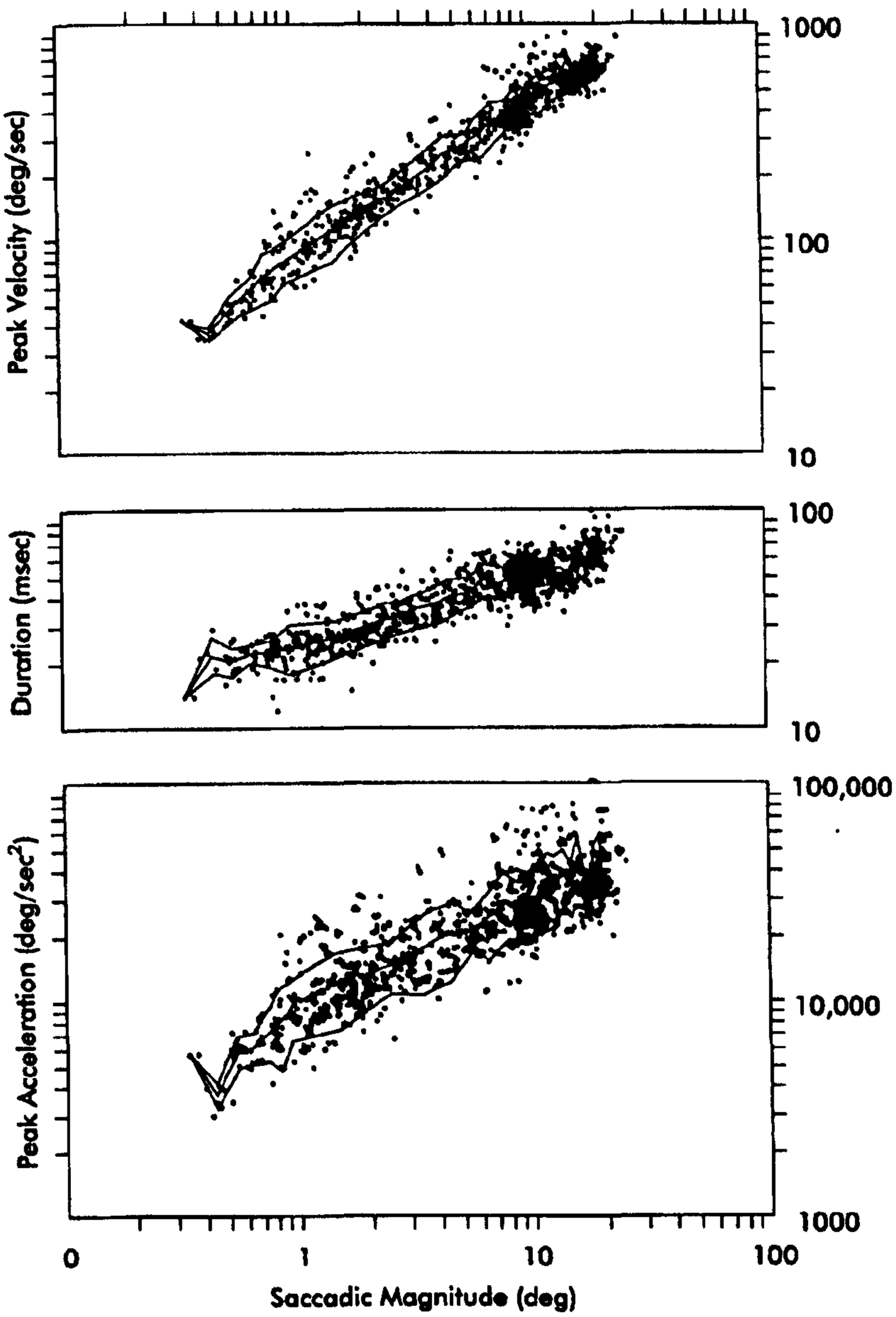


Figure 1.10. Saccadic main sequence diagrams showing peak velocity, duration, and the first peak accelerations as functions of saccadic eye movements of 13 subjects with normal vision (from Bahill et al, 1981).

1.3.1.d. Saccades: Amplitude

The ideal ocular motor re-fixation response is a single eye movement that rapidly and accurately reaches the target and abruptly stops. About 85% of all naturally occurring saccades are less than 15 degrees in amplitude (Bahill et al 1975b).

A saccadic re-fixation may be either *normometric*, that is the correct amplitude to gain re-fixation, or, *dysmetric*, that is an inaccurate eye movement (Bahill & Troost, 1979). A normometric saccade consists of a single, accurate movement having appropriate gain and dynamics. The underlying neural controller signal consists of a single, precisely matched pulse-step combination.

Normal individuals frequently show small degrees of saccadic dysmetria. Most commonly this appears as an undershoot (*hypometria*) of the target by about 10 per cent of what should have been the full amplitude of the saccade (Becker & Fuchs, 1969). The error may increase in older subjects and with fatigue (Bahill and Stark, 1975). The latter is of interest in that one of the anecdotal observations reported during the clinical cover-uncover test, and something that will be further discussed in this thesis, is that patients with a poorly compensated phoria may make more than one eye movement to regain binocular fixation. Subjects manifesting a poorly compensated (or associated) phoria are said to be more likely to complain of asthenopia or visual fatigue (Yekta et al,1987).

When normal subjects undershoot the target, they usually make a corrective saccade with a latency of 100 to 130 ms less than normal (Becker & Fuchs, 1969). Such corrective movements can occur even when the target is extinguished before the initial saccade is completed and this suggests that a non-visual signal can provide information about whether the initial movement is to be accurate or requires an additional movement to be instituted. This information might be proprioceptive using afferent signals from the extraocular muscles or by means of a mechanism for the monitoring of efferent ocular motor commands.

1.3.1.e. Saccades: Latency

Typical values for saccadic reaction time are of the order of 180 to 220 ms measured from target appearance (Baloh and Honrubia, 1976) although, as has already been mentioned, corrective saccades may have a shorter latency. Factors that contribute to this delay include:

Afferent or visual neurosensory delay of approximately 50 ms, that includes neural transmission time from the retina to visual cortex to high-level centres involved in the saccadic decision-making process;

Efferent or neuromotor delay of approximately 30 ms that includes neural transmission time within higher-level centres involved in the saccadic decision- making process as well as lower-level signal processing within the midbrain;

Computational delay of approximately 50 ms, that, along with the first two factors listed, may be regarded as non-cognitive; and

Decision-making processing delay of at least 50 ms for the brain to decide whether or where to change gaze in the field, thus involving higher-level cognitive processing (Becker & Jurgens, 1979). Table 1.2 lists some factors that affect saccadic latency.

Increase	Decrease
Very small (< 30 min) or large (> 10 degree target eccentricities	Target predictability
Increased target uncertainty	Increased target luminance
Increased target complexity	Increased target contrast
Increased age in adulthood	Heightened attention
Inability to disengage attention	Forewarning period
Decreased motivation	Fixation disengagement (gap effect)

Table 1.2. Factors that affect saccadic latency (adapted from Ciuffreda & Tannen, 1995).

I. Procrastination

Whilst a small saccade may last only 20ms there may have been a delay of some 200ms before the eye movement starts. Carpenter (1981) argues that it is unlikely that more than 12 ms of this latent period is due to delays in the final common oculomotor pathways, and, in good photopic conditions, the purely visual part of the delay is approximately 50 ms at most. Although nerve conduction is slow, this cannot explain the long latencies often measured for saccades. Carpenter concludes that the brain is deliberately delaying a process that could be executed much more rapidly and calls this *procrastination.* He argues that saccades are a mixed blessing in that while they enable the eye to bring a new target onto the fovea, vision is much reduced during movement.

II. Fixation disengagement and express saccades

Saslow (1967) showed that reaction time decreased to about 140 ms when the stimulus at fixation is extinguished before target onset (gap condition) than when it is extinguished after target onset (overlap condition). Similar findings have been reported by Becker (1972), Cohen & Ross (1977), Iwasaki (1990), Mayfrank et al (1986), Reulen (1984), Ross & Ross (1980), and Ross & Ross (1981). These have been termed *express saccades.*

Fischer & Ramsperger (1984) also described these extremely fast reaction times (short latency) for saccades under gap conditions. Human subjects were asked to execute a saccade from a central fixation point to a peripheral target at the time of onset. When the fixation point is turned off some time (≈ 200 ms) before target onset such that there is a gap when subjects see nothing, the distribution of their saccadic reaction times is bimodal with one narrow peak around 100 ms (express saccades) and another peak around 150 ms (regular saccades) measured from the onset of the target. Fischer & Boch (1983) have reported even faster reaction times of the order of 75 ms.

The relative number of express saccades reportedly decreases for subjects over the age of 50 years (Mayfrank et al, 1986).

Mayfrank et al (1986) examined the effect of luminance of the fixation point on the percentage of express saccades and showed an increasing number of express saccades with increasing luminance from nil at threshold to the maximum percentage at 0.8 log units above threshold.

It has been suggested that this gap effect may be due to facilitation of attentional disengagement before a saccade (Braun & Breitmeyer, 1988; Fischer & Breitmeyer, 1987; Fischer & Weber, 1993). However, Tam & Stelmach, (1993) compared and examined together the contribution of the attentional and ocular sampling systems to saccadic latencies. Three possibilities were considered: saccadic latencies are determined by the ocular sampling system, by the attentional sampling system, or jointly by both systems. They concluded that ocular and ocular-attentional explanations provided a satisfactory account for their data, but that the attentional explanation on it's own failed to account for the data. Tam & Ono (1994) argue that attentional processes play little or no role in explaining the gap effect and favour an ocular motor processing explanation. Kingstone & Klein, (1993); and Klein et al (1992) appear to support this theory having shown that the gap effect appeared to depend more on whether the foveated stimulus was extinguished than on whether an attended stimulus was extinguished.

Possible pathways for express saccades

Only the collicular pathway may be described as a sub-cortical loop, bypassing the cortex. Whilst this shortest connection was believed to mediate express saccades described in humans, Fischer & Ramsperger (1984) showed that express saccades are abolished without the striate cortex. However, express saccades survive ablations of the Frontal Eye Fields (Schiller et al, 1987). Thus it may be postulated that even the shortest pathway in the generation of visually guided saccades includes the striate cortex and the superior colliculus.

III. Auditory cues

Ross & Ross (1981) investigated the effect of auditory onset and offset stimuli. Their findings suggest that both auditory-onset and auditory-offset warnings facilitated saccadic responses. Unlike the results with visual warnings there was no evidence that auditory-onset warning exerted an interfering effect on saccadic responses when the auditory stimulus onset occurred after target onset.

Nevertheless, the effect of auditory warning requires consideration because the stepper motors used to drive the occluders used for the cover test in this study were not silent.

1.3.1.f. Saccades: Suppression and omission

Despite the eyes travelling at high velocity during a saccade, individuals have the impression or illusion of clear, uninterrupted vision during the movement. This is in part due to an elevation in visual threshold that actually occurs not only during the saccade but also immediately before and after (Zuber & Stark, 1966). This phenomenon is termed *saccadic suppression*. However, suppression cannot explain the phenomenon completely and the work of Matin et al (1972) and Campbell & Wurtz (1978) has shown that a more important factor is that of visual masking or *saccadic omission* which occurs due to the presence of either an immediately preceding or succeeding visual fixation.

1.3.2 Vergence eye movements

Vergence eye movements driven by disparity only are relatively slow, disjunctive movements of the eyes. They are used to track objects moving in depth to maintain a fused and single percept (Schor & Ciuffreda, 1983).

1.3.2.a. Vergence: Neural control

Fusional vergence eye movements are initiated by binocular retinal disparity information. Neurons in the striate and prestriate cortex are capable of eliciting

convergence or divergence via the internal saggital stratum of the corticotectal tract and pretectal olivary nuclei. Decussation occurs to the accessory oculomotor nuclei including the rostral interstitial nucleus of the medial longitudinal fasiculus (RiMLF). Cells in this area show increased activity preceding a vergence eye movement and are non-active prior to version eye movements (Leigh & Zee, 1991; Ruskell, 1998). These cells may project directly to the oculomotor nuclei. Both the abducens (VI) and oculomotor (III) nuclei are active in both version and vergence eye movements (Leigh and Zee, 1991).

The final motorneuronal controller signal is not a simple step of neural discharge but rather a small and broad pulse combined with a step having characteristics appropriate for the relatively slow vergence response (Ciuffreda & Tannen, 1995).

The following three types of midbrain neural cells have been described as being crucial to overall vergence control (Mays et al, 1986; Zee & Levi, 1989):

Vergence burst neurons fire immediately prior to and during the vergence response. It is believed that these cells encode peak vergence velocity and effectively define neurologically the vergence "main sequence" peak velocity/amplitude relationship (Ciuffreda & Tannen, 1995). They probably act as input to a neural integrator.

Vergence tonic neurons probably carry the immediate output of the vergence neural integrator and fire just before a vergence movement. The firing rate is proportional to vergence angle. Tonic convergence cells are much more common than tonic divergence cells (Ciuffreda & Tannen, 1995).

Vergence-burst-tonic neurons probably associated with the combined burst and tonic signals described above and may therefore be the "near response cells" that input directly into the oculomotor neurons (Zhang et al, 1991).

1.3.2.b. Vergence: Classification

Maddox (1893) suggested that there were four components of vergence and Morgan (1980) presents an elegant review of the Maddox classification.

Maddox believed that the total vergence required to bifixate an object in space could be factored into various components. He termed the initial component *"tonic convergence"*. He suggested that if all innervation were to cease, the *anatomical position of rest* of the eye would be one of divergence and this has been shown in cadavers to be of the order of 15 to 25° (Stutterheim, 1934). This divergent resting position has also been confirmed under general anaesthesia (Meyers, 1951) and muscle relaxants such as curare (Drucker et al, 1951).

The anatomical position of rest cannot be determined under normal conditions, and thus, the magnitude of tonic vergence cannot be measured. However, one may deduce that deficient tonic convergence results in exophoria and excessive tonic convergence results in esophoria. The anatomical position of rest may vary throughout life as the ocular structures involved in eye movements change (Abraham, 1951; Abraham, 1964).

I. Tonic vergence

Even in the absence of visual feedback information, the eyes of a conscious subject are moved by tonic vergence from this position of *anatomical rest* to a position that is more convergent and which may be termed the *physiological position of rest.* This more convergent position can be determined by the estimation of the distance phoria. However, the period of time for which the eyes are allowed to dissociate will have a bearing on this measurement (Marlow, 1924; Peli & McCormack, 1983; Barnard & Thomson, 1995a; Rosenfield et al, 1997a).

Maddox (1893) identified two non-visual causes of tonic convergence. The first is the tonus exhibited by living striated muscle. The second is what he termed "converging innervation" and what would be described today as *the central control mechanism of vergence*. This has a tonic activity of its own and is also affected by sleep, drowsiness, alcohol (Adams, 1978; Hogan and Linfield, 1983; Miller et al, 1986) anaesthetic agents, and ultimately by death (Morgan, 1980).

II. Disparity vergence

What Maddox defined as "reflex convergence" or the "fusional supplement" has been termed *fusional vergence* or, in more recent years, *disparity vergence* (Stark et al, 1980). Disparity driven vergence is induced when there is a difference between similar targets seen by the two eyes. Although vergence can be elicited by presenting disparate dissimilar targets to the two eyes, fusion is not achieved (Mitchell, 1970).

III. Accommodative vergence

The third category of vergence may be described as *accommodative vergence.* During near fixation accommodative effort will invoke a vergence response that will correlate with the amplitude of accommodative effort and will vary between subjects. Kenyon et al (1980a, 1980b, and 1980c) has shown the presence of accommodative vergence in the absence of disparity vergence.

IV. Proximal vergence

What Maddox described as "psychic" vergence is now termed *proximal vergence*. Proximal vergence refers to the change in vergence angle of the eyes caused solely by the perceived nearness of an object (Ciuffreda & Tannen, 1995).

Stark et al (1980) argued that both proximal and tonic vergence are rather minor phenomena.

V. Interactions between types of vergence

Implicit in the Maddox hierarchy are two assumptions (Semmlow & Hung, 1980). Firstly, accommodative convergence adds algebraically to other controller systems to produce a binocular response. Secondly, the value of accommodative convergence during binocular fixation is the same as that observed monocularly during dissociation. Semmlow & Hung (1980) argue that the shape of the zone of clear, single vision provides support for this assumption. Assuming that the maximum and minimum attainable

magnitude of the fusional component is a constant, or at least independent of accommodative stimulus, then the vergence limits may be explained as the sum of this component and the monocular phoria. Further support may be found in the fixation disparity measurements of Martens and Ogle (1959). Their findings that binocular AC/A was similar to monocular AC/A suggest the presence of an active and additive accommodative convergence component of binocular vergence. Semmlow & Venkiteswaran (1976) showed that step-like changes in accommodative stimulation will produce small binocular accommodative convergence movements. Semmlow & Wetzel (1980) found that accommodative stimulation enhanced the dynamics of the vergence response and the faster response could be explained by a simple addition of fusional and accommodative convergence dynamics.

Stark et al (1980) argue that disparity vergence is a complete example of a closed loop visual feedback system with well defined binocular stimulus and response, be it convergence or divergence. Added to this disparity vergence is the accommodative vergence synkinesis. Although disparity vergence is the primary and prominent component of ordinary vergence, accommodative vergence is also co-acting and may dominate the response in clinical conditions such as strabismus and amblyopia when disparity vergence may be absent. They suggest that the general term "fusional vergence" should be superseded by the term "*disparity vergence*" to describe the vergence component caused by a disparity stimulus.

1.3.2.c. Vergence: Characteristics

I. Velocity

The peak velocity of vergence movements has been shown to increase in proportion to its response amplitude. This peak velocity versus amplitude of vergence is known as the *main sequence*, a term also used to describe the characteristics of saccadic eye movements, and has been described for disparity-only stimulation (Bahill et al, 1975a; Bahill & Stark, 1979). Others have shown that the main sequence relationship described by Bahill et al

holds with the addition of blur and proximity to the viewing conditions (Semmlow et al, 1986; Semmlow et al, 1993; Hung et al, 1994) (see Figure 1.11).

The classical investigations of Westheimer (1954) and Rashbass & Westheimer (1961) suggested that disparity driven vergence velocities were much slower than saccadic peak velocities. Saccadic peak velocities were found to be in the order of hundreds of degrees/second compared to vergence velocities which were of the order of only tens of degrees/second. This difference was used as evidence for separate neuro-physiological systems controlling vergence and saccadic eye movements.

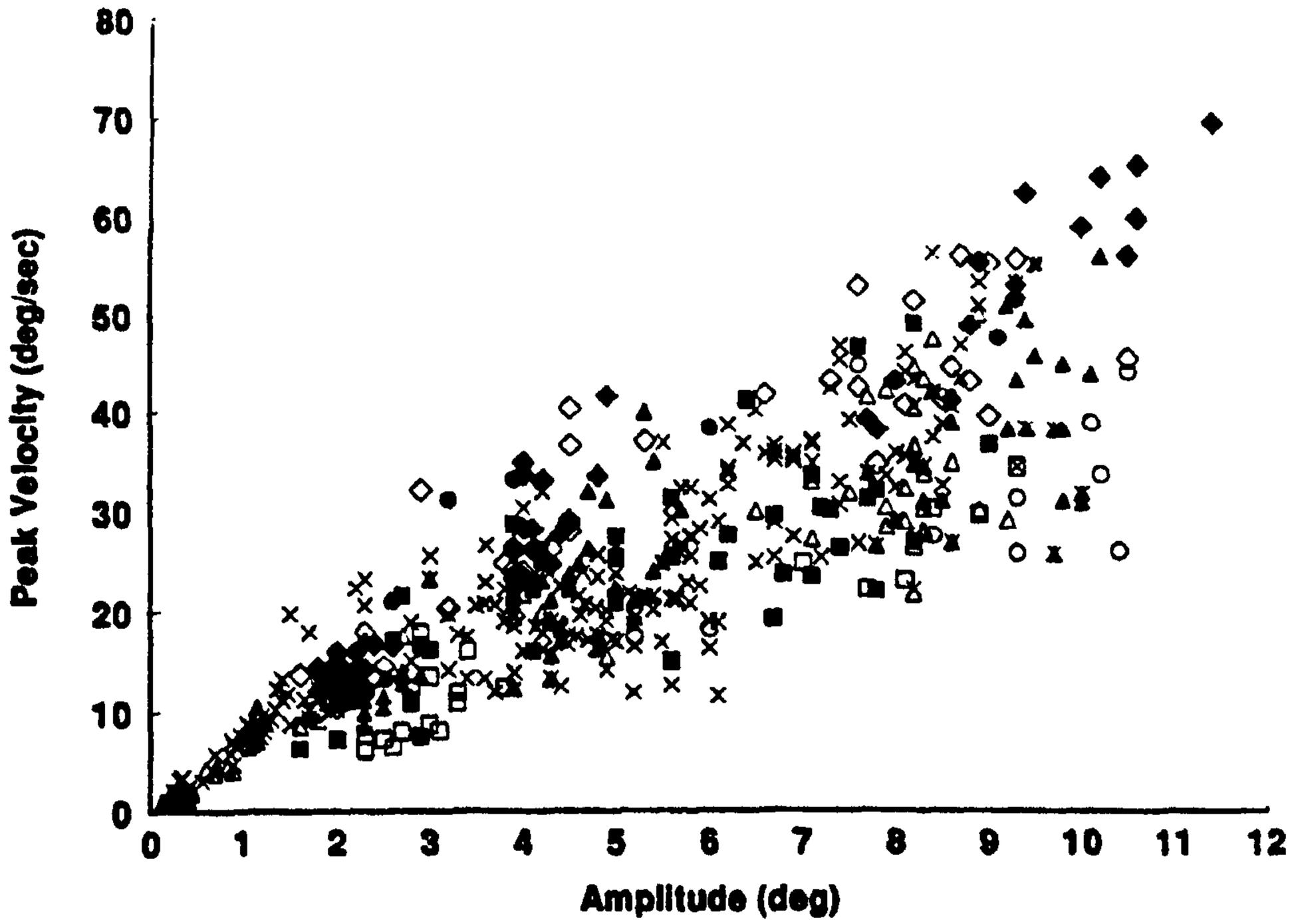


Figure 1.11. Main sequence disparity vergence responses for a variety of stimulus conditions in one subject. Filled symbols, Instrument space environment (that is, disparity stimulation only); open symbols, free-space environment (that is, disparity plus blur plus proximity; X disparity-only standard step stimulation in instrument space (from Hung GK, Ciuffreda KJ, Semmlow JL, Hornig JL, 1994).

Some investigators have challenged these results, reporting faster velocities than expected (Enright, 1984; Erkelens et al, 1989a; Erkelens et al, 1989b). Hung et al (1994) discussed possible methodological reasons for this discrepancy but also suggested that the enriched stimulus cues

present under the free space (versus instrument space) viewing environment may be a factor.

Disparity only driven vergence has been shown to produce much slower vergence velocities than those associated with voluntary shifts of gaze. (Rashbass & Westheimer, 1961; Erkelens, 1987). Collewijn et al (1995) argued that these low vergence velocities are not representative for vergence associated with voluntary shifts in gaze. Rather, they are probably typical for corrective vergence movements that serve to eliminate errors in binocular foveation that may occur in between gaze shifts. One might hypothesise that recovery from a clinical cover test may also be a special case producing this type of movement.

Oohira (1993) investigated vergence eye movements associated with and without saccades in both dark and illuminated rooms. Vergence eye movements became faster when associated with saccades. Varying the presence of visual cues for depth perception had no effect on vergence velocity. Oohira also reported that blinking speeded up divergence.

More recently, Collewijn et al (1995) studied the dynamics of version and vergence during voluntary gaze-shifts. They found that pure vergence was almost never observed; divergence, especially was always associated with saccades. Likewise, horizontal saccades were never strictly conjugate but they always contained a transient divergence-convergence sequence.

Erkelens, Steinman & Collewijn (1989b) measured an approximately linear relationship between vergence velocity and vergence amplitude with a slope of the order of 5 degrees/second per degree of vergence. Thus natural vergence shifts of 20 degrees between real targets were found to have peak velocities of the order of 100 degrees/second. This is of a different order of magnitude to that described by Rashbass & Westheimer (1961).

II. *Latency*

It has been reported that convergence responses, in general, have faster dynamics than the divergence responses (Zuber & Stark, 1968; Mitchell,

1970). Westheimer and Mitchell (1956) found vergence response latencies of approximately 200 ms. Krishnan et al (1973) reported fusional vergence latencies of between 130 and 250 ms for temporally unpredictable stimuli. They also found a prediction operator in the system, that, under certain conditions, produced very short latencies (as short as 10 ms). However, unlike previous reports, they found that divergence latencies tended to be shorter than convergence latencies. Tam & Ono (1994) measured latencies of vergence eye movements to a target that was positioned at a different depth from an initially fixated stimulus in gap and overlap conditions. As with saccades, vergence latencies were shorter under gap conditions. Barnard & Thomson (1995a) reported very short latencies, with exophoric recovery latencies tending to be shorter than esophoric recoveries, which contradicts the findings of Krishnan et al (1973). However, Barnard & Thomson presented combined vergence and saccadic data. Schoessler (1980) reported vergence latencies that were larger for squinters than non-squinters and these were especially long for those strabismic patients with anomalous correspondence.

1.3.2.d. Factors affecting vergence

I. *The effect of fixation target characteristics on vergence*

Marr and Poggio (1979) and Mowforth et al (1981) studied the effect of target spatial frequency information on initiation of vergence eye movements. Marr and Poggio suggested that vergence eye movements to large disparities are initiated by low spatial information with higher spatial frequency being more relevant to smaller disparities. Mowforth et al (1981) found that vergence could be initiated to larger disparities with stimuli of higher spatial frequencies than found by Marr and Poggio.

There have been a number of studies investigating the effect of central and eccentric stimuli.

It has long been held that stimuli in the peripheral visual field may act as a stronger stimulus than more central targets (Burian, 1939). Burian reported

that targets as small as 1° positioned 12° from the fovea could initiate a fusional movement. Sullivan & Kurtesz (1979) measured motor and sensory responses to simultaneously presented peripheral and central stimuli and found that there was a fusional preference to peripheral fusion targets. Nauheim (1957) compared negative fusional amplitudes with three types of stimuli: a central fixation point; a central fixation point the same size as above surrounded by an annulus subtending approximately 5.5°; vertical lines subtending 1.2° and positioned approximately 5.5° from the central fixation point. This study found the annulus to be the most effective stimulus to fusion with the central fixation target alone giving the poorest fusional amplitudes. However, in their study Ludvigh et al (1965) investigated the relative effects of presenting stimuli of identical parameters at the fovea and peripheral to the fovea and concluded that for their targets central fusion stimuli were stronger than peripheral stimuli. Cooper et al (1992) also investigated the role of various stimulus parameters that influence motor fusion responses for peripheral as compared to central fusional stimuli. Their results indicated that a central stimulus equal in size to a peripheral fusion stimulus dominated the fusional response independent of the amount of retinal eccentricity of the peripheral target. A second experiment indicated that the central stimulus dominated even when the peripheral stimulus was larger. However, when the peripheral stimulus was changed in shape to an annulus that surrounded the central stimulus, the peripheral stimulus was always stronger. Kertesz & Hampton (1981) reported that fusional vergence response is often reduced in the absence of central fusion stimulus.

Georgievski (1994) investigated the effects of masking of central and peripheral binocular fields on horizontal fusional amplitudes and on the duration of jump-fusional disparity convergence movements. The method of this study was novel and warrants some discussion. The fixation target used for the experiment was a 6/12 letter on a white background superimposed on a full sized Bailey-Lovie LogMAR chart at a distance of 3 m from the subject. Fusional amplitudes and jump convergence responses were recorded under full field, peripheral field only and central field only

whilst the eye movements were monitored using a limbal reflection infra-red eye movement recorder. Peripheral masking was achieved using a thin tube providing 8 degrees of central binocular field. The manner in which central masking was obtained is open to some comment. This was obtained by bleaching the central 8 degrees of retina using a Visuoscope the target of which was fixated for 30 seconds prior to each measurement. The luminance of the ophthalmoscope was not stipulated. Margrain (1996) has suggested that for a 6/12 high contrast letter, recovery from the bleaching will be occurring after about 4 seconds, with the field being almost fully recovered after about 10 seconds. This contradicts Georgievski who states that complete visual re-adaptation did not occur for at least several minutes. He does not state how long each measurement took.

Georgievski found that, under central stimulation, the fusional amplitudes were significantly lower than those under peripheral stimulation. However, in view of the criticisms above it is possible that the 'peripheral field only' measurements were not valid as such in that the central field may have been recovering sooner than the author postulated.

II. The effect of drugs on vergence

A number of studies have shown that pharmacological agents including alcohol have an effect on the vergence system.

Ethanol has been shown to produce an esophoric shift for distance and either an exophoric shift or a minimal change for near vision. Both results may be described as a shift towards the tonic vergence position (Powell, 1938; Colson, 1940; Brecher et al, 1955; Masters, 1964; Hogan & Linfield, 1983; Hogan & Gilmartin, 1985).

The time taken to attain fusion for far and near targets has been shown to increase as a monotonic function of intoxication (Brecher et al, 1955; Masters, 1964; Miller, 1991).

1.3.2.e. Control of disparity vergence

A number of models have been proposed to describe disparity vergence eye movement control. Many of the models include a leaky integrator in the vergence control system (Zuber & Stark,1968; Krishnan & Stark, 1977).

It had been suggested that, unlike saccadic eye movements, vergence movements are controlled by *continuous visual feedback* (i.e. it is a closed-loop system) (Toates, 1974; Zuber & Stark, 1968; Krishnan & Stark, 1977). However, more recently it has been suggested that a fully *pre-programmed or open-loop initial control strategy* is involved (Hung et al, 1986; Semmlow et al, 1986). An open-loop system is one in which none of the output is fed back to the input.

Semmlow et al (1993) investigated the stimulus features that elicit or influence the ocular motor response to disparity stimulation and their findings support a theory of a combination of both open and closed-loop control. They examined the convergence response to a step, a step followed by a target disappearance, and a pulse followed by target disappearance. The target was a thin vertical bright line (0.25° in width) and either 2 or 10° in height, produced on an oscilloscope screen presented haploscopically against an otherwise totally dark background. A pinhole arrangement eliminated blur-driven accommodation and the authors stated that care was taken to eliminate other cues to vergence. The stimuli, having different amplitudes (1,2, 4 and 8°) and disappearance times (50, 100, and 200 ms), were selected randomly and presented along with occasional divergent stimuli to minimise prediction and voluntary vergence. They reported that the dynamic characteristics of the initial portion of the response were essentially the same, even when the target disappeared before the movement took place. The magnitude of the initial response depended on the stimulus amplitude, but was not influenced by either stimulus duration or target height. The initial response appeared to be active over a well-defined time period of about 200 ms, after which the response appeared to be mediated by a visually-guided control component. The authors conclude that the transient portion of the oculomotor response to a disparity is dominated by an open-loop response that lasts at least 200 ms

and is likely to be pre-programmed. The motor programme of this initial response is strongly influenced by the stimulus amplitude, but is relatively insensitive to the other stimulus features tested.

Jones (1980) suggested that there was an interdependence between fusion-initiating and fusion-sustaining components during fusional vergence. The results of Semmlow et al (1993) also support a *dual-mode theory of vergence control* in which an initial pre-programmed (open-loop) control component is followed by a feedback (closed-loop) controlled component which reduces any remaining disparity.

Interestingly, it was Hoffmann & Bielschowsky (1900), cited by Schor & Ciuffreda (1983), who perhaps first alluded to a dual-mode system when in 1900 they stated " the important factor concerning fusional movements is not the movement that leads to sensory fusion per se but the prolonged tonic fundamental innervation of the eye muscles that is adapted for the condition of single vision."

These two components to disparity vergence may also be termed ***fast fusional vergence*** and ***slow fusional vergence***. Fast fusional vergence is responsible for rapidly altering the degree of vergence. The slow fusional vergence sustains vergence for longer periods of time.

Fast fusional vergence is capable of reducing retinal image disparity within one second (Rashbass & Westheimer,1961) to less than 28 seconds of arc (Riggs & Niehl,1960; Hebbard, 1962). Hung et al (1986) described the fast system as a leaky integrator controller with a time constant of approximately 10 seconds. This means that the time for the exponential response to a step input to reach 63% of its final value is approximately ten seconds. The time course of relaxation for the fast mechanism has been described as being approximately 10-15s (Ludvigh et al, 1964; Krishnan & Stark,1977). However, Rosenfield et al (1997) in their discussion stated that that fast vergence will dissipate within 1-2 seconds.

For sustained vergence gaze, the ***slow fusional vergence system***, with a time constant of greater than a minute, gradually subsumes responsibility. The motor output of the fast system controller inputs and drives this slow

"adaptive" system which in turn inputs into the fast system controller producing a cycle. As the slow system continues to be innervated, the fast system innervation decays, with the sum of the slow and fast system outputs being constant in order to sustain binocular fixation. As the slow system takes over responsibility for the output it reduces 'stress' on the fast system and gradually resets the motor zero position (the phoria and fixation disparity) leading to what is termed vergence (or prism) adaptation (Schor, 1979a; Schor, 1980; Ciuffreda & Tannen, 1995).

The time course for decay of slow fusional vergence can be extremely prolonged with a long-decay time constant reported to be greater than 30 seconds (Schor, 1979b), minutes, hours or even days (Marlow,1924; Ellerbrock,1950; Ogle & Prangen,1953; Rosenfield et al, 1997).

1.3.2.f. Models of the vergence system

A number of models have been proposed to 'explain' the vergence system. Schor (1980) described fusional vergence eye movements as being controlled by a comnination of fast-acting and slow-acting mechanisms.

Hung & Semmlow (1980) developed a dual-inter-active model of the vergence system and adaptive loops have been added by Hung (1992). No-one to date has successfully incorporated all four vergence components of vergence into a physiologically sound, quantitatively based static or dynamic model (Ciuffreda & Tannen, 1995). However they argue that that whilst proximal vergence may have considerable influence on the static vergence system under open-loop viewing conditions, it becomes negligible under normal closed-loop viewing conditions in which disparity and blur predominate. The model described by Hung (1992) shown in Figure 1.12, has been described by Ciuffreda and Tannen (1995) as being a useful descriptive static model.

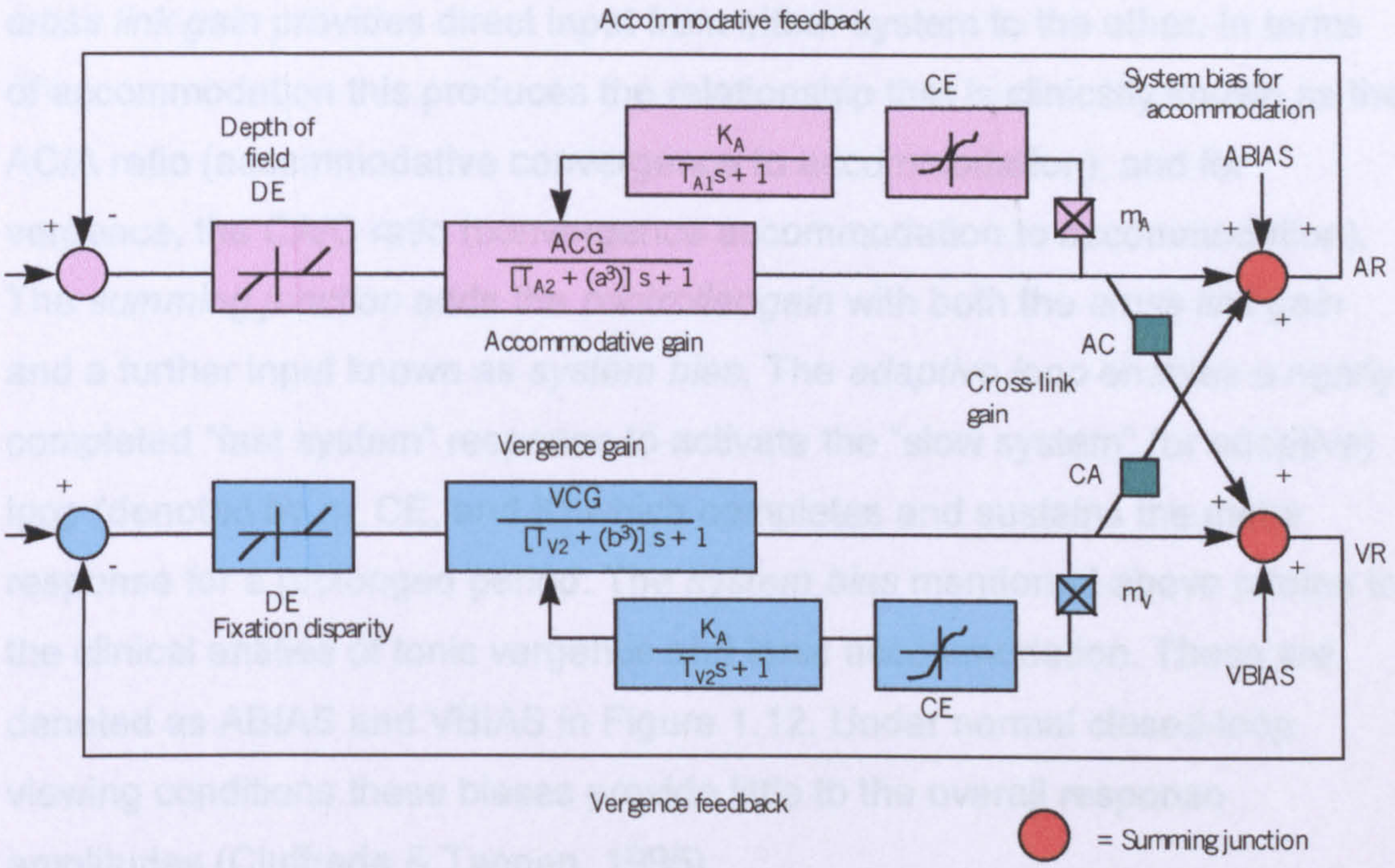


Figure 1.12. Static model of accommodation and vergence. See text for details. (from Hung 1992).

Figure 1.12 shows the accommodative and disparity loops having similar components and the discussion that follows is adapted from Hung (1992) and Ciuffreda and Tannen (1995). Time delays (latencies) of the vergence (≈ 200 ms) (Westheimer & Mitchell, 1956) and accommodation (≈ 350 ms) (Fincham, 1961) loops were neglected in this model.

The input to the system is a stimulus change for either disparity vergence (VS) or accommodation (AS) summing with a negative feedback value from the *output* (VR & AR respectively). The difference between the input and the negative feedback values represents the initial system error. A *dead space elemen*t (DE) allows for a small neuro-sensory error to exist without producing adverse perceptual consequences. In vergence terms this may be correlated with a fixation disparity and in terms of accommodation, with depth of field. Such errors are tolerated and would not produce a corrective input. The final system error is the initial system error minus the dead space element. The *controller gain* represents the open-loop gain of either accommodation (ACG) or disparity vergence (VCG). These gain elements multiply the respective final system errors and the results input to three further components: the *cross-link gain, summing junction* and *adaptive loop*. The

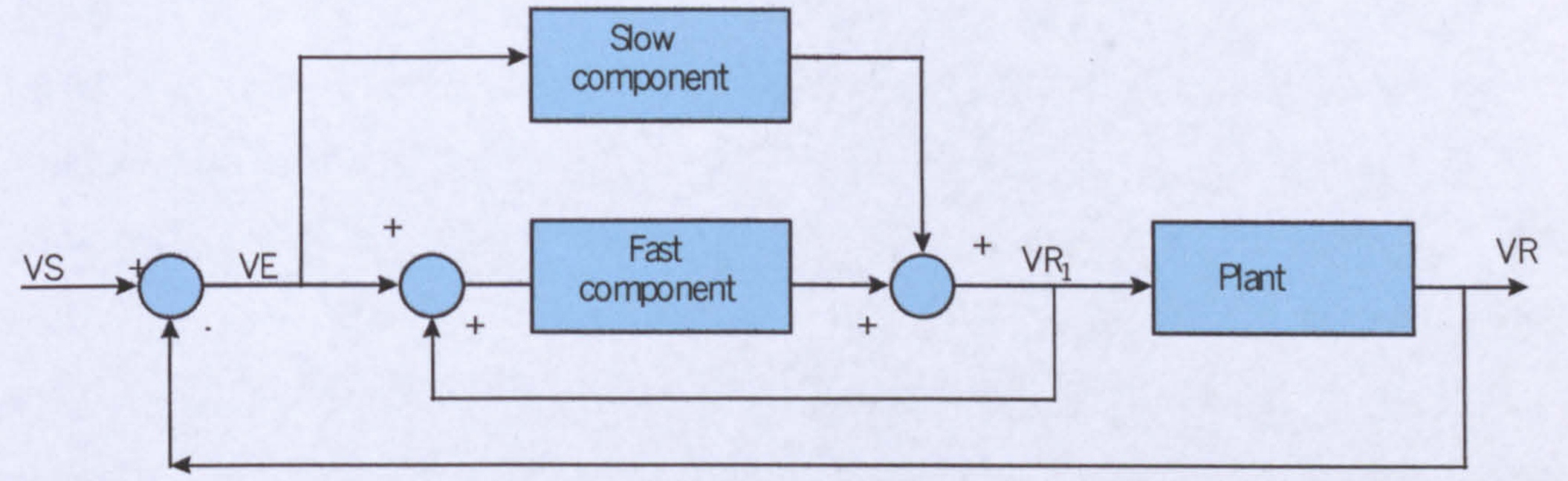
cross link gain provides direct input from either system to the other. In terms of accommodation this produces the relationship that is clinically known as the AC/A ratio (accommodative convergence to accommodation), and for vergence, the CA/C ratio (convergence accommodation to accommodation). The *summing junction* adds the *controller gain* with both the *cross link gain* and a further input known as *system bias.* The *adaptive loop* enables a nearly completed "fast system" response to activate the "slow system" (or adaptive) loop (denoted by m, CE, and K) which completes and sustains the motor response for a prolonged period. The *system bias* mentioned above relates to the clinical entities of tonic vergence and tonic accommodation. These are denoted as ABIAS and VBIAS in Figure 1.12. Under normal closed-loop viewing conditions these biases provide little to the overall response amplitudes (Ciuffreda & Tannen, 1995).

The final *output* for either accommodation (AR) or vergence (VR) provides negative feedback to the initial summing junction at the left of Figure 1.12 and the cycle repeats itself until a stable steady-state response for both systems is established.

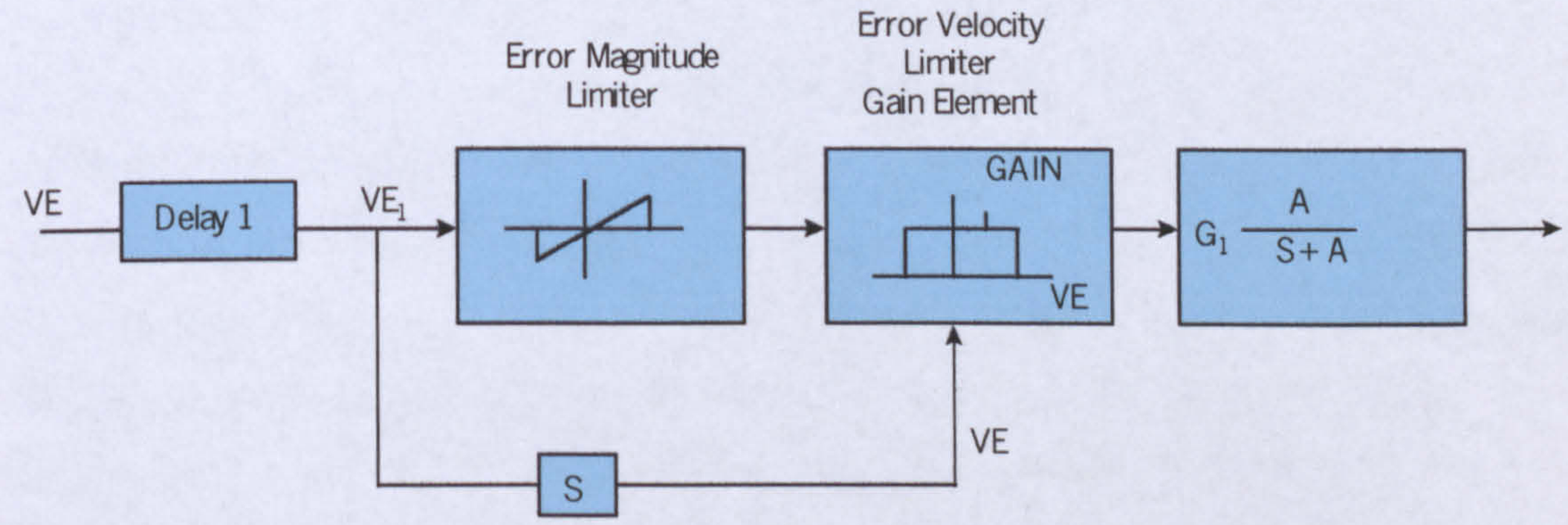
Whereas Figure 1.12 illustrates a static model of the interaction of accommodation and vergence, Figure 1.13 (Hung et al, 1986) illustrates a dynamic model for disparity vergence alone and the following explanation is from both Hung et al (1986) and Ciuffreda & Tannen, 1985).

This model has two main components: the *fast* and *slow components.* The *fast* component has a latency of approximately 200 ms and is used to track perceived target velocity of fast moving targets (> ~ 2 degrees/second) such as occurs with rapid ramps, fast sinusoids, pulses and steps. The fast component is triggered by, and then samples the moving target. A predictor operator then predicts the future target position, such as where it will be 500 ms later, based on target position and velocity at the time of sampling, and sends a command to make an appropriate motor response. The fast component's motor output approximates an exponential. The *slow component* is used to track slowly moving targets (> 2 degrees/second) and is driven by vergence error. The latency is approximately 50 ms for predictable and 200 ms for unpredictable stimuli. This component uses continuous visual feedback

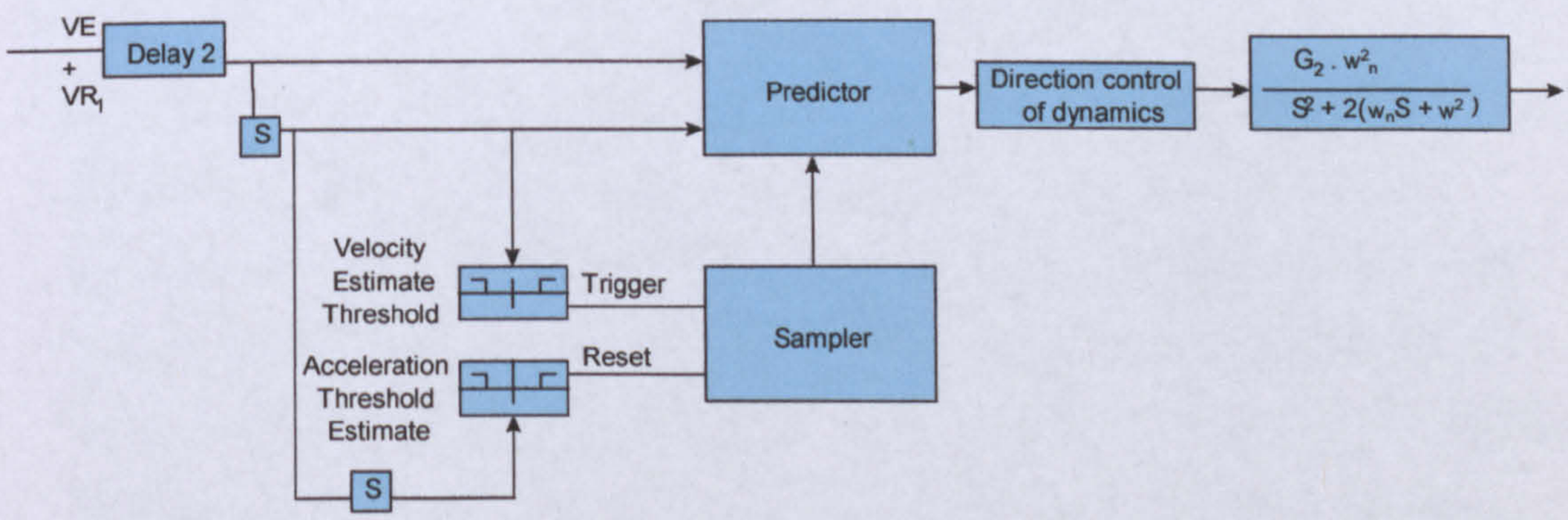
and enables correction of any slow accumulating vergence error especially following slightly inaccurate rapid step or ramp responses. This *slow component* is not to be confused with the "slow" adaptive controller discussed previously with regard to the static model of vergence control.



A



B



C

Figures 1.13. Diagrams describing a dynamic model of disparity vergence only (from Hung et al 1986).

The following discussion and explanation of this model is taken from Hung et al (1986) and Ciuffreda & Tannen (1995). Figure 1.13-A shows an overall dynamic model of the disparity vergence system showing slow and fast

components in forward loop. The responses from both components are summed to give VR_1. Internal positive feedback from VR_1 is summed with vergence error (*VE*) to give an estimate of target position. *Plant* represents mechanical properties of the eyeball and muscles and is assumed to have unity gain for the vergence stimulation. Vergence response (*VR*) is subtracted from vergence stimulus (*VS*) to give vergence error. Figure 1.13-B shows the slow component in forward loop. Vergence error (*VE)* is delayed by 200 ms (*Delay 1)* to produce delayed vergence error (VE_1). An error magnitude limiter (up to 1 degree) and an error velocity limiter gain element (up to 2 degrees/second) simulate the range of slow component dynamics. Figure 1.13-C shows the fast component in forward loop. The vergence error (*VE*) is summed with VR_1 to give an estimate of target position. The delay element (*Delay 2*) represents the effective delay throughout the fast component. An estimated target velocity above a threshold of 1.7 degrees/second is used in the model to trigger the *sampler*. The *sampler* enables the *predictor* to use the estimated target position and velocity to predict the future position of the stimulus. After triggering, the threshold is increased slightly and if the estimated stimulus velocity remains constant, the sampler repeats every 0.5 seconds. A sudden large change in velocity will cause the sampler to be reset.

I. Interactions between the saccadic and vergence systems

It is often assumed that the vergence system is independent from the saccade system (Yarbus, 1957; Alpern, 1969). Enright (1984) and Erkelens et al (1989b) have questioned this. Hyson et al (1983) reported vergence saccades during fusion of misaligned random dot stereograms. (Enright (1984) reported that when binocular fixation is shifted between two targets that require change in vergence as well as an alteration in visual direction, a large proportion of the total change in vergence occurred during the saccades. Further, saccades which contributed strongly to (or fully mediated) an intended vergence change showed significant binocular differences in saccadic excursions (as much as 40-50%). This suggests that these eye movements were not fully yoked as the term 'conjugate' implies. Rather, the eyes behave in such situations as though visual

information from each eye is processed separately prior to the saccade, in order to generate the neural signals which control open-loop saccadic movement of the eye. Hung and Ciuffreda (1996) described a schematic model of saccade-vergence interactions in humans.

Collewjin et al (1988), using a search coil recording technique showed that the abducting eye accelerates faster, reaches a higher peak velocity and terminates its saccade earlier than its fellow adducting eye. This produces an intrasaccadic divergence error of up to 3 degrees which reduces in amplitude after peak velocity has been reached because the adducting eye decelerates more slowly. By the end of the saccade, a disparity of 0.3° may still exist.

Bahill et al (1976) reported subtle differences in the movement characteristics of each eye during saccades and suggested that these were violations of Hering's Law. Ono et al (1978), Kenyon et al (1980c), and Enright (1984) showed that binocular saccades can be disjunctive with the saccade being of different amplitudes for each eye. Erkelens (1989a) reported that the characteristics for the two eyes can differ considerably from the "main sequence" described by Bahill et al (1975).

The question as to whether unequal saccades violate Hering's law of equal innervation has been discussed by Kenyon et al (1980c). Simulations of vergence and saccade interactions were designed to obey Hering's law and showed the same inequality of saccades when superimposed on vergences as shown in their patients. They concluded that the inequality of saccades is not due to a violation of Hering's law and suggest a biomechanical interaction in the muscle globe plant.

More recently, Collewijn et al (1995) studied the dynamics of voluntary, horizontal gaze-shifts between pairs of continuously visible, real three-dimensional targets. They found that pure vergence was almost never observed and were usually associated with saccades. They also reported that horizontal saccades were never strictly conjugate but always contained a transient divergent-convergence sequence. They concluded that, within subjects' manual working gaze-shift related vergence is largely and

preferentially achieved by disjuntive saccades. Pure vergence gaze-shifts without saccades are avoided and, when forced, such isolated vergence is slower than in any combination with saccades. The cover test may be viewed as an extreme example of manipulation of disparity as the sole input stimulus for an eye movement. The subsequent movements are involuntary and such disparity-only-driven vergence has been shown to produce much slower vergence velocities than those associated with voluntary shifts of gaze (Rashbass & Westheimer,1961; Erkelens,1987).

1.3.2.g. Vergence (or prism) adaptation

The vergence system senses disparity between the retinal images in the two eyes and uses feedback to rotate the two eyes until fusion is obtained (Westheimer & Mitchell, 1956). If the vergence loop is opened, for example during a cover test, vergence will decay towards a tonic value ranging from -1 to +1 metre angle (MA) (Alpern, 1969; Hung & Semmlow, 1980) with a time constant in the order of 10 s (Krishnan & Stark, 1977; Schor, 1979a; Fisher et al, 1988).

The magnitude of tonic vergence appears to change transiently immediately after a period of sustained vergence (Fisher et al, 1990; Owens & Liebowitz, 1980; Owens & Wolf-Kelly, 1987; Schor, 1986) and an increase in vergence duration causes a more sustained effect (Fisher et al, 1990). This transient change has been termed *vergence adaptation* (Sethi, 1986). A number of other studies have confirmed the presence of an adaptation mechanism within the vergence system (Schor, 1979a; Henson & North, 1980) and as has seen, the Hung (1992) model incorporates an adaptive loop (see Figure 1.12).

A relatively slow decay of the vergence response after a period of sustained vergence output has also been shown by Schor (1980) who measured fusional vergence after periods of 5 seconds and 60 seconds of vergence. The decay of fusional vergence after 60 seconds of vergence was significantly slower than after 5 seconds. This is illustrated in Figure 1.14. The incomplete relaxation of convergence after long-term stimulation and convergence indicates the effect of slow vergence,

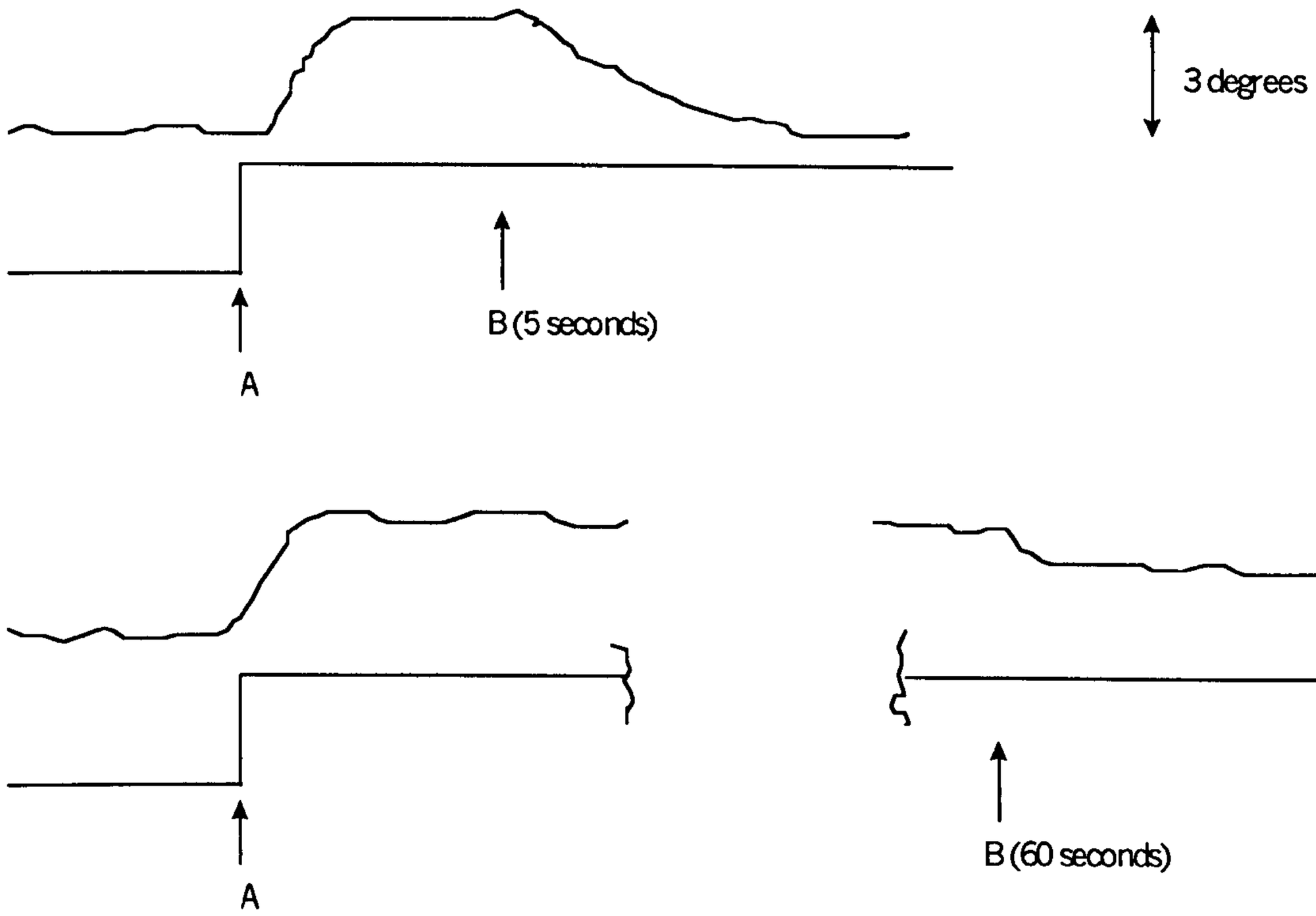


Figure 1.14. Eye movement traces showing time constants for relaxation of fusional vergence after stimulating convergence for 5 seconds (upper trace) and 60 seconds (lower trace). A = onset of stimulation; B = occlusion of one eye (from Schor C, 1980).

If a prism is placed before one eye whilst fusion is still maintained, there will be an immediate change in the phoria amplitude by an amount approximately equal to the prism power. However, within a short time (minutes or even seconds) the phoria amplitude will revert back to the "pre-prism" values. This "resetting" to maintain an optimal or habitual level of ocular alignment has been termed *phoria (*or *prism) adaptation* (Henson & North, 1980).

Prism adaptation reflects the contribution of slow fusional vergence to the overall vergence response, and is usually assessed immediately after the removal of a sustained stimulus (Rosenfield, 1997a). As has already been discussed, the total fusional vergence response can be subdivided into two temporal components; a rapidly acting fast component which typically acts within 1 second to reduce retinal disparity, and a slow component which acts to maintain the net fusional vergence response over an extended period of time (Schor, 1979a; Schor, 1980). The initial response to disparity is mediated by fast fusion. If the stimulus is maintained for a period of time then the fast

fusion will initiate a slow fusional vergence response with a concurrent reduction in fast fusional vergence (Hung, 1992).

In clinical terms, if an orthophoric patient fixates a distant target and a base-out prism is placed before one eye, a fast fusional vergence response will be initiated in order to fuse the disparate images. When the patient maintains binocular vision through the prism for a few seconds and is then dissociated using the cover test, the fast fusional response will decay rapidly. On removal of the cover there will be an exophoric recovery eye movement of an amplitude that approximates to the amplitude of the prism. If this procedure is repeated but this time the prism is left in place for several minutes, then fast fusional vergence will stimulate a slow vergence response. If the prism is kept in place while a conventional cover-uncover test is performed, then there may not be any movement on removing the cover and the eyes will appear approximately orthophoric. As well as *prism vergence*, this phenomenon has also been termed as "eating prism" (Campos & Catellani, 1978).

Rosenfield (1997b) argued that prism adaptation would cause dissociation tests that are only of a few seconds duration, to provide an inaccurate measure of heterophoria amplitude as a substantial portion of slow fusional vergence may remain. This has been confirmed for the cover test (Barnard & Thomson, 1995a) where eye movement recordings have shown that, in some cases, vergence may still be decaying 10 seconds after occlusion commenced.

Henson & North (1980) and Leigh & Zee (1991) discussed the need for an adaptive mechanism to deal with situations such as changes in orbital mechanics with age. For example, the gradual loss of fat that occurs with age (Weale, 1963) will cause a change in orbital resistance to muscle action and will arguably affect the degree of innervation required to carry out a saccade or vergence eye movement. Henson & North (1980) argued that to deal with these changes there must be an adaptive mechanism that monitors any difference between the intended position of the eye and its true position and make appropriate adjustments to the innervational output of the ocular motor control system.

Winn et al (1994) compared vergence adaptation to a 6$^{\Delta}$ vergence stimulus for subjects aged between 19 and 85 years and found a decline in adaptation with age.

Sethi & North (1987) investigated the time course of the adaptive response between different prism values and for subjects with different fusional amplitudes. Heterophoria measurements were made using a flashed Maddox rod technique after the subject was given 15 s of binocular viewing followed by 15 s of occlusion. Henson & North (1980), North & Henson (1982) and Sethi & Henson (1984) have described this technique previously. They demonstrated that the rates of adaptation decreased as the prismatic effect was increased. However, the amount of adaptation increased with the increase in the amplitude of the prism until this was greater than the fusional amplitude.

North and Henson (1981) investigated the magnitude of adaptation to horizontal and vertical prisms in subjects with abnormal binocular vision and/or asthenopia. They reported that symptomatic patients almost invariably exhibited reduced adaptation. Fisher et al (1987) measured vergence adaptation before, during, and immediately after a 45-min period of near vision work in both symptomatic and asymptomatic individuals. They reported that the pre-task levels of tonic vergence and the post-task rate of decay of vergence adaptation were equivalent for the two groups. However, there was some suggestion of a difference in the rate of adaptive onset, with the asymptomatic group reaching the maximum degree of adaptation earlier in the inducing period.

North & Henson (1982) demonstrated that the success of orthoptic exercises in relieving symptoms is associated with an improved ability to adapt to prism-induced heterophoria.

1.4 Vergence anomalies

Both esophoria and exophoria may be classified according to whether the vergence manifested is greater for distance or near. So, if a patient has a poorly compensated esophoria for distance vision but relatively less esophoria, which is well compensated, for near vision, then this is termed *divergence insufficiency.* Similarly, if the patient is relatively esophoric at near compared to distance, and the near esophoria is poorly controlled, then this is termed *convergence excess.* Conversely, *divergence excess* and *convergence insufficiency* describe the conditions where the patient is relatively exophoric for distance and near respectively and in both cases the exophoria is poorly compensated.

Ocular motor imbalances may categorised in a number of different ways.

Firstly, they may be described as manifest (heterotropia or strabismus) or latent (heterophoria). Either of these may be concomitant, in which the amplitude of deviation remains constant in all directions of gaze, or incomitant, in which the amplitude will change according to the direction of gaze and depending on which eye is fixating. The latter occurs when an under-action or over-action of a specific extraocular muscle or muscles is present. When no deviation or latent deviation is present, this is known as orthophoria.

Strabismus may be constant or intermittent. Heterophoria may become decompensated under certain conditions and the patient may then exhibit a manifest heterotropia.

When a strabismus first becomes manifest, the patient is likely to experience diplopia. Whilst this may be intractable under certain circumstances, such as a new strabismus developing in an adult, other sensory strategies may be employed by the visual system to eliminate the diplopic disturbance. These strategies are suppression of the visual image for one eye and/or the development of a new sensory relationship between the sensory retinal elements of the two eyes. The latter is termed anomalous retinal correspondence and may have the advantage of providing some semblance

of a pseudo-binocularity including stereopsis. Generally, suppression tends to occur in divergent strabismus (exotropia) of early onset and anomalous retinal correspondence in early onset, relatively small and stable amplitude convergent strabismus (esotropia). Diplopia usually only occurs in relatively late-onset strabismus.

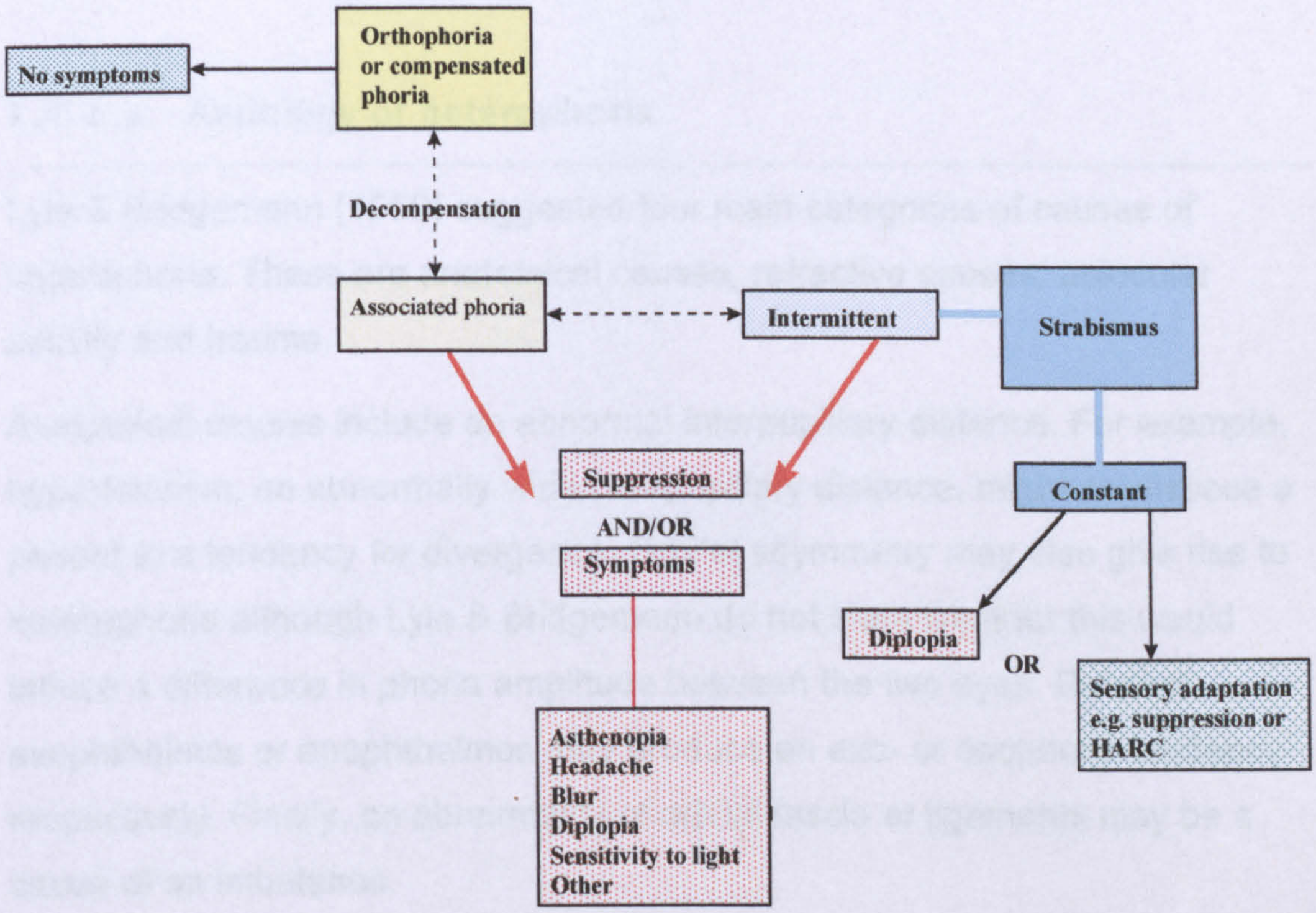


Figure 1.15. A simple schematic diagram illustrating the possible relationships between various categories of ocular motor imbalances and their possible sequelae.

1.4.1 Heterophoria

Stevens (1886), cited by von Noorden, (1990) introduced the term *heterophoria* which is derived from the Greek words, *heteros,* meaning different from; and *phora*, meaning bringing or carrying.

Heterophorias may be described as ocular deviations kept latent by the fusion mechanism. If sensory fusion is suspended, or in some patients 'embarrassed' then a deviation in the visual axes will appear. This deviation is

termed a heterophoria or phoria. It has also been defined as the locus of intersection of the lines of sight, measured with respect to the object of regard, in the absence of fusional vergence response (Rosenfield, 1997). Fry (1964) described the phoria position as "the position taken by the visual axes relative to one another, in the absence of all stimuli to fusion". The presence of a leaky integrator (Zuber & Stark, 1968; Krishnan & Stark, 1977) allows for the return to the "phoria position" upon the application to one eye of an occluder in the cover test (Stark et al, 1980).

1.4.1.a. Aetiology of heterophoria

Lyle & Bridgemann (1959) suggested four main categories of causes of heterophoria. These are anatomical causes, refractive causes, uniocular activity and trauma.

Anatomical causes include an abnormal interpupillary distance. For example, hypertelorism, an abnormally wide interpupillary distance, might predispose a patient to a tendency for divergence. Orbital asymmetry may also give rise to heterophoria although Lyle & Bridgemann do not state whether this would induce a difference in phoria amplitude between the two eyes. Relative exophthalmos or enophthalmos may produce an exo- or esophoric tendency respectively. Finally, an abnormality of orbital fascia or ligaments may be a cause of an imbalance.

Refractive causes relate to the relationship between accommodation and convergence with, for example, uncorrected hypermetropia having a tendency to induce a shift towards esophoria.

The repeated and prolonged use of one eye, for example by a watchmaker, is also suggested as being a possible cause of heterophoria.

1.4.1.b. Prevalence and distribution of heterophoria

There appears to be a high prevalence of distance orthophoria in the population despite a large number of mechanical, neural, and sensory variables (Schor & Ciuffreda, 1983) and it has been suggested that this

apparent *orthophorization* is due to vergence adaptation (Ogle & Prangen, 1953; Carter, 1965). Crone & Hardjowijoto (1979) suggest that orthophorization (or prism adaptation) is abnormal in heterophoria. Dowley (1990) looked at a sample of 925 subjects and demonstrated a significantly non-normal ($p = < 0.05$) frequency distribution (see Figure 1.16) which supported the theory of orthophorization.

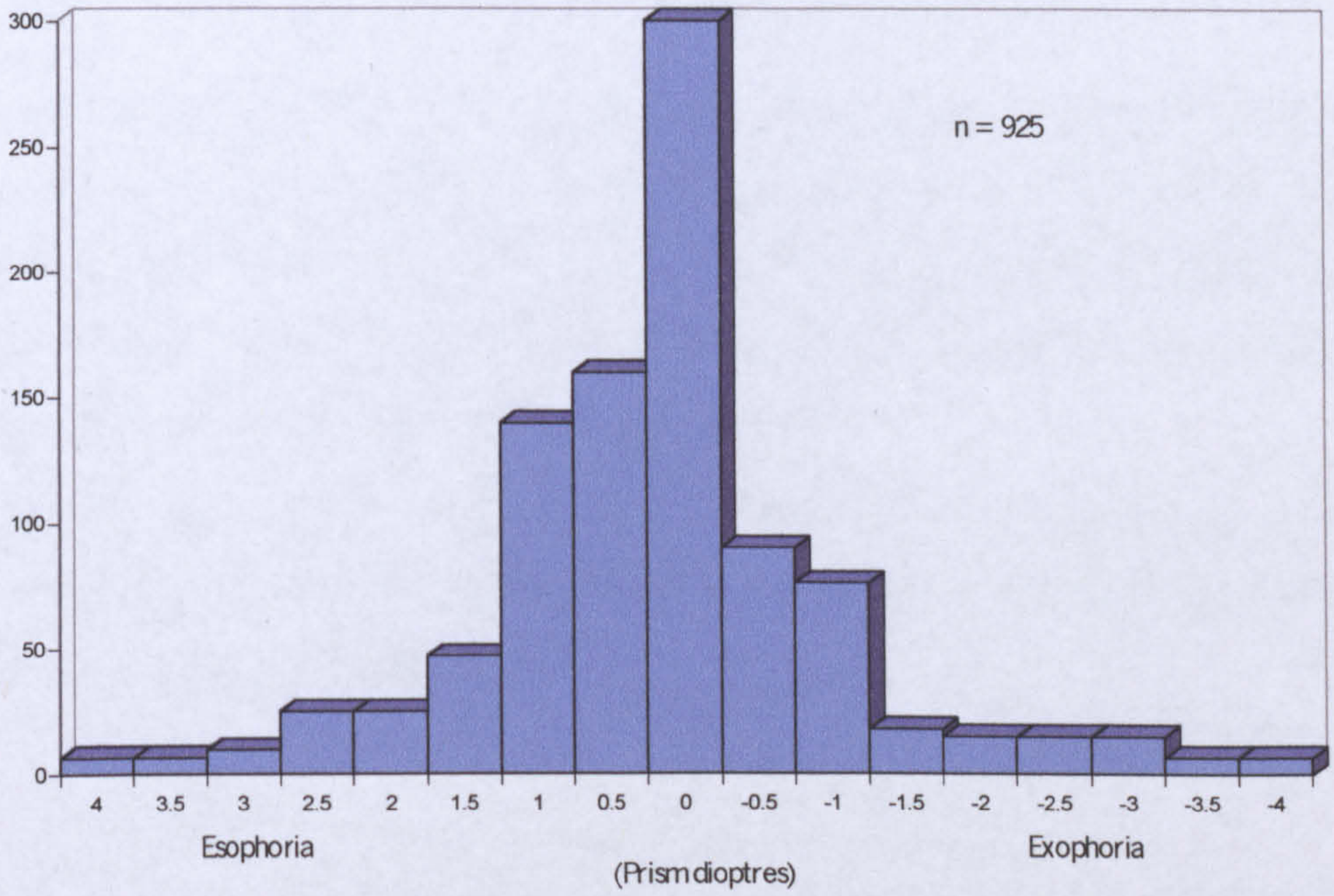


Figure 1.16. Frequency distribution of 925 symptom-free heterophoric subjects (from Dowley D, 1990).

Dowley (1990) suggests that orthophoria is achieved when vergence adaptation is unsaturated and the total deviation is well within a comfortable operating range for vergence adaptation and that distance heterophoria arises when vergence adaptation is partially saturated.

Eames & Cambridge (1933) measured near phoria for 212 non-presbyopes and found an average phoria of 0.4 $^{\Delta}$ exophoria. They also reported that the average phoria amplitude for a sample of 90 presbyopes was 7^{Δ} exophoria although they did not specify the method used nor whether or not the amplitude was measured with a near reading addition in place.

It is interesting to note that manifest exo-deviations are reportedly more prevalent amongst females (Gregerson, 1969; Krzystkowa & Pajakowa,

1972), particularly as some authors have suggested a psychogenic cause for some exo-deviations (Knapp, 1958; Taylor, 1990) whilst others have reported a higher prevalence of psychosomatic visual anomalies in females (Mantyjarvi, 1981; Barnard, 1989).

An increase in exophoria for near vision with age has been described by a number of researchers (Morgan & Peters, 1951; Snydacker, 1962; Sheedy & Saladin, 1975; Freier & Pickwell, 1983). Snydacker (1962) found that the amount of exophoria increased in amplitude by 1.5^{Δ} for every 20 years of age.

Yekta et al (1989) reported an increase in exophoria, associated heterophoria and fixation disparity with increasing age. They also found no evidence that a presbyopic reading addition accelerates an increase in fixation disparity or associated phoria with age and they concluded that there is an adaptation of these parameters to the reading addition.

Graefe (1967) (cited by von Noorden, 1990) reported an increase in the amplitude of deviation for near fixation under "the influence of bright light" in those patients with exophoria.

1.4.1.c. Measurement of heterophoria amplitude

There are numerous ways of measuring the amplitude of heterophoria. In practice the results vary depending on the test used. These variations may be a result of differences in the central or peripheral stimulus to fusion including the size and complexity of the visual field or the fixation targets, the nature of the borders in the field of view, luminance differences, and various levels of proximal vergence effects (Daum, 1983a). In addition, the ability of the test to control accommodation and the length of time that fusion is disrupted will also affect measurements (Griffin, 1982). The adoption of a standard, universal method of measuring the amplitude of deviation is neither likely nor desirable but it is useful to have some understanding of the validity and reliability of tests employed in clinical practice.

1.4.1.d. Objective methods for assessing heterophoria

There is some anecdotal confusion amongst clinicians as to exactly what is meant by the term "cover test." There is a tendency in ophthalmology to equate "cover test" with what might be more fully described as the *alternate cover test* whereas in optometry the term is often used to mean the *cover-uncover test* or *unilateral intermittent occlusion.* For the purposes of this discussion, whenever the term "cover test" is employed, it is used to describe the *cover-uncover test.*

The cover test is the most commonly performed and most important test relating to binocular vision and enables the clinician to detect the presence of heterophoria or hetereotropia and to measure the size of the deviation (Barnard & Thomson, 1995a; Franklin, 1997). An investigation of eye movement characteristics during the cover test forms a large component of this thesis.

The alternate cover test is usually used to assess phoria amplitude. The patient fixates a target at the required distance and an occluder is placed before one eye for a short period of time and then rapidly moved in front of the fellow eye without giving any time for binocular vision to occur. As the cover is removed from the eye, any movement is noted. This process is repeated until the practitioner is satisfied with the observation. Evans (1997) described the *alternate cover test* as a transfer of the cover from one eye to the other and back several times. von Noorden (1990) described it as a test used in conjunction with prisms to neutralise the movement observed during this alternating covering procedure. This technique reportedly produces a larger amplitude of phoria as compared to the *cover-uncover test.* Cooper (1992) and Pickwell (1997) also suggested that the degree of deviation usually increases during the alternate cover test allowing 'the full extent of the deviation to be seen'.

Cooper (1992) discussed the clinical implications of vergence adaptation in terms of the cover test and observed that with repeated alternate occlusion, the angle of deviation often increases and that the increase in the angular measurement is dependant on:

Mallett (1966) also believed that heterophoric headaches usually accompany the visual act promoting the stress but suggested that esophoric induced headache may not commence until the following day. The duration of the headaches is variable.

Diplopia: Mallett (1966) suggested that diplopia occurs frequently in uncompensated exophoria and convergence insufficiency but is rarely experienced in esophoria.

Blurring : This may occur when the oculo-motor imbalance has an accommodative element (Mallett, 1966). A further symptom or sign, to be exact, is an interference in binocular visual acuity in the presence of horizontal associated phoria reported by Jenkins et al (1994).

Asthenopia: This may be described as a generalised discomfort and irritability of the eyes. The term is derived from the Greek: σ θενος, strength; and ϖψ, eye. Clarke (1893) described asthenopia as a symptom or group of symptoms that result from straining some part of the eye apparatus. It is worth noting that Sheedy & Saladin (1978) carried out an investigation into the association of symptoms with measures of ocular motor deficiencies. The questionnaire used in the study seeks to rank asthenopic symptoms but does not use headache as a separate symptom.

Photophobia: An intolerance to bright light has been reported for patients with exo-deviations (Lyle & Bridgemann, 1959; Eustace et al, 1973; von Noorden, 1990).

Other symptoms: Anecdotal reports of general ocular fatigue and strain, blepharitis, lacrimation, irritation, conjunctival hyperaemia (especially nasally), changes in head posture, distortion and difficulty in judging distances have also been suggested (Gibson, 1955; Lyle & Bridgemann, 1959; Purcell, et al, 1983).

Suppression: Deep central suppression can develop after a poorly compensated heterophoria has been present for some time. Suppression may cause a lessening or complete resolution of symptoms. Jampolsky (1964) suggested that manifest exo-deviations usually begin as an exophoria that

may deteriorate into intermittent and constant exotropia as suppression develops.

1.5.2 The relationship between binocular function and symptoms

Over the years, various vergence criteria have been suggested as prerequisites for binocular comfort.

Donders (1864) (cited by Birnbaum, 1993) was the first to use a graphical form to portray the relationship between accommodation and convergence. He analysed the limits of accommodation that could be elicited at given levels of convergence, and postulated that accommodation could be comfortably sustained at a given fixation distance only if positive relative accommodation was greater than negative relative accommodation. Birnbaum (1993) goes on to cite Landolt (1886) who added the ranges of relative convergence to the graph, and proposed that not more than one third of the absolute range of convergence could be maintained without asthenopia.

The use of Sheard's criterion (Sheard, 1957) is a method of prescribing prism that has been popular in the USA. Worrell et al (1971) described the criterion as 'a classical, easily applied, and commonly used method to determine the amount of prism to be incorporated in spectacles for patients with neuromuscular imbalance and/or asthenopia'. Sheard claimed that for patient comfort, the demand on fusion (phoria) should be no greater than half the fusional ability (opposing relative fusional response). Specifically, for comfort, the amount of prism required equals two thirds of the phoria minus one-third the compensating duction. If the result is positive, prism is needed in the amount represented by the result; if negative, no prism is needed. The compensating duction is base-out to blur (or break if there is no blur) for exophoria and base-in for esophoria.

Percival's criterion suggests that vergence demand should lie in the middle third of clear binocular single vision. This is sometimes known as the middle third technique (Solomons, 1978).

Mallett (1966), in his discussion on symptoms and signs of heterophoria, states that the degree of heterophoria may bear little relationship with the

patient's symptoms. He observes that a patient with less than 2Δ lateral heterophoria may have severe discomfort and that higher degrees may be fully compensated. Here there is an allusion to a hypothesis that decompensated phoria is likely to cause symptoms. Fixation disparity and associated phoria (the degree of prism required to eliminate the fixation disparity) have been thought to be an indicator of decompensation of the heterophoria (Mallett, 1974).

Using a double blind experimental procedure, Worrell et al (1971) evaluated prism prescribed on the basis of Sheard's criterion for 43 patients with muscular imbalance and asthenopic complaints. An evaluation of symptoms appeared to be made simply on patient preference to one of two pairs of spectacles. No attempt was made to evaluate symptoms in detail. They reported that the pre-presbyopic exophores did not prefer spectacles that contained prism, whereas the majority of the presbyopic group did.

Sheedy & Saladin (1977) examined two groups of subjects in an attempt to establish which optometric measures could be used to identify patients with ocular motor symptoms. The sample consisted of two groups of pre-presbyopic subjects. The first group comprised of 32 subjects selected from 50 optometry students with no ocular symptoms. The presence and strength of symptoms was evaluated by questionnaire and interview. Details of the questionnaire and how the asthenopia was scored were not included in the paper. The authors stated that the study was double masked in that neither the subject nor the investigator had available the diagnostic data. The second group was comprised of 28 patients from the orthoptic clinic complaining of symptoms associated with near vision and diagnosed as having a lateral binocular ocular motor deficiency. They measured horizontal phoria, vergence ranges and forced vergence fixation disparity curves. It should be noted that the Sheedy disparometer used as a fixation disparity test does not provide a good fusional lock. Eleven clinical indicators or variables were derived from the data and discriminant analysis was used to determine which tests or group of tests best discriminated between the two groups and for exophoric and esophoric sub-samples. They reported that Sheard's criterion was the best discriminator for the exophoric group, and the amplitude of heterophoria was

the best discriminator for the esophoric group. The next best discriminator was a fixation disparity variable.

Sheedy & Saladin (1978) criticised their study as producing a biased symptomatic group as this group may have been affected by the referral criteria used by individual practitioners. Sheedy & Saladin (1978) repeated their previous study and removed any possible referral bias by dividing 103 optometry students into symptomatic and non-symptomatic groups based entirely on results of a symptom questionnaire. The average asthenopia level of the symptomatic group might be expected to be less severe than that of the symptomatic group in their previous study because the subjects were deliberately not drawn from a clinic population. An asthenopia scale of 1 to 8 was determined for both distance and near vision. Phoria, fusion ranges and forced fixation disparity curves were measured for each subject at 6 m and 40cm. Only the near data was analysed as there were few high asthenopia ratings for distance. Subjects with an asthenopia rating of 1 were analysed as one group (n = 44) and subjects with ratings of 4 - 8 were treated as the second group (n = 33). Ratings of 2 and 3 were treated as borderline asthenopes and were excluded from the analysis. The data were analysed using discriminant analysis. Discriminant analysis may be used to distinguish statistically between two or more groups, here symptomatic and asymptomatic, on the basis of a set of discriminating variables. For each variable, an F value is calculated on the basis of the values of the variables in the two populations (Sheedy & Saladin, 1978). Seventeen variables were selected or calculated from the clinical test results to serve as the discriminating variables.

Sheedy & Saladin reported that:

- Sheard's criterion is the best discriminator for the entire sample and was a better discriminator for exo deviations compared to eso deviations
- Fixation disparity variables were good discriminators between the two groups
- Percival's criterion was the best discriminator for the esophores

They argued that implicit in Sheard's criterion is the concept of an opposing vergence compensating for a deviation. The concept that positive vergence is a more active process than negative vergence, together with the clinical impression that positive fusional vergence is more easily trained than negative vergence, is then meaningful in terms of Sheard's criterion proving to be a better discriminator of symptoms in the exo population than in the eso group. Similarly, implicit in Percival's criterion is the concept that visual comfort is found in the middle of the vergence range that suggests a passivity of vergence. The superiority of Percival's criterion as a discriminator for eso deviations implies that the negative vergence is indeed passive.

Teitelbaum et al (1985) examined ninety optometry students and divided them into a symptomatic and an asymptomatic group on the basis of case history. One student was eliminated due to suppression. All subjects were correctable to 6/6 acuity. Symptoms reported by the subjects and used for categorisation included headache, asthenopia with near work, blurring or doubling of vision at near. However, no further details were given as to how these symptoms were graded. A forced vergence fixation disparity curve was generated for each subject using a Disparometer. Slopes were calculated for each curve. In addition, each curve was labelled steep or flat. They reported that the gradient of the curves did not correlate well with the presence of symptoms. An independent t-test found no significant difference between the two groups, contrary to the findings of Sheedy & Saladin (1978).

Pickwell & Hampshire (1981) tested 455 patients and found that poor convergence movements were associated with decompensated exophoria for near vision as diagnosed on the Mallet unit. No evidence was found to associate an NPC greater than 10 cm with a high incidence of symptoms. However, poor jump-convergence movements were more frequently associated with symptoms.

Pickwell et al (1987) measured fixation disparity in a group of 40 binocular and mainly symptom-free subjects, at a good and at a poor level of illumination. Measurements were taken for near vision (40 cm) without prism, with 4^{Δ} in, and with 4^{Δ} out. In a second procedure, associated phoria was measured for fifty-seven subjects at the high illumination level and then they were asked to

read for 10 minutes in reduced illumination. They were then asked to report any visual discomfort. A third experiment was carried out on 20 subjects to note discomfort after more prolonged reading under reduced illumination. The phoria at near was measured using the Mills test and the Mallett near vision fixation disparity unit was also used at 40 cm

They concluded that a reduction in illumination, when added to prism stress will increase fixation disparity. The stress created by asking subjects to read in reduced illumination for half an hour resulted in the mean associated heterophoria being increased, and over half the subjects reported symptoms of stress. They further concluded that fixation disparity is changed by the visual stress employed in the study in some subjects, and in near vision is increased to a more marked exo-disparity. Most of this increase occurs in the first ten minutes

Yekta et al (1987) investigated ocular motor characteristics in eighty-four young adults in the morning and again in the afternoon. They reported an increase in the amplitude of associated phoria and fixation disparity at the end of the working day and that this seems to be related to the increase in the visual symptoms and discomfort. They found that associated phoria and fixation disparity showed a better correlation with visual symptoms than dissociated phoria readings and that as associated phoria and fixation disparity increase towards the end of the working day, the visual symptoms increased.

Jenkins et al (1989) examined two groups of patients, one under 40 years of age and the other 40 years and over. They used a method based on 'signal detection theory' to determine whether a value could be found for dissociated heterophoria (measured with the Mills test), or associated heterophoria (measured with the Mallett unit), which could predict which patients had symptoms due to decompensated heterophoria in normal routine investigation. Patients were categorised as either being symptomatic or symptom-free further grading. Measurements of dissociated phoria had little value in terms of discriminating between symptomatic and asymptomatic patients and they concluded that the measurement of heterophoria amplitude is not useful as a routine procedure. Jenkins et al. (1989) found that the

Mallett test had a sensitivity of 75% and a specificity of 78% for detecting symptomatic heterophoria, and that this test eliminated the need to plot a forced vergence disparity curve."In the case of associated heterophoria, for the under 40 years age group, patients with a value of 1^{Δ} or more were more likely to have symptoms than not, and one-third of patients with close-work problems had a value of 2^{Δ} or more. It was rare to find any asymptomatic patient with a value as high as this. In the 40 years and over age group 64% of patients with an associated phoria of 2^{Δ} or more were symptomatic.

Feldman et al (1992) demonstrated significantly higher ratings of asthenopia after induced vergence compared to version. However, they were unable to show a significant correlation between asthenopia scores before and after induced vergence in their sample of normal subjects (n = 30). Possible reasons noted for this result were that subjects in their sample were largely asymptomatic with restricted asthenopia scores. They also suggested that the experimental task was not representative of the tasks normally carried out by the subjects environment and that the experimental intervention may not have been prolonged enough to produce stress.

As noted previously, anomalies of prism adaptation may be correlated with symptomatology (North & Henson, 1981).

1.5.3 Grading systems for symptoms

Feldman et al (1992) described two questionnaires for grading symptoms. The first questionnaire was completed prior to the experimental treatment being carried out. This asked the subjects to rate the severity of his/her daily asthenopic symptoms while doing a variety of near point tasks in the natural environment. Ratings ranged from asymptomatic to severe on a 5-point rating scale. The 10th question invited the subject to comment on any other visual difficulties experienced whilst doing close work. The second questionnaire was completed by the subject following either vergence or version training. This presented questions that related to symptoms directly associated with the recently completed training but utilised the same 5-point rating scale.

Yekta et al (1987) produced a visual symptom score by allocating one point for each symptom reported by the subject. They then classified subjects into five groups as follows:

Group 1 symptom-free subjects with no symptom score

Group 2 symptomatic subjects with 1 or 2 symptoms

Group 3 symptomatic subjects with 3 or 4 symptoms

Group 4 symptomatic subjects with 5 or 6 symptoms

Group 5 symptomatic subjects with 7 or 9 symptoms

1.6 The treatment of binocular vision anomalies

Practitioners have a number of treatment options available for managing associated phoria and convergence anomalies. These include orthoptics or vision therapy, optical intervention (both lenses and prisms) and surgery.

1.6.1 Prismatic correction

The use of prisms may be considered when orthoptic treatment is inappropriate because of ill-health, or due to lack of time or incentive on the part of the patient. There are a variety of ways of determining the amplitude of prism. A common method of prescribing in the UK is to determine the amount of prism required to eliminate a fixation disparity. The minimum prism required to neutralise the fixation disparity may be prescribed (Mallett, 1966).

Evans (1997) suggested an objective method of determining the prism power whereby the practitioner determines the power of the weakest prism required to produce a quick and smooth recovery following the cover test. This method presupposes that a poorly compensated heterophoria does produce a slow and jerky recovery during the cover test. This supposition is based on anecdotal evidence and will be investigated in this thesis.

Prisms may also be used in the treatment of divergent strabismus (Ravault et al, 1968; Berard, 1968). Ravault et al suggested that the elimination of

suppression is a major effect of constantly wearing a prismatic correction in those patients for whom prismatic treatment eliminates a manifest deviation.

The relief of vertical heterophoria with prism has been reported to aid compensation of exophoria (London & Wick, 1987).

Prism adaptation commonly occurs in patients with normal binocular vision. However, most patients with binocular vision anomalies that are causing symptoms exhibit abnormal prism adaptation (North & Henson, 1981). Whilst this may be an indication for prescribing prisms, North & Henson (1982; 1992) showed that some patients who had abnormal adaptation to prisms before receiving orthoptic exercises had normal prism adaptation after treatment.

Winn (1994) demonstrated that prism adaptation reduces with age and suggested that this may at least partly explain a claim that older patients do not respond as well as younger patients to orthoptic exercises.

1.6.2 Refractive correction

Consideration should be made to whether correcting a patient's ametropia or anisometropia may facilitate an improvement in binocular function. Blurring of one or both eyes may precipitate poorer compensation of a heterophoria. Whilst Ukwade & Bedell (1993) found that small degrees of blur and reduction of contrast would have inconsequential effects on fixation ability or vergence, Dwyer & Wick (1995) found that even correcting low degrees of ametropia can dramatically improve vergence and accommodative function for some patients.

Modifying the spectacle prescription may alleviate ocular motor stress. This effectively employs the relationship between accommodation and accommodative vergence to either increase or decrease convergence. For a patient with a poorly compensated esophoria at near or a convergence excess, an additional positive lens may be used to reduce accommodative demand and thus reduce accommodative convergence. This will have the effect of reducing the amplitude of an esophoria. An additional positive lens will cause blurring of distance vision and so a bifocal type lens is often employed. The addition in the near segment provides the patient with the

benefit of a reduced accommodative requirement for near vision. Similarly, incorporating a binocular negative lens addition (Caltrider & Jampolsky, 1983) may alleviate a poorly compensated exophoria. This will produce an additional accommodative demand and so stimulate accommodative vergence. The fact that this technique requires additional accommodation means that it may only be used for patients with adequate accommodation. In young patients this technique may be employed to help control associated exophoria at distance and near as well as divergence excess and convergence insufficiency.

It should be noted that patients may adapt to spherical lenses just as they can adapt to prisms (North & Henson, 1985). Patients should therefore be allowed to wear the proposed prescription for 3-5 minutes and the ocular motor balance re-assessed to ensure that adaptation has not taken place (Evans, 1997).

Dwyer (1992) assessed the refraction, vergence, and accommodative status of 144 consecutive patients and found that 39% presented with ametropia. Of these, nearly 80% manifested a vergence and/or accommodative anomaly. Dwyer & Wick (1995) recommended that refractive correction should be used for such patients and suggested there was a possibility that this type of intervention will cause passive resolution of the binocular disorder.

1.6.3 Orthoptic exercises

In the UK the term 'orthoptics' has been used to describe eye exercises to treat ocular motor disorders such as vergence anomalies. Orthoptics is a term used both by the 'optometric profession' and by 'hospital orthoptists'. In the USA the term 'orthoptics' tends to describe treatment offered by the separate orthoptist profession and 'vision therapy' (or 'visual therapy') is used to describe exercise regimes prescribed by optometrists. For most exercise regimes the two terms probably describe very similar treatment philosophies. For the purpose of this discussion the two terms will be used interchangeably to allow for the various usage's in the international literature.

The term 'orthoptics' is derived from the Greek *ορο s* meaning "straight", and οπτικοζ meaning "pertaining to sight". Orthoptics has been used for many

centuries to treat ocular motor disorders. Flick (1937) (cited by Giles, 1943) traced a reference to treatment of strabismus using a mask as far back as the Ebers papyrus dated about 1650 BCE. Flick suggested that the first orthoptic treatment was carried out by Aeginets (AD 625-690).

1.6.3.a. Techniques

The types of vision training or orthoptic exercises are very wide and varied. Some of the most commonly prescribed exercises for convergence insufficiency and exophoric conditions are "push-up", "jump convergence" and "physiological diplopia techniques". A combination of these three exercises was used for treating the subjects in this present study and is described below.

I. Push up (pencil-to-nose) exercises

The patient is asked to look at a fine tip of a pencil placed at about 50 cm or well outside the range of NPC. The pencil is then moved towards the eyes until it appears to double, or the practitioner observes that one eye has ceased to converge (Evans, 1997). The exercise is repeated a number of times with the aim of developing convergence and reducing the NPC. If the patient does not appreciate diplopia then the practitioner should assist the patient to develop awareness of physiological diplopia before proceeding. Once the patient understands the exercise, instructions can be given for the patient to carry out the exercises at home on a daily basis.

II. Jump convergence exercises

The patient moves a small, detailed target in as close as possible before it becomes blurred, double, or one eye diverges. The target is held stationary and the patient relaxes accommodation and convergence to view a distance target. Fixation is then changed from the distance to the near target. The patient should be aware of physiological diplopia (Evans, 1997). This cycle is repeated a prescribed number of times.

III. Physiological diplopia techniques

There are a number of variations of this category of exercise. For example, the patient can be instructed to hold two different colour pencils at different distances on the median line against a plain background. The patient is instructed to look at the further pencil that should be seen singly. The nearer pencil should appear double. The patient is then instructed to view the near target that should then be single. The further target should then be seen in diplopia. This procedure is then repeated with the pencils being brought closer to the patient.

Another variation includes the use of a string on which a number of small beads have been threaded. The patient is instructed to hold the string so that one end is touching the tip of the nose and the other end is at arm's length. The patient fixates on the farthest bead that should be seen singly. The patient should also perceive the string and the remainder of the beads in diplopia with the separation of the two images becoming greater as the string gets closer to the nose. The patient then re-fixates to the next bead. Now this one should be seen singly, with the furthest bead and the nearer beads all being seen in diplopia. The diplopic images of the string should appear to cross through the single bead. The patient is instructed to change fixation and advance from bead to bead until the nearest bead is seen singly.

A similar technique uses a card on which a line and a series of dots have been drawn as an alternative to the string and the beads.

IV. Other techniques

Development of fusional reserves may be carried out using haploscopic instruments or with vectographs (Rosner & Rosner, 1990; Evans, 1997). Afandor (1982), Goldrich (1982), Scheiman et al (1983) and Letourneau & Giroux (1984) have all reported success in treating large exo-deviations using ocular motor biofeedback therapy. Computerised vergence therapy has also been employed in the treatment of ocular motor disorders (Sommers et al, 1984; Daum et al, 1987).

1.6.3.b. Efficacy

Orthoptic exercises have been employed as a conventional or "orthodox" method of treatment for many years. It is widely accepted that fusional reserve training exercises can be an effective method of treating positive fusional reserve dysfunction such as convergence insufficiency (Davies, 1956; Kratka & Kratka, 1956; Passmore & MacLean, 1957; Norn, 1966; Wick, 1977; Cooper & Duckman, 1978; Kertesz, 1982; Daum,1982; Pantano, 1982; Cooper et al, 1983; Cohen & Soden, 1984; Daum, 1984a; Daum, 1984b). Other studies purport to show that patients receiving vergence training are effectively relieved of difficulties associated with other ocular motor anomalies (Ludlam & Kleinman, 1965; Sanfillipo & Clahane, 1970; Hoffman, 1973).

Goodson & Rahe (1981) studied the effects of vision training on various optometric parameters, including vergence ranges, in a group of US Air Force pilots with normal binocular vision. There was a control group who did not undergo any vision training and the study was single (practitioner) masked but there was no placebo control in operation for the treated group. They reported significant ($p < 0.05$) increases in the trained group for base-out vergence to blur and break and base in vergence to recovery.

Daum (1982) studied the time course and the magnitude of changes in the horizontal vergence system of subjects with normal binocularity following regimes of commonly used vergence training techniques. Thirty-five healthy adults aged 22 to 28 years underwent a full examination of their refractive, binocular and ocular motor systems. They all then underwent visual training which consisted of two sessions (totalling 4 min/day) of "push up" exercises, flip prism or jump vergence exercises (2 min), variable vectograph exercises (2 min) and vergence training on a major amblyoscope (2 min). All but the latter exercise were carried out at home and for five days a week for three weeks. Analysis of the results showed positive fusional ranges to be significantly increased after 1week of training with an even greater effect measurable after three weeks. Six months after training ceased, the effect of the exercises was still apparent but the vergence facility was found to have

decreased. Whilst negative fusional ranges were shown to have increased, this training was less effective.

The long-term effect of exercises reported in normal subjects (Daum, 1982) has also been demonstrated for CI by Pantano (1982) whose study suggested a long-term cure for convergence insufficiency. Two hundred and seven patients with CI were compared on the basis of their success category immediately after training. Those that had been released as "cured" maintained the same result after six months and two years. Of those patients in the "partially cured" group, 79% remained asymptomatic after six months, but only 11% were asymptomatic after two years. The "failure" group received no symptomatic relief and even the improved convergence skills were not maintained six months post-therapy. Only those patients who were able to achieve both voluntary and fusional convergence and who had learned to relax accommodation adequately while converging so that they had altered their AC/A ratio could maintain their status over time.

Daum (1983b) compared the effectiveness of step and jump (phasic) vergence training with sliding and push-up (tonic) vergence procedures in a group of thirty-four healthy asymptomatic adults who were divided randomly into two groups. One group carried out phasic vergence training and the other group tonic vergence training. Whilst both groups made substantial gains in convergence and divergence ranges, the phasic group showed the greatest improvement. It is worth noting that Pickwell & Stephens (1975) suggested that jump (phasic) convergence may be of more clinical significance than a poor NPC and Pickwell & Hampshire (1981) demonstrated a stronger association between symptoms and poor jump convergence than with poor NPC.

Daum (1982) and Vaegan (1979) have also demonstrated the efficacy of repeated, short training periods.

However, there are surprisingly few studies that look critically at the efficacy of the various techniques. There are even less that utilise double masked placebo controlled trials such as employed by Daum (1986c) and the

matched-subjects control group crossover design used to reduce placebo effects in the study described by Cooper et al (1983).

Cooper et al (1983) used a matched-subjects control group crossover design in a study of the effects of automated fusional reserve convergence training on a group of seven patients with CI. All patients showed significant increases in vergence ranges with concurrent marked reduction of symptoms after training. Their results clearly demonstrated the effectiveness of fusional reserve training in reducing asthenopia in these patients. Subsequent accommodation and vergence training using traditional orthoptic procedures yielded further reduction of asthenopia, as well as an increase in the positive fusional ranges.

Daum (1986c) conducted a double-blind placebo-controlled study to investigate the effects of positive fusional reserve training. Four experimental protocols were selected and five subjects were assigned randomly to each of the four experimental groups. Each subject in each group spent a total of 120 minutes over a three week period doing fusional reserve training. Group A trained in twelve 10-minute sessions; Group B in six 20-minute sessions; Group D was the control group and carried out version rather than vergence exercises. All of the training sessions took place within the optometry clinic and consisted of positive fusional vergence training on the synoptophore. Each of the test groups showed increases in their positive fusional vergence ability at both distance and near with the group undertaking the shorter sessions demonstrating the largest overall increases. This group was also the only group to show significant increases in positive blur findings and negative fusional reserves. The control group did not show a significant change in vergence amplitudes. It is worth noting that whilst this study suggested that it is possible to increase the positive fusional vergences it did not aim to show that increasing positive fusional vergence is effective in dealing with the asthenopic symptoms produced by vergence dysfunction.

Although younger patients may tend to respond more positively to such exercises than older patients (Winn et al, 1994), Wick (1977), using well defined and rigorous criteria for success, reported a 93% cure rate following the treatment of 191 presbyopes aged from 45 to 89, having asthenopic

symptoms associated with convergence insufficiency, near point exophoria, and/or fusional vergence deficiencies. Similar results have been obtained for adults over the age of 60 years with convergence insufficiency (Cohen & Soden, 1984). This suggests that practitioners should not rely on patients' ages in determining whether orthoptic exercises should be prescribed. Indeed, Grisham (1988) suggested that because of their maturity, older patients might demonstrate the self-discipline necessary to follow through with visual therapy.

1.6.4 Surgery

Surgery may be required for large angle deviations, as well as various incomitant anomalies that have not or are unlikely to respond to other treatment methods. A fuller description of these procedures is outside of the scope of this thesis and the reader is recommended to von Noorden (1990) and Coffey et al (1992).

1.7 Summary

This chapter reviewed horizontal vergence and version eye movements, methods of assessing ocular motor balance, and methods of treating horizontal ocular motor anomalies.

Whilst the cover test is widely employed by clinicians, this introduction has shown there have been very few objective studies that have examined the nature of eye movements during the test.

Chapter 2. Method for assessing eye movements during the cover test

2.1 Introduction

The *cover test* is probably the most commonly performed and arguably one of the most important tests relating to binocular vision enabling the clinician to detect the presence of heterophoria or hetereotropia and to measure the size of the deviation (Barnard & Thomson, 1995a; Franklin, 1997). Despite the clinical importance commonly attributed to the cover test there is very little published research describing the nature of the eye movements made by subjects during this procedure.

The main aim of this study was to investigate the characteristics of eye movements during the cover test with the objective of providing some information to explain anecdotal observations made by practitioners relating to such characteristics as speed and smoothness of the recovery eye movements following occlusion.

2.2 Methods

2.2.1 Automated cover test

In order to obtain precise information about the nature of eye movements during the cover test, it was necessary to develop a system to a) cover each eye rapidly and in a repeatable manner and b) monitor eye movements accurately.

2.2.1.a. Development of apparatus

In a pilot study, pen motors were used to drive shutters to cover the subjects' eyes. These were found to be noisy causing the subjects to blink excessively which introduced artefacts into the eye movement recordings.

The use of liquid crystal shutters was investigated as a possible method for producing a very rapid, silent occlusion. These devices respond in less than 2 milliseconds from high to low transmission and less than 5 milliseconds in the reverse direction. However, the optical density of the shutters in the 'darkened' state was inadequate to provide complete occlusion and therefore they were rejected in favour of a mechanical system.

In the only previous study of this type, Peli and McCormack (1983) used an electromechanical occluder controlled by a manual switch. The possible use of a manual switch was rejected for this study, as it would not allow for precise control of the temporo-spatial status of the cover. However, the use of a mechanically driven occluder was investigated and decided upon.

Polystyrene covers driven by two independent computer-controlled stepper motors were used to occlude the eyes. To ensure that occlusion was rapid, the stepper motors were geared to rotate at an angular velocity of approximately $1500^{o}s^{-1}$. The time taken to obtain complete occlusion of the visual field was approximately 38ms.

Figure 2.1 shows a subject positioned on the apparatus with the occluders in the uncovered position.

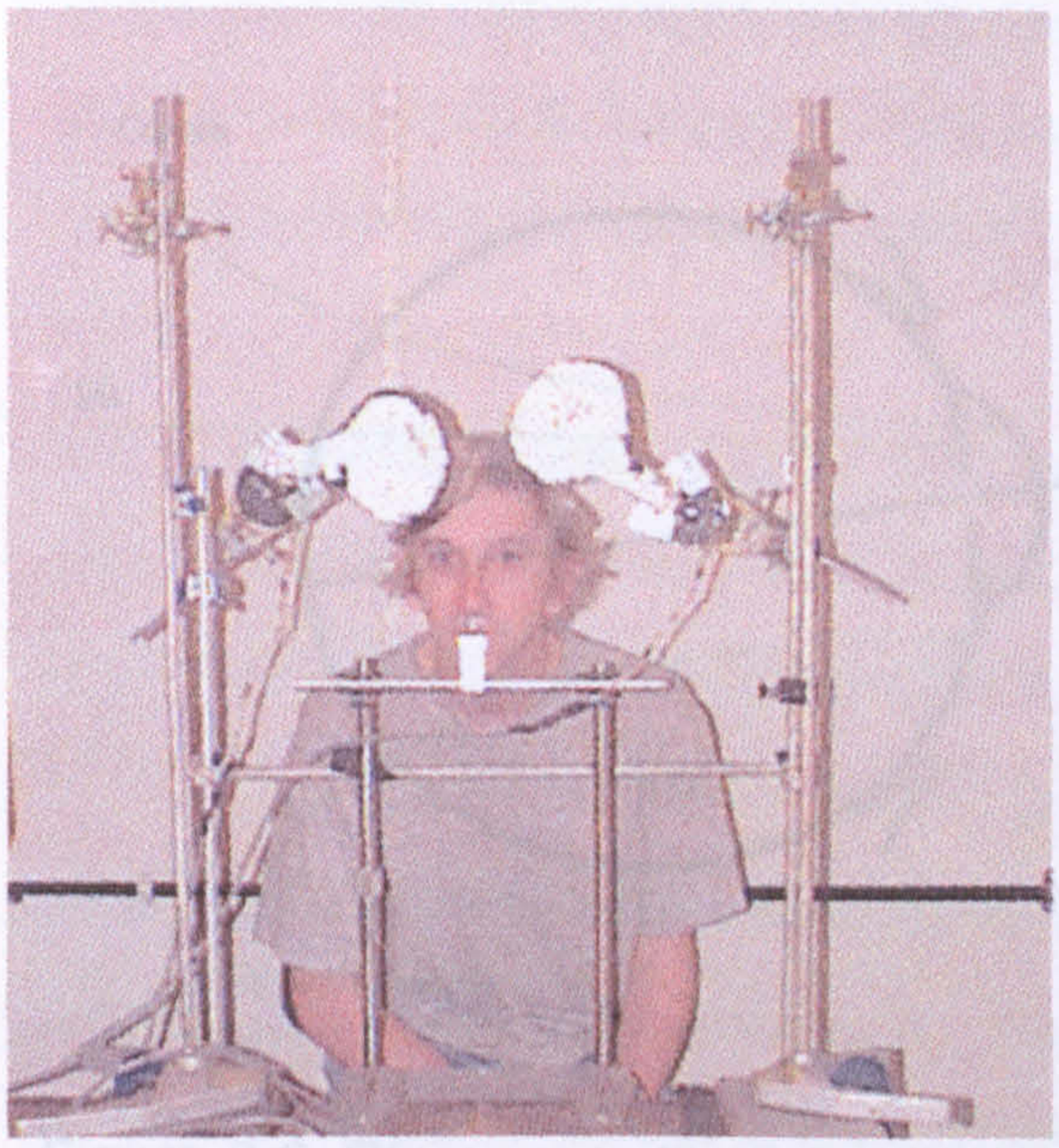


Figure 2.1. Photograph of a subject positioned on the dental bite with the occluders both in the uncovered position.

2.2.1.b. Eye movement recording system

Eye movements were simultaneously recorded for both eyes using an infrared photoelectric method (Haines). Carpenter (1977) has discussed the general principles of this technique. The eye movement recorder consisted of a pair of low-wattage infra-red photo diodes (peak wavelength 880 nm) for each eye mounted on a trial frame and directed at the iris-scleral border, one on each side of the iris (see Figure 2.2). The photo diodes were chopped at 10 kHz to reduce the effect of ambient light.

Simultaneous right and left eye recordings made it possible to analyse binocular eye movements.

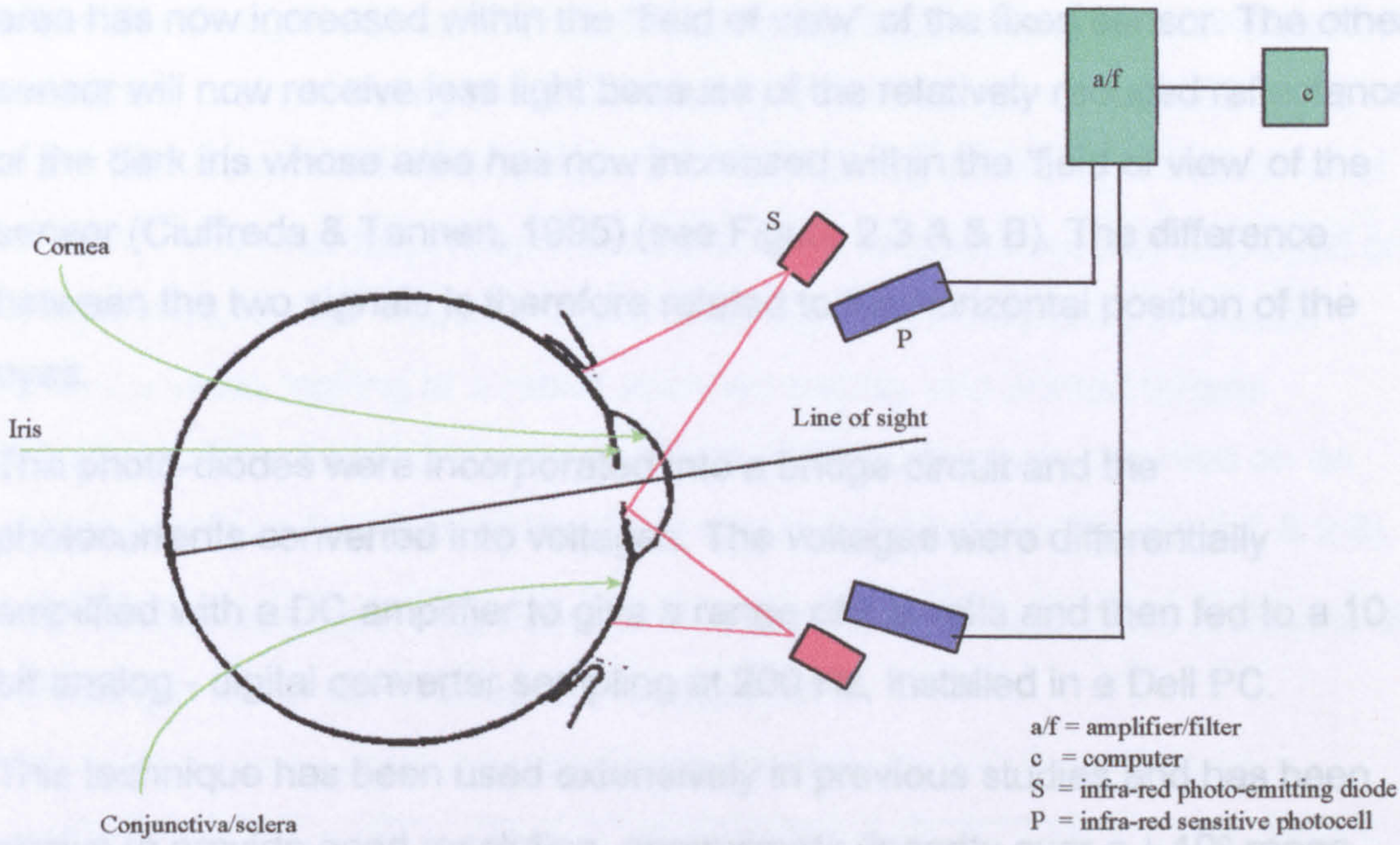


Figure 2.2. Diagram to illustrate the arrangement of an infrared limbal reflection system for recording eye movements.

The infrared sources illuminate the limbal regions of the eye and this is shown schematically in Figure 2.3 A.

Infrared photosensors are positioned in the trial frame to collect light from these same regions (see Figure 2.2).

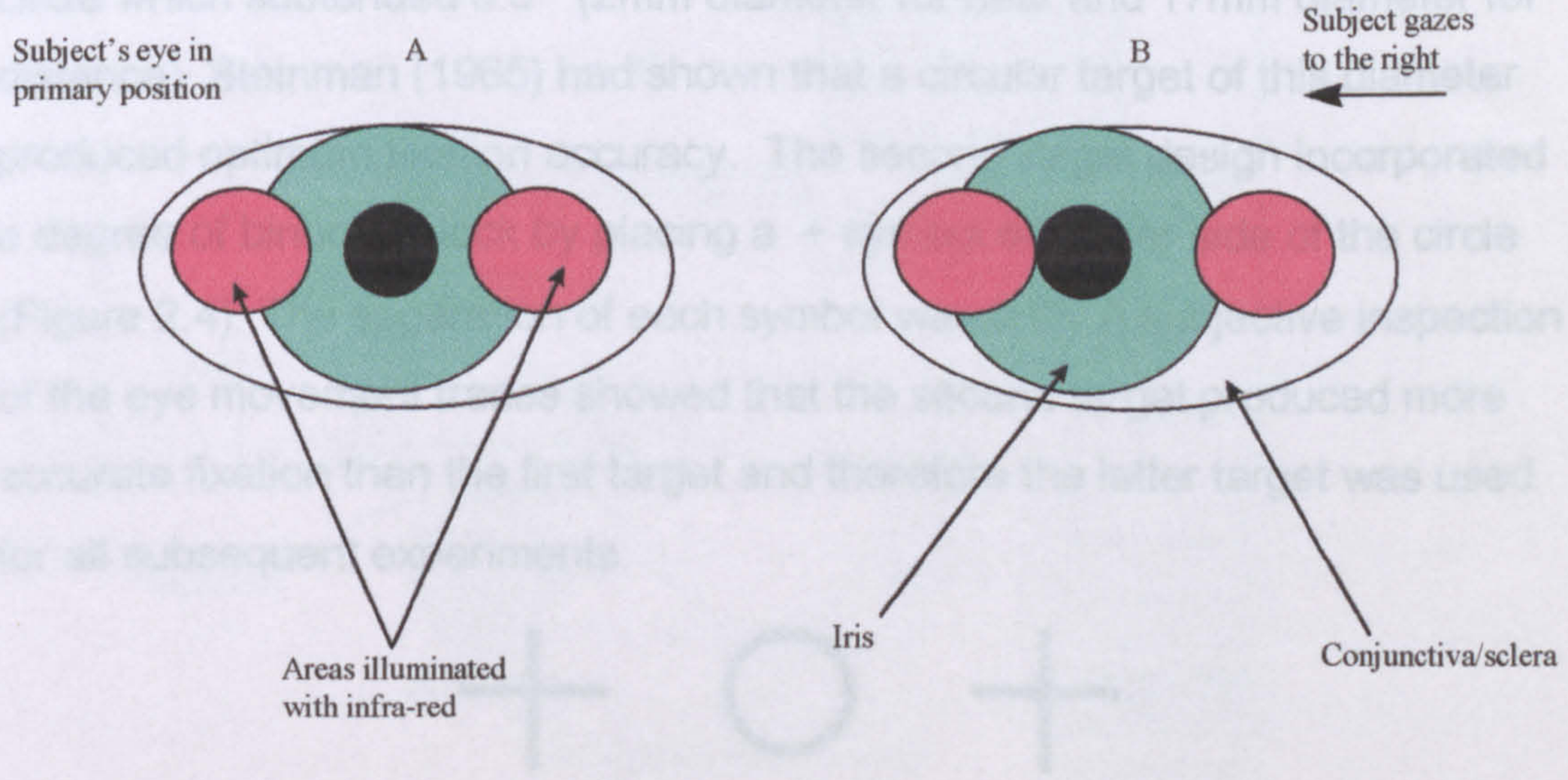


Figure 2.3. Diagrams to illustrate areas of iris/sclera illuminated by infrared light by a pair of infrared emitting diodes.

As the eye rotates horizontally from the mid-line, one sensor will receive more infrared light because of the higher reflectance of the 'white' sclera, whose

area has now increased within the "field of view" of the fixed sensor. The other sensor will now receive less light because of the relatively reduced reflectance of the dark iris whose area has now increased within the 'field of view' of the sensor (Ciuffreda & Tannen, 1995) (see Figure 2.3 A & B). The difference between the two signals is therefore related to the horizontal position of the eyes.

The photo-diodes were incorporated into a bridge circuit and the photocurrents converted into voltages. The voltages were differentially amplified with a DC amplifier to give a range of ± 5 volts and then fed to a 10 bit analog - digital converter sampling at 200 Hz, installed in a Dell PC.

This technique has been used extensively in previous studies and has been shown to provide good resolution, approximate linearity over a ± 10° range and ease of use in a clinical type setting. Infrared light is used as visible light may disturb the patient and affect eye movements.

2.2.1.c. Fixation targets

A pilot study was carried on a small number of subjects using two types of fixation target for both distance and near. The first target consisted of a black circle which subtended 0.3° (2mm diameter for near and 17mm diameter for distance). Steinman (1965) had shown that a circular target of this diameter produced optimum fixation accuracy. The second target design incorporated a degree of binocular lock by placing a + symbol on either side of the circle (Figure 2.4). The separation of each symbol was 0.6°. A subjective inspection of the eye movement traces showed that the second target produced more accurate fixation than the first target and therefore the latter target was used for all subsequent experiments.



Figure 2.4. Diagram illustrating the design of the fixation target used during the automated cover test.

The distant' target was printed on acetate and attached to a white sheet which provided a uniform background subtending a visual angle of 40° x 40°. The sheet was illuminated by a halogen floodlight giving an average luminance of 65 cdm^{-2}. The near target was identical in most respects but was displayed on a high-resolution 20'' monitor. There is no significant difference in fixation accuracy when looking at a raster scanned display and printed targets (Thomson WD, personal communication). The monitor was mounted on an optical bench so that it could be slid in and out of position (Figures 2.5 & 2.8).

The monitor was used in preference to a printed target due to the problems of providing uniform illumination at this distance.

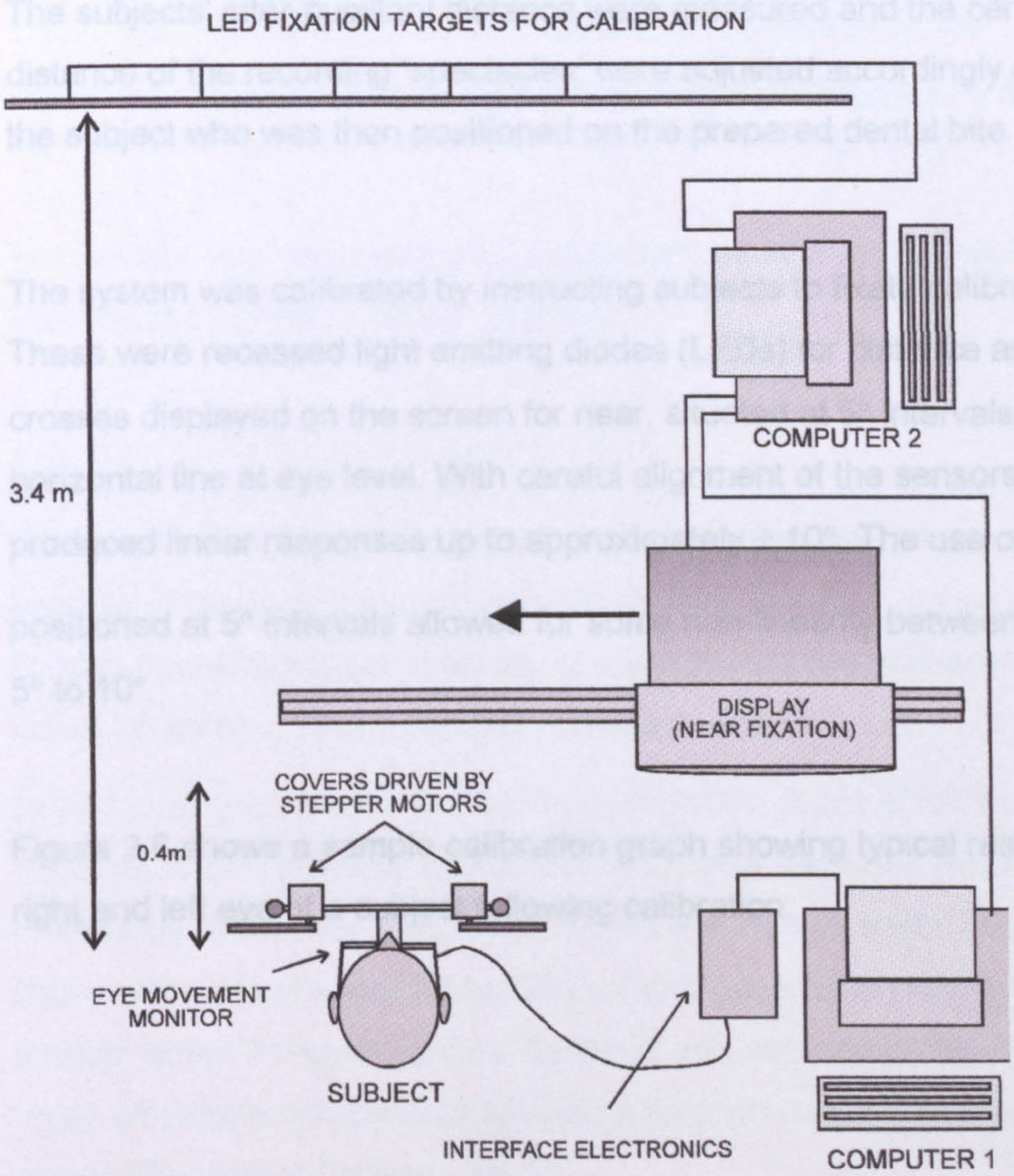


Figure 2.5. Schematic representation of the equipment used during the automated cover test. The subject is viewing the distance fixation target. The computer screen used to present the near fixation target could be seen slid to one side on the optical bench.

2.2.1.d. Positioning the subject and calibration of system

For each subject a dental impression was made using a proprietary thermoplastic impression compound and attached to a metal plate. Each plate could then be attached to an adjustable bar. The dental bite was used to eliminate head movements during eye movement recording. Figure 2.1 illustrates a subject positioned on the dental bite.

After the subject was positioned at the apparatus, the height of the bar was adjusted so that the pupil of the subject's left eye was at the same height as the fixation target (which was the same for the distance and near targets).

The subjects' inter-pupillary distance were measured and the centration distance of the recording 'spectacles' were adjusted accordingly and fitted to the subject who was then positioned on the prepared dental bite.

The system was calibrated by instructing subjects to fixate calibration targets. These were recessed light emitting diodes (LEDs) for distance and small crosses displayed on the screen for near, situated at 5° intervals along a horizontal line at eye level. With careful alignment of the sensors, the system produced linear responses up to approximately ± 10°. The use of targets positioned at 5° intervals allowed for some non-linearity between 0° to 5° and 5° to 10°.

Figure 2.6 shows a sample calibration graph showing typical results for the right and left eye of a subject following calibration.

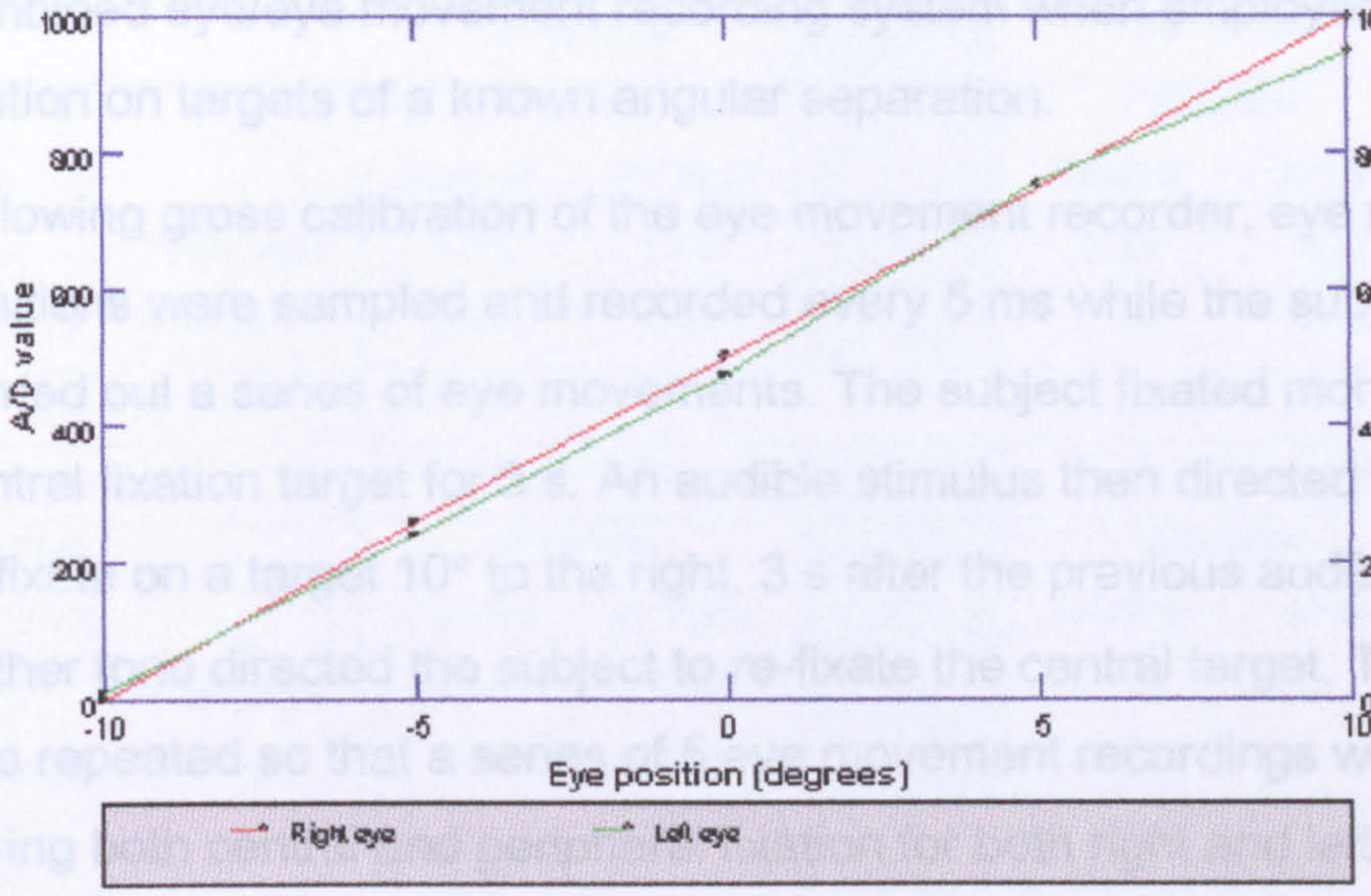


Figure 2.6. Sample calibration graph showing typical results for the right and left eye of a subject following calibration (A/D = analog to digital).

Eye position was ascertained for subsequent recordings by means of linear interpolation between calibration points.

2.2.1.e. Reliability of measurements

The suppliers of the eye movement monitor claimed that the system was capable of measuring eye movements of 'less than 0.1°'. It was difficult to confirm these claims because any study to assess the precision of the system would be confounded by variation in fixation accuracy.

During attempted steady fixation on a stationary object of regard, the eye does not remain perfectly motionless. Both slow and rapid small-amplitude involuntary eye movements occur. These are *tremors* (high frequency (~ 50 Hz) movements of about 20 seconds of arc); s*low drifts* (slow movements through about 5 minutes arc per second); and *microsaccades* (which have a mean amplitude of 5 minutes arc with a duration range of between 10 to 25 msec) (Ciuffreda & Tannen, 1995).

Whilst the possibility was considered of carrying out a repeatability experiment using an artificial eye, this was rejected because it would produce other artefacts and results would be of limited value.

Nevertheless, an experiment was carried out to explore the reliability of the combined eye/eye movement recording system when employed to measure fixation on targets of a known angular separation.

Following gross calibration of the eye movement recorder, eye movement positions were sampled and recorded every 5 ms while the subject (author) carried out a series of eye movements. The subject fixated monocularly on a central fixation target for 3 s. An audible stimulus then directed the subject to re-fixate on a target 10° to the right. 3 s after the previous audible stimulus a further tone directed the subject to re-fixate the central target. This procedure was repeated so that a series of 5 eye movement recordings were obtained during both central and peripheral fixation for both right and left eyes. The procedure was repeated for fixation targets positioned 10° to the left and 5° to right and left.

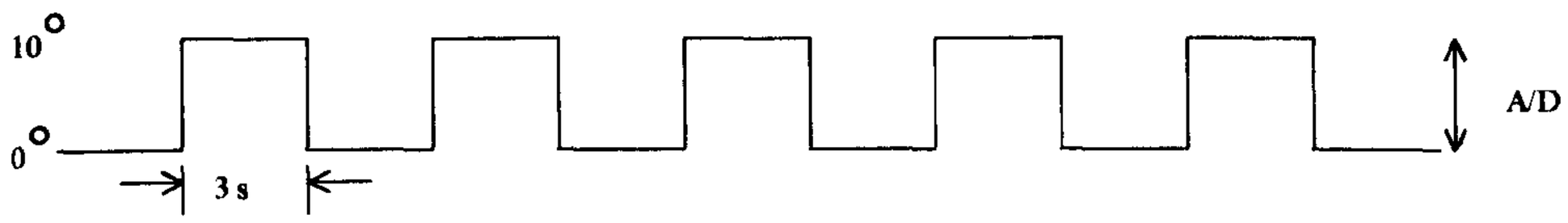


Figure 2.7 Schematic representation of the pattern of eye movements during repeated fixation between a central target (0°) and a target 10° from the centre.

Subsequent analysis was carried out to determine the mean A/D value of each central and peripheral fixation position. These data were used to convert the A/D values into degrees (Figure 2.7). The standard deviation of the eye position was then calculated for each fixation position.

The results are presented in Table.2.1.

		10° to right			10 to left	
Fixation		Fixation			Fixation	
Accuracy		*Central*	*10° right*		*Central*	*10° left*
SD of	Right eye	0.59	0.48		0.29	0.27
mean (°)	Left eye	0.99	0.33		0.62	0.24

		5° to right			5° degrees to left	
Fixation		Fixation			Fixation	
Accuracy		*Central*	*5° right*		*Central*	*5° left*
SD of	Right eye	0.26	0.37		0.13	0.21
mean (°)	Left eye	0.65	0.69		0.36	0.32

Table 2.1. Fixation accuracy of the eye movement recording system/subject combination.

The mean of all the above standard deviations of fixation accuracy measures was 0.43 °. This implies that 95% of the time fixation was within ± 0.86° of the fixation target and represents the combined variability of the eyes and the eye movement recording system. Up to approximately ± 0.5° may explained by fixational eye movements (Ciuffreda & Tannen, 1995). This compares to the approximately 1° resolution of EOG recording techniques (Collewijn, 1999), and claims of a 15" sensitivity for the electromagnetic scleral coil technique (Robinson, 1963). The latter equipment, although more sensitive, was not available to the author.

Ludvigh (1949) and Romano & von Noorden (1971) found that under optimal conditions of illumination and with co-operative patients, the smallest observable change in eye position during the cover test is approximately 2^{Δ}.

2.2.1.f. Automated cover test experiments

For the automated cover test experiments, eye movements were recorded during a distance and near automated cover test while the patient fixated the high contrast targets positioned at either 340 cm or 40 cm respectively. Two networked computers were used to control the experiment (see Figure 2.5).

The first computer was used to acquire the eye movement data while the second computer was used to drive the stepper motors, calibration LEDs and the display screen used for near fixation.

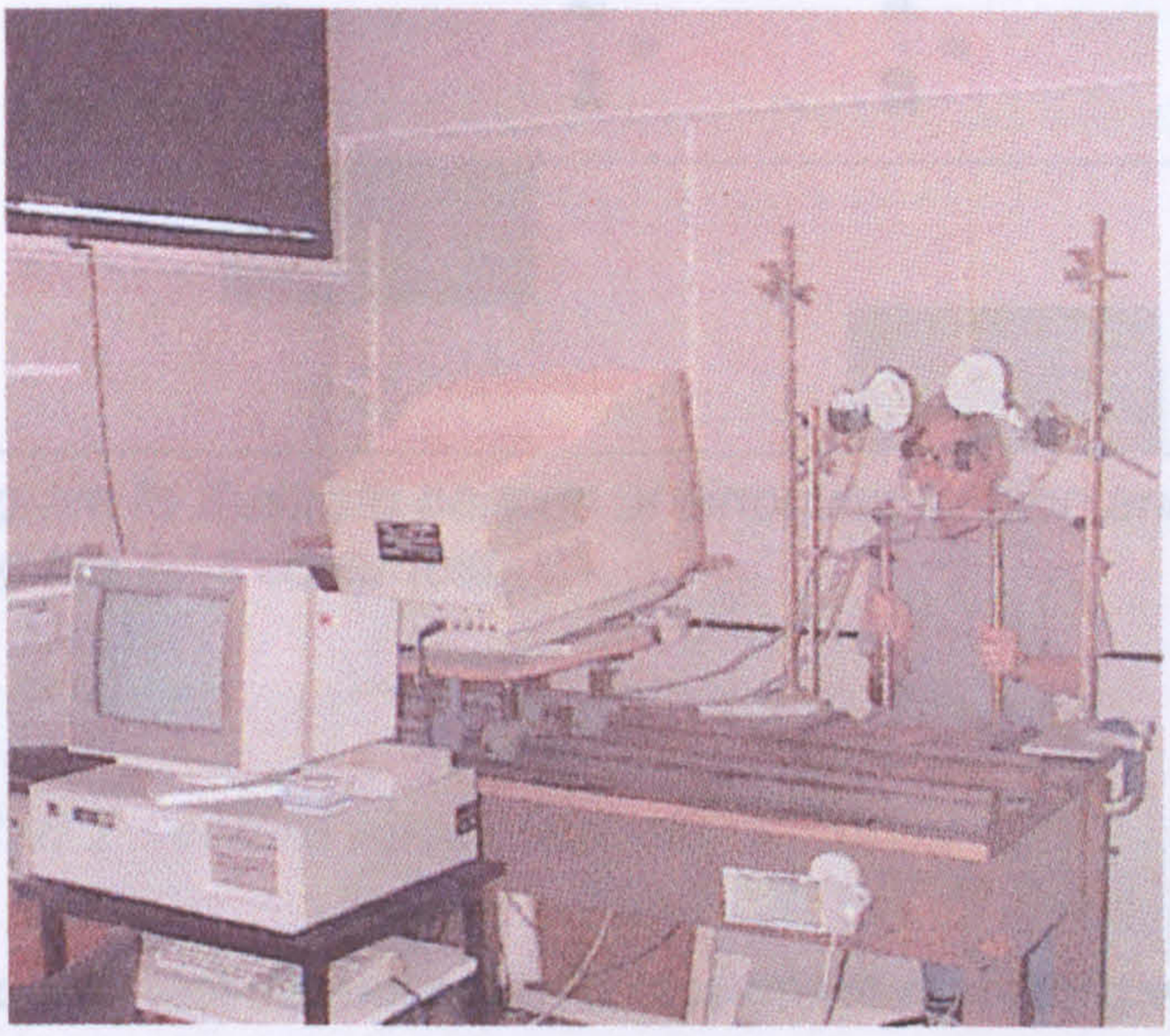


Figure 2.8. Photograph showing the equipment used during the automated cover test. The set up shown is for distance fixation with the near display screen situated out of position.

A total of three cover test protocols were used in the pilot studies. These were a 10 s cover test, a 2 s cover test and an alternate cover test.

I. Automated 10 s cover test

Having set up the subject and calibrated the eye movement recorder for distance, the subject was instructed to fixate the distance target for approximately 30 seconds. The examiner then reminded the subject to concentrate on the fixation target and commenced the automated cover test cycle that was as follows:

- 0 - 4 s no occlusion
- 4 - 14 s right eye occluded
- 14 - 20 s no occlusion
- 20 - 30 s left eye occluded
- 30 - 36 s no occlusion

This is illustrated in Figure 2.9.

The procedure could be repeated for near, with the VDU screen positioned approximately 40 cm from the cornea.

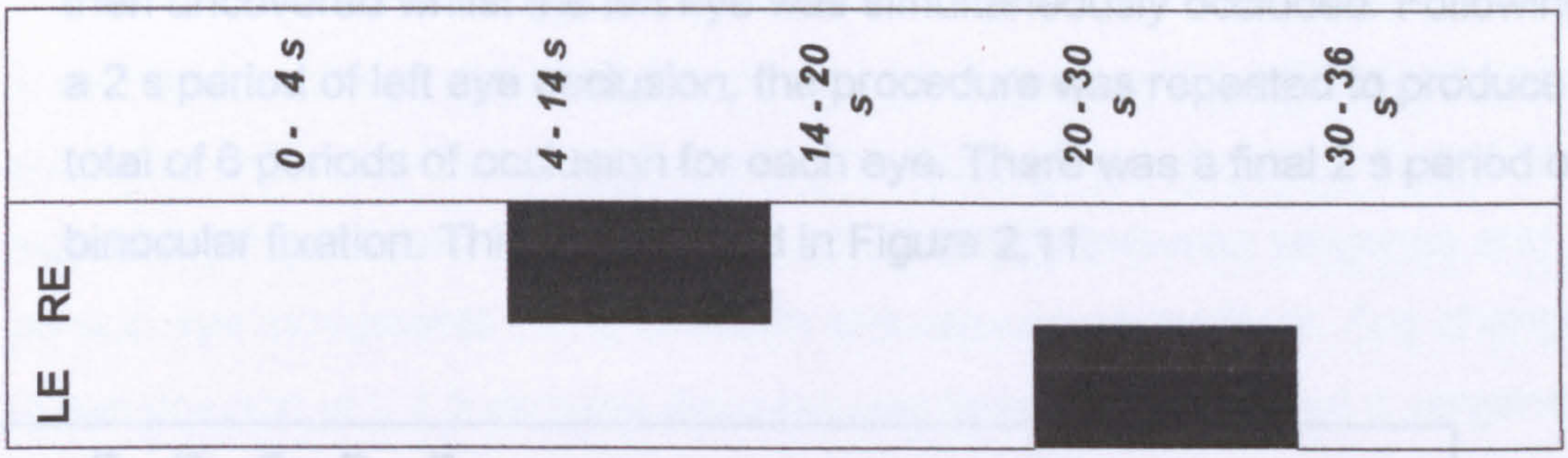
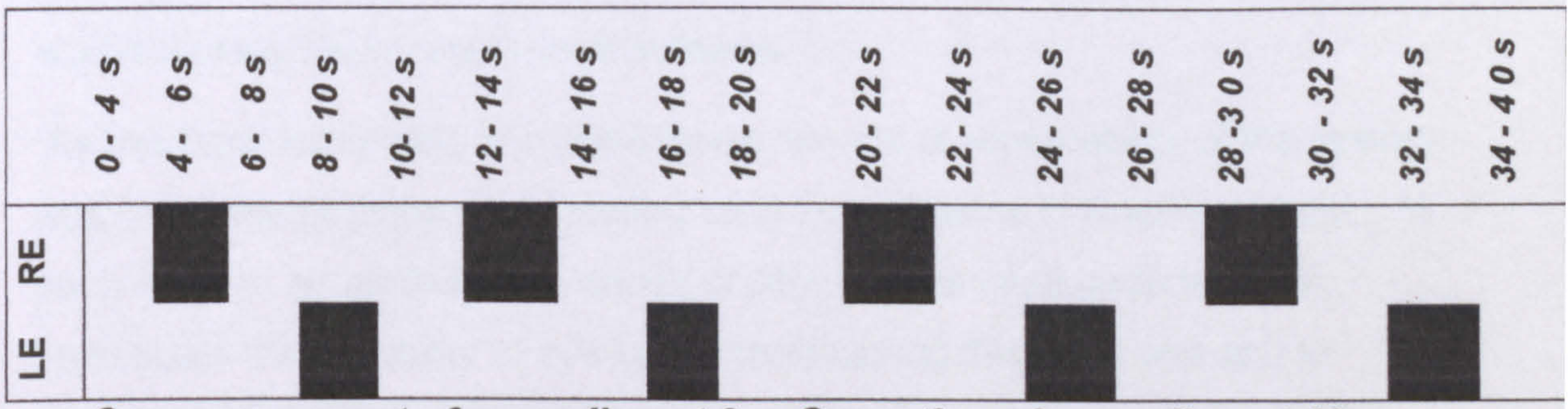


Figure 2.9. Diagram to illustrate periods of binocular fixation and unilateral occlusion during the 10 s cover test.

II. Automated 2 second cover test

For the 2 s cover test sequence, eye movement recording was carried out over a 40 s period. During this time, the computer software operated the automated occluders so as to produce a cycle of 2 s unilateral intermittent occlusion. After an initial 4 s of binocular fixation, there was a repetitive cycle of 2s of right eye occlusion, 2 s of binocular fixation and 2 s left eye occlusion. This cycle was repeated a number of times as described in Figure 2.10.



Commencement of recording at 0 s. Cessation of recording at 40 s

■= period of occlusion

Figure 2.10. Diagram to illustrate periods of binocular fixation and unilateral occlusion during the 2 s cover test.

III. Automated alternate cover test

The conventional, manual alternate cover test has previously been described in Chapter 1.

For the automated alternate cover test, following an initial 4 s period of binocular fixation, the right eye was occluded for 2 s. The right eye was then uncovered whilst the left eye was simultaneously occluded. Following a 2 s period of left eye occlusion, the procedure was repeated to produce a total of 6 periods of occlusion for each eye. There was a final 2 s period of binocular fixation. This is illustrated in Figure 2.11.

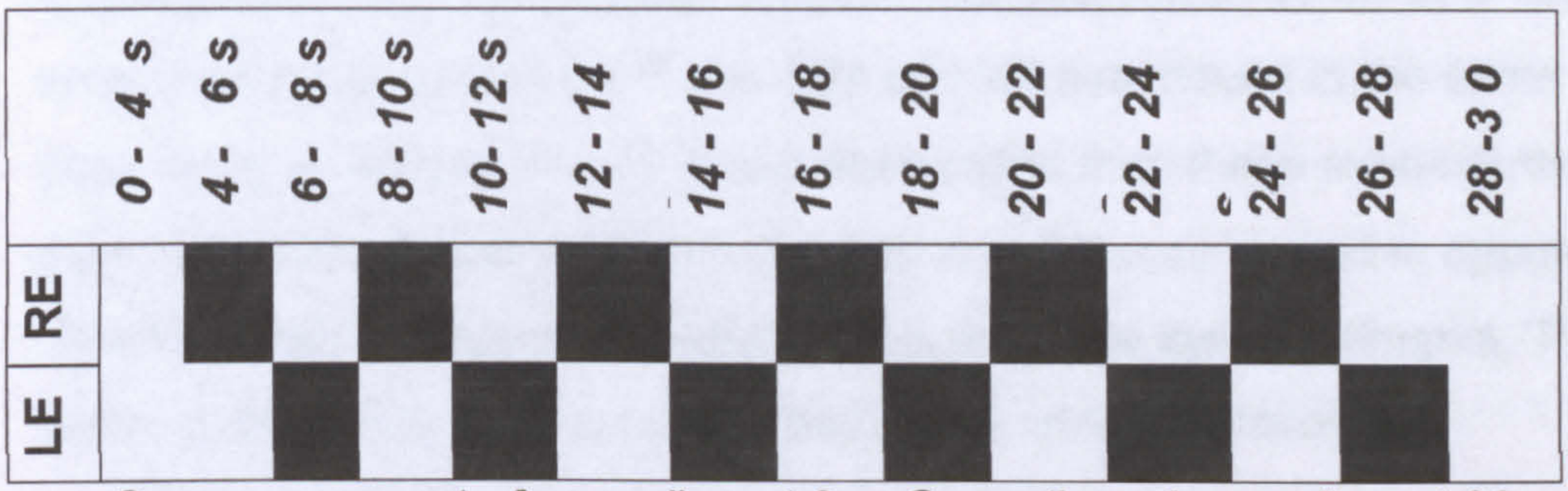


Figure 2.11. Diagram to illustrate periods of binocular fixation and unilateral occlusion during the alternate cover test

2.3 Data analysis

Analysis of each eye movement trace was carried out using a Windows based analysis program written specifically for the study. The software included an algorithm programmed to differentiate automatically between vergence and version eye movements using direction and velocity parameters. Any change in eye position of ≥ 1.5 degrees /second was deemed to be either a vergence or saccadic eye movement. If the right and left eye moved in the same direction at a velocity of ≥ 20.0 degrees/second then these movements were deemed to be saccades. Where the right and left eyes moved in opposite directions then these were deemed to be vergence eye movements. The subsequent traces were automatically colour coded as follows:

- Red = dextroversion (rightward saccade)
- Green = laevoversion (leftward saccade)
- Blue = convergence
- Yellow = divergence

The colour coding was used for guidance although the operator's interpretation was the final arbiter.

The summary measures were recorded in a Microsoft Excel spreadsheet for analysis using Excel and Unistat software.

As has been explained, a precise measurement of repeatability of the system was unattainable because of confounding by variations in fixation accuracy of each subject. Nevertheless, a series of pilot studies were undertaken to investigate the variability of eye movements during the cover test and to investigate the effects of cover/uncover protocols.

2.3.1 Determination of an optimal cover/uncover protocol

2.3.1.a. Introduction

Peli & McCormack (1983) reported that eyes might take up to 8 s to reach a position of equilibrium during the cover test. Cooper (1992) suggested that the

alternate cover test would produce larger phoria amplitudes than a conventional cover test. As has previously been explained in Chapter 1, contemporary wisdom suggests that the eye should be occluded for about 2 s.

A pilot study was carried out to compare the results of a 2 s, 10 s and alternate automated cover test. The author is not aware of any previous comparative studies of this type. The results of this study are presented here because measurements were repeated a number of times thus producing some limited repeatability data.

2.3.1.b. Method

For the purpose of this experiment, a reliable observer (an optometry lecturer/research student) was selected who fulfilled the following criteria:

- asymptomatic;
- no manifest deviation (heterotropia)as assessed by the cover test;
- no history of binocular vision anomalies or orthoptic treatment;
- VA of 6/6 or better with each eye;
- a refractive error of between -0.50 DS (red on duochrome) and +0.75 DS (green on duochrome) with no more than 0.50 DS of spectacle astigmatism and no more than 0.50 D of anisometropia;
- aged between 18 and 35 years;
- not wearing contact lenses;
- no history of ocular disease.

These criteria were used for all subjects recruited for the pilot studies and subsequently for the 'normal' group discussed in Chapter 3. No subjects manifested were either aware of or manifested any obvious incomitancy. A Hess or Lees screen was not carried out to detect any subtle anomalies.

Two second, 10 second and alternating automated cover test routines were carried out with a 5 minute rest between each routine. During each rest, the observer left the room and looked out of the window into the distance to

encourage relaxation of accommodation. The three routines were repeated to produce the six different order combinations (Latin square) as shown in Table 2.2 All of the routines were completed over a ten day period and were carried out between 1000 and 1200 hours. The equipment was re-calibrated before each cover test cycle.

Order of procedures	1st	2nd	3rd
	10 s	2 s	Alternate
	10 s	Alternate	2 s
	2 s	10 s	Alternate
	2 s	Alternate	10 s
	Alternate	10 s	2 s
	Alternate	2 s	10 s

Table 2.2. List of showing the 18 permutations of order of carrying out the three types of cover test.

2.3.1.c. Results

Data was extracted from all 18 eye movement recording sequences. Measurements were taken of phoria amplitude, latency for recovery, and recovery times for each occlusion cycle.

I. The effect of repeated 2 s intermittent monocular occlusion on phoria amplitude

The mean phoria amplitude of all 6 combinations was calculated for each of the four cycles of the 2 s cover test procedure.

Figure 2.12 describes the mean (of 6 permutations) phoria amplitude^{RL} (^{RL} = mean of right and left measurents) for each cycle of the 2 s distance cover test.

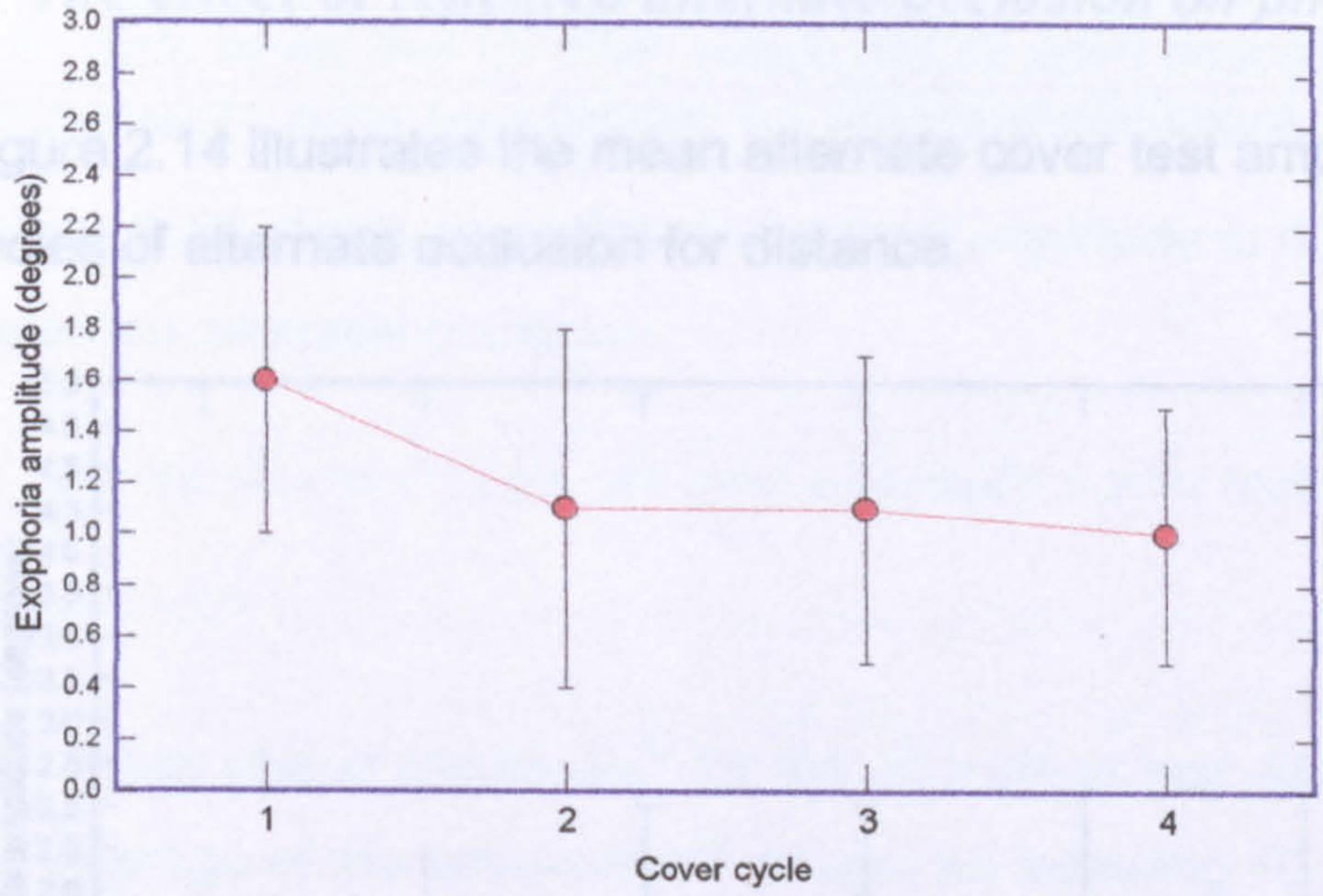


Figure 2.12. Scatter plot showing mean phoria amplitude^{RL} for each cycle of the repeated 2 s distance cover test. (Each datum point describes the average of 6 measurements; error bars denote ± 1 standard deviation).

Figure 2.13 describes the mean (of 6 permutations) phoria amplitude^{RL} for each cycle of the 2 s near cover test.

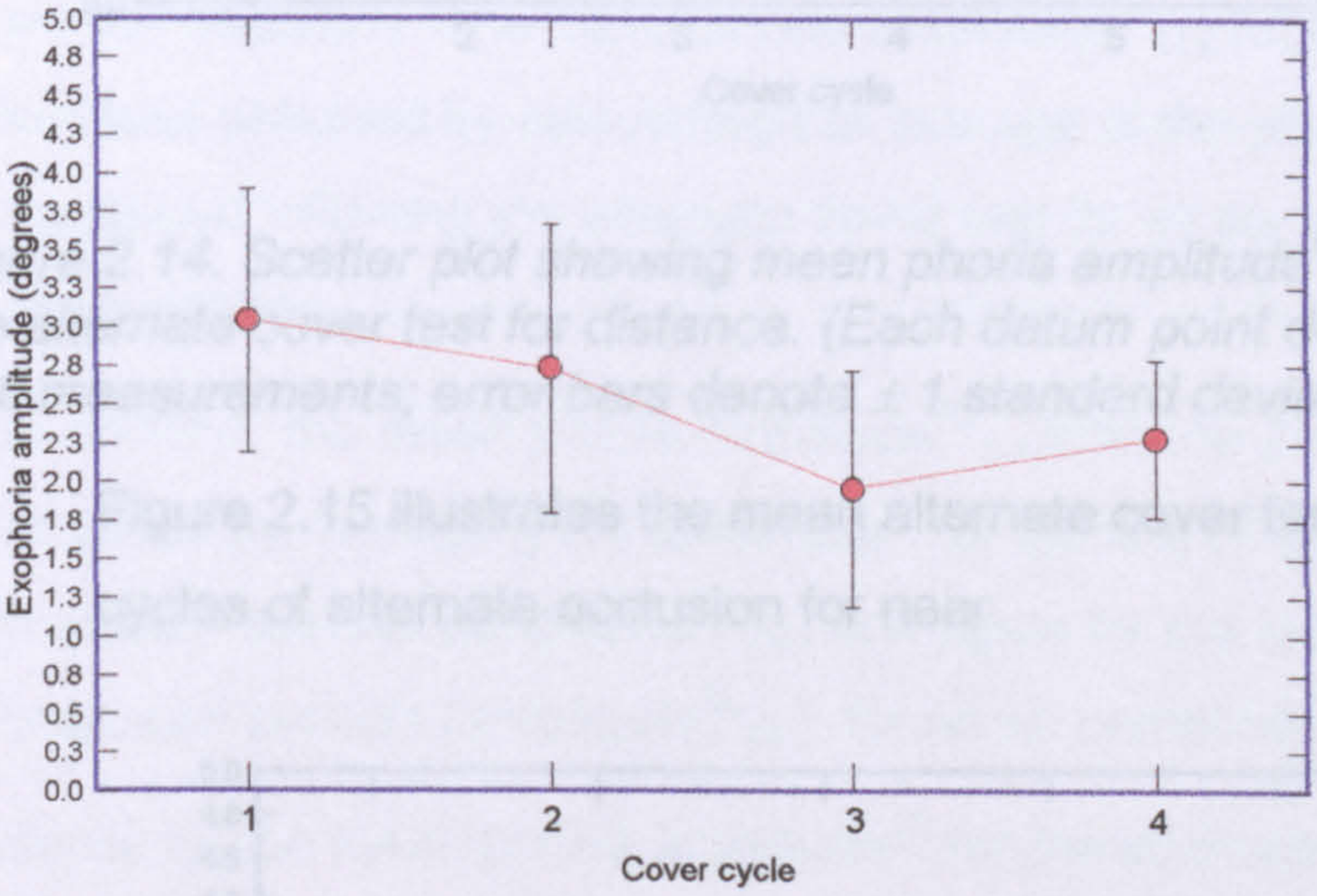


Figure 2.13. Scatter plot showing mean phoria amplitude^{RL} for each cycle of the repeated 2 s near cover test. (Each datum point dscribes the average of 6 measurements; error bars denote ± 1 standard deviation).

Figures 2.12 and 2.13 show a trend suggesting a reduction in phoria amplitude^{RL} with the number of times the cover test is carried out.

II. The effect of repeated alternate occlusion on phoria amplitude

Figure 2.14 illustrates the mean alternate cover test amplitude over six cycles of alternate occlusion for distance.

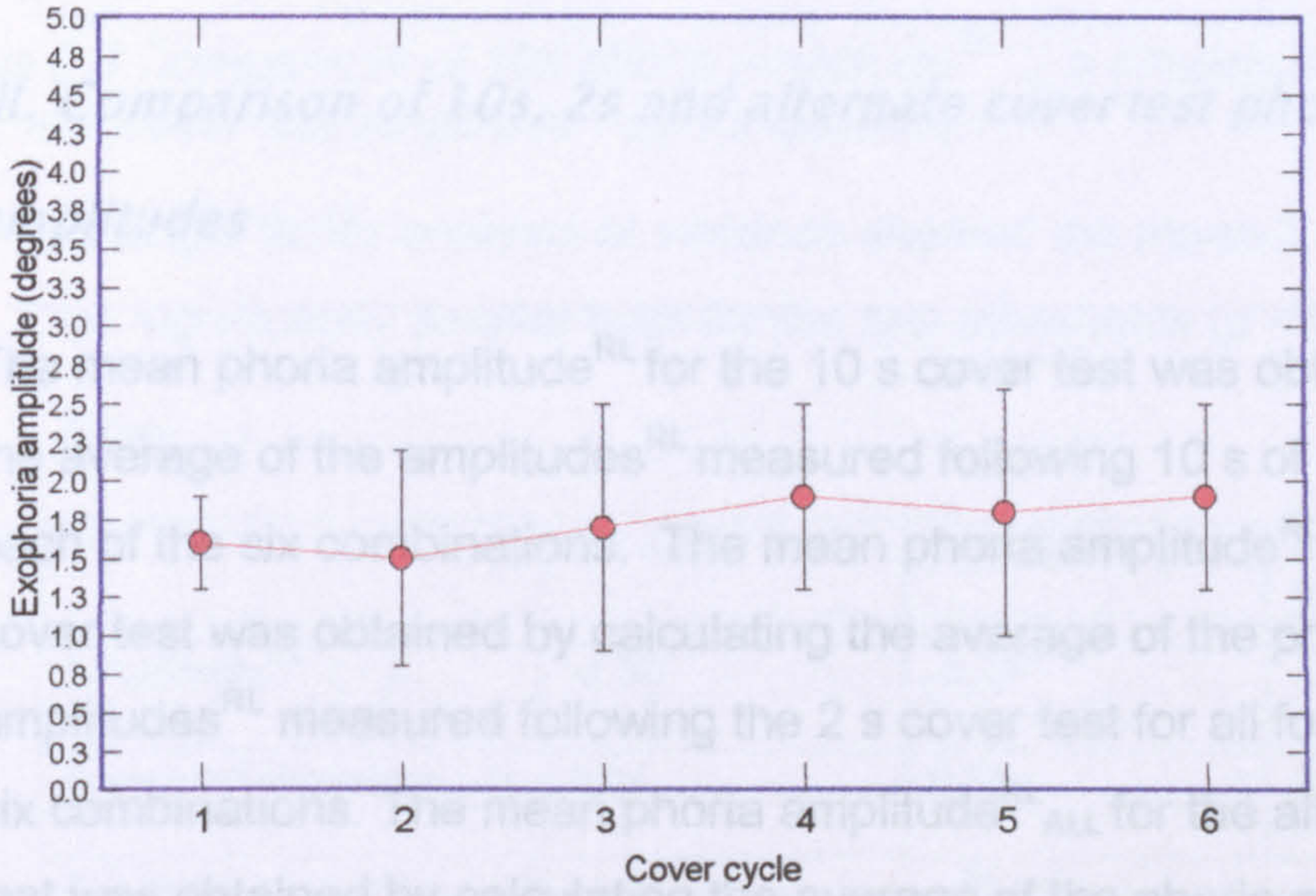


Figure 2.14. Scatter plot showing mean phoria amplitude^{RL} for each cycle of the alternate cover test for distance. (Each datum point describes the average of 6 measurements; error bars denote ± 1 standard deviation).

Figure 2.15 illustrates the mean alternate cover test amplitude over six cycles of alternate occlusion for near.

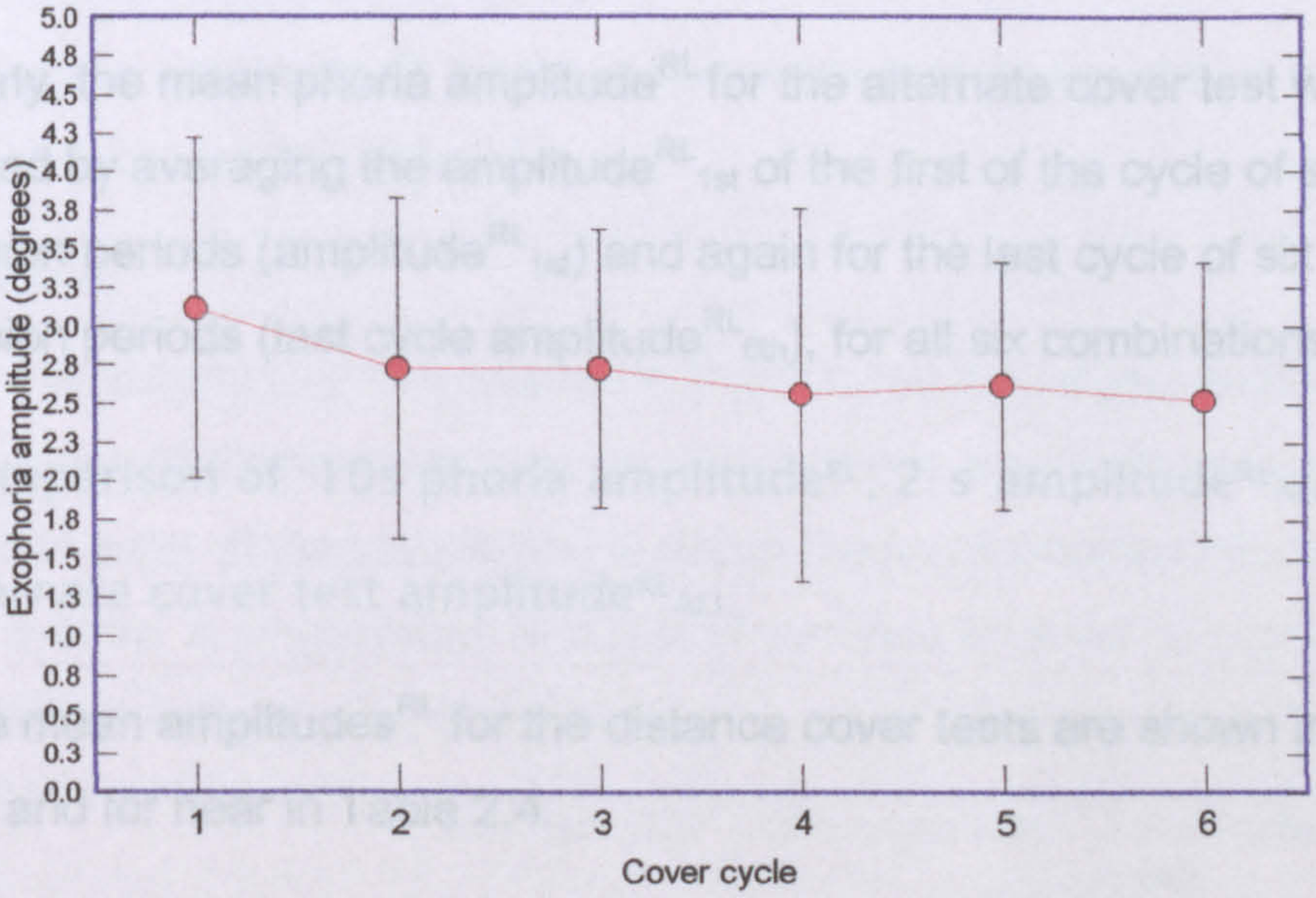


Figure 2.15. Scatter plot showing mean phoria amplitude^{RL} for each cycle of the alternate cover test for near. (Each datum point describes the average of 6 measurements; error bars denote ± 1 standard deviation).

Figure 2.15 shows a trend for the phoria amplitude to decrease slightly with repeated alternate occlusion.

III. Comparison of 10s, 2s and alternate cover test phoria amplitudes

The mean phoria amplitude^{RL} for the 10 s cover test was obtained by taking the average of the amplitudes^{RL} measured following 10 s of occlusion, at each of the six combinations. The mean phoria amplitude$^{RL}_{ALL}$ for the 2 s cover test was obtained by calculating the average of the phoria amplitudes^{RL} measured following the 2 s cover test for all four cycles and six combinations. The mean phoria amplitude$^{RL}_{ALL}$ for the alternate cover test was obtained by calculating the average of the phoria amplitudes^{RL} measured following the alternate cover test for all six cycles and six combinations.

In addition, the mean phoria amplitude$^{RL}_{1st}$ from the 2 s cover test was obtained by averaging the amplitude^{RL} of the first of the cycle of four occlusion periods (amplitude$^{RL}_{1st}$) and again for the last cycle of four occlusion periods (amplitude$^{RL}_{4th}$), for all six combinations.

Similarly, the mean phoria amplitude^{RL} for the alternate cover test was obtained by averaging the amplitude$^{RL}_{1st}$ of the first of the cycle of six occlusion periods (amplitude$^{RL}_{1st}$) and again for the last cycle of six occlusion periods (last cycle amplitude$^{RL}_{6th}$), for all six combinations.

Comparison of 10s phoria amplitude^{RL}, 2 s amplitude$^{RL}_{ALL}$, and alternate cover test amplitude$^{RL}_{ALL}$

The mean amplitudes^{RL} for the distance cover tests are shown in Table 2.3 and for near in Table 2.4.

Distance

	2 s cover test	10 s cover test	Alternate cover test
Amplitude^{RL} (°)	-1.17	-1.96	-1.73
Standard deviation (°)	0.69	1.26	0.63

Table 2.3 Comparison of 10s phoria amplitude^{RL}, 2 s amplitude$^{RL}{}_{ALL,,}$ and alternate cover test amplitude$^{RL}{}_{ALL}$ for distance.

A single factor analysis of variance showed the mean 2 s amplitude to be significantly smaller than for the two other tests ($p < 0.05$).

Near

	2 s cover test	10 s cover test	Alternate cover test
Amplitude^{RL} (°)	-2.50	-3.33	-2.72
Standard deviation (°)	0.84	0.91	0.94

Table 2.4 Comparison of 10s phoria amplitude^{RL}, 2 s amplitude$^{RL}{}_{ALL,,}$ and alternate cover test amplitude$^{RL}{}_{ALL}$ for near.

A single factor analysis of variance showed no significant differences between the three tests.

Comparison of 10s phoria amplitude^{RL}, firs t cycle 2 s amplitude $^{RL}{}_{1st}$ and first cycle alternate cover test amplitude $^{RL}{}_{1st}$.

The mean amplitudes^{RL} for the distance cover tests are shown in Table 2.5 and for near in Table 2.6.

Distance

For this subject the 10 s amplitude ^{RL} produced the largest amplitude. However, all three procedures produced phoria amplitudes of the same clinical order of magnitude and to within the resolution/reproducibility of the system. A single factor analysis of variance showed no significant difference between the three tests.

	2 s cover test	10 s cover test	Alternate cover test
Amplitude^{RL} (°)	-1.62	-1.96	-1.55

Standard deviation (°)	0.59	1.26	0.30

Table 2.5. Comparison of 10 s distance amplitude^{RL} and first cycle 2 s and first cycle alternate distance cover test phoria amplitudes $^{RL}{}_{1st}$.

Near

There were similar findings for the near cover test with all three cover test procedures produced phoria amplitudes of the same clinical order of magnitude.

	2 s cover test	**10 s cover test**	**Alternate cover test**
Amplitude^{RL} (°)	-3.04	-3.33	-3.17
Standard deviation (°)	0.85	0.91	1.13

Table 2.6. Comparison of 10 s near amplitudes^{RL} and first cycle 2 s and first cycle alternate near cover test phoria amplitudes $^{RL}{}_{1st}$.

A single factor analysis of variance showed no significant difference between the three tests.

Comparison of 10s amplitude^{RL}, last cycle 2 s amplitude $^{RL}{}_{4th}$, and last cycle alternate cover test phoria amplitudes $^{RL}{}_{6th}$

The mean amplitudes^{RL} for the distance cover test are shown in Table 2.7 and for near in Table 2.8.

Distance

A comparison of the 10 s and last cycle measures of the 2 s and alternate cover test for distance showed the 10 s cover test and the alternate cover test producing the largest amplitudes^{RL} and the 2 s cover test producing the smallest.

	2 s cover test	**10 s cover test**	**Alternate cover test**
Amplitude^{RL} (°)	-0.98	-1.96	-1.94
Standard deviation (°)	0.59	1.26	0.57

Table 2.7. Comparison of 10 s distance amplitude^{RL} , last cycle 2 s amplitude$^{RL}{}_{4th}$, and last cycle alternate distance cover test phoria amplitudes$^{RL}{}_{6th}$

Although the differences were of interest, all three procedures produced phoria amplitudes of a similar clinical order of magnitude. A single factor analysis of variance showed no significant difference between the three tests.

Near

A comparison of the 10 s and last cycle measures of the 2 s and alternate cover test for near showed the 10 s cover test produced the largest amplitude^{RL} and the 2 s cover test produced the smallest. However, again all three procedures produced phoria amplitudes of a similar order of magnitude and a single factor analysis of variance showed no significant difference between the three tests.

	2 s cover test	**10 s cover test**	**Alternate cover test**
Amplitude^{RL} (°)	-2.28	-3.33	-2.52
Standard deviation (°)	0.51	0.91	0.90

Table 2.8. Comparison of 10 s near amplitude^{RL} , last cycle 2 s amplitude^{RL} 4th, and last cycle alternate distance cover test phoria amplitudes^{RL} 6th.

IV. Latency

10 s cover test

Distance

Of the 12 possible latency measures, 1 was not available due to a blink at the critical time. 2 further measures were discarded because they were apparently < 60 ms (see page 22). The mean latency for the remaining data (n = 9) was 170 ms (SD = 50 ms; range = 100 ms to 230 ms).

Near

For near, 2 latency data were unavailable due to blinks and 1 was discarded because the time was < 60 ms. The mean latency for the remaining data (n = 9) was 190 ms (SD = 120 ms; range = 90 ms to 400 ms).

Table 2.9 lists the average distance and near latency time for the right and left eyes at each cycle of the 2 s cover test. Standard deviations are shown in brackets. Each latency time listed was the average of 6 measures unless the standard deviation is followed by a superscript annotation showing the number of measures available for analysis. Missing data was due to blinks occurring at critical times in the recording procedure.

	Distance		Near	
Cover cycle	**Right eye**	**Left eye**	**Right eye**	**Left eye**
1	190 (SD = 70) [4]	300 (SD = 290) [4]	100 (SD = 20)	170 (SD = 10)
2	170 (SD = 50) [5]	200 (SD = 40) [2]	140 (SD = 10)	110 (SD = 30) [5]
3	170 (SD = 30) [3]	210 (SD = 60) [2]	120 (SD = 10)	100 (SD = 10) [5]
4	180 (SD = 60) [4]	180 (SD = 70) [4]	130 (SD = 10) [5]	110 (SD = 20) [5]

Table 2.9 Mean latency times (ms) following 2 s cover test.

Distance

Of the 48 possible latency measures, 20 were unavailable due to blinks. The mean latency for the remaining data (n = 28) was 200 ms (SD = 120 ms; range = 90 ms to 730 ms).

Figure 2.16 illustrates the mean latency with repeated occlusion during the distance 2 s cover test.

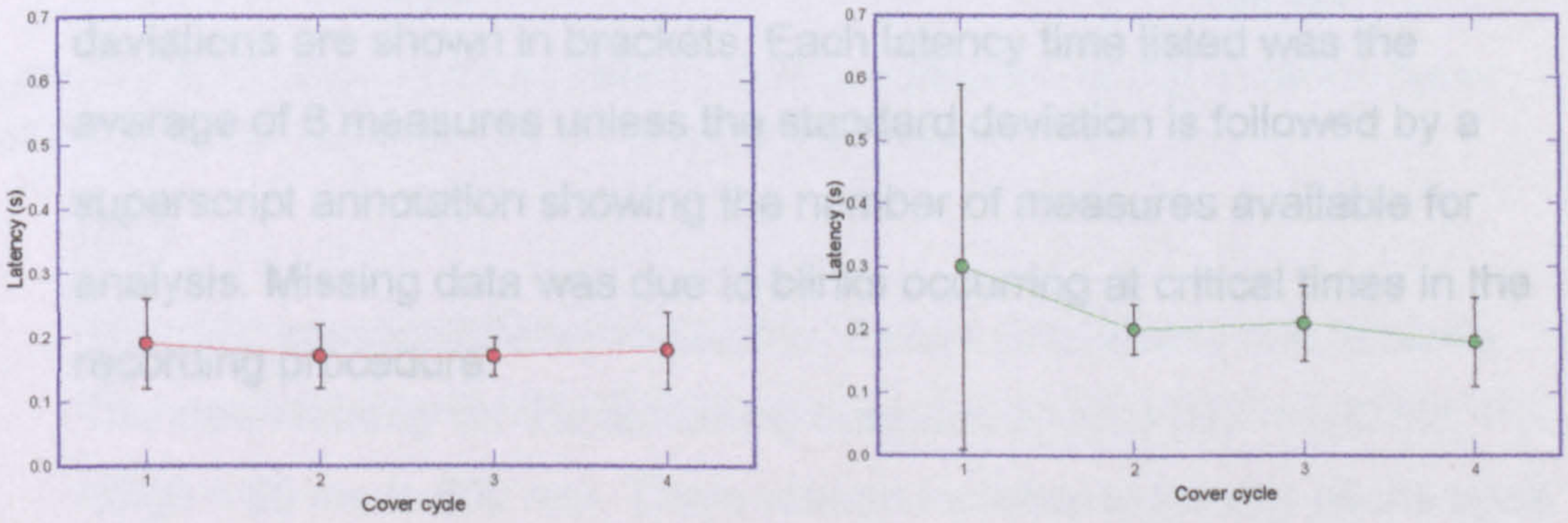


Figure 2.16. Change in mean latency as the distance 2s cover test cycle progresses. (Error bars denote ± 1 standard deviation).

Near

Of the 48 possible latency measures, 4 were unavailable due to blinks. The mean latency for the remaining data (n = 44) was 120 ms (SD = 30 ms; range = 80 ms to 180 ms).

Figure 2.17 illustrates the mean latency with repeated occlusion during the near 2s cover test.

These results did not suggest any trends for a change in latency time with repeated 2 s cover tests.

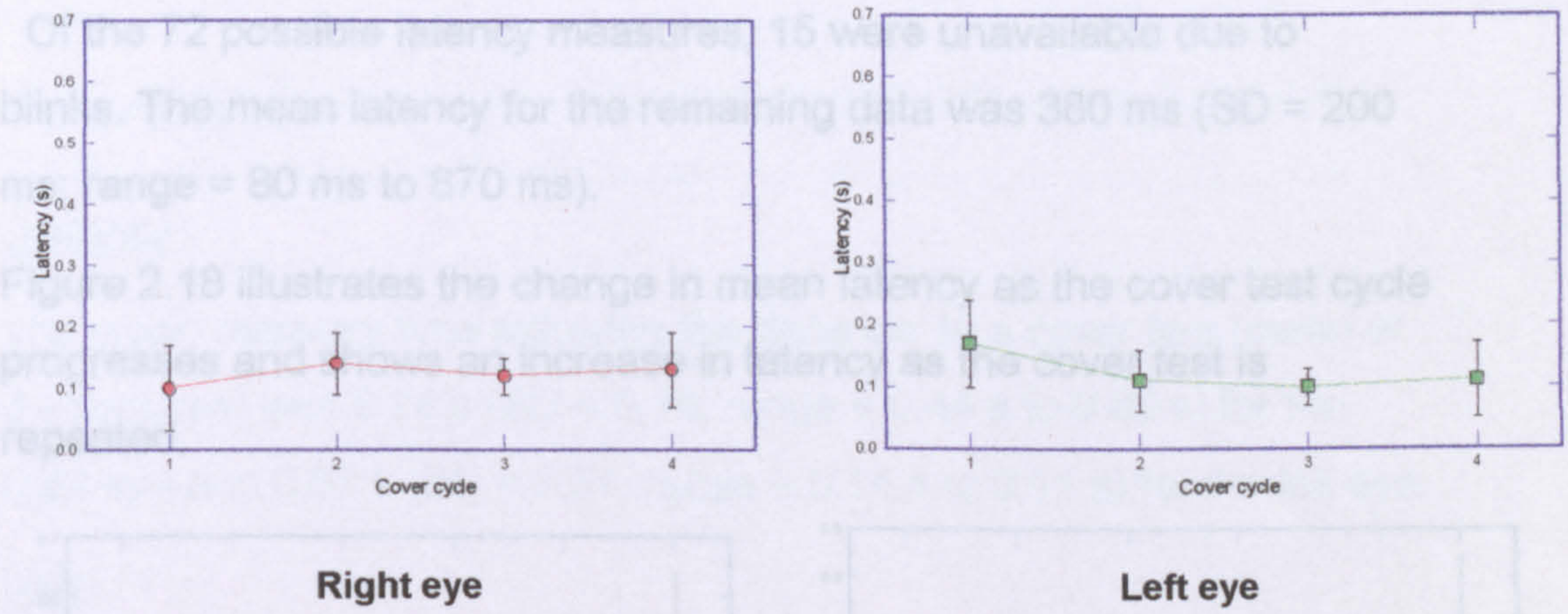


Figure 2.17.Change in mean latency as the near 2s cover test cycle progresses. (Error bars denote ± 1 standard deviation).

Alternate cover test

Table 2.10 lists the average distance and near latency times, for the right and left eyes, at each cycle of the alternate cover test. Standard deviations are shown in brackets. Each latency time listed was the average of 6 measures unless the standard deviation is followed by a superscript annotation showing the number of measures available for analysis. Missing data was due to blinks occurring at critical times in the recording procedure.

	Distance		Near	
Cover cycle	**Right eye**	**Left eye**	**Right eye**	**Left eye**
1	170 (SD = 110) [5]	140 (SD = 40) [4]	110 (SD = 10)	260 (SD = 270)
2	220 (SD = 20) [4]	240 (SD = 60) [5]	190 (SD = 80) [5]	180 (SD = 70)
3	300 (SD = 50)	420 (SD = 280) [5]	240 (SD = 80) [5]	310 (SD = 70) [5]
4	390 (SD = 100) [5]	480 (SD = 100) [5]	300 (SD = 130)	450 (SD = 80)
5	440 (SD = 130) [5]	610 (SD = 70) [4]	360 (SD = 190)	440 (SD = 210)
6	550 (SD = 90)	690 (SD = 150) [3]	530 (SD = 120)	520 (SD = 230)

Table 2.10. Mean latency times (ms) following the alternate cover test.

Distance

Of the 72 possible latency measures, 15 were unavailable due to blinks. The mean latency for the remaining data was 380 ms (SD = 200 ms; range = 80 ms to 870 ms).

Figure 2.18 illustrates the change in mean latency as the cover test cycle progresses and shows an increase in latency as the cover test is repeated.

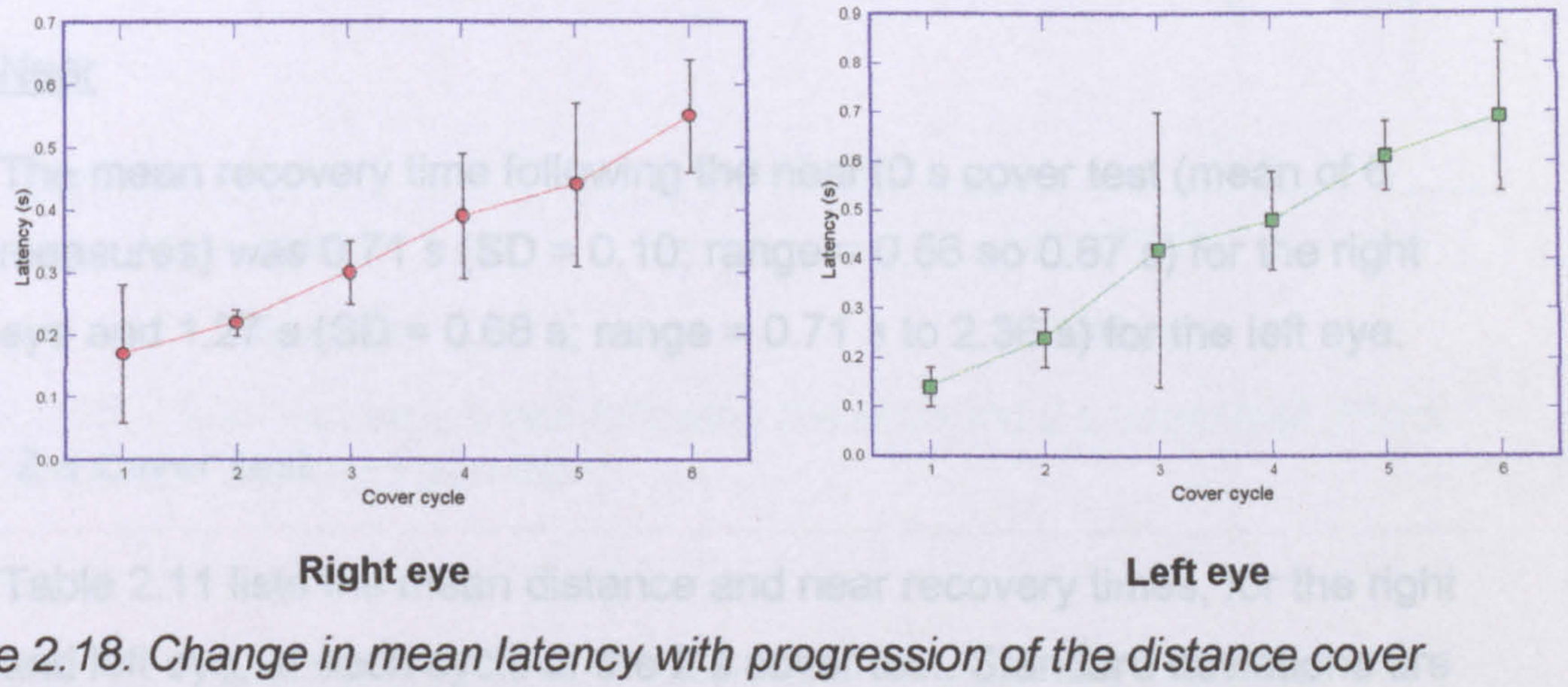
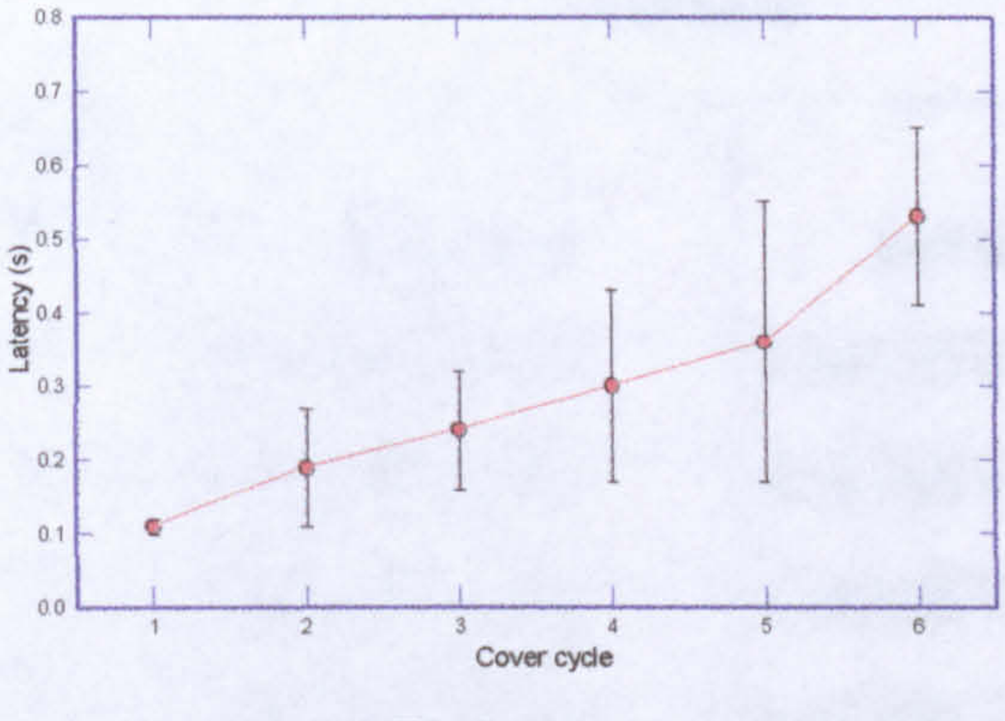
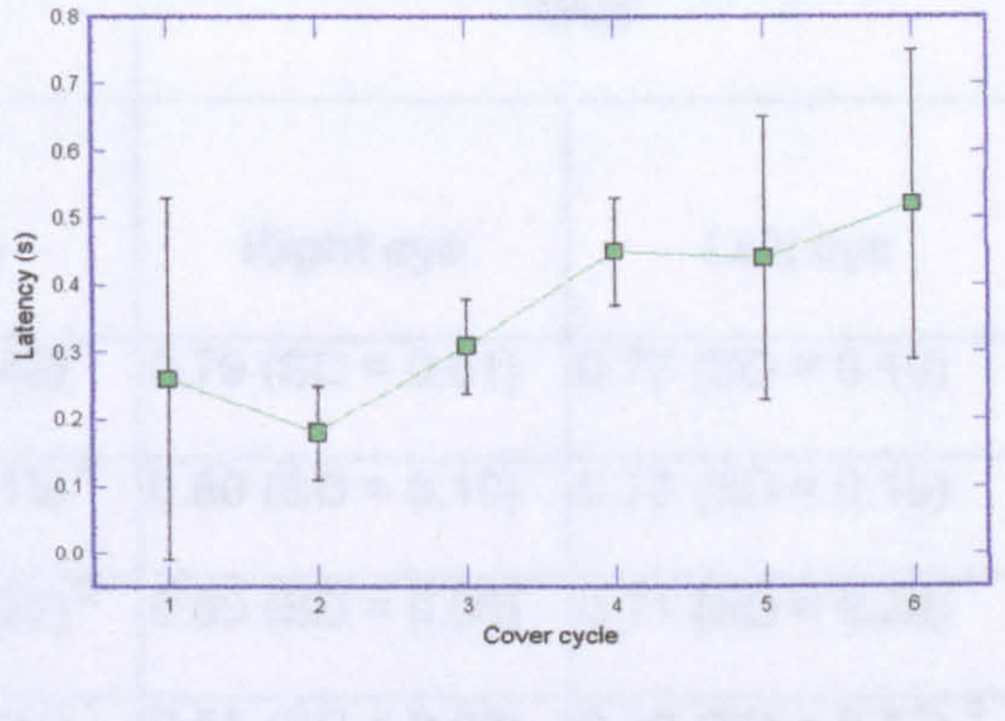


Figure 2.18. Change in mean latency with progression of the distance cover test cycle (Error bars denote ± 1 standard deviation).

Near

Of the 72 possible latency measures, 3 were unavailable due to blinks. The mean latency for the remaining data was 340 ms (SD = 200 ms; range = 80 ms to 800 ms). There was an increase in latency as the cycle of the cover test progressed and this is shown in Figure 2.19.





Right eye **Left eye**

Figure 2.19. Change in mean latency with progression of the near cover test cycle. (Error bars denote ± 1 standard deviation).

V. Recovery time

10 s cover test

Distance

The mean recovery time following the distance 10 s cover test (mean of 6 measures) was 0.74 s (SD = 0.16; range = 0.44 s to 0.88 s) for the right eye and 0.67 s (SD = 0.37; range = 0.14 s to 0.11 s) for the left eye.

Near

The mean recovery time following the near10 s cover test (mean of 6 measures) was 0.71 s (SD = 0.10; range = 0.56 so 0.87 s) for the right eye and 1.27 s (SD = 0.68 s; range = 0.71 s to 2.36 s) for the left eye.

2 s cover test

Table 2.11 lists the mean distance and near recovery times, for the right and left eye, at each cycle of the 2 s cover test. Standard deviations are shown in brackets. Each recovery time listed was the average of 6 measures unless the standard deviation is followed by a superscript annotation showing the number of measures available for analysis. Missing data was due to blinks occurring at critical times in the recording procedure.

	Distance		Near	
Cover cycle	**Right eye**	**Left eye**	**Right eye**	**Left eye**
1	0.55 (SD = 0.15)	0.76 (SD = 0.42) [3]	0.79 (SD = 0.01)	0.77 (SD = 0.10)
2	0.46 (SD = 0.15)	0.75 (SD = 0.15) [4]	0.80 (SD = 0.10)	0.78 (SD = 0.15)
3	0.52 (SD = 0.13)	0.50 (SD = 0.22) [5]	0.69 (SD = 0.06)	0.71 (SD = 0.26)
4	0.54 (SD = 0.15)	0.51 (SD = 0.24) [4]	0.66 (SD = 0.02)	0.79 (SD = 0.17) [5]

Table 2.11. Recovery times (s) following 2 s cover test.

Distance

Figure 2.20 illustrates the mean recovery time for each eye at each of the four cycles of the distance 2 s cover test.

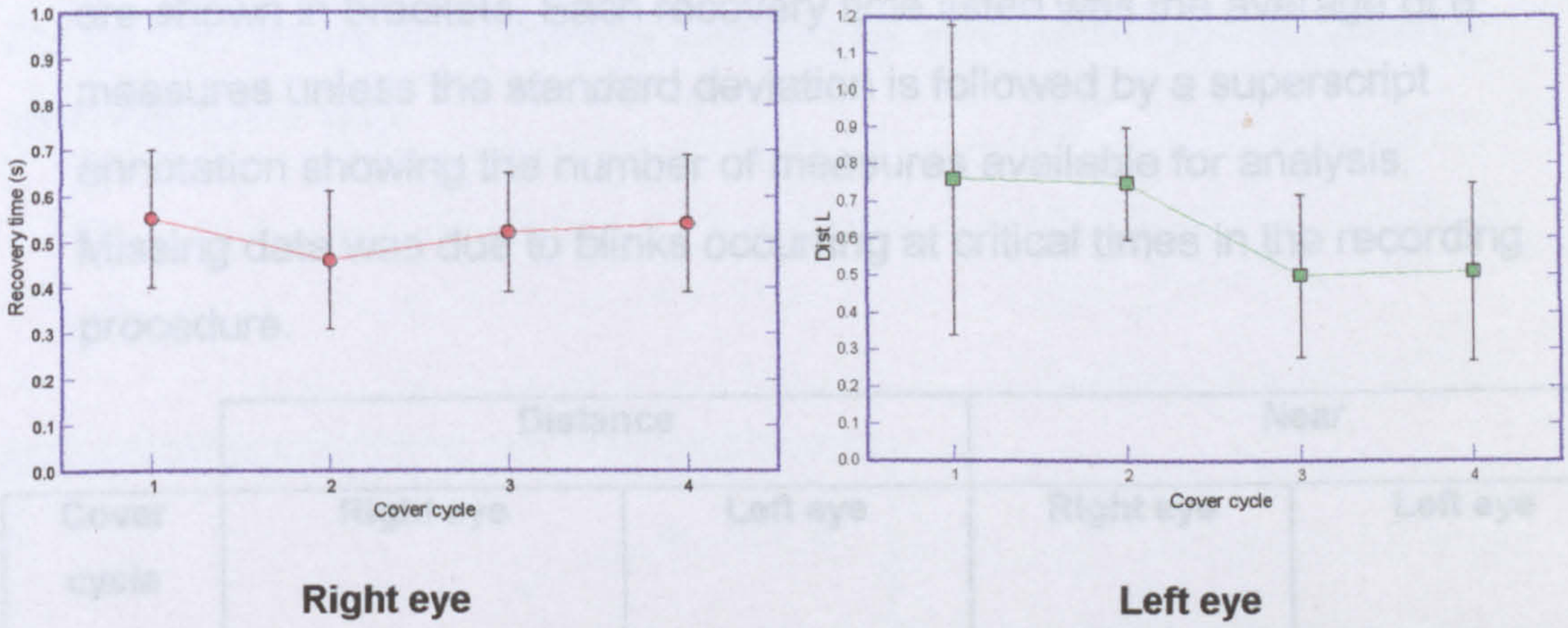
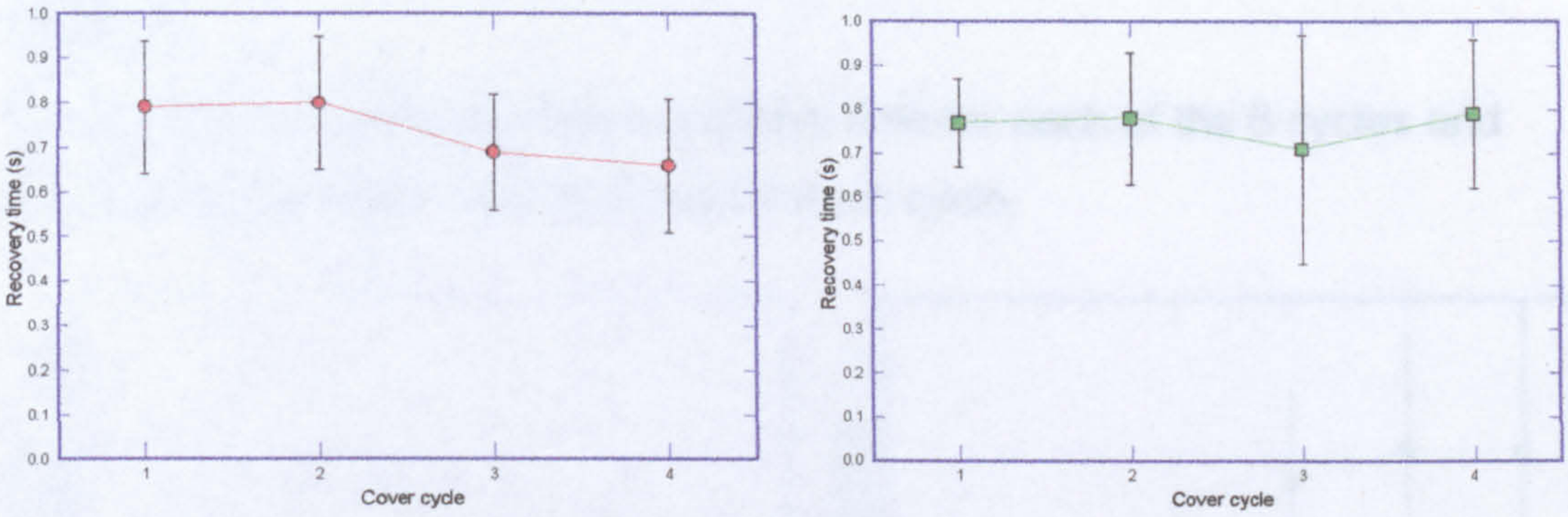


Figure 2.20. Mean recovery times following the distance 2 s cover test. (Error bars denote ± 1 standard deviation).

Near

Figure 2.21 illustrates the mean recovery time for each eye at each of the four cycles of the near 2 s cover test.



Right eye **Left eye**

Figure 2.21. Mean recovery times following the near 2 s cover test. (Error bars denote ± 1 standard deviation).

Alternate cover test

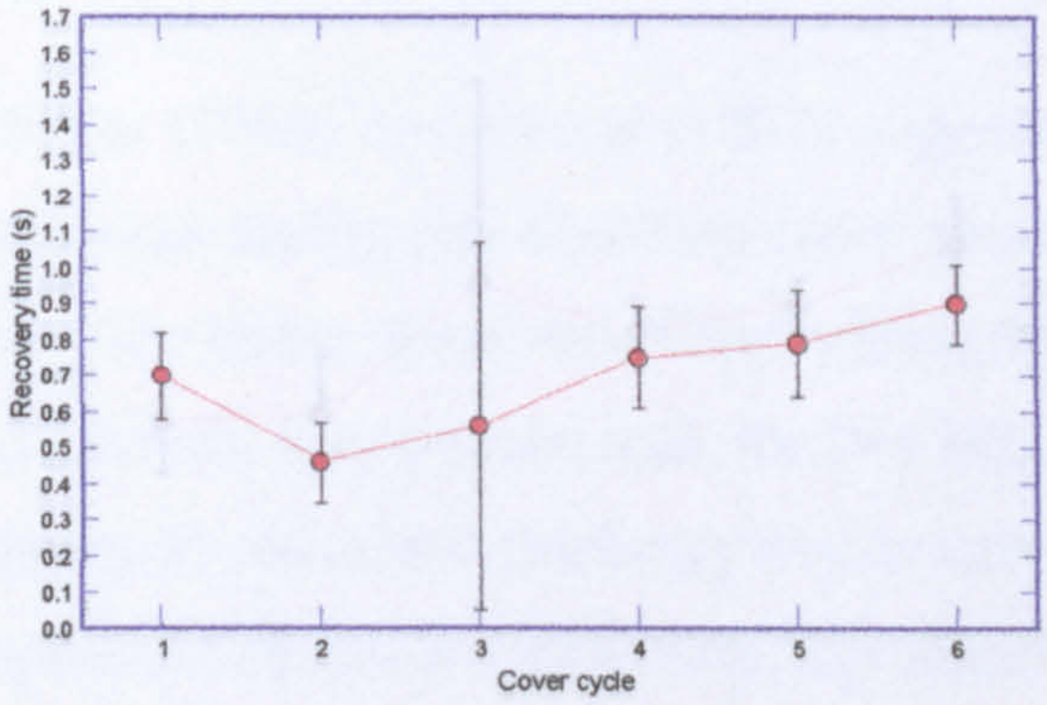
2.12 lists the average distance and near recovery time for the right and left eyes at each cycle of the alternate cover test. Standard deviations are shown in brackets. Each recovery time listed was the average of 6 measures unless the standard deviation is followed by a superscript annotation showing the number of measures available for analysis. Missing data was due to blinks occurring at critical times in the recording procedure.

	Distance		**Near**	
Cover cycle	**Right eye**	**Left eye**	**Right eye**	**Left eye**
1	0.70 (SD = 0.12)	0.48 (SD = 0.23) [5]	0.49 (SD = 0.12) [5]	0.79 (SD = 0.55)
2	0.46 (SD = 0.11)	0.57 (SD = 0.17)	0.50 (SD = 0.14) [5]	0.57 (SD = 0.13)
3	0.56 (SD = 0.51)	0.76 (SD = 0.18)	0.80 (SD = 0.47) [5]	0.73 (SD = 0.20) [5]
4	0.75 (SD = 0.14)	1.07 (SD = 0.35) [5]	0.69 (SD = 0.08)	0.85 (SD = 0.22)
5	0.79 (SD = 0.15)	1.19 (SD = 0.47) [4]	0.75 (SD = 0.06)	0.87 (SD = 0.11)
6	0.90 (SD = 0.11)	1.18 (SD = 0.57)	0.89 (SD = 0.11)	1.04 (SD = 0.28)

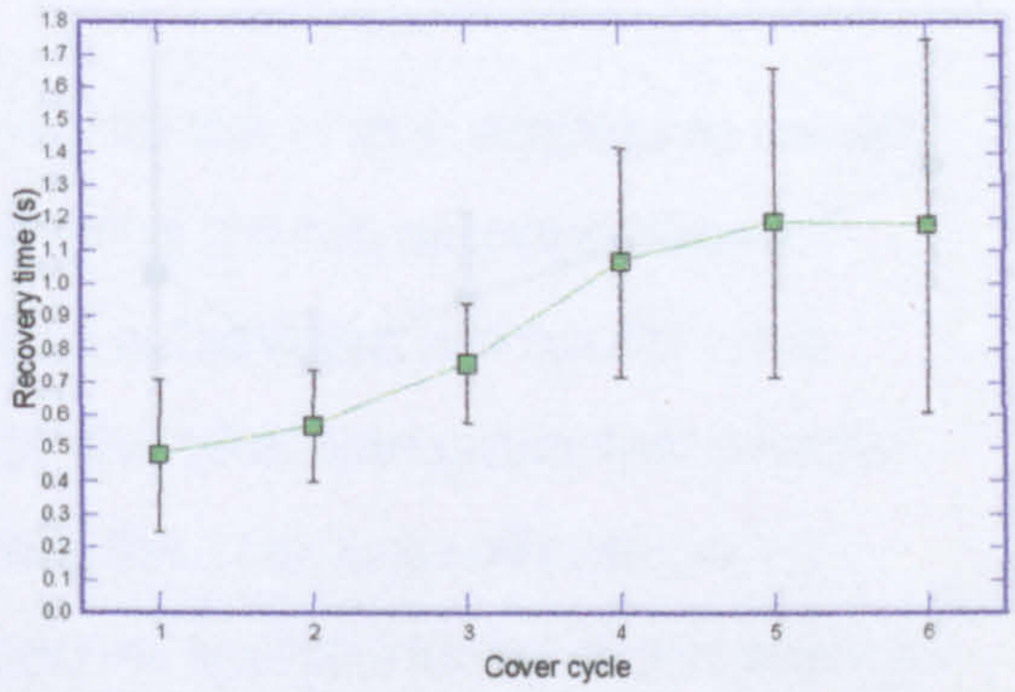
Table 2.12. Recovery times (s) following the alternate cover test.

Distance

Figure 2.22 illustrates the mean recovery time for each of the 6 cycles and also shows the mean latency times for each cycle.



Right eye



Left eye

Figure 2.22. Change in mean recovery times with repeated distance alternate cover test. (Error bars denote ± 1 standard deviation).

Figure 2.23 shows both recovery and latency times with repeated distance alternate cover test.

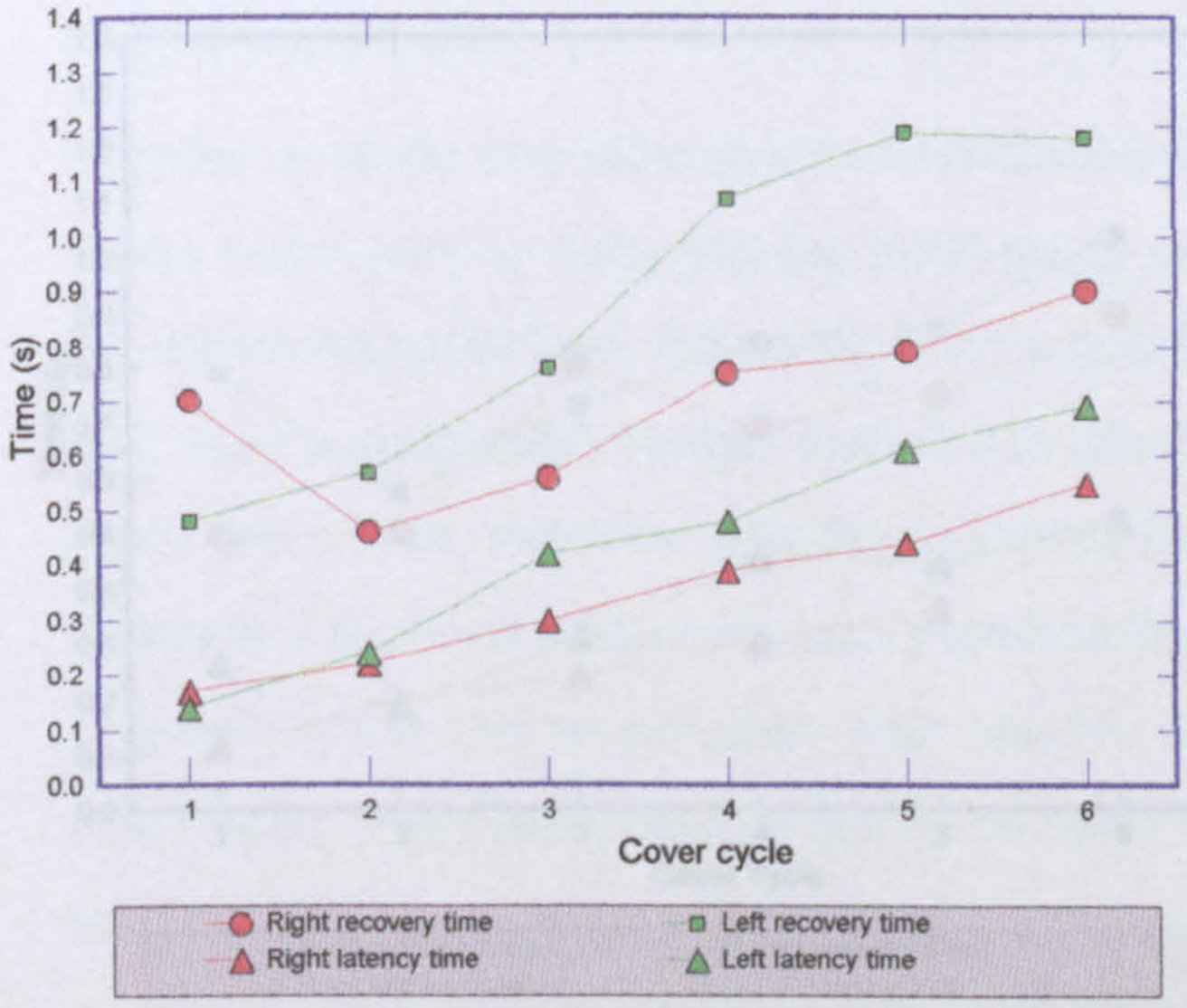


Figure 2.23. Change in mean recovery and latency times with repeated distance alternate cover test.

Near

Figure 2.24 illustrates the mean recovery, for each eye, at each of the six cycles of the near alternate cover test.

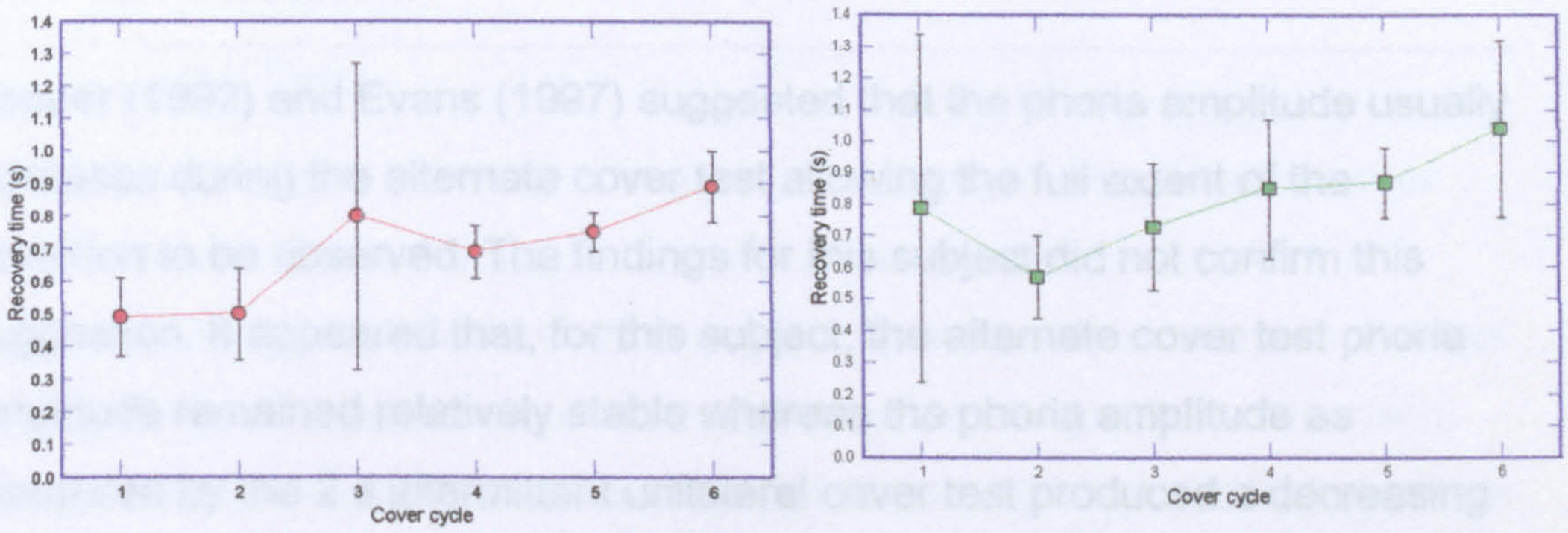


Figure 2.24. Change in mean recovery times with repeated near alternate cover test. (Error bars denote ± 1 standard deviation).

Figure 2.25 shows both recovery and latency times with repeated near alternate cover test.

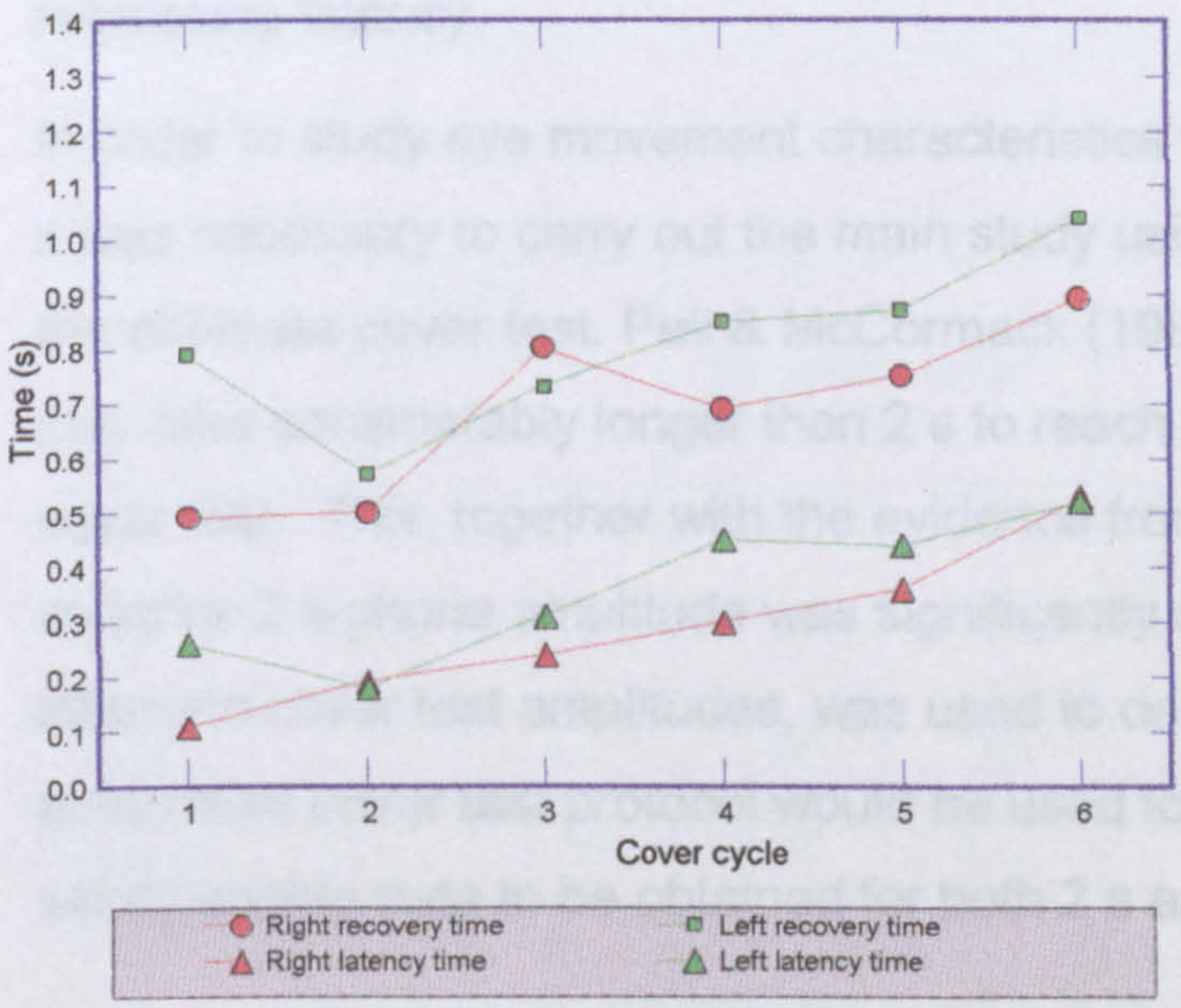


Figure 2.25. Change in mean recovery and latency times with repeated near alternate cover test.

As with the distance alternate cover test, there is an apparent increase in latency time and total recovery time as the alternate cover test is repeated.

2.3.1.d. Discussion

Cooper (1992) and Evans (1997) suggested that the phoria amplitude usually *increases* during the alternate cover test allowing the full extent of the deviation to be observed. The findings for this subject did not confirm this suggestion. It appeared that, for this subject, the alternate cover test phoria amplitude remained relatively stable whereas the phoria amplitude as measured by the 2 s intermittent unilateral cover test produced a decreasing amplitude with the number of cycles.

An interesting finding was that of the increasing recovery time produced by repeated alternate occlusion. This appeared to be mostly due to an increasing latency time with repeated occlusion. The alternate cover test does not elicit disparity vergence to produce a recovery movement. Re-fixation is carried out by means of saccades. Possible explanations for increased saccadic latency include 'inability to disengage attention' and 'decreased motivation' (Ciuffreda & Tannen, 1995). A further study is warranted on a larger population to determine whether this finding is common and to determine the cause of the increasing latency.

In order to study eye movement characteristics following removal of the cover it was necessary to carry out the main study using the cover test rather than the alternate cover test. Peli & McCormack (1983) suggested that some eyes may take considerably longer than 2 s to reach a stable position during the cover test. This, together with the evidence from this pilot study that the distance 2 s phoria amplitude was significantly smaller than the 10 s and alternate cover test amplitudes, was used to determine that the 10 s automated cover test protocol would be used for the main experiment. This would enable data to be obtained for both 2 s and 10 s periods of occlusion.

2.3.2 Experiment to assess the repeatability of eye movement characteristics during the cover test.

2.3.2.a. Method

Repeated measures were taken on one subject to give some indication of repeatability of phoria amplitude, latency of first recovery movement, and recovery time. Eye movement recording during the automated cover test was carried out on 7 separate days. The subject was a postgraduate optometry student. All the measurements took place between 1000 and 1600 hours over a 10 day period. The subject was asked not to drink any alcohol on those days that recordings took place.

The distance and near 10 s cover test was carried out and data were extracted to produce the average 10 s phoria amplitude^{RL}, the average recovery time^{RL}, and latency times for the right and left eye. The mean and standard deviations for each set of data were calculated and these are shown in Table 2.13. Only one measure of right eye distance latency was available due to blinks at the critical time in the recording.

2.3.2.b. Results

	Phoria amplitude^{RL} (°)		Recovery time^{RL} (s)		Latency time (s)			
	Distance	**Near**	**Distance**	**Near**	**RE Distance**	**LE distance**	**RE near**	**LE near**
N	7	7	7	7	-	6	5	6
Mean	-0.37	-2.4	1.01	1.21	Nd	0.26	0.18	0.19
Standard deviation	0.26	0.30	0.75	0.44	-	0.07	0.03	0.06

Table 2.13. Data for repeated measures of parameters for a single subject.

This data provides some information as to the combined repeatability of the recording system and the subject. The low standard deviations for both phoria

amplitude and for latency suggest that the experimental apparatus had the potential for obtaining repeatable spatial and temporal data when the parameters being measured are themselves repeatable.

2.3.3 Variation of eye movement characteristics during the cover test over a 6 hour period

2.3.3.a. Introduction

A pilot study was carried out to investigate whether phoria amplitudes and recovery times might vary during the day. The reason for this study was to determine whether it might be necessary to be specific about the time of day that procedures in the main study were to be carried out (Barnard & Thomson, 1995b).

Although the possibility of conducting a study over a full diurnal period was contemplated, this had to be rejected for practical reasons.

Yekta et al (1987) assessed the binocular vision of 84 young adult subjects aged between 15 and 47 years. A criticism of this study is the large age range that appeared to include subjects of presbyopic age. 63 of the subjects complained of visual symptoms associated with close working and reading. Amplitude of dissociated phoria was measured using a modified Mills test at the start (0730 hours) and at the end (1330 hours) of the working day. In 36 subjects (43%) the amplitude of dissociated phoria was unchanged and in 7 subjects (8%) it had decreased. These statistics did not differentiate between exophoric and esophoric subjects. However, they reported a significant statistical difference ($p < 0.001$) in the means of dissociated exophoria at the start (mean $= 4.38^{\Delta}$) and end of the working day (5.17^{Δ}).

2.3.3.b. Method

The three 'normal' subjects (*C, M and T)* used for this study were male postgraduate optometry students.

Eye movement recording was carried out for both distance and near cover tests at 1000 hours, 1200 hours, 1400 hours and 1600 hours. Each subject was asked to continue with his normal work schedule in between each test and was asked not to consume any alcohol.

2.3.3.c. Results

I. Phoria amplitudes

Right and left 10 second phoria amplitudes were extracted from the eye movement recording data and phoria amplitudes^{RL} were calculated.

Distance

Figure 2.26 shows the distance phoria amplitudes for each subject at the four times over the six hour period.

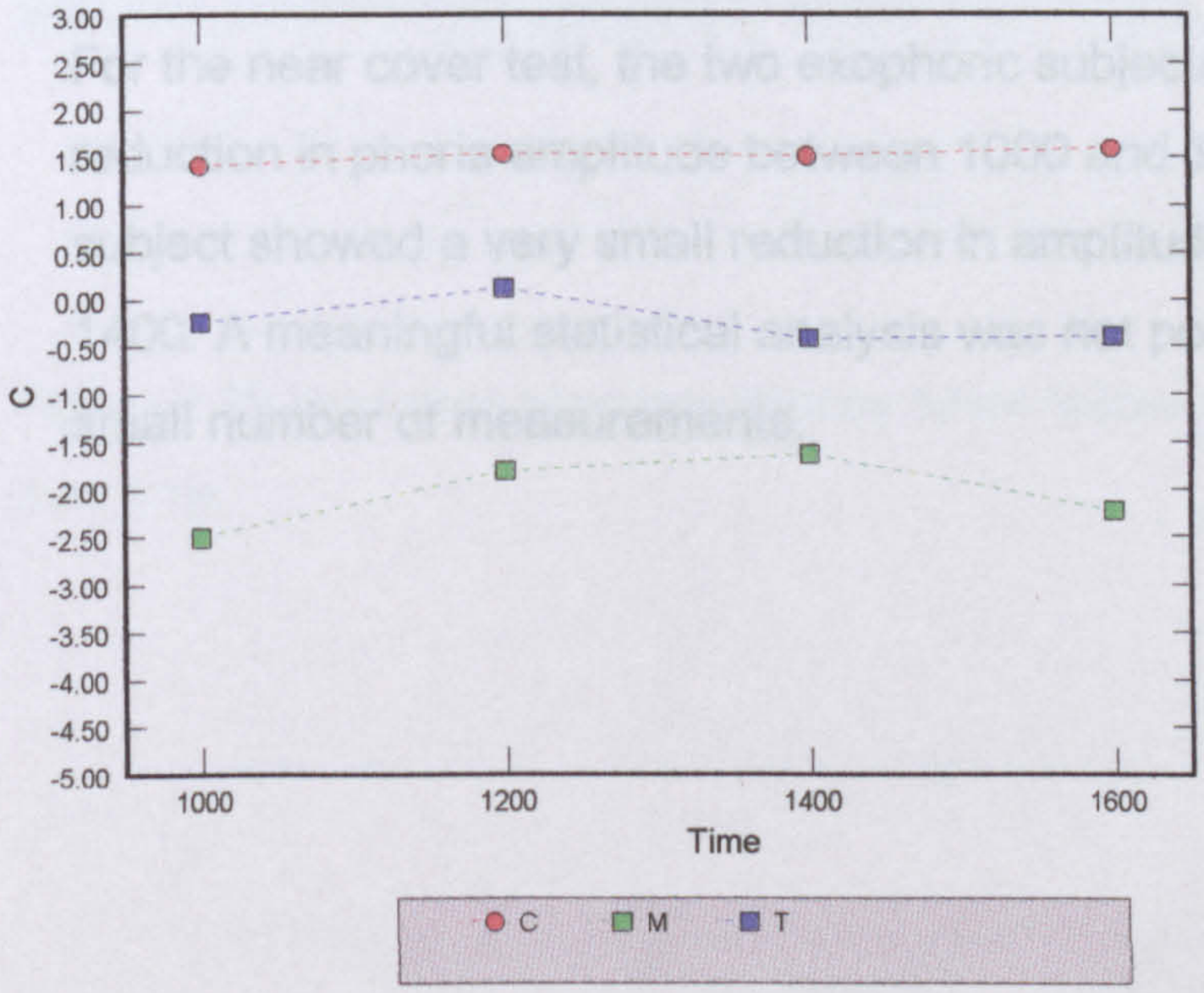


Figure 2.26. Scatter plot showing variation of distance phoria amplitude with time for 3 subjects.

The phoria amplitudes appeared to remain constant to within approximately 0.5° over the 6 hour period. A meaningful statistical analysis was not possible because of the small number of measurements.

Figure 2.27 illustrates the near phoria amplitudes for each subject at the four times over the six hour period.

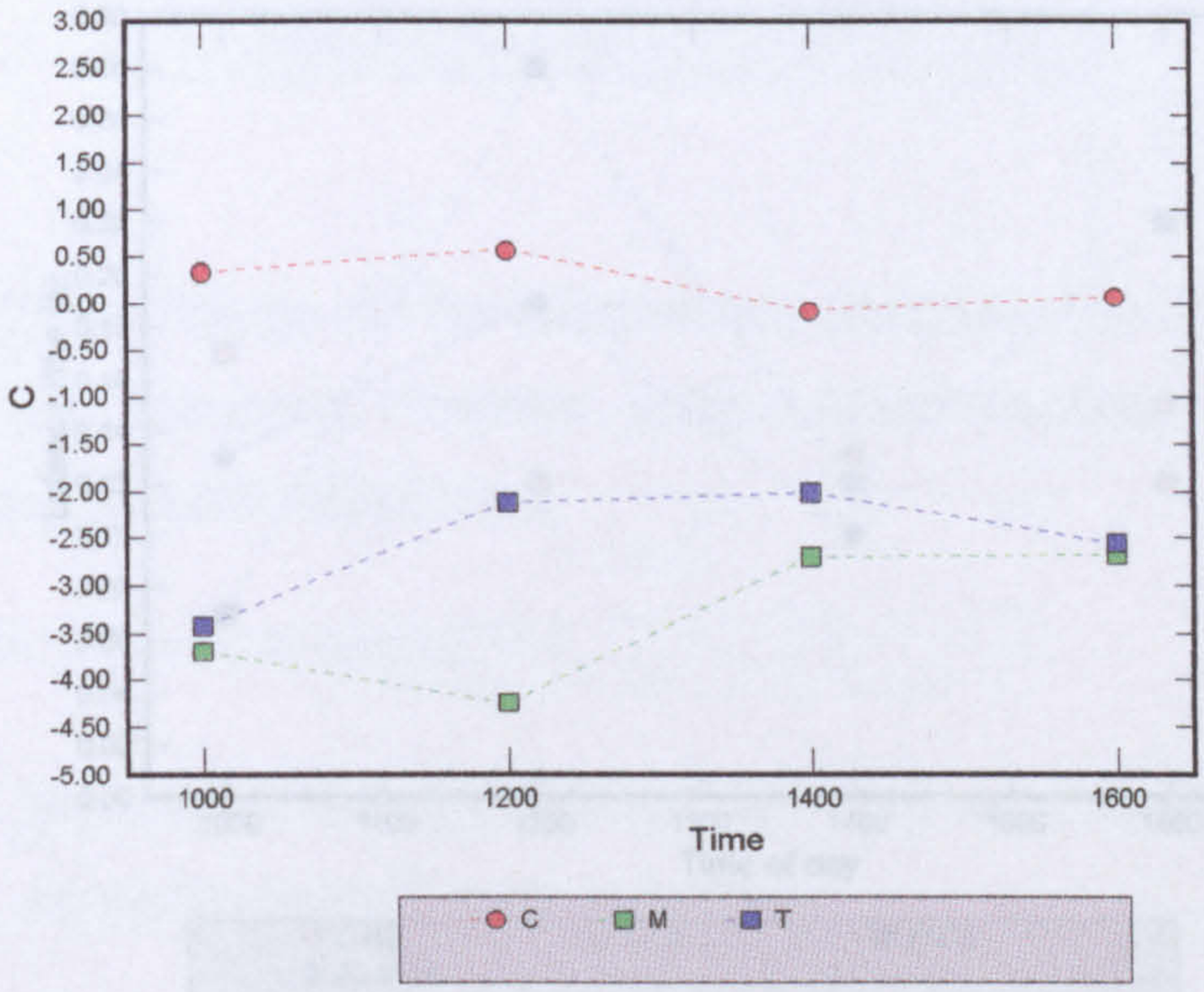


Figure 2.27. Scatter plot showing variation of near phoria amplitude with time for 3 subjects.

For the near cover test, the two exophoric subjects showed a small reduction in phoria amplitude between 1000 and 1400. The esophoric subject showed a very small reduction in amplitude between 1200 and 1400. A meaningful statistical analysis was not possible because of the small number of measurements.

II. Latency times

Distance

Figure 2.28 illustrates recovery latency times following the distance cover test, over time. Latency data was not available for subject *T* except for 1600 hours and this subject is therefore not shown in this figure. A meaningful statistical analysis was not possible because of the small number of measurements.

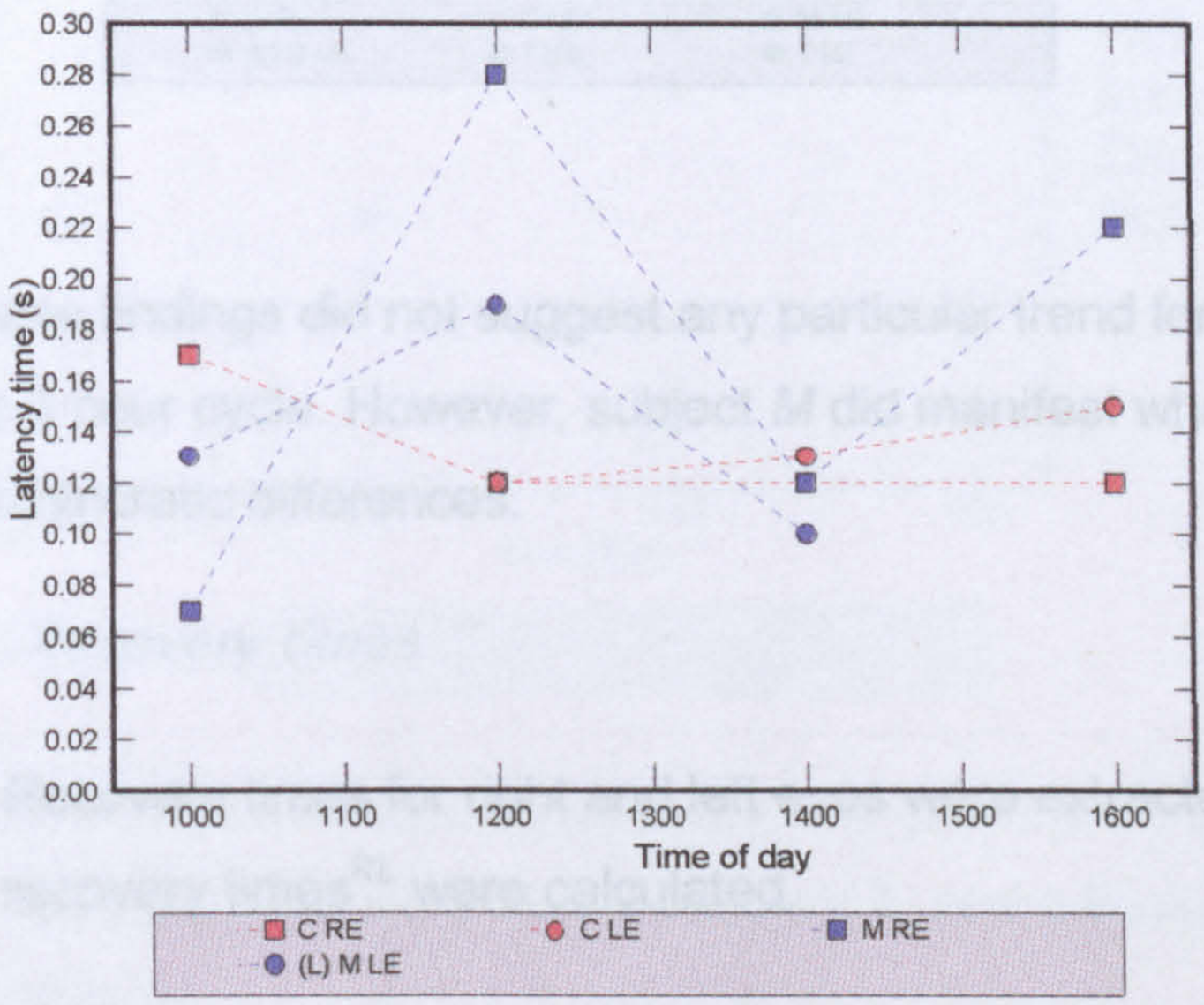


Figure 2.28. Scatter plot showing right and left eye latency times following the distance cover test for subjects C and M (RE = right eye; LE = left eye).

Near

Figure 2.29 illustrates recovery latency times following the near cover test, over time.

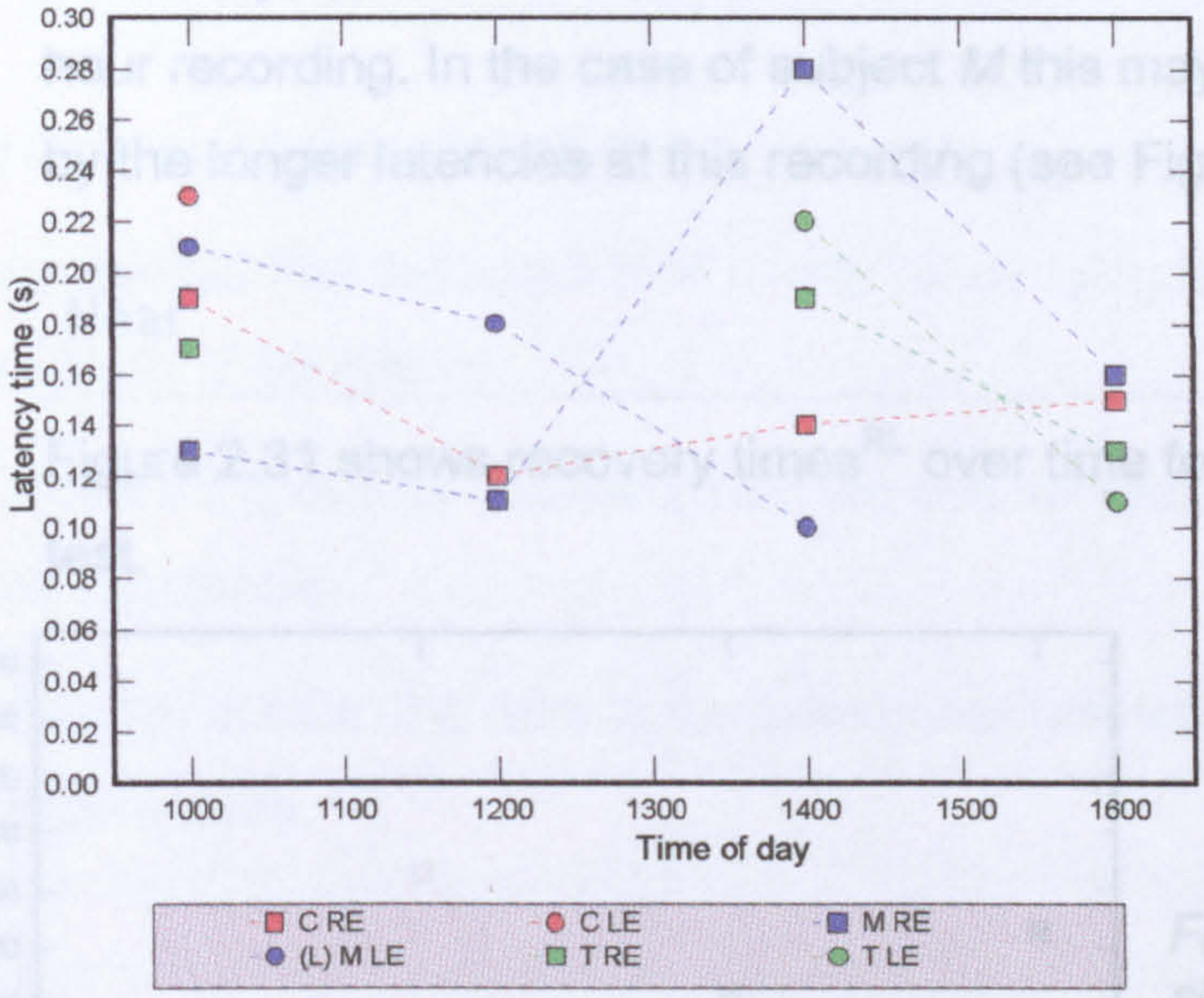


Figure 2.29. Scatter plot showing right and left eye latency times following the near cover test.

These findings did not suggest any particular trend for latency times over the 6 hour cycle. However, subject *M* did manifest what appeared to be idiosyncratic differences.

III. Recovery times

Recovery times for right and left eyes were extracted and the average recovery times^{RL} were calculated.

Distance

Figure 2.30 shows recovery times^{RL} over time following the distance cover test.

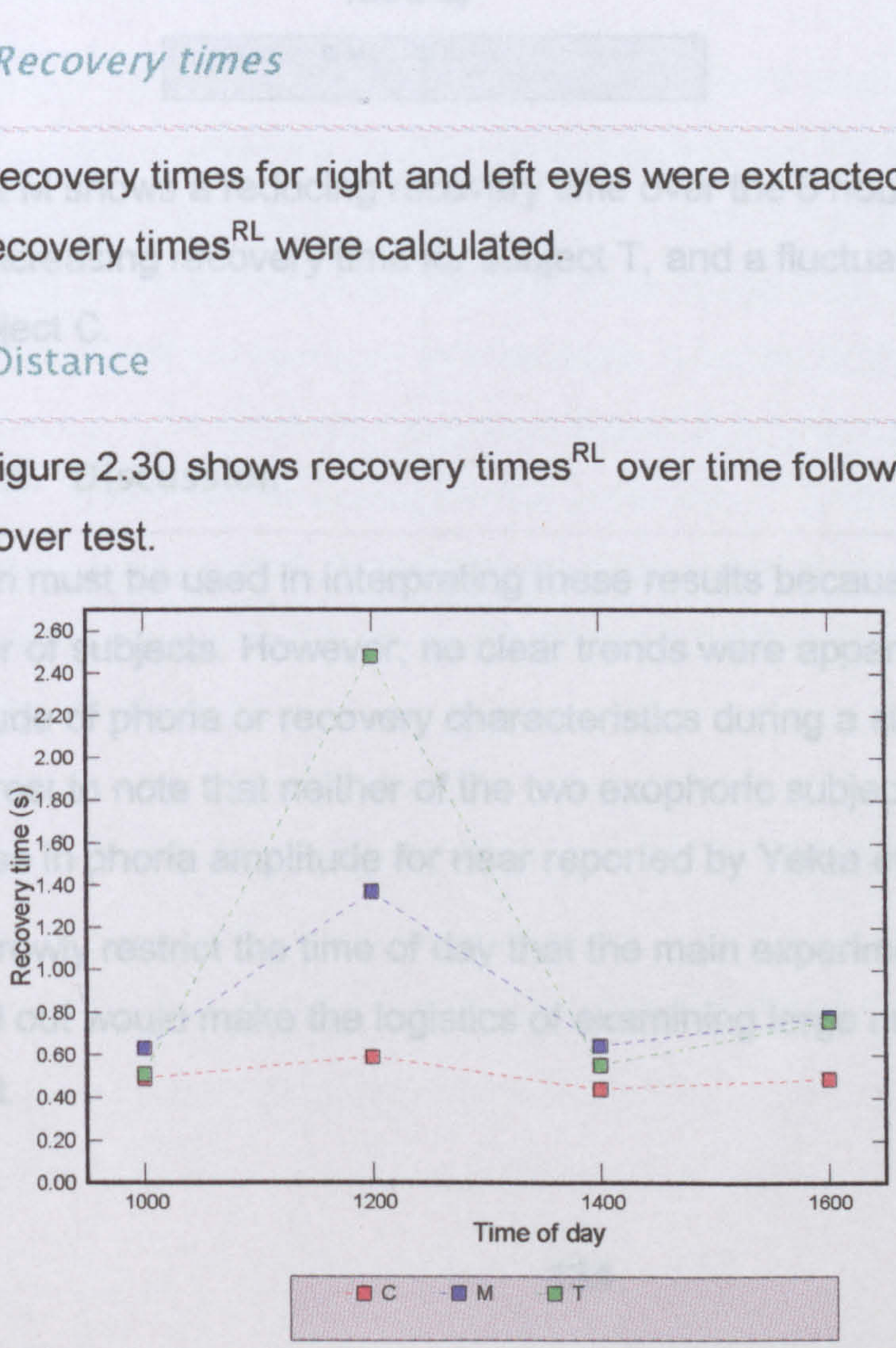


Figure 2.30. Scatter plot showing recovery time ^{RL} following the distance cover test.

Both subject *M* and *T* showed an increase in recovery time^{RL} at the 1200 hour recording. In the case of subject *M* this may possibly be explained by the longer latencies at this recording (see Figure 2.28).

Near

Figure 2.31 shows recovery times^{RL} over time following the near cover test.

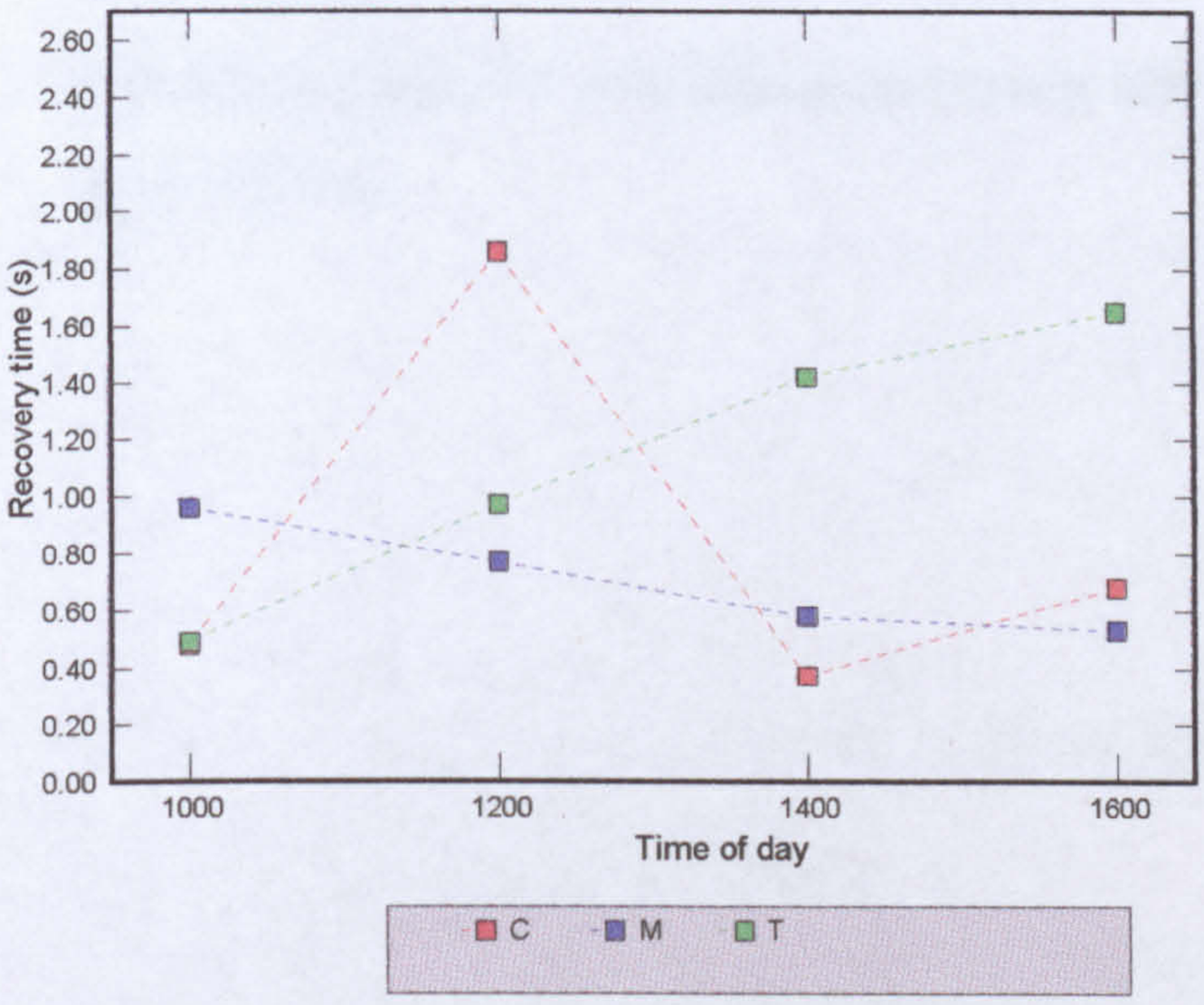


Figure 2.31. Scatter plot showing recovery time ^{RL} following the near cover test.

Subject M shows a reducing recovery time over the 6 hour period, compared to an increasing recovery time for subject T, and a fluctuating recovery time for subject C.

2.3.3.d. Discussion

Caution must be used in interpreting these results because of the very small number of subjects. However, no clear trends were apparent in either the amplitude of phoria or recovery characteristics during a six hour period. It was of interest to note that neither of the two exophoric subjects showed the increase in phoria amplitude for near reported by Yekta et al (1987).

To narrowly restrict the time of day that the main experiments were to be carried out would make the logistics of examining large numbers of subjects difficult.

2.3.4 Conclusions

The results of these pilot studies:

- suggested that the exact time of day for carrying out the experiments was unlikely to be critical.
- showed the results obtained from the eye movement recording system to be repeatable.
- determined that the 10 s automated cover test would be used for the main experiments.

Chapter 3. Eye movement characteristics during the cover test in a population of 100 normal subjects

The aim of the first experiment was to investigate the eye movement characteristics during the 10 s cover test of a group of 'normal' subjects. 100 'normal' subjects were recruited to the study and underwent eye movement recording during both distance and near automated 10 s cover test. This has been described in Chapter 2. 39 subjects also underwent a battery of other optometric tests and, despite claiming to be asymptomatic, they were invited to complete a symptom questionnaire.

3.1 Subjects

100 subjects meeting the 'normal' criteria described in Chapter 2 were recruited to the study:

Following completion of a recruitment questionnaire (see Appendix 1), subjects likely to meet the criteria were examined by the author. Unaided vision and refraction were obtained. A conventional cover test was carried out to rule out the presence of strabismus. If any subject showed a one Snellen line difference of VA between the two eyes, then the protocol demanded that foveal fixation was checked using the Keeler Specialist ophthalmoscope eccentric fixation target to exclude the possibility of a microtropia. No such subjects were observed. Subjects were then scheduled to attend for the automated cover test procedure. All measurements were carried out between 0900 and 1800 hours.

3.2 Results

The following parameters were extracted from the eye movement recordings:

- The maximum phoria amplitude reached during the 10 seconds of occlusion (T^{max})

- The amplitude of phoria after 2 seconds occlusion (P^2)
- The amplitude of phoria after 10 seconds of occlusion (P^{10})
- The time at which the maximum phoria amplitude was reached (T^{max})
- The time at which the amplitude measured at 10 seconds was reached (T^{10})
- The latency of recovery measured from when the occluder theoretically passed across the visual axis to the commencement of the first recovery movement (L)
- The time taken for the eye to regain fixation on removal of the occluder (R)
- The number and type of eye movements during the recovery phase

Figure 3.1 illustrates schematically some of the parameters listed above.

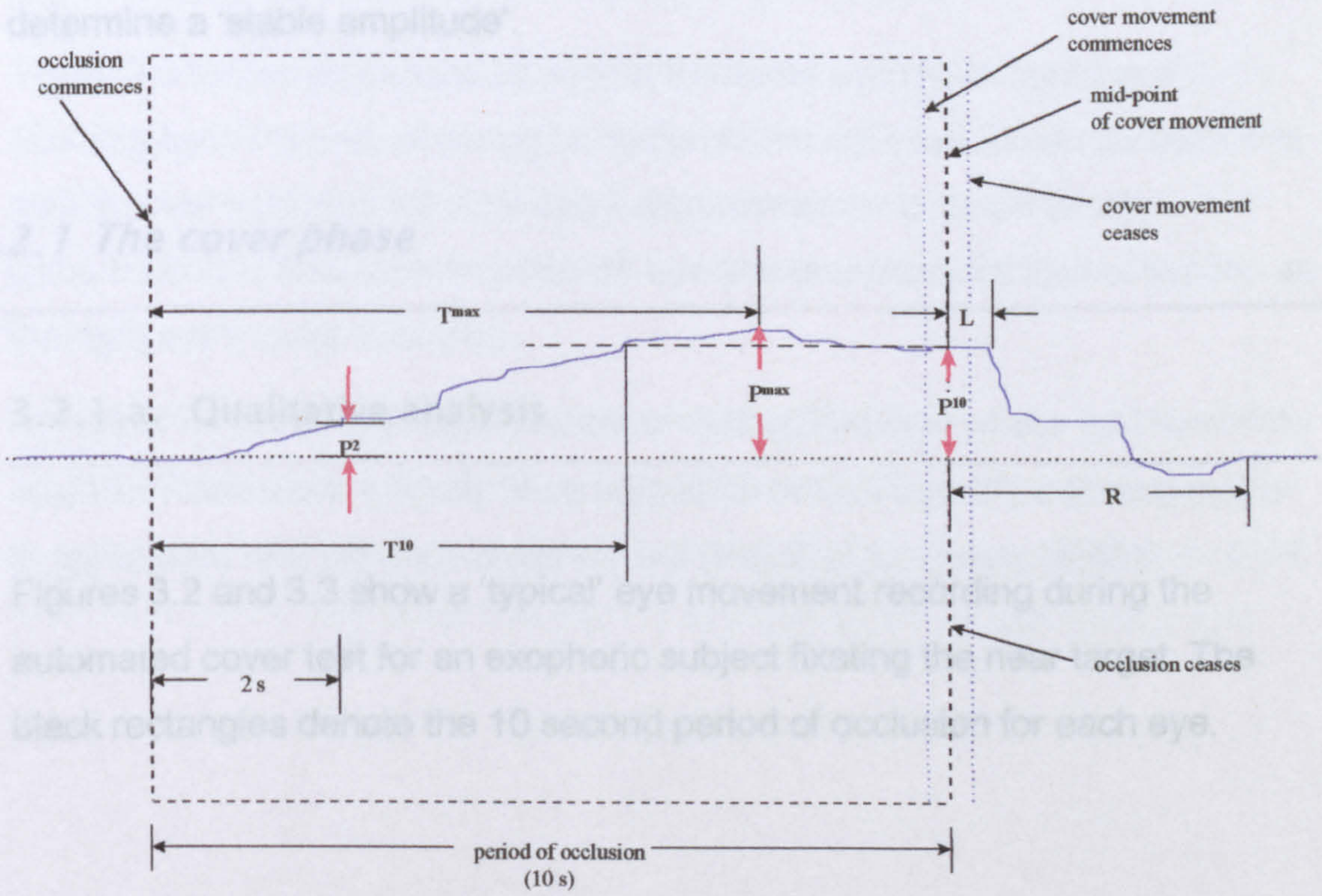


Figure 3.1. Schematic diagram showing various parameters extracted from the eye movement traces (not to scale). The continuous blue line denotes an eye movement trace.

It is evident from the list above that three different measures of phoria amplitude were extracted from the data and this warrants further explanation.

Firstly, the amplitude of phoria was measured when each eye had been occluded for 2 seconds. This arbitrary period was chosen because conventional wisdom, as supported by Evans (1997), is that approximately 2 seconds is the optimum period and indeed, in this author's view is likely to be an approximation to the period most commonly employed by practitioners.

The amplitude was also measured at the 10 second position this being the point at which the cover commenced uncovering that eye. 10 seconds is also arbitrary but there needs to be a limit in terms of data acquisition by the computer. This period does exceed the 8 seconds of occlusion utilised by Peli & McCormack (1983) and thereby extends the range of investigation.

Lastly, the position of maximum phoria amplitude was measured. This additional parameter will enable some conclusions to be made on the optimum time that an eye should be occluded during the cover test in order to determine a 'stable amplitude'.

3.2.1 The cover phase

3.2.1.a. Qualitative analysis

Figures 3.2 and 3.3 show a 'typical' eye movement recording during the automated cover test for an exophoric subject fixating the near target. The black rectangles denote the 10 second period of occlusion for each eye.

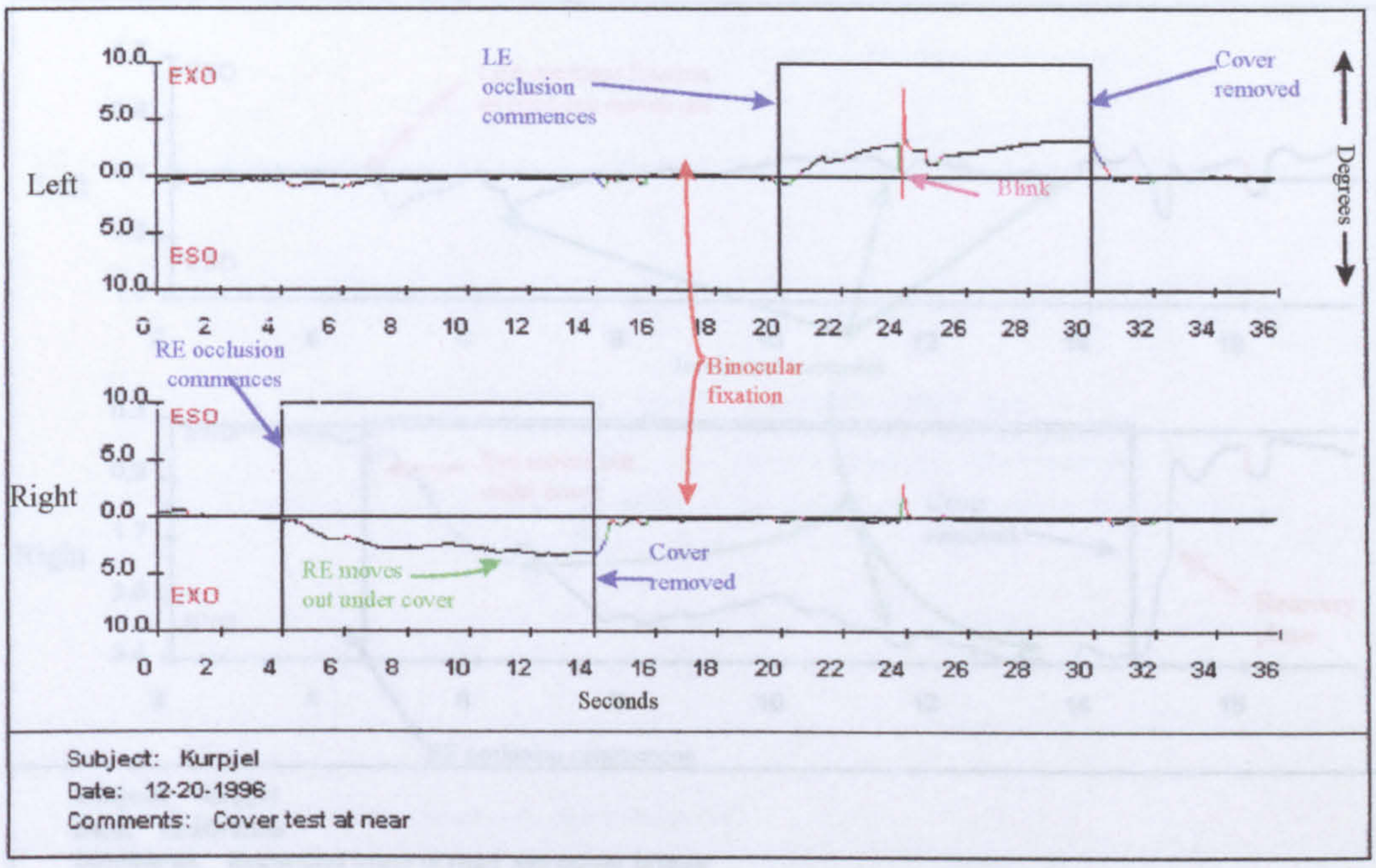


Figure 3.2. An example of an eye movement recording during the near cover test.

Figure 3.3 shows an expanded view of the same eye movement trace showing eye movements during occlusion of the right eye. It can be seen that as the cover occludes the right eye it commences an outward (exo) movement. It is also clear that the left eye shows a transient loss of fixation as the right eye moves outwards.

A number of 'irrelevant' saccades occur during the cover phase and for most subjects these were typically of an amplitude of less than 1°, a finding that is in agreement with previous observations (Peli & McCormack, 1983).

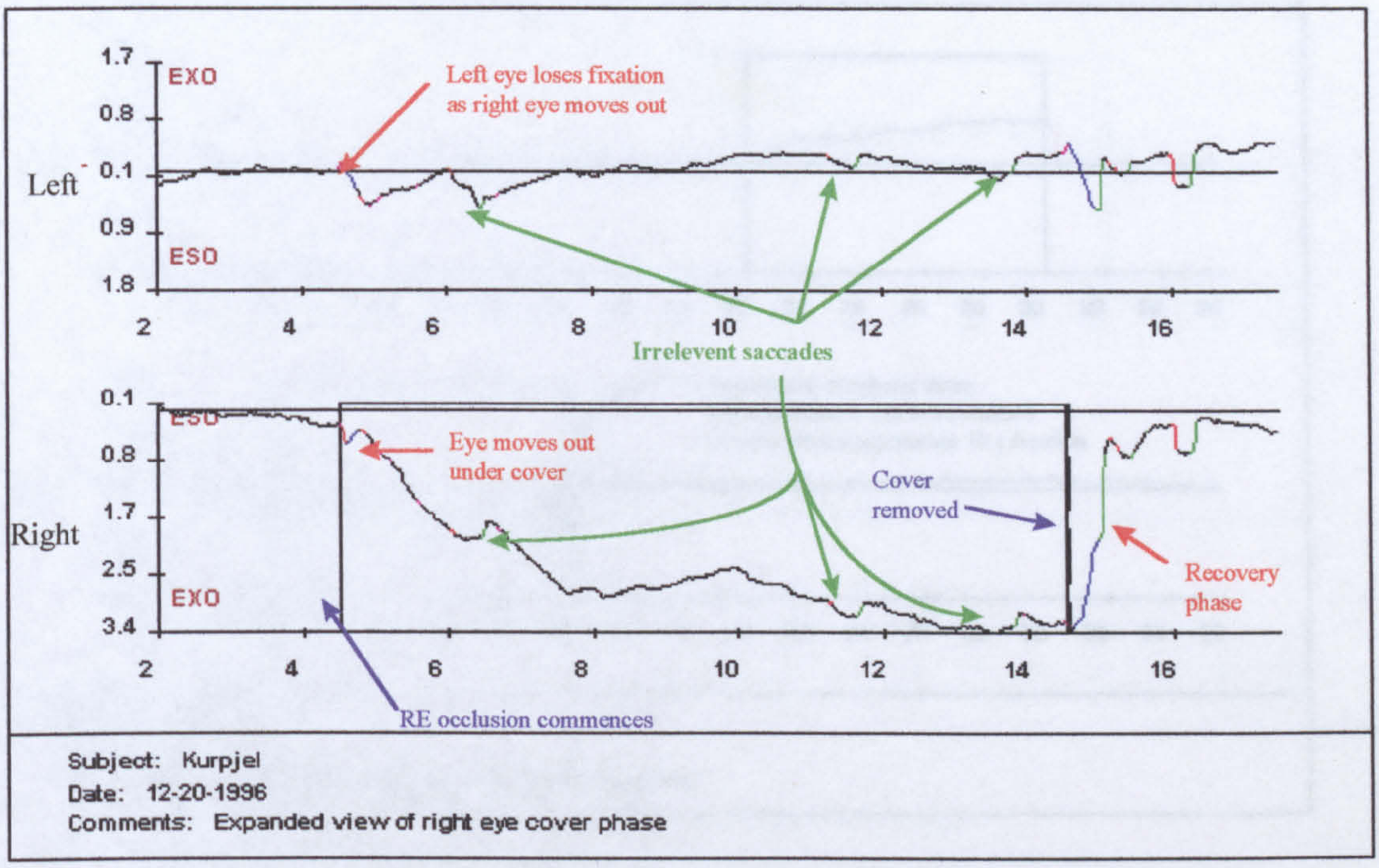


Figure 3.3. Expanded view of the right eye cover phase.

The eye movement profiles under the cover were varied. In some cases the eye would move rapidly to a position of rest and the amplitude remain stable until the cover was removed at 10 seconds. In other cases the eye would gradually drift more slowly reaching a position of rest in an 'exponential' manner.

Figure 3.4 illustrates the eye movements during the near cover test of another exophoric subject which demonstrates a different pattern of movement under the cover. In this case it took considerably longer than 2 seconds to reach the maximum phoria amplitude and there is little movement of the fixating eye.

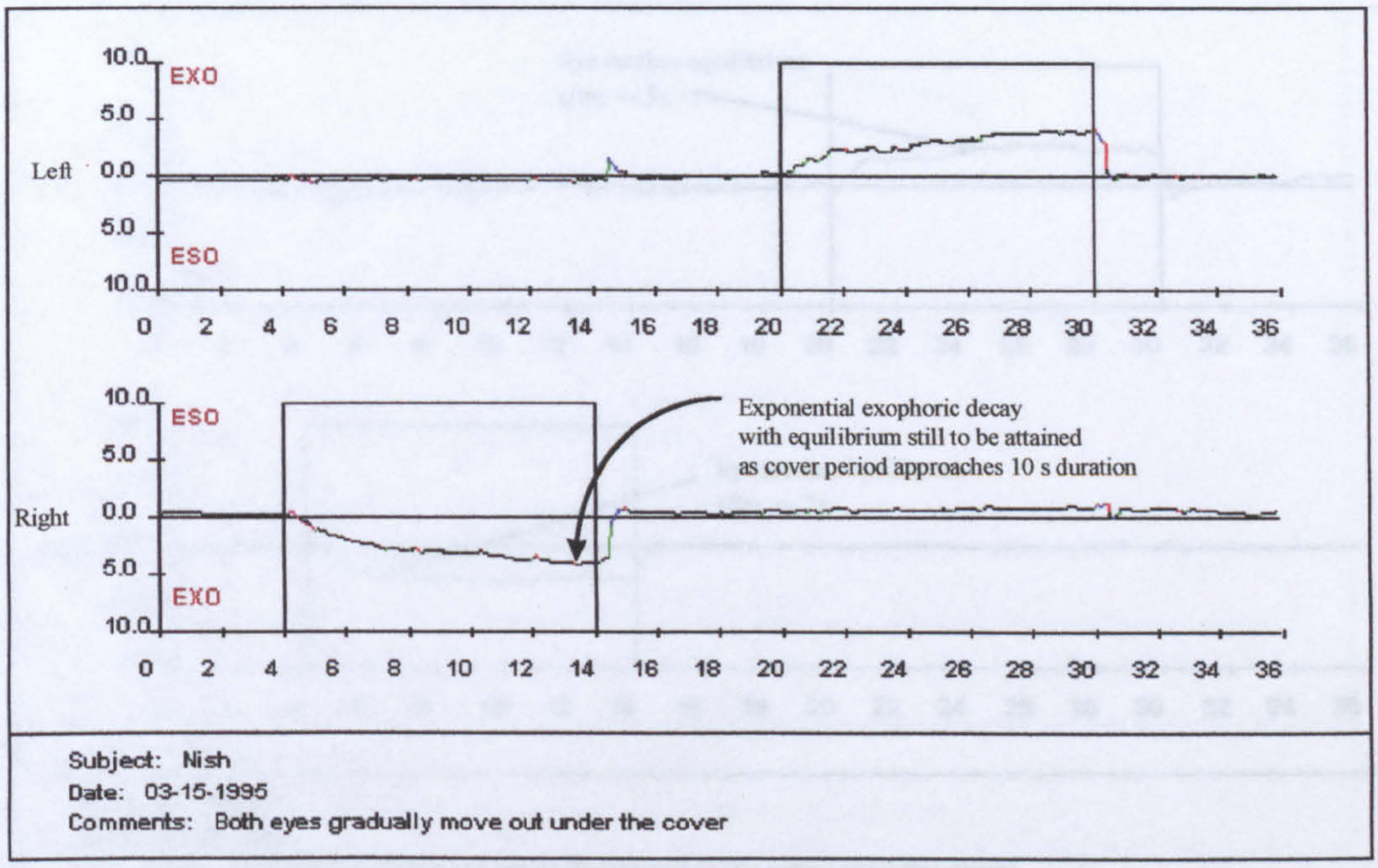


Figure 3.4. Eye movement trace showing a slow exophoric drift while each eye is occluded.

This is seen more easily seen in Figure 3.5.

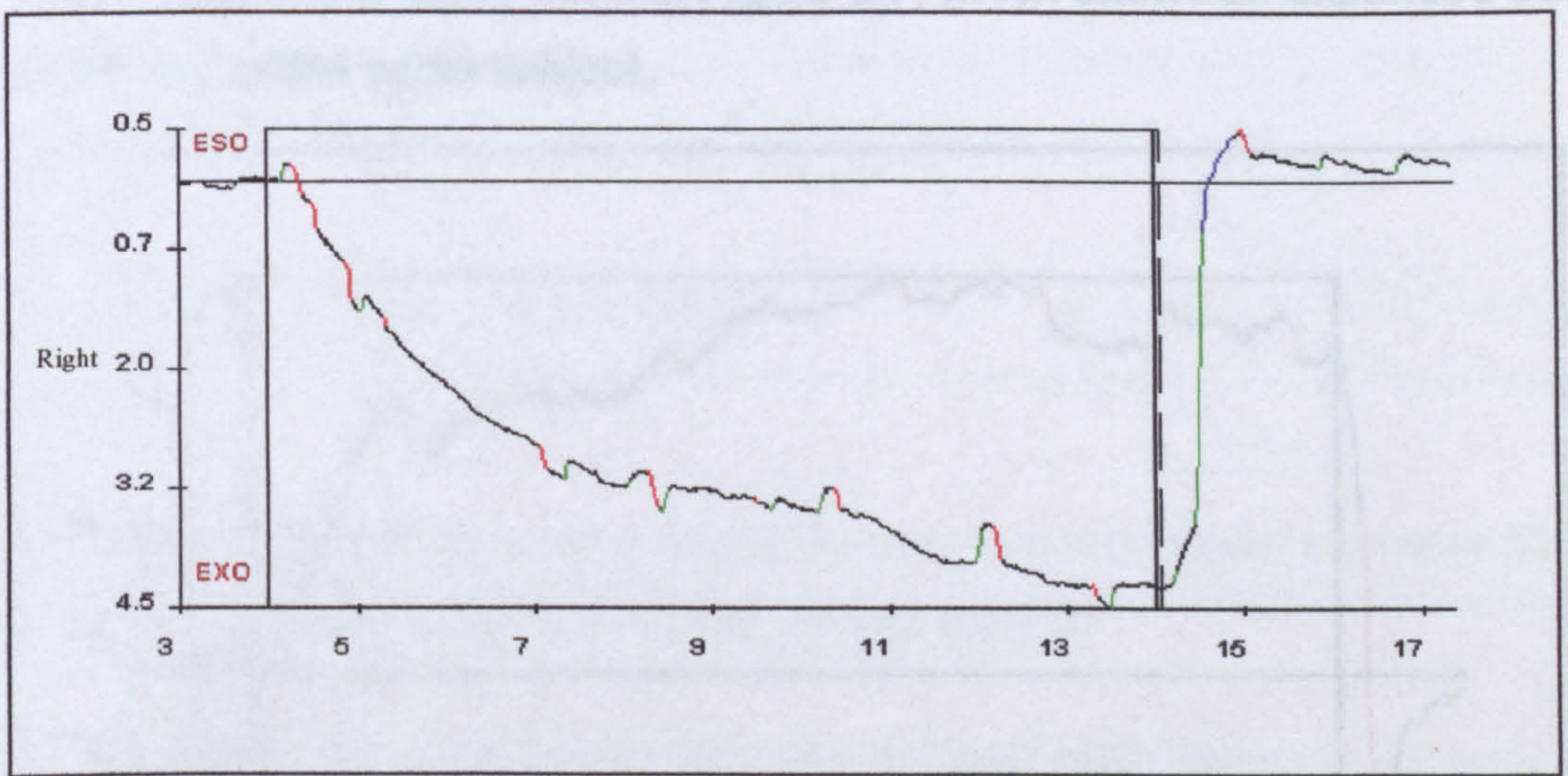


Figure 3.5. Eye movement trace for the right eye of the same subject as in Figure 3.4, showing an expanded view of the eye movement profile under the cover.

In contrast, the trace shown in Figure 3.6 illustrates an exophoric subject whose eyes moved outwards under the cover much more rapidly, reaching the stable amplitude of phoria long before the 10 seconds of occlusion was complete.

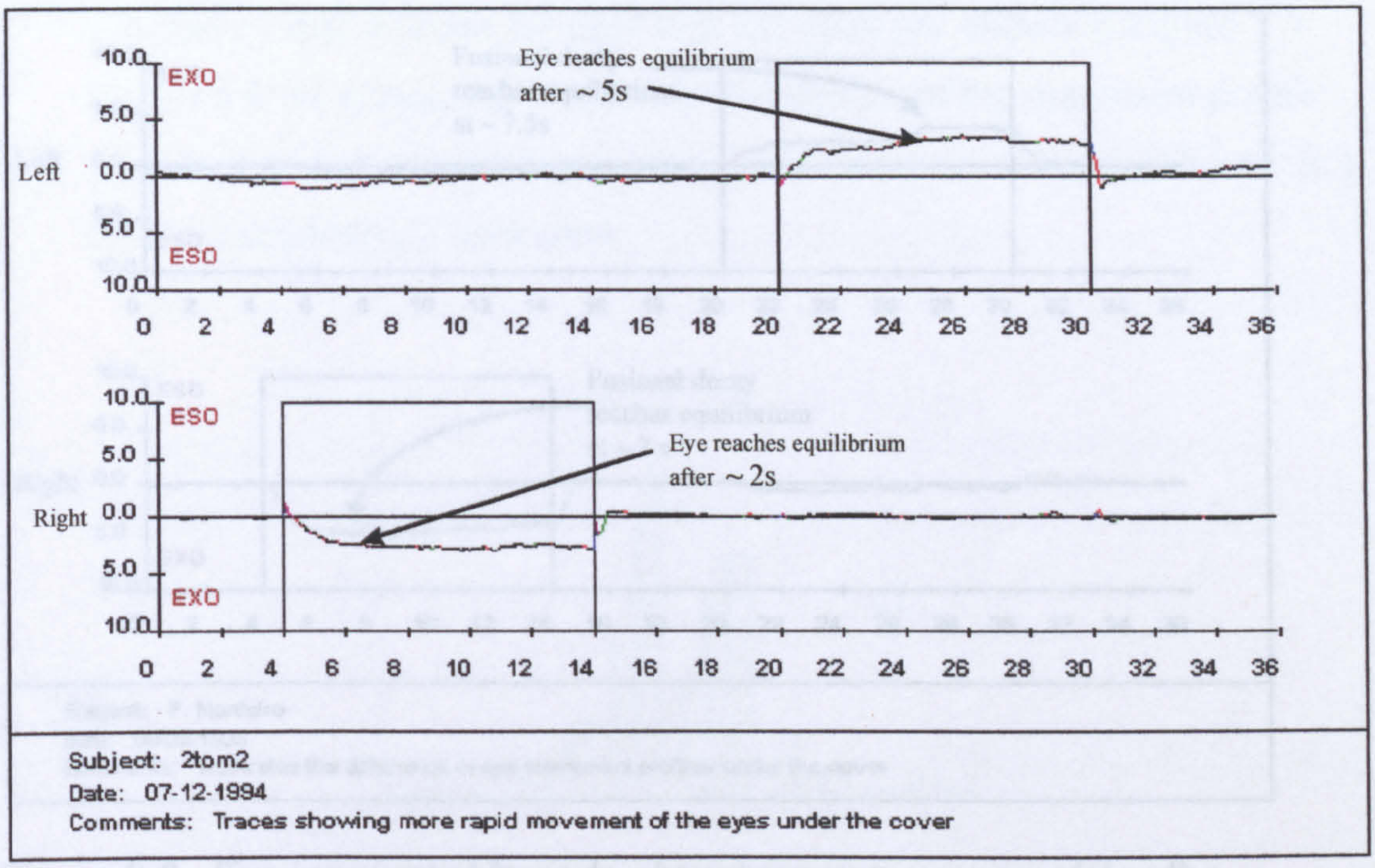


Figure 3.6. Eye movement trace showing a more rapid exophoric fusional decay while each eye is occluded.

This is seen more easily seen in Figure 3.7, which shows an expanded trace of left eye of the same subject.

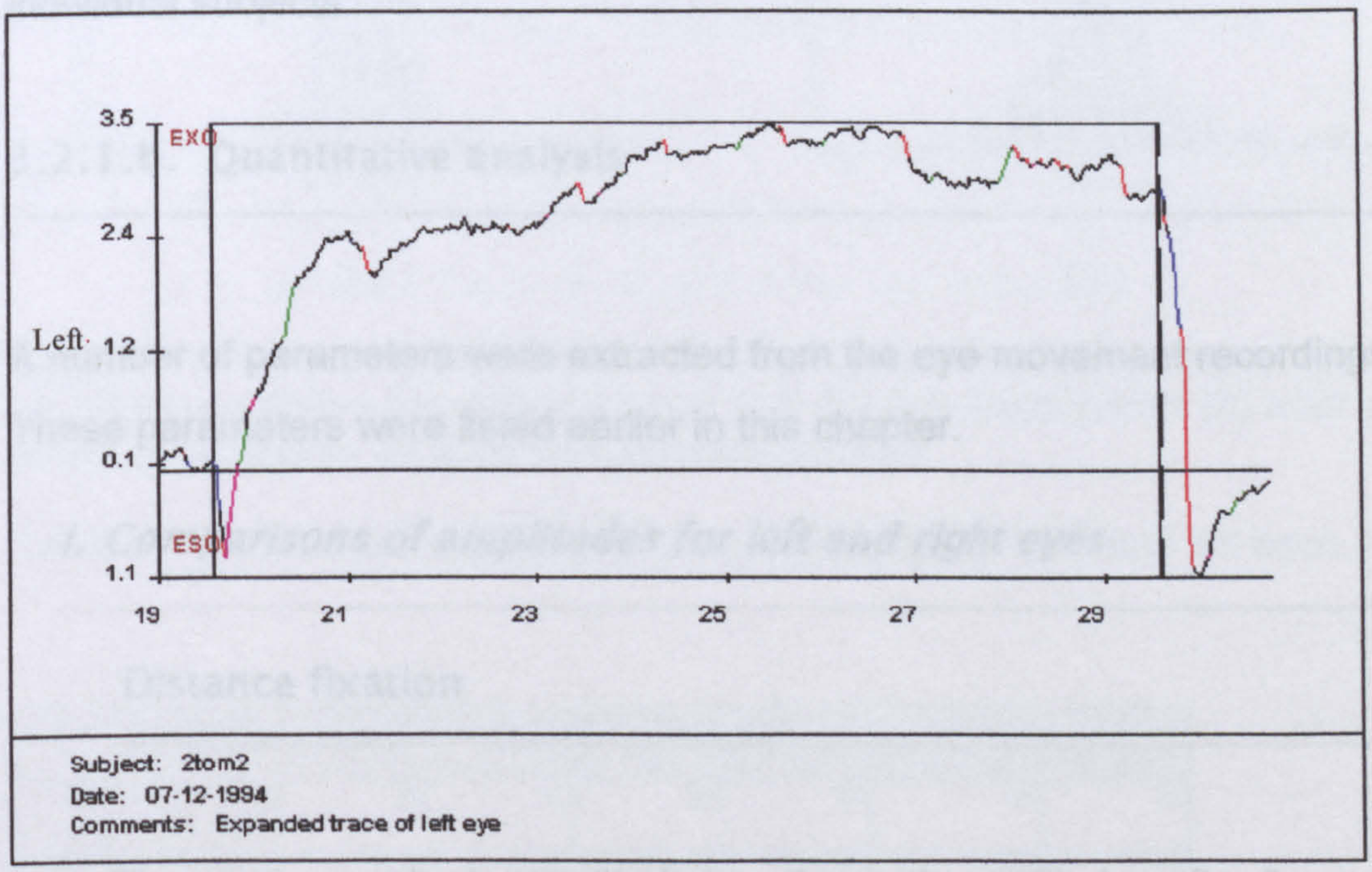


Figure 3.7. Expanded view of Figure 3.6 showing eye movements of the left eye under the cover.

Some subjects showed a different pattern of eye movements under the cover of <u>each</u> eye and this is illustrated by Figure 3.8.

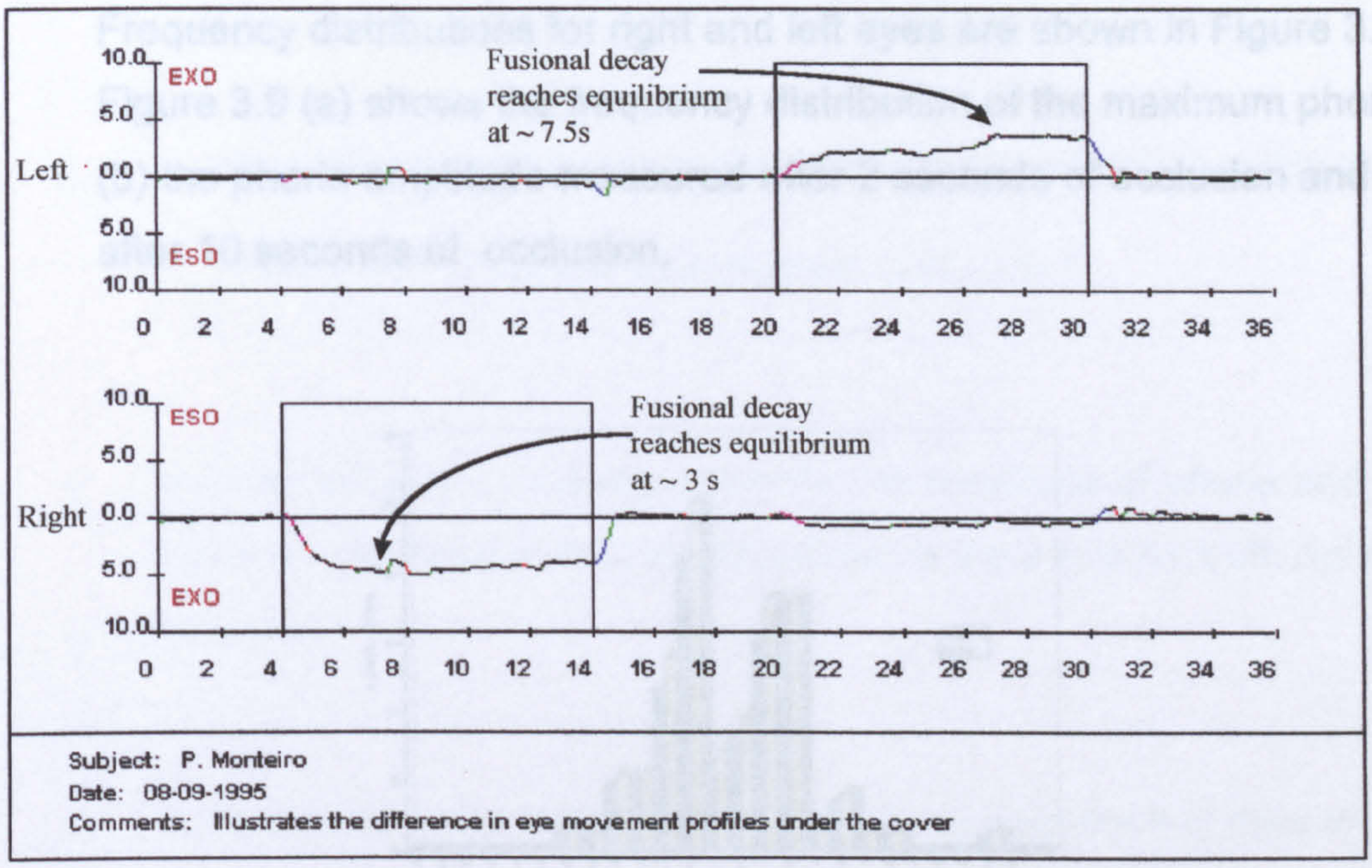


Figure 3.8. Eye movement trace to show how one eye completes the dissociation movement to reach the maximum amplitude more quickly than the other.

It may be concluded that, not only does the fusional decay profile under the cover vary between subjects but it may also vary between each eye of individual subjects.

3.2.1.b. Quantitative analysis

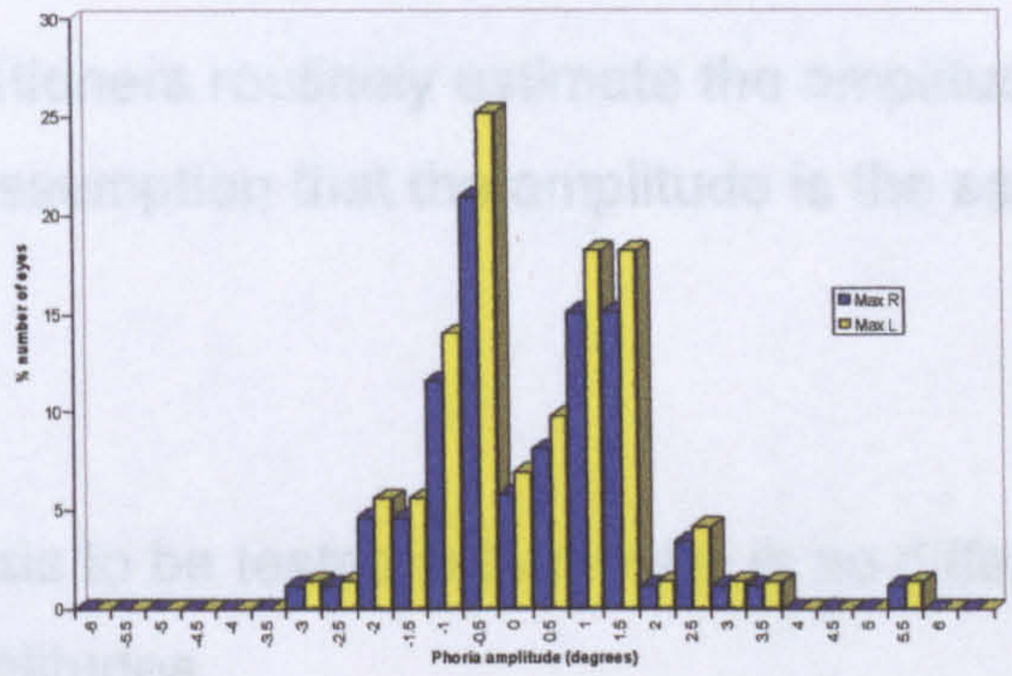
A number of parameters were extracted from the eye movement recordings. These parameters were listed earlier in this chapter.

I. Comparisons of amplitudes for left and right eyes

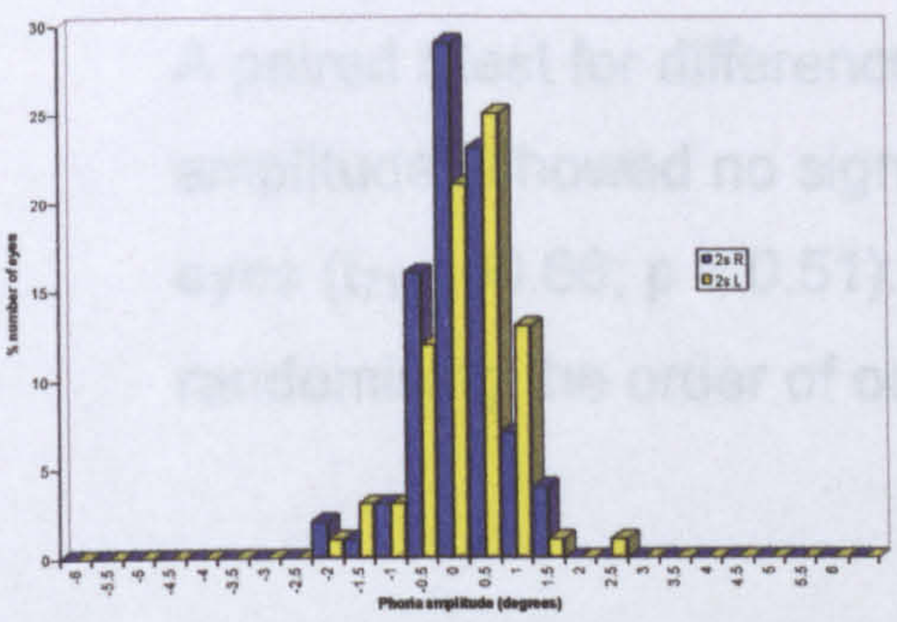
Distance fixation

The maximum phoria amplitude together with amplitudes after 2 s and 10s of occlusion were measured during distance fixation for each eye during the 10 second period of occlusion.

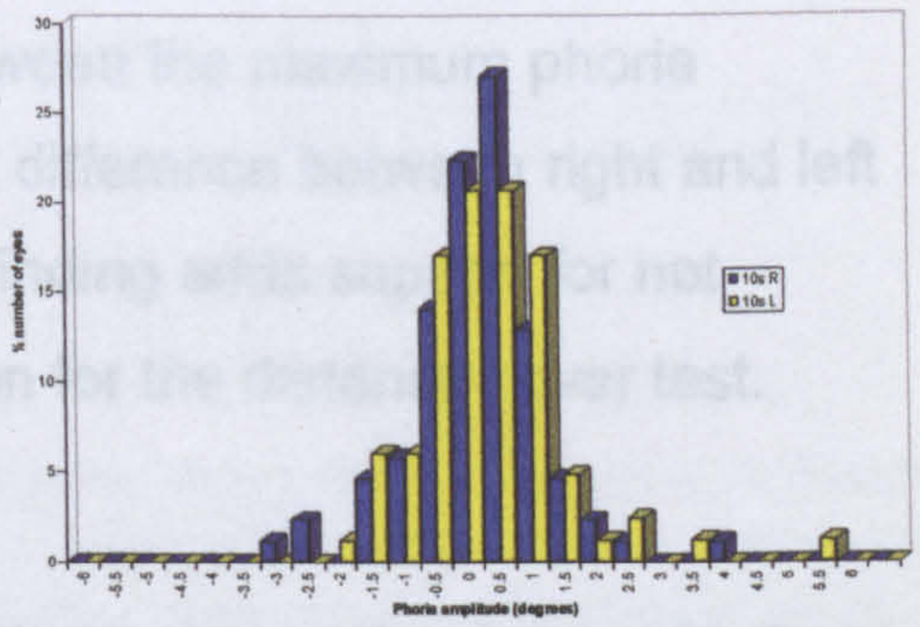
Frequency distributions for right and left eyes are shown in Figure 3.9. Figure 3.9 (a) shows the frequency distribution of the maximum phoria (b) the phoria amplitude measured after 2 seconds of occlusion and (c) after 10 seconds of occlusion.



(a)



(b)



(c)

Figure 3.9. Frequency distributions of phoria amplitudes (degrees) of right and left eyes during the distance cover test (a) maximum phoria (b) after 2 seconds occlusion (c) after 10 seconds occlusion.

Table 3.1 summarises the mean phoria amplitudes for right and left eyes.

	Max RE	Max LE	2s RE	2s LE	10s RE	10s LE
No. of eyes	86	83	85	80	85	82
Mean	-0.15	-0.05	-0.14	-0.15	-0.09	0.04
Standard Deviation	1.21	1.44	0.65	0.71	1.03	1.13

Table 3.1. Summary of mean maximum, 2 second and 10 second distance phoria amplitudes (degrees) for right and left eyes.

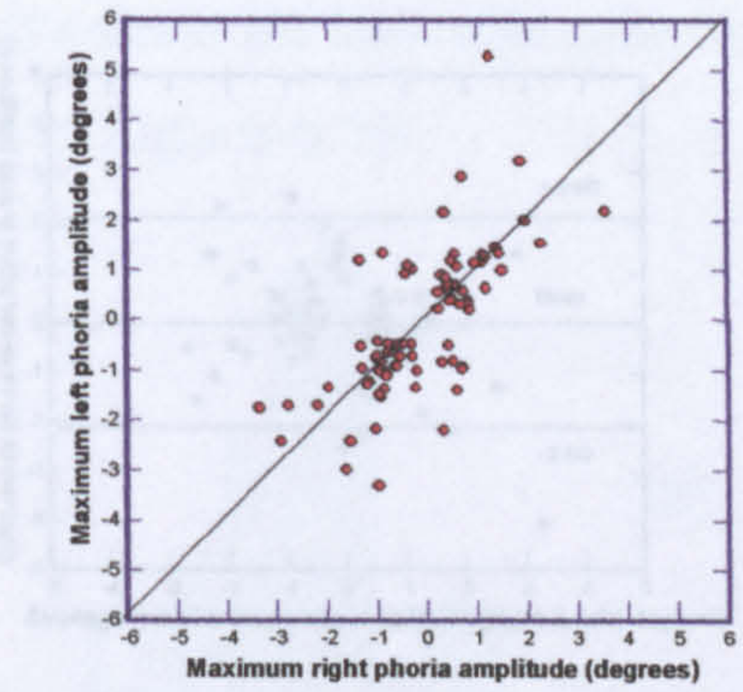
Relationship between right and left eye phoria amplitudes during the distance automated cover test

Most practitioners routinely estimate the amplitude of phoria and make an assumption that the amplitude is the same for both right and left eyes.

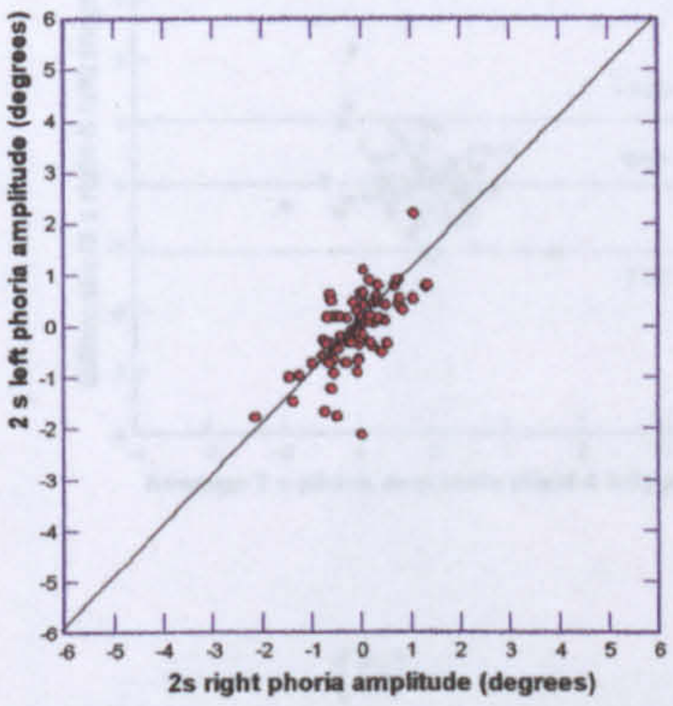
A hypothesis to be tested is that there is no difference in right and left phoria amplitudes.

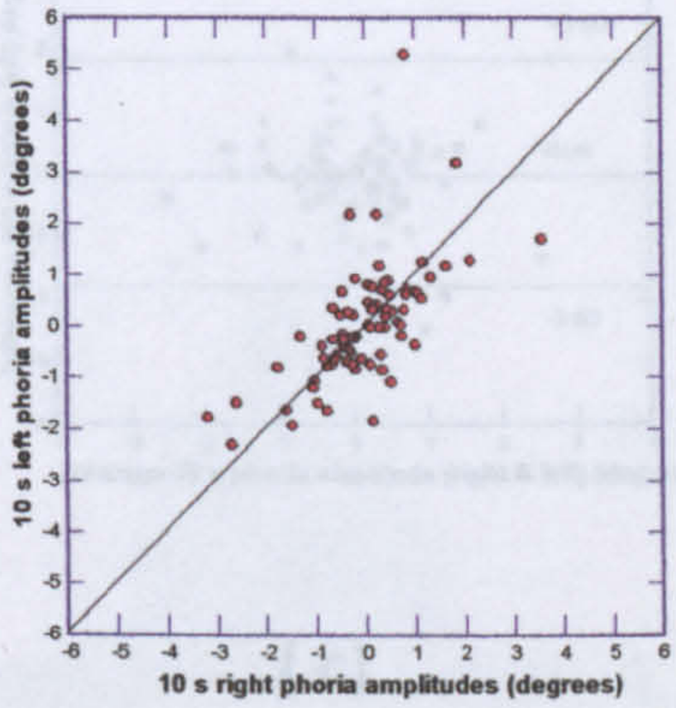
A paired t-test for differences between the maximum phoria amplitudes showed no significant difference between right and left eyes (t_{71} = -0.66; p = 0.51). This finding adds support for not randomising the order of occlusion for the distance cover test.

Figure 3.10 illustrates the correlation between the right and left eyes of individual subjects during the cover test procedure.



(a)



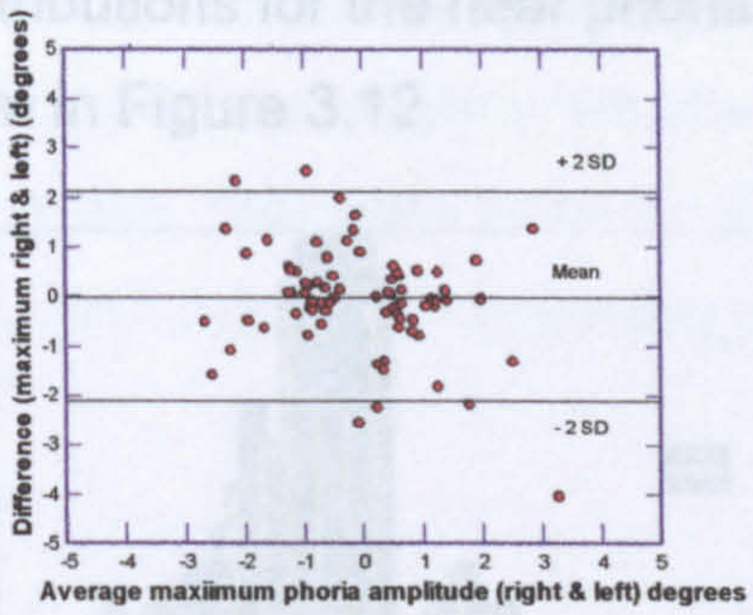


(b) **(c)**

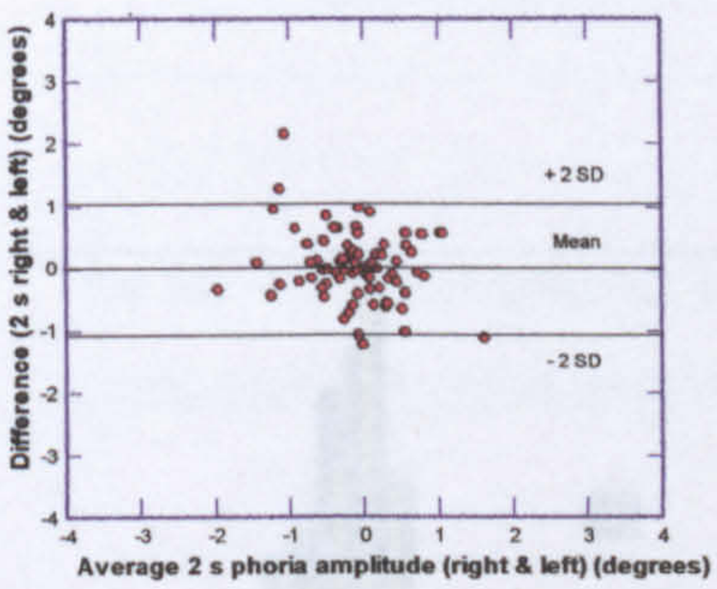
Figure 3.10. Diagram showing relationship between right and left eye phoria amplitudes (degrees) during the distance cover test in the normal group (a) maximum amplitudes (R^2 = 51%; n = 81), (b) after 2 seconds occlusion (R^2 = 43%; n = 79), and (c) after 10 seconds occlusion (R^2 = 42%, n = 81). All show a 1-Tail probability of < 0.001.

These results suggest that there is a strong association between right and left amplitudes.

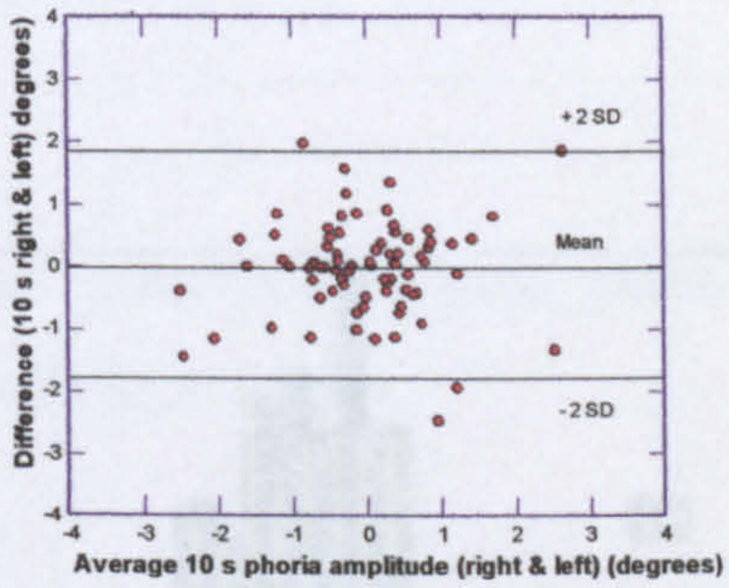
To investigate if the difference between eyes was related to phoria amplitude, the difference between the two eyes was plotted against the average of the two eyes. This is shown in Figure 3.11.



(a)



(b)



(c)

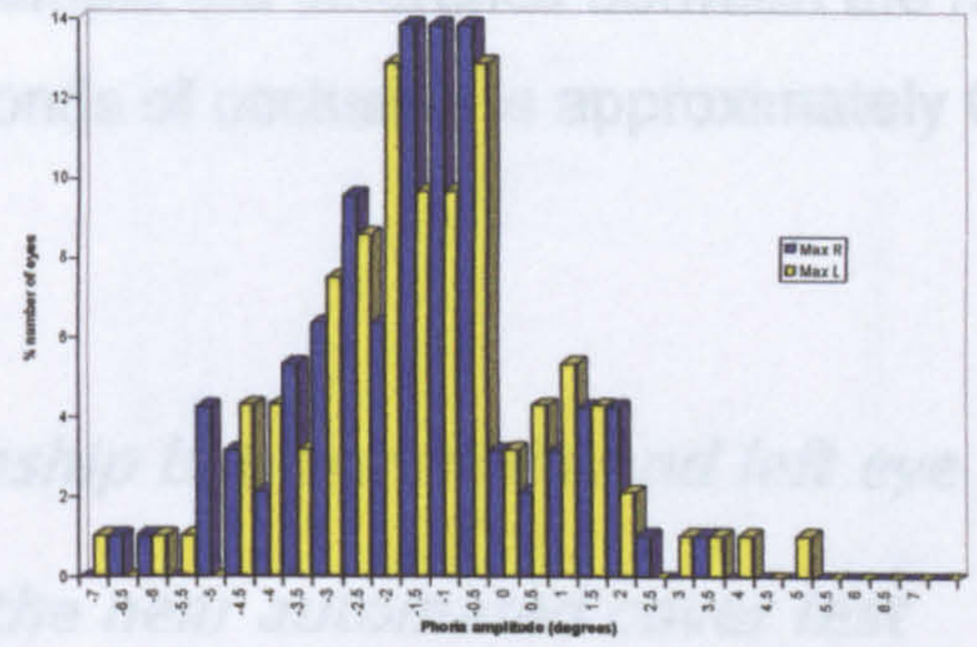
Figure 3.11. Relationship between the difference between right and left eye phoria amplitudes and their average for the distance cover test. (a) maximum phoria amplitudes, (b) 2 second amplitudes and (c) 10 second amplitudes.

The mean difference (right - left) for the maximum, 2 s and 10 s amplitudes were -0.04°, 0.02° and -0.08° respectively. These biases of less than 0.1° are not of any clinical significance. The limits of agreement shown by the ± 2 SD lines on the graphs, gave ranges of 2.06° to - 2.14° for the maximum phoria, 1.13° to -1.09° for the 2 second phoria, and 1.73° to -1.89° for the 10 second phoria. These graphs show no obvious change in the difference between eyes with phoria amplitude.

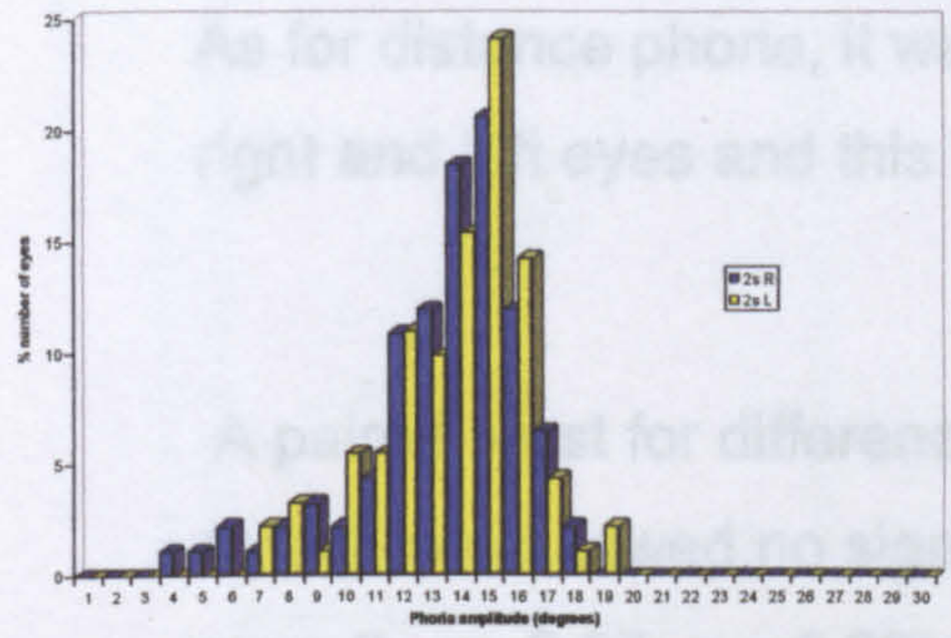
Near fixation

The maximum phoria amplitude together with amplitudes after 2 s and 10s of occlusion were measured during near fixation for each eye during the 10 second period of occlusion.

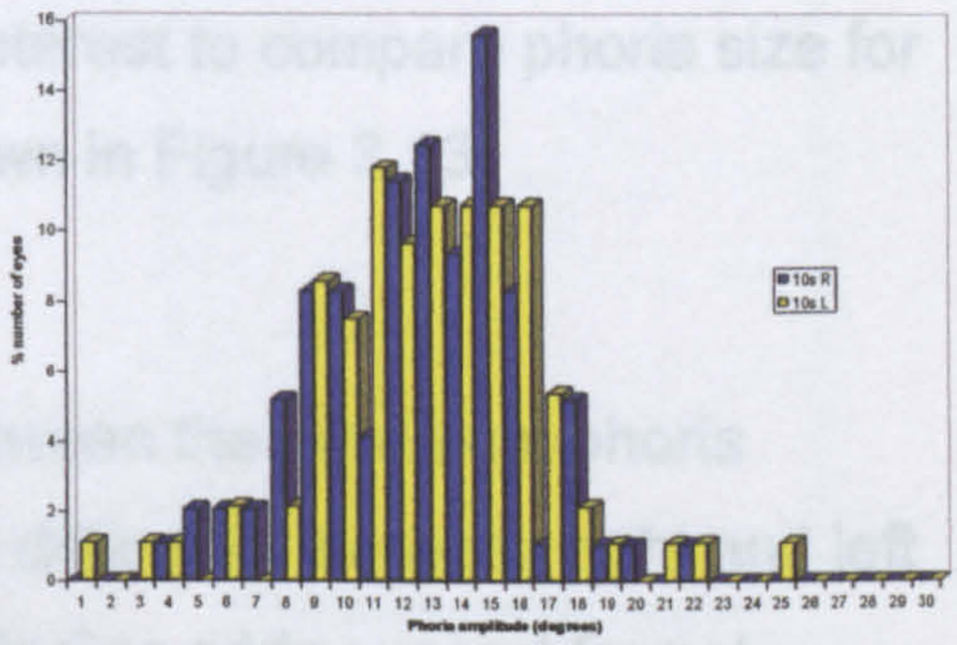
The frequency distributions for the near phoria amplitudes for right and left eyes are shown in Figure 3.12.



(a)



(b)



(c)

Figure 3.12. Frequency distributions (%) of phoria amplitudes (degrees) of right and left eyes during the near cover test (a) maximum phoria (b) after 2 seconds occlusions, (c) after 10 seconds occlusion.

It is apparent from these diagrams that there is a greater variance for the 10 s measurement compared to the 2 s measurement. There does not appear to be an obvious difference between right and left eye amplitudes and this is discussed further in the next section.

Table 3.2 summarises the mean phoria amplitudes for right and left eyes.

	Max RE	Max LE	2s RE	2s LE	10s RE	10s LE
No. of eyes	94	93	92	91	96	93
Mean	-1.74	-1.60	-1.03	-0.89	-1.42	-1.31
Standard Deviation	1.95	2.11	1.41	1.26	1.75	1.87

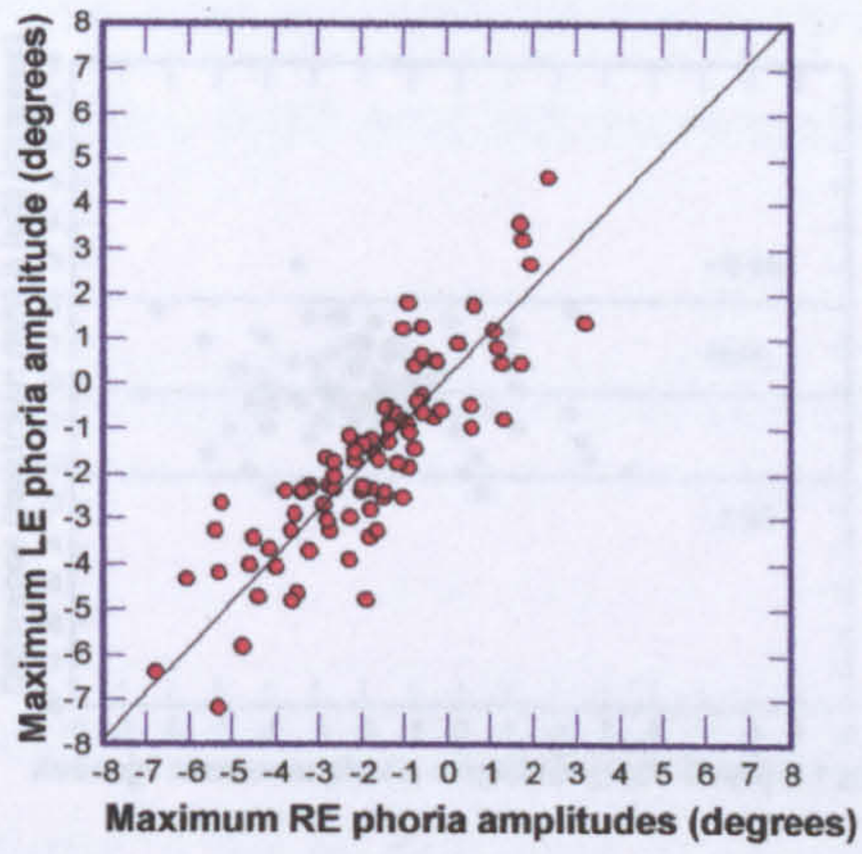
Table 3.2. Summary of mean maximum, 2 second and 10 second distance phoria amplitudes (degrees) for right and left eyes.

As for the distance cover test, the differences between the means of phoria amplitude for the three measurements are not large in clinical terms. For example the difference between the mean amplitudes after 2 and 10 seconds of occlusion is approximately 0.5° (or ~ 1^{Δ}).

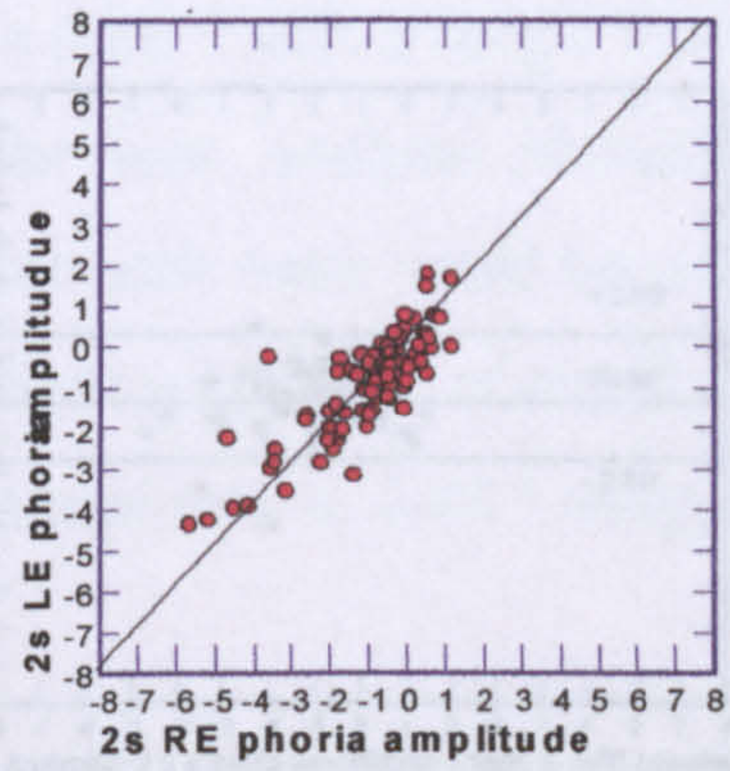
Relationship between right and left eye phoria amplitudes during the near automated cover test

As for distance phoria, it was of interest to compare phoria size for right and left eyes and this is shown in Figure 3.13.

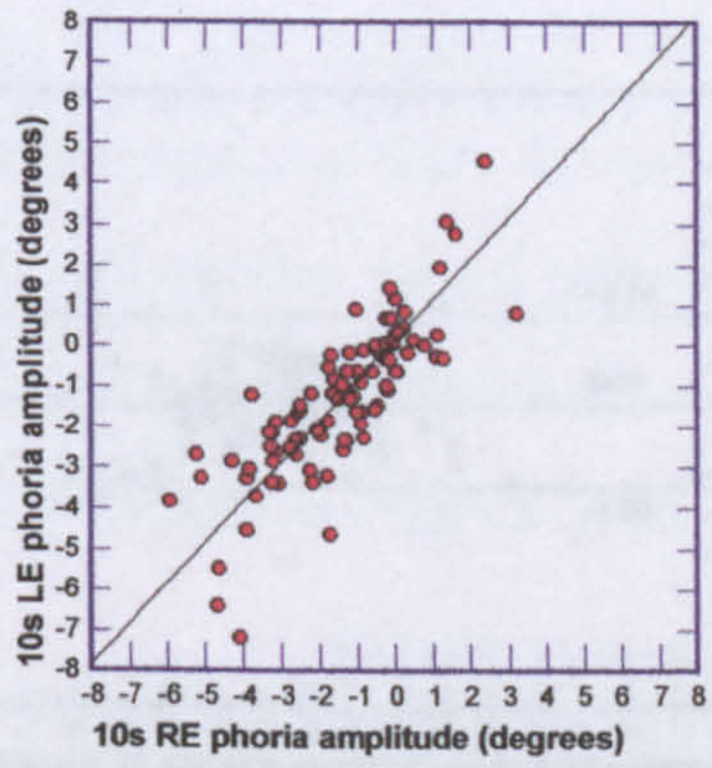
A paired t-test for differences between the maximum phoria amplitudes showed no significant difference between right and left eyes (t_{71} = -0.87; p = 0.39). This finding adds support for not randomising the order of occlusion for the near cover test.



(a)



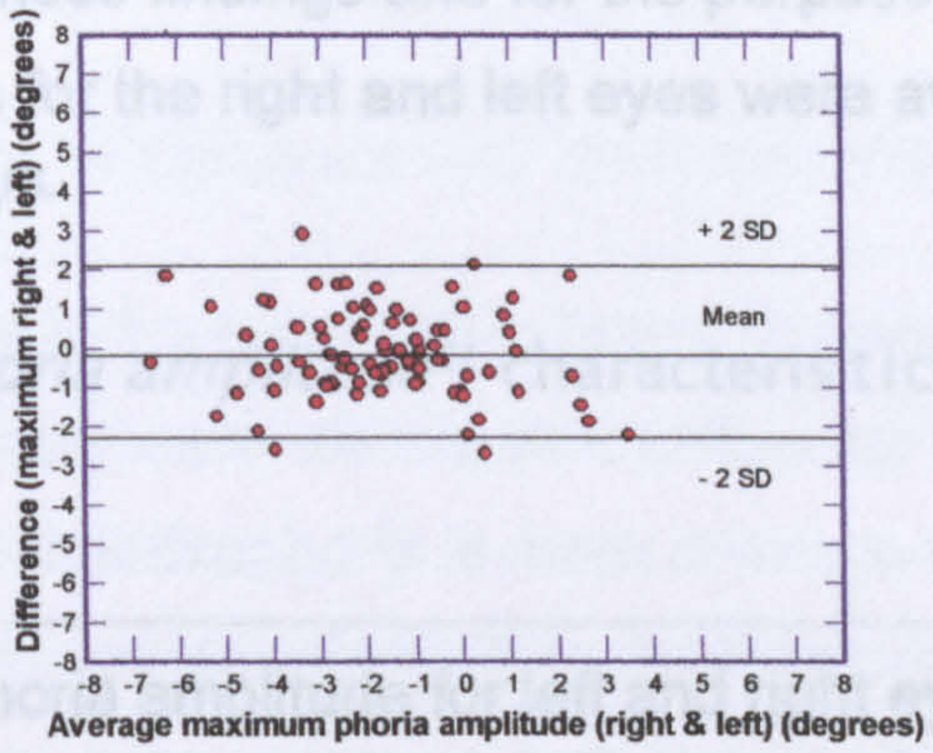
(b)



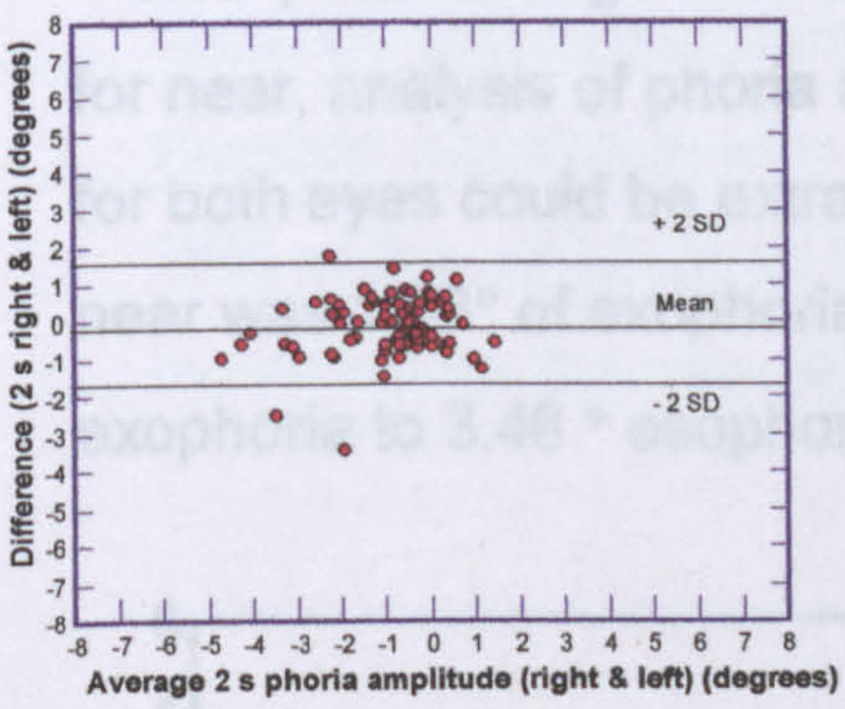
(c)

Figure 3.13. Diagram showing relationships between right and left eye phoria amplitudes (degrees) during the near cover test in the normal group (a) maximum amplitudes (Pearson R^2 = 73%, n= 91) (b) after 2 seconds occlusion (R^2 = 70%, n = 87) and (c) after 10 seconds occlusion (R^2 = 67%, n = 93). All show a 1-Tail probability of < 0.001.

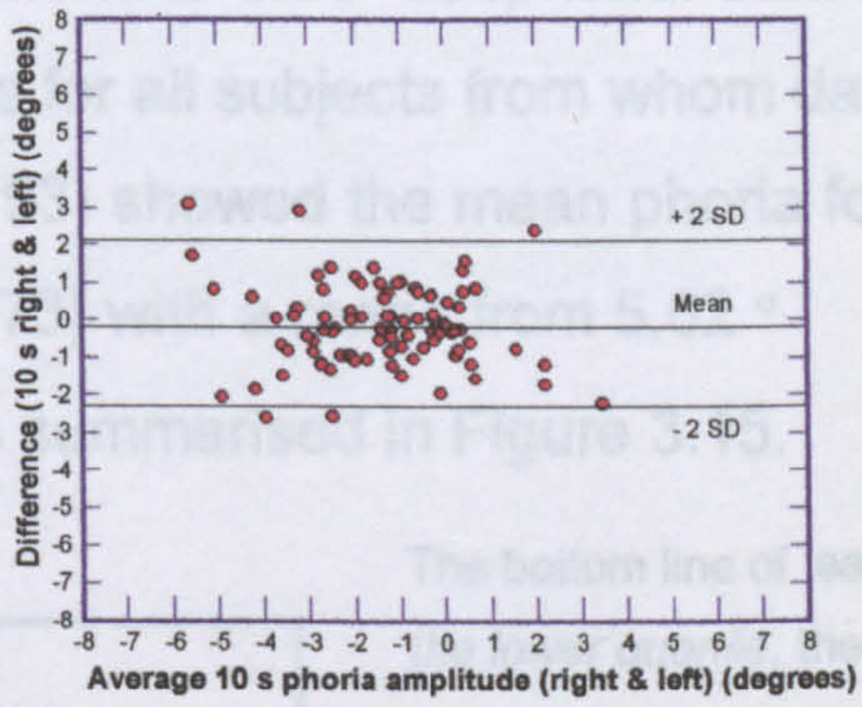
A further analysis was carried out to compare the difference between the right and left amplitude against the mean of the right and left amplitudes and this is shown in Figure 3.14.



(a)



(b)



(c)

Figure 3.14. Relationship between the difference between right and left eye phoria amplitudes and their average for the near cover test. (a) maximum phoria amplitudes, (b) 2 second amplitudes, and (c) 10 second amplitudes.

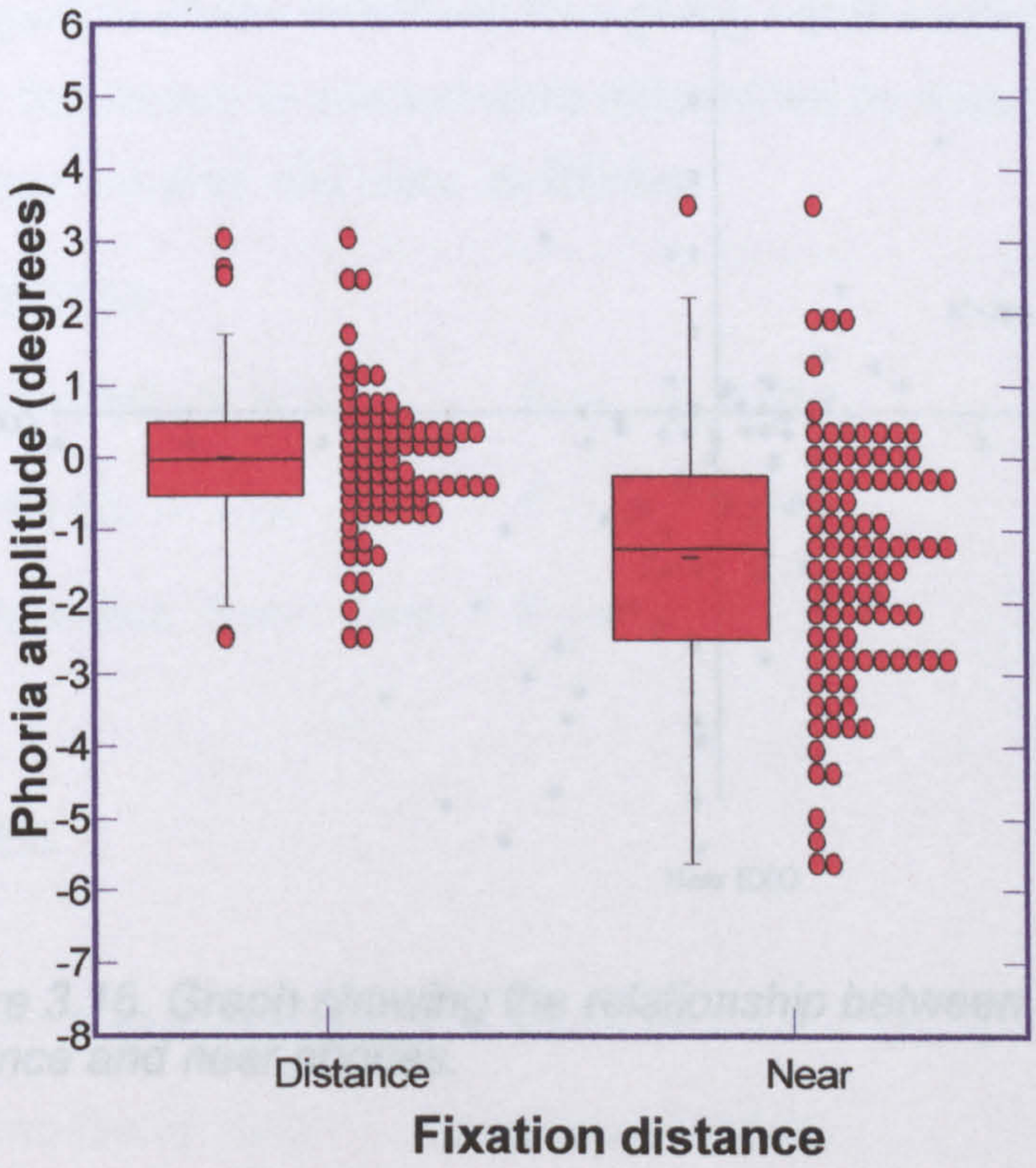
The mean of differences (right - left) for the maximum, 2 s and 10 s amplitudes were -0.14°, -0.14 ° and -0.13° respectively. The limits of agreement shown by the ± 2 SD lines on the diagrams, gave ranges of 2.08 ° to - 2.36 ° for the maximum phoria, 1.46° to -1.74° for the 2 second phoria, and 2.09° to -2.35° for the 10 second phoria. These graphs show no obvious change in the difference between eyes with phoria amplitude.

In clinical practice, most practitioners assume the amplitude of phoria to be equal in both eyes and just one measurement is recorded in the notes. The results of the statistical analyses suggest that this is a reasonable assumption, at least for “normal” young adult subjects.

On the basis of these findings and for the purpose of some specific analyses, results for the right and left eyes were averaged to give *phoria amplitude*^{RL}.

Summary of *phoria amplitude*^{RL} characteris tics for the normal group

Combined 10 s phoria amplitude for left and right eyes was calculated (*10 s phoria amplitude*^{RL}) for all subjects from whom data for both eyes could be extracted. The mean phoria for distance (n = 80) was 0.00° (SD = 0.98°) with a range from 2.51° exophoria to 3.03° esophoria. Similarly, for near, analysis of phoria amplitudes for all subjects from whom data for both eyes could be extracted (n = 93) showed the mean phoria for near was 1.38° of exophoria (SD = 1.73) with a range from 5.62 ° exophoria to 3.48 ° esophoria. This is summarised in Figure 3.15.



The bottom line of each box denotes *the lower quartile*, the middle line denotes the *median* and the top line denotes the *upper quartile*.

The lower whisker denotes the *lower adjacent value* (This is equal to the maximum of (i) lower quartile minus 1.5 times the inter-quartile range and (ii) the minimum observation). Any values below this are *outliers* and are plotted individually. The upper whisker denotes *the upper adjacent value*. This is equal to the minimum of (i) upper quartile plus 1.5 times the inter-quartile range and (ii) the maximum observation. Any values above this are *outliers* and are plotted individually.

The frequency distribution is shown as a dot plot. Dots outside of whiskers denote 'outliers'.

Figure 3.15. Box and whisker and dot plot illustrating the distribution of phoria amplitude^{RL} *for distance and near.*

Comparison of distance and near amplitudes[RL]

Figure 3.16 shows the relationship between distance and near phorias. The preponderance of points below the horizontal axis reflects the fact that exophoria is more common than esophoria for near fixation. The cluster of points in the lower right quadrant indicates that it is not uncommon to find a small esophoria in the distance and exophoria at near. The scarcity of points in the upper left quadrants indicates that it is uncommon to find an exophoria in the distance and an esophoria at near (at least in emmetropic subjects). A Pearson correlation was calculated and from this R^2 determined. Only 26% of the variability of the distance and near data could be explained by the association of the two variables.

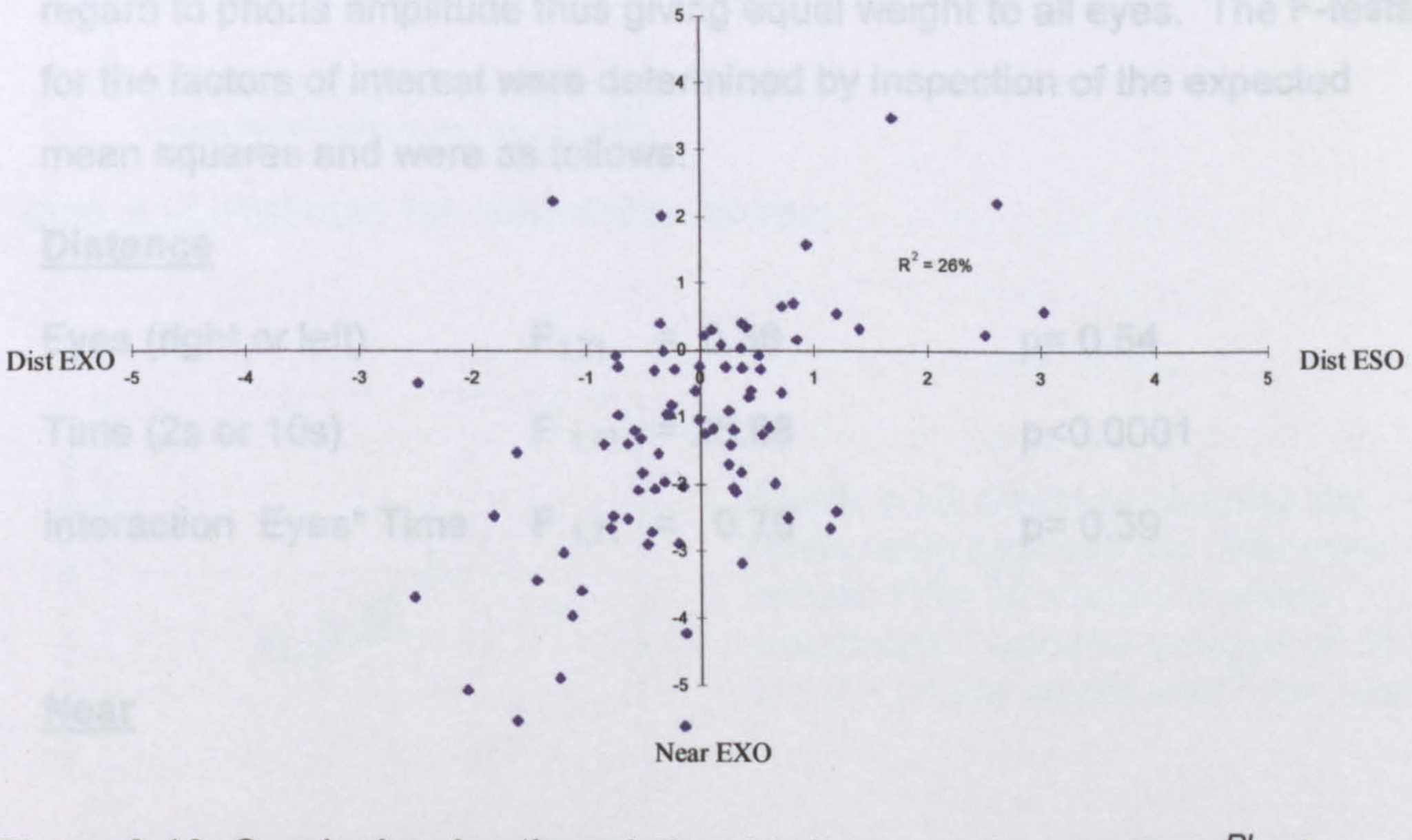


Figure 3.16. Graph showing the relationship between the amplitude[RL] of distance and near phorias.

II. Relationship between the 2 second and 10 second phoria amplitudes for each subject

A hypothesis to be investigated is that there was no difference in a subject's phoria amplitude at 2 and 10 seconds of occlusion for either distance or near cover tests.

This hypothesis was tested using a three-way factorial analysis of variance. The three factors were eyes (right, left), time (2 s, 10s), and subjects. Eyes and time were considered fixed effects and subjects were considered as random effects. Only subjects for whom a full set of data were available were included in the analysis (n = 72 for distance; n = 71 for near). In order to remove the negating effect of exophoric amplitudes being positive and esophoric amplitudes being negative, each 2 s and 10 s amplitude was transformed into a ratio compared to the maximum phoria amplitude for that eye. This treatment also had the effect of standardising subjects with regard to phoria amplitude thus giving equal weight to all eyes. The F-tests for the factors of interest were determined by inspection of the expected mean squares and were as follows:

Distance

Eyes (right or left)	$F_{1,71} = 0.38$	p= 0.54
Time (2s or 10s)	$F_{1,71} = 25.88$	p<0.0001
Interaction Eyes* Time	$F_{1,71} = 0.75$	p= 0.39

Near

Eyes (right or left)	$F_{1,70} = 0.25$	p= 0.62
Time (2s or 10s)	$F_{1,70} = 27.42$	p<0.0001
Interaction Eyes* Time	$F_{1,70} = 0.01$	p= 0.94

No difference was observed in phoria amplitude between right and left eyes for either distance or near. There were statistically significant differences between 2 s and 10 s amplitudes. For distance, the mean 2 s amplitude was 36% of the maximum amplitude while the 10 s was 62%. For near, the mean 2 s amplitude was 43% of the maximum amplitude while the 10 s was 67%.

This relationship is also illustrated in Figures 3.16 & 3.17, which plot the difference between the 10 s and 2 s *phoria amplitudes*^{RL} against the average of 10 s and 2 s *phoria amplitudes*^{RL} for distance and near.

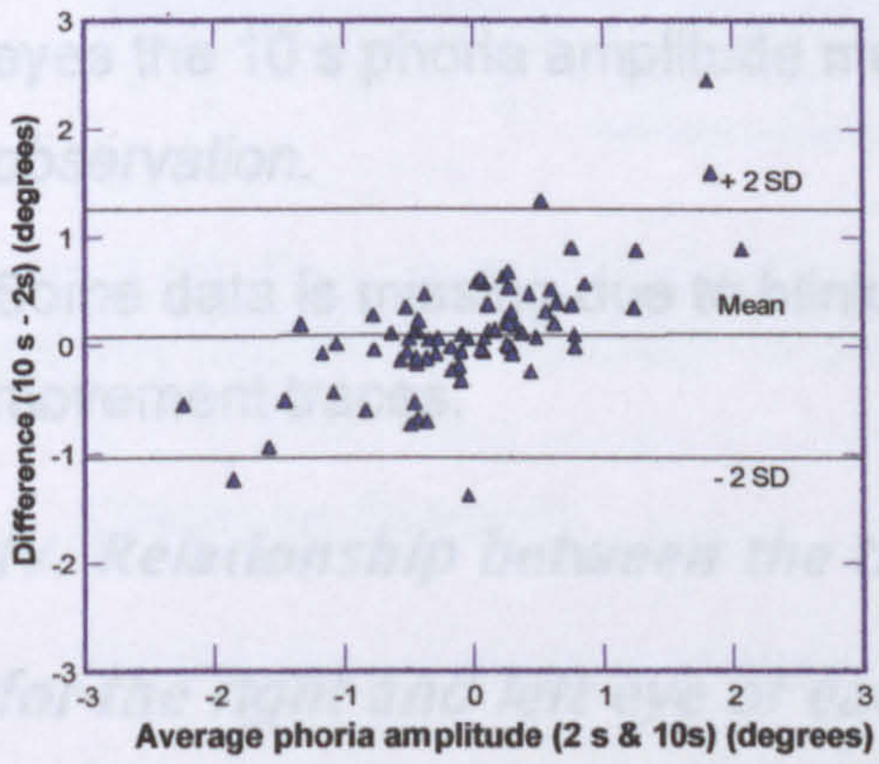


Figure 3.17. Diagram showing the relationship between the difference between the 10 s and 2 s phoria amplitudes^{RL} *and the average of 10 s and 2 s phoria amplitudes*^{RL} *for distance.*

Figure 3.18 illustrates the relationship for near.

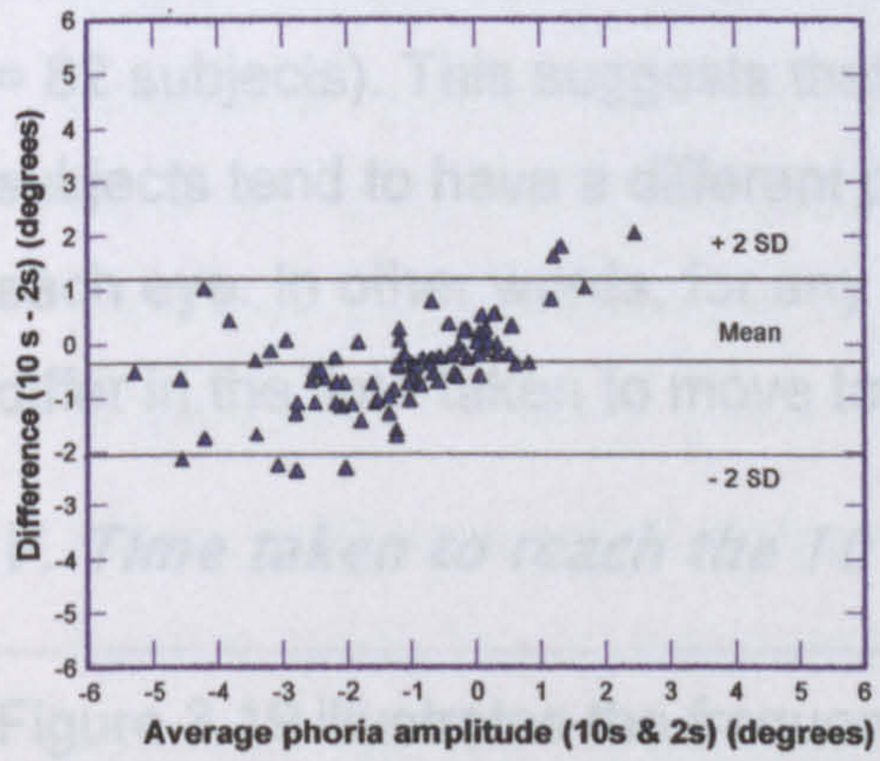


Figure 3.18. Diagram showing the relationship between the difference between the 10 s and 2 s phoria amplitudes^{RL} *and the average of 10 s and 2 s phoria amplitudes*^{RL} *for near.*

These results suggest that the difference between the 10 s and 2 s amplitudes increases with the amplitude for both exo and eso deviations.

It would be helpful from a clinical viewpoint to establish the minimum duration of occlusion required for the eyes to adopt a position of relative equilibrium.

Further information to help address this question may be found by analysing the mean times for the subjects' eyes to reach the 10 second phoria amplitude.

III. Time taken to reach the 10 s amplitude

In the experiment, the duration of the occlusion was 10 s. This was sufficient for most eyes to reach a position of equilibrium. However, in a few cases the eyes were still moving when the cover was removed. For these eyes the 10 s phoria amplitude measurement is described as a *censored observation.*

Some data is missing due to blinks obscuring critical periods in the eye movement traces.

IV. Relationship between the time taken to reach the 10 s amplitude for the right and left eye of each subject

There was a very weak relationship between the right and left eyes of subjects for the time taken to reach the phoria amplitude measured at 10 seconds for both distance (R^2 = 6%; n = 78 subjects) and near (R^2 = 6%; n = 82 subjects). This suggests that, during the distance cover test, many subjects tend to have a different profile of movement under the cover for each eye. In other words, for any one subject, the right and left eyes may differ in the time taken to move towards their positions of rest.

V. Time taken to reach the 10 s amplitude

Figure 3.19 illustrates the frequency distributions of the time taken to reach the phoria amplitude measured following 10 s occlusion. The distributions appear to be bimodal for both distance and near fixation.

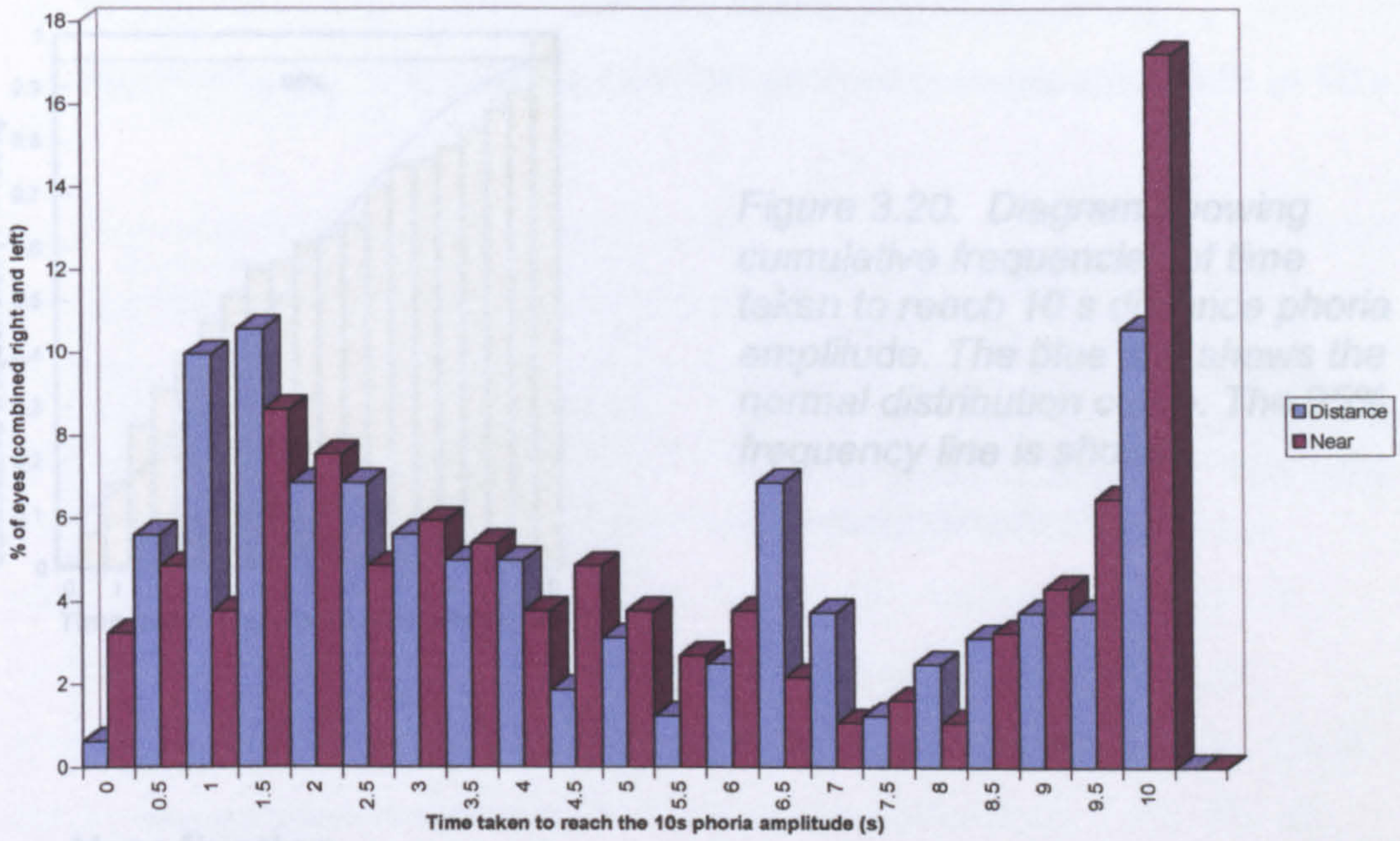


Figure 3.19. Frequency distributions of the time taken to reach the phoria amplitude measured following 10 s occlusion.

Distance fixation

For the distance cover test on the normal group (n = 161 eyes), the mean time to reach the10 s phoria position was 4.36 s (SD = 3.27 s). These results excluded eyes for which the phoria amplitude at 10 s was measured to be 0.00 degrees. For exophoric eyes (n = 80) the mean time to reach the 10 s amplitude was 3.57 s (SD = 2.58 s) and for esophoric eyes (n = 81) the mean time was 5.13 s (SD = 3.41 s). A Mann-Whitney U Test showed a significant difference between the esophoric and exophoric eyes for the time taken to reach the 10 s amplitude (p = 0.004). These results are summarised in Table 3.3.

Figure 3.20 shows cumulative frequencies for the duration required for eyes to reach the 10 s distance phoria amplitude. The data includes eyes that had not yet achieved a stable position by10 s. Whilst 75% of the sample had reached a stable amplitude by 7 s, approximately 12% of eyes had not reached a stable amplitude by 10 s.

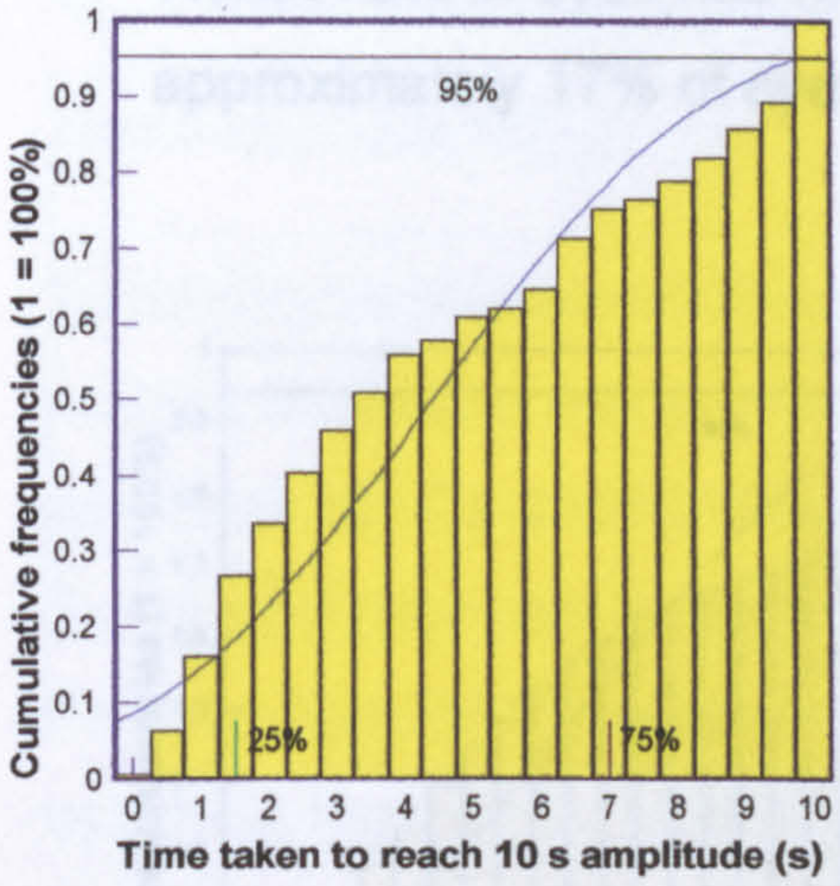


Figure 3.20. Diagram showing cumulative frequencies of time taken to reach 10 s distance phoria amplitude. The blue line shows the normal distribution curve. The 95% frequency line is shown.

Near fixation

For the near cover test (n = 186) the mean time was 4.96 s (SD = 3.48 s). For exophoric eyes (n = 149) the mean time to reach the 10 s amplitude was 5.15 s (SD = 3.38 s) and for esophoric eyes (n = 37), the mean time was 4.20 s (SD =3.86 s). There was no significant difference in this respect between the esophoric and exophoric eyes for the near cover test (p = 0.20). These results are summarised in Table 3.3.

Distance			Near		
All amplitudes	***Mean time to reach 10s amplitude (s)***	***p ****	***All amplitudes***	***Mean time to reach 10s amplitude (s)***	***P****
All eyes	4.36 ±3.27 (n = 161)	0.004	All eyes	4.96 ± 3.48 (n = 186)	0.20
Exo eyes	3.57 ±2.58 (n = 80)		Exo eyes	5.15 ± 3.38 (n = 149)	
Eso eyes	5.13 ±3.41 (n = 81)		Eso eyes	4.20 ± 3.86 (n = 37)	

*Mann-Whitney U Test for difference between the exophoric and esophoric groups (± data = 1 standard deviation).

Table 3.3. Mean times for eyes to reach the 10 s phoria amplitude during occlusion.

Figure 3.21 shows the cumulative frequencies for the duration required for eyes to reach the 10 s near phoria amplitude. The 10 s cumulative data includes eyes that had not achieved a stable position at 10 s).

Whilst 75% of eyes had reached a stable amplitude by 9 s, approximately 17% of eyes had not reached a stable amplitude at 10 s.

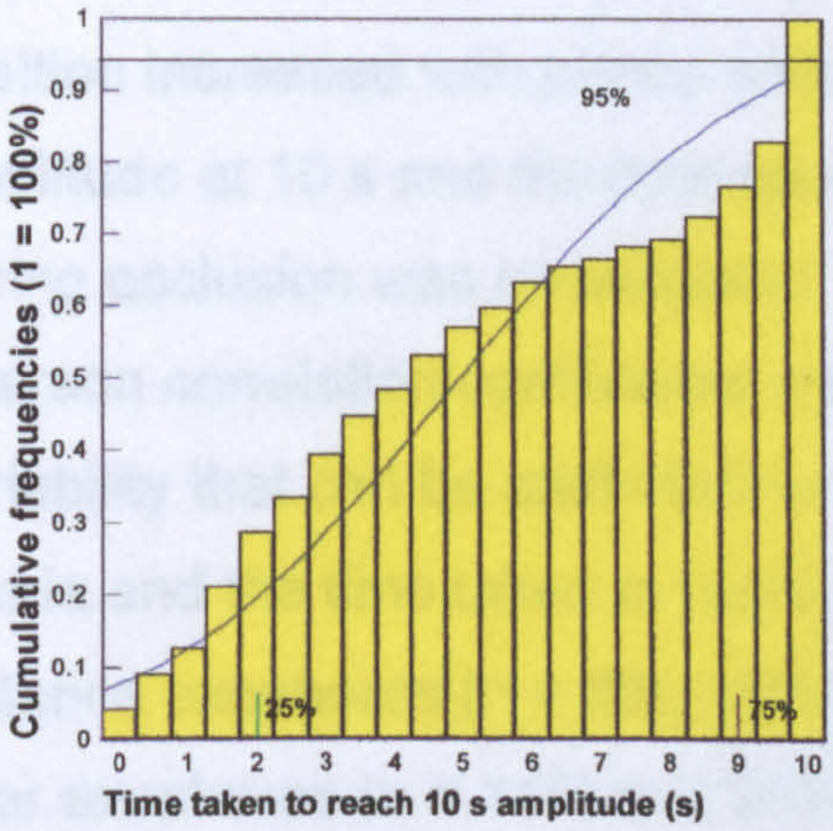


Figure 3.21. Diagram showing cumulative frequencies of time taken to reach 10 s near phoria amplitude. The blue line shows the normal distribution curve. The 95% frequency line is shown.

For distance, 9 esophoric eyes took 10 seconds to reach the maximum amplitude recorded under the cover[2]. However, 6 of these eyes manifested a phoria of less than 1degree amplitude and caution must be used with phoria amplitudes of such a small order because of the presence of normal saccades made during fixation. For the exophoric group only 1 eye took 10 seconds to reach the maximum phoria position (1.3 degrees).

For near exophoria, 20 eyes took 10 seconds to reach the maximum amplitude[2]. Of these, 3 eyes manifested amplitudes of less than 1 degree.

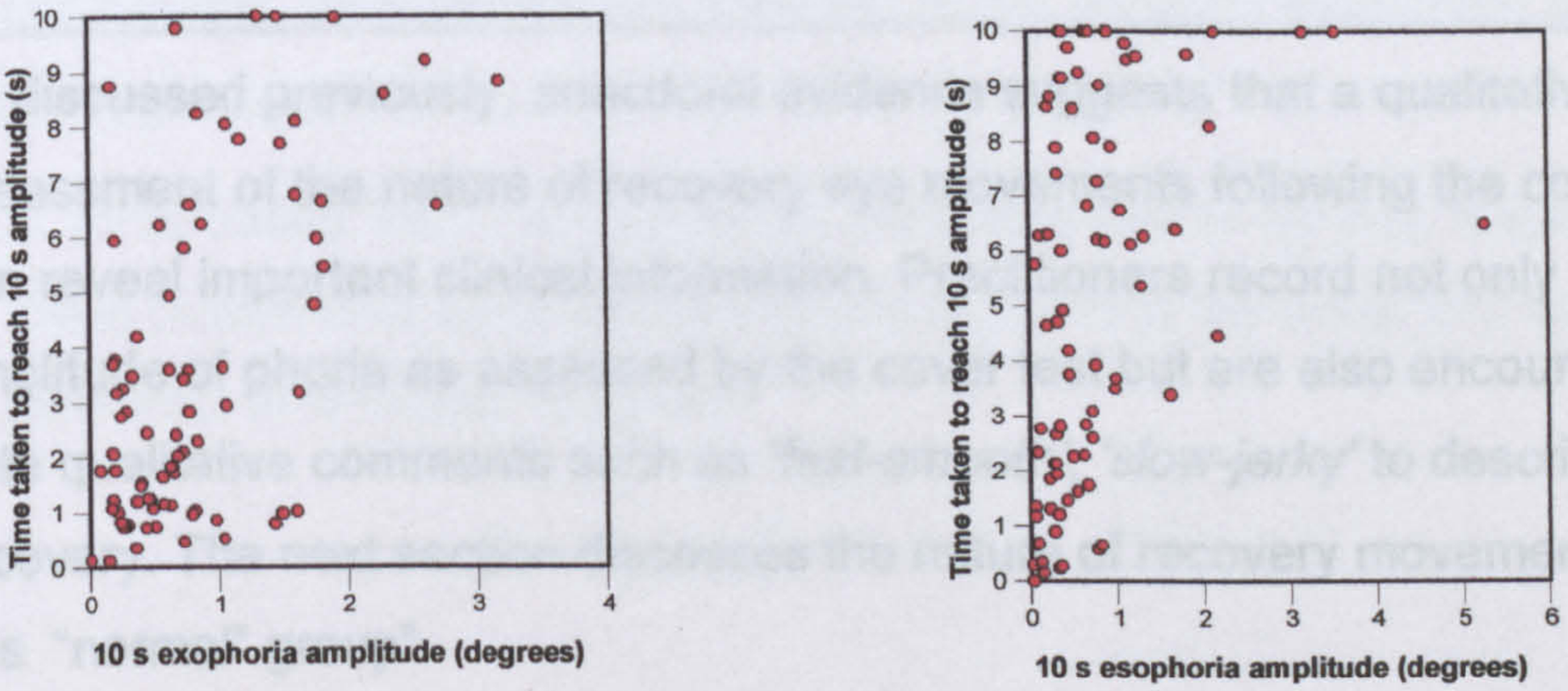
Of the near esophoric group 3 eyes took 10 seconds to reach the maximum phoria position during the 10 s period of occlusion[2]. One of these had esophoric amplitude of just 0.09 degrees and so should be ignored. The other two eyes were 3.2 and 4.6 degrees.

---

[2] Includes *censored* observations in which the eyes were still moving towards a position of rest as the occluder was removed.

VI. Correlation between 10 s phoria amplitude and the time taken to reach that amplitude

To determine whether the time taken for the occluded eye to reach a stable position increased with phoria amplitude, the relationship between phoria amplitude at 10 s and the time taken for the eye to reach that position during occlusion was investigated. Some data was missing due to blinks. Pearson correlation coefficients were calculated and the percentage variability that can be attributed to an association between the size of phoria and the time taken to reach that amplitude (R^2) was 34% for distance exophores (n = 82), 15% for distance esophores (n = 82), 6% for near exophores (n = 149) and 25% for near esophores (n = 38). For all distance phorias R^2 was 45% (n = 164) and for all near phorias, 10% (n = 187) ($p < 0.001$ for all groups). These findings suggest only a weak to moderate association between amplitude and time taken to reach that amplitude and this is also illustrated in Figure 3.22 for distance fixation and Figure 3.23 for near fixation.



(a) **(b)**

Figure 3.22. Scatter diagram illustrating the relationship between 10 s distance phoria amplitude and the time taken to reach that amplitude. (a) exophoric eyes and (b) esophoric eyes.

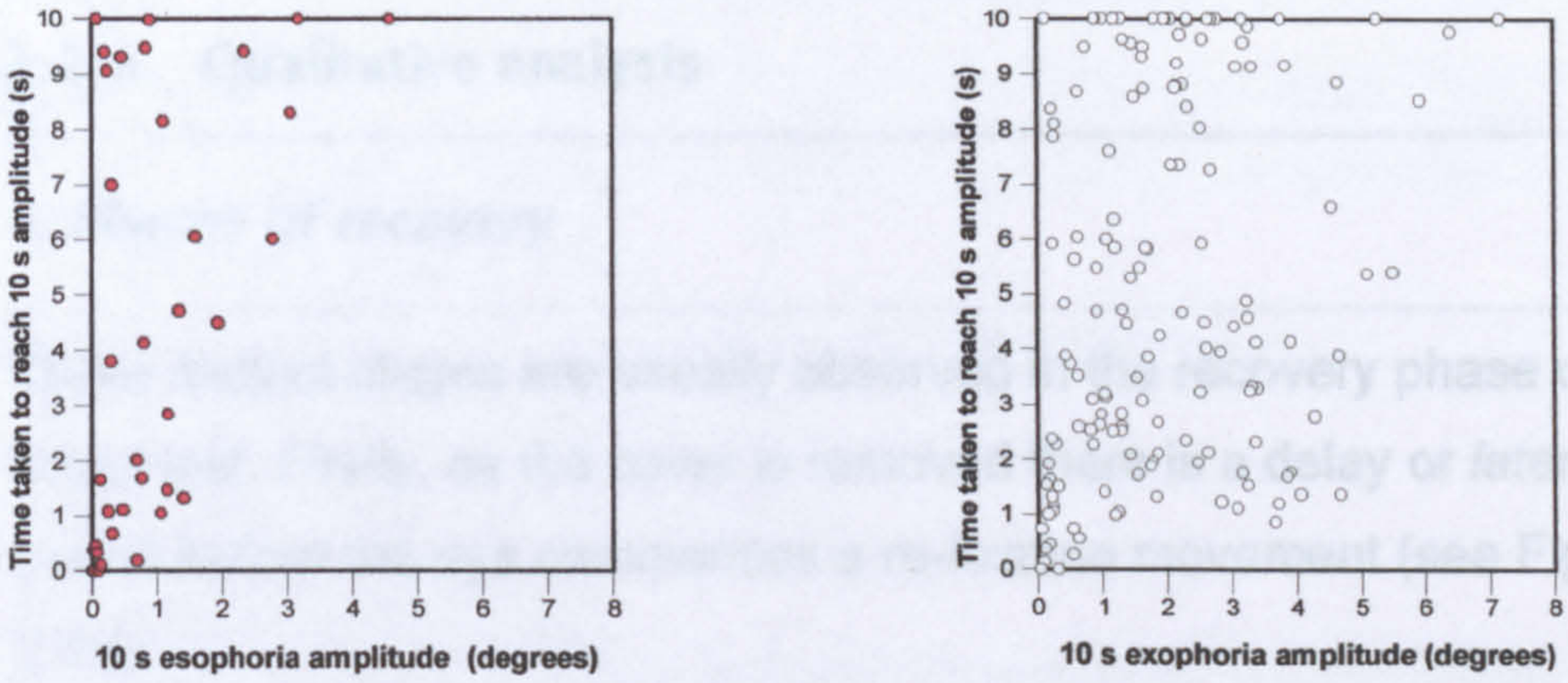


Figure 3.23. Scatter diagram illustrating the relationship between 10 s near phoria amplitude and the time taken to reach that amplitude. (a) esophoric eyes and (b) exophoric eyes.

The preceding findings suggest that larger phorias may not necessarily take longer to reach their resting point during dissociation when compared to smaller phorias and that for any one subject, one eye may show a different fusional decay profile than its fellow.

3.2.2 The recovery phase

As discussed previously, anecdotal evidence suggests that a qualitative assessment of the nature of recovery eye movements following the cover test can reveal important clinical information. Practitioners record not only the amplitude of phoria as assessed by the cover test but are also encouraged to note qualitative comments such as '*fast-smooth*', '*slow-jerky*' to describe the recovery. The next section discusses the nature of recovery movements for this "normal" group".

3.2.2.a. Qualitative analysis

I. Phases of recovery

Three distinct stages are usually observed in the recovery phase of the cover test. Firstly, as the cover is removed there is a delay or *latency* period before the eye commences a re-fixation movement (see Figure 3.24).

The cover commences movement at zero minus 19 ms (in this case 13.81 s into the recording procedure) and crosses the centre of the pupil at 14.00 s. The cover ceases movement at zero plus 19 ms (in this case 14.19 s). The latency for the commencement of recovery was taken as the time from when the occluder passed through the centre of the pupil during the uncover phase, to the start of the recovery movement. The latter was taken as the point in time when the eye being uncovered was judged to make its first movement towards the fixation target. This was judged visually with the aid of the colour coding algorithm incorporated in the analysis program.

Recovery movements frequently involved saccades as well as vergence movements. An example of a saccade as the initiating movement is shown in Figure 3.26.

In some cases, very short latencies were recorded (< 60 ms). It is likely that these movements were initiated before removal of the cover and were incidental to binocular recovery.

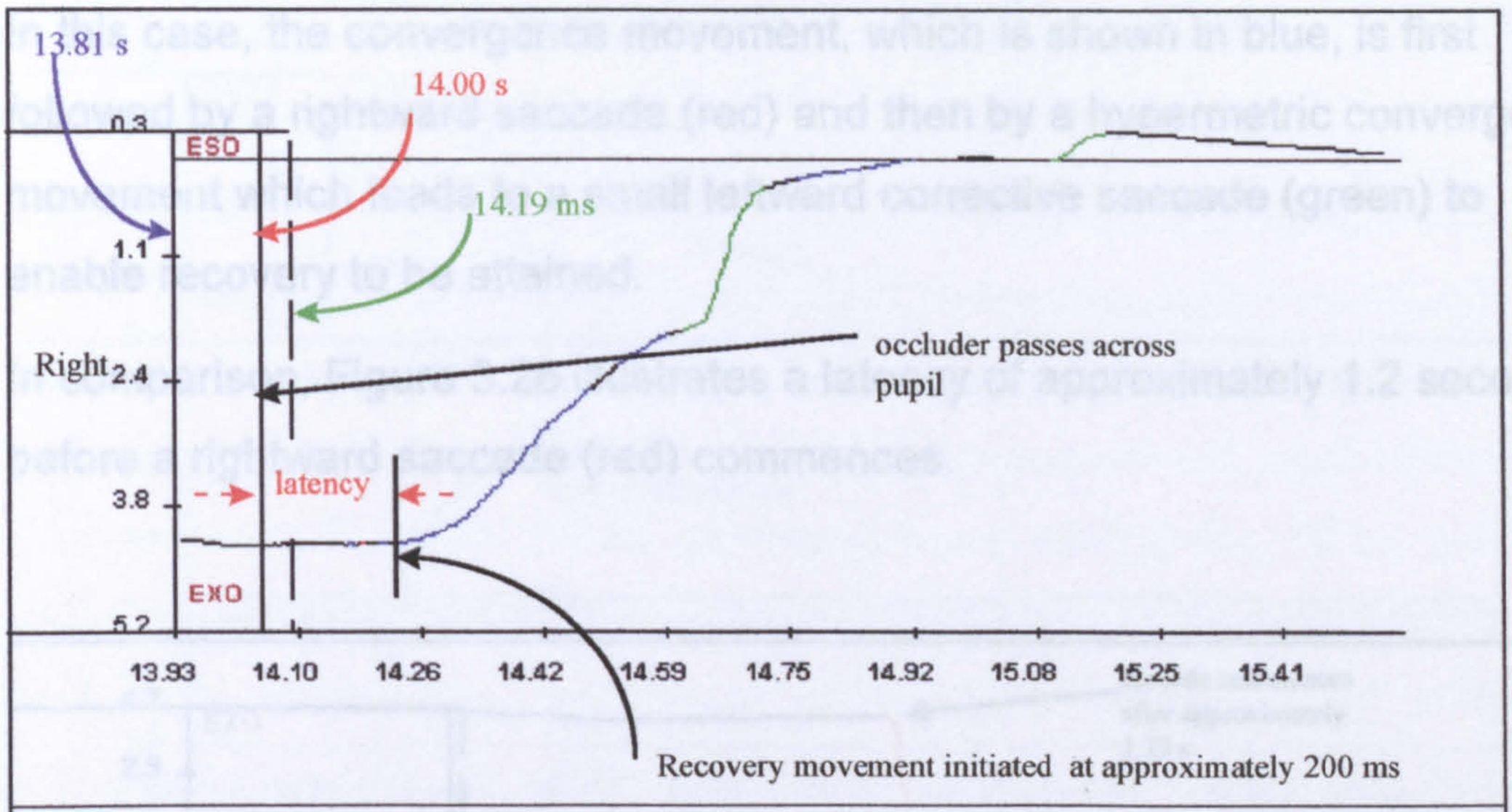


Figure 3.24. Eye movement trace illustrating the latency for a recovery movement following removal of the cover.

Latencies varied significantly between subjects. Figures 3.24, 3.25 & 3.26 illustrate further examples of 'average', 'long' and 'short' latencies.

Figure 3.25 shows a typical 200 ms latency for the left eye.

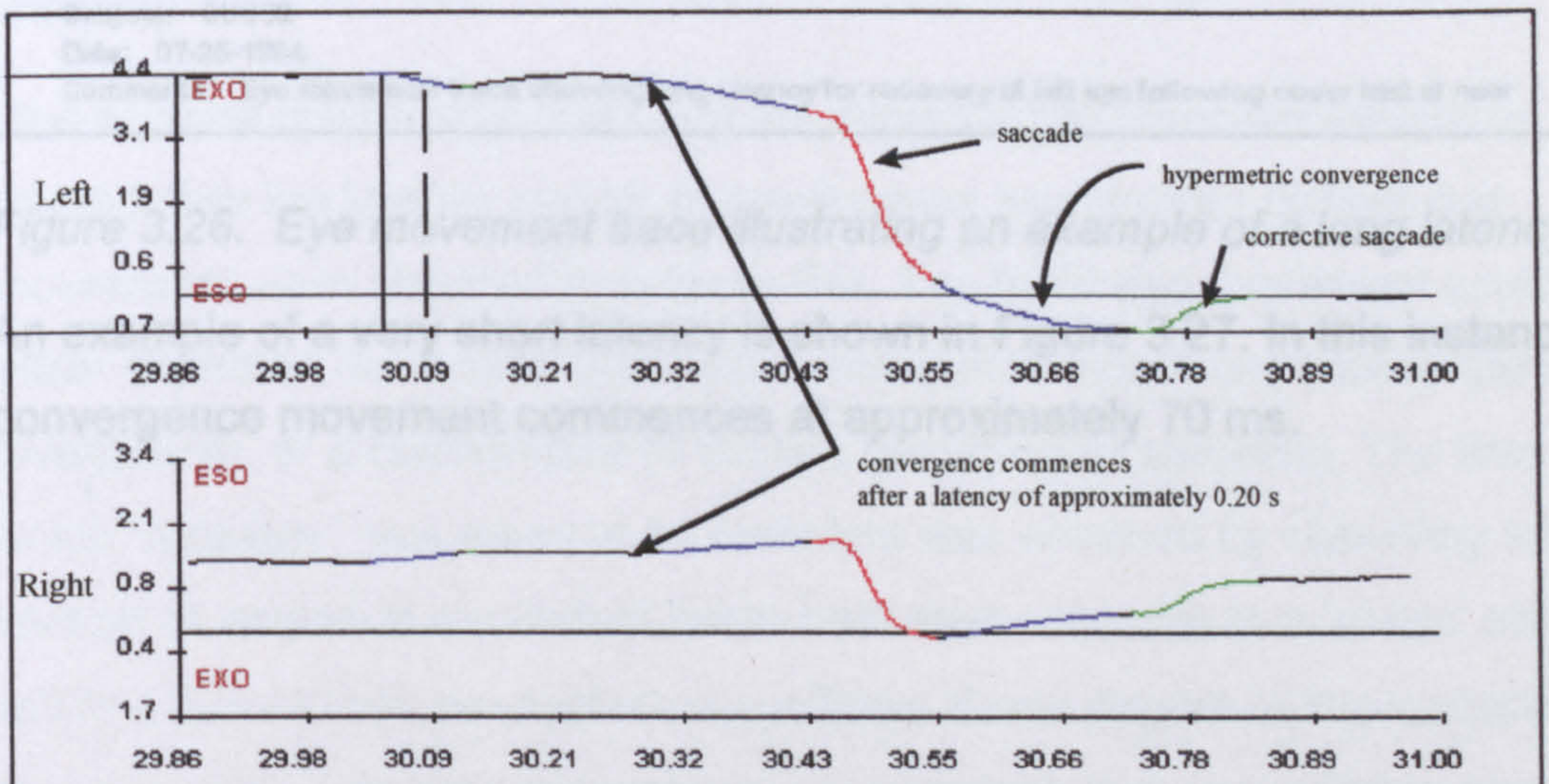


Figure 3.25. Eye movement trace illustrating an example of an 'average' latency.

In this case, the convergence movement, which is shown in blue, is first followed by a rightward saccade (red) and then by a hypermetric convergence movement which leads to a small leftward corrective saccade (green) to enable recovery to be attained.

In comparison, Figure 3.26 illustrates a latency of approximately 1.2 seconds before a rightward saccade (red) commences.

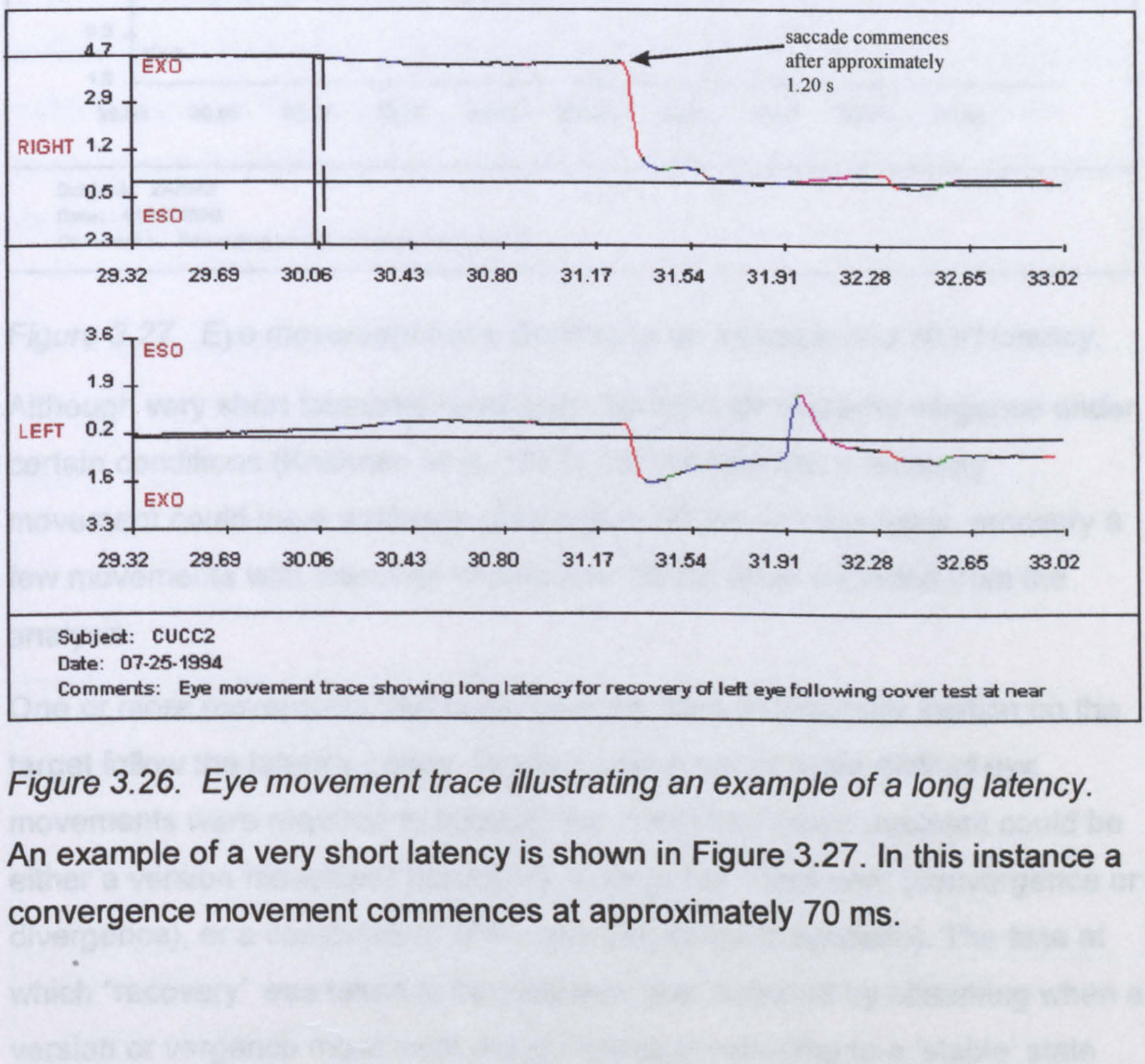


Figure 3.26. Eye movement trace illustrating an example of a long latency.

An example of a very short latency is shown in Figure 3.27. In this instance a convergence movement commences at approximately 70 ms.

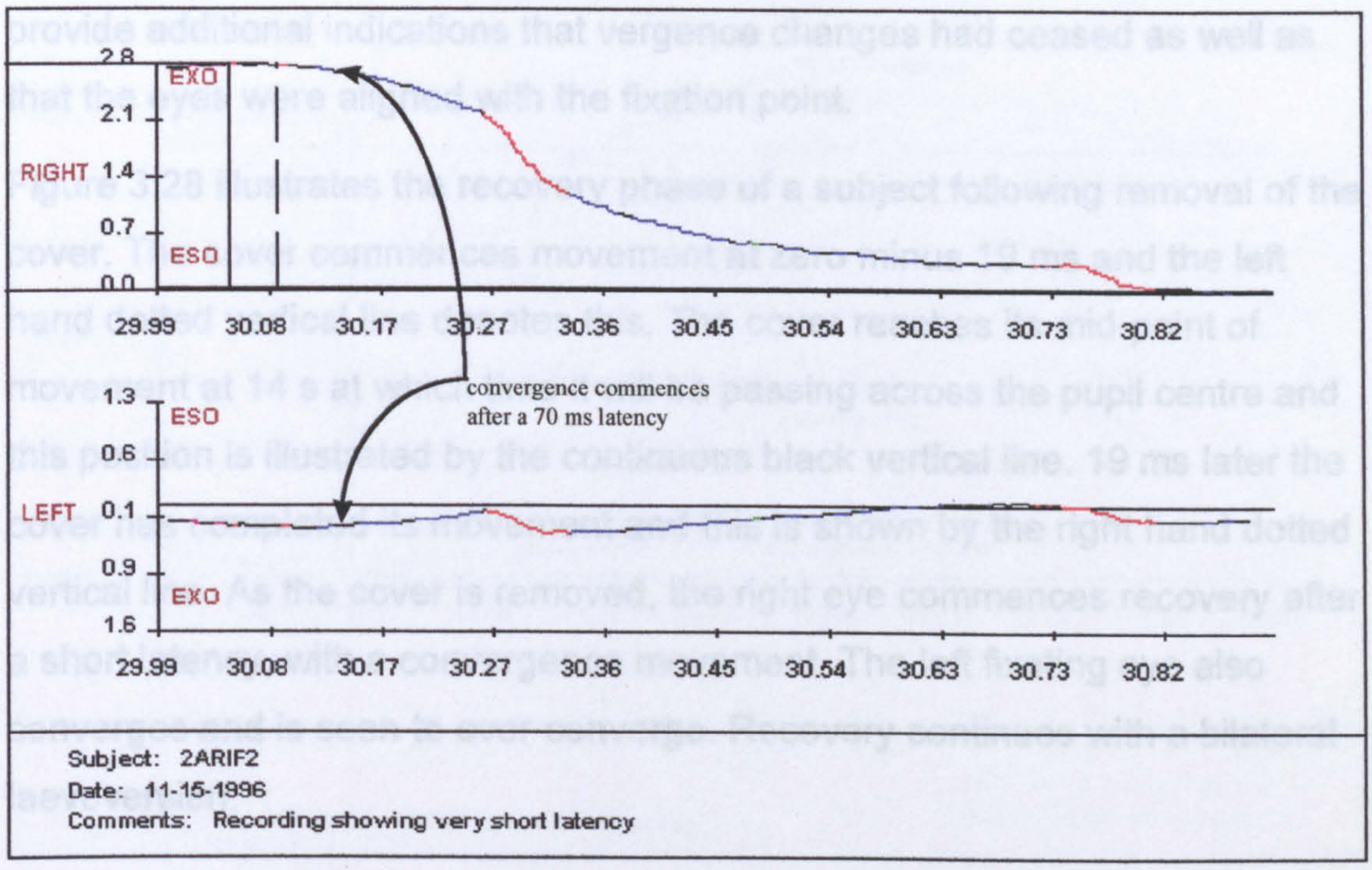


Figure 3.27. Eye movement trace illustrating an example of a short latency.

Although very short latencies have been reported for disparity vergence under certain conditions (Krishnan et al, 1973) it is unlikely that a recovery movement could have a latency of less than 60 ms. On this basis, recovery a few movements with latencies of less than 60 ms were excluded from the analysis.

One or more movements that bring the eyes back to binocular fixation on the target follow the latency period. In many cases two or more distinct eye movements were required to achieve this. The initial eye movement could be either a version movement (saccade), a vergence movement (convergence or divergence), or a combination of the two (asymmetric saccade). The time at which "recovery" was taken to be complete was obtained by observing when a version or vergence movement led to both eyes returning to a 'stable' state within $\pm 0.5°$ of their pre-occlusion positions. Some degree of interpretation was necessary because of involuntary movements that occur during normal binocular 'fixation'. Frequently the eyes overshot the fixation point and a further corrective saccade or vergence was observed. In these cases, the corrective saccades were included in the total observed recovery time. In some instances the right and left eye movement traces were subtracted to

provide additional indications that vergence changes had ceased as well as that the eyes were aligned with the fixation point.

Figure 3.28 illustrates the recovery phase of a subject following removal of the cover. The cover commences movement at zero minus 19 ms and the left hand dotted vertical line denotes this. The cover reaches its mid-point of movement at 14 s at which time it will be passing across the pupil centre and this position is illustrated by the continuous black vertical line. 19 ms later the cover has completed its movement and this is shown by the right hand dotted vertical line. As the cover is removed, the right eye commences recovery after a short latency, with a convergence movement. The left fixating eye also converges and is seen to over-converge. Recovery continues with a bilateral laevoversion.

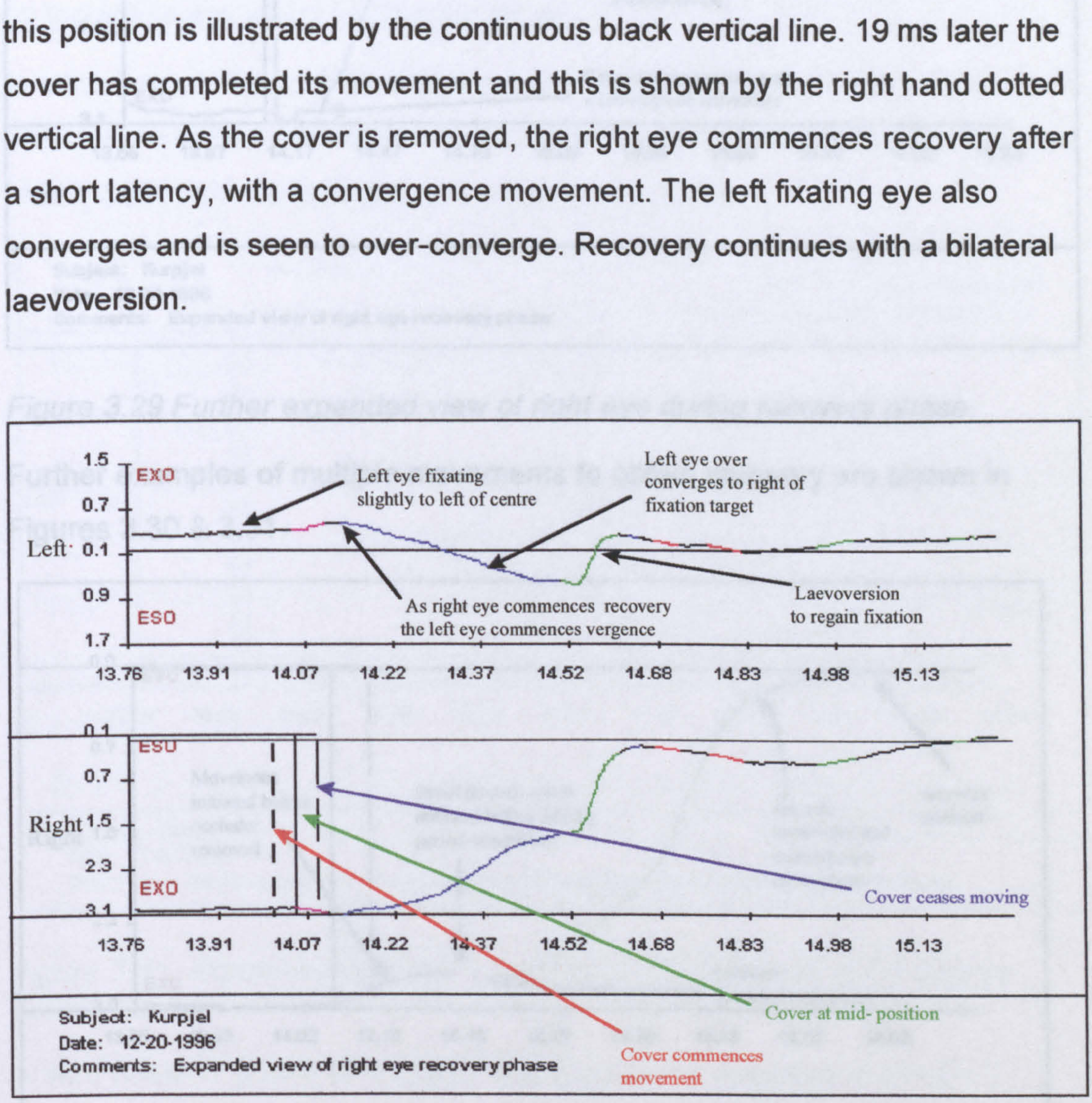


Figure 3.28. Expanded view of the recovery phase of the right eye following removal of the cover illustrating the associated movements of the fixating eye.

Figure 3.29 presents a further expanded view of the recovery phase of the same right eye and illustrates a two stage recovery to regain fixation. In this case the initiating recovery movement is a convergence which, as will be seen, is typical for near exophores. The second movement is a saccade.

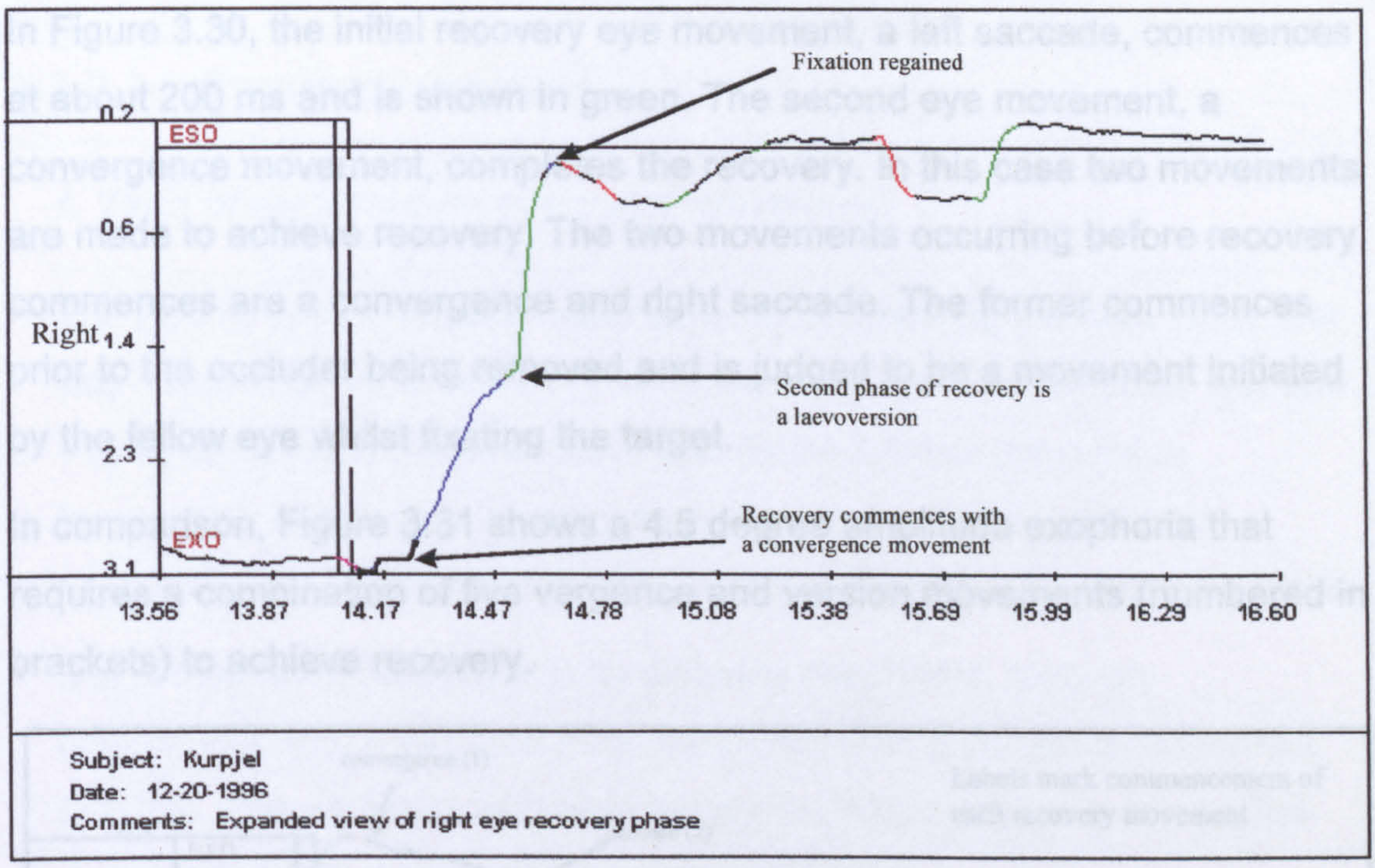


Figure 3.29 Further expanded view of right eye during recovery phase.

Further examples of multiple movements to obtain recovery are shown in Figures 3.30 & 3.31.

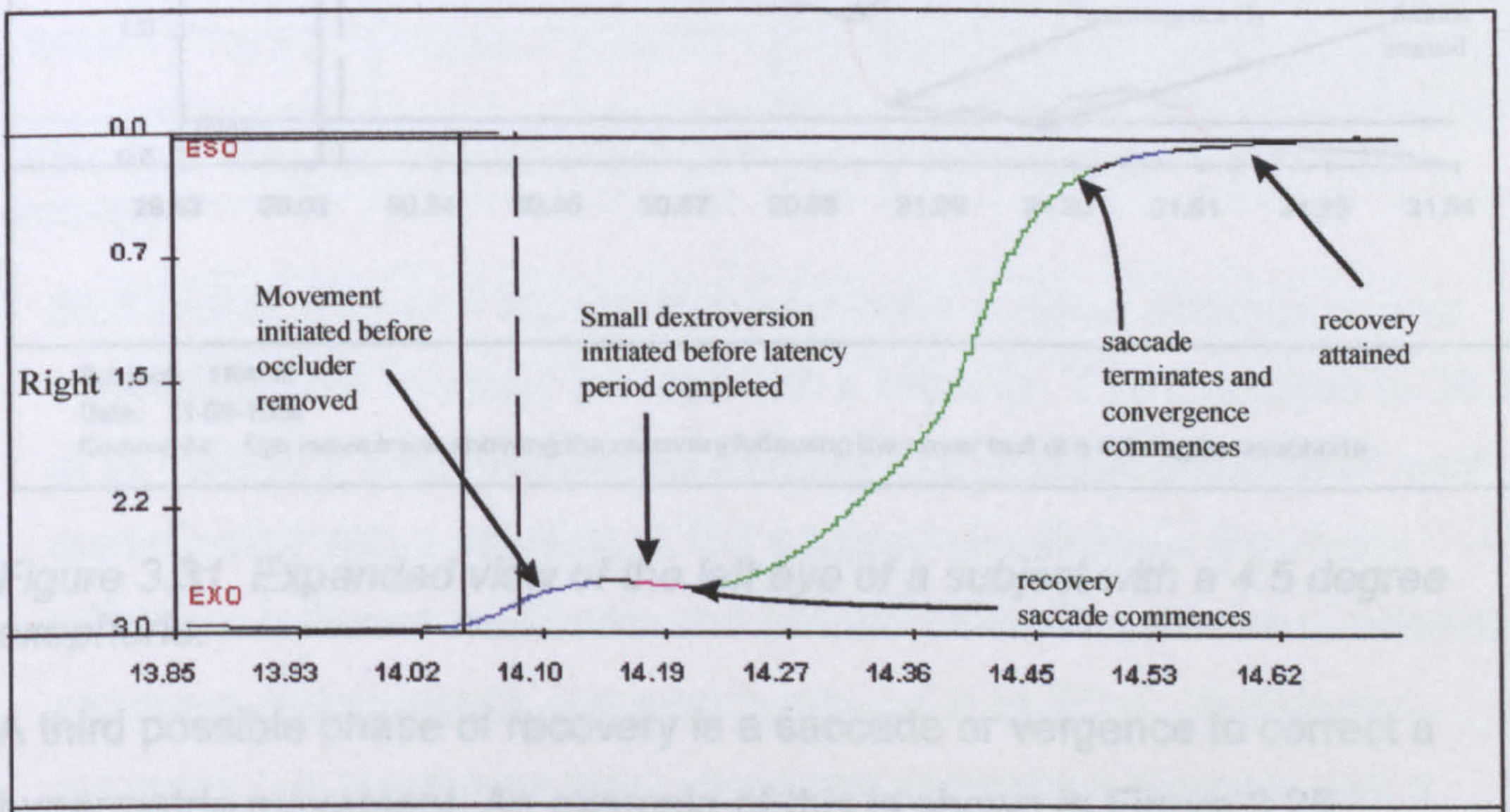


Figure 3.30. Expanded view of the right eye of a subject with 3 degrees exophoria taking two movements to achieve recovery.

In Figure 3.30, the initial recovery eye movement, a left saccade, commences at about 200 ms and is shown in green. The second eye movement, a convergence movement, completes the recovery. In this case two movements are made to achieve recovery. The two movements occurring before recovery commences are a convergence and right saccade. The former commences prior to the occluder being removed and is judged to be a movement initiated by the fellow eye whilst fixating the target.

In comparison, Figure 3.31 shows a 4.5 degree amplitude exophoria that requires a combination of five vergence and version movements (numbered in brackets) to achieve recovery.

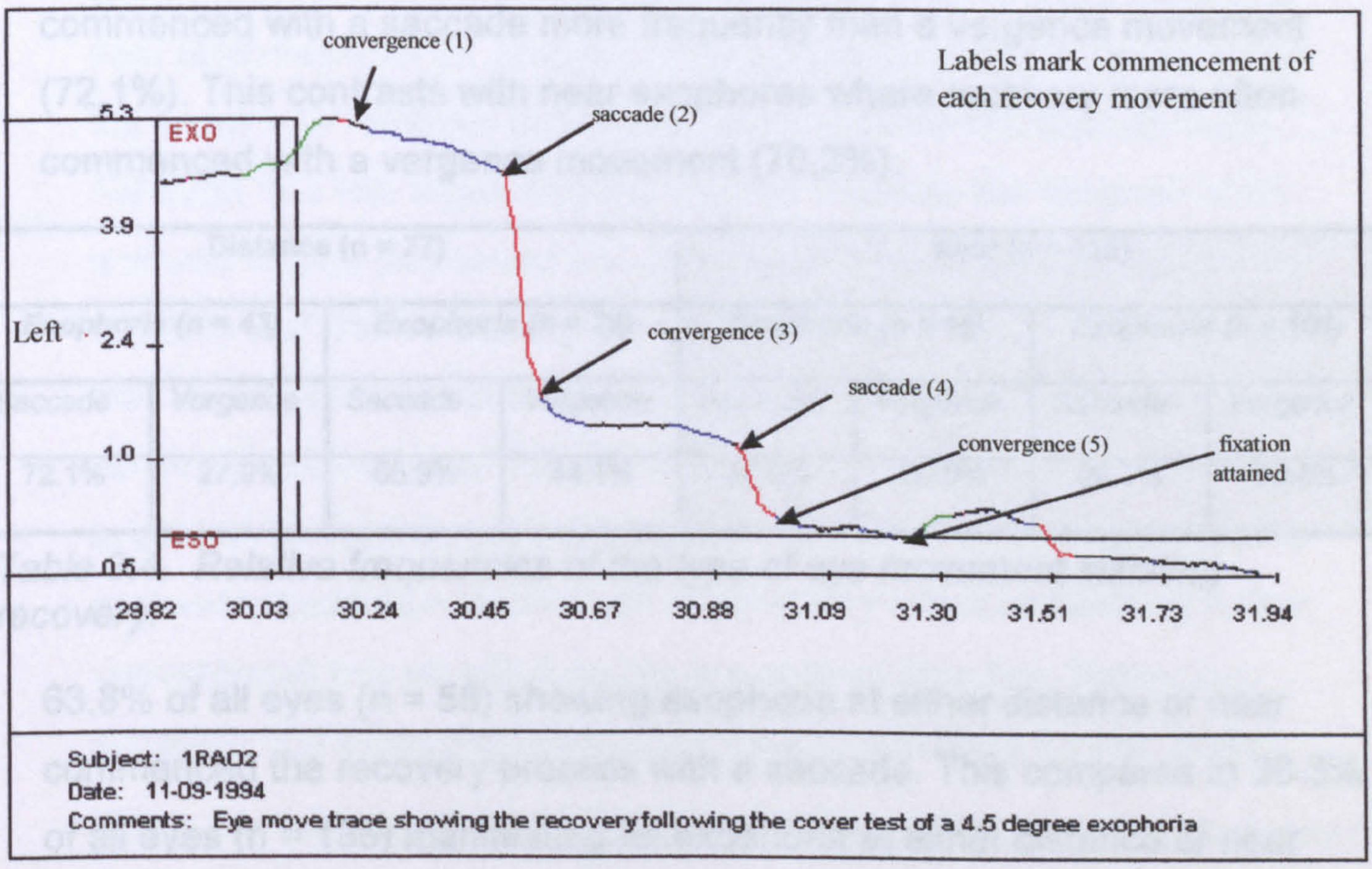


Figure 3.31. Expanded view of the left eye of a subject with a 4.5 degree exophoria.

A third possible phase of recovery is a saccade or vergence to correct a hypermetric movement. An example of this is shown in Figure 3.25.

3.2.2.b. Quantitative analysis

I. Type of initiating recovery eye movement

The type of eye movement initiating recovery following the near cover test was determined for a total of 77 eyes following the distance cover test and 116 eyes following the near cover test. Table 3.4 lists the relative frequencies of recovery initiating saccades and vergence amongst esophores and exophores following both the distance and near cover tests.

It is of interest to observe that for distance esophores, recovery commenced with a saccade more frequently than a vergence movement (72.1%). This contrasts with near exophores where recovery more often commenced with a vergence movement (70.3%).

Distance (n = 77)				**Near (n = 116)**			
Esophoria (n = 43)		***Exophoria (n = 34)***		***Esophoria (n = 15)***		***Exophoria (n = 101)***	
Saccade	*Vergence*	*Saccade*	*Vergence*	*Saccade*	*Vergence*	*Saccade*	*Vergence*
72.1%	27.9%	55.9%	44.1%	40.0%	60.0%	29.7%	70.3%

Table 3.4. Relative frequencies of the type of eye movement initiating recovery.

63.8% of all eyes (n = 58) showing esophoria at either distance or near commenced the recovery process with a saccade. This compares to 36.3% of all eyes (n = 135) manifesting an exophoria at either distance or near commencing with a saccade. A Chi squared test shows a significant difference between exophores and esophores for frequencies of initiating saccades and vergence eye movements ($\chi^2 = 24.37$; $p < 0.001$) and suggests that esophores more commonly commence recovery with a saccade and exophores with a vergence eye movement.

II. Latency

Distribution

Latency data was measured whenever possible. Missing data was common primarily due to a blink just as the cover was removed. Of the 400 potential measurements it was possible to extract latencies for 187 eyes (combined right and left) from the combined distance and near data. Of these 72 measurements related to distance and 115 to near fixation.

It is of interest to note that more measurements were possible for the near latencies than for distance. There were more blinks observed as the cover was removed during the distance cover test than for near. Although one can argue that this may be because the distance cover test was always performed first, the general impression given both by subjects and from analysing the entirety of eye movement traces, was that for some reason the subjects had more difficulty in suppressing blinks for distance and that this was not only an order effect. Hypotheses to explain this phenomenon include the possibilities that convergence and accommodation might have an inhibitory effect on blinking or that convergence has an effect on the pre-corneal tear film. The author suggests that these hypotheses are areas warranting further study.

The mean latency of the first recovery movement for all traces that could be analysed (n = 187) was 290 ms (range 70 ms to 6780 ms; SD = 580 ms).

Analysis of the results for the distance cover test only (n = 72) showed a mean latency of 260 ms (range 70 ms to 980 ms; SD = 150 ms).

For the near cover test (n = 115) the mean latency was longer than for distance (310 ms) (range 70 ms to 6780 ms; SD = 730 ms).

Figure 3.32 illustrates the frequency distribution of these latencies for distance and near fixation.

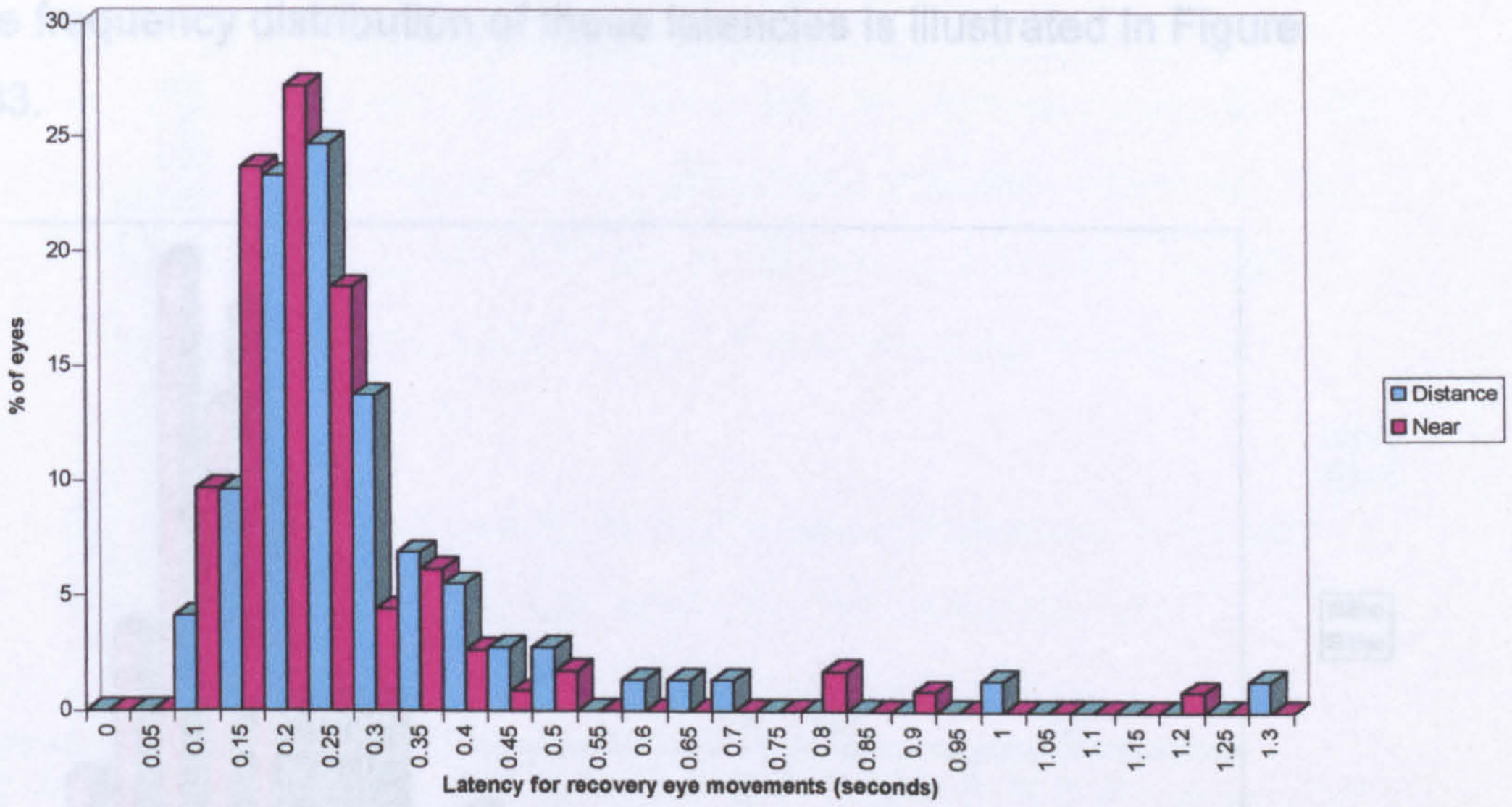


Figure 3.32. Frequency distributions (%) for latency of recovery movement following removal of the occluder for all eyes in the normal group for distance and near. Two outlying near latencies (4100 ms and 6780 ms) are omitted (see text).

As the data was not normally distributed, a Mann-Whitney U Test was applied and showed a significant difference between distance and near latencies ($p < 0.001$; difference between medians = 0.05 s; 95% confidence intervals 0.02 s to 0.07s).

It was thought that the difference between the latencies for distance and near fixation might be related to the fact that exophoria was more common for near fixation. To examine this possibility, the latencies for exophoric and esophoric recovery movements were examined separately.

Comparison of Eso and Exo deviations

Distance

The mean latency for exophoric eyes (n = 31) was 220 m s (range 70 ms to 670 ms; SD = 110 ms). The mean latency for esophoric eyes (n = 42) was 310 ms (range 70 ms to 1260 ms s; SD = 220 ms).

The frequency distribution of these latencies is illustrated in Figure 3.33.

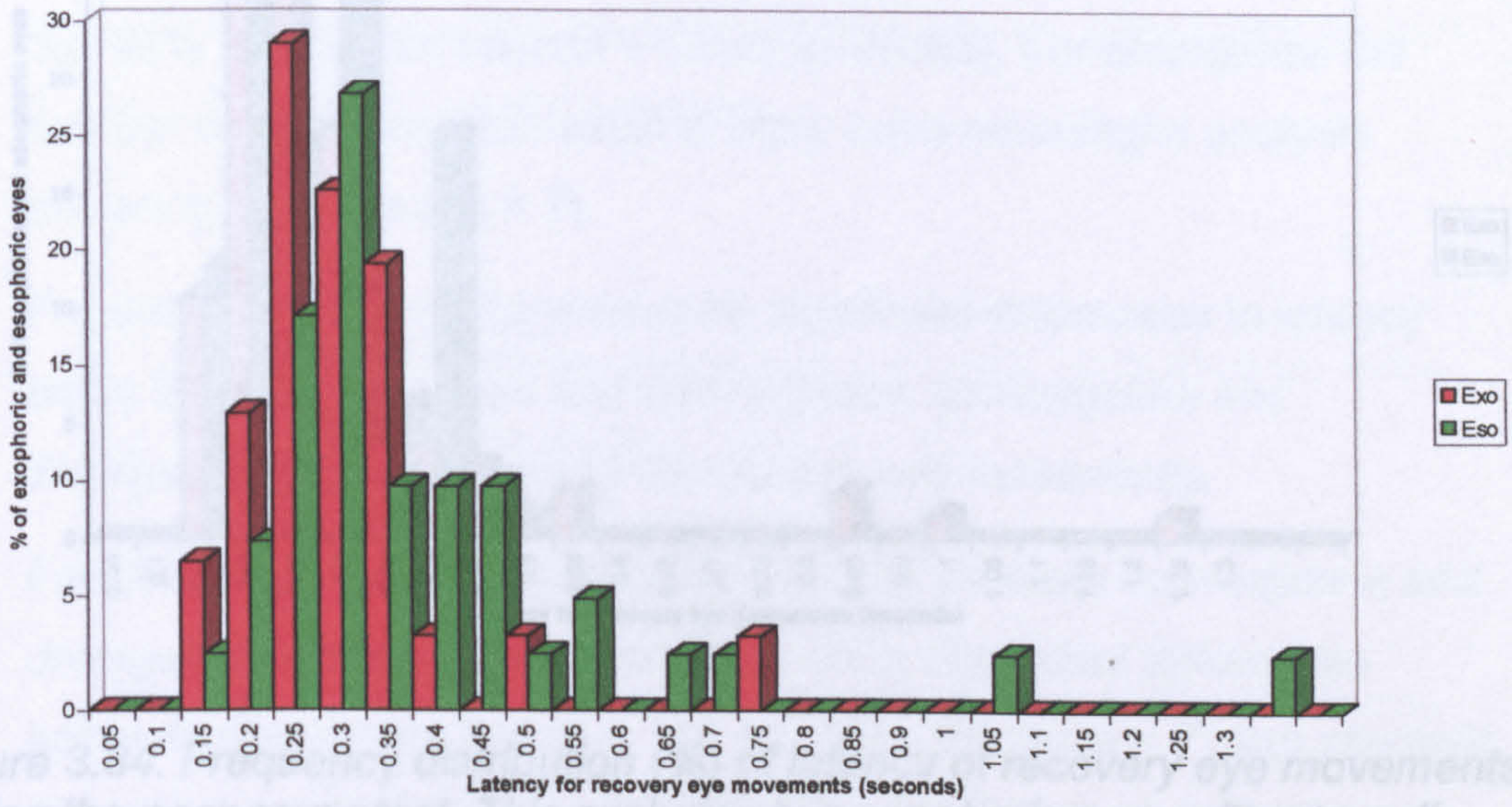


Figure 3.33. Frequency distributions (%) for latency of recovery eye movements during the distance cover test.

A Mann-Whitney U Test showed that the difference between distance exophoric and esophoric latencies was statistically significant (p = 0.01; difference between medians = 60 ms; confidence intervals = 10 ms to 110 ms).

Near

Figure 3.34 shows the frequency distribution of latency times for esophores and exophores following the near cover test.

The mean latency for recovery eye movement amongst near exophores (n = 96) was 220 ms (range 70 ms to1200 ms; SD = 170 ms).

The mean latency of the recovery movement for near esophores (n = 19) was 0.747 s (range 0.08 s to 6780 s; SD = 1.72 s). The median was 220 ms. Two eyes showed a very long latency before recovery, namely 4100 ms and 6780 ms. If the two long latencies are discarded the mean of the modified group is 200 ms (range 80 ms to 340 ms; SD = 80 ms).

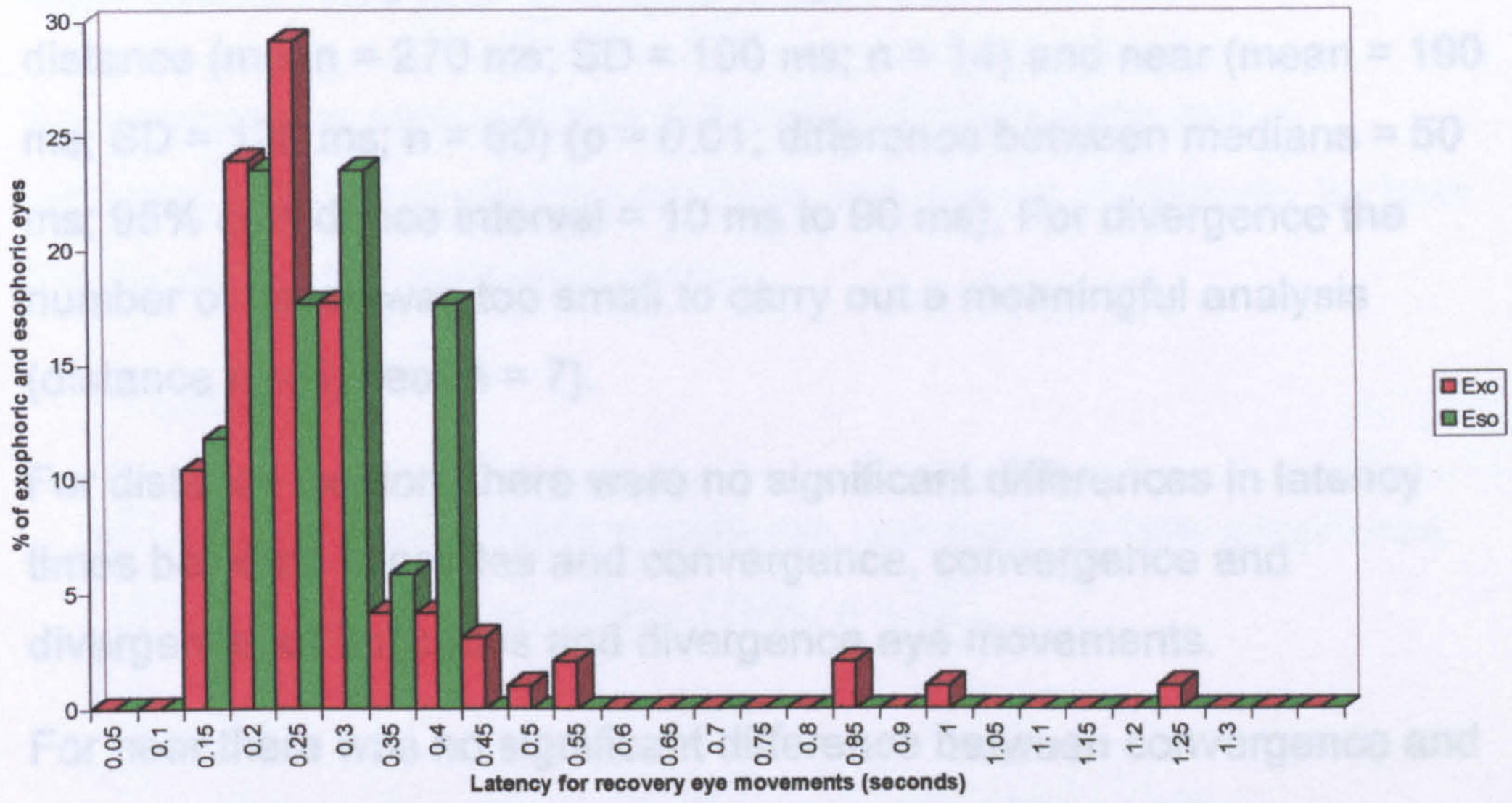


Figure 3.34. Frequency distribution (%) of latency of recovery eye movements during the near cover test. This excludes two esophoric eyes with saccadic latencies of 6780 ms and 4100 ms.

For near latencies, a Mann-Whitney U Test showed no significant difference between exophoric and esophoric latencies.

A Mann-Whitney U Test was carried out to compare latencies for all exophores (distance and near) compared to all esophores. The two outlying esophores were excluded from the analysis. There was a significant difference between groups ($p = 0.002$; difference between medians = 50 ms; 95% confidence intervals = - 20 ms to 80 ms).

Comparison of saccadic and vergence laten cies

A further analysis was carried out to compare latency times of initiating vergences and saccades. Some data was excluded because of difficulties in categorising the type of eye movement initiating recovery. The two esophoric eyes with saccadic latencies of 6780 ms and 4100 s were also excluded.

Distance and near eye movement latencies were compared for version, convergence, and divergence eye movements. Each group showed a non-normal distribution. A Mann-Whitney U Tests showed no significant difference between distance (n = 45) and near (n = 33) saccadic latency

times. For convergence there was a significant difference between distance (mean = 270 ms; SD = 190 ms; n = 14) and near (mean = 190 ms; SD = 120 ms; n = 60) (p = 0.01; difference between medians = 50 ms; 95% confidence interval = 10 ms to 90 ms). For divergence the number of cases was too small to carry out a meaningful analysis (distance n = 5; near n = 7).

For distance fixation, there were no significant differences in latency times between saccades and convergence, convergence and divergence, or saccades and divergence eye movements.

For near there was no significant difference between convergence and divergence latencies. However, there were significant differences between divergence (mean = 160 ms; SD = 50 ms; n = 7) and saccadic latencies (mean = 270 ms; SD = 140 ms; n = 30)(p = 0.01; difference between medians = - 100 ms; 95% confidence interval = -190 ms to -30 ms), and between convergence and saccadic latencies (p < 0.001; difference between medians = 100 ms; 95% confidence limit = 50 ms to 140 ms).

Comparison of latencies of adducting and abducting saccades

There was no significant difference between distance and near latencies for either adducting or abducting saccades.

The mean latency time for adducting saccades was 260 ms (SD = 120 ms) compared to 350 ms (SD = 270 ms) for abducting saccades. Ranges were 90 ms to 670 ms for adduction and 80 ms to 1260 ms for abduction. A comparison of latency times for all eyes (combined distance and near) showed a difference between adducting saccades (n = 35) and abducting saccades (n = 43) (Mann-Whitney U Test p = 0.09; difference between medians = 40 ms; 95% confidence interval = -100 ms to 10 ms).

Correlation with phoria amplitude

An analysis was carried out to determine whether there was a relationship between phoria amplitude and latency of recovery for both the esophoric and exophoric groups.

Distance

R^2 for latency with phoria amplitudes was poor for both exophores (18%; n = 32) and esophores (18%; n = 42).

Near

R^2 for latency with phoria amplitudes was poor for both exophores (13%; n = 96) and esophores (21%; n = 19).

As might be expected, these results suggest there is no strong relationship between phoria amplitude, for either esophoria or exophoria, and latency for recovery following both the distance and near cover test for normal subjects.

III. Number of recovery movements

Distribution

For distance fixation (n = 69 eyes) the median number of distinct eye movements made to achieve recovery in the normal group (exophores and esophores), including those made to correct for an overshoot (hypermetric vergence or saccade), was 2 with a range of 1 to 5. The mean was 1.7 (SD = 0.8) This compares with a median of 2 movements following the near cover test. The range for near was from 1 to 8 with a mean of 2.8 (SD = 1.0; n = 115 eyes). These distributions are illustrated in Figure 3.35.

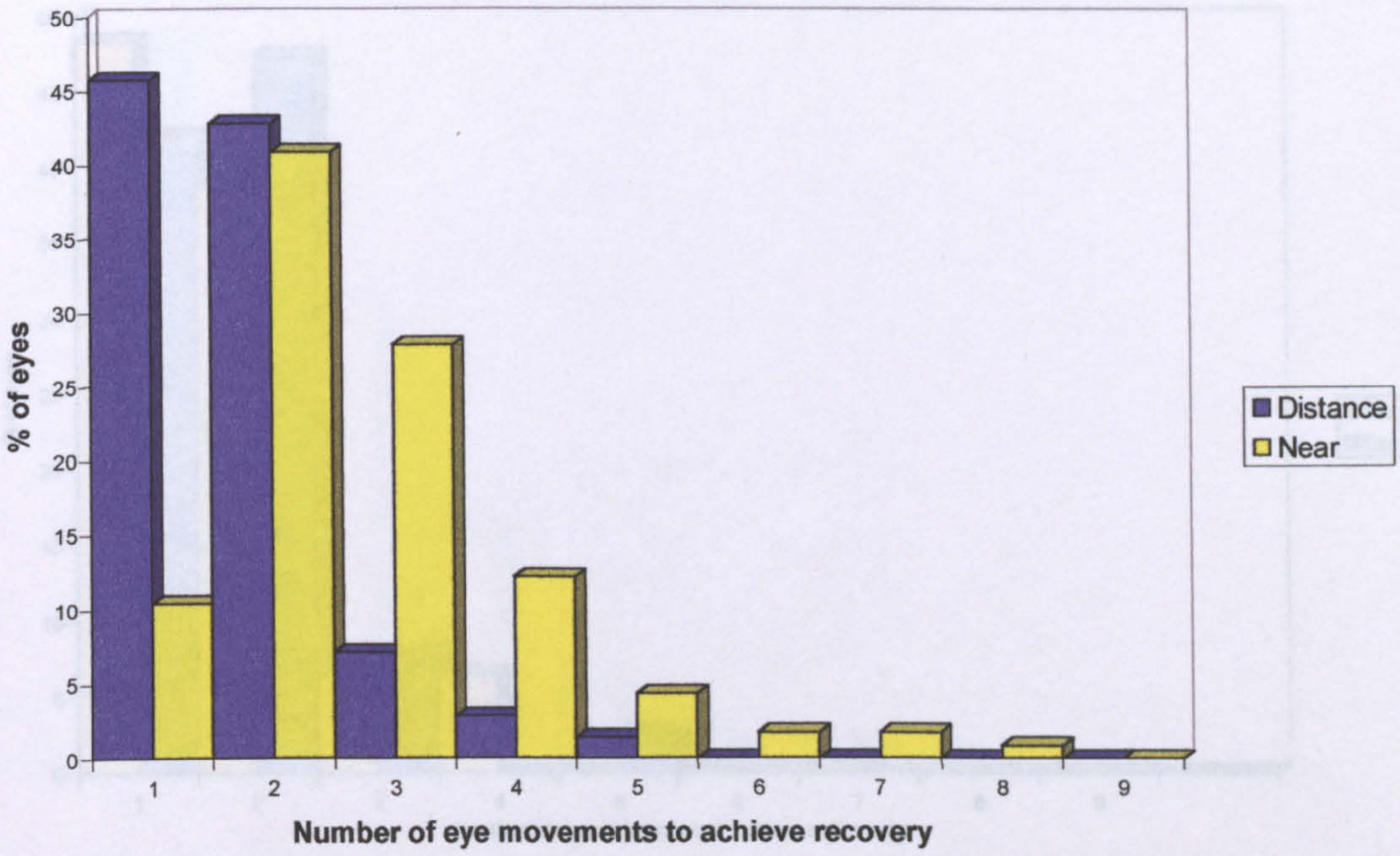


Figure 3.35. Frequency distributions of number of eye movements to achieve recovery following the automated cover test for distance and near fixation.

A Mann-Whitney U Test showed a significant difference between distance and near fixation for the number of eye movements made to achieve recovery ($p < 0.001$; 95% confidence interval = 1.00 to 1.00).

Comparison of Eso and Exo deviations

To determine whether exophores and esophores differed in the number of recovery eye movements made to achieve recovery, a comparison of the two groups was carried out for distance and near.

Distance

For distance exophores (n = 31 eyes), the median number of distinct eye movements made to achieve recovery in the normal group was 2, with a range of 1 to 4. The mean was 1.7 (SD = 0.86). For distance esophores (n = 38), the median was 2, range 1 to 5, and mean 1.7 (SD = 0.83). These distributions are illustrated in Figure 3.36.

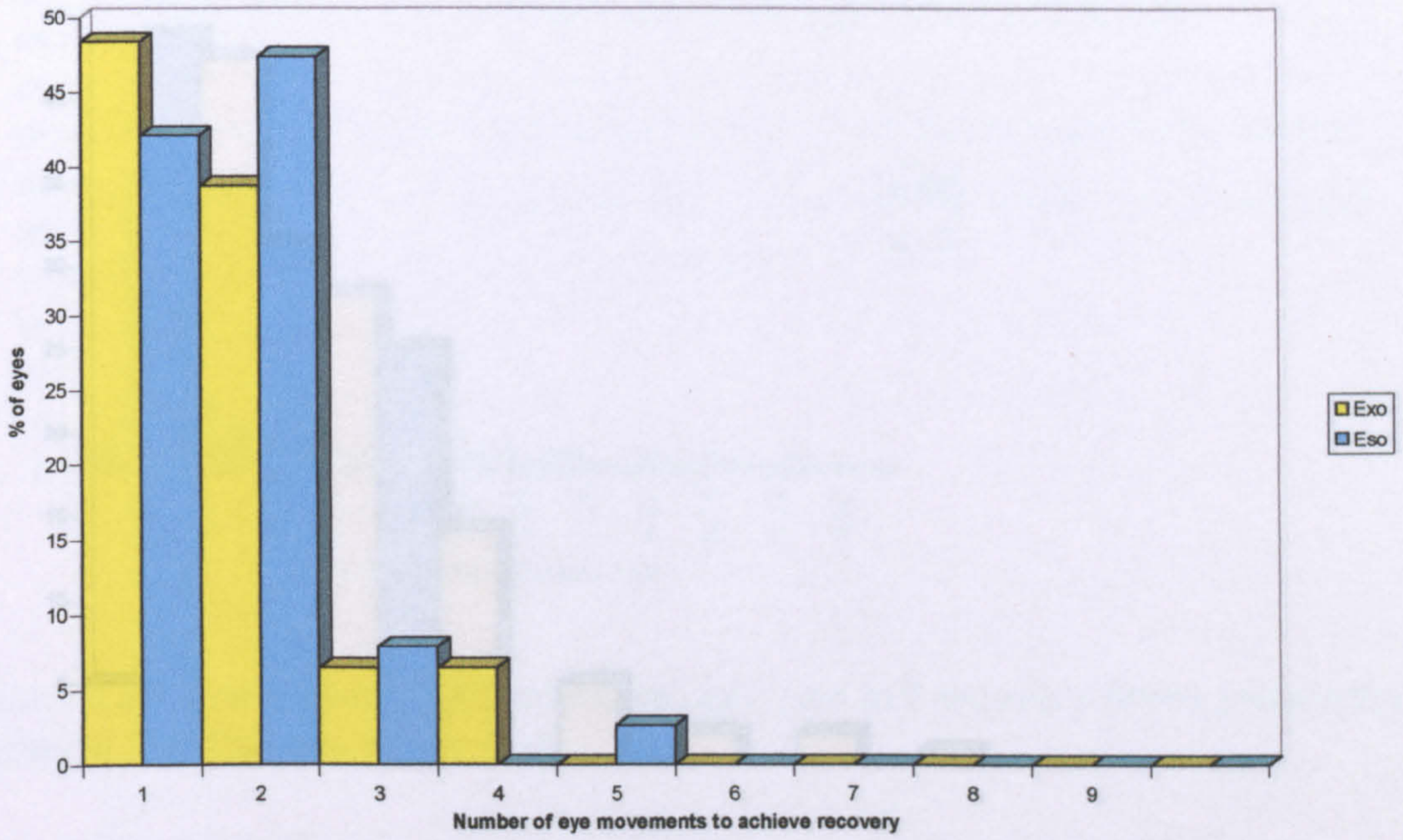


Figure 3.36. Frequency distributions of number of eye movements to achieve recovery following the distance automated cover test for exophores and esophores.

There was no statistical difference between the number of eye movements to achieve recovery for exophores and esophores (Mann-Whitney U Test p = 0.75).

Near

For near exophores (n = 99 eyes), the median number of distinct eye movements made to achieve recovery in the normal group was 3, with a range of 1 to 8. The mean was 2.9 (SD = 1.32). For near esophores (n = 16), the median was 2, range 1 to 3, and mean 1.8 (SD = 0.8). These distributions are illustrated in Figure 3.37.

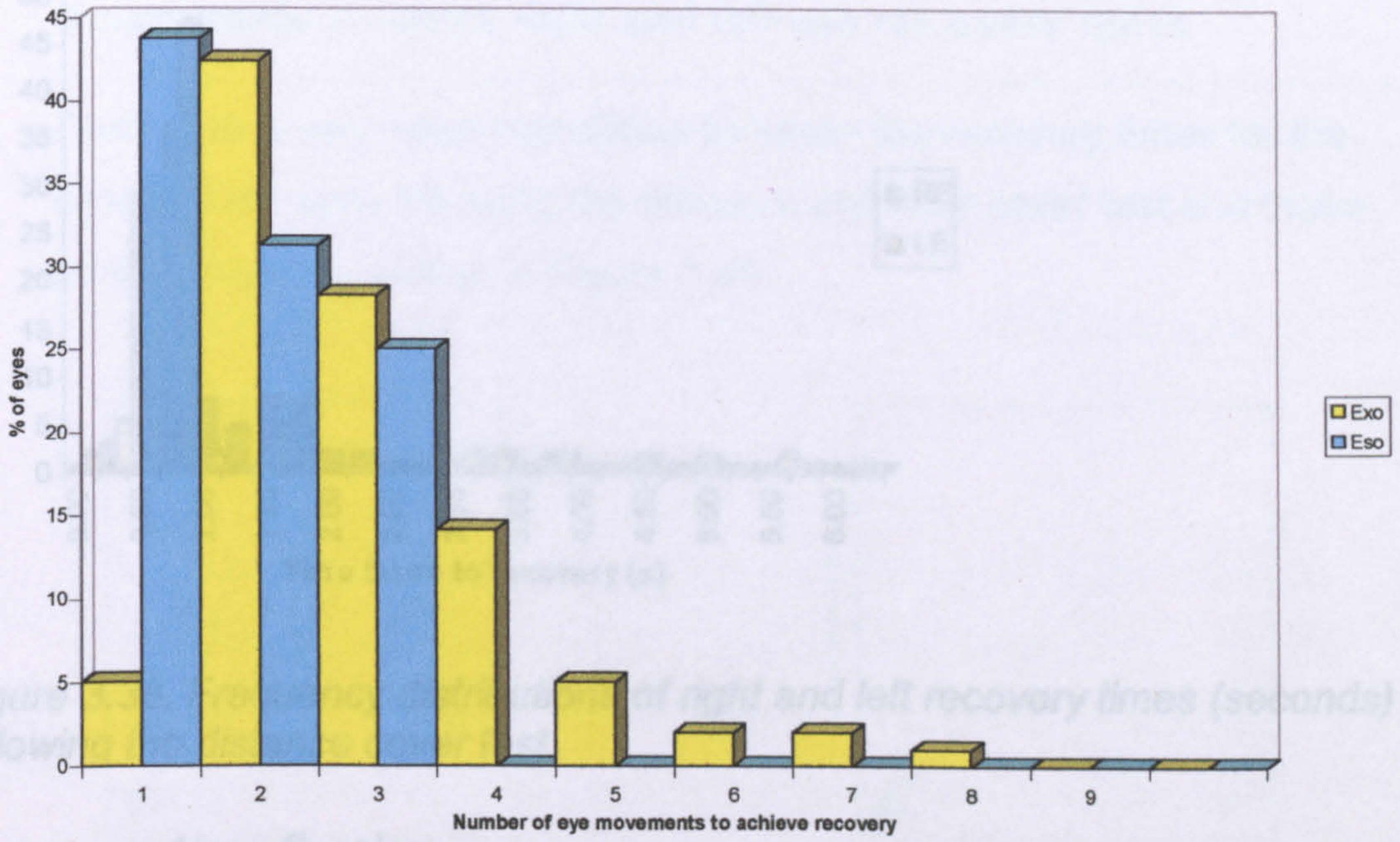


Figure 3.37. Frequency distributions of number of eye movements to achieve recovery following the near automated cover test for exophores and esophores.

For the near cover test, a Mann-Whitney U Test showed a significant statistical difference for the number of eye movements to achieve recovery between exophores and esophores (p = 0.001; 95% confidence interval = 0.00 to 1.00) (exo > eso).

IV. Recovery time

Distribution

Figures 3.39 and 3.40 illustrate the frequency distributions of recovery times for right and left eyes for distance and near fixation respectively.

Distance fixation

The mean recovery time for all eyes (n = 131) following the distance cover test was 860 ms (SD = 880 ms). The minimum time for recovery was 150 ms and the maximum was 5.41 s.

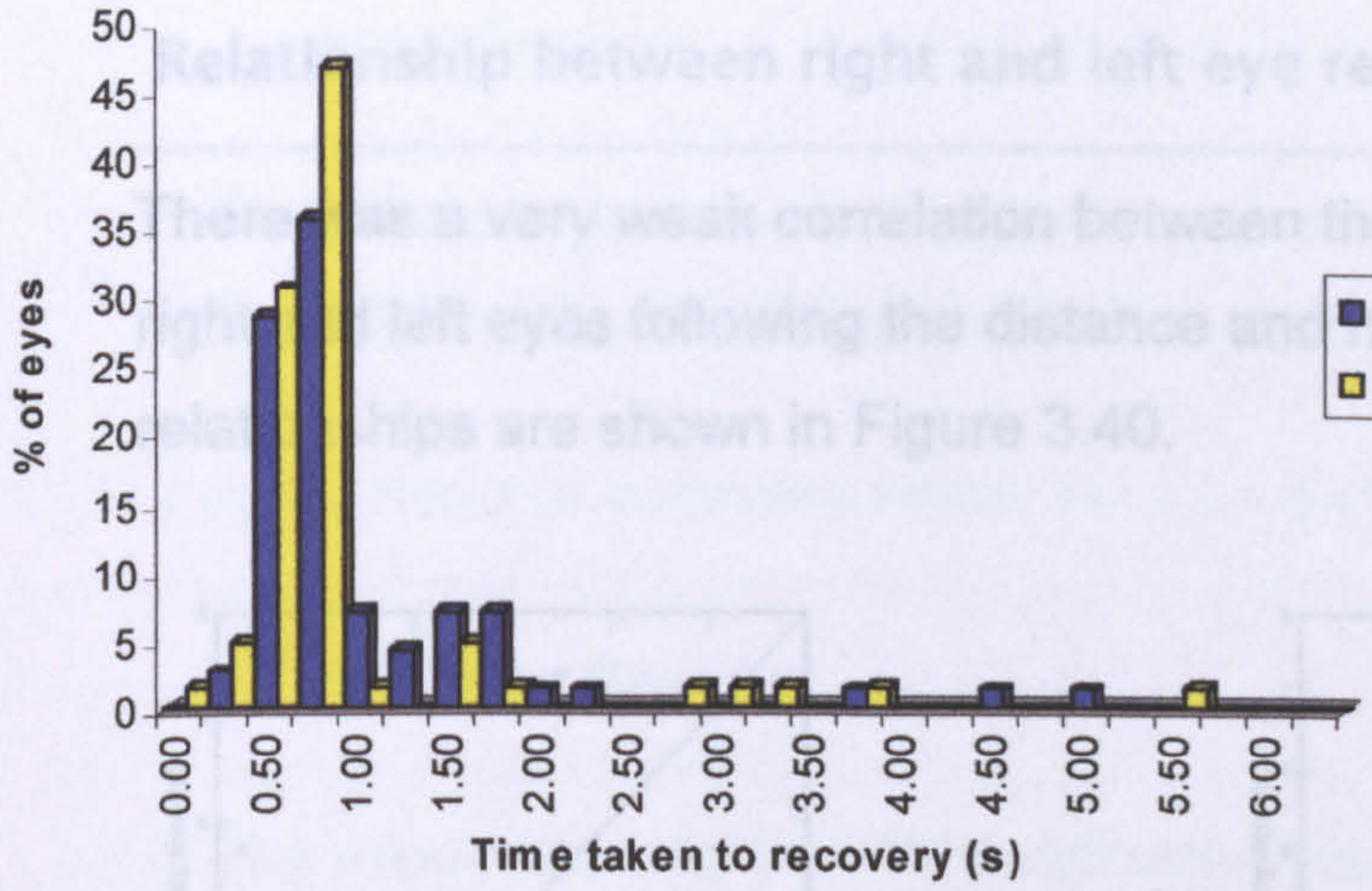


Figure 3.38. Frequency distributions of right and left recovery times (seconds) following the distance cover test.

Near fixation

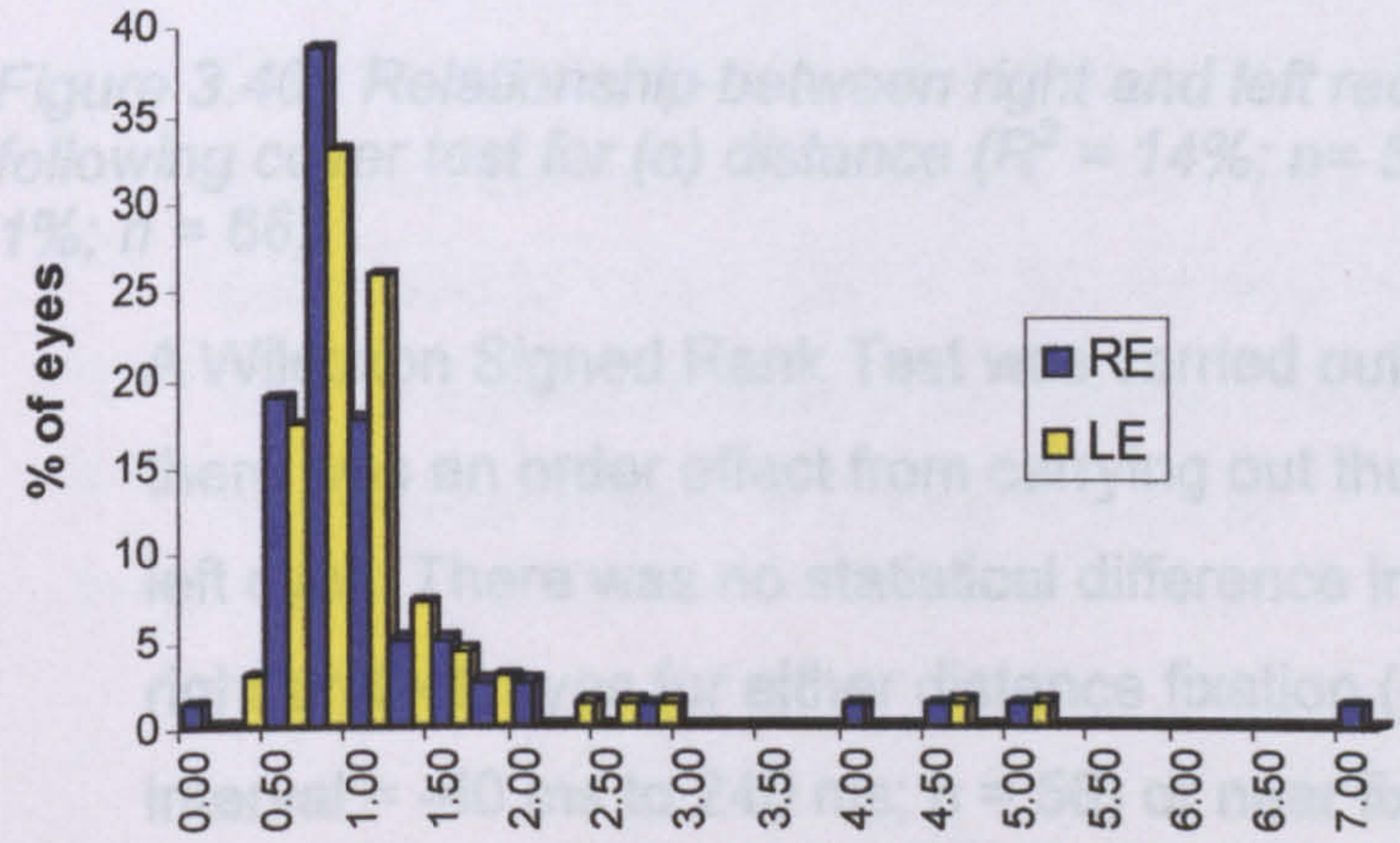


Figure 3.39. Frequency distributions of right and left recovery times (seconds) following the near cover tests in the normal group

The mean recovery time for all eyes (n = 149) following the near cover test was 1.05 s (SD = 1.21). The minimum time for recovery was 160 ms and the maximum was 7.75 s.

Relationship between right and left eye rec overy times

There was a very weak correlation between the recovery times for the right and left eyes following the distance and near cover test and these relationships are shown in Figure 3.40.

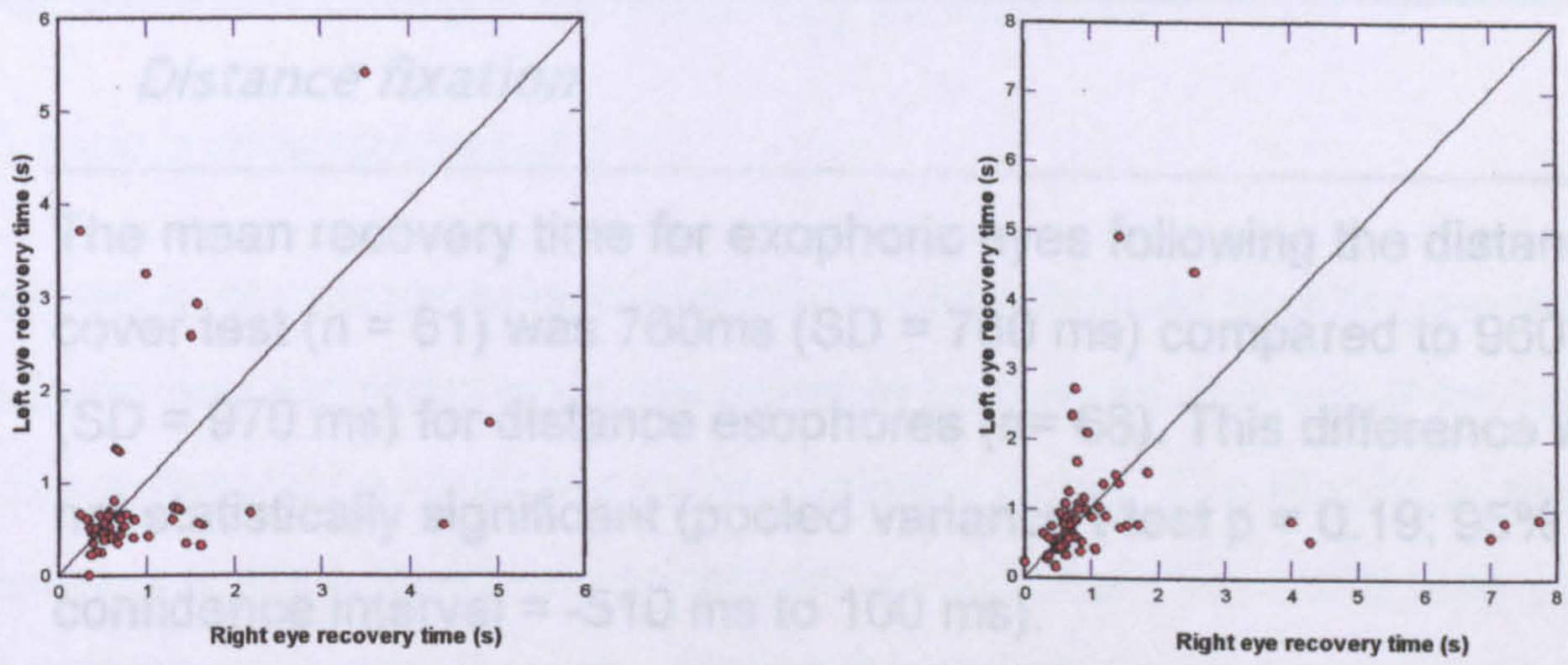


Figure 3.40. Relationship between right and left recovery times (seconds) following cover test for (a) distance (R^2 = 14%; n= 56) and (b) near (R^2 = < 1%; n = 66).

A Wilcoxon Signed Rank Test was carried out to test the hypothesis that there was an order effect from carrying out the cover test on right before left eyes. There was no statistical difference in recovery time between right and left eyes for either distance fixation (p = 0.19; 95% confidence interval = -40 ms to 240 ms; n = 56) or near fixation (p = 0.95; 95% confidence interval = -100 ms s to 100 ms; n = 66).

Relationship between recovery time and ph oria amplitude

Distance fixation

For the distance cover test there was a very weak relationship between amplitude of phoria and time taken for recovery for both exophoric eyes (Kendall Rank R^2 = 1%; n = 61) and esophoric eyes (R^2 = 3%; n = 68).

Near fixation

The relationship was also weak for near exophoric eyes (Kendall Rank R^2 = 6%; n = 124) and near esophoric eyes (R^2 = 26%; n = 24).

Comparison of recovery times for Eso and Exo deviations

Distance fixation

The mean recovery time for exophoric eyes following the distance cover test (n = 61) was 760ms (SD = 760 ms) compared to 960 ms (SD = 970 ms) for distance esophores (n= 68). This difference was not statistically significant (pooled variance t-test p = 0.19; 95% confidence interval = -510 ms to 100 ms).

Near fixation

The mean recovery time for recovery time for exophoric eyes following the near cover test = 1.04 s (SD = 1.13) (n = 124) compared to 1.05 s (SD = 1.55) for near esophores (n = 24). There was no significant difference between the two groups (p = 0.98; 95% confidence interval = -0.54 s to 0.53 s).

3.2.2.c. Summary of results

There are a number of findings from this study of 100 "normal" subjects that will be of interest to clinicians. Some of these findings confirm what practitioners have intuitively believed for many years. Other findings suggest that a modification to long held views may be necessary.

I. A single measurement ?

Most eye care practitioners in clinical practice assume that the amplitude of phoria for any patient is normally equal in both eyes. It is usual for just one measurement to be recorded in the patient's notes. The results of this study suggest that it is indeed reasonable for practitioners to record their phoria estimations on "normal" young adult subjects making the assumption that the measure is similar for both right and left eyes.

II. Phoria distribution

For emmetropes aged 18 to 35 years, exophoria is more common than esophoria for near fixation. It is not uncommon to find a small esophoria in the distance and exophoria at near. It is uncommon to find an exophoria in the distance and an esophoria at near.

III. Period of occlusion

In general, the difference between the phoria amplitude measured after 2 s occlusion and 10 s occlusion increases with the phoria amplitude. However, larger amplitude phorias may not necessarily take longer to reach a position of equilibrium during dissociation when compared to smaller phorias. In addition, for any one subject, one eye may show a different fusional decay profile to its fellow.

The evidence that there are significant differences between the 2s and 10s phoria amplitudes for individuals in the "normal group" and also that the mean times to reach the 10 second amplitude are greater than 2 seconds, suggests that, in many cases, the eye had not reached a state of

equilibrium at 2 s. This is of clinical importance because the usual period of occlusion used during the conventional cover test is approximately 2 s. Some eyes take 10 seconds or more to reach the maximum amplitude. It may be concluded that a longer period of occlusion than the commonly used 2 seconds is necessary to allow for the covered eye to reach equilibrium for all magnitudes of phoria.

IV. Type of recovery eye movement

Esophores more commonly commence recovery with a saccade and exophores with a vergence eye movement. The difference between groups was significant (p = 0.006).

V. Latency for recovery

There was a significant statistical difference between latencies for recovery for distance and near fixation (near > distance) (p < 0.001; difference between medians = 50 ms; confidence intervals 20 ms to 70 ms).

There was a statistically significant difference in latencies between exophores and esophores for distance fixation (esophores > exophores) (p = 0.01). There was no significant difference between exophores and esophores for near.

There was also a significant difference between distance and near convergence eye movement latency periods with the convergence latencies being shorter at near (p = 0.01). However, there was no significant difference between distance and near saccadic latencies.

For near, both divergence and convergence latencies were statistically significantly shorter than saccadic latencies (p = 0.01 and p < 0.001 respectively).

There was a difference in latency periods between adducting saccades and abducting saccades (abducting > adducting) (p = 0.09).

VI. Number of eye movements to achieve recovery

There was a significant difference between distance and near fixation for the number of eye movements to achieve recovery (near > distance) ($p < 0.001$).

For near fixation, there was a significant statistical difference between the number of eye movements to achieve recovery for exophores and esophores (exophores > esophores) ($p = 0.001$).

VII. Recovery times

There was not a strong association between phoria amplitude and recovery time. This is because the total recovery time is a combination of latency time, the number of distinct movements made to achieve recovery, and the type of each individual movement (i.e. vergence or saccade).

There was a very weak correlation between the recovery times for the right and left eyes following the distance and near cover test. However, a paired t-test did not show a statistical difference in recovery time between right and left eyes for either distance fixation ($p = 0.45$) or near fixation ($p = 0.20$).

VIII. Recovery times and number of eye movements

This study has shown for the first time, for a large 'normal' population, the nature of eye movements of the occluded eye once the cover is removed.

One of the anecdotal, subjective observations sometimes reported by practitioners is a "slow and jerky" recovery following the cover test.

The results of this study help provide explanations for some of these anecdotal reports. Although recovery times were likely to have been affected by the latency of the initial movement and also by the type of eye movement (saccade or vergence), they were also a function of the number of movements required to achieve refixation and this ranged from 1 to 8 movements. For the first time, the nature of a 'jerky' recovery has been demonstrated.

Chapter 4. The relationship between the resultsof a range of clinical binocular vision tests and eye movement characteristics during the cover test

4.1 Introduction

As well as the automated cover test for distance and near, previously discussed in Chapter 3, 39 consecutive subjects of the "normal" group underwent an additional battery of ocular motor investigations. The purpose of this was to identify any relationships between the results of those tests and eye movement characteristics during the automated cover test.

Although analysis using multilinear regression would have been the ideal, this was not feasible because of the limited number of subjects in this study and the large number of co-variables.

Although these subjects were included in this study on the basis that they did not suffer from symptoms during near work, they were nevertheless invited to complete a questionnaire designed to determine the profile of any discomfort they believed they suffered during close work. The questionnaire was based on the design of Feldman et al (1992) and is shown in Appendix 2. Feldman and Cooper (1998) have demonstrated the reliability and validity of a similar asthenopia questionnaire to the one used in this study.

The relationships of symptom score and eye movement characteristics during the cover test are discussed in Chapter 6.

4.2 Method

Each subject was invited to complete the symptom questionnaire. The questionnaire consisted of 7 questions relating to various symptoms. The subject responded by choosing one of a choice of 5 answers relating to severity of that symptom. This enabled the author to grade each symptom from 0 to 4 in order of severity, and produce a total "symptom score" for each subject.

After completing the questionnaire a further battery of tests was carried out by the author in the following order:

- **Associated phoria (Mallett fixation disparity test) for distance and near**

  The Mallett fixation test (Mallett, 1964; Mallett, 1966) has been alluded to in Chapter 1. This test is designed to detect fixation disparity and may be used for both distance and near and has been described by Evans (1997). In both tests, there is a central fixation target, the word OXO, seen with both eyes, and two cross-polarised monocular markers known as Nonius strips positioned in line with the X, one seen with each eye. Dissociation of the monocular markers is obtained by the use of cross-polarised filters worn by the patient. The background surrounding the markers and OXO is seen binocularly which aids 'binocular lock'. The test is designed to detect a fixation disparity which appears to the patient as a displacement of one or both markers from their alignment with X. Prisms are then introduced by the practitioner to re-align the marker. The amplitude of the prism was a measure of the associated phoria.

- **Automated cover test routine for distance and near**

  This has been described in Chapter 2.

- **Near fusional reserves using variable prisms**

  A variable prism stereoscope was employed to gradually increase the power, at a rate of approximatel 1^{Δ}/s, from zero, of a pair of Risley prisms

through which the subject viewed a vertical line target. The patient was requested to state at which point the target first appeared blurred *(blur point)* and then when it was first seen in diplopia *(break point)*. At that point the power of the prism was gradually decreased until the target was again seen singly *(recovery)*. For each subject, negative fusional reserves (base in) were measured before positive fusional reserves (base out).

- **Near point of convergence**

  The *near point of convergence (NPC)* for each subject was investigated using the *push-up test* with a RAF rule. The procedure has previously been described in Chapter 1.

- **Maddox rod**

  This test for measuring the amplitude of phoria for distance with the Maddox rod test has been discussed in Chapter 1. A standard red Maddox rod was employed.

- **Maddox wing**

  This test for measuring the amplitude of phoria for near with the Maddox wing has been discussed in Chapter 1. In those cases where subjects reported instability of the position of the arrow, a cover was placed before the left eye for approximately 2 seconds and the subject was asked to report the position of the arrow immediately on removal of the occluder.

- **Conventional cover test for distance and near**

  The procedure for the cover test has been discussed in Chapter 1. Each eye was occluded for approximately 2 seconds for both the distance and near cover test. The testing distances were 600 cm and approximately 40 cm for distance and near respectively. It should be noted that this compares to distances of 340 cm and 40 cm used during the automated cover test.

- **AC/A ratio (gradient test)**

  The gradient test was used to assess the change in phoria amplitude with change in accommodation. The method used employed a Maddox wing.

The amplitude of phoria was recorded and the measurement repeated with a + 2 D lens before each eye. It should be noted that changes in accommodation cannot be controlled precisely with lenses. However, the test is employed in clinical practice and the test was included for completeness.

- **Amplitudes of accommodation**

Measurement of right and left amplitudes of accommodation were made using the *Donders* or *push up* technique (Reading, 1988). Each eye was tested monocularly. The subject was instructed to view the N5 print of a RAF rule target. The target was moved, at a rate of approximately 1D/s, from about 30 cm towards the subject who was instructed to try and keep the print clear and to report when the print first appeared to blur. The point at which blur was first reported was noted and the target pushed a little closer towards the subject and then slowly retracted until the subject reported the print clear again. The reciprocal of the latter distance in metres was recorded as the amplitude of accommodation.

- **TNO stereotest**

The TNO test for stereoscopic vision was used to assess the stereo-acuity of each subject. This test consists of stereograms in which the half images have been superimposed and printed in roughly complementary colours. These anaglyphs are viewed through filters that transmit mainly either one or other of the printed colours and these images evoke the perception of an image in depth (Walraven, 1975). The test plates were well illuminated and the testing distance employed was approximately 40 cm. This test enables stereo-acuity to be graded from 480" to 15".

- **Near binocularity (Mallett suppression test)**

The Mallett test for foveal suppression (or 'binocular status') presents rows of progressively smaller letters. In each row, the central, or central pair of letters is seen by both eyes, providing binocular lock (Evans, 1997). The letters to the left and right of the central targets are cross-polarised. The targets are viewed through cross-polarised filters and if there is

suppression some of the letters will not be read by the patient. The letters subtend 14', 10', 7' and 5' of arc.

4.3 Results

Quantitative data will be presented to describe the results of the tests listed above. Eye movement characteristics during the automated cover test of this group will not be described independently in this chapter because these results formed part of the results described in the previous chapter. However, the eye movement recording data will be used to explore relationships with the results of some of the other tests carried out.

4.3.1 Symptoms

One of the criteria for recruitment to the study was that the subjects did not consider themselves symptomatic during near vision tasks. However, in common with a group of symptomatic patients, who will be discussed in Chapters 5 and 6, this group was invited to complete a symptom questionnaire (see Appendix 2). For each question, a maximum of '4' points was allocated for a response to choice number 5 and a minmum of '0' for a response to choice number 1. A total number of seven questions produced a maximum possible score of '28' points.

It is of interest to observe that although these subjects did not consider themselves to be symptomatic, the questionnaire revealed that symptoms were common as may be seen from Figure 4.1.

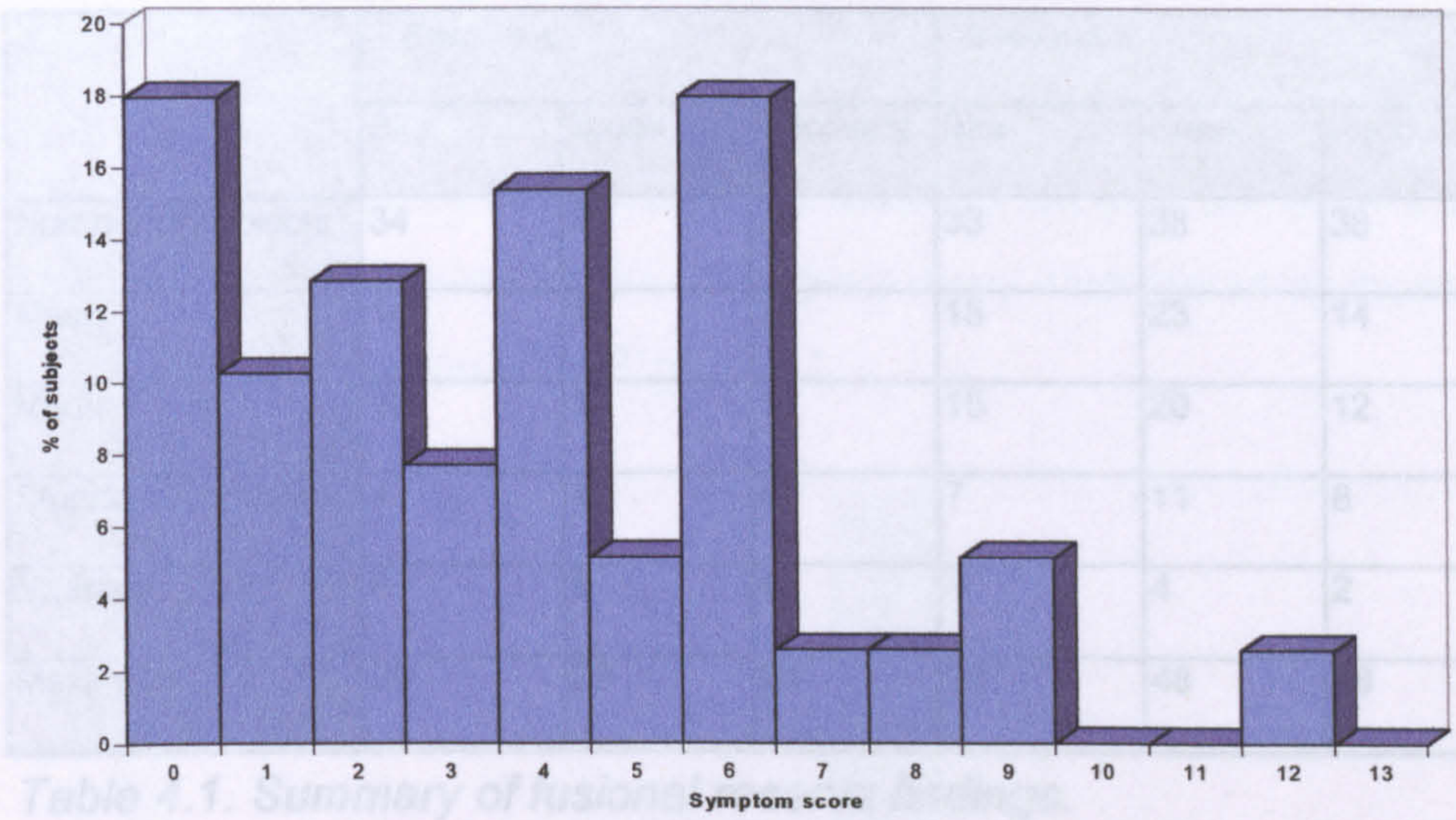


Figure 4.1. Frequency distribution of near symptom scores.

The mean symptom score (n = 39 subjects) was 3.7 (SD 3.0) with a range from 0 to 12).

These findings serve to illustrate the highly individual subjectivity of symptoms. What may be described as being a problem by one subject will not be perceived as a complaint or symptom by another.

4.3.2 Associated phoria

Associated phoria was estimated by neutralising reported fixation disparity on the Mallett unit using prisms in 0.25^{Δ} steps.

For distance vision, 2 subjects manifested 0.5^{Δ} esophoric slip, 1 subject 0.25^{Δ} esophoric slip, and 1 subject 0.5^{Δ} exophoric slip. For near vision 2 subjects manifested 0.5^{Δ} esophoric slip and 2 subjects reported 0.5^{Δ} exophoric slip.

4.3.3 Near fusional reserves

Table 4.1 and Figure 4.2 show summaries of the fusional reserve characteristics as measured with the variable prism stereoscope.

	Base in Δ			Base out Δ		
	Blur	Break	Recovery	Blur	Break	Recovery
Number of subjects	34	39	39	33	38	38
Mean	14	16	12	15	23	14
Median	12	16	12	15	20	12
Standard Deviation	4	4	4	7	11	8
Minimum	8	8	4	4	4	2
Maximum	28	28	24	32	48	38

Table 4.1. Summary of fusional reserve findings.

These findings are similar to the fusional reserve data from a normal population derived by Pickwell (1965) and cited by Evans (1997) (Table 4.2).

	Base in Δ			Base out Δ		
	Blur	Break	Recovery	Blur	Break	Recovery
Average values	11-15	18-24	7-15	14-20	18-24	7-15

Table 4.2. Average values of horizontal fusional reserves for near (33 – 40 cm) (after Pickwell, 1965).

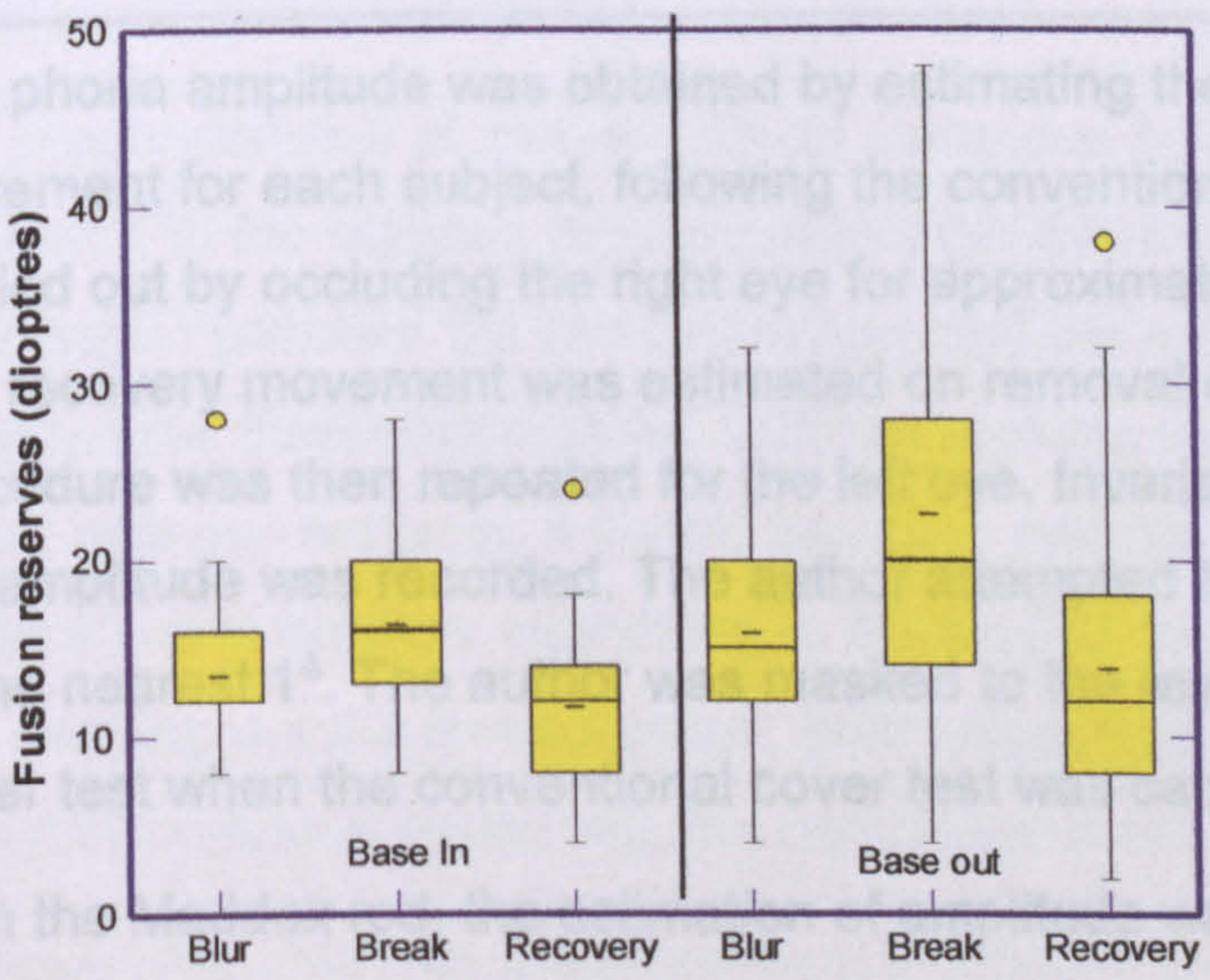


Figure 4.2. Box and whisker plot illustrating the distribution of blur/break/recovery fusional reserve measurements.

4.3.4 Near point of convergence (NPC)

The mean near point of convergence was 6.9 cm (n = 38; SD = 2.2). The range was from 3cm to 12 cm. The frequency distribution of these measurements is shown in Figure 4.3.

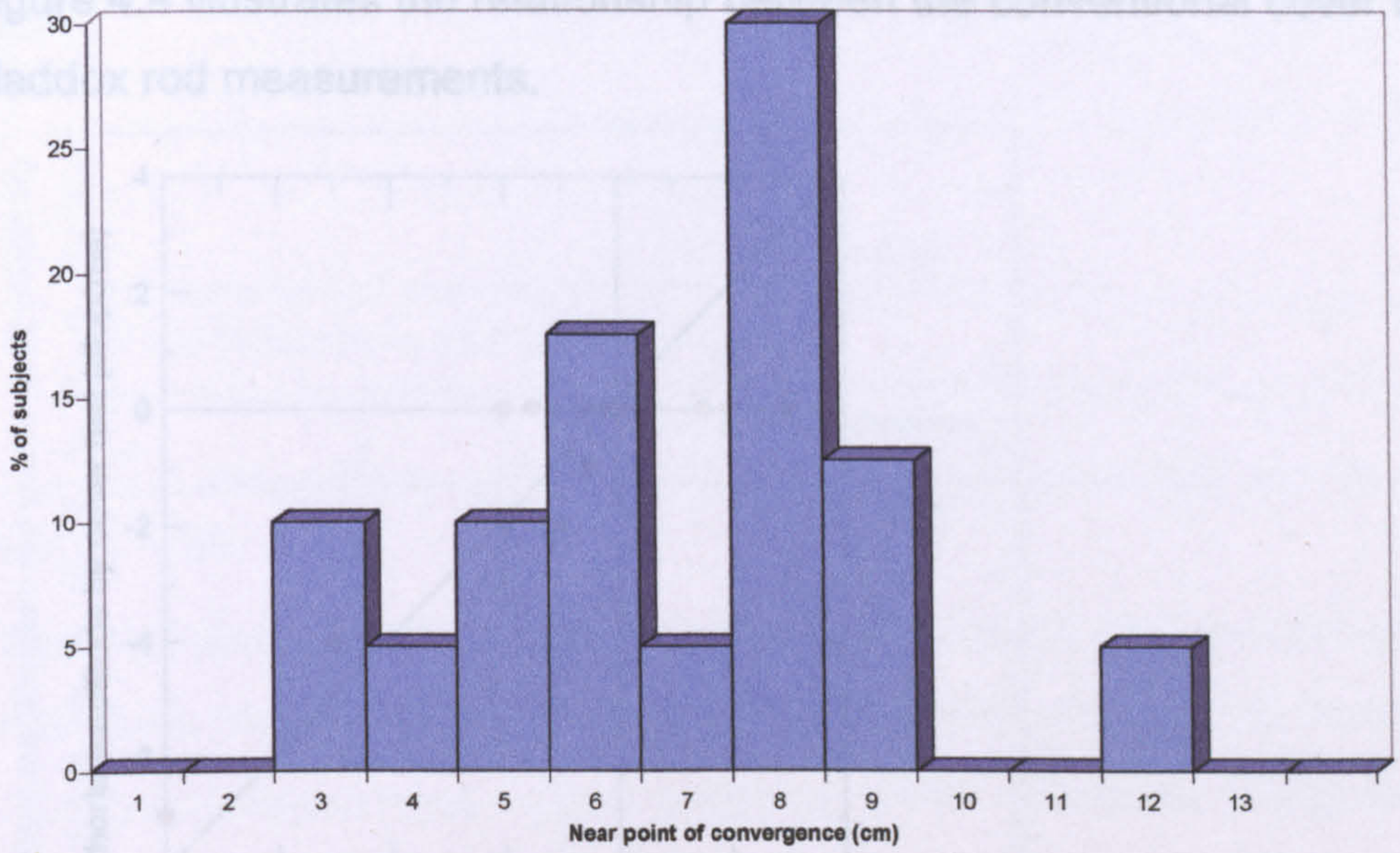


Figure 4.3. Frequency distribution of near point of convergence (cm).

4.3.5 Conventional cover test, Maddox rod and Maddox wing

The phoria amplitude was obtained by estimating the size of the recovery movement for each subject, following the conventional cover test. This was carried out by occluding the right eye for approximately 2 s. The amplitude of any recovery movement was estimated on removal of the cover. The procedure was then repeated for the left eye. Invariably, a single estimation of the amplitude was recorded. The author attempted to estimate the amplitude to the nearest 1^{Δ}. The author was masked to the results of the automated cover test when the conventional cover test was carried out.

With the Maddox rod, the estimation of amplitude was made using prisms to align the spotlight and the Maddox rod induced red line reported by the subject. Prisms were left in place for as brief a time as possible to minimise the effects of prism adaptation. Estimation was made to the nearest 0.25^{Δ}

4.3.5.a. Distance

The conventional cover test showed a mean phoria of 0.47$^{\Delta}$ exophoria (n = 39; SD = 1.37 $^{\Delta}$). This compared to the Maddox rod findings of a mean of 0.27 $^{\Delta}$ exophoria (n = 39; SD = 1.89$^{\Delta}$).

Figure 4.4 illustrates the relationship between the conventional cover test and Maddox rod measurements.

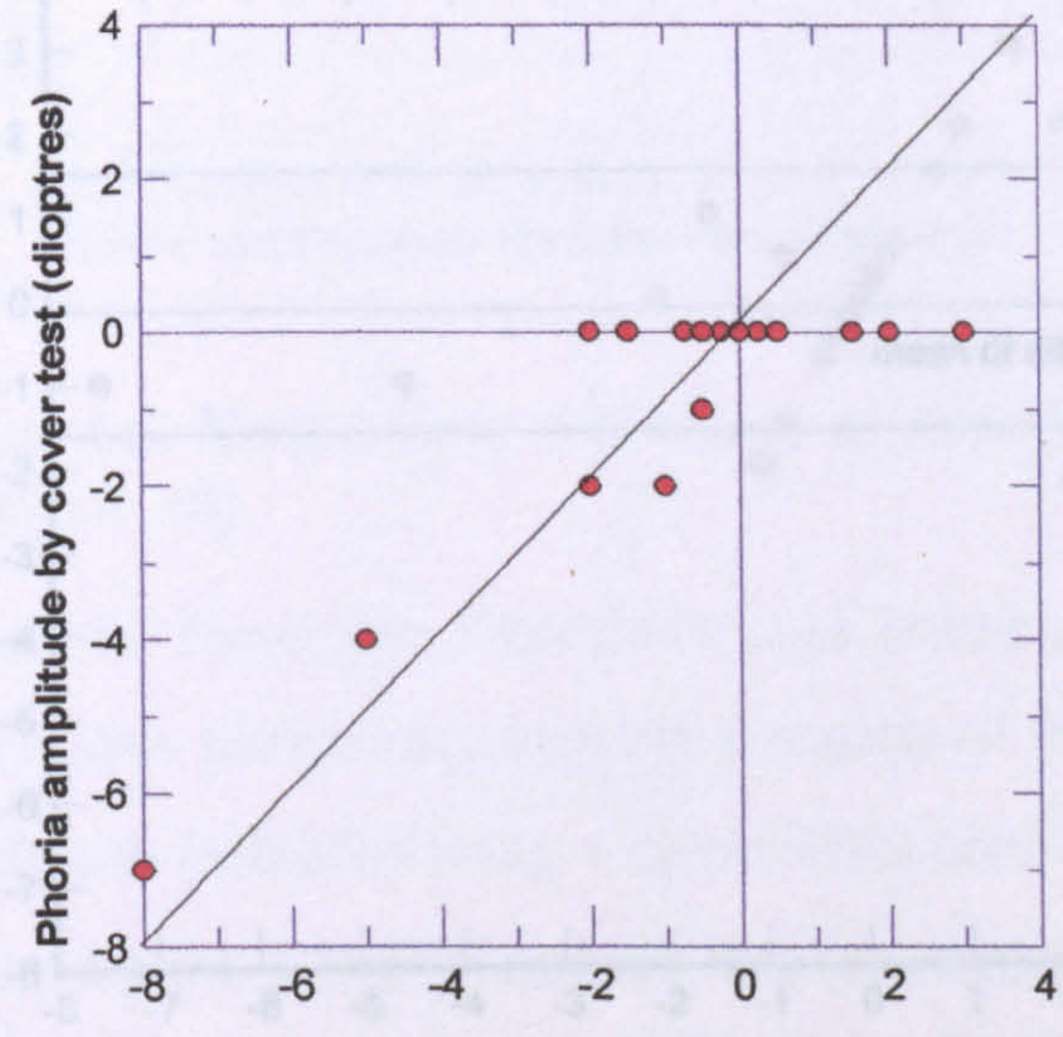


Figure 4.4. Distance phoria measurements by conventional cover test and Maddox rod (n = 39 subjects).

The preponderance of zero amplitude measures by conventional cover test compared to the Maddox rod may reflect the difference in the time period of dissociation between the two techniques and the resolution of the observer. Although the dissociation period with the Maddox rod was not controlled and varied between subjects, it should be noted that the routine of placing the Maddox rod in position, explaining to the subject the requirement of the test, and carrying out the measurement, took longer than the approximately 2 s period of occlusion period with the conventional cover test.

However, the data may also represent a difficulty in observing relatively small eye movements during the cover test as carried out by the author.

Figure 4.5 illustrates the difference between distance phoria measurements assessed by the Maddox rod and the conventional cover test plotted against the average of the two test methods.

The mean of differences (Maddox rod – cover test) was 0.08 $^{\Delta}$. The limits of agreement shown by the ± 2 SD lines in the diagram gave a range of 1.83$^{\Delta}$ to –1.65$^{\Delta}$.

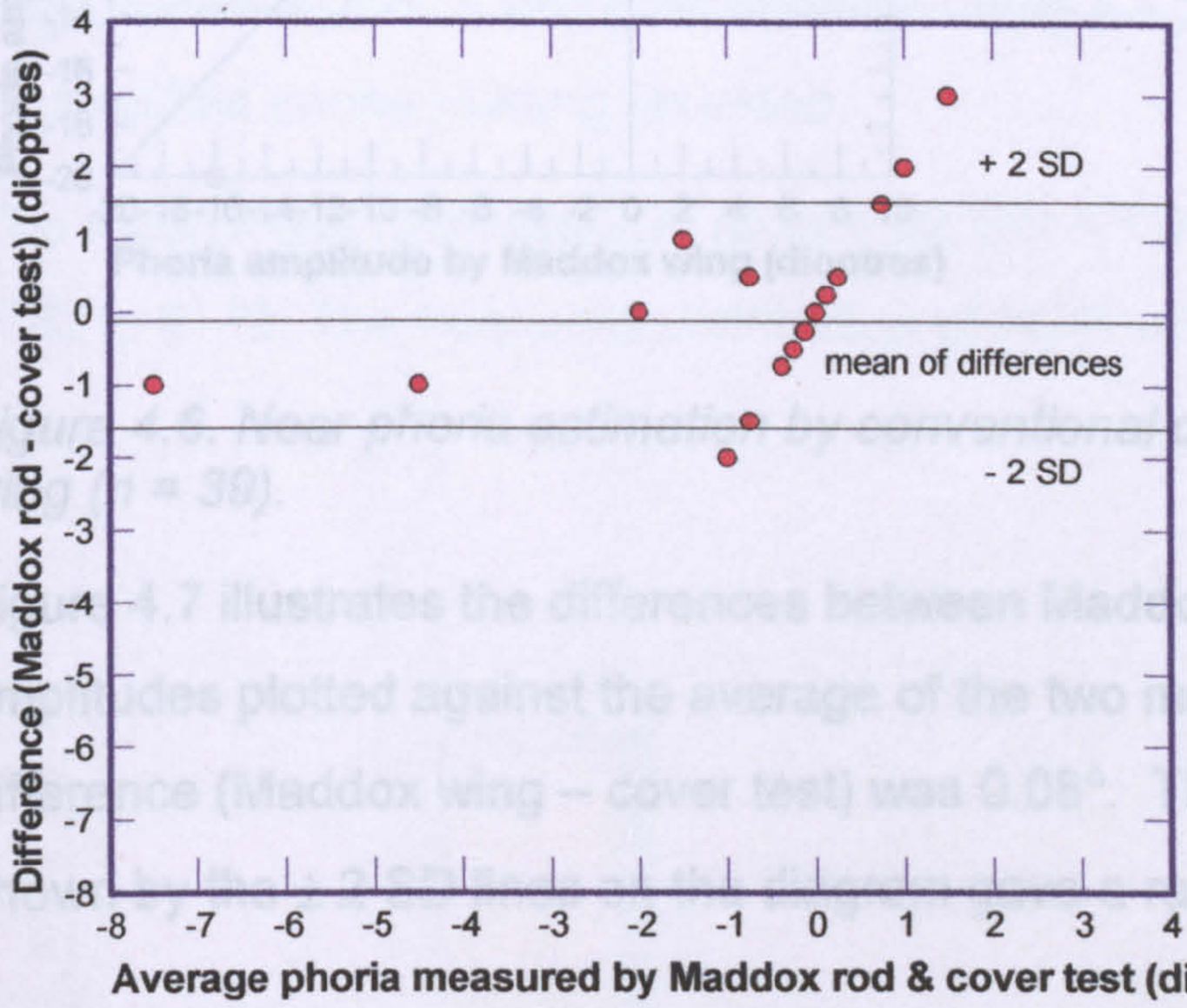


Figure 4.5. Difference between distance phoria amplitude measurements with the conventional cover test and with the Maddox rod plotted against the average of the results for the two test methods (n= 39 subjects).

These results suggest that there was poor agreement between the conventional cover test and Maddox rod findings for distance vision when carried out by this investigator.

4.3.5.b. Near

The conventional cover test showed a mean phoria of 2.5$^{\Delta}$ exophoria (n = 39; SD = 4.2$^{\Delta}$). This compared to the Maddox Wing findings of a mean of 2.4$^{\Delta}$ exophoria (n = 39; SD = 3.8$^{\Delta}$).

Figure 4.6 illustrates the relationship between conventional cover test findings for near fixation and Maddox wing results. The diagram shows a wider distribution of phoria amplitudes for 40 cm as compared to distance.

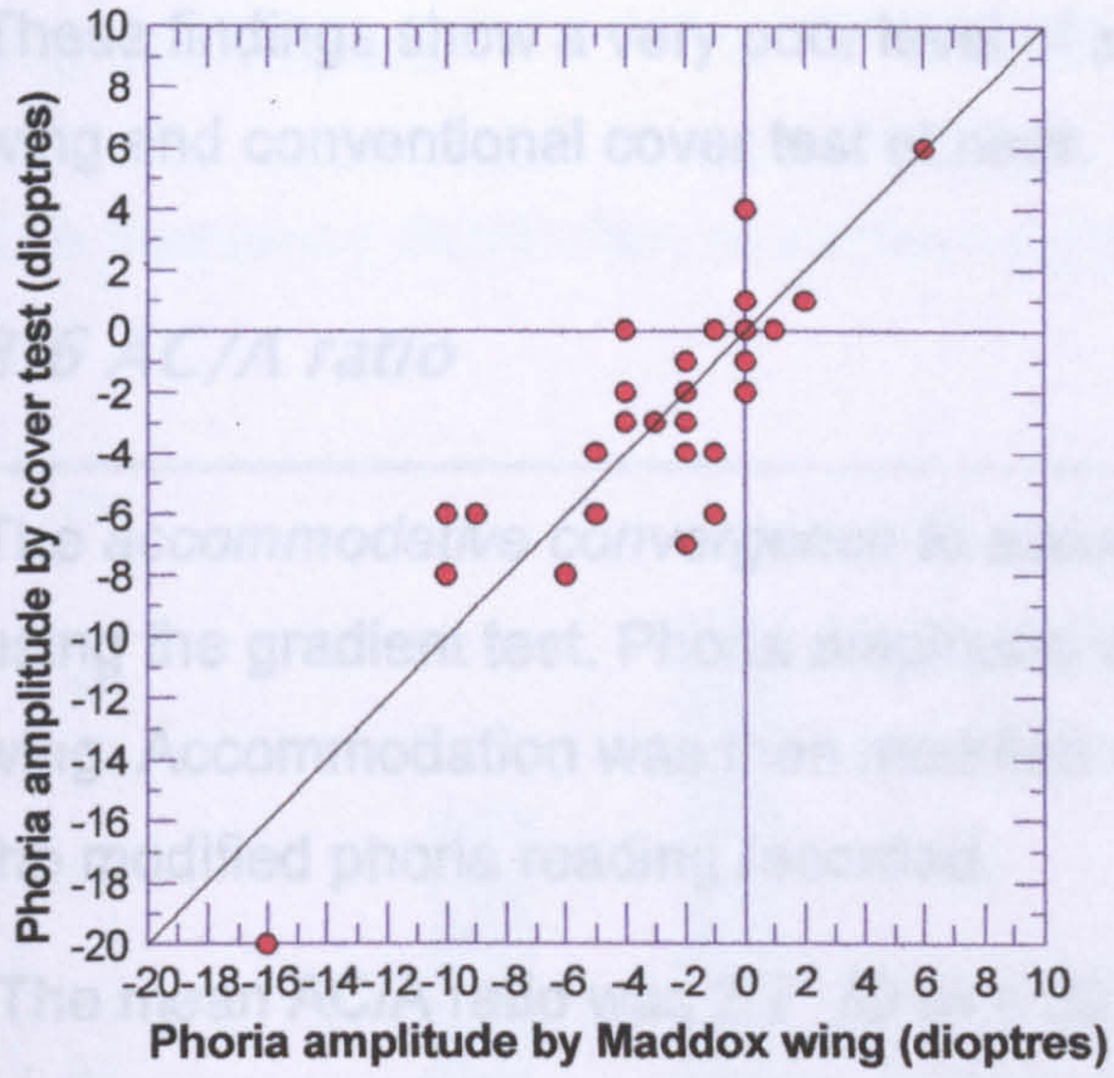


Figure 4.6. Near phoria estimation by conventional cover test and Maddox wing (n = 39).

Figure 4.7 illustrates the differences between Maddox wing and cover test amplitudes plotted against the average of the two methods. The mean difference (Maddox wing – cover test) was 0.08^{Δ}. The limits of agreement shown by the ± 2 SD lines on the diagram gave a range of 4.38^{Δ} to -4.22^{Δ}.

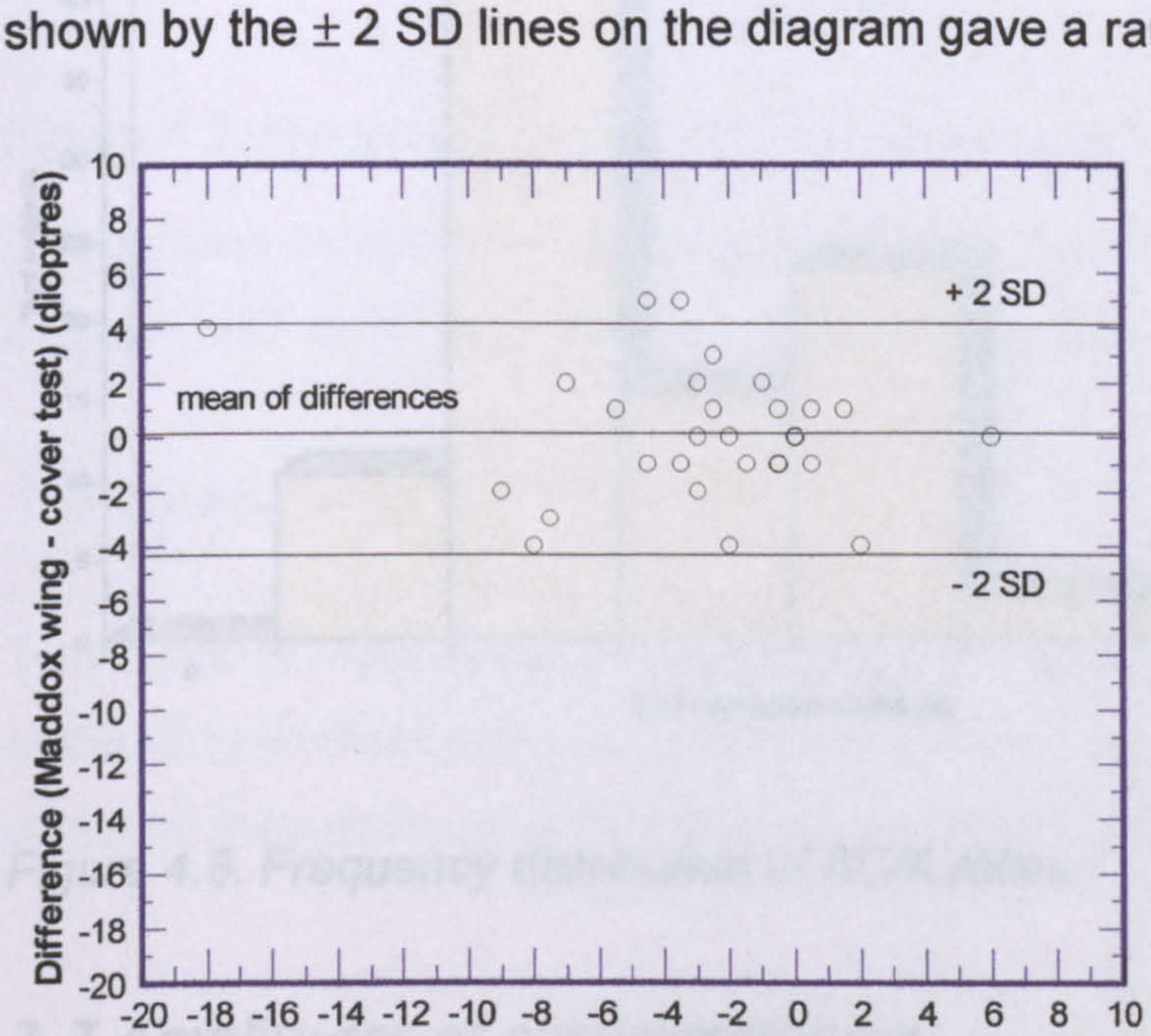


Figure 4.7. Difference between near phoria amplitude measurements with the conventional cover test and with the Maddox wing plotted against the average of the results for the two test methods (n = 39).

These findings show a very poor level of agreement between the Maddox wing and conventional cover test at near.

4.3.6 AC/A ratio

The *accommodative convergence to accommodation ratio* was measured using the gradient test. Phoria amplitude was first measured using a Maddox wing. Accommodation was then modified using a + 2 D binocular addition and the modified phoria reading recorded.

The mean AC/A ratio was 2.7^{Δ} /D (n = 39; SD = 1.2^{Δ} /D). The range was from 1^{Δ} /D to 6^{Δ} /D. The frequency distribution of AC/A ratios is shown in Figure 4.8.

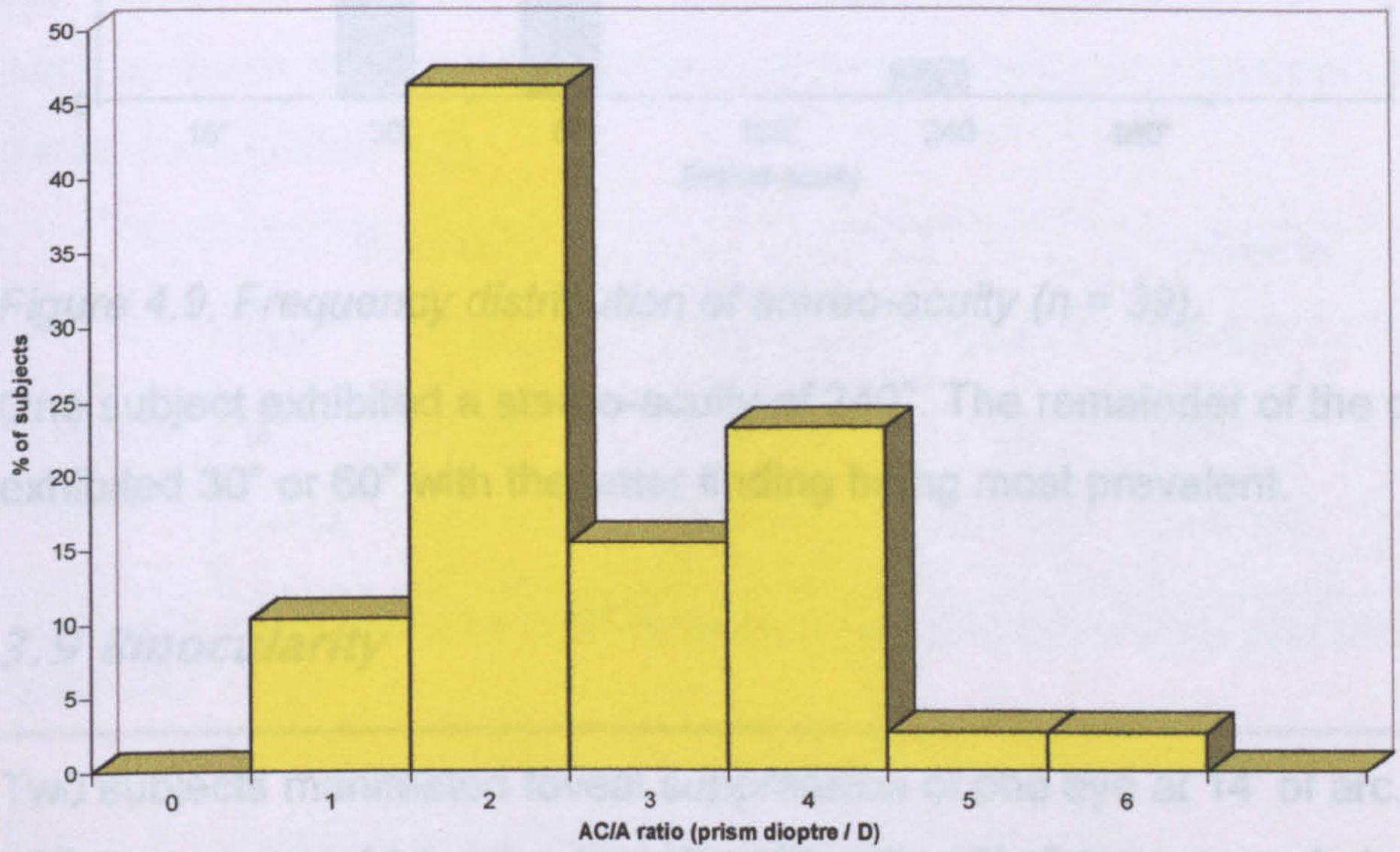


Figure 4.8. Frequency distribution of AC/A ratios.

4.3.7 Amplitudes of accommodation

Amplitude of accommodation was measured to the nearest 0.5 D for each eye using a RAF rule (n = 39 subjects). The mean amplitude for the right eye was 8.6 D (SD 1.7 D) compared to 8.7 D (SD =1.7D) for the left eye. The range for all eyes was from 5.5 D to 13.0D.

4.3.8 Stereopsis

The frequency distribution of stereo-acuity for 39 subjects is shown in Figure 4.9.

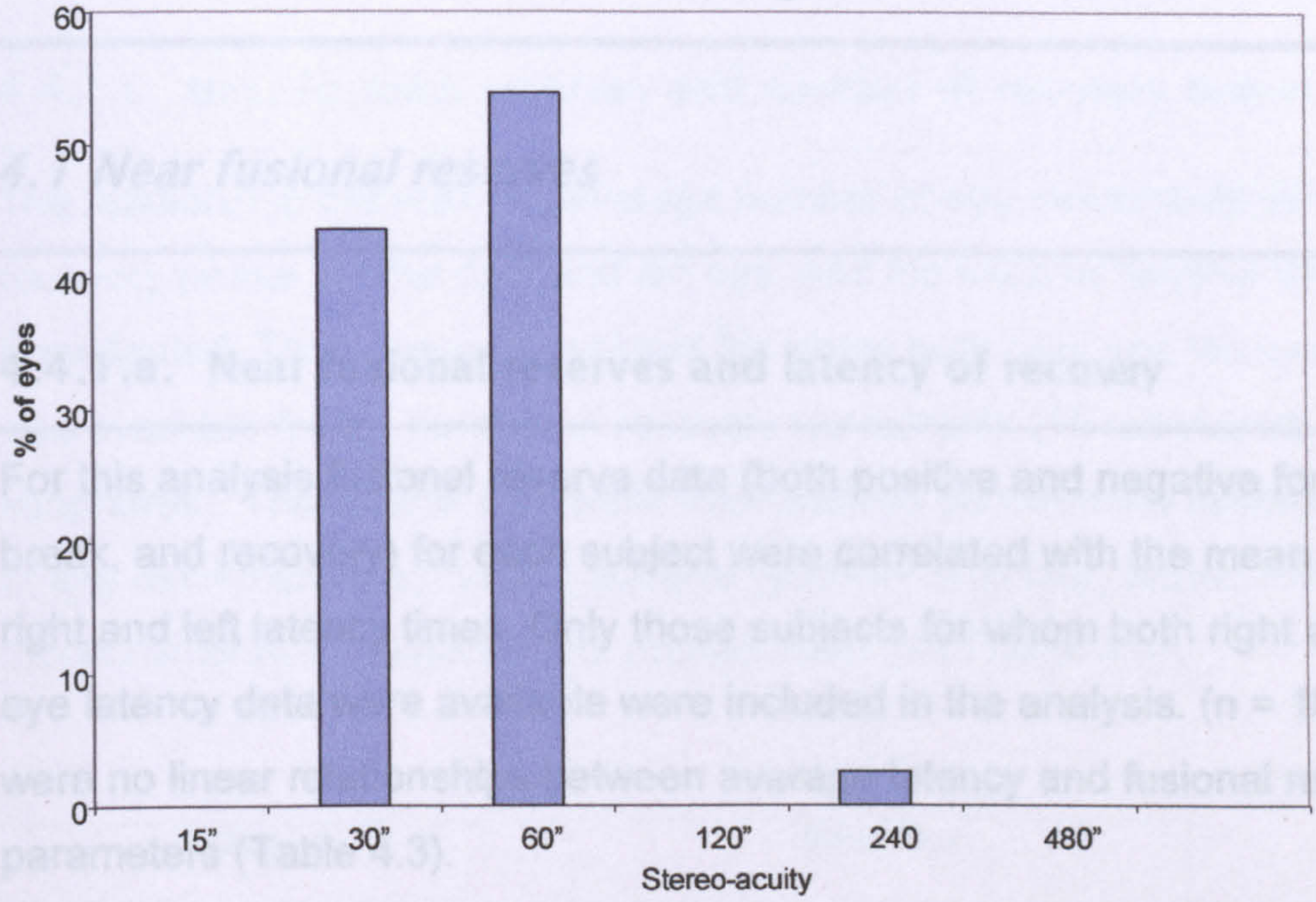


Figure 4.9. Frequency distribution of stereo-acuity (n = 39).

One subject exhibited a stereo-acuity of 240". The remainder of the population exhibited 30" or 60" with the latter finding being most prevalent.

4.3.9 Binocularity

Two subjects manifested foveal suppression of one eye at 14' of arc. As both of these eyes could resolve targets subtending 5' of arc monocularly, it was deduced they were exhibiting foveal suppression of 9' of arc.

4.4 Relationship between results of clinical tests and eye movement characteristics during the automated cover tests

4.4.1 Near fusional reserves

4.4.1.a. Near fusional reserves and latency of recovery

For this analysis fusional reserve data (both positive and negative for blur, break, and recovery) for each subject were correlated with the mean of the right and left latency times. Only those subjects for whom both right and left eye latency data were available were included in the analysis. (n = 18). There were no linear relationships between average latency and fusional reserve parameters (Table 4.3).

	Base out			Base in		
	Blur	**Break**	**Recovery**	**Blur**	**Break**	**Recovery**
Spearman Rank R^2	19% n = 14	5% n = 17	4% n = 17	< 1% n = 16	2% n = 168	< 1% n = 18

Table 4.3. Spearman Rank R^2 data for average latency time and fusional reserves.

4.4.1.b. Near fusional reserves and recovery time

I. Relationship between positive and negative fusional reserves (prism dioptres) and time for recovery following the near cover test

For this analysis fusional reserve data (both positive and negative for blur, break, and recovery) for each subject were correlated with the mean of the right and left recovery times. Only those subjects for whom both right and

left eye recovery time data were available were included in the analysis (n = 24).

There was no linear relationship between average recovery time and any fusional reserve parameter (Spearman Rank R^2 < 5% for all cases).

4.4.1.c. Near fusional reserves and number of recovery movements

The relationship between the average number of eye movements during the recovery phase, for the right and left eye, and the fusional reserve data was investigated. There were 17 subjects for whom both right and left eye data was available for the number of recovery movements (15 exophores and 2 esophores). There were very weak relationships between the average number of eye movements required to achieve recovery, and blur, break, and recovery measurements for *negative* and *positive* reserves (Table 4.4).

Base In *Spearman Rank R^2*			**Base Out** *Spearman Rank R^2*		
Blur	**Break**	**Recovery**	**Blur**	**Break**	**Recovery**
15% (n =15)	6% (n = 17)	15% (n = 17)	10% (n = 13)	5% (n = 17)	1% (n = 17)

Table 4.4. Relationships between the number of recovery movements and fusional reserves.

4.4.2 Near point of convergence

4.4.2.a. NPC and near phoria amplitude^{RL}

There was no linear relationship between NPC and amplitude of phoria^{RL} (Pearson R^2 = < 1%; n = 34 subjects).

4.4.2.b. NPC and latency of recovery

There was no linear relationship between NPC and latency of recovery following the near automated cover test (Pearson R^2 = < 1%; n = 44 eyes).

4.4.2.c. NPC and number of recovery movements

There was no linear relationship between NPC and the number of eye movements required to achieve recovery following the near automated cover test (Spearman R^2 = < 1%; n = 40 eyes).

4.4.2.d. NPC and recovery time

There was a very poor correlation between NPC and recovery time following the near automated cover test (Pearson R^2 = < 1%; n = 54 eyes)

4.4.3 Associated phoria

The number of eyes manifesting an associated phoria for either distance or near was too small (< 5) to allow meaningful statistical analyses of the relationships between groups with and without associated and phoria amplitude, latency of recovery, number of eye movements to achieve recovery, and recovery time.

4.4.4 Comparison of the conventional cover test and the automated cover test phoria amplitudes

The results of the conventional cover test were compared with the phoria amplitudes measured after both 2 seconds and 10 seconds of automated occlusion. As has been noted previously, the test distance was slightly different for the two procedures. However, the difference in convergence to a target at a distance of 600 cm (0.17 MA) compared to 340 cm (0.29 MA) is approximately 0.12 MA. For a subject with a 60 mm inter-pupillary distance, this is equivalent to approximately 13.4' of arc, which is clinically insignificant.

However, the closer test distance during the automated cover test would influence accommodation. For a typical AC/A ratio of 3^{Δ}/D the effect of 0.3 D of accommodation would be 1^{Δ}.

4.4.4.a. Distance

Figure 4.10 shows the relationship between the distance phoria amplitudes measured with the conventional cover test and by automated cover test.

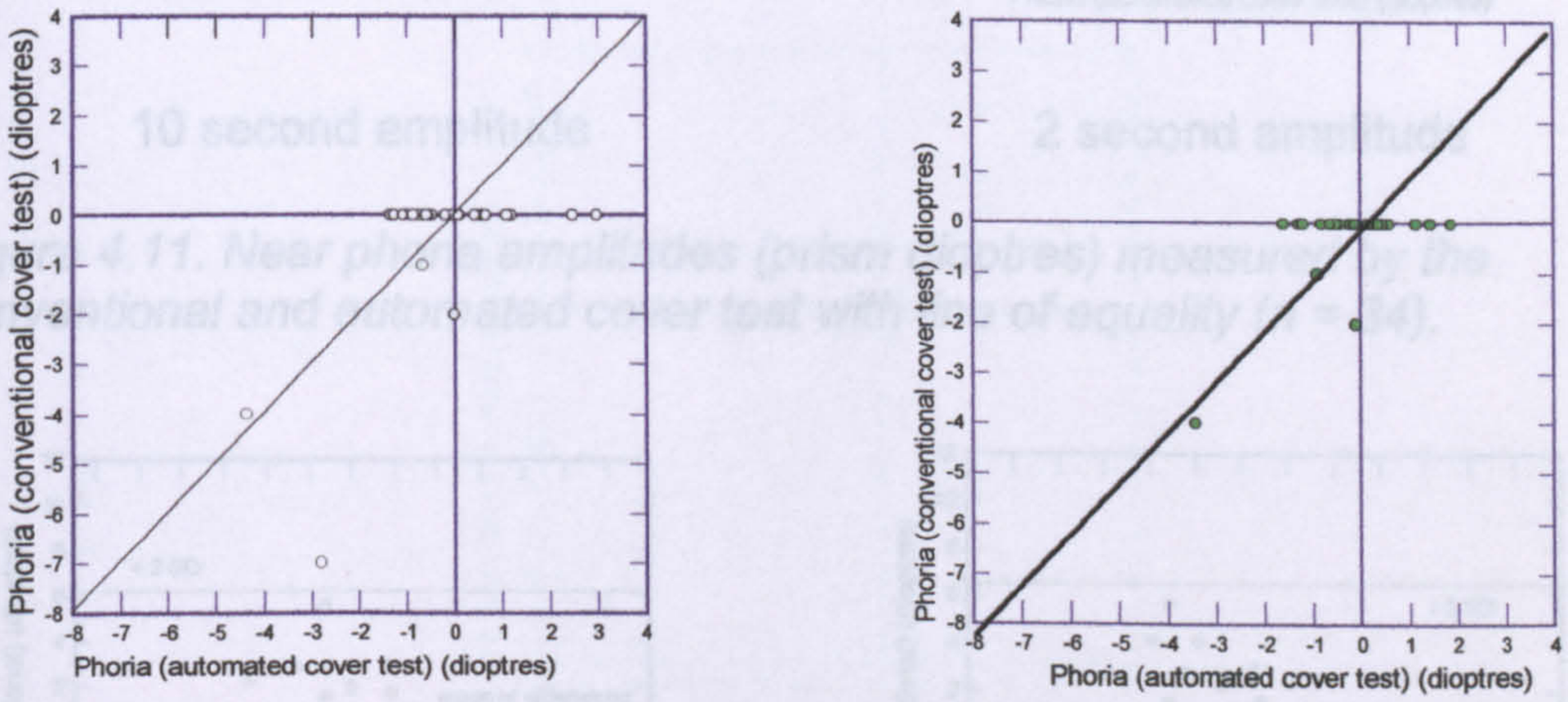
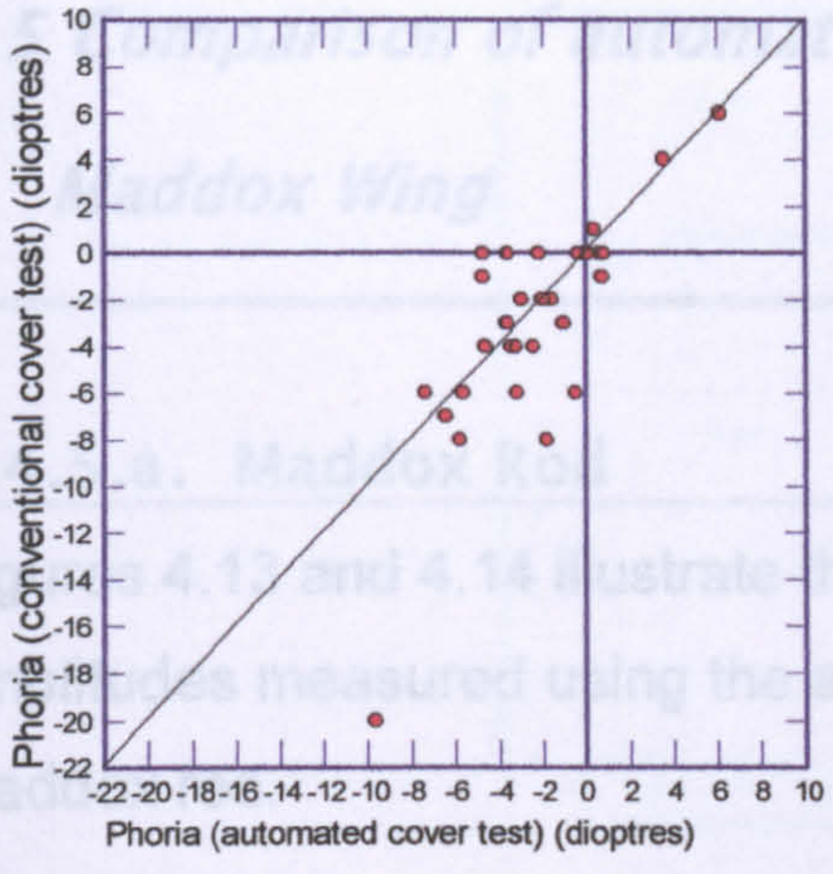


Figure 4.10. Distance phoria amplitudes (prism dioptres) measured by the conventional and automated cover test with line of equality (n = 27).

As with the comparison between Maddox rod and the conventional cover test, the findings shown in Figures 4.10 illustrate a poor level of agreement between conventional and automated cover tests.

4.4.4.b. Near

Figures 4.11 and 4.12 show the relationship between the near phoria amplitudes measured with the conventional cover test and by automated cover test.



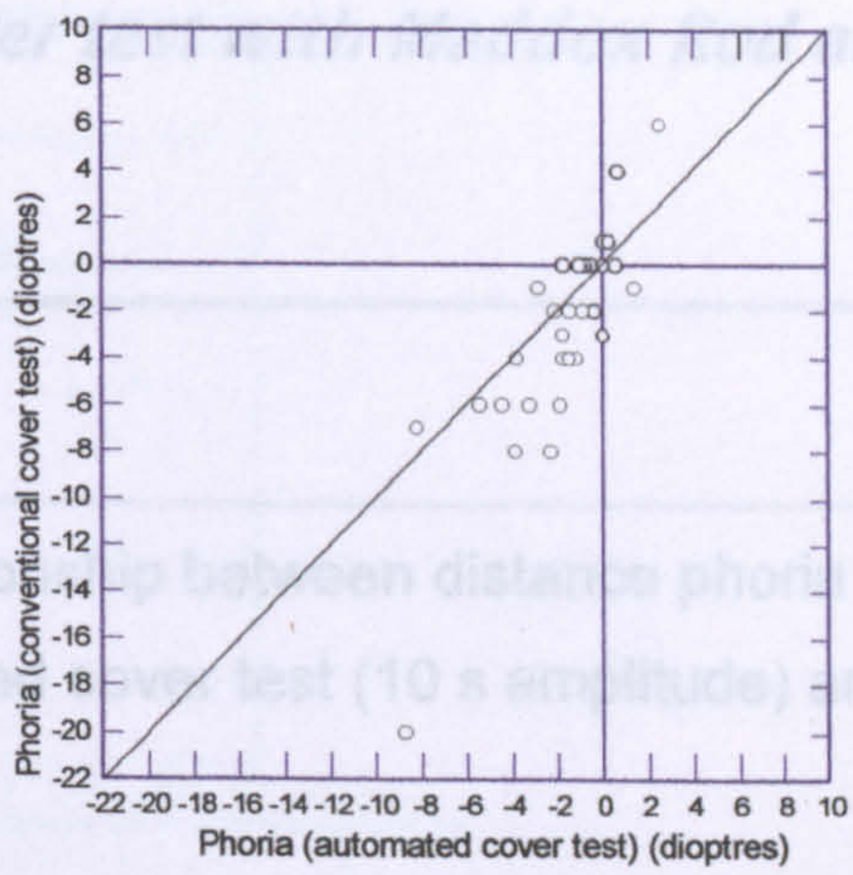
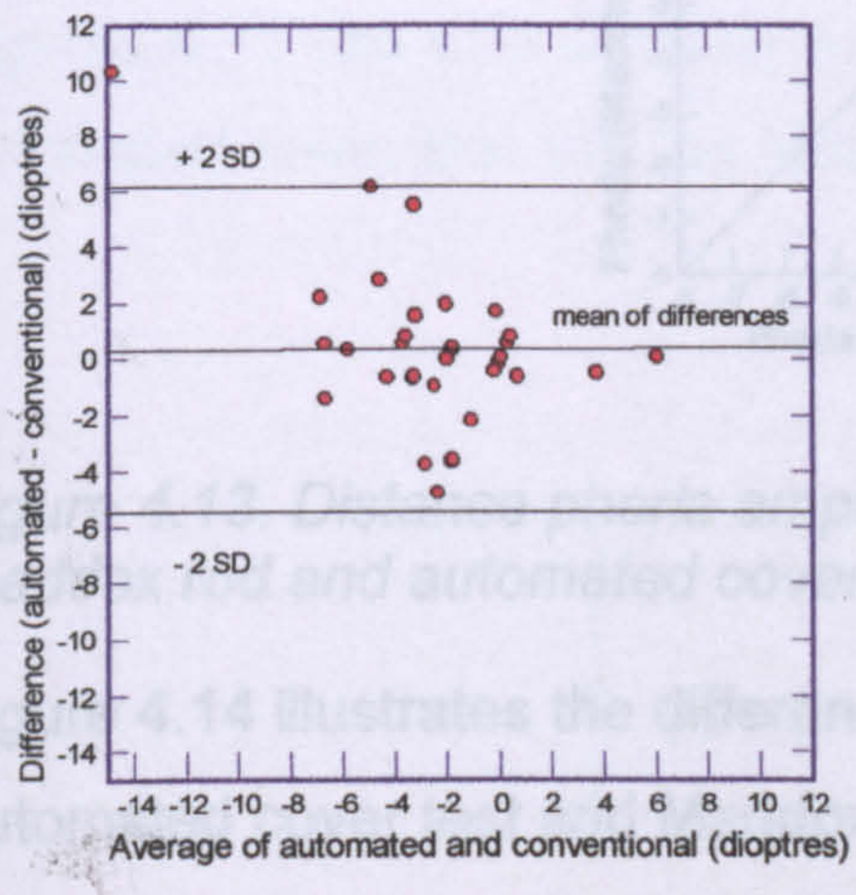


Figure 4.11. Near phoria amplitudes (prism dioptres) measured by the conventional and automated cover test with line of equality (n = 34).



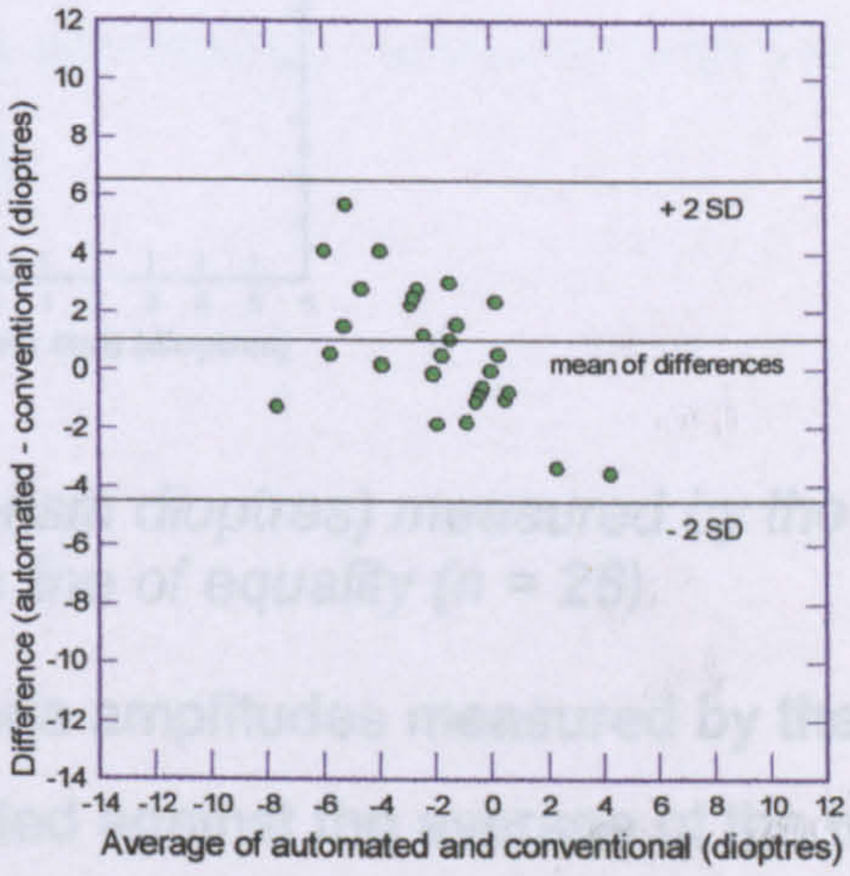


Figure 4.12. Difference between near phoria measurements with the conventional and automated cover tests plotted against the average.

Figure 4.12 demonstrates very poor agreement between the two methods for near when carried out by this investigator. The mean of differences between automated and conventional cover tests was 1.00^{Δ} for the 2 s amplitude and 0.36^{Δ} for the 10 s amplitude. The limits of agreement shown by the ± 2 SD lines were 6.42^{Δ} to -5.42^{Δ} for the 2 s amplitude and 6.06^{Δ} to -5.34^{Δ} for the 10 s amplitude.

4.4.5 Comparison of automated cover test with Maddox Rod and Maddox Wing

4.4.5.a. Maddox Rod

Figures 4.13 and 4.14 illustrate the relationship between distance phoria amplitudes measured using the automated cover test (10 s amplitude) and the Maddox rod.

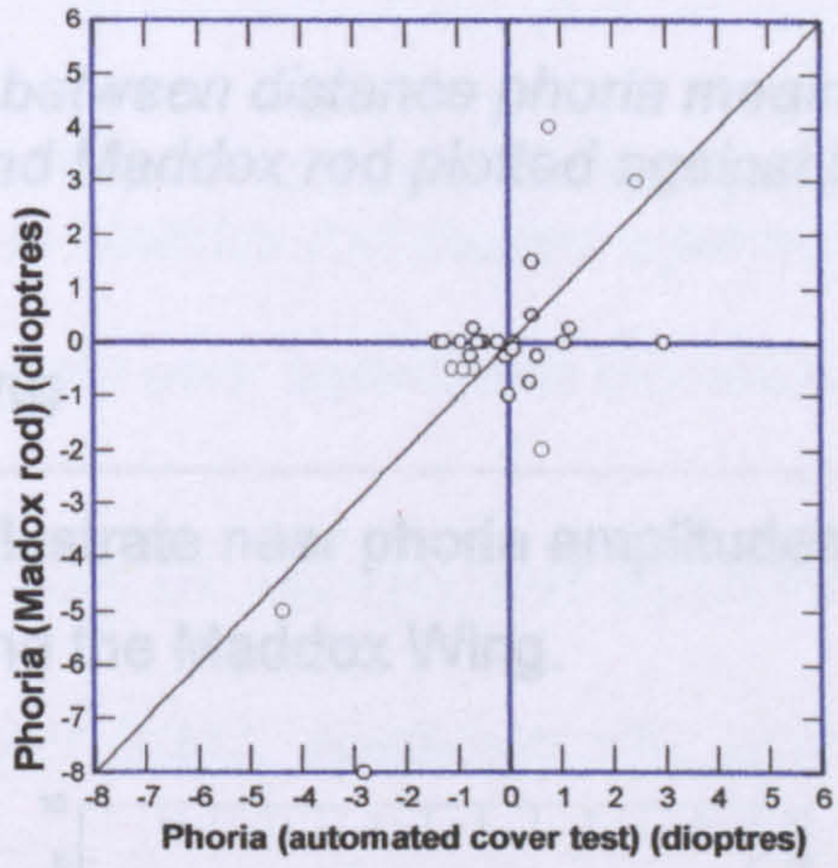


Figure 4.13. Distance phoria amplitudes (prism dioptres) measured by the Maddox rod and automated cover test with line of equality (n = 28).

Figure 4.14 illustrates the difference in phoria amplitudes measured by the automated cover test and Maddox rod plotted against the average of the two methods. The mean of the differences was – 0.07$^{\Delta}$ and the limits of agreement, shown by the ± 2 SD lines on the diagram, were 1.49$^{\Delta}$ to –1.63$^{\Delta}$.

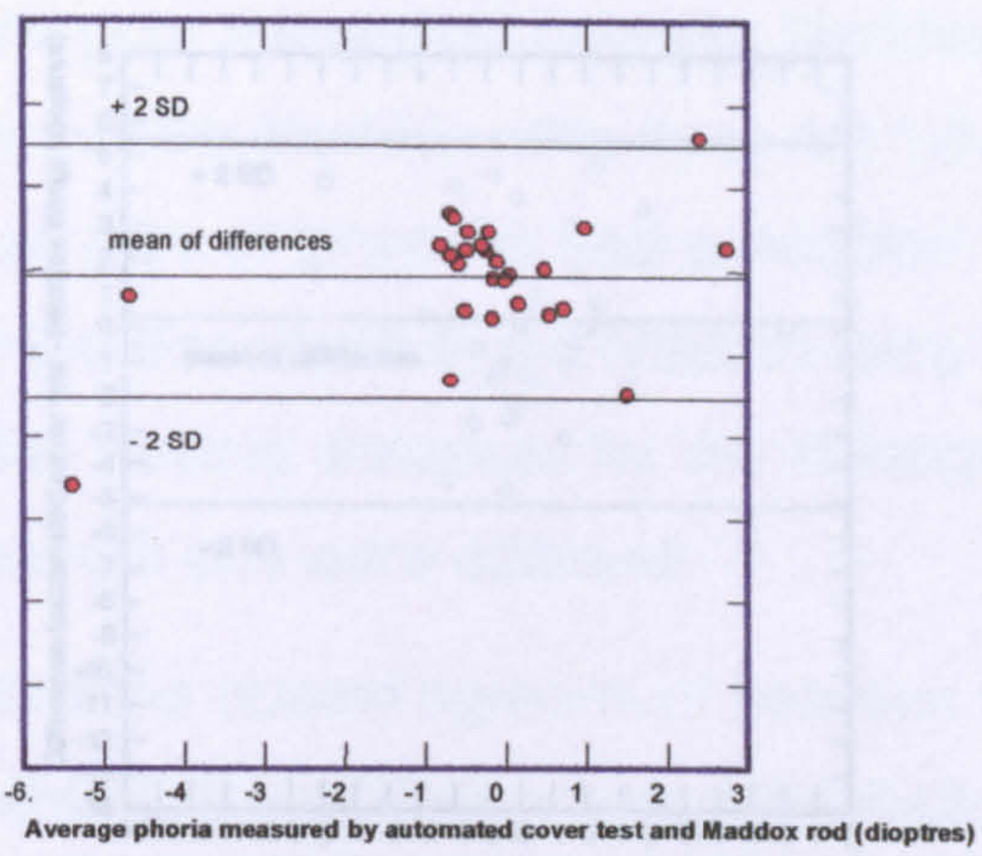


Figure 4.14. Difference between distance phoria measurements with the automated cover test and Maddox rod plotted against the average.

4.4.5.b. Maddox Wing

Figures 4.15 and 4.16 illustrate near phoria amplitudes measured with the automated cover test and the Maddox Wing.

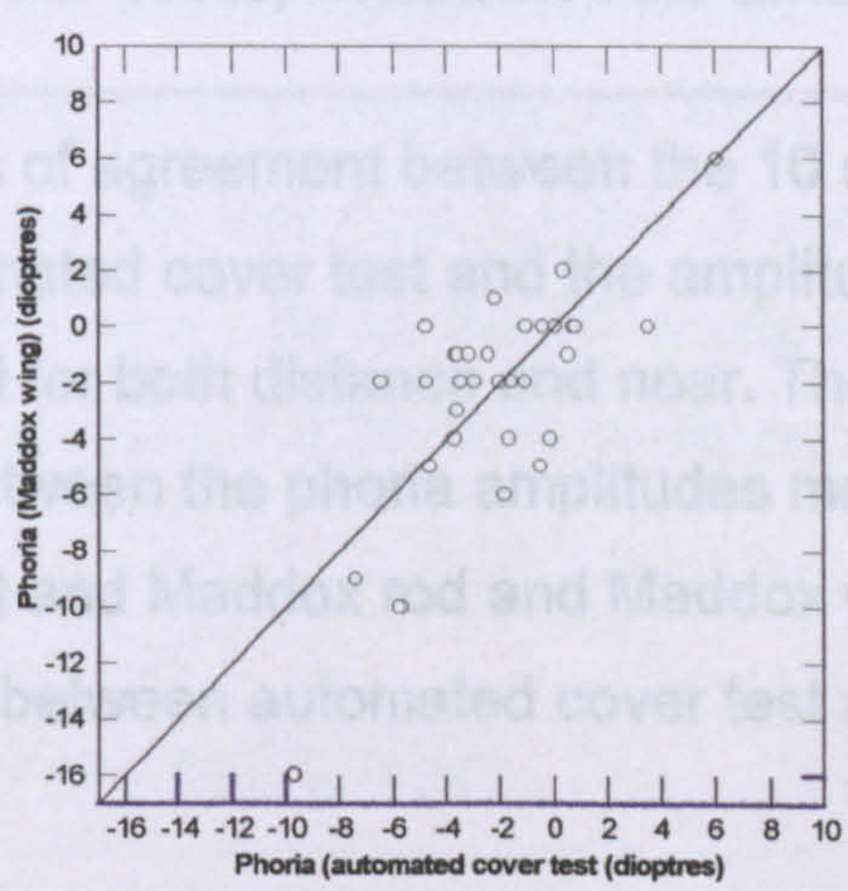


Figure 4.15. Near phoria amplitudes (prism dioptres) measured by the automated cover test and the Maddox wing with line of equality (n = 35).

The mean of differences between the two methods was 0.24^{Δ}. The limits of agreement, as shown by the ±2 SD lines in Figure 4.16, were 5.58^{Δ} to 5.10^{Δ}.

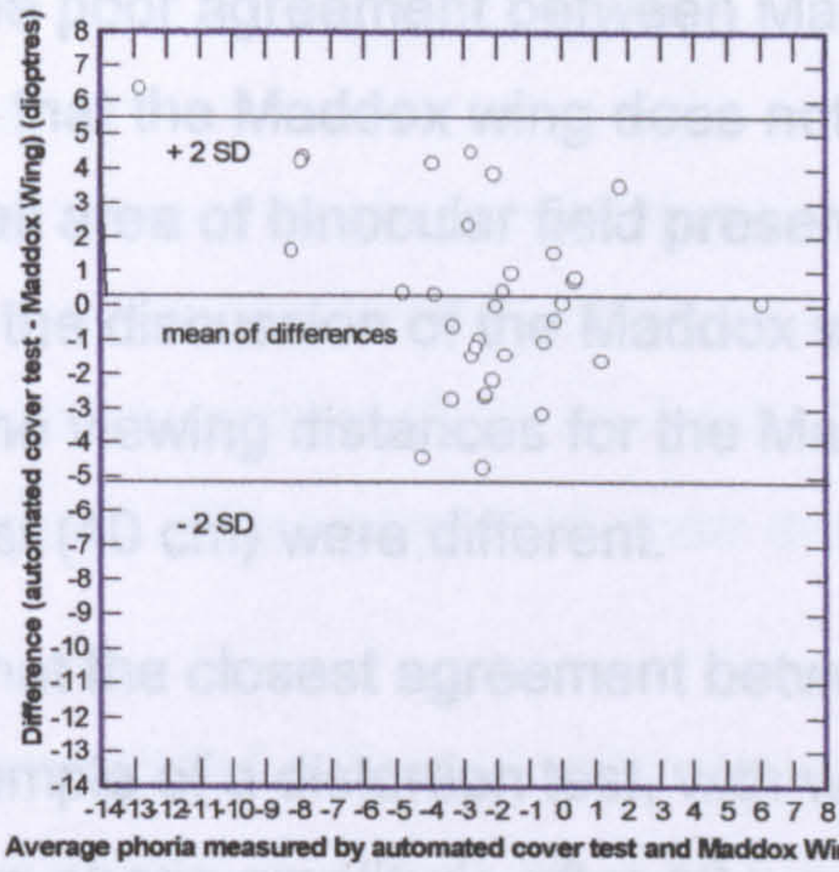


Figure 4.16 Difference between near phoria measurements with the automated cover test and Maddox rod plotted against the average.

These findings suggest very poor agreement between these two techniques.

4.4.6 Discussion of levels of agreement between automated and conventional cover tests, Maddox rod and Maddox Wing

There were poor levels of agreement between the 10 s phoria amplitudes measured by the automated cover test and the amplitude measured by the conventional cover test for both distance and near. There were also poor levels of agreement between the phoria amplitudes measured by the conventional cover test and Maddox rod and Maddox wing for distance and near respectively, and between automated cover test and Maddox wing for near.

The poor agreement between automated and conventional cover tests may have been due to the difference in dissociation time for the two techniques. However, the level of agreement was also poor between the conventional cover test and the phoria amplitude measured with the automated cover test after 2 s occlusion at near. As the investigator was occluding the eye for about 2 s during the conventional cover test, this suggests a possibility that this investigator may have been inaccurate in estimating the size of the phoria with the conventional cover test at near.

A possible reason for the poor agreement between Maddox wing and automated cover test is that the Maddox wing does not fully dissociate the eyes, there being a small area of binocular field presented. This has been alluded to previously in the discussion of the Maddox wing and Mills test in Chapter 1. In addition the viewing distances for the Maddox wing (30 cm) and the automated cover test (40 cm) were different.

It is of interest to note that the closest agreement between tests occurs with the Maddox rod, an example of a distortion test, with which the eyes are dissociated fully, and the phoria amplitude after 10 s occlusion during the automated cover test.

4.4.7 Stereopsis

4.4.7.a. Stereo-acuity and recovery time

There was no significant statistical difference between the group of subjects manifesting 60" of arc and another manifesting 30" of arc, for recovery times following the automated cover test. The number of eyes for which recovery time data was available was n = 34 for the 60" group and n = 21 for the 30" group. (Mann-Whitney U test p = 0.98; 95% confidence interval = -0.17 s to 0.19 s).

4.4.7.b. Stereo-acuity and latency

There was no significant statistical difference between the group of subjects manifesting 60" of arc and another manifesting 30" of arc, for latency times following the automated cover test. The number of eyes for which latency time data was available was n = 28 for the 60" group and n = 16 for the 30" group. (Mann-Whitney U test p = 0.41; 95% confidence interval = -0.03 s to 0.06 s).

4.5 Summary of results

This chapter has described the relationship between a number of tests of binocular function and eye movement characteristics during the cover test, in a group of "normal " subjects. The sample size was small so results should be viewed with caution.

Most tests were poorly correlated with each other and show little association with any aspect of eye movements during the cover test. There was no linear relationship between NPC and recovery time following the automated cover test. There was neither a relationship between measures of fusional reserves and recovery time nor between fusional reserves and the number of recovery eye movements following the cover test.

Whilst the 10 s phoria amplitude measured with the automated cover test showed a poor level of a agreement with the conventional cover test and the Maddox Wing, the former showed a closer level of agreement with the Maddox rod for the measurement of horizontal phoria amplitude. A possible reason for Maddox rod measurements showing agreement with the automated cover test results is that the dissociation period with the Maddox rod needs to be prolonged enough to enable the clinician to neutralise the phoria with prisms. In other words, both techniques allow time for the eye to move towards a position of rest.

The number of subjects manifesting an associated phoria was too small to enable a statistical evaluation with regard to various eye movement characteristics.

Chapter 5. Eye movement characteristics during the cover test in a group of 30 subjects referred for treatment of binocular vision anomalies

5.1 Introduction

The aim of this part of the study was to record the eye movement characteristics during the automated cover test, of a group of subjects referred to the study for treatment of horizontal ocular motor anomalies, and to compare those characteristics with those of the 'normal' group.

As part of the initial protocol, formal arrangements, including Ethical Committee agreement, had been made with the Orthoptic Department at Moorfields Eye Hospital for the referral of patients to the study with convergence insufficiency prior to treatment commencing. Despite the full co-operation of the orthoptist team at Moorfields, no patients were referred to the study over a two-year period. It was concluded that the incidence of patients of the age and other characteristics demanded by the protocol was lower in this hospital than had been assumed by both the author and the orthoptist team. A criticism of this study is that a formal estimation of the incidence of suitable patients attending that Department had not been carried out in advance of this study.

5.2 Subjects

Under the circumstances, a review of the study proposal had to be made at this point. It was decided that a more generalised recruitment protocol would be employed. Supervising clinicians in the primary care optometry clinics at City University were invited to refer to the study any horizontal heterophoria or convergence anomaly particularly if the patient reported near vision symptoms. In addition a number of subjects were recruited by inviting

students in the wider university population to attend if they suffered asthenopic type symptoms during or following near vision work.

The criteria for inclusion in the study were as follows:

- a diagnosed or suspected horizontal binocular vision anomaly with or without symptoms suspected of being associated with this anomaly
- no constant heterotropia as assessed by the cover test
- VA of 6/6 or better with each eye;
- a refractive error of between -0.50 DS (red on duochrome) and +0.75 DS (green on duochrome) with no more than 0.50 DS of spectacle astigmatism and no more than 0.50 D of anisometropia;
- aged between 18 and 35 years;
- not wearing contact lenses;
- no history of ocular disease.

A total of 30 subjects were recruited to this phase of the study. 29 subjects met the refractive criteria. 1 subject was +1 D anisometropic but was uncorrected when she entered the study. 1 subject presented with a minimal symptom score but with a diagnosed CI. No Brown's or Duane's syndromes were observed.

The data obtained from this group was subsequently compared to that of the "normal" group previously described in Chapter 3.

5.3 Methods

The method employed has been discussed in Chapters 2 and 3.

5.4 Results

A number of parameters were extracted from the eye movement recordings. These parameters were listed in the previous chapter.

5.4.1 The cover phase

5.4.1.a. Phoria amplitudes

I. Comparisons of amplitudes for left and right eyes

Distance fixation

The mean 10 s phoria amplitude for right eyes was –0.59 degrees (n = 27; SD = 1.46) compared to –1.07 degrees (n = 27; SD = 2.3) for left eyes.

Frequency distribution

Frequency distributions for right and left eyes for amplitudes after 10 seconds of occlusion are shown in Figure 5.1.

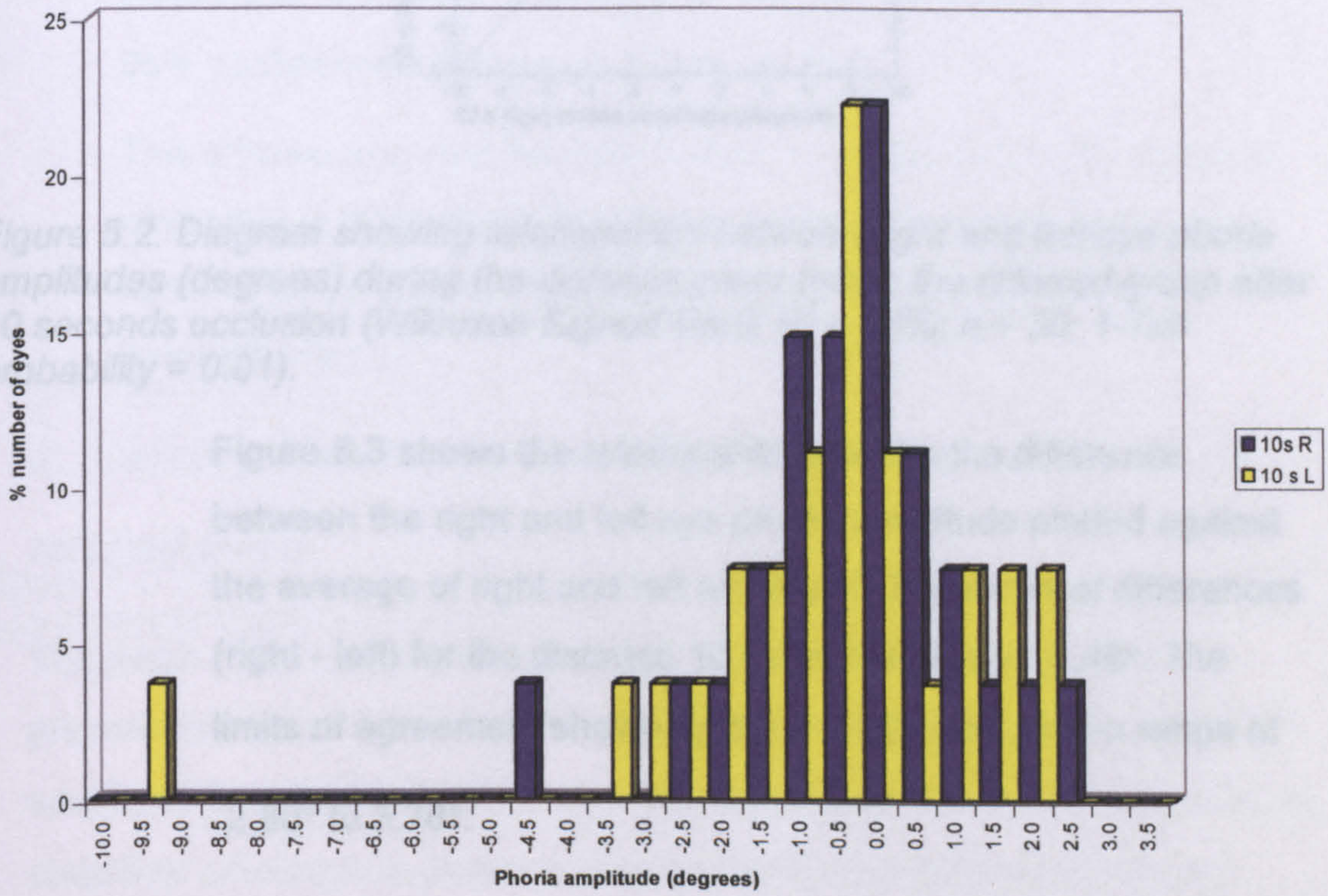


Figure 5.1. Frequency distributions (%) of phoria amplitudes (degrees) of right and left eyes during the distance cover test after 10 seconds occlusion.

Relationship between right and left eye 10 s phoria amplitudes during the distance automated cover test

Because the data distribution appeared skewed, a Wilcoxon Signed Rank test was employed. There was no significant difference between right and left eye phoria amplitude for distance 10 s phoria amplitude ($p = 0.11$).

Figure 5.2 describes the relationship between right and left phoria amplitudes following 10 s of occlusion in the referred group.

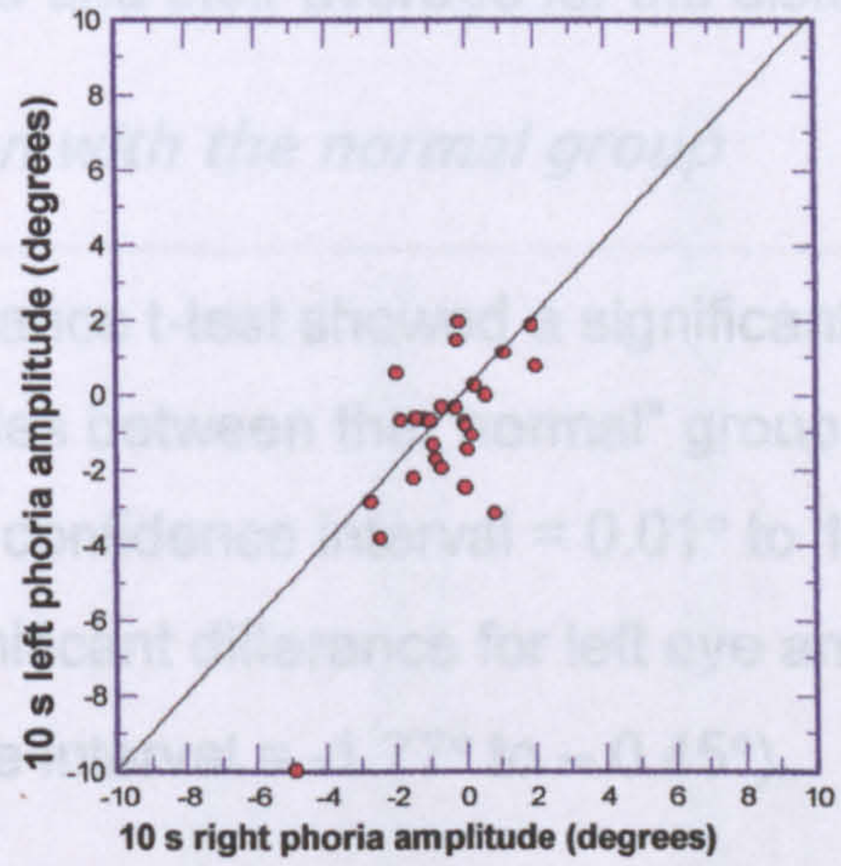


Figure 5.2. Diagram showing relationships between right and left eye phoria amplitudes (degrees) during the distance cover test in the referred group after 10 seconds occlusion (Wilcoxon Signed Rank $R^2 = 19\%$; $n = 30$; 1-Tail probability = 0.01).

Figure 5.3 shows the relationship between the difference between the right and left eye phoria amplitude plotted against the average of right and left amplitude. The mean of differences (right - left) for the distance 10 s amplitudes was 0.48°. The limits of agreement shown by the ± 2 SD lines gave a range of -2.80° to 3.76°.

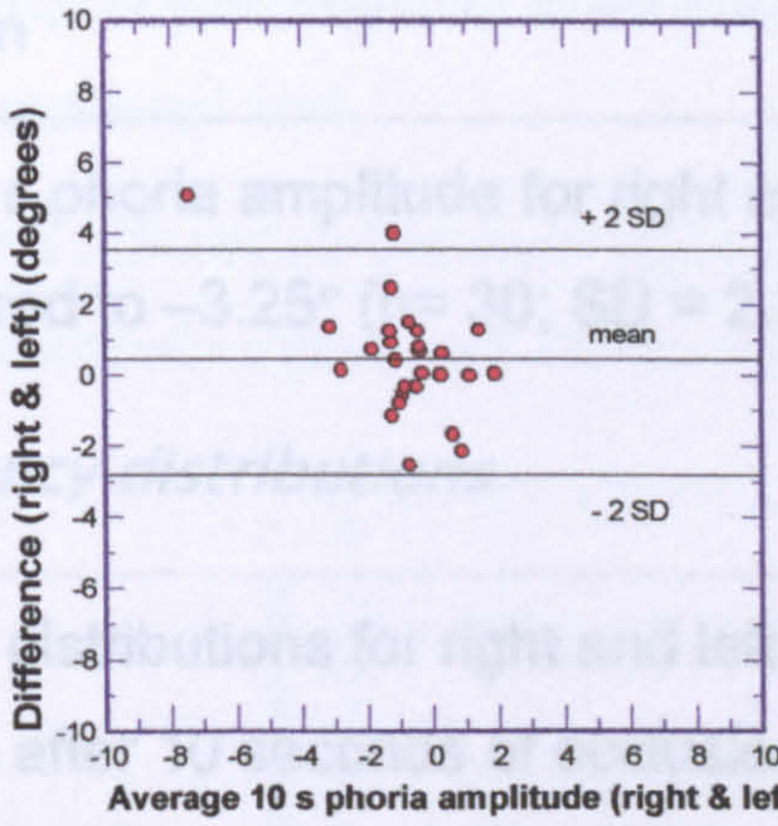


Figure 5.3. Relationship between the difference between right and left eye 10 second phoria amplitude and their average for the distance cover test.

Comparison with the normal group

A separate variance t-test showed a significant difference for right eye phoria amplitudes between the "normal" group and the referred group ($p = 0.05$; 95% confidence interval = 0.01° to 1.00°). There was a statistically significant difference for left eye amplitudes ($p = 0.00$; 95% confidence interval = -1.77° to – 0.45°).

This difference in eye movement characteristics during the distance cover test is of interest because the patients were included in the referred group because of symptoms associated with **near** heterophoria or CI.

II. Summary of distance phoria amplitude^{RL} characteristics for the referred group

The mean distance phoria amplitude^{RL} for the referred group at first presentation was –0.83° (n = 27; SD = 1.75°) with a range of phoria amplitude from 7.45° exophoria to 1.89° esophoria. There was a significant statistical difference in distance phoria amplitude between the referred population (n = 27) and the normal group (n = 80) (separate variance t-test p = 0.03; 95% confidence interval = 0.28 ° to 1.38°).

Near fixation

The mean 10 s phoria amplitude for right eyes was -2.86° (n = 30; SD = 2.28°) compared to –3.25° (n= 30; SD = 2.31°) for left eyes.

Frequency distributions

Frequency distributions for right and left eyes for near phoria amplitudes after 10 seconds of occlusion are shown in Figure 5.4. The frequency for neither right nor left eyes appear to be normally distributed.

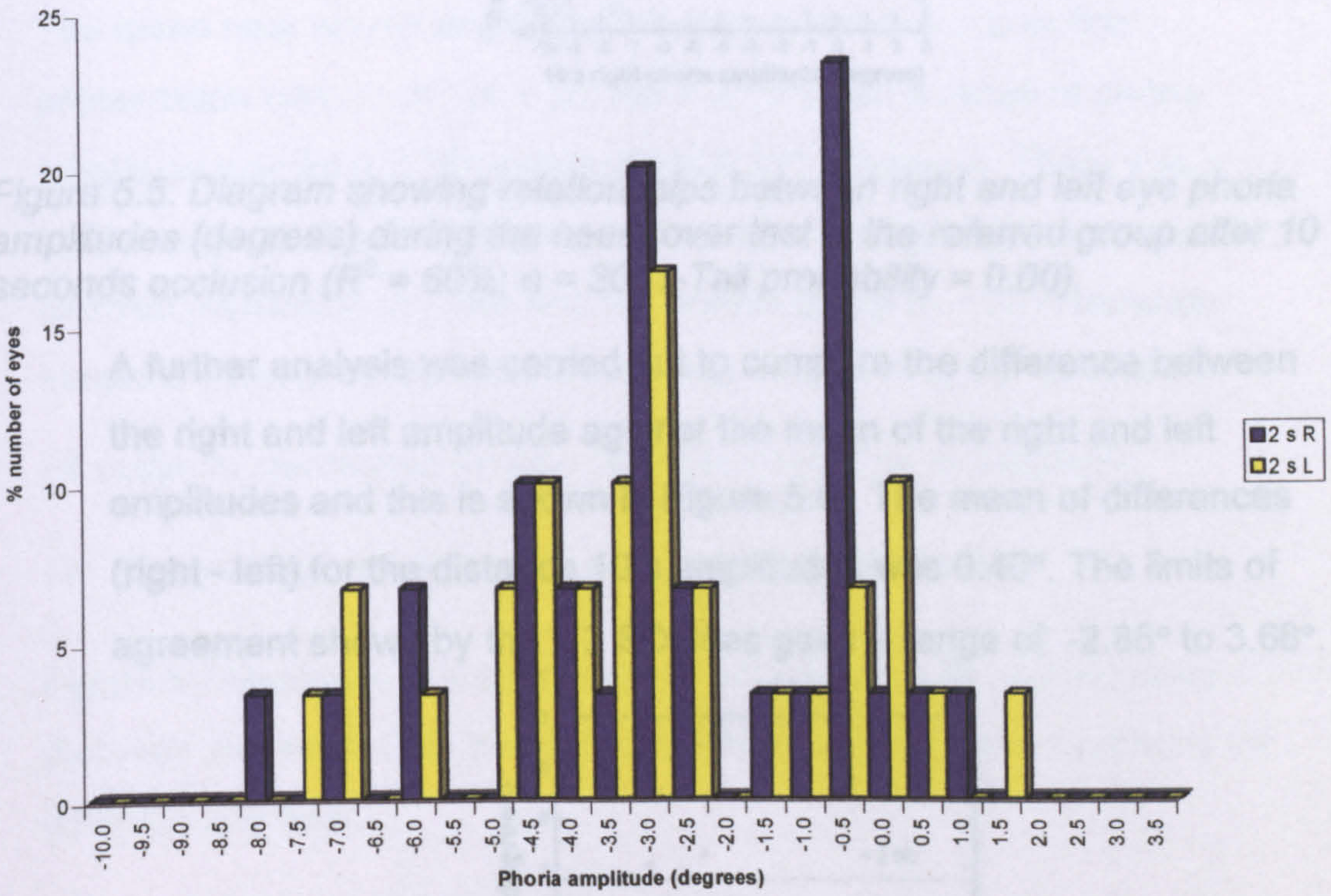


Figure 5.4. Frequency distributions (%) of phoria amplitudes (degrees) of right and left eyes during the near cover test after 10 seconds occlusion.

Relationship between right and left eye phoria amplitudes during the near automated cover test

Figure 5.5 shows the relationship between right and left phoria amplitudes following 10 s of occlusion. Left phoria amplitudes show a relative exophoric tendency compared to the right amplitudes. The

reason for this is possibly due to an order effect caused by the right eye being occluded first.

However, the difference between right and left eye phoria amplitudes was not statistically significant (Wilcoxon Signed Rank test p = 0.3).

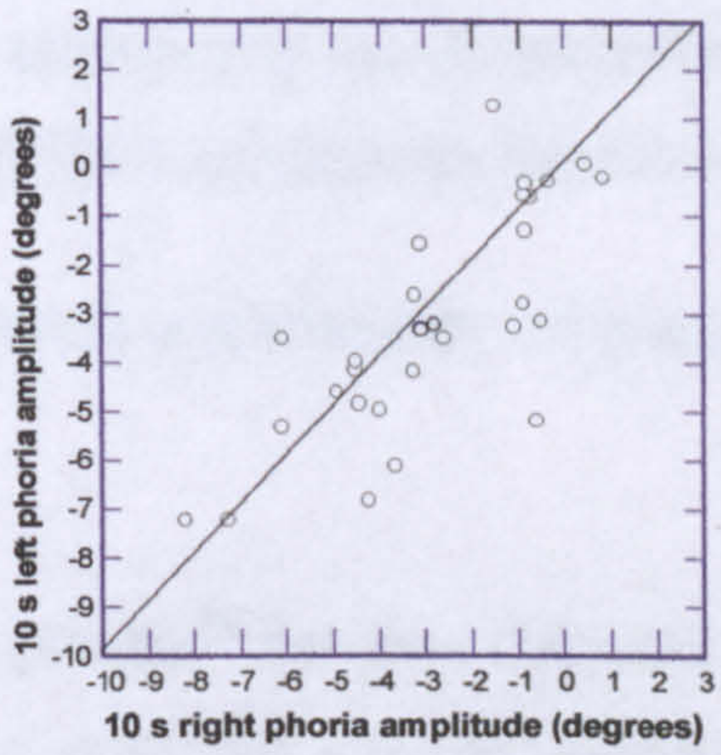


Figure 5.5. Diagram showing relationships between right and left eye phoria amplitudes (degrees) during the near cover test in the referred group after 10 seconds occlusion (R^2 = 60%; n = 30; 1-Tail probability = 0.00).

A further analysis was carried out to compare the difference between the right and left amplitude against the mean of the right and left amplitudes and this is shown in Figure 5.6. The mean of differences (right - left) for the distance 10 s amplitudes was 0.40°. The limits of agreement shown by the ± 2 SD lines gave a range of -2.88° to 3.68°.

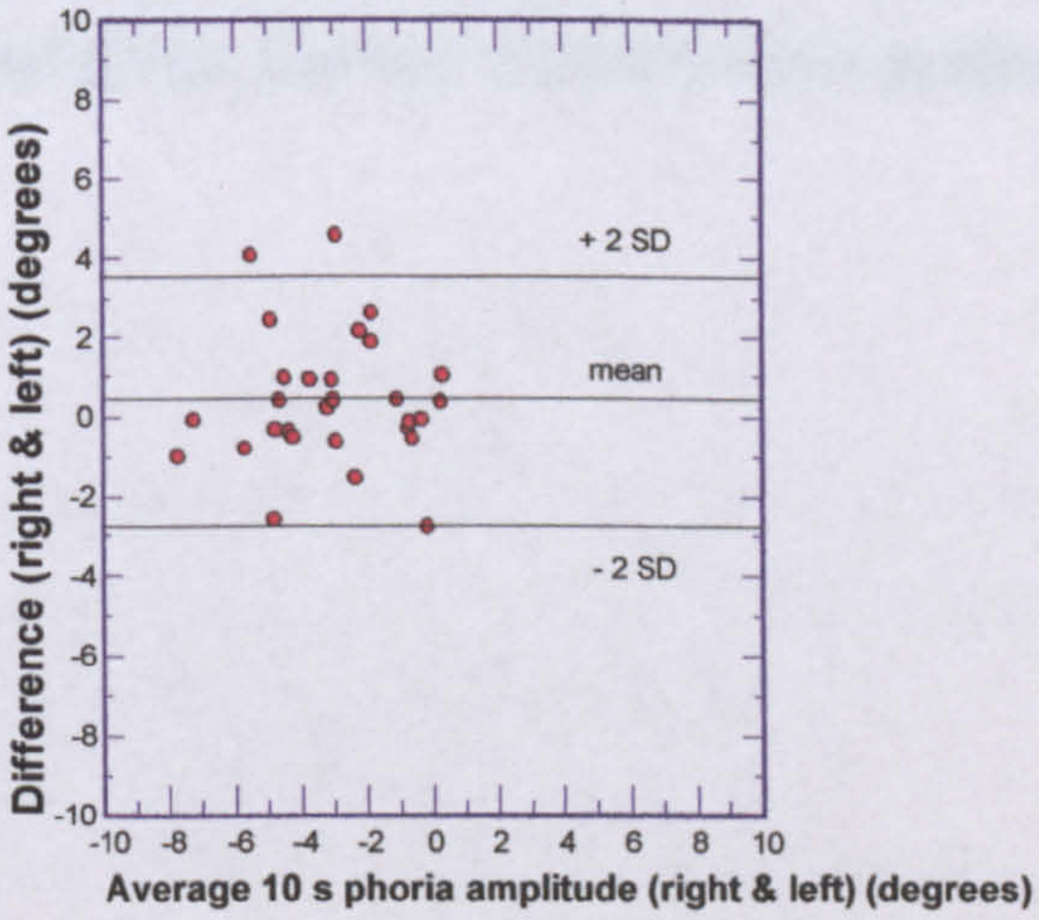


Figure 5.6. Relationship between the difference between right and left eye 10 s phoria amplitudes and their average for the near cover test.

Comparison with the normal group

A separate variance t-test showed a significant statistical difference for right eye phoria amplitudes between the "normal" group and the referred group (p = 0.000; 95% confidence interval = -2.22 ° to – 0.66°). There was a statistically significant difference for left eye amplitudes (p = 0.00; 95% confidence interval = -2.77° to –1.12°).

III. Summary of near phoria amplitude^{RL} characteristics for the referred group

The mean near phoria amplitude^{RL} for the referred group at first presentation was –3.06° (n = 30; SD = 2.15°) with a range of phoria amplitude was from 7.72° exophoria to 0.31° esophoria. There was a statistically significant difference in near phoria amplitudes between the referred population (n = 30) and the normal group (n = 93) (separate variance t-test p = 0.00; 95% confidence interval = -2.46° to –0.90°).

28 subjects manifested exophoria at near and 2 were esophoric.

IV. Comparison of distance and near amplitudes^{RL}

Figure 5.7 illustrates a preponderance of subjects with exophoria for distance and near. Only two of the referred subjects were esophoric for distance and near.

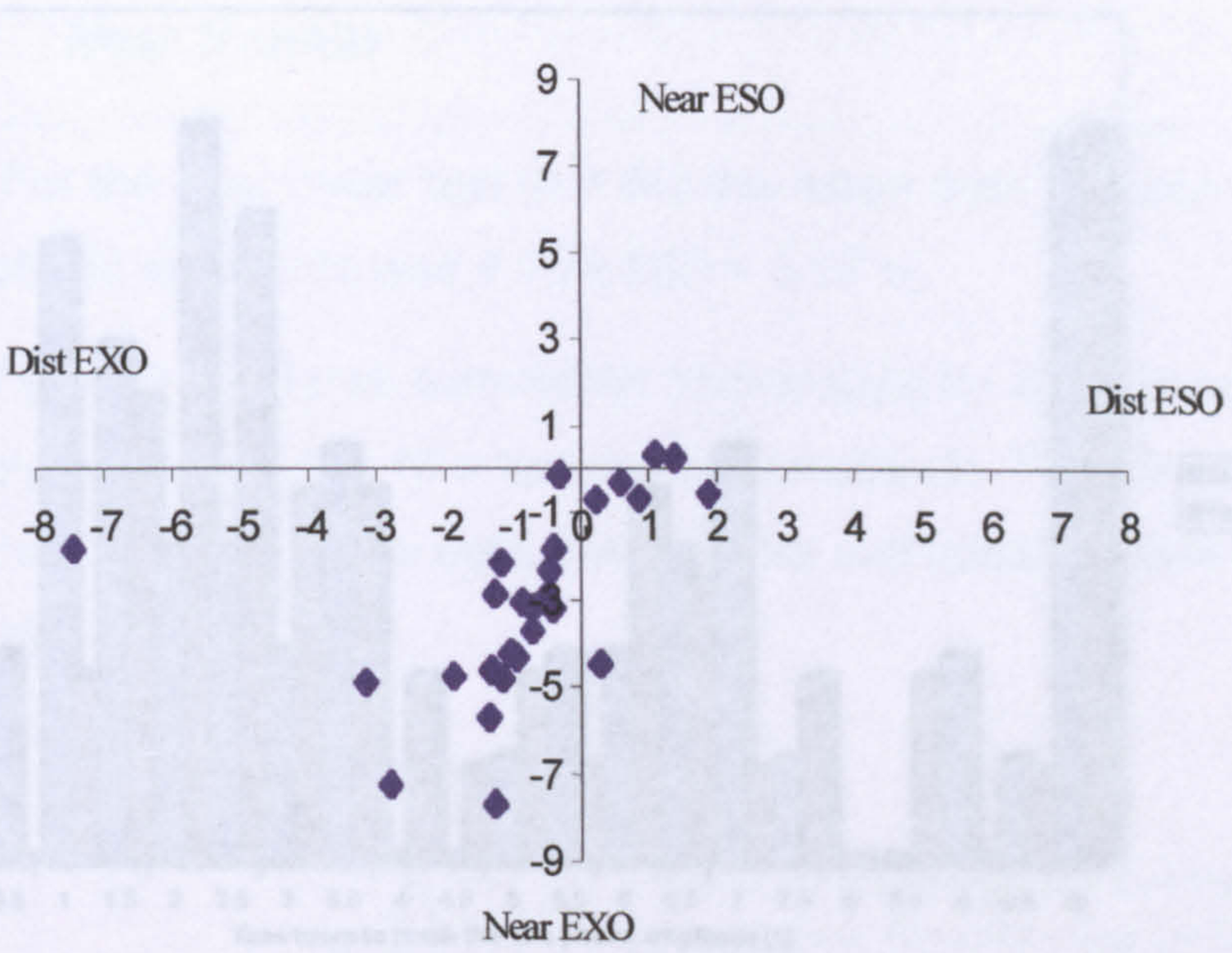


Figure 5.7. Graph showing the relationship between the amplitude^{RL} of distance and near phorias in the referred group.

V. Time taken to reach the 10 s amplitude

Relationship between the time taken to reach the 10 s amplitude for the right and left eye of each subject

As with the normal group, the referred group showed weak relationship between right and left eyes for the time to reach the phoria amplitude for both distance R^2 = 1 %; n = 24 subjects) and near (R^2 = 7%; n = 27 subjects).

Time taken to reach the 10 s amplitude

Figure 5.8 illustrates the frequency distributions of the time taken to reach the phoria amplitude measured following 10 s occlusion. As with the normal group, the distributions were not normal.

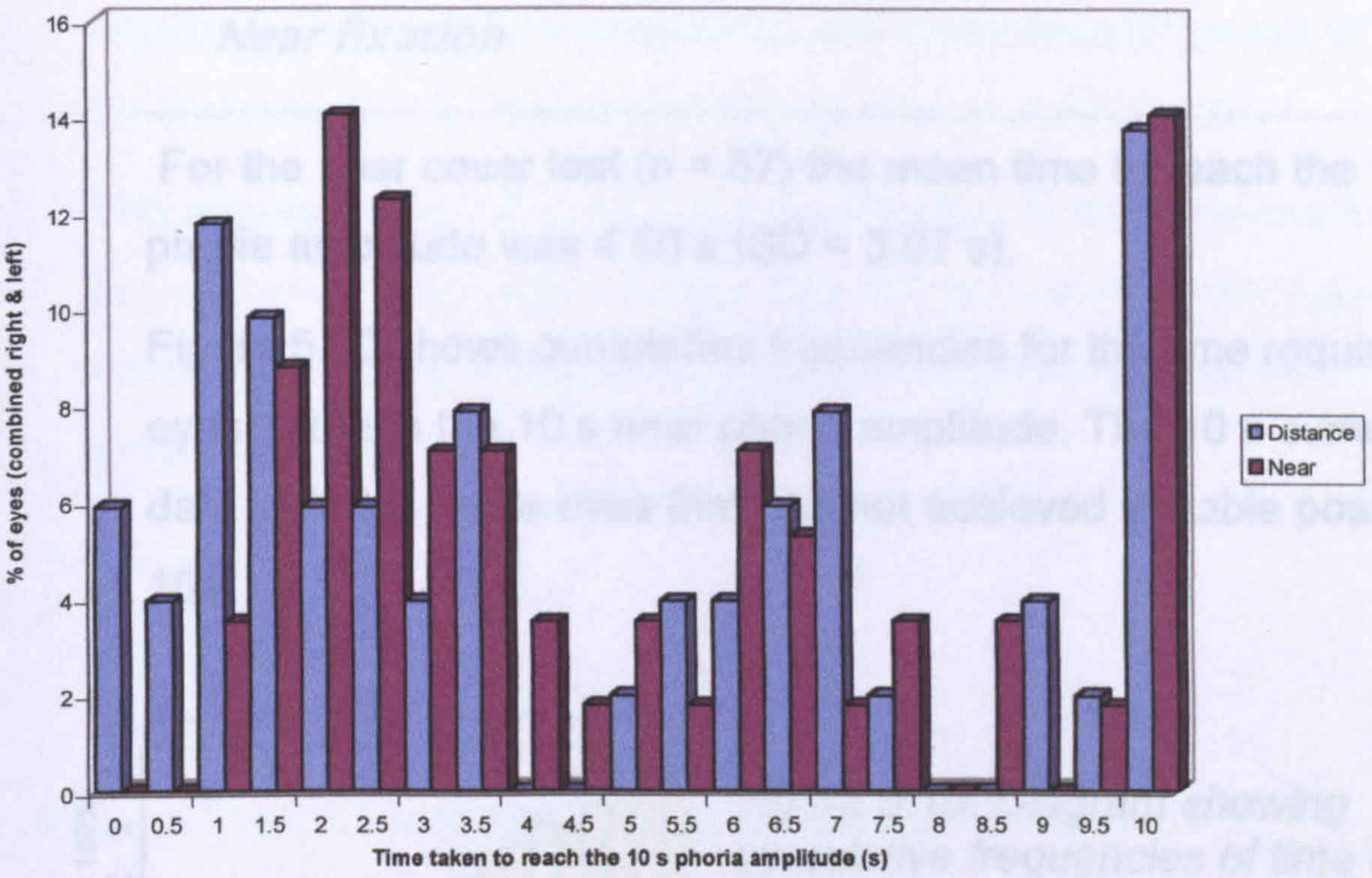


Figure 5.8. Frequency distributions of the time taken to reach the phoria amplitude measured following 10 s occlusion.

Distance fixation

For the distance cover test on the referred group (n = 51 eyes), the mean time to reach the 10 s phoria position was 4.25 s (SD = 3.42 s). Figure 5.9 shows the cumulative frequencies for the duration required to reach the 10 s phoria amplitude. The 10 s cumulative data includes eyes that had not achieved a stable position at 10 s.

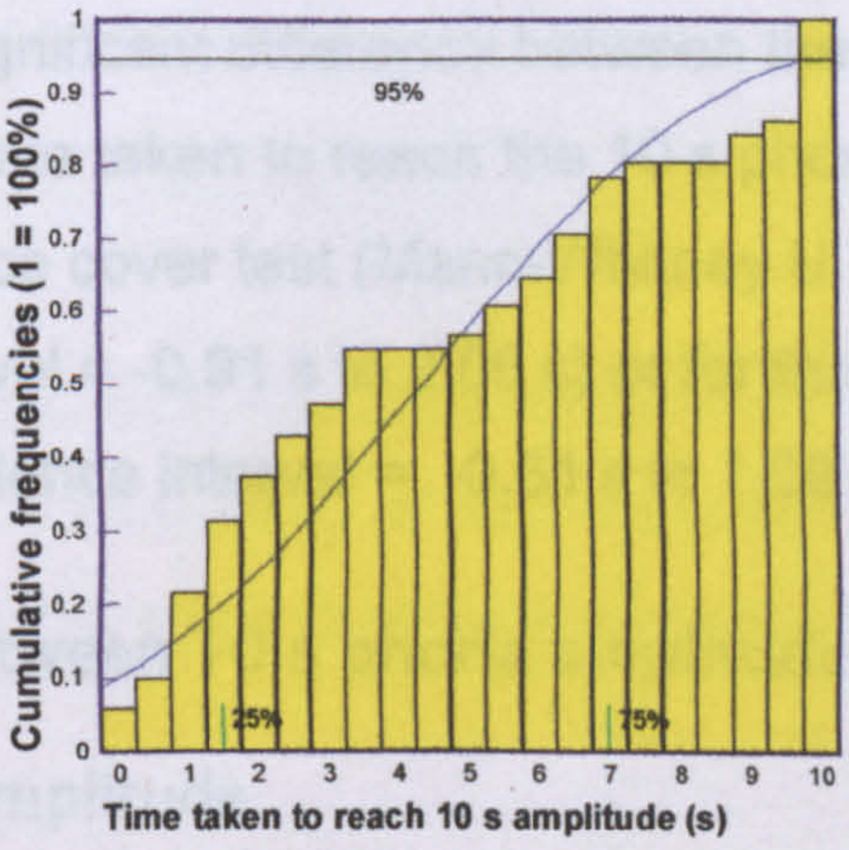


Figure 5.9. Diagram showing cumulative frequencies of time taken to reach 10 s distance phoria amplitude during the distance cover test. The blue line shows the cumulative normal distribution curve. The 95% frequency line is shown.

Near fixation

For the near cover test (n = 57) the mean time to reach the 10 s phoria amplitude was 4.50 s (SD = 3.07 s).

Figure 5.10 shows cumulative frequencies for the time required for eyes to reach the 10 s near phoria amplitude. The 10 s cumulative data includes those eyes that had not achieved a stable position at 10s.

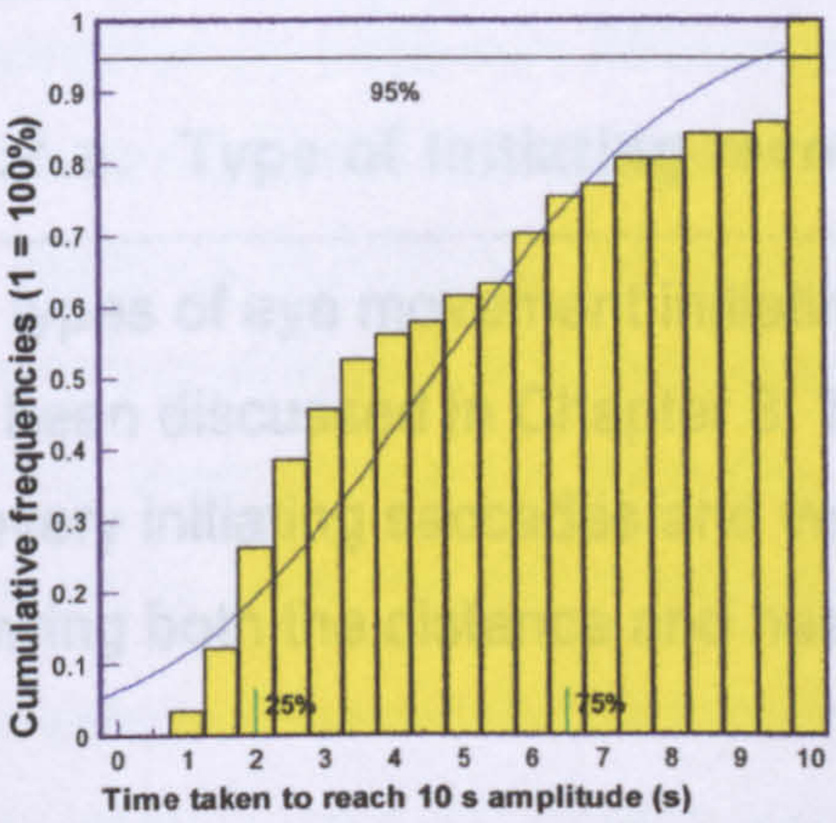


Figure 5.10. Diagram showing cumulative frequencies of time taken to reach 10 s near phoria amplitude. The blue line shows the cumulative normal distribution curve. The 95% frequency line is shown.

Comparison of time taken to reach 10 s phoria amplitude (combined right and left eyes) between the normal and referred group

There was no significant difference between the normal and referred groups, for the time taken to reach the 10 s phoria amplitude position, for either the distance cover test (Mann-Whitney U Test p = 0.91; 95% confidence interval = -0.91 s to 2.06 s) or for the near cover test (p = 0.72; 95% confidence interval = -0.61 s to 1.08 s).

Correlation between 10 s phoria amplitude and the time taken to reach that amplitude

The relationship between phoria amplitude at 10 s (combined esophoria and exophoria) and the time taken for the eye to reach that position was investigated. The percentage variability that can be attributed to an association between the size of phoria and the time taken to reach that

amplitude for combined esophores and exophores (Pearson R^2) was 7% for distance (n = 51 eyes) and 1% for near (n = 57 eyes).

The results for the referred group confirm the finding observed for the normal group that larger phorias may not necessarily take longer to reach their resting point during dissociation when compared to smaller amplitude phorias.

5.4.2 The recovery phase

5.4.2.a. Type of initiating recovery eye movement

The types of eye movement initiating recovery following the near cover test has been discussed in Chapter 3. Table 5.1 lists the relative frequencies of recovery initiating saccades and vergence amongst esophores and exophores following both the distance and near cover tests.

Distance (n = 39)				**Near (n = 56)**			
Esophoria (n = 10)		***Exophoria (n = 29)***		***Esophoria (n = 2)***		***Exophoria (n = 54)***	
Saccade	*Vergence*	*Saccade*	*Vergence*	*Saccade*	*Vergence*	*Saccade*	*Vergence*
50.0%	50.0%	13.8%	82.2%	50.0%	50.0%	29.6%	70.4%

Table 5.1. Relative frequencies of type of eye movement initiating recovery in the referred group.

For near exophoric eyes there was a similarity to the results of the normal group (see Table 3.4). The number of near esophoric eyes for which data could be obtained was too small to provide statistical inference.

5.4.2.b. Latency

I. Distribution

Latency data was measured whenever possible. Of the potential measurements it was possible to determine 89 latencies for eyes (combined right and left) from the combined distance and near data. Of these measurements, 38 related to distance and 51 to near.

The mean latency of the first recovery movement for all data that could be extracted (n = 89) was 290 ms (range 80 ms to 6210 ms; SD = 660 ms). The eye that showed a 6000 ms latency was the right eye of a subject with a distance exophoria. This particular measurement describes an eye with a manifest strabismus at the commencement of the eye movement recording. If this measurement is excluded then the mean latency was 230 ms (range 80 ms to 1480 ms; SD = 190 ms).

Analysis of the results for the distance cover test only (excluding the 6000 ms latency) (n = 37) gave a mean latency of 240 ms (range 80 ms to 1480 ms; SD = 240 ms). For the near cover test (n = 51) the mean latency was 210 ms (range 80 ms to 1060 ms; SD = 150 ms).

Figure 5.11 illustrates the frequency distribution of these latencies for distance and near.

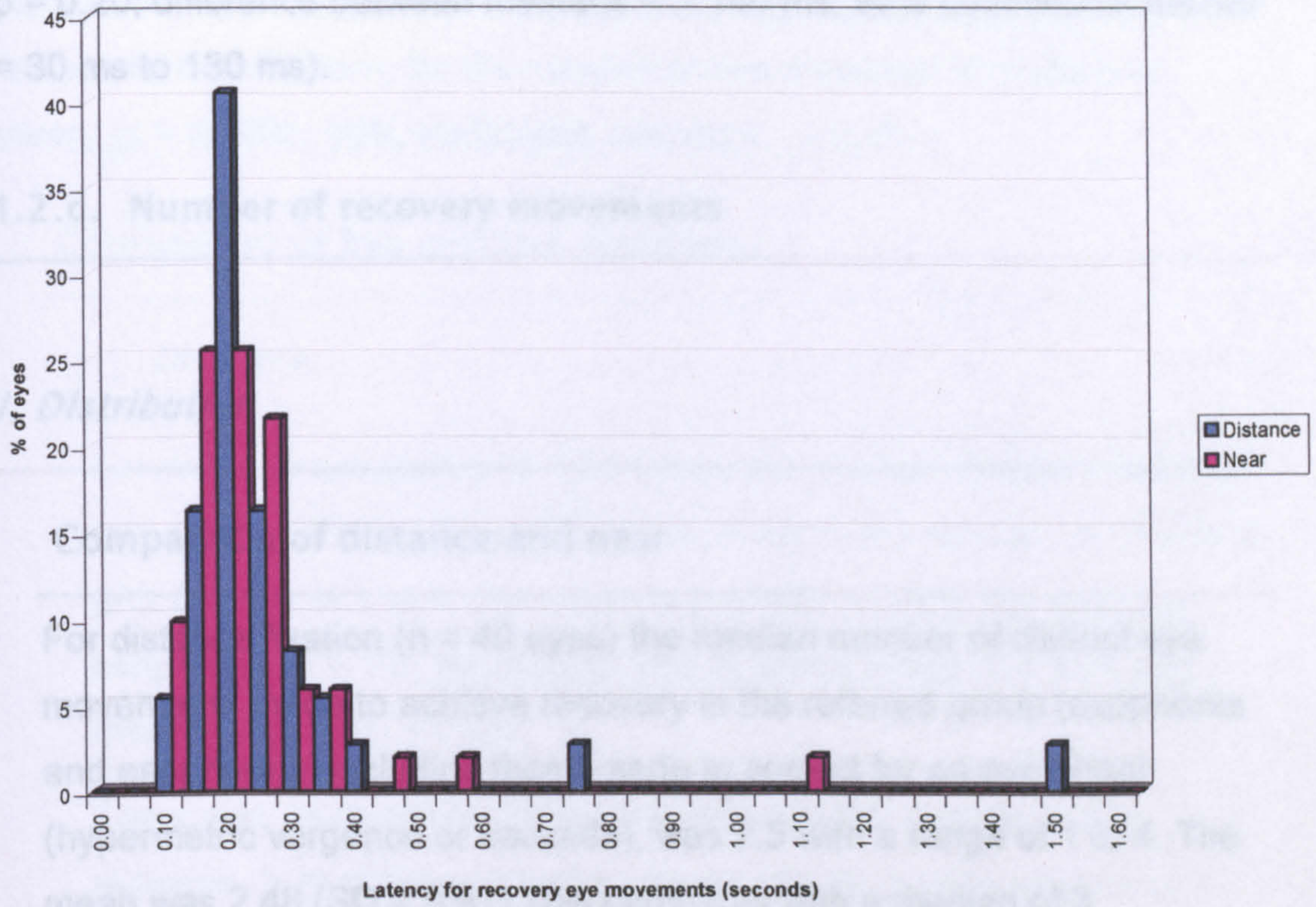


Figure 5.11. Frequency distributions (%) for latency of recovery movement following removal of the occluder for all eyes in the referred group for distance and near. An outlying distance latency was omitted.

There was no significant difference between distance and near latencies (Mann-Whitney U Test p = 0.34; difference between medians = 20 ms; 95% confidence interval = -20 ms to 0.50 ms).

II. Correlation of Eso and Exo deviations

A Mann-Whitney U Test was carried out to compare latencies for all exophores (distance and near) (n = 80) compared to all esophores (n = 13). There was no significant difference between groups p = 0.69; difference between medians = -10 ms; 95% confidence interval = -60 ms to 40 ms).

III. Comparison with the normal group

A Mann-Whitney U Test was carried out to compare latencies for all available data for subjects in the referred group (distance and near) (n = 88) with all available data for the subjects in the normal group (n = 185). The three cases of abnormally long latencies alluded to previously for both groups were excluded. There was no significant difference between groups

p = 0.20; difference between medians = - 140 ms; 95% confidence interval = 30 ms to 130 ms).

5.4.2.c. Number of recovery movements

I. Distribution

Comparison of distance and near

For distance fixation (n = 40 eyes) the median number of distinct eye movements made to achieve recovery in the referred group (exophores and esophores), including those made to correct for an overshoot (hypermetric vergence or saccade), was 2.5 with a range of 1 to 4. The mean was 2.48 (SD = 0.91) This compares with a median of 3 movements following the near cover test. The range for near was from 1to 8 with a mean of 3.59 (SD = 1.60; n = 51 eyes). These distributions are illustrated in Figure 5.12.

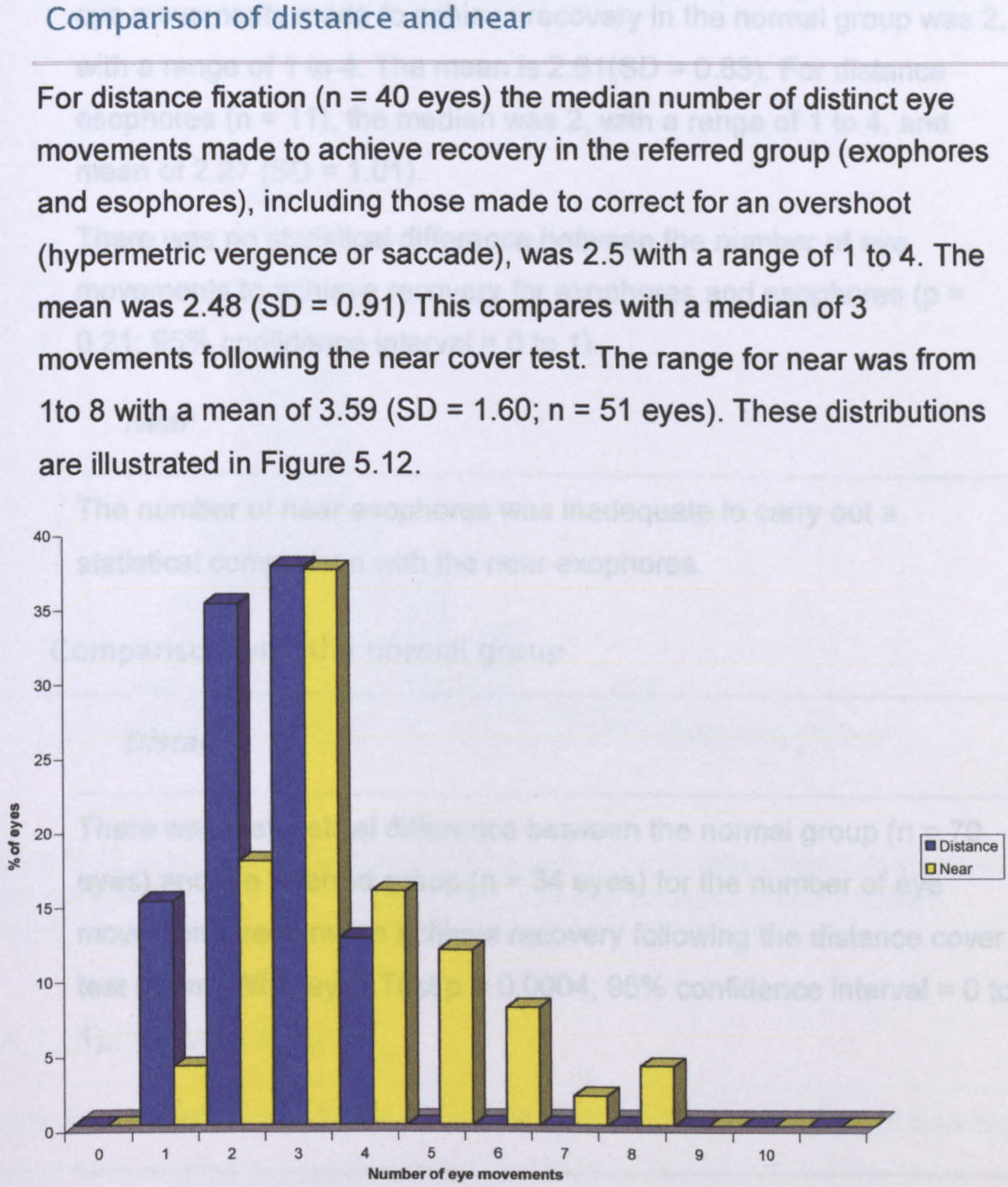


Figure 5.12. Frequency distributions of number of eye movements to achieve recovery following the automated cover test for distance and near fixation.

A Mann-Whitney U Test showed a statistically significant difference between distance and near fixation, for the number of eye movements to achieve recovery (p = 0.0002; 95% confidence interval = -1 to 0).

Comparison of Eso and Exo deviations

Distance

For distance exophores (n = 28 eyes), the median number of distinct eye movements made to achieve recovery in the normal group was 2, with a range of 1 to 4. The mean is 2.61(SD = 0.83). For distance esophores (n = 11), the median was 2, with a range of 1 to 4, and mean of 2.27 (SD = 1.01).

There was no statistical difference between the number of eye movements to achieve recovery for exophores and esophores (p = 0.21; 95% confidence interval = 0 to 1).

Near

The number of near esophores was inadequate to carry out a statistical comparison with the near exophores.

Comparison with the normal group

Distance

There was a statistical difference between the normal group (n = 70 eyes) and the referred group (n = 34 eyes) for the number of eye movements required to achieve recovery following the distance cover test (Mann-Whitney U Test p = 0.0004; 95% confidence interval = 0 to 1).

Near

There was a statistically significant difference between the normal group (n = 115 eyes) and the referred group (n = 51 eyes) for the number of eye movements required to achieve recovery following the

near cover test (Mann Whitney p = 0.0002; 95% confidence interval = 0 to 1).

Figure 5.13 illustrates the frequency distributions of the two near groups.

For near exophores only, there was a significant difference between the normal group (n = 96 eyes and the referred group (n = 48 eyes) for the number of eye movements required to achieve recovery (Mann-Whitney U test p = 0.002; 95% confidence interval = 0 to 1 movement).

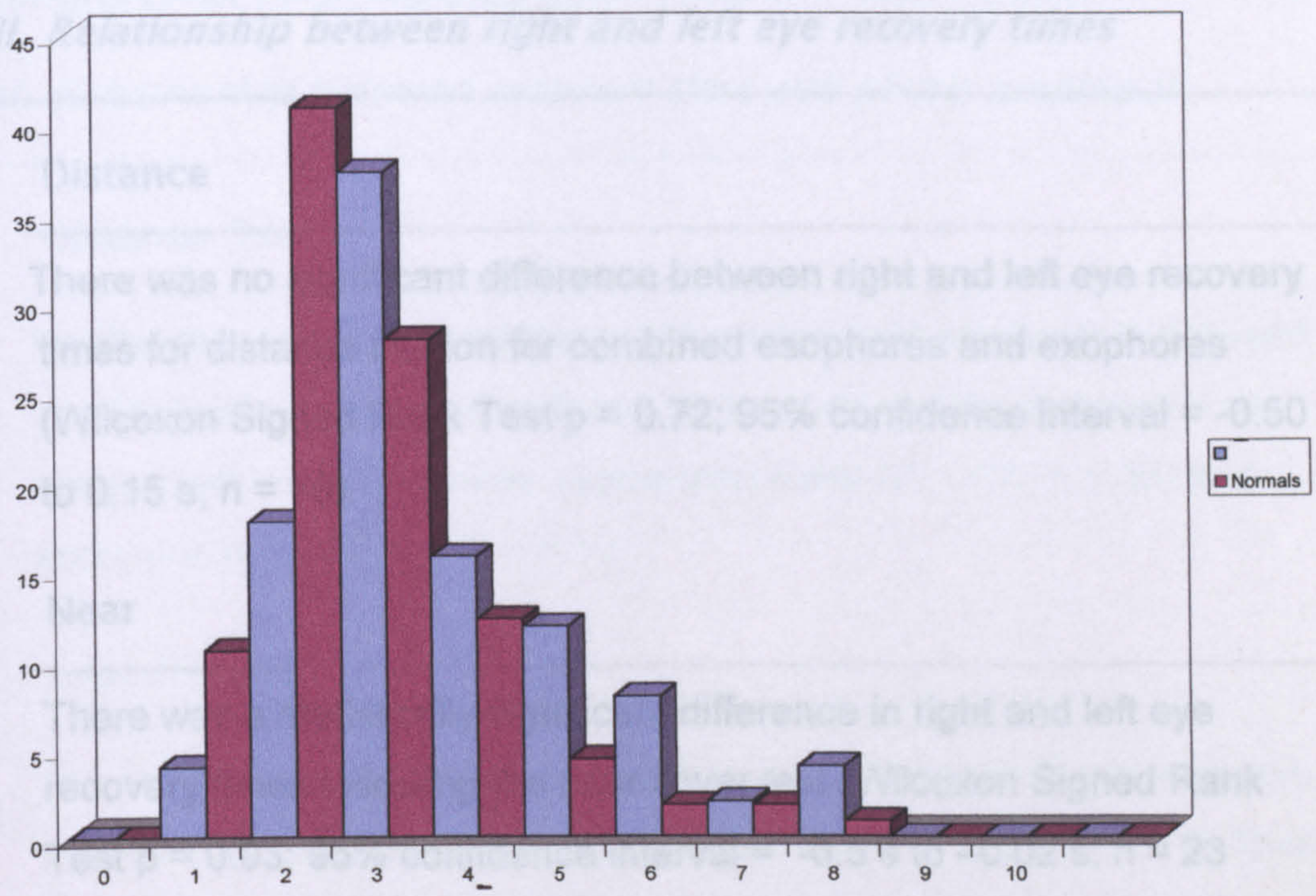


Figure 5.13. Frequency distributions of number of eye movements to achieve recovery following the near automated cover test for normal and referred groups.

5.4.2.d. Recovery time

There was one case of a left eye of an exophoric subject that failed to take up fixation following the removal of the cover. This eye was excluded from the statistical data in terms of recovery time.

I. Distance fixation

The mean recovery time for all eyes (n = 45) following the distance cover test was 1.00 s (SD = 1.17 s). The minimum time for recovery was 0.17 s and the maximum was 6.57 s.

II. Near fixation

The mean recovery time for all eyes (n = 53) following the near cover test was (SD = 1.16 s). The minimum time for recovery was 0.31 s and the maximum was 6.67 s.

III. Relationship between right and left eye recovery times

Distance

There was no significant difference between right and left eye recovery times for distance fixation for combined esophores and exophores (Wilcoxon Signed Rank Test p = 0.72; 95% confidence interval = -0.50 s to 0.15 s; n = 19).

Near

There was a statistically significant difference in right and left eye recovery times following the near cover test (Wilcoxon Signed Rank Test p = 0.03; 95% confidence interval = -0.5 s to –0.02 s; n = 23 subjects).

Figure 5.14 further illustrates the relationship between right and left recovery times following the near automated cover test.

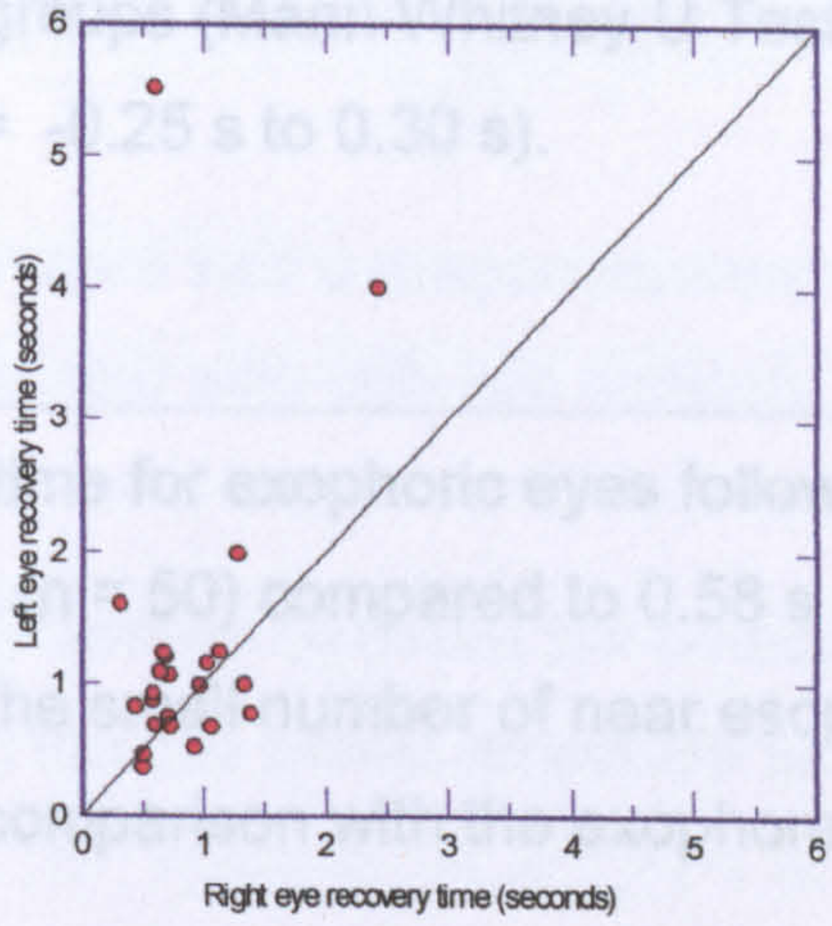


Figure 5.14. Relationship between right and left recovery times (seconds) following the near cover test (Spearman Rank R^2 = 3%; n = 23).

IV. Relationship between recovery time and phoria amplitude

Distance fixation

For the distance cover test there was a very weak relationship between amplitude of phoria and time taken for recovery for the distance cover test for both exophoric eyes (Spearman Rank R^2 = 4%; n = 33) and esophoric eyes (R^2 = 4%; n = 12)

Near fixation

The relationship between recovery time and phoria amplitude was also very weak for near exophores (Spearman Rank R^2 = 8%; n = 51). There was recovery time data available for only three near esophoric eyes and this was insufficient for a statistical test.

V. Comparison of recovery times for Eso and Exo deviations

Distance fixation

The mean recovery time for exophoric eyes (n = 33) following the distance cover test was 0.93 s (SD = 1.08) compared to 1.18 s (SD = 1.43) for distance esophores (n = 12). There was no significant

difference between groups (Mann Whitney U Test p = 0.49; 95% confidence interval = -0.25 s to 0.30 s).

Near fixation

The mean recovery time for exophoric eyes following the near cover test = 1.17 s (SD = 1.18) (n = 50) compared to 0.58 s (SD = 0.23 s) for near esophores (n = 3). The small number of near esophores did not lend itself to a statistical comparison with the exophores.

VI. Comparison of recovery times between the normal and referred group

Distance

There was no significant difference in recovery times between the normal and referred groups following the distance cover test (Mann-Whitney U Test p = 0.15; 95% confidence interval = -0.18 s to 0.03 s).

Near

There was a borderline difference in recovery times between groups for normal exophoric eyes (n = 124) and referred exophoric eyes (n = 50) for the near cover test (Mann-Whitney U Test p = 0.09; 95% confidence interval = -0.01 to 0.24) with the referred group being slower.

5.5 Summary of results

As with the normal group, there was no significant difference between right and left eye phoria amplitudes for either distance or near.

There was a statistically significant difference in near phoria amplitudes between the referred population and the normal group. Although phoria amplitude itself was not a criterion for referral of a subject to the study, it is hypothesised that the presence of a large amplitudes of phoria was likely to

raise the index of suspicion that a subject's symptoms were correlated with an ocular motor anomaly.

Among the referred group there was a preponderance of subjects with exophoria for distance and near with only two subjects manifesting esophoria for distance and near.

There was no significant difference between the normal and referred groups for the time taken to reach the 10s phoria amplitude for position for either distance or near.

As with the normal group, vergence eye movements were more prevalent than saccades as the initiating recovery eye movement amongst exophores for near.

There was no significant difference between the normal and referred groups for latency of recovery for distance and near.

There was a statistically significant difference between distance and near fixation, for the number of eye movements to achieve recovery.

It is likely that a recovery made up of multiple eye movements would appear to an observer viewing the eyes subjectively during the conventional cover test, as a '*jerky*' recovery. It was therefore of interest to note that there was a statistical difference between the normal group and the referred group, for the number of eye movements required to achieve recovery following both the distance and near cover tests.

There was only a very weak relationship between phoria amplitude and recovery time.

There was no significant difference in recovery times between the normal and referred groups following the distance cover test. However, there was a difference following the near cover test ($p = 0.09$).

Important limitations of this phase of the study were that the subjects were selected and made up a somewhat diverse group. Nevertheless it was of interest to study the characteristics of these mostly symptomatic subjects.

Chapter 6. The relationship between symptom scores and ocular motor characteristics

The aim of this of these analyses was to determine any correlation between symptom scores for near vision and ocular motor characteristics including eye movement characteristics during the near cover test.

In Chapter 4 it was shown that some of the subjects who described themselves as being asymptomatic when recruited, reported a range of near vision related symptoms when invited to complete a symptom questionnaire.

Data from the 'normal' group were therefore combined with the data for the referred group in order to investigate the relationship between eye movement characteristics during the cover test and symptoms.

Figure 6.1 illustrates the frequency distribution of symptom scores for the combined groups.

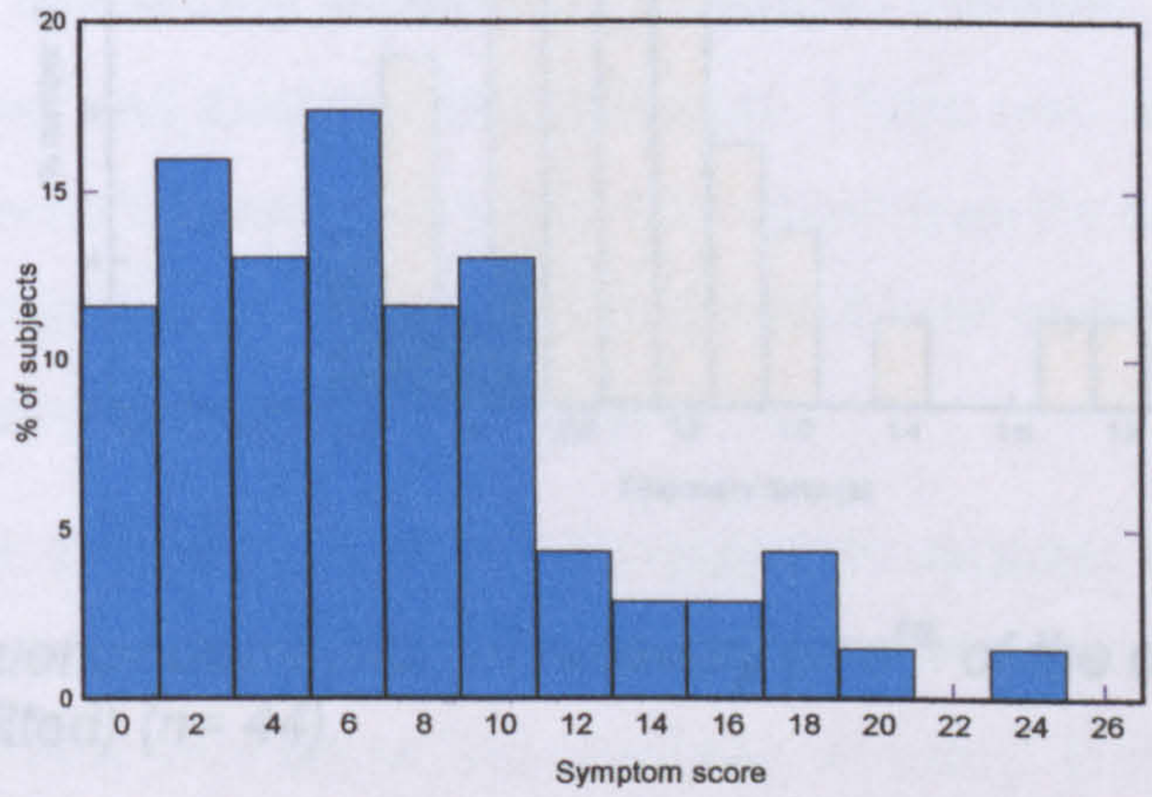


Figure 6.1. Frequency distribution of symptom scores for the combined groups (n = 69 subjects).

Caution must be exercised when interpreting all analyses presented in this chapter because of the fact that the subjects were drawn from two distinct and selected groups rather than from randomly selected subjects.

6.1 Symptom scores and recovery time

6.1.1 Average of right and left eye recovery times (recovery time^{RL})

To investigate the relationship between symptoms and recovery time the average of right and left recovery times for the combined groups were analysed. Data was excluded for those subjects for whom data for one eye was missing. The average recovery time^{RL} was 0.93 s (n = 44; SD 0.57 s). The range was from 0.32 s to 3.21 s. Figure 6.2 illustrates the frequency distribution of recovery time^{RL} with two outlying data omitted (3.21 s and 3.06 s). It should also be noted one subject who failed to make recovery movement with one eye was also omitted for all the following recovery time analyses.

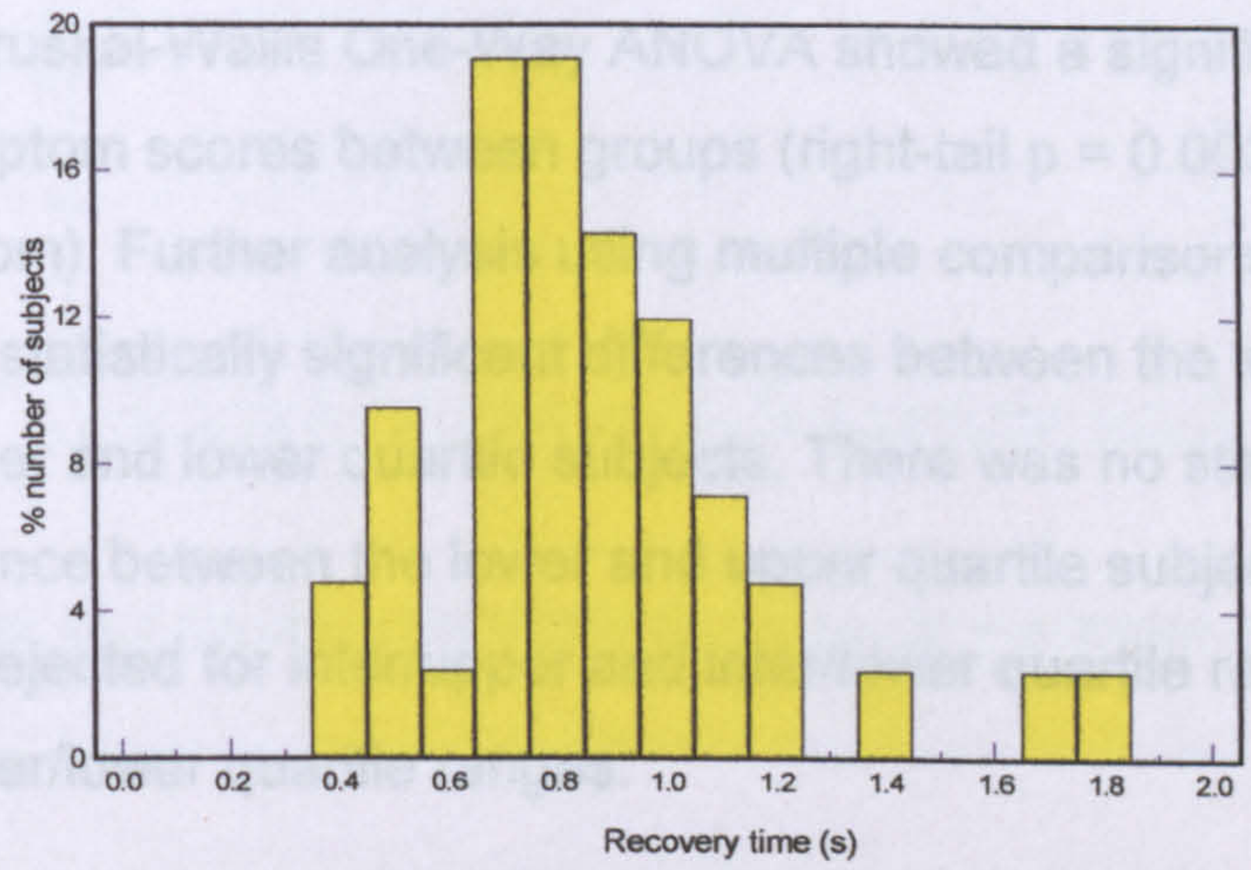


Figure 6.2. Frequency distribution of recovery time^{RL} of the combined groups (two outliers omitted) (n= 44).

There was no linear relationship between recovery time^{RL} and symptom score (Pearson R^2 = < 1%). Figure 6.3 shows symptom scores plotted against recovery times^{RL}.

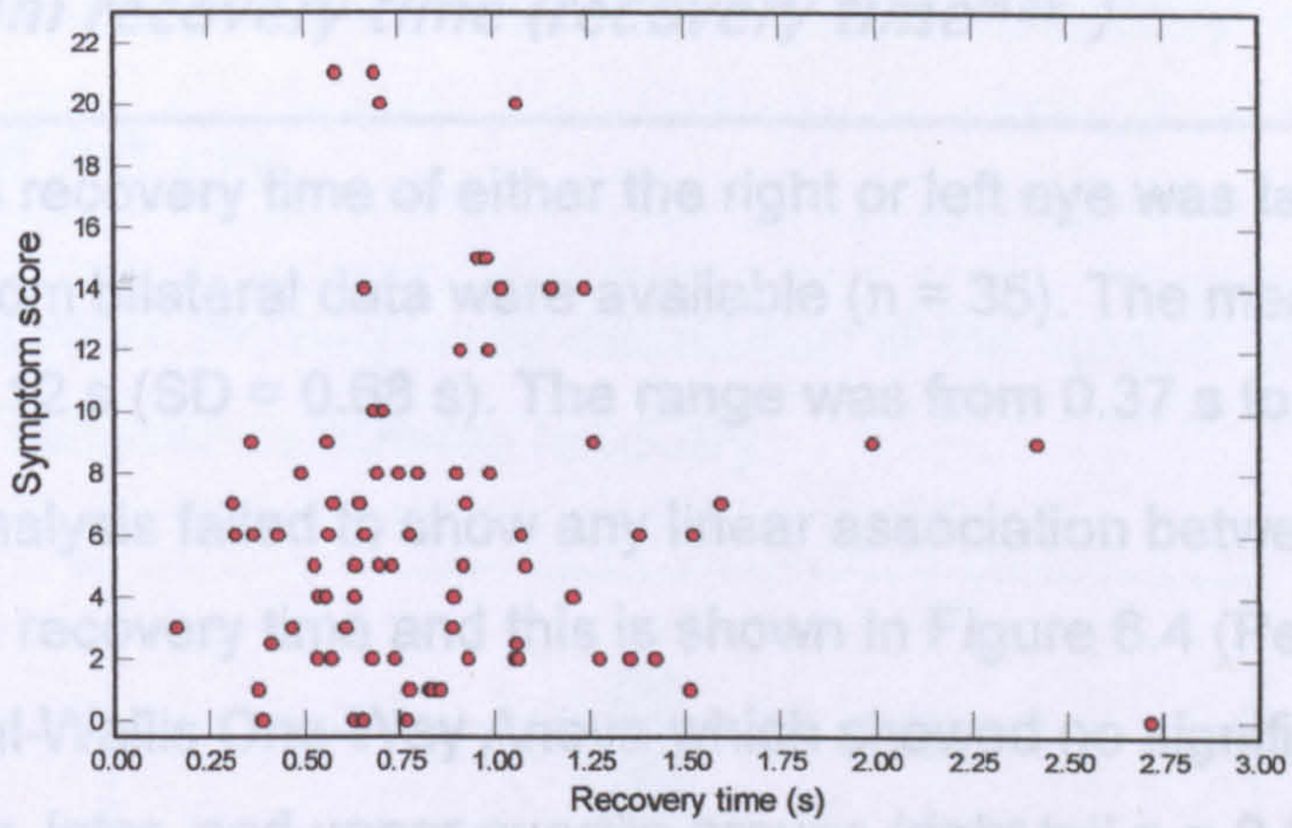


Figure 6.3. Scatter plot showing distribution of symptom scores as a function of recovery time.

Symptom scores were grouped into three categories based upon the lower quartile n = 11), upper quartile (n = 11), and interquartile (n = 22) recovery time^{RL} data. A Kruskal-Wallis One-Way ANOVA showed a significant difference in symptom scores between groups (right-tail p = 0.003 for 2 degrees of freedom). Further analysis using multiple comparisons (95% Dunn interval) showed statistically significant differences between the inter-quartile and both the upper and lower quartile subjects. There was no statistically significant difference between the lower and upper quartile subjects. The null hypothesis was rejected for inter/upper and inter/lower quartile ranges but accepted for upper/lower quartile ranges.

These results are interesting in that they appear to suggest that subjects who manifest a “fast recovery” or a “slow recovery”, are likely to suffer from less symptoms than those who show “intermediate recovery” times of between approximately 0.6 and 1.0 s following the cover test.

One hypothesis to explain these findings is that the binocular visual system of subjects showing particularly longer recovery times following removal of the cover, may have a strategy for producing suppression thereby avoiding symptoms during periods of visual stress. However, an argument against this hypothesis was that the author found no evidence for such a strategy in terms of the near Mallett unit binocularity test.

6.1.2 Maximum recovery time (recovery time^{max})

The maximum recovery time of either the right or left eye was taken for each subject for whom bilateral data were available (n = 35). The mean recovery time^{max} was 1.12 s (SD = 0.68 s). The range was from 0.37 s to 4.00 s.

Regression analysis failed to show any linear association between symptoms and maximum recovery time and this is shown in Figure 6.4 (Pearson R^2 = < 1%). A Kruskal-Wallis One-Way Anova which showed no significant difference between lower, inter, and upper quartile groups (right tail p = 0.65).

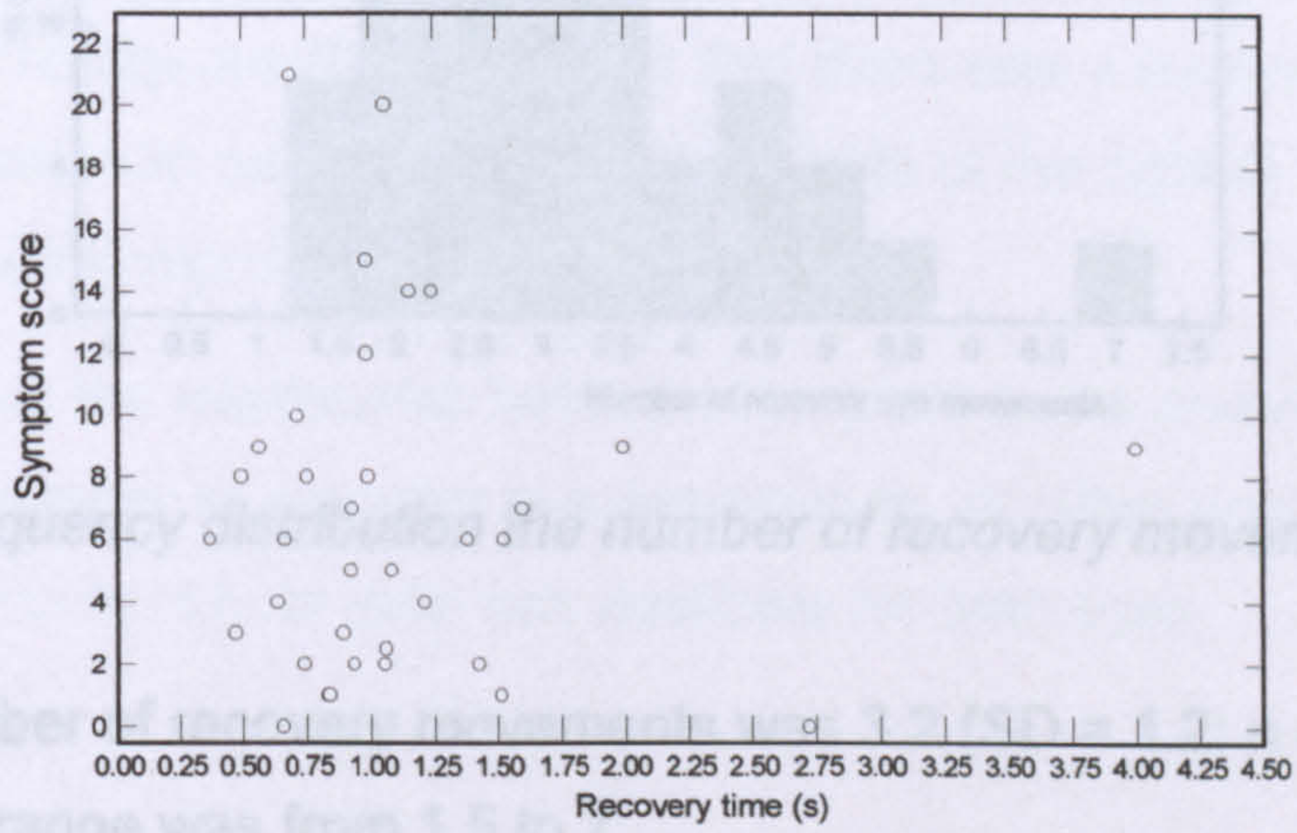


Figure 6.4. Scatter plot showing relationship between symptom score and recovery time^{max}.

6.2 Symptom scores and number of eye movements to achieve recovery

It is often stated that a multi-stage recovery following removal of the cover is indicative of a poorly compensated phoria which may be associated with symptoms.

Symptom scores were examined in relation to the average of the right and left eye number of eye movements required to achieve recovery for each subject. Subjects were included in the analysis only if recovery movement data were available for both eyes.

6.2.1 Number of eye movements to achieve recovery - average of right and left eye

Figure 6.5 illustrates the frequency distribution of the number of recovery movements^{RL} required to achieve recovery.

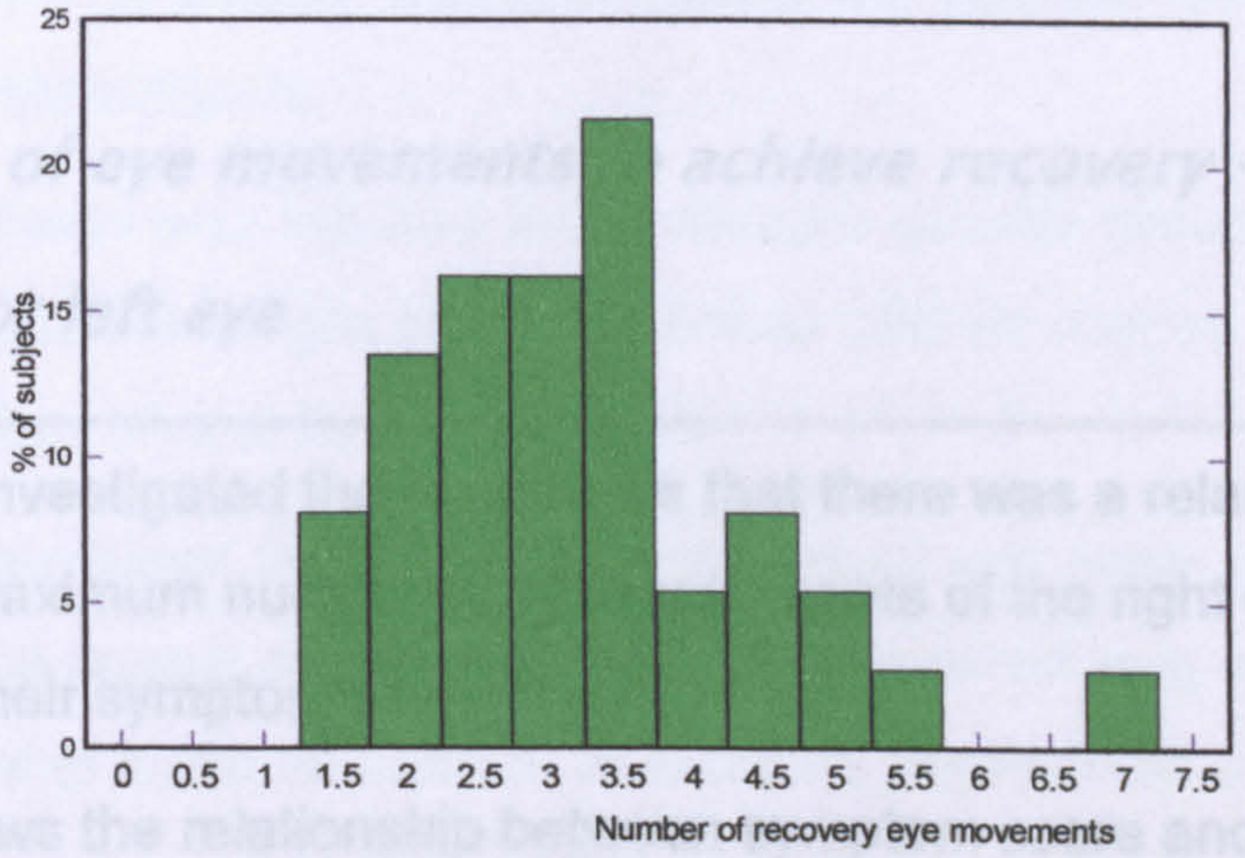


Figure 6.5. Frequency distribution the number of recovery movements^{RL} (n = 37 subjects).

The mean number of recovery movements was 3.2 (SD = 1.2; n = 37 subjects). The range was from 1.5 to 7.

Figure 6.6 shows the very weak linear relationship between symptom scores and the number of recovery eye movements (Pearson R^2 = 10%).

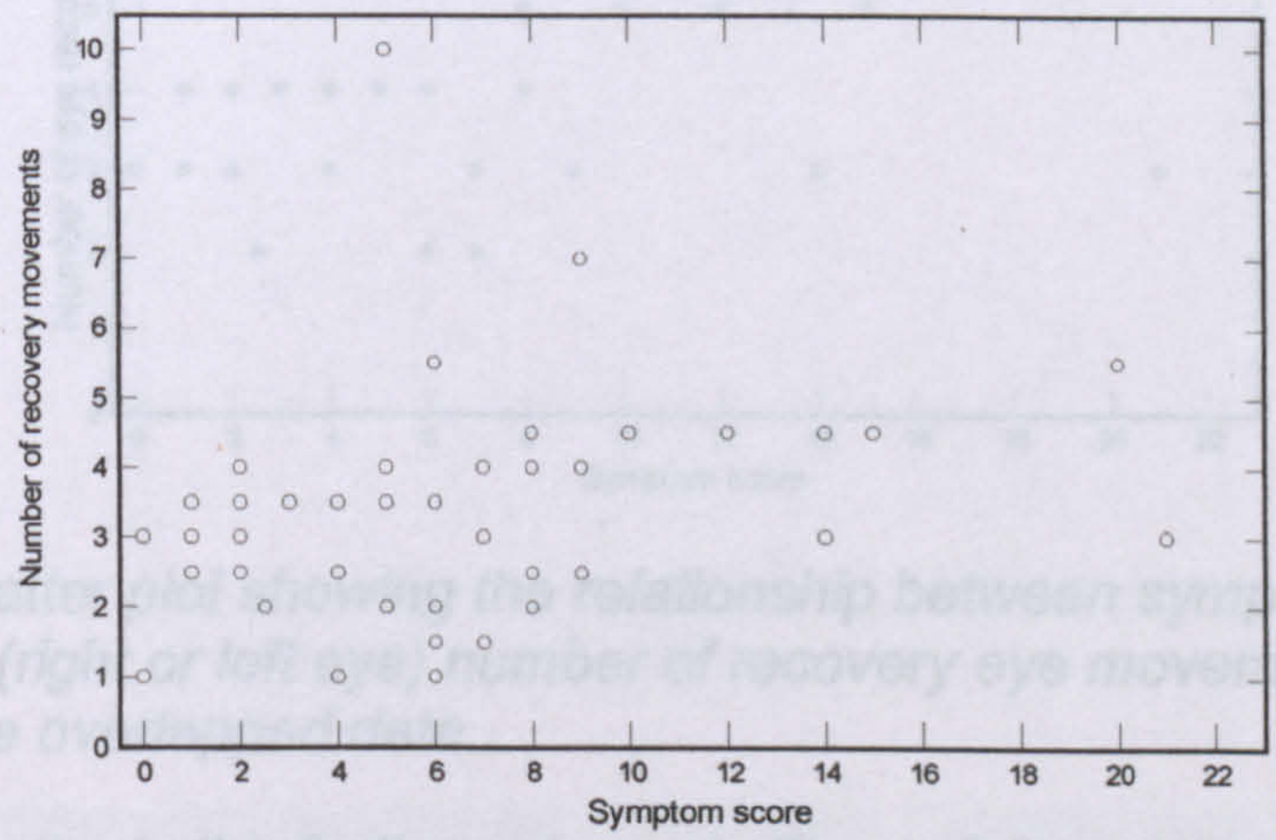


Figure 6.6. Scatter plot showing the relationship between symptom scores and the number of eye movements to achieve recovery. Some points describe overlapped data.

To confirm the apparent lack of association between symptom score and the number of recovery movements, symptom scores were grouped into three categories based upon the lower quartile n = 8), upper quartile (n = 9), and interquartile (n = 20) number of recovery eye movement^{RL} data. There was no difference in symptom score between groups (Kruskal-Wallis One-Way ANOVA right tailed p = 0.71 for 2 degrees of freedom).

6.2.2 Number of eye movements to achieve recovery - the greater of right or left eye

This analysis investigated the hypothesis that there was a relationship between the maximum number of eye movements of the right or left eye of subjects and their symptom scores.

Figure 6.7 shows the relationship between symptom score and the greater of the maximum number of recovery eye movements, of either right or left eye, for those subjects for whom data was available for both eyes.

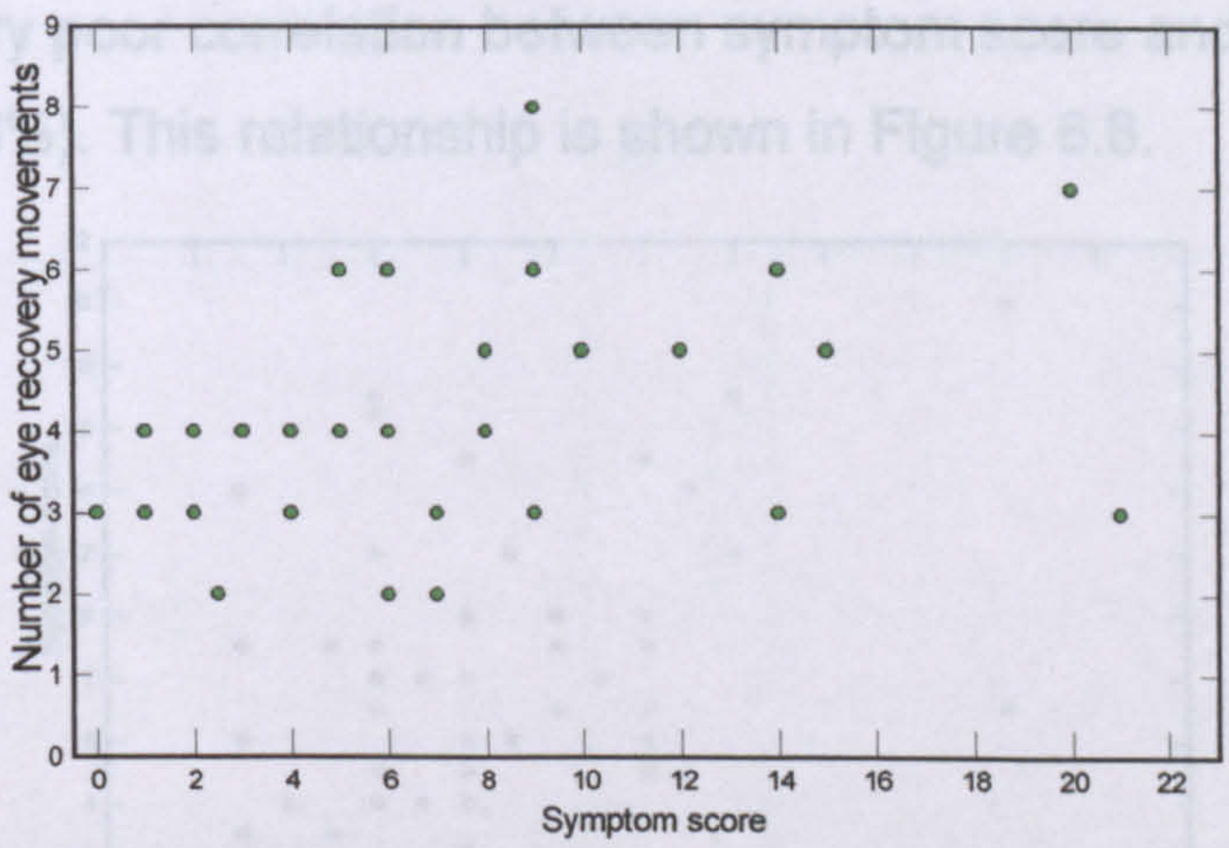


Figure 6.7. Scatter plot showing the relationship between symptom score and the maximum (right or left eye) number of recovery eye movements. Some points describe overlapped data.

To further investigate the findings shown in Figure 6.7, symptom scores were grouped into three categories based upon the lower quartile (2 movements; n = 6), inter-quartile (3 or 4 movements; n = 23) and upper quartile (5 or more movements; n = 8) number of recovery eye movement^{max} data.

A Kruskal-Wallis One-Way ANOVA showed a significant difference between groups (right-tail p = 0.02 for 2 degrees of freedom).

Further analysis using multiple comparisons (95% Dunn interval) showed a statistically significant difference between the interquartile and the upper quartile subjects. There was neither a statistically significant difference between the lower and upper quartile subjects nor between the interquartile and lower quartile subjects.

The null hypothesis was rejected for inter/upper quartile groups but accepted for inter/lower quartile ranges and upper/lower quartile ranges for the maximum number of recovery movements.

However, the combined findings of both maximum and mean number of recovery eye movement analyses did not provide convincing evidence of a strong relationship between number of recovery movements and symptom scores.

6.3 Symptom scores and NPC

There was a very poor correlation between symptom score and NPC (Pearson R^2 = 8%). This relationship is shown in Figure 6.8.

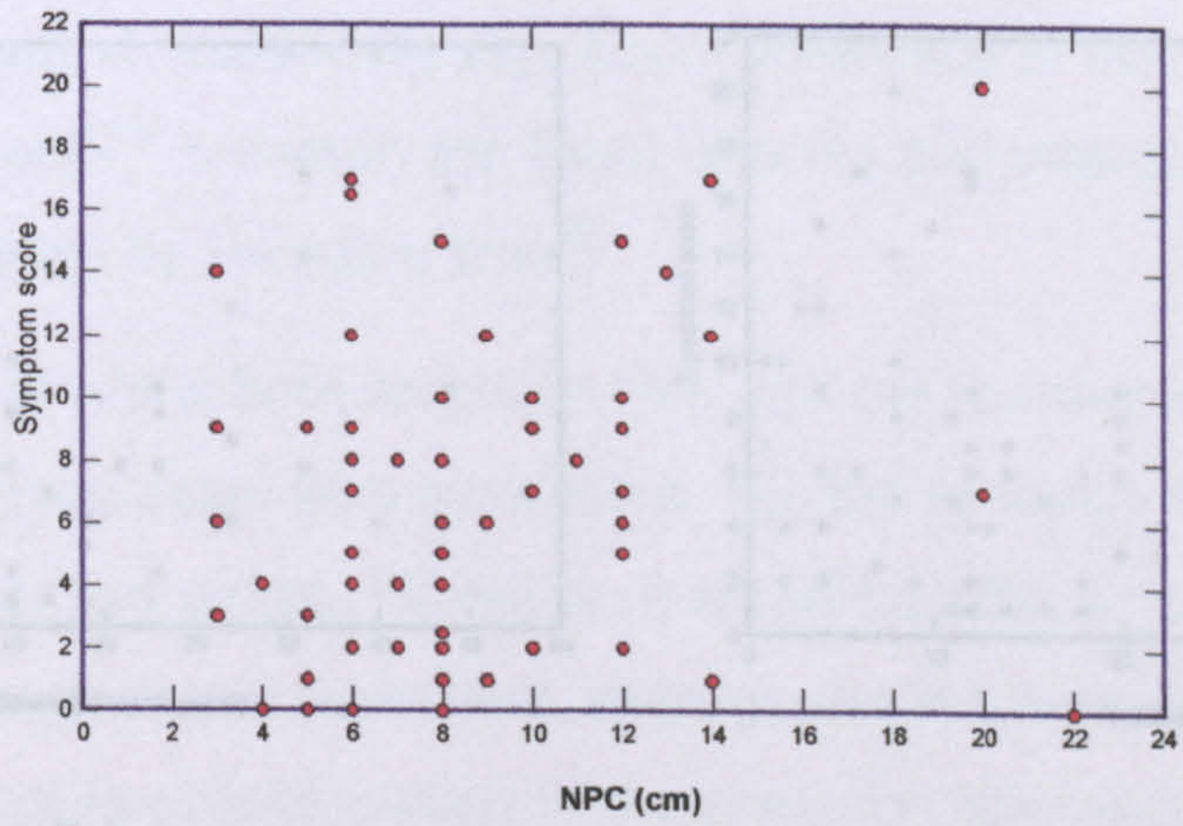


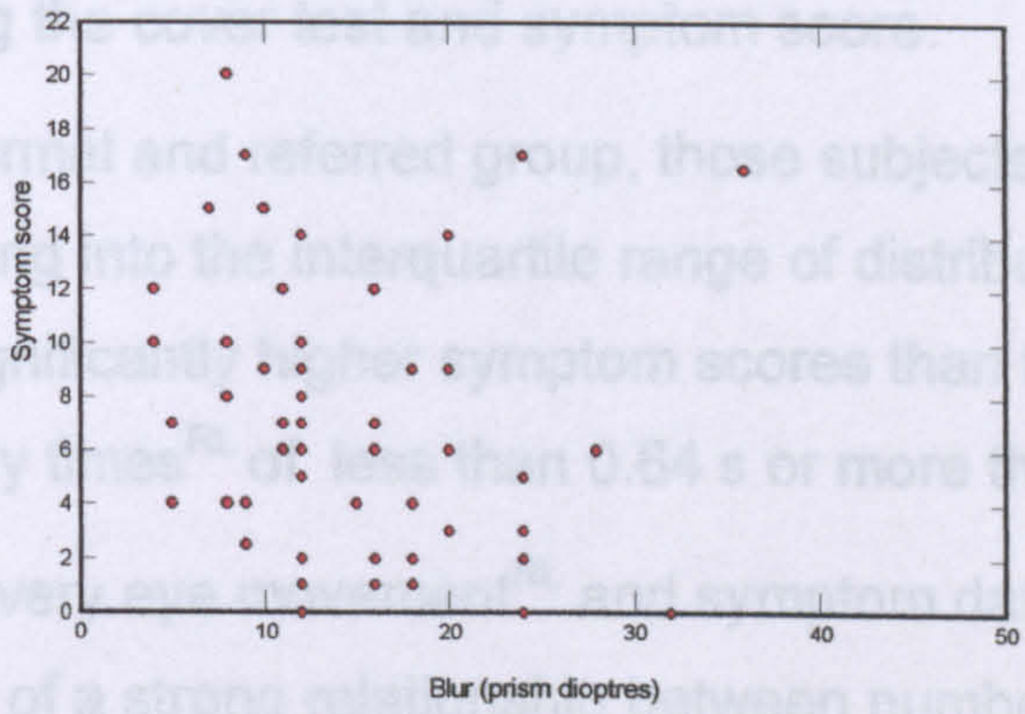
Figure 6.8. Scatter plot showing the relationship between symptom score and NPC.

An NPC of 10 cm or greater has often been associated with the diagnosis of 'convergence insufficiency' (see Chapter 1: Table 1.4). To test whether there was a difference in symptom score between subjects with NPCs of < 10 cm

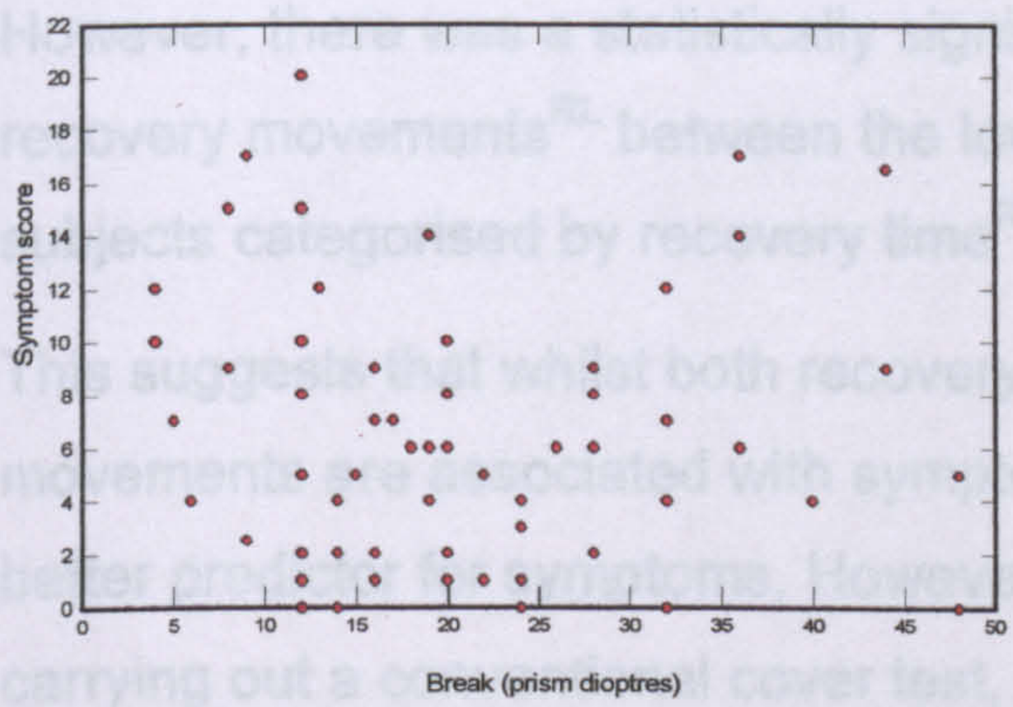
and those with ≥10 cm, subjects were organised into one group whose NPC were < 10 cm (n = 46) and a second group whose NPCs were ≥10 cm (n = 21). A Pooled Variance t–test showed a significant difference in symptom scores between groups (p = 0.005; 95% confidence interval = - 7 to – 1) with the 10 cm and greater group manifesting the higher mean symptom scores.

6.4 Symptom scores and fusional reserves

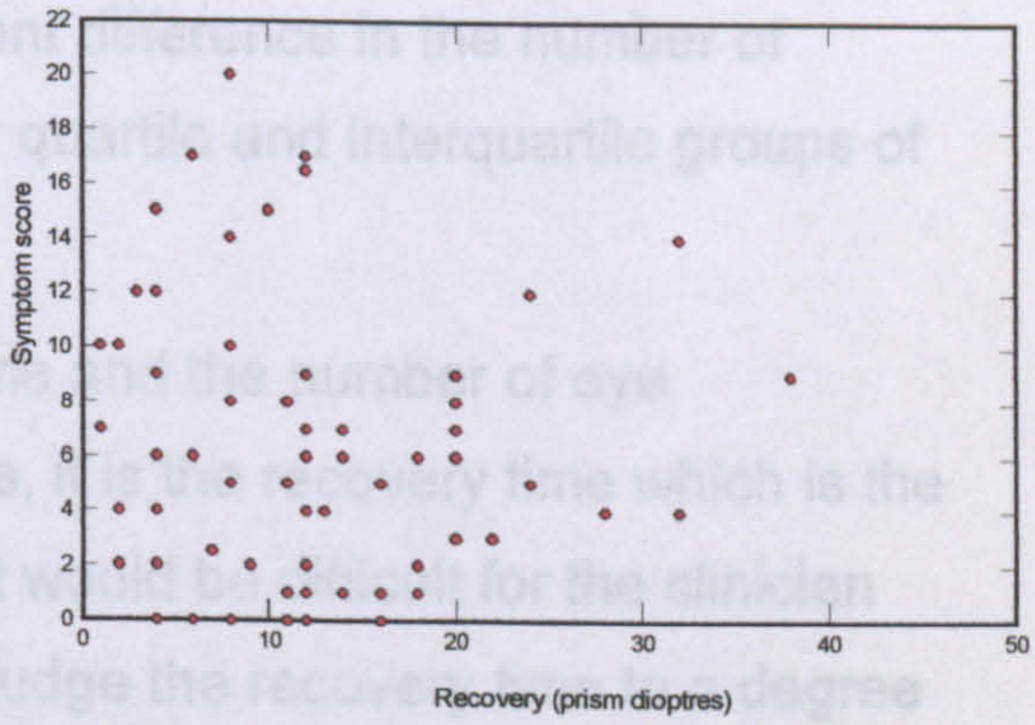
There was no linear relationship between symptom scores and each of the convergenct blur, break and recovery fusional reserves. This is shown in Figure 6.9.



(a)



(b)



(c)

Figure 6.9 (a) Relationship between symptom score and blur (R^2 = 4 %); (b) Relationship between symptom score and break (R^2 = < 1%) and (c) Relationship between symptom score and recovery (R^2 = <1 %).

6.5 Symptom scores and associated phoria

Subjects were grouped into those with associated phoria (n = 16) and those without (n = 53). A Pooled Variance t-test showed that the group with associated phoria had significant higher symptom scores (2 tail probability p = 0.003; 95% confidence interval = 1.5 to 7).

6.6 Summary

Overall there was very poor correlation between any aspect of eye movement characteristics during the cover test and symptom score.

For the combined normal and referred group, those subjects manifesting recovery times^{RL} falling into the interquartile range of distribution (0.64s to 0.96 s), produced significantly higher symptom scores than those subjects who showed recovery times^{RL} of less than 0.64 s or more than 0.96 s.

Analyses of the recovery eye movement^{RL} and symptom data did not provide convincing evidence of a strong relationship between number of recovery movements and symptom scores.

However, there was a statistically significant difference in the number of recovery movements^{RL} between the lower quartile and interquartile groups of subjects categorised by recovery time^{RL}.

This suggests that whilst both recovery time and the number of eye movements are associated with symptoms, it is the recovery time which is the better predictor for symptoms. However, it would be difficult for the clinician carrying out a conventional cover test, to judge the recovery time to a degree of accuracy to enable categorisation of the precision afforded by the automatic cover test.

Subjects with a NPC of 10 cm or more manifested significantly higher symptom scores than those with a NPC of <10 cm.

There was also a significant difference in symptom scores between subjects with and without associated phoria (symptom scores greater in the associated phoria group).

Chapter 7. The effect of a regime of orthoptic treatment on ocular motor characteristics

7.1 Introduction

Evidence for the effectiveness of various orthoptic treatments is partly anecdotal and has been discussed previously in Chapter 1. One of the aims of this study was to try to provide objective evidence relating to the efficacy of a specific regime of orthoptic treatment. The only valid way of achieving this would be by means of a double masked placebo controlled study. Unfortunately, it was not possible to obtain Research & Ethical Committee approval for such a study. Furthermore it proved very difficult to recruit suitable subjects.

This small study did not aim to compare the efficacy of various treatment methods. Accordingly, a standard orthoptic regime of exercises was initially prescribed for all patients. The exercises chosen for inclusion are arguably the most commonly employed by optometrists in general, rather than by those in specialist optometric practice, in the UK. Such exercises tend to be simple and require little or no equipment.

Daum (1983b) compared the effectiveness of step and jump (phasic) vergence training with sliding and push-up (tonic) vergence procedures and had found phasic therapy the most efficacious. In addition, Pantano (1982) had found that only those patients who were able to achieve both voluntary and fusional convergence and who had learned to relax accommodation adequately while converging could maintain any improvement over time. Daum (1982) and Vaegan (1979) demonstrated the efficacy of repeated short training periods.

Taking these factors into account, a combination of a minimum of 10x "target" to nose (tonic) exercises, 10x jump convergence (phasic), and 5 minutes of physiological diplopia exercises was prescribed to all subjects, to be carried out daily.

A priority during the management of these subjects was to achieve successful treatment of their clinical anomaly and, where applicable, the elimination or reduction of reported symptoms. Accordingly, treatment methods were modified at review appointments, as necessary. These modifications are listed in Table 7.2.

The number of subjects recruited for this part of the study was small and was reduced even further by subjects dropping out of the study. In view of this statistical analyses were limited. However, anecdotal descriptions of the effect of treatment on individuals will be presented.

7.2 Methods

After undergoing the eye movement recording and the other investigative procedures previously described, each subject were offered a regime of orthoptic exercises designed to be carried out by the subjects. All subjects entering the study agreed to carry this out.

Each subject received training from the author in the techniques to be used and was issued with a diary sheet for recording the number of push-ups and jumps, and the number of minutes of physiological diplopia exercises carried out each day. Push-ups and jumps were to be carried out using the tip of a pen or pencil as a target. Physiological diplopia exercises were to be carried out using a "dot-card". A card was dispensed to each subject for this purpose.

Subjects were instructed to keep a diary of exercises carried out for presentation at each appointment. It was stressed that they were to be honest with their recording but it should be noted that the author had to rely on the subjects' objectivity as there was no other way of checking compliance to the regime that was designed to be carried out at home.

Each subject agreed to return for review after one month. At each review appointment, the subject completed a further symptom questionnaire and underwent a full battery of investigations including the automated cover test. If symptoms had satisfactorily resolved they were told to cease the exercises and return a month later to review whether the effect of the treatment was

sustained. If however, at the first review, symptoms were not satisfactorily resolved, the subjects were told to continue with treatment or the treatment was modified.

7.3 Subjects

The inclusion criteria for subjects have been described previously in Chapter 5. 30 subjects initially entered the study. Of these, 7 failed to attend the second appointment and despite follow-up did not make a further appointment. 2 subjects who attended the second appointment stated that they had not carried out the exercises and requested that they be discharged from the study.

The age range of the 21 remaining subjects was from 18 to 35 years with an average age of 24 years.

7.3.1 Anecdotal control group

While it was not possible to obtain ethical approval for a full placebo controlled study, a group that was termed an 'anecdotal control group' was studied. Subjects *mayli, jadoo, nazmo, sselb, vicup* and *vicurt* were subjects who complained of symptoms but showed no obvious anomalies consistent with CI or any other binocular or ocular motor anomaly. They were included in the study as a small control group to give an indication as to the likely placebo effect of the regime of exercises. However, there was a possibility that the normal battery of ocular motor tests did not detect an anomaly and caution should be used when considering the use of this group.

7.4 Results

Table 7.1 lists data describing some of the characteristics of the referred subjects. In this table, phoria amplitudes^{RL} have been converted to prism dioptres. Diagnoses of convergence insufficiency was made when the near point of convergence was 10 cm or greater. Subjects with uncertain diagnoses

were allocated an "?" in Table 7.1. One was described as showing a divergence excess.

Because of the small numbers of subjects, and differences between presenting symptoms and signs, and the subsequent treatment regime, it was generally not possible to apply meaningful statistical analyses to the subjects as a group. Instead, results are presented as a series of case studies. Some statistics are shown for interest but must be viewed with caution.

Subject	Diagnosis	Near symptom score	Dist. amp^{RL} (to nearest ½ Δ)	Near amp^{RL}. (to nearest ½ Δ)	Near recovery time RE (s)	Near recovery Time LE (s)	No. moves RE (near)	No. moves LE (near)	Dist associated phoria(Δ)	Near associated phoria (Δ)	NPC (cm)	Fusional reserves in (Δ)	Fusional reserves out (Δ)	Amplitude of accommodation (D)	AC/A (Δ/D)	Stereoacuity (")	Binocularity (')	Follow-up
adeoj	CI	1	-1	-5½	1.12	1.24	3	4	½ L so	½ R xo	14	10/16/08	16/24/14	R.10.5 L. 10.5	2	60	5	Y
allyo	CI	7	1½	-1	0.31	1.60	1	2	0	½ L xo	10	08/13/11	16/16/12	R.10.0 L. 10.0	2	60	5	Y
airey	?	6	p	-9½	0.68	1.21	5	6	0	0	3	20/20/04	26/28/16	R. 9.0 L 9.0	2	120	5	Y
aisha	CI	2	-½	-3	nd	1.28	nd	3	0	1½ R xo	10	16/20/18	24/28/02	R. 9.0 L. 8.5	2	60	5	Y
arif	?	8	-1	-5	0.70	0.75	4	4	0	0	6	12/16/12	12/12/11	R.10.0 L.10.0	2	30	5	Y
cheal	CI	20	-1	-5½	0.71	1.06	4	7	¼ xo OU	0	20	08/16/12	08/12/08	R. 5.0 L. 5.0	2	60	5	N
fatma	CI	8	3	-1	0.90	nd	2	nd	0	½ R xo	11	08/20/12	08/12/08	R. 7.0 L. 8.0	4	60	5	Y
gejon	CI	0	-1	-6½	1.06	0.68	3	4	0	0	22	04/12/08	nd /12/04	R. 5.0 L. 5.0	0.5	30	5	N
gurao	CI	6	-3	-8	1.38	0.78	3	4	2 R xo	3 L xo	12	12/16/12	08/12/08	R. 4.0 L. 5.0	nd	30	5	Y
jadoo	?	12	2	½	0.84	nd	3	nd	½ R so	0	6	16/20/12	16/32/24	R.12.0 L. 11.0	1	60	5	Y
jashb	CI	9	-2	-13½	2.42	4.00	2	2	0	0	12	14/16/12	nd /08/04	R. 7.0 L. 6.0	4	60	5	N
jimh	CI	24	-1½	-7½	0.59	0.69	3	3	0	½ L xo	16	12/18/12	09/10/07	R. 6.5 L. 6.5	4	30	5	Y
jocox	DE	8	-13	-3	0.49	0.38	3	2	R. supp	0	7	nd /20/ nd	nd /20/ nd	R.12.0 L.12.0	2	120	5	Y
kadep	CI	14	½	-8	1.02	1.16	3	3	0	¼ xo ou	13	12/19/05	12/19/08	R.10.0 L. 12.0	1	60	5	Y
kurp	CI	7	-1½	-5	6.67	nd	5	nd	0	½ L xo	20	12/20/16	05/05/01	R. 3.5 L. 4.0	2	60	5	Y
levy	CI	5	p	-5	0.64	1.08	4	3	0	0	12	nd /12/08	nd /22/16	R. 6.5 L. 6.5	3	60	5	N
mayli	?	5	½	0	0.92	0.53	8	2	0	½ R xo	6	nd /12/11	nd /12/11	R. 9.5 L. 9.5	1	60	5	Y
mdob	CI	15	-2	-8	0.96	0.99	5	3	0	2 L xo	12	15/16/13	07/08/04	R. 6.5 L. 6.5	3	30	5	Y
naco	CI	10	-5½	-8½	1.33	1.00	nd	nd	0	0	10	12/20/12	12/20/2	R. 6.0 L. 8.0	2	60	5	N
nazmo	?	15	½	-1	0.50	0.47	3	2	½ L xo	½ L so	8	nd /14/10	10/12/10	R. 8.0 L. 8.0	2	60	5	Y
neth	CI	10	2½	-½	0.38	nd	2	-	0	0	12	04/08/04	08/12/08	R.12.0 L. 12.0	2	60	5	Y
oltay	CI	17	p	-2	nd	0.60	nd	3	½ R so	1 R xo	14	08/12/09	09/09/06	R. 8.0 L. 0.0	2	30	5	N
sbash	CI	9	-5	-13½	1.27	1.99	6	8	1½ L xo	1 L xo	10	12/14/12	12/16/12	R. 9.0 L. 9.0	2	30	5	Y*
shmeh	?	10	-1	-4	0.71	0.69	5	5	½ L xo	0	8	16/6/15	04/04/01	R. 8.0 L. 7.0	2	60	5	Y
siwh	CI	12	-2	-8½	0.58	0.88	3	6	0	1 L xo	14	08/16/08	11/13/04	R. 7.5 L. 7.5	4	60	5	Y
sselb	?	10	-2	-4	0.59	5.52	3	3	0	0	6	10/14/08	24/36/12	R. 10.5 L. 9.5	5	60	5	Y
vcurt	?	9	1	-½	0.36	nd	2	nd	0	0	6	08/10/08	10/28/12	R.10.0 L. 10.0	2	60	5	Y
vicox	CI	7	-2	-7½	0.58	0.93	3	3	½ L xo	1 R xo	8	12/20/12	11/32/20	R. 8.0 L. 8.0	1	60	5	Y
vicup	?	16.5	-2	-10	0.43	0.83	1	3	0	0	6	nd /16/14	36/44/12	R.12.0 L.12.0	2	60	5	Y
yelena	?	14	-2	-5	0.66	1.24	3	6	0	0	3	08/16/10	20/36/32	R. 8.0 L. 7.0	1	120	5	N

N = failed to attend follow-up appointment
Y* = attended for follow-up but had not done exercises
Y = attended at least one follow-up appointment
P = poor or incomplete traces

L = left eye
R = right eye
OU = both eyes
nd = no data

xo = exophoria supp = suppression (Mallett)
so = esophoria CI = convergence insufficient
+ = esophoria DE = divergence excess
- = exophoria ? = uncertain diagnosis

Table 7.1. Data table showing clinical data of referred subjects at first presentation. Automated cover test phoria amplitudes^{RL} are shown to the nearest ½ Δ.

Table 7.2 lists those subjects who attended for at least one review having carried out the regime of orthoptic exercises, and provides a résumé of treatment progression.

Subject	Diagnosis	1 month review	2 month review	3 month review	4 month review	5 month review
adeoj	CI	C	*D			
allyo	CI	C	*D			
airey	?	*	*	*	-	C♣
aisha	CI	*•	C	*D		
arif	?	*	DNA			
fatma	CI	*	*	* D		
gurao	CI	*	*	* ♣		
jadoo	?	*ψ	* D			
jimh	CI	*	♣			
kadep	CI	*	DNA			
kurp	CI	*	C	* D		
mayli	?	*	C	*	DNA	
mdob	CI	*	C	*D		
nazmo	?	*	*D			
neth	CI	*	D			
shmeh	?	*•	*•	DNA		
siwh	CI	*	C	*D		
sselb	?	C o D				
vcurt	?	*	-	C♠	*D	
vicox	CI	*	*	C sph	*D Δ	
vicup	?	*• t ♣				

* = attended review
C = to cease treatment following this appointment
♣ = subject requested discharge from study
DNA = failed to keep follow-up review
D = discharged
ψ = reported exercise regime not carried out but no close-work undertaken during the interim period
■ = see case studies for modified treatment for this subject
♠ = subject reported that fluorescent lights at work have been removed
sph = -1.00DS spectacles prescribed
Δ = base in prisms prescribed
• = subject reported exercises carried out less than 50% (i.e. 3 days per week or less)
o = Intuitive overlay prescribed
t = Intuitive Colorimetry carried out and Precision tint prescribed

Table 7.2. Table listing progression of treatment for those subjects who attended at least one follow-up appointment.

7.4.1 Subjects diagnosed as CI

12 subjects were diagnosed as manifesting one or more characteristics of CI.

7.4.1.a. Symptom score

Table 7.3 describes changes in symptom score after one month and, where applicable, two months of orthoptic treatment had been carried out. Symptom scores at one month after cessation of exercises are included, where available.

Subject	Symptom score at presentation (A)	Symptom score after 1 month (B)	Symptom difference (B – A)	Symptom score after 2 months of exercises (C)	Symptom difference (C – A)	Symptom score 1 month after ceasing exercises (D)	Regression of symptoms (D – B or C)
adeoj	1	0	-1	na	na	0	0
allyo	7	1	-6	na	na	0	0
aisha	2	8	+6	8 #	+6	11	+3
fatma	8	5	-3	5	-3	na	na
gurao	10	6	-4	5	-5	na	na
jimh	24	21	-3	21	-3	na	na
kadep	14	7	-7	na	na	na	na
kurp	7	1	-6	1	-6	1	0
mdob	15	4	-11	0	-15	0	0
neth	10	10	0	8	-2	na	na
siwh	12	12	0	2	-10	3	1
vicox	7	8	+1	7	0	na	na

\# = subject reported "inertia of accommodation" resolved
na = not applicable for this subject

Table 7.3. Symptom profiles of the CI subjects over time.

6 subjects with CI underwent a minimum of one month of exercises and were followed up one month after ceasing the treatment.

4 subjects (*allyo, kurp, mdob,* and *siwh*) reported noticeable reductions in symptom scores after one or two months. In terms of symptom score, these subjects appeared to be successful and were named ***group*** α.

1 subject (*adeoj)* had presented with a low symptom score (= 1) having been referred solely because of a remote NPC. This subject reported a resolution of symptoms following the treatment but was not included in *group* α.

Subjects *aisha*, *gurao, jimh, kadep, neth* and *vicox* were still complaining of symptoms at the time they left the study and must be considered failures in terms of their symptom score. The treatment for subject *fatma* had to be

modified before achieving a satisfactory resolution of symptoms. These subjects were termed ***group*** β.

7.4.1.b. Recovery time

Table 7.4 lists recovery time (RT) following the automated cover test of CI subjects.

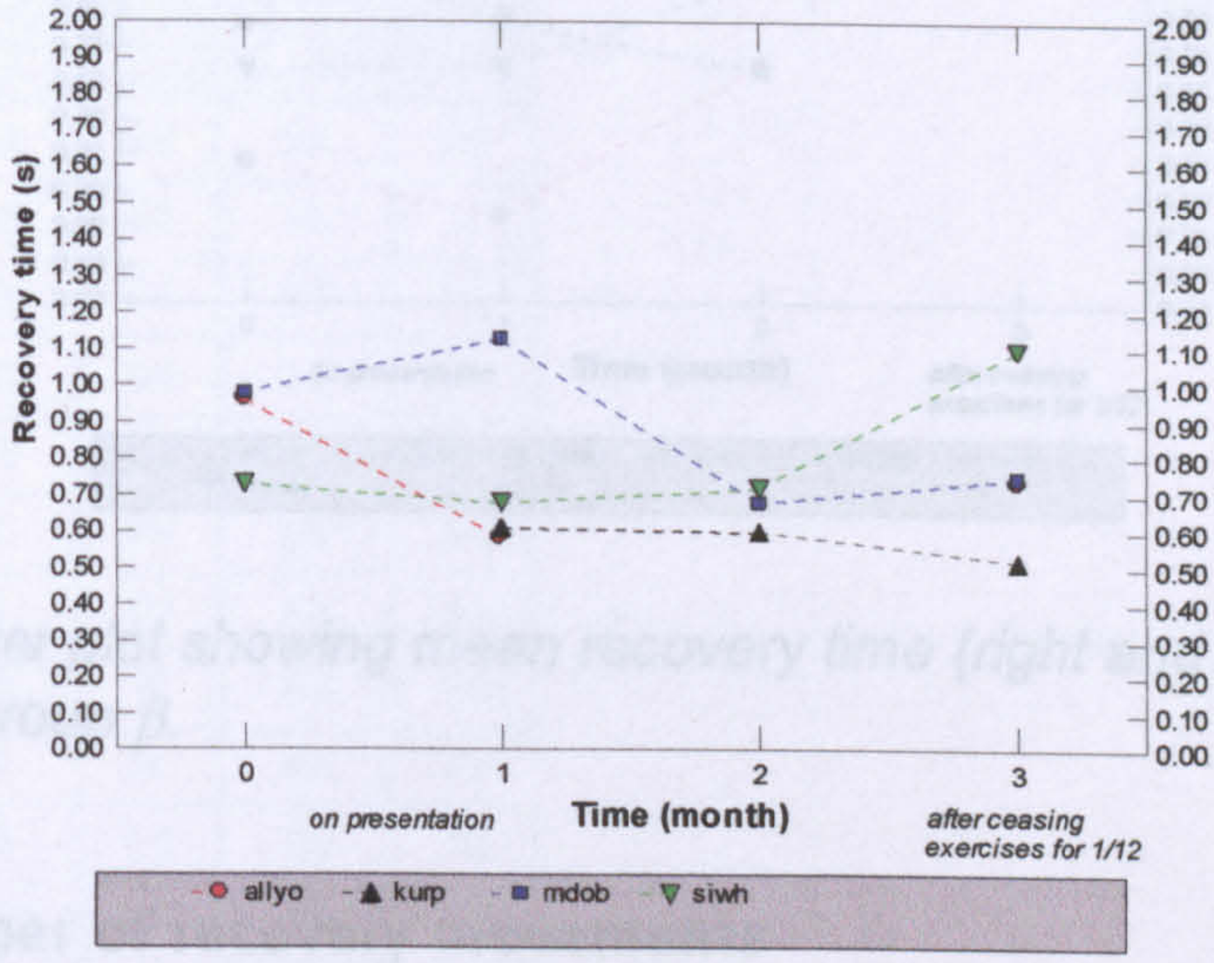
Subject	RE RT at presentation	LE RT at presentation	RE RT after 1 month	LE RT after 1 month	RE RT after 2 months of	LE RT after 2 months	RE RT 1 month after ceasing	LE RT 1 month after ceasing
adeoj	1.12	1.25	0.86	1.52	na	na	nd	nd
allyo	0.31	1.6	0.55	0.60	na	na	0.92	0.55
aisha	nd	1.28	0.62	0.96	0.44	1.88	0.60	3.00
fatma	0.90	nd	0.78	0.97	3.05	0.66	na	na
gurao	1.38	0.78	1.03	0.51	0.71	0.56	na	na
jimh	0.59	0.69	0.70	0.6	1.11	0.66	na	na
kadep	1.02	1.16	0.93	0.58	na	na	na	na
kurp	6.67	nd	0.57	0.65	0.66	0.54	0.48	0.56
mdob	0.96	0.99	0.85	1.41	0.64	0.72	0.79	0.70
neth	0.38	nd	0.38	0.10	0.66	nd	na	na
siwh	0.58	0.88	0.65	0.71	0.63	0.80	0.89	1.31
vicox	0.58	0.93	0.92	1.76	nd	fc	na	na

nd = no data
na = not applicable to this subject
fc= failed to make a recovery

Table7.4. Recovery times(s) of CI subjects following the automated near cover test over time.

Figure 7.1 illustrates the mean of the right and left recovery time for each subject in *group* α. Along the X-axis, 0 denotes the point at which the subject first presented, 1 denotes the first review after one month of exercises, 2 denotes the review after two months of exercises, and 3 denotes the review one month after ceasing the exercises. For all subsequent figures, the numbers along the X-axis denote the month of review. In Figure 7.1, the

presentation datum for subject *kurp* has been omitted because one eye failed to recover following the cover test and the fellow eye took over 6 s to recover. Subject *allyo* ceased treatment after the first review.



Along the X-axis, 0 denotes the point at which the subject first presented, 1 denotes the first review after one month of exercises, 2 denotes the review after two months of exercises, and 3 denotes the review one month after ceasing the exercises. For all subsequent figures, the numbers along the X-axis denote the month of review.

Figure 7.1. Scatter plot showing mean near recovery time (right and left eyes) for subjects within group α.

Although there appears to be a trend towards a reduction in average recovery time by the end of the treatment period for three subjects, subject *siwh* did not show this tendency.

Figure 7.2 illustrates the mean of the right and left recovery time for each subject in *group β.* Apart from *gurao,* individuals within *group β* showed no tendency for a decrease in recovery time over the treatment. Indeed, there appeared to be a trend towards increased recovery times for these subjects. For the purposes of this figure, monocular data was used when data for both eyes was not available.

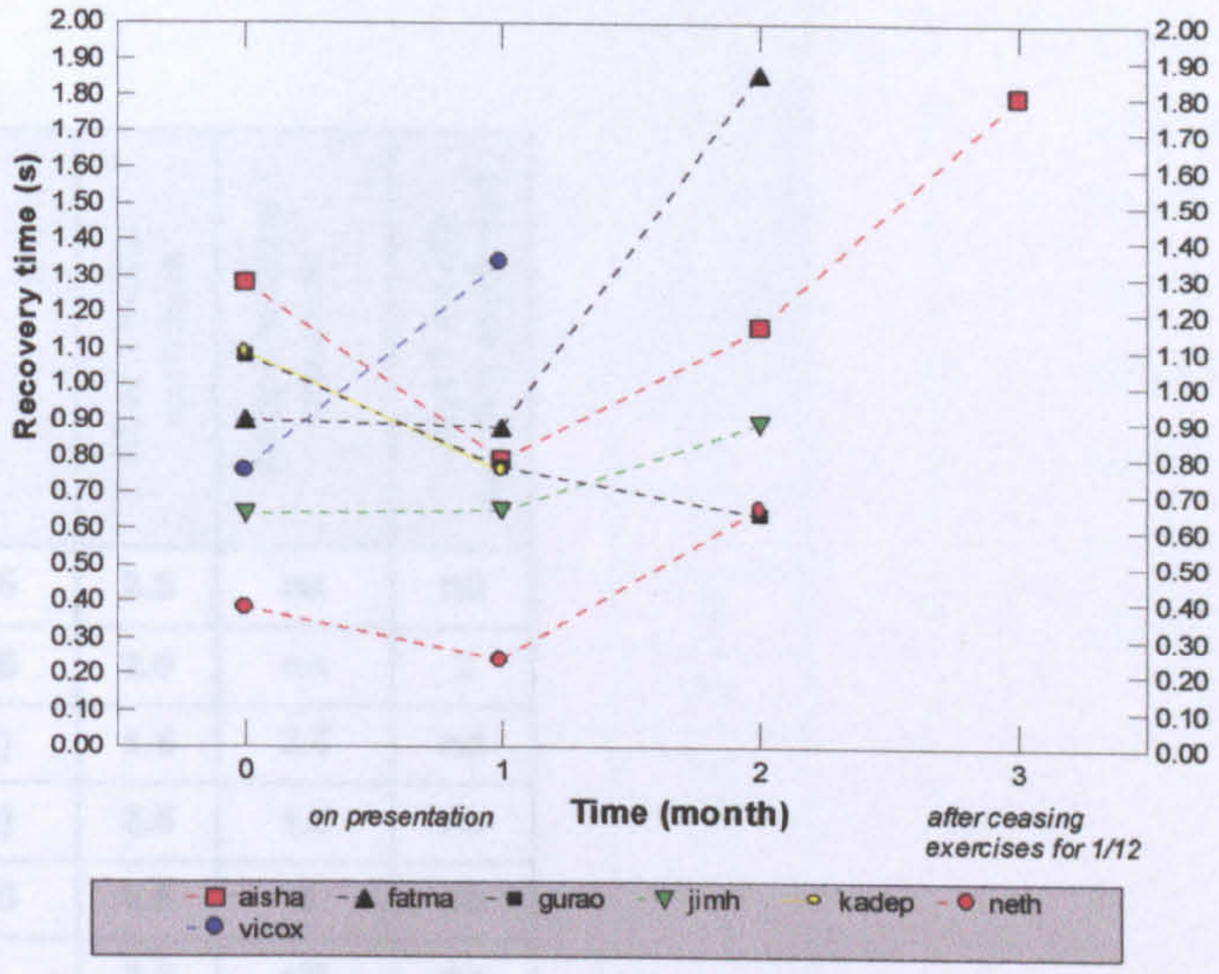


Figure 7.2. Scatter plot showing mean recovery time (right and left eyes) for subjects within group β.

7.4.1.c. Number of recovery movements

Table 7.5 lists the mean number of recovery eye movements (right and left eye) made by each subject following the automated cover test. Where bilateral data was not available, the available monocular data are included in brackets. Some data are missing due to difficulty in interpreting traces due to blinks.

Subject	At presentation	After 1 month exercises	After 2 months exercises	After 1 month ceasing exercises
adeoj	3.5	2.5	na	nd
allyo	1.5	2.5	na	2
aisha	(3)	1.5	3.5	nd
fatma	(2)	3.5	1.5	na
gurao	3.5	4.5	3	na
jimh	3	3.5	(3)	na
kadep	3	2.5	na	na
kurp	(5)	4	2.5	1.5
mdob	4	4	3.5	4.5
neth	(2)	1.5	(2)	na
siwh	4.5	4	4.5	3.5
vicox	3	3.5	(fc)	na

na = not applicable for this subject
nd = no or inconclusive data
fc = failed to recover

Table 7.5. Mean number of eye movements made to achieve recovery.

Examination of this data did not reveal any individual trends in terms of recovery characteristics for either *group* α *or* β.

7.4.1.d. NPC

Table 7.6 shows changes in NPC measures with orthoptic treatment.

10 of the 12 subjects showed a reduction in NPC after one month of treatment. The mean NPC before treatment commenced was 13.0 cm (SD = 2.8 cm) and 8.5 cm (SD = 3.3 cm) after one month of treatment. There was a significant statistical difference between means (Wilcoxon signed rank test p = 0.006).

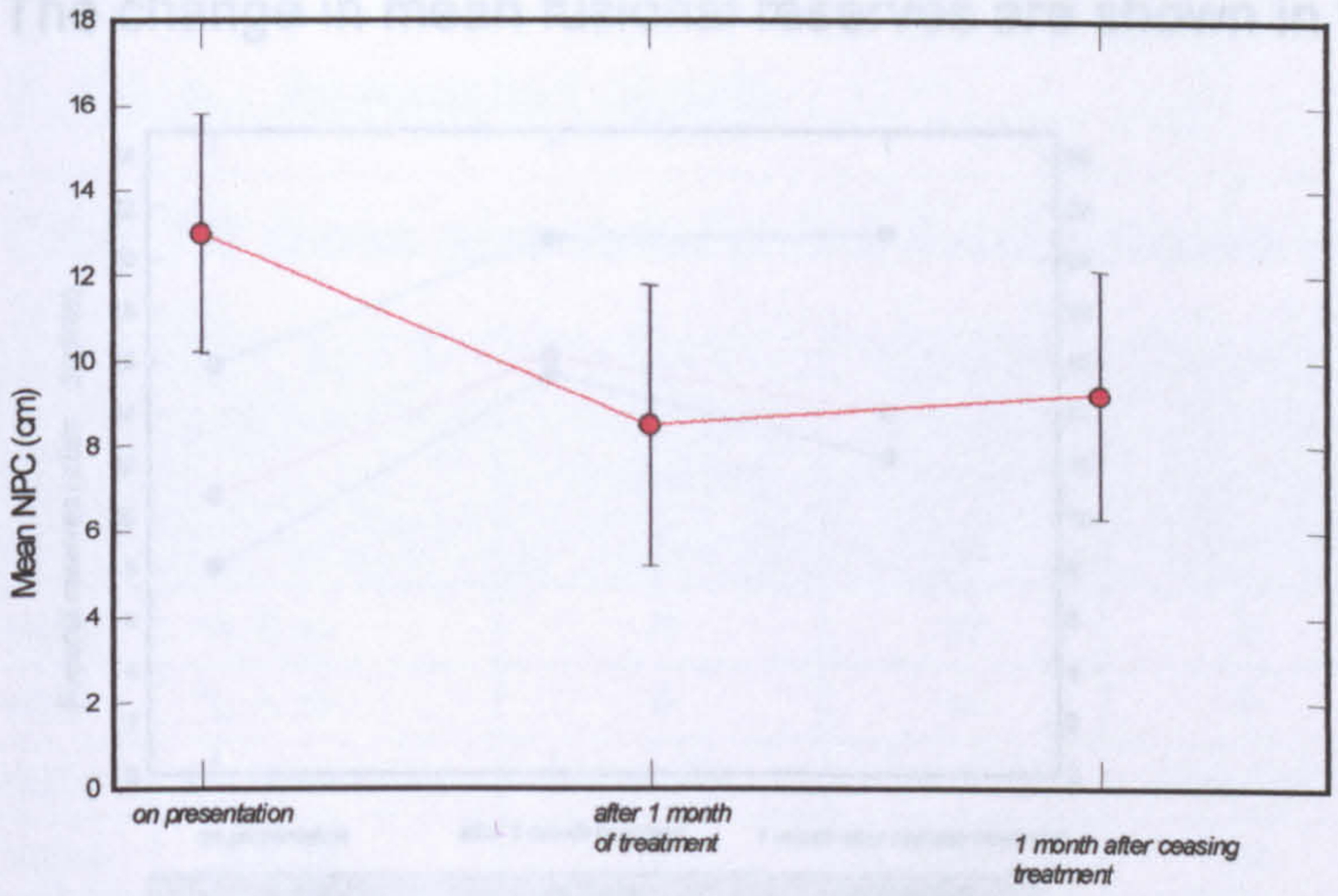
Subject	NPC at presentation (A)	NPC after 1 month (B)	NPC difference (B – A)	NPC after 2 months of exercises (C)	NPC difference (C – A)	NPC 1 month after ceasing exercises (D)	Regression of NPC (D – B, or D – C where applicable)
adeoj	14	6	-8	na	na	6	0
allyo	10	6	-4	na	na	7	+1
aisha	10	6	-4	6	-4	6	0
fatma	11	14	+3	12	+1	na	na
gurao	12	8	-4	8	-4	na	na
jimh	16	14	-2	14	-2	na	na
kadep	13	4	-9	na	na	na	na
kurp	20	7	-13	7	-13	8	+1
mdob	12	7	-5	6	-6	6	0
neth	12	12	0	12	0	na	na
siwh	14	10	-4	9	-5	8	-1
vicox	12	8	-4	9	-3	na	na

na = not applicable to this subject

Table 7.6. NPC over time for the CI group.

For those subjects who were reassessed one month after ceasing treatment (n = 6), there was a significant statistical difference (p = 0.03) between the mean NPC before commencing treatment (mean = 13.3 cm; SD = 3.7 cm) and after one month of treatment (mean = 6.5 cm; SD = 1.6 cm). There was no difference (p = 0.85) between means after one month of treatment and after ceasing exercises for one month. Some caution should be used in interpreting these results as improvements in NPC might be explained by subjects 'learning' to do the test better.

Figure 7.3 shows the mean NPC on presentation, after one month of treatment and one month after ceasing the exercises.



The data on presentation and after one month of treatment depict the mean for 12 subjects; the datum after cessation of the exercises depicts the mean for 6 subjects. Error bars depict standard deviations.

Figure 7.3. Graph showing mean NPC before treatment commenced, after one month of treatment and after treatment had ceased for one month.

Positive fusional reserves

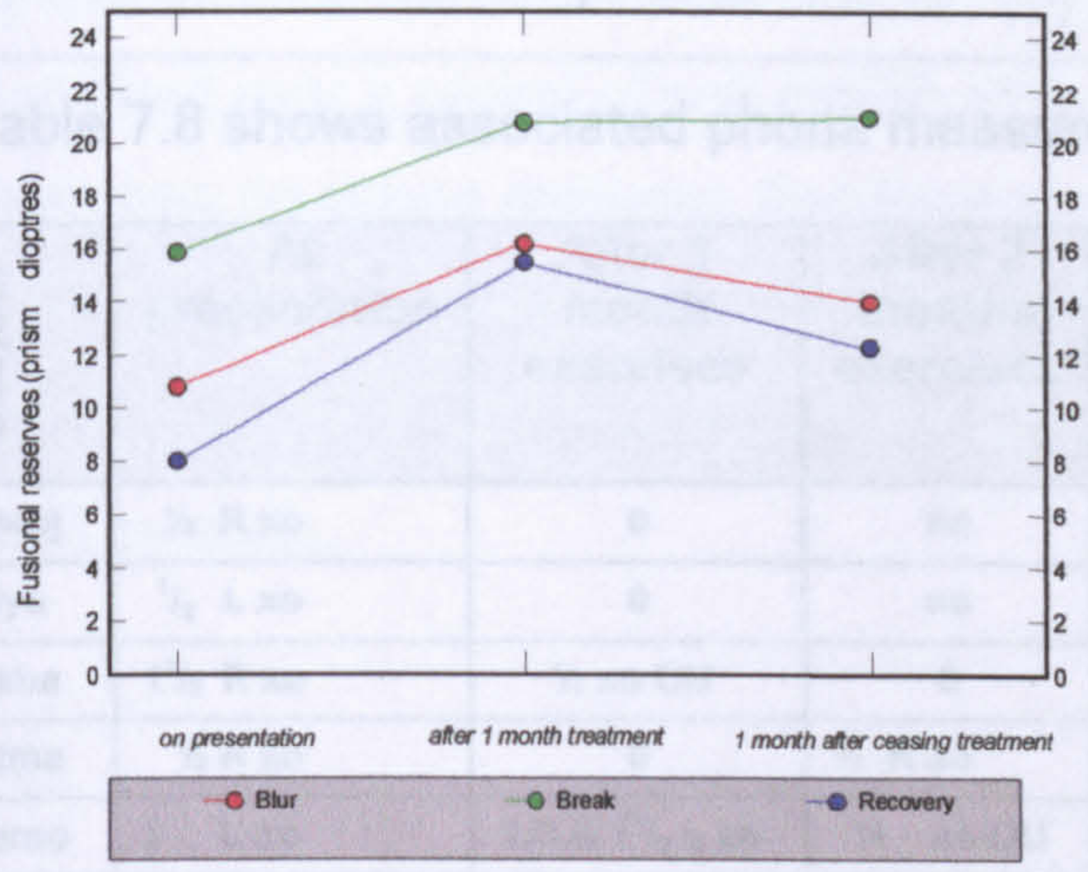
Table 7.7 shows positive fusional reserves (blur/break/recovery) over time.

Subject	Positive fusional reserves (blur/break/recovery) (Δ)						
	At presentation (A) (A1/A2/A3)	After 1 month (B)	Change (B – A)	After 2 months of exercises (C) (C1/C2/C3)	Change (C- A)	After 1 month of ceasing exercises (D) (D1/D2/D3)	Regression (D – B) (or D –C when applicable)
adeoj	16/24/14	24/26/18	08/02/04	na	na	#/16/14	na/-10/-04
allyo	#/16/12	14/28/16	#/12/04	na	na	16/22/04	02/-06/-12
aisha	24/28/02	30/36/28	06/08/26	24/36/12	00/08/10	#/30/28	na/-06/16
fatma	08/12/08	10/14/10	02/02/02	12/20/14	04/08/06	na	na
gurao	08/12/08	10/14/06	02/02/-2	#/18/16	na/06/08	na	na
jimh	09/10/07	11/12/11	02/02/04	06/06/02	-03/-04/-05	na	na
kadep	12/19/08	28/32/28	14/13/20	na	na	na	na
kurp	05/05/01	20/24/20	15/19/19	#/32/16	na/27/15	#/30/08	Na/-02/-08
mdob	07/08/04	#/20/18	na/12/14	10/12/10	03/04/06	12/12/08	02/00/-2
neth	08/12/08	08/08/02	00/00/-06	04/08/06	-04/-04/-02	na	na
siwh	11/13/04	10/14/12	-01/01/08	14/18/12	03/05/08	14/18/12	00/00/00
vicox	11/32/20	13/22/17	02/-10/-3	12/20/16	01/-12/-04	na	na

\# = *blur point not observed by subject*
na = *not applicable to this subject*

Table 7.7. Positive fusional reserves (blur/break/recovery) over time.

The change in mean fusional reserves are shown in Figure 7.4.



	Blur		Break		Recovery	
	SD	*n*	*SD*	*n*	*SD*	*n*
On presentation	5.3	11	8.3	12	5.3	12
After 1 month	7.9	11	8.7	12	7.9	12
After ceasing	2.0	3	7.5	6	8.4	6

Figure 7.4. Graph showing mean fusional reserves (prism dioptres) on presentation, after 1 month of treatment and 1 month after cessation of exercises (standard deviations (SD) and the number of subjects (n) are shown).

There was a statistically significant difference between means, for fusional reserves on presentation and after one month of treatment, for blur (Wilcoxon signed rank test $p = 0.01$), break ($p = 0.05$), and recovery ($p = 0.03$).

Analysis of the limited data for break and recovery showed no statistically significant difference between means for measures after one month of treatment and one month after cessation of treatment (break $p = 0.20$; recovery $p = 0.10$).

7.4.1.e. Associated phoria

Table 7.8 shows associated phoria measurements over time.

Subject	At presentation	After 1 month exercises	After 2 months exercises	After 1 month ceasing exercises
adeoj	½ R xo	0	na	0
allyo	1/2 L xo	0	na	0
aisha	1 1/2 R xo	½ xo OU	0	½ xo OU
fatma	½ R xo	0	½ R so	na
gurao	3 L xo	1 R & 1 1/2 L xo	½ xo OU	na
jimh	1/2 L xo	0	0	na
kadep	1/4 xo OU	0	na	na
kurp	1/2 L xo	1/2 L xo	0	1/2 L xo
mdob	2 L xo	0	0	0
neth	0	0	0	na
siwh	1 L xo	½ L xo	0	½ L xo
vicox	1 R xo	1 ½ L xo	1 ½ R xo	na

Note: paradoxical esophoric slip occurring at 2months for subject fatma

na = not applicable to this subject

Table 7.8. Changes in associated phoria.

The mean associated phoria at presentation was 1^{Δ} exophoria (n = 12) compared to 0.5^{Δ} after one month of treatment (n = 12). There was a statistically significant difference between means (Wilcoxon signed rank p = 0.02). There were data for 6 subjects who were reassessed one month after ceasing the exercises. For these subjects there was a significant statistical difference in mean associated phoria amplitude on presentation and one month after ceasing treatment (Wilcoxon signed rank test p = 0.05).

Comment

The statistics presented above must be viewed with extreme caution because of the very limited number of subjects.

Nevertheless, the findings are of interest and suggest that the regime of orthoptic exercises carried out by these subjects caused a reduction in the mean NPC and associated phoria amplitudes, and an improvement in mean

fusional reserves after one month of treatment. The evidence suggested that, for this small group of subjects with CI, the mean improvements were largely sustained over a period of one month after ceasing the exercises. However, there was no strong evidence that the exercises affected either recovery time or the number of recovery movements during the cover test.

In terms of symptoms, this regime was judged to be successful in only 33.3% of CI subjects.

7.4.2 Discussion of individual case histories

7.4.2.a. GROUP α

I. Case history

Subject	*Allyo*
Age	21 years-old
Sex	Female
Occupation	Optometry undergraduate
Symptoms	Very troublesome headaches about 25% of the occasions that she carried out close-work, as well as aching or watering eyes, blurred near vision, and tired eyes and/or loss of concentration 25% of the time
Reported compliance	Full
Resolution of symptoms	Resolved by 1 month review at which time she requested cessation of exercises
Follow-up	Reported that the amelioration of the symptoms was sustained one month after cessation
Outcome	The subject described the resolution of her symptoms as a successful outcome and was discharged

Figures 7.5 and 7.6 show the reduction in NPC and symptom score, and mean recovery time accompanying the reduction of symptom score.

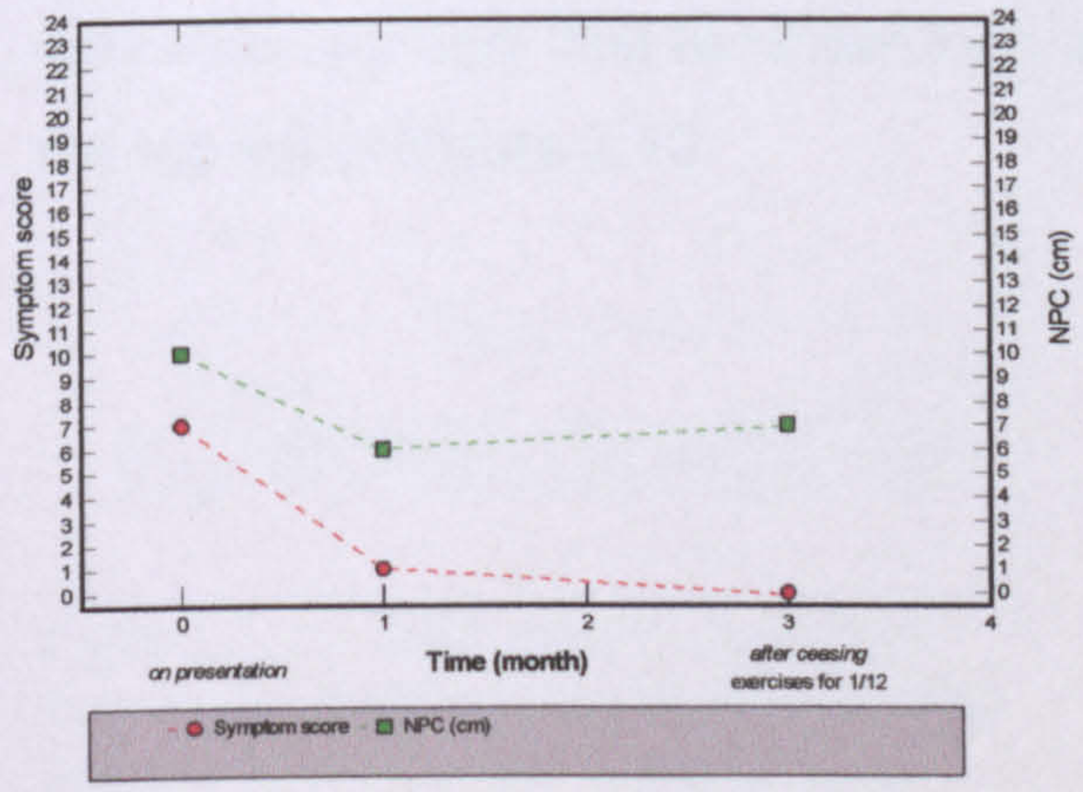


Figure 7.5. Changes in symptom score and NPC over the treatment period for subject allyo.

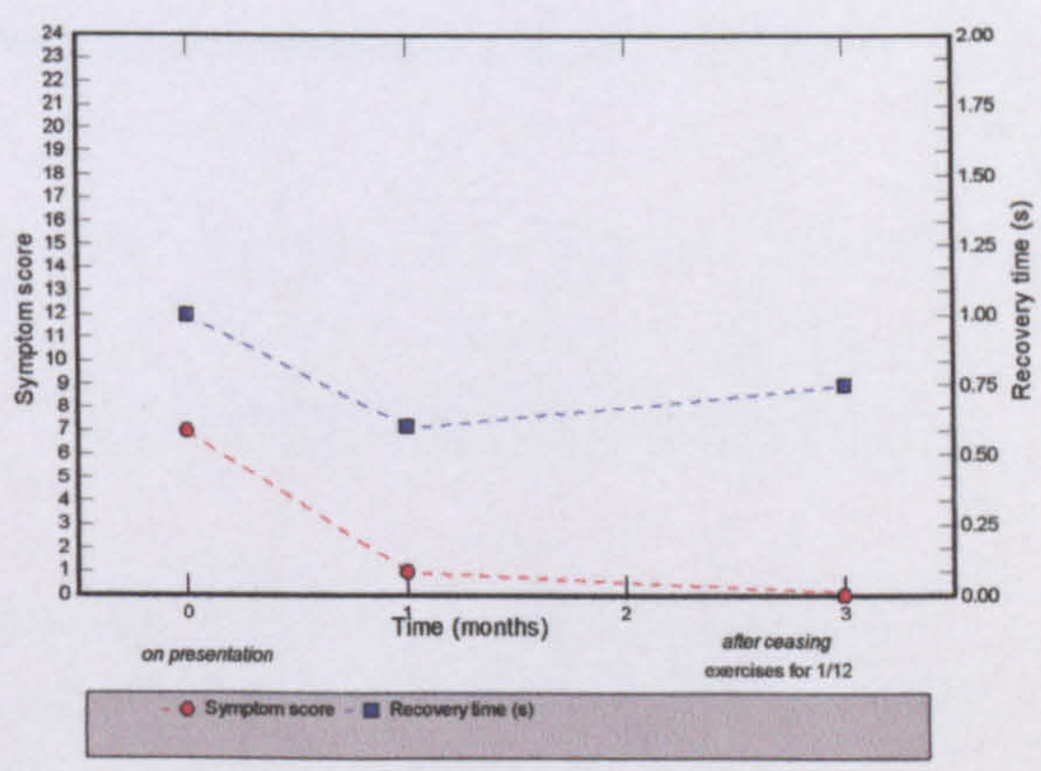


Figure 7.6.Changes in symptom score and recovery time over the treatment period for subject allyo.

Figure 7.7 illustrates the improvement in fusional reserves after one month of treatment accompanying the reduction in symptom score. The subject could not differentiate between blur and break on presentation and only the break datum is shown. There appeared to be a regression of fusional reserves after ceasing the exercises for one month. This was not accompanied by an increase in symptom score. Figure 7.8 describes the elimination of associated phoria that was sustained following cessation of the exercises.

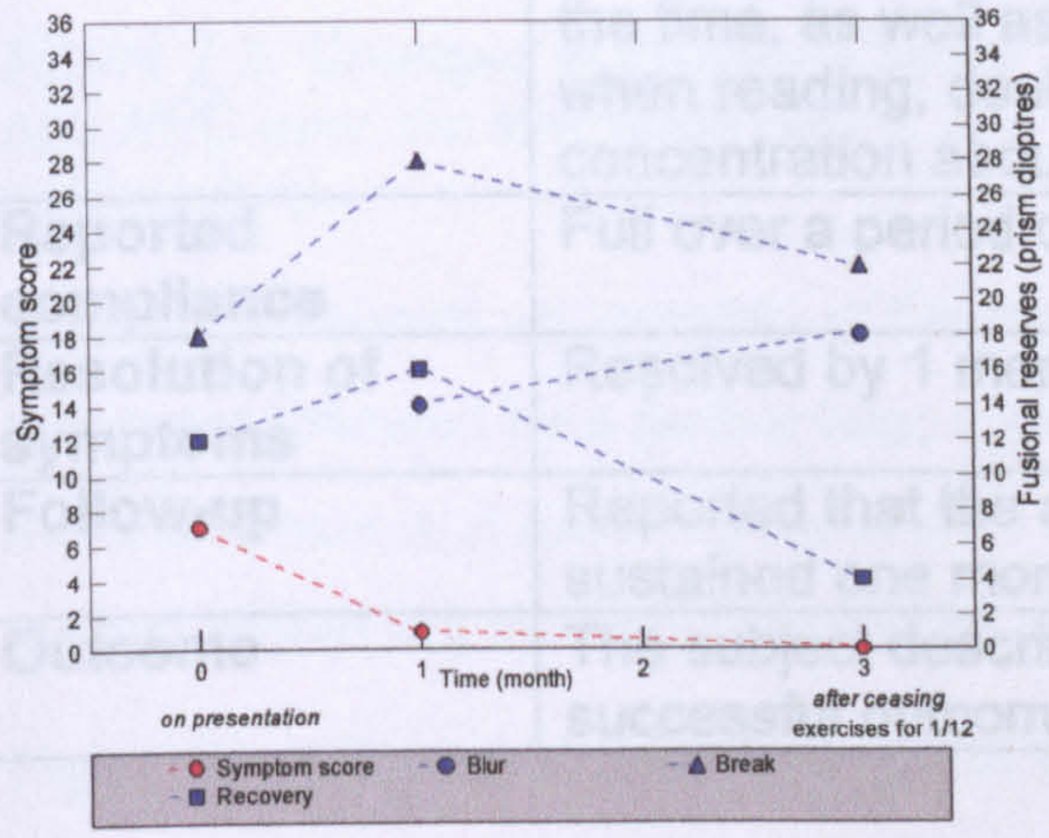


Figure 7.7. Changes in symptom score and fusional reserves over the treatment period for subject allyo.

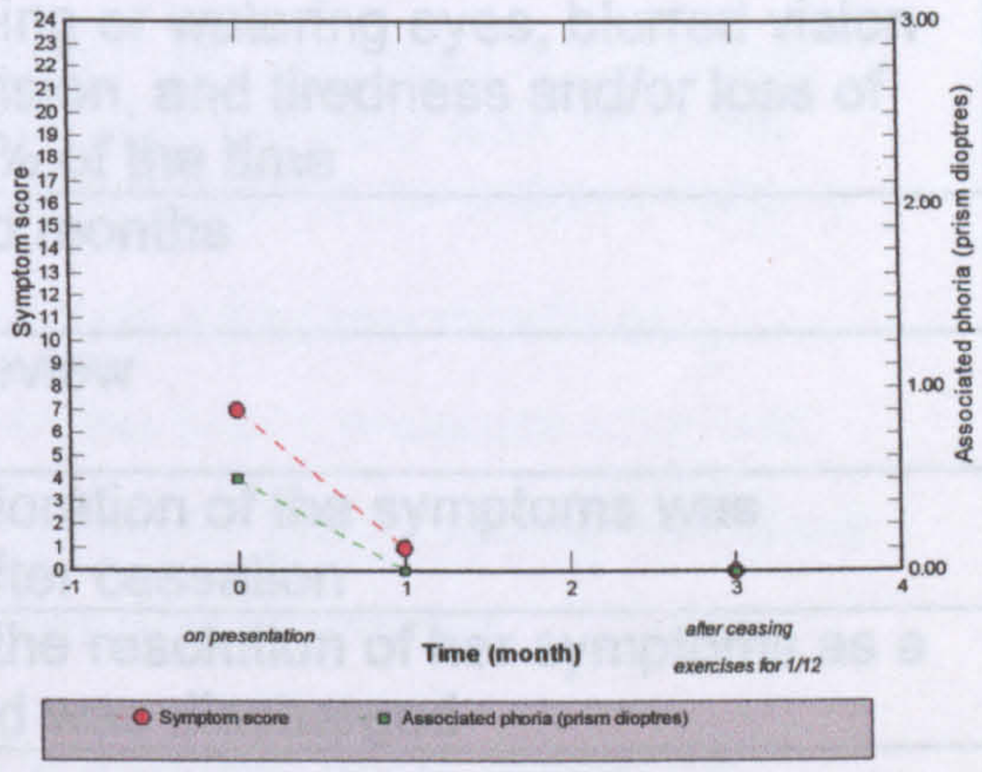


Figure 7.8. Changes in symptom score and associated phoria over the treatment period for subject allyo.

II. Case history

Subject	*Kurp*
Age	24 years-old
Sex	Female
Occupation	Optometry undergraduate
Symptoms	She complained of discomfort after about 2 hours of close work. This took the form of mild headaches about 25% of the time, as well as aching or watering eyes, blurred vision when reading, double vision, and tiredness and/or loss of concentration about 25% of the time
Reported compliance	Full over a period of two months
Resolution of symptoms	Resolved by 1 month review
Follow-up	Reported that the amelioration of the symptoms was sustained one month after cessation
Outcome	The subject described the resolution of her symptoms as a successful outcome and was discharged

Figure 7.9 shows the improvement in NPC that was accompanied by a reduction in symptom score. Whilst the NPC had regressed slightly following cessation of the exercises, the symptom score remained static.

Figure 7.10 shows little change in recovery time between the 1 and 2 month reviews and at the review one month after she had ceased treatment. The very slow recovery time recorded for one eye at presentation is alluded to at the top left of Figure 7.10.

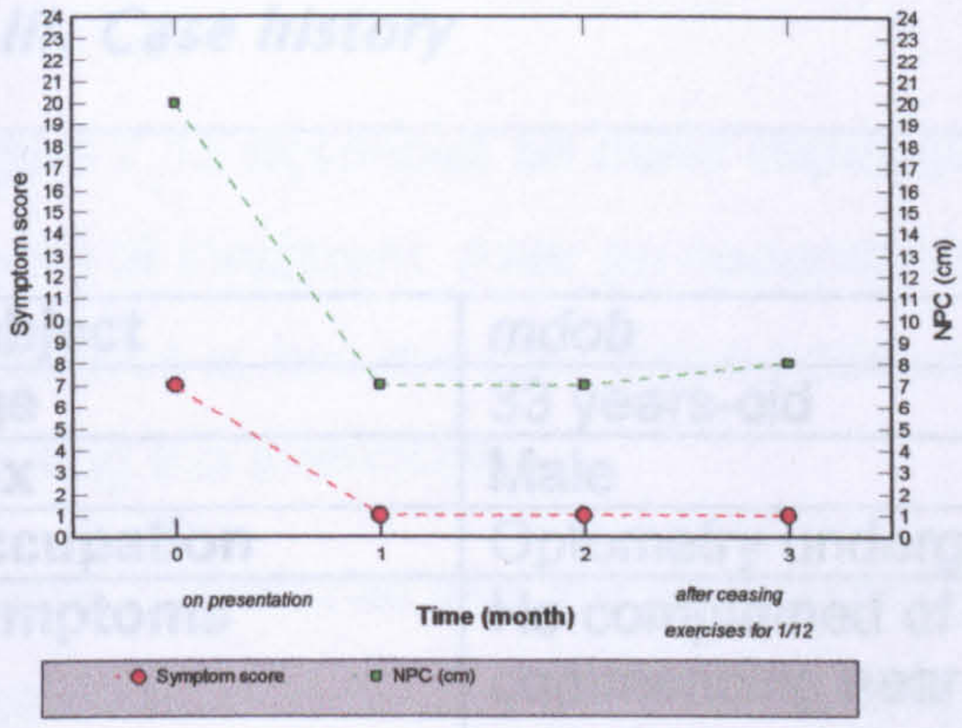


Figure 7.9. Changes in symptom score and NPC over the treatment period for subject kurp.

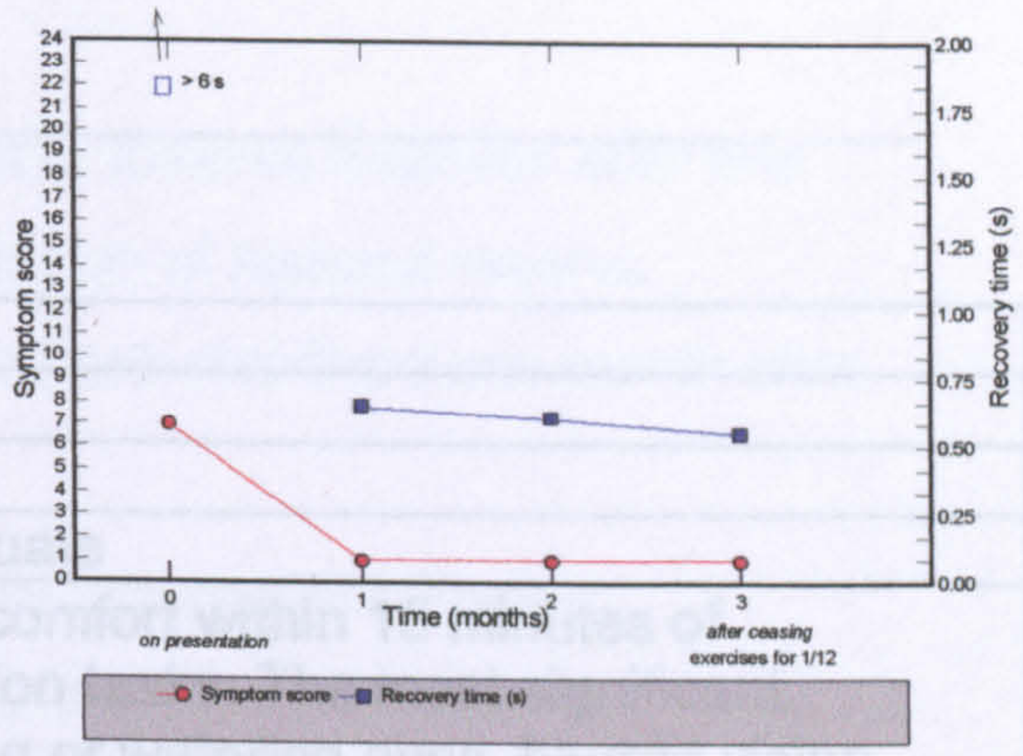


Figure 7.10. Changes in symptom score and recovery time over the treatment period for subject kurp.

Figure 7.11 shows an increase in fusional reserves after one month of treatment followed by a further improvement in the break measure after two months. Break and recovery measures had regressed after ceasing exercises for one month but the improvement in symptoms was sustained.

Figure 7.12 illustrates the alleviation of the 1^{Δ} exophoric slip at the second review. This had manifested itself again after ceasing exercises for one month.

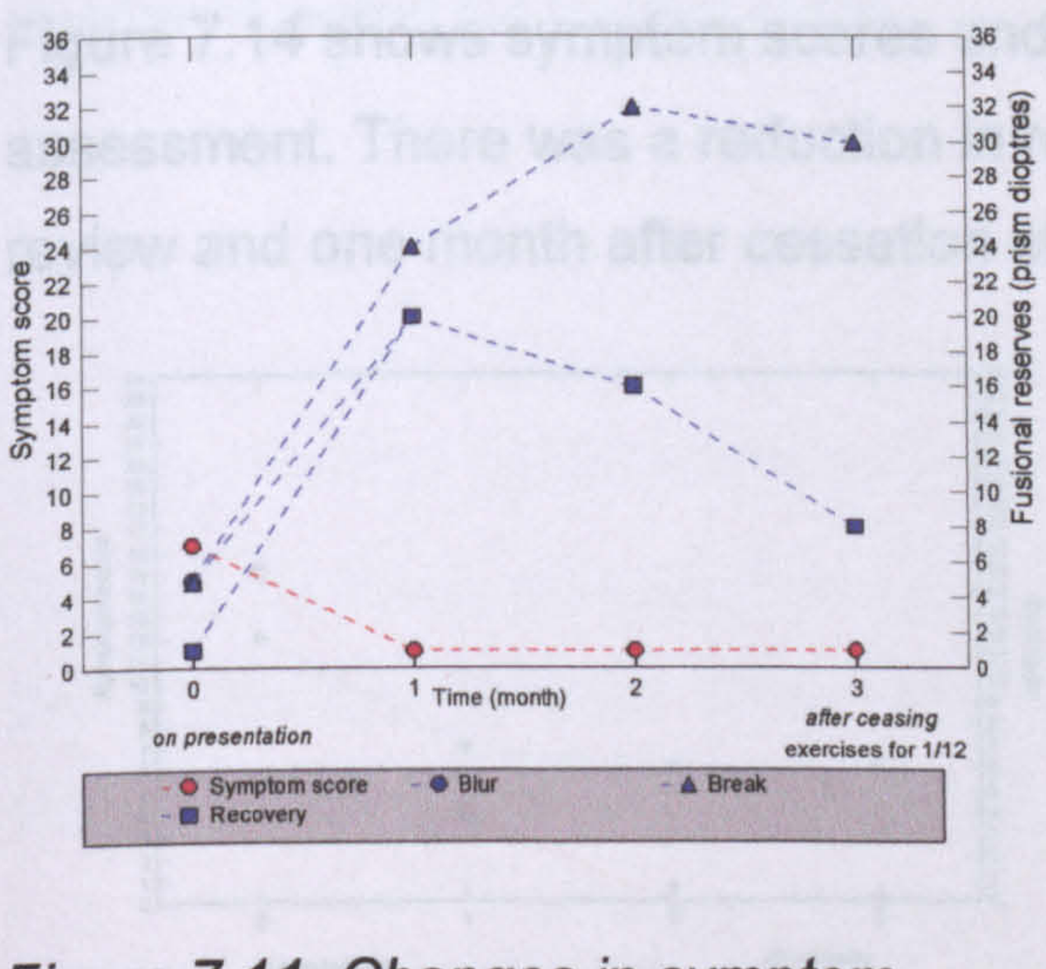


Figure 7.11. Changes in symptom score and fusional reserves over the treatment period for subject kurp.

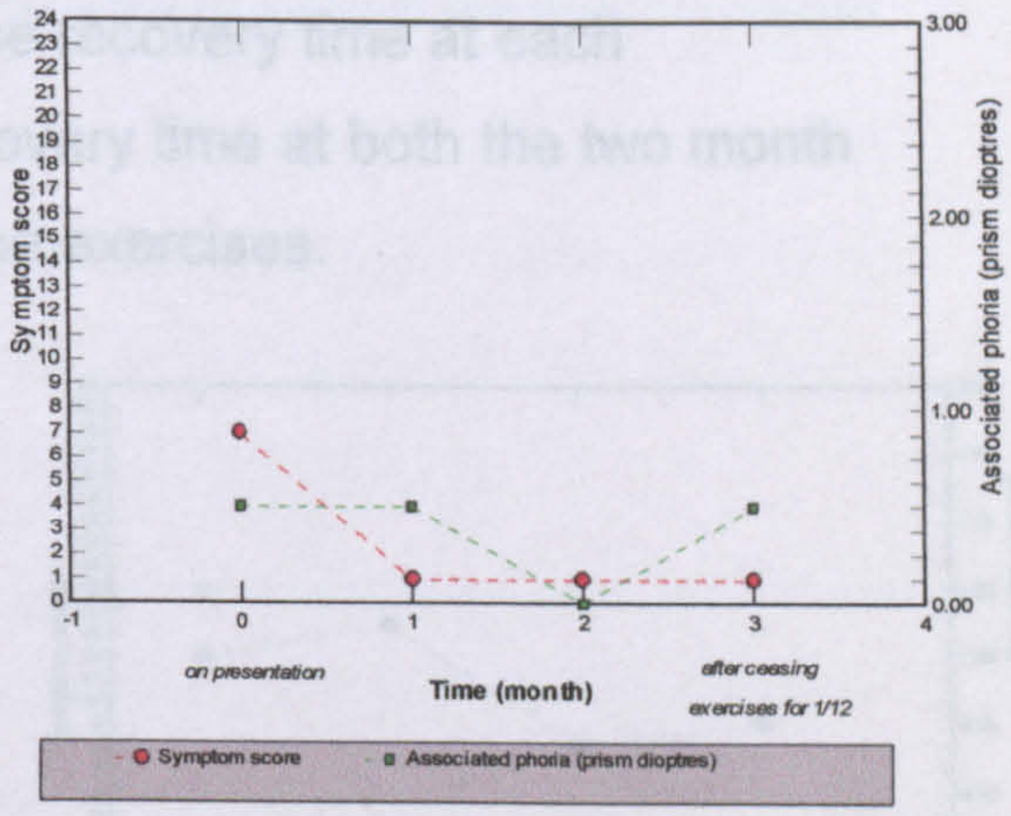


Figure 7.12. Changes in symptom score and associated phoria over the treatment period for subject kurp.

III. Case history

Subject	*mdob*
Age	33 years-old
Sex	Male
Occupation	Optometry undergraduate
Symptoms	He complained of discomfort within 15 minutes of commencing near vision tasks. The most significant symptoms were aching or watering eyes, blurred vision when reading, and tired eyes and/or loss of concentration about 75% of the time
Reported compliance	Full compliance
Resolution of symptoms	His initial symptom score of 15 was reduced to 4 after 1 month of treatment and to zero after two months
Follow-up	Reported that the amelioration of the symptoms was sustained one month after cessation
Outcome	The subject described the resolution of his symptoms as a successful outcome and was discharged

Figure 7.13 shows an improvement in NPC accompanied by the reduction in symptoms after one month of treatment. Both NPC and symptom resolution was maintained after ceasing the exercises for one month.

Figure 7.14 shows symptom scores and the recovery time at each assessment. There was a reduction in recovery time at both the two month review and one month after cessation of the exercises.

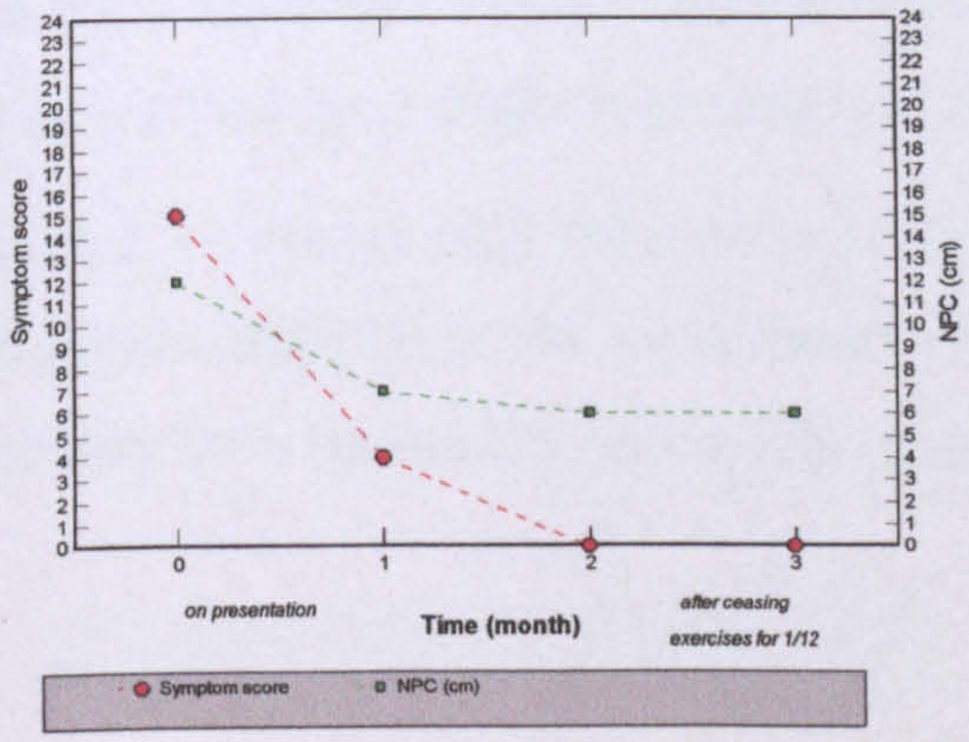


Figure 7.13. Changes in symptom score and NPC over the treatment period for subject mdob.

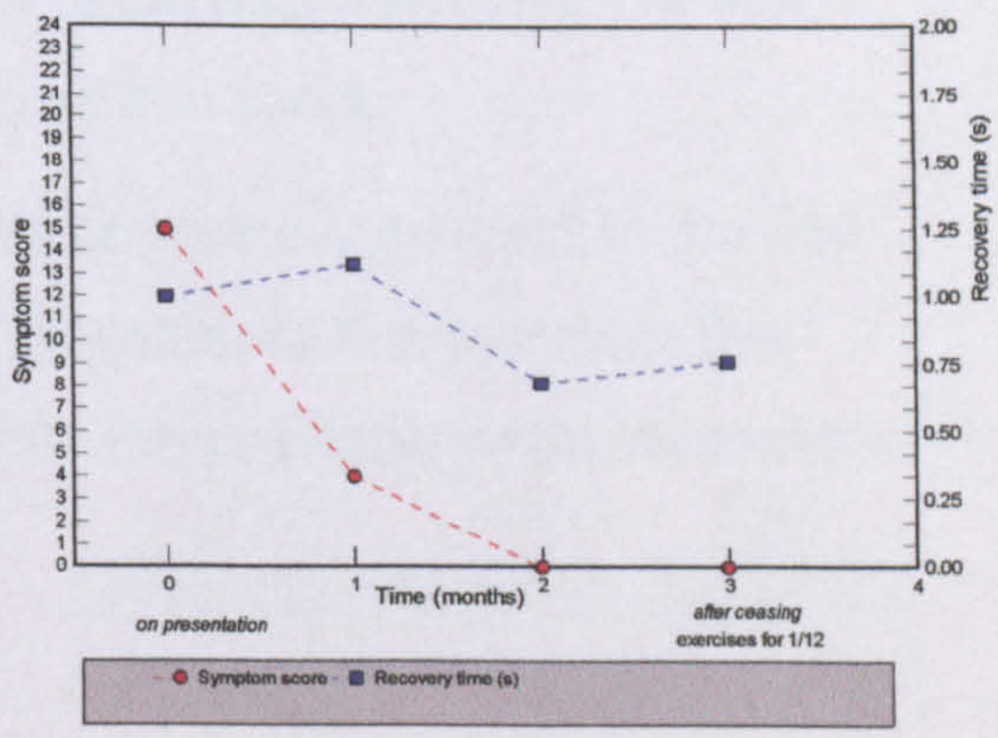


Figure 7.14. Changes in symptom score and recovery time over the treatment period for subject mdob.

Figure 7.15 describes an initial improvement in fusional reserves after one month of treatment. After an apparent regression of fusional reserve measures at two months, these appeared to have stabilised one month after ceasing the exercises.

Figure 7.16 shows that the associated phoria that was manifest at presentation appeared to be resolved by the one month review. There was no regression thereafter.

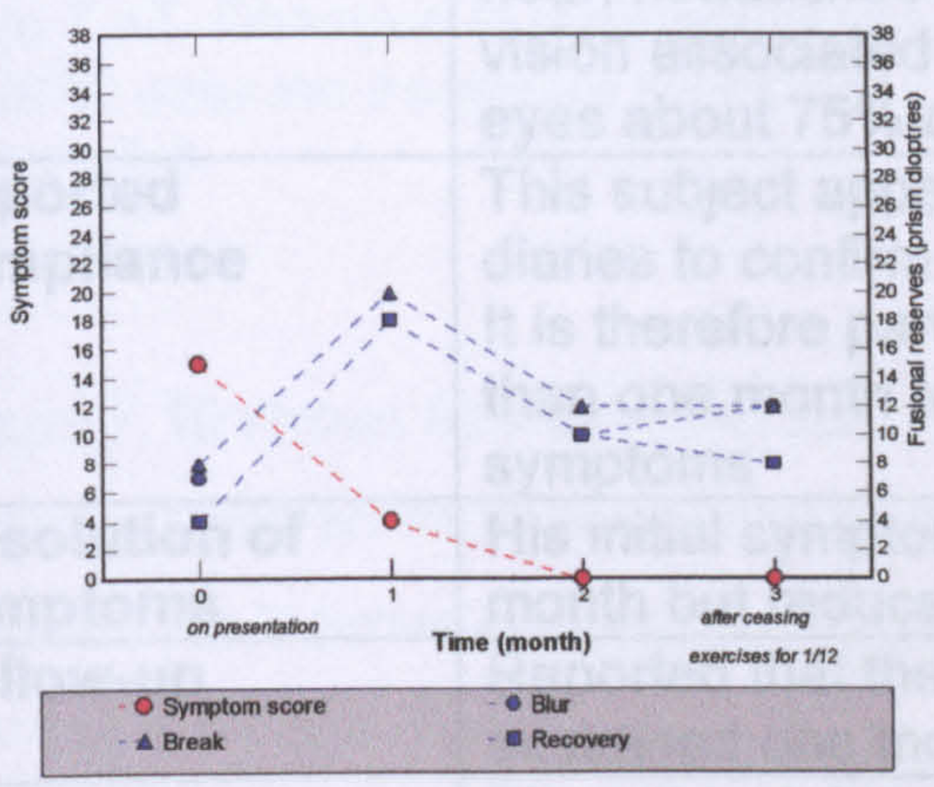


Figure 7.15. Changes in symptom score and fusional reserves over the treatment period for subject mdob.

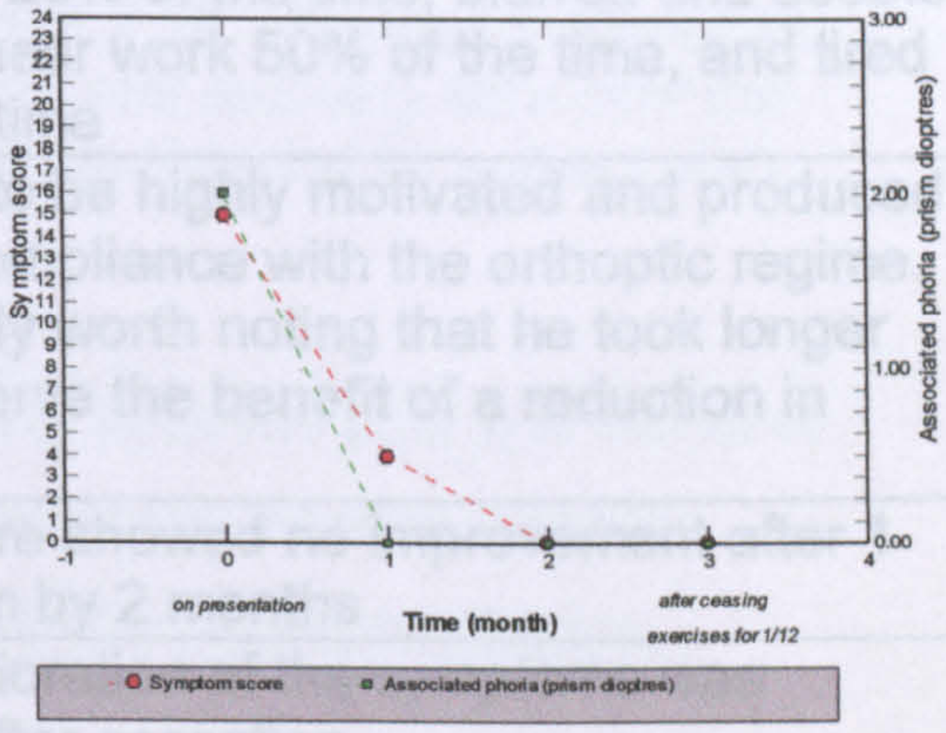


Figure 7.16. Changes in symptom score and associated phoria over the treatment period for subject mdob.

IV. Case history

Subject	*siwh*
Age	19 years-old
Sex	Male
Occupation	Optometry undergraduate
Symptoms	His symptoms associated with close-work included discomfort after carrying out near vision tasks for about an hour, headaches about 25% of the time, blurred and double vision associated with near work 50% of the time, and tired eyes about 75% of the time
Reported compliance	This subject appeared to be highly motivated and produced diaries to confirm full compliance with the orthoptic regime. It is therefore particularly worth noting that he took longer than one month to observe the benefit of a reduction in symptoms
Resolution of symptoms	His initial symptom score showed no improvement after 1 month but reduced from by 2 months
Follow-up	Reported that the amelioration of the symptoms was sustained one month after cessation
Outcome	The subject described the resolution of his symptoms as a successful outcome and was discharged

Figure 7.17 shows no improvement in the symptom score after one month of treatment despite a slight reduction in the NPC. The NPC improved slightly by two months and this was accompanied by a significant reduction in symptoms. The NPC improved further despite cessation of the exercises and this was accompanied by a slight regression of the symptom score.

Figure 7.18 shows little difference in recovery at presentation and at the first two review appointments. One month after cessation of the exercises the recovery time appeared noticeably longer than the previous measurements.

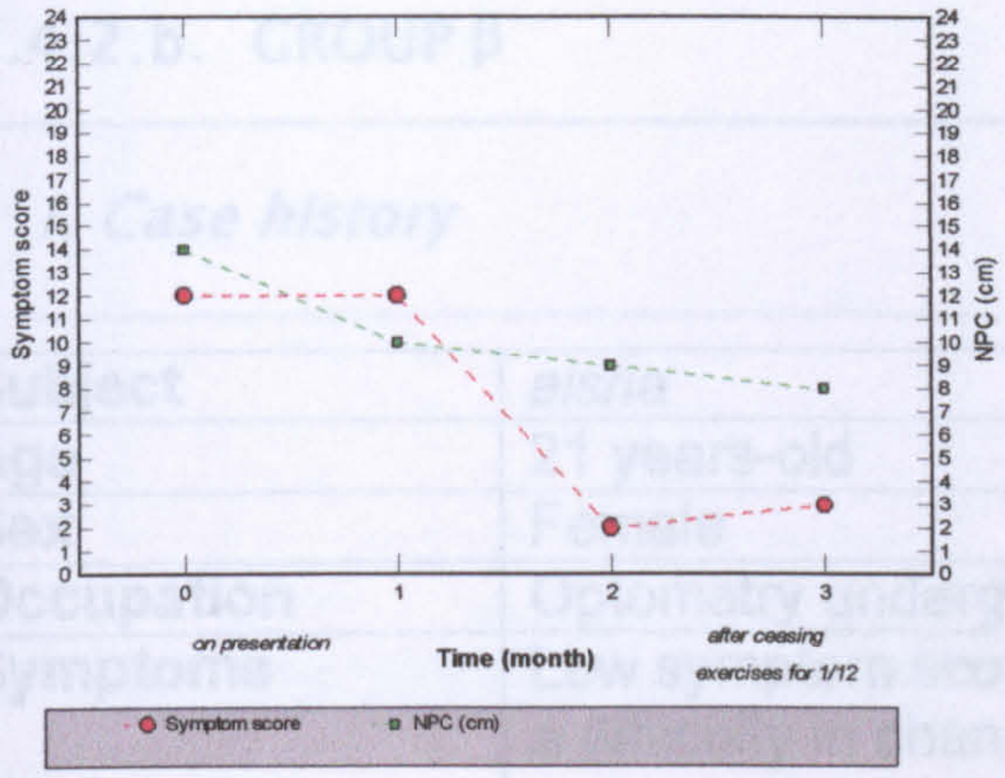


Figure 7.17. Changes in symptom score and NPC over the treatment period for subject siwh.

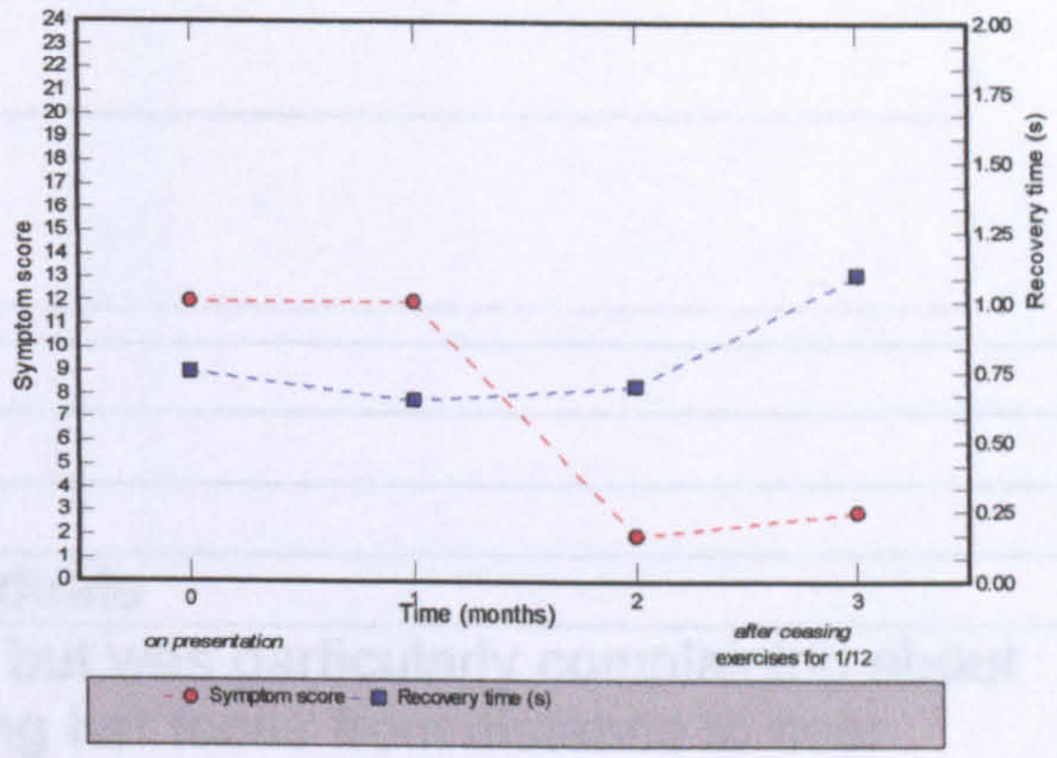


Figure 7.18. Changes in symptom score and recovery time over the treatment period for subject siwh.

Figure 7.19 shows little improvement in fusional reserves after one month of treatment with an improvement blur and break measures after a further month. This improvement appeared to be sustained after ceasing the exercises for one month.

For this subject there was a reduction in the amplitude of associated phoria after one month and again after two months of treatment. This is illustrated in Figure 7.20 which also shows the slight regression of both associated phoria amplitude and symptoms after cessation of the exercises for one month.

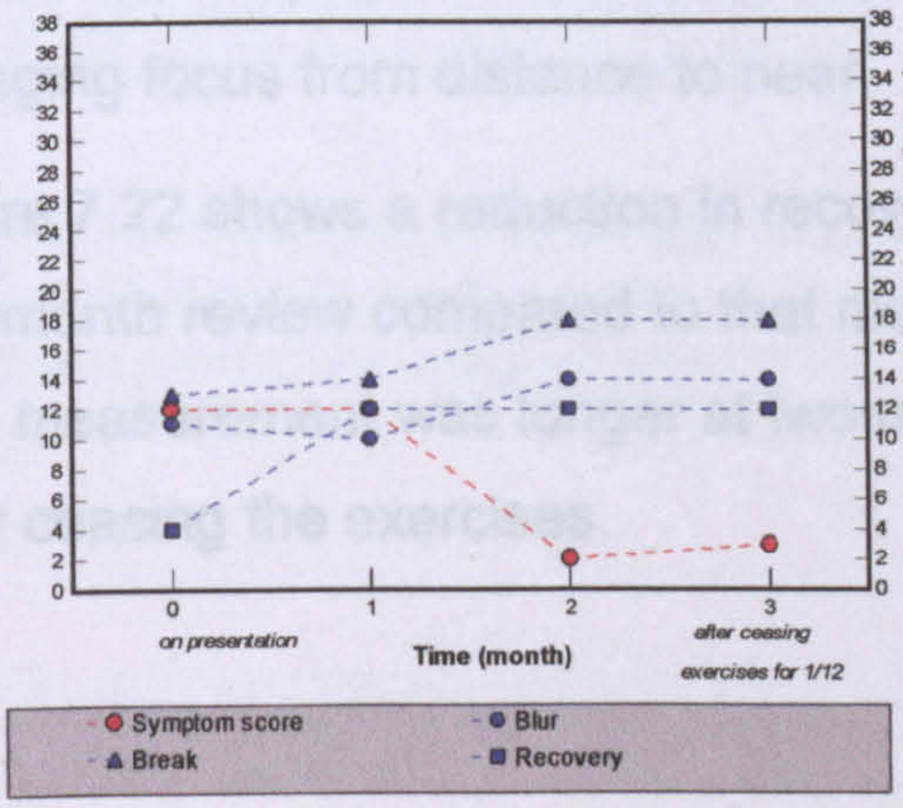


Figure 7.19. Changes in symptom score and fusional reserves over the treatment period for subject siwh.

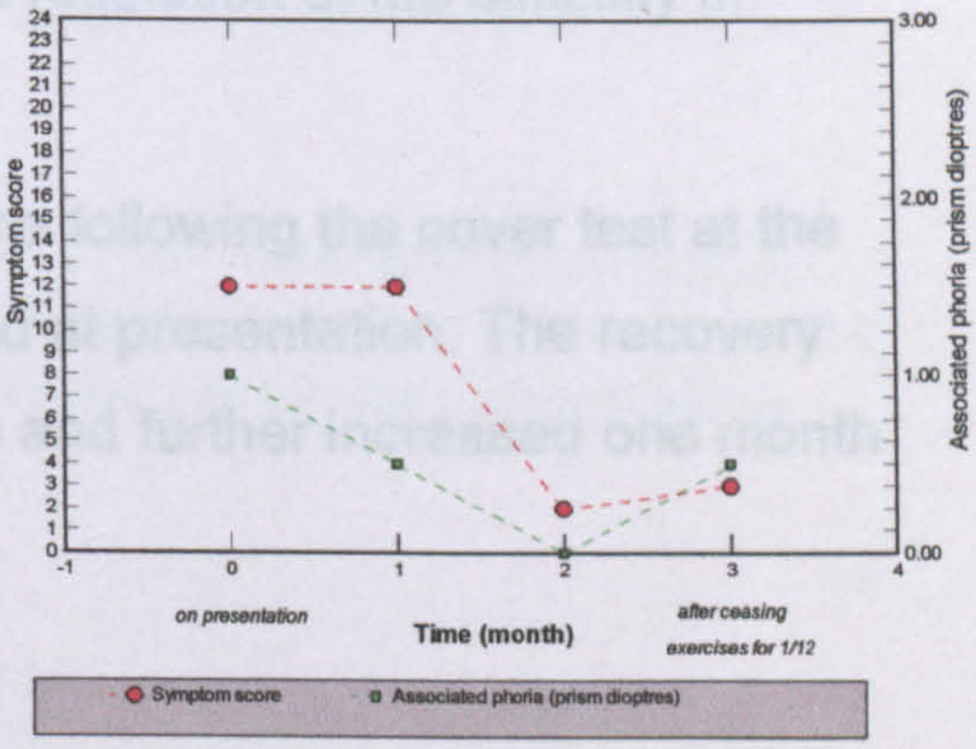


Figure 7.20. Changes in symptom score and associated phoria over the treatment period for subject siwh.

7.4.2.b. GROUP β

I. Case history

Subject	*aisha*
Age	21 years-old
Sex	Female
Occupation	Optometry undergraduate
Symptoms	Low symptom score but was particularly complaining about a difficulty in changing her focus from distance to near vision and vice versa. This was diagnosed as inertia of accommodation
Reported compliance	Compliance with the treatment regime was uncertain as she reported that in the first month she had not carried out the exercises every day
Resolution of symptoms	Worsened
Follow-up	Symptom score highest one month after ceasing exercises
Outcome	This subject considered the exercise regime to be a success with regards to the presumed inertia of accommodation. The increase in other near vision symptoms were attributed by the subject to stress

Figure 7.21 shows an improvement in NPC after one month accompanied by an increase in symptom score. The NPC remained stable at both two months and at the review one month after cessation of exercises. She reported the highest symptom score one month after cessation of the exercises. Despite the increased symptom score, she reported a resolution of the difficulty in changing focus from distance to near.

Figure 7.22 shows a reduction in recovery time following the cover test at the one month review compared to that measured at presentation. The recovery time measurement was longer at two months and further increased one month after ceasing the exercises.

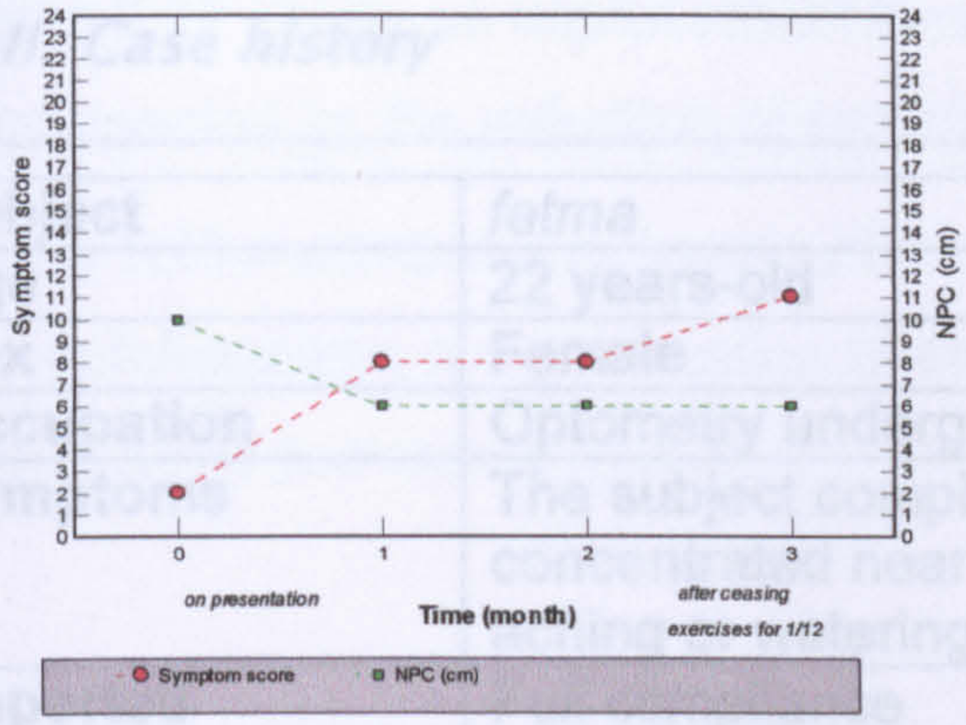


Figure 7.21. Changes in symptom score and NPC over the treatment period for subject aisha.

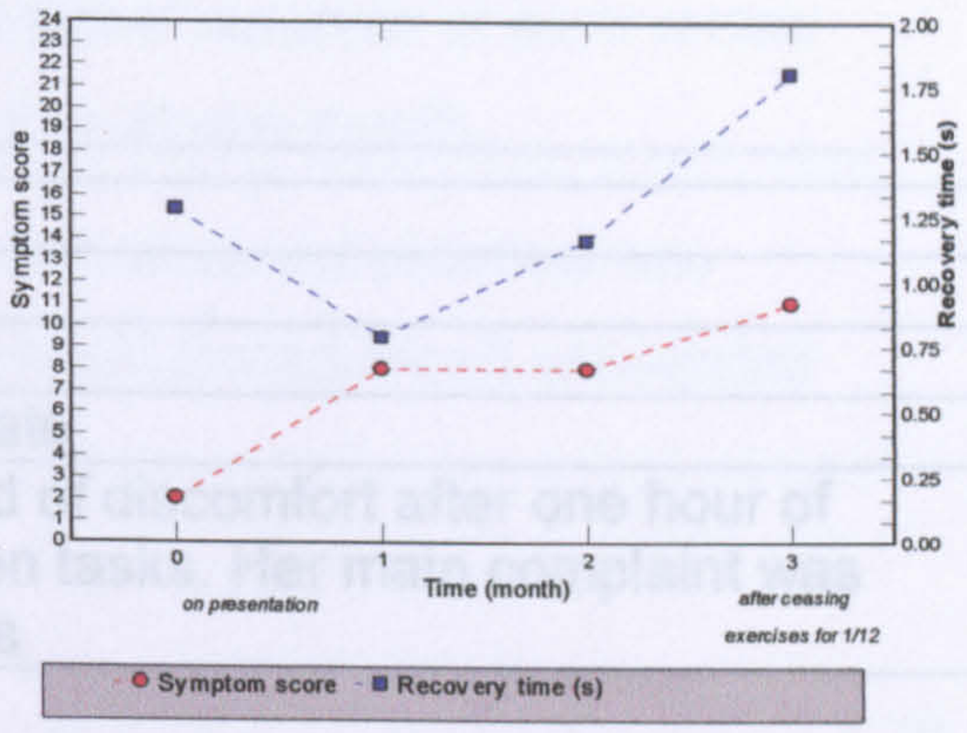


Figure 7.22. Changes in symptom score and recovery time over the treatment period for subject aisha.

Figures 7.23 and 7.24 show variable measures of fusional reserves and associated phoria over the treatment period.

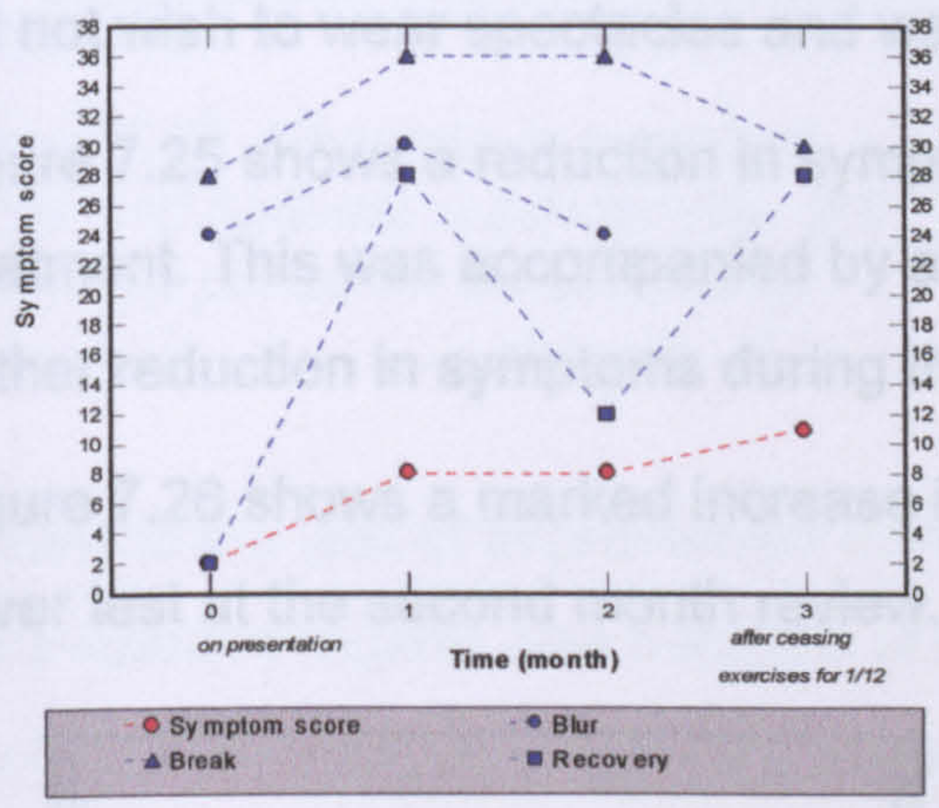


Figure7.23. Changes in symptom score and fusional reserves over the treatment period for subject aisha.

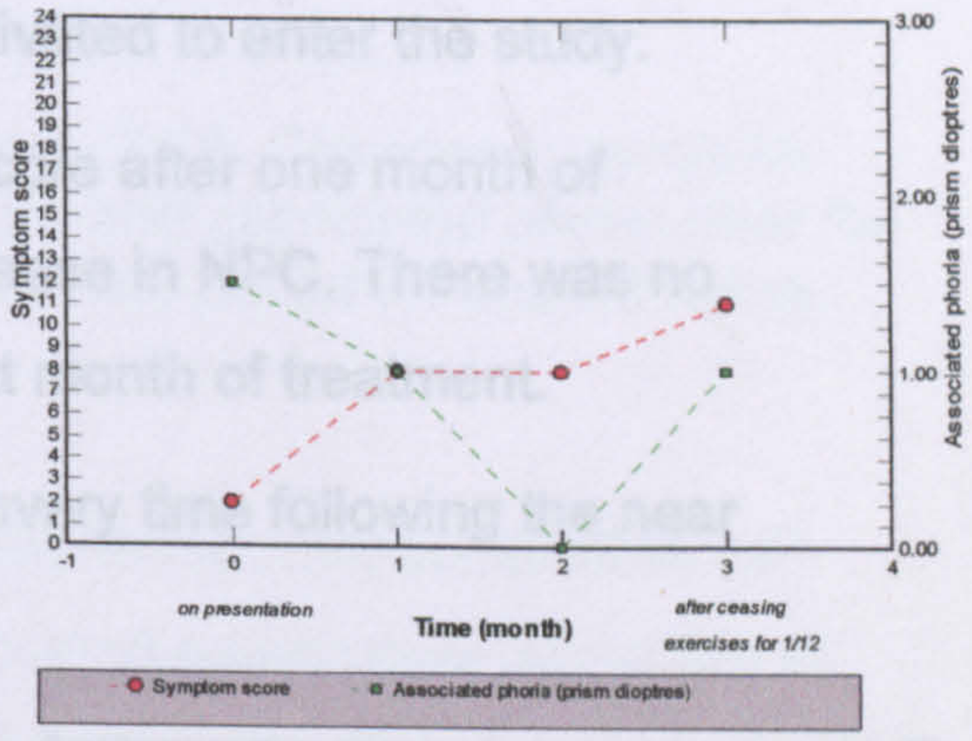


Figure 7.24. Changes in symptom score and associated phoria over the treatment period for subject aisha.

II. Case history

Subject	*fatma*
Age	22 years-old
Sex	Female
Occupation	Optometry undergraduate
Symptoms	The subject complained of discomfort after one hour of concentrated near vision tasks. Her main complaint was aching or watering eyes
Reported compliance	Full compliance
Resolution of symptoms	Part-resolution with exercises
Follow-up	-
Outcome	Exercises unsuccessful but symptoms alleviated by refractive intervention

This subject's refraction was R. +0.75/-0.25 x 90 L. plano. Although this degree of anisometropia did not comply with the study's criteria, the subject did not wish to wear spectacles and was motivated to enter the study.

Figure 7.25 shows a reduction in symptom score after one month of treatment. This was accompanied by an increase in NPC. There was no further reduction in symptoms during the next month of treatment.

Figure 7.26 shows a marked increase in recovery time following the near cover test at the second month review.

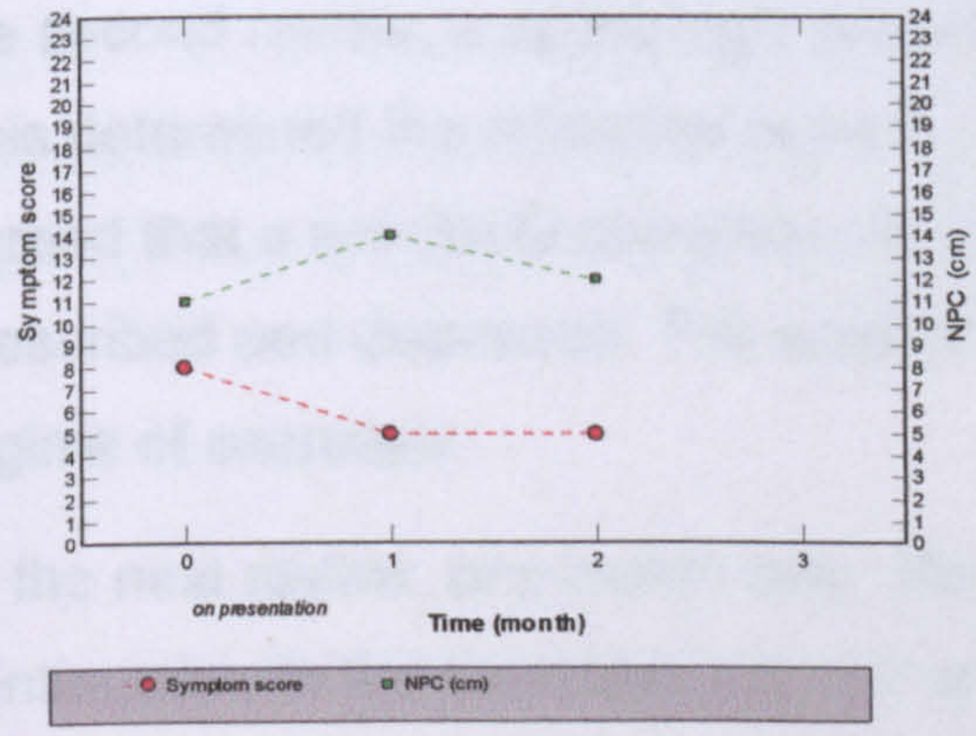


Figure 7.25. Changes in symptom score and NPC over the treatment period for subject fatma.

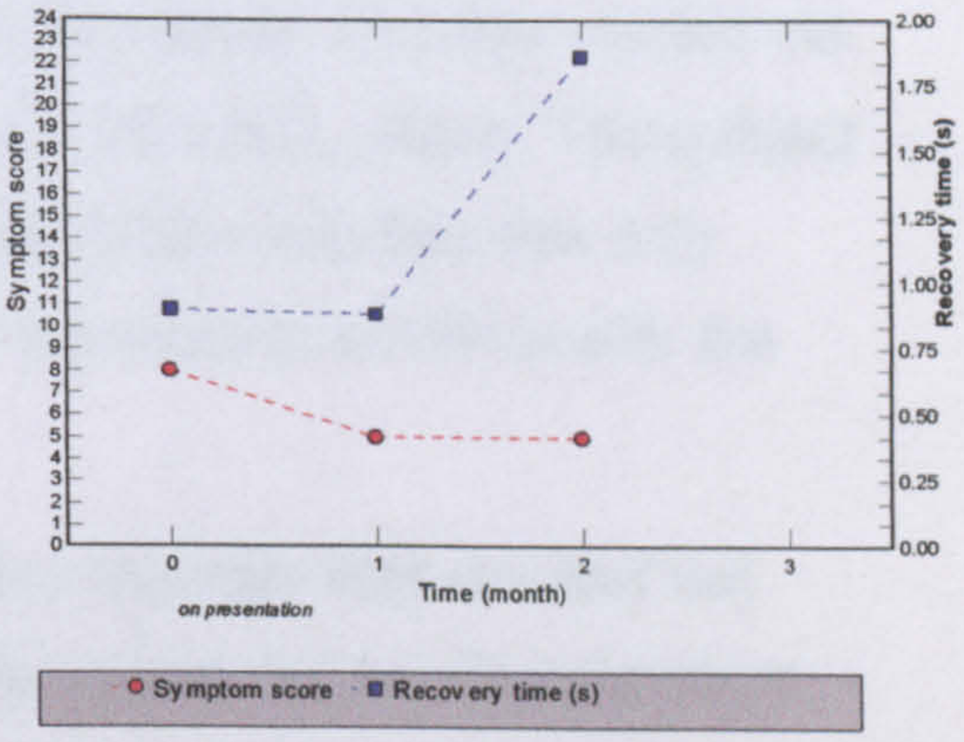


Figure 7.26. Changes in symptom score and recovery time over the treatment period for subject fatma.

Figure 7.27 shows an improvement in near fusional reserves at each review accompanied by the reduction of symptom score at one month.

Figure 7.28 describes a reduction of exophoric associated phoria at one month followed by a manifestation of an esophoric (paradoxical) associated phoria at the two month review.

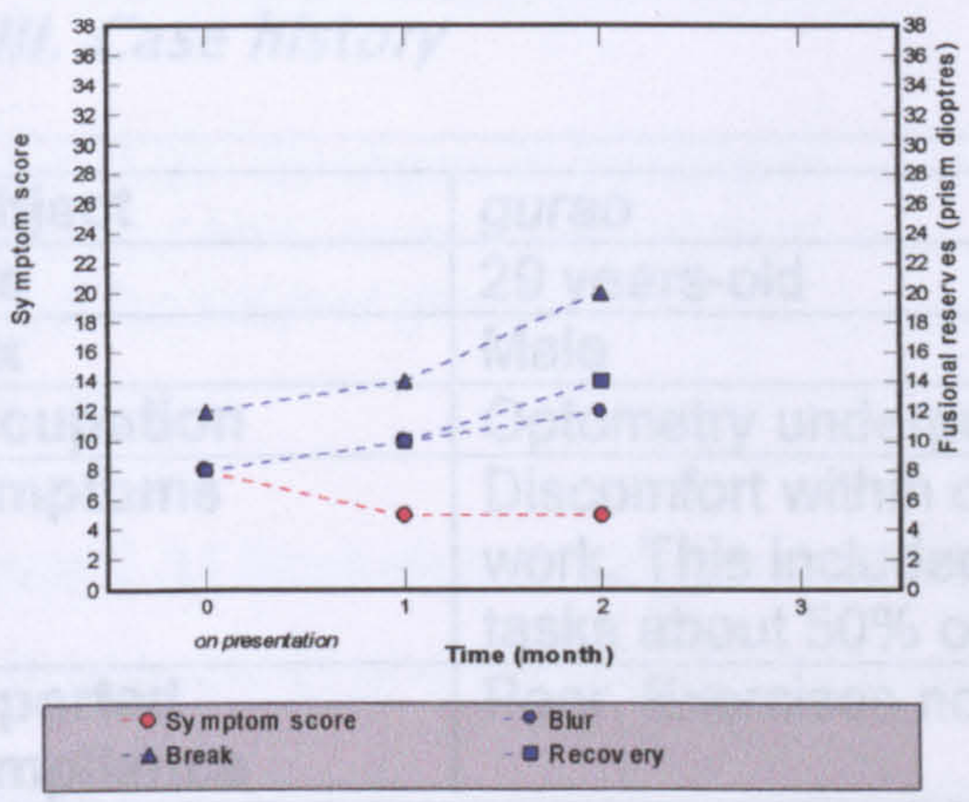


Figure 7.27. Changes in symptom score and fusional reserves over the treatment period for subject fatma.

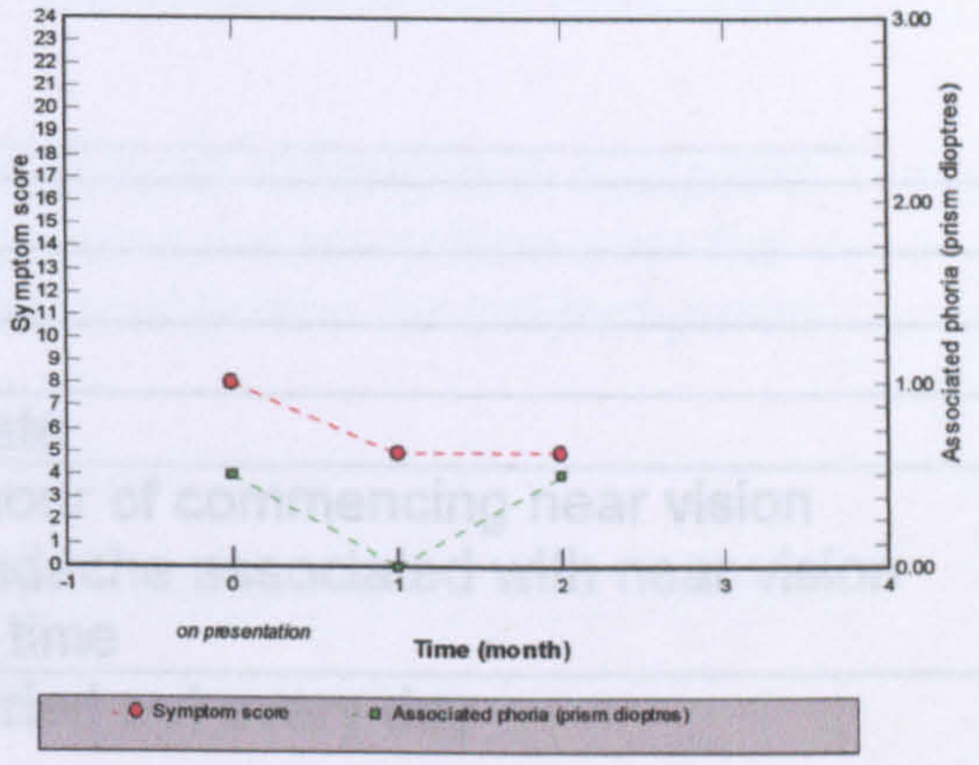


Figure 7.28. Changes in symptom score and associated phoria over the treatment period for subject fatma.

In view of the lack of improvement in NPC despite reported compliance with the treatment regime, and because of the reported paradoxical retinal slip at the second review, a cycloplegic refraction (Tropicamide 1%) was carried out. This determined the refraction to be R. +1.00/-0.25 x 90 L. plano. The subject agreed that a spectacle correction would be advisable and this was duly prescribed and dispensed. The subject was instructed to continue with the regime of exercises.

At the next review, one month later, the subject reported that she had not continued with the exercises but had worn the spectacles for all close work. There had been an almost complete resolution of her symptom (symptom score = 1). Her NPC was 12 cm. There was no reported associated phoria. The subject requested discharge from the study.

Comment

Whilst this subject had manifested a remote NPC, the clinical picture was also complicated by the uncorrected anisometropia. Correction of the latter coincided with a reported reduction of symptoms. The regime of orthoptic exercises had not produced a comparable satisfactory reduction in symptoms.

III. Case history

Subject	*gurao*
Age	29 years-old
Sex	Male
Occupation	Optometry undergraduate
Symptoms	Discomfort within one hour of commencing near vision work. This included headache associated with near vision tasks about 50% of the time
Reported compliance	Poor. Exercises not carried out every day
Resolution of symptoms	Reduction but not resolution of symptoms
Follow-up	Carried out exercises for an additional month before requesting discharge
Outcome	Unsuccessful

Figure 7.29 shows a reduction in NPC accompanied by an improvement in symptom score.

Figure 7.30 shows a reduction in recovery time following the cover test accompanied by the reduction in symptom score. The recovery time datum for the three month review represents the left eye only.

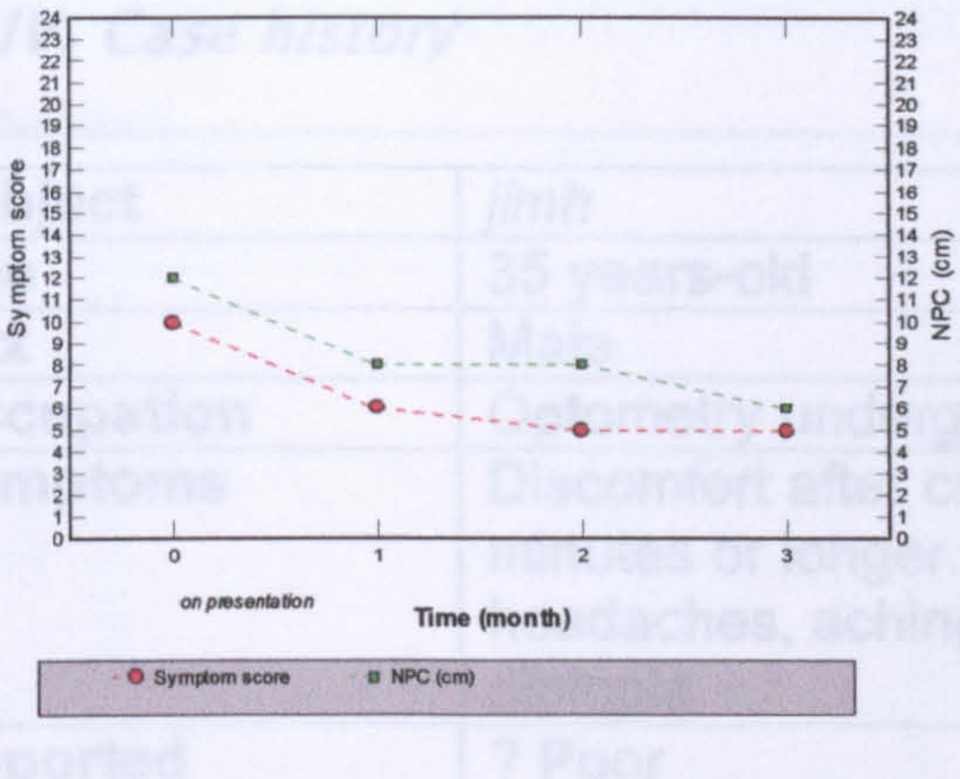


Figure 7.29. Changes in symptom score and NPC over the treatment period for subject gurao.

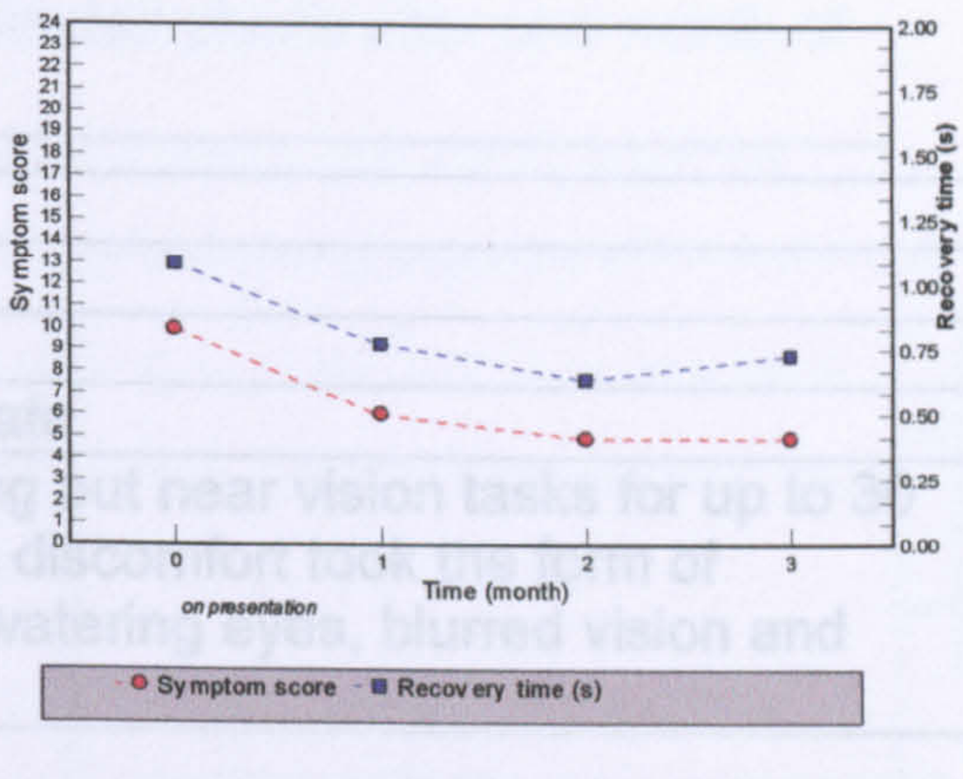


Figure 7.30. Changes in symptom score and recovery time over the treatment period for subject gurao.

Figure 7.31 illustrates an improvement in the fusional reserve break measure after one month and two months of treatment.

Figure 7.32 shows a reduction in the amplitude of associated phoria over the first two months of treatment. There was no further improvement after a third month of exercises and the subject left the study still manifesting an associated phoria and complaining of noticeable symptoms.

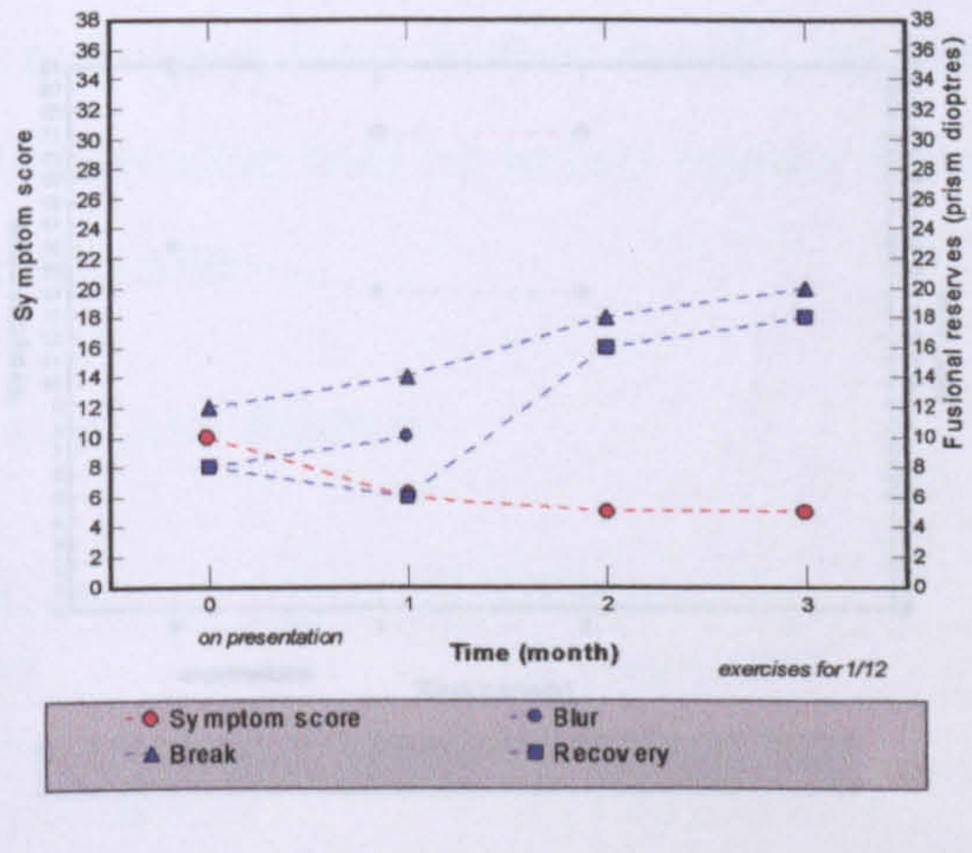


Figure 7.31. Changes in symptom score and fusional reserves over the treatment period for subject gurao.

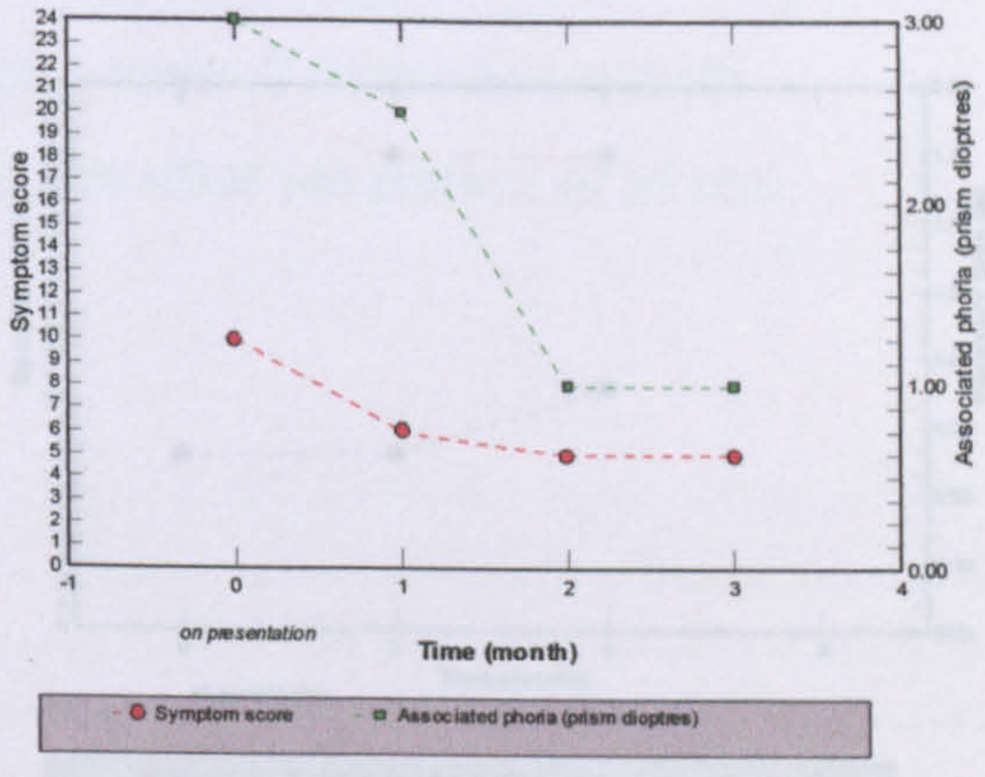


Figure 7.32. Changes in symptom score and associated phoria over the treatment period for subject gurao.

IV. Case history

Subject	*jimh*
Age	35 years-old
Sex	Male
Occupation	Optometry undergraduate
Symptoms	Discomfort after carrying out near vision tasks for up to 30 minutes or longer. This discomfort took the form of headaches, aching or watering eyes, blurred vision and diplopia
Reported compliance	? Poor
Resolution of symptoms	No significant improvement
Follow-up	Discharged himself
Outcome	Unsuccessful

Figure 7.33 illustrates the high symptom score which only showed a marginal improvement over the two month treatment period. This was accompanied by a very modest improvement in NPC.

Figure 7.34 shows a slight increase in recovery time following the cover test over the same period

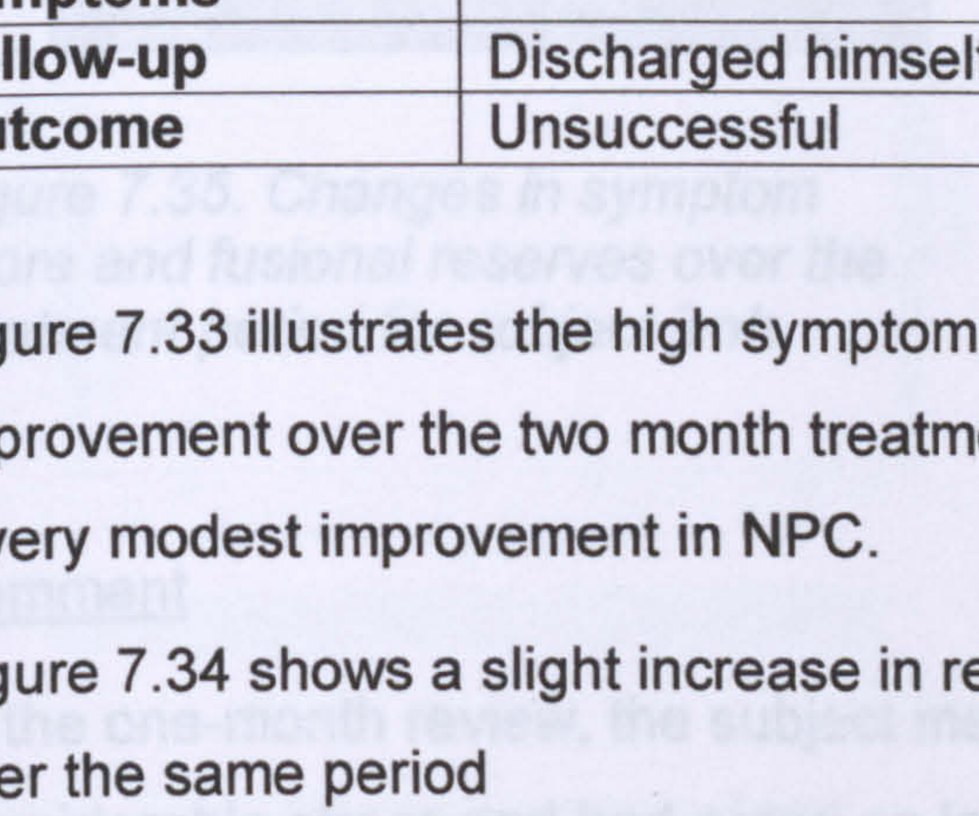


Figure 7.33. Changes in symptom score and NPC over the treatment period for subject jimh.

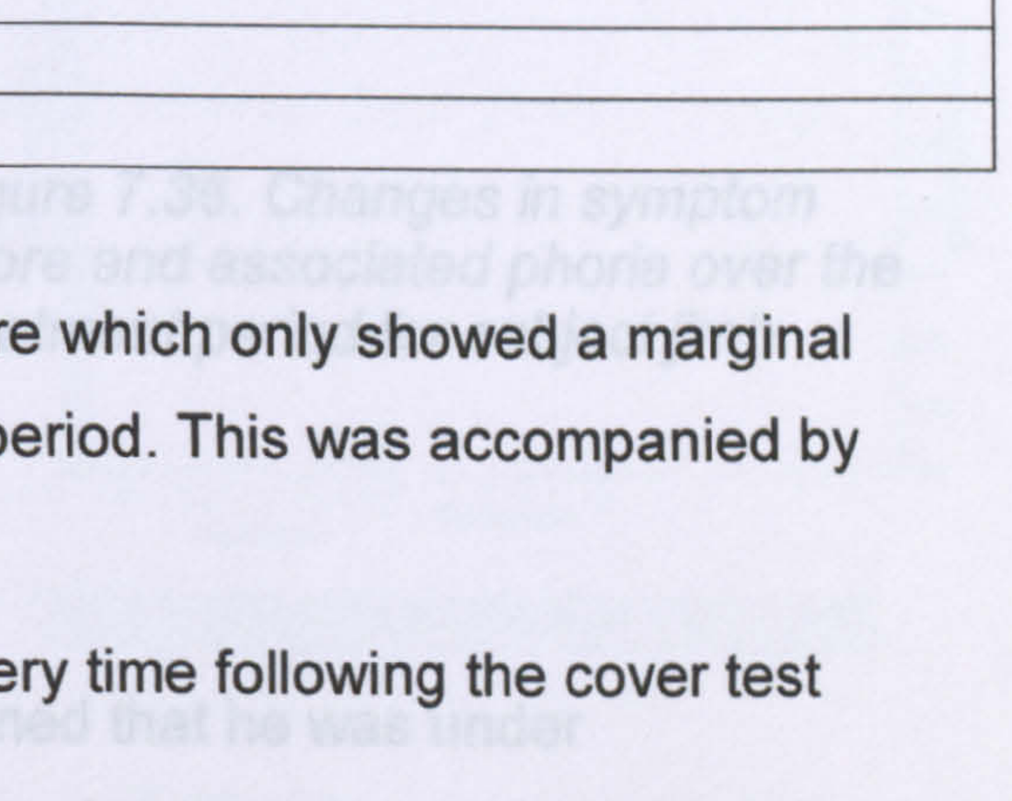


Figure 7.34. Changes in symptom score and recovery time over the treatment period for subject jimh.

Figure 7.35 shows a modest increase in fusional reserves at the one month review followed by a marked regression at the two month review.

Figure 7.36 shows the elimination of an associated phoria after one month of treatment.

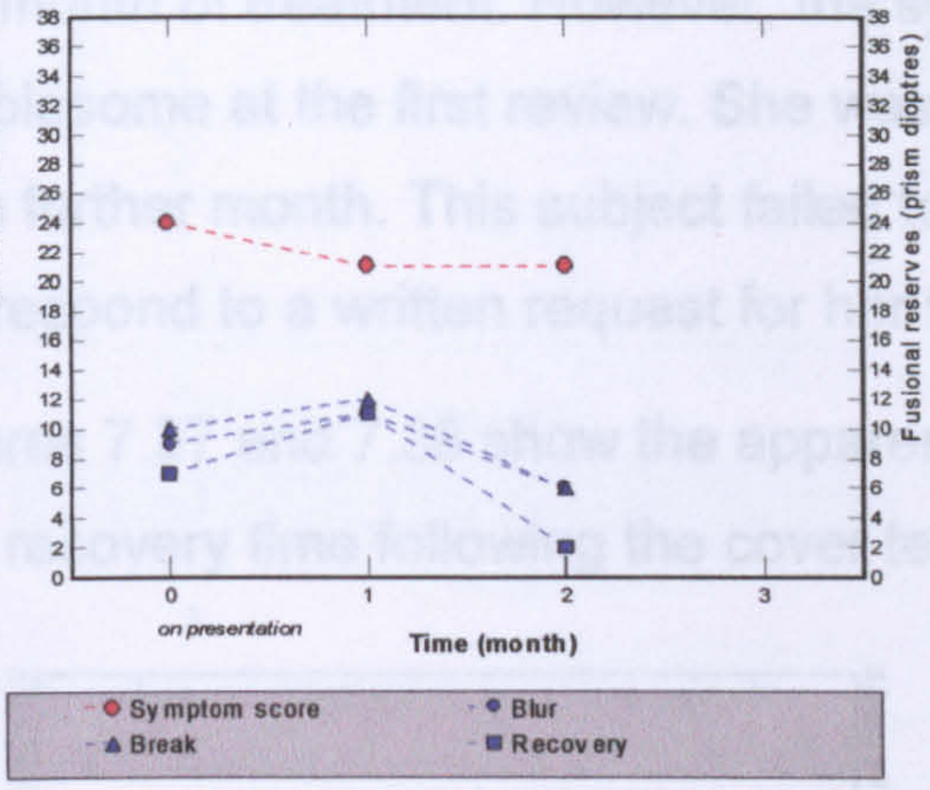


Figure 7.35. Changes in symptom score and fusional reserves over the treatment period for subject jimh.

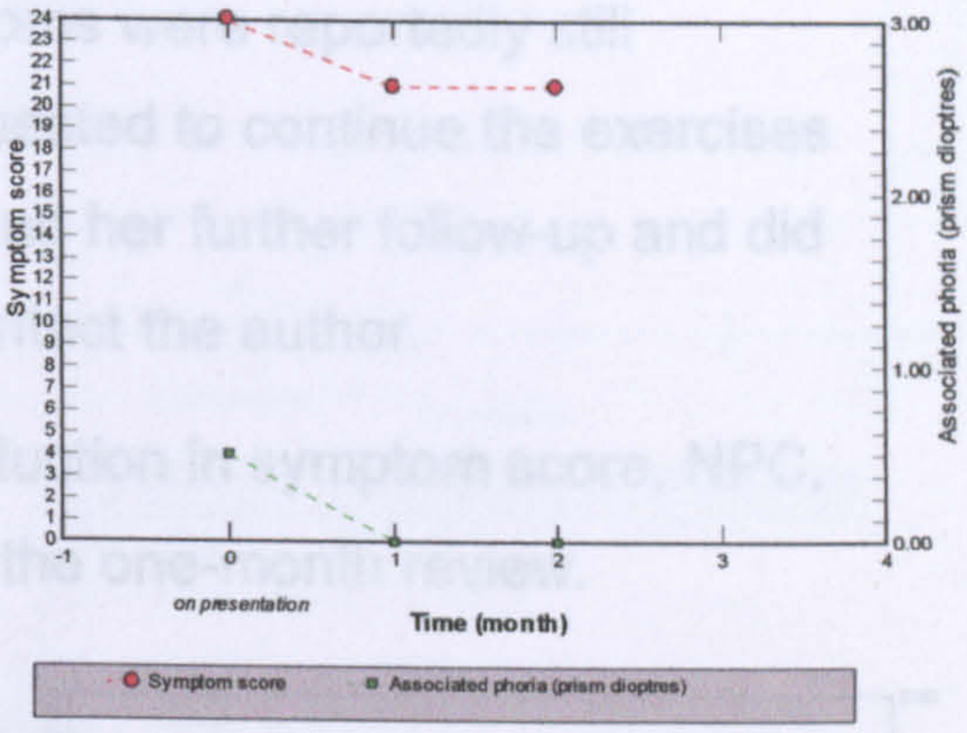


Figure 7.36. Changes in symptom score and associated phoria over the treatment period for subject jimh.

Comment

At the one-month review, the subject mentioned that he was under considerable stress and had noted an increase in symptoms as his forthcoming marriage approached. Whilst he was encouraged to continue with the exercises for a further month, he discharged himself at the two month review stating that he would review his symptoms after his period of stress had passed.

V. Case history

Subject	*kadep*
Age	18 years-old
Sex	Female
Occupation	School student
Symptoms	Discomfort after 15 minutes of close work .This took the form of severe headaches, aching and/or watering eyes, blurred vision, and tiredness or loss of concentration
Reported compliance	Uncertain
Resolution of symptoms	Reduction in symptom score after 1 month
Follow-up	Nil
Outcome	Unsuccessful

This subject showed a reduction in both symptom score and NPC after the first month of treatment. However, the symptoms were reportedly still troublesome at the first review. She was requested to continue the exercises for a further month. This subject failed to attend her further follow-up and did not respond to a written request for her to contact the author.

Figures 7.37 and 7.38 show the apparent reduction in symptom score, NPC, and recovery time following the cover test at the one-month review.

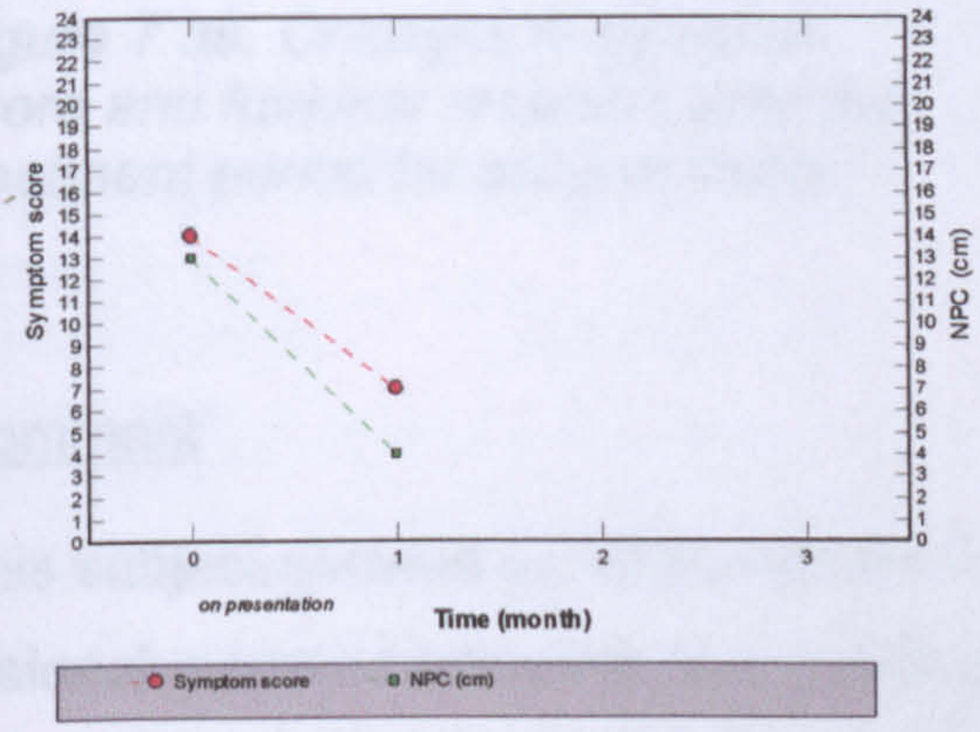


Figure 7.37. Changes in symptom score and NPC over the treatment period for subject kadep.

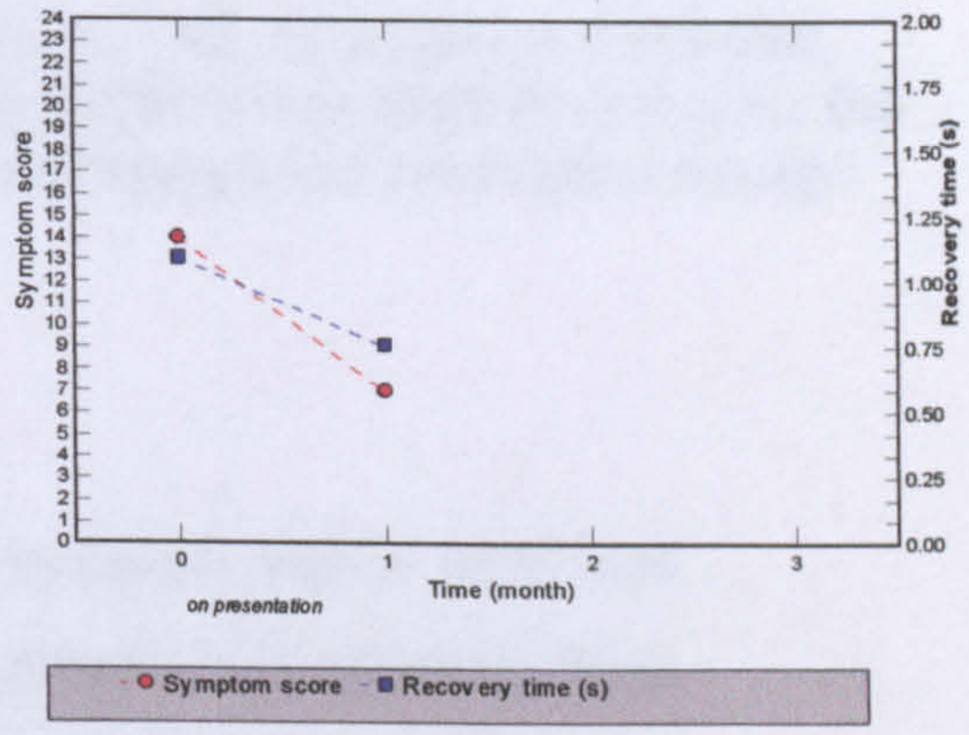


Figure 7.38. Changes in symptom score and recovery time over the treatment period for subject kadep.

Figure 7.39 illustrates an increase in blur/break/ and recovery fusional reserve measures at the one-month review.

The subject's associated phoria appeared alleviated at the one-month review and this is shown in Figure 7.40.

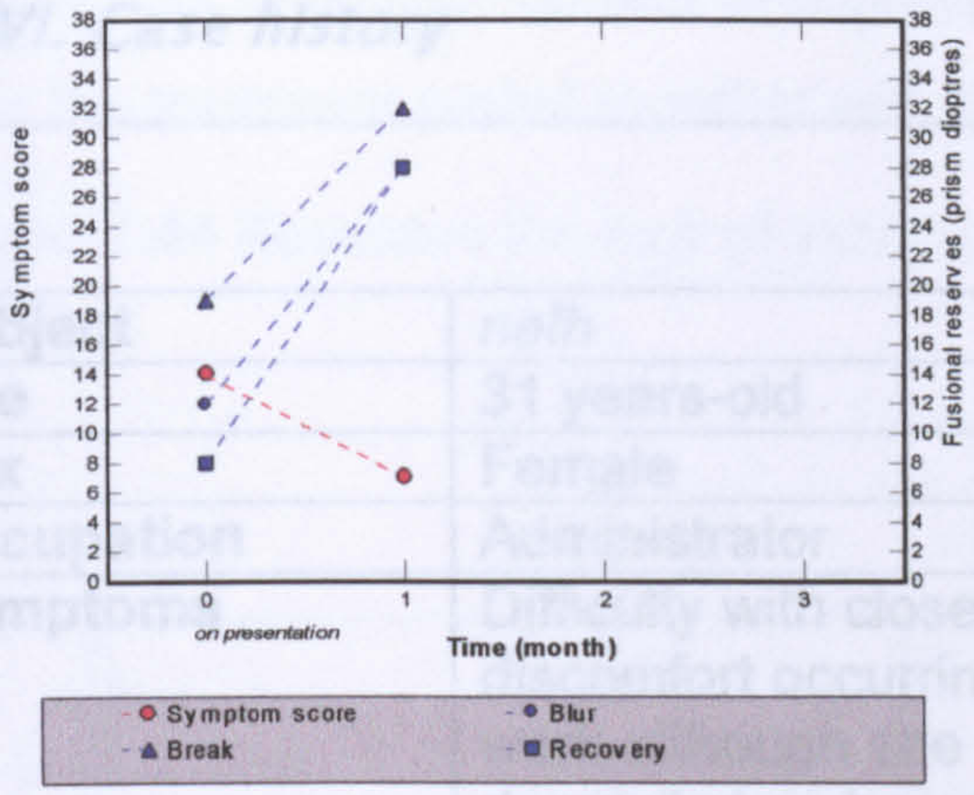


Figure 7.39. Changes in symptom score and fusional reserves over the treatment period for subject kadep.

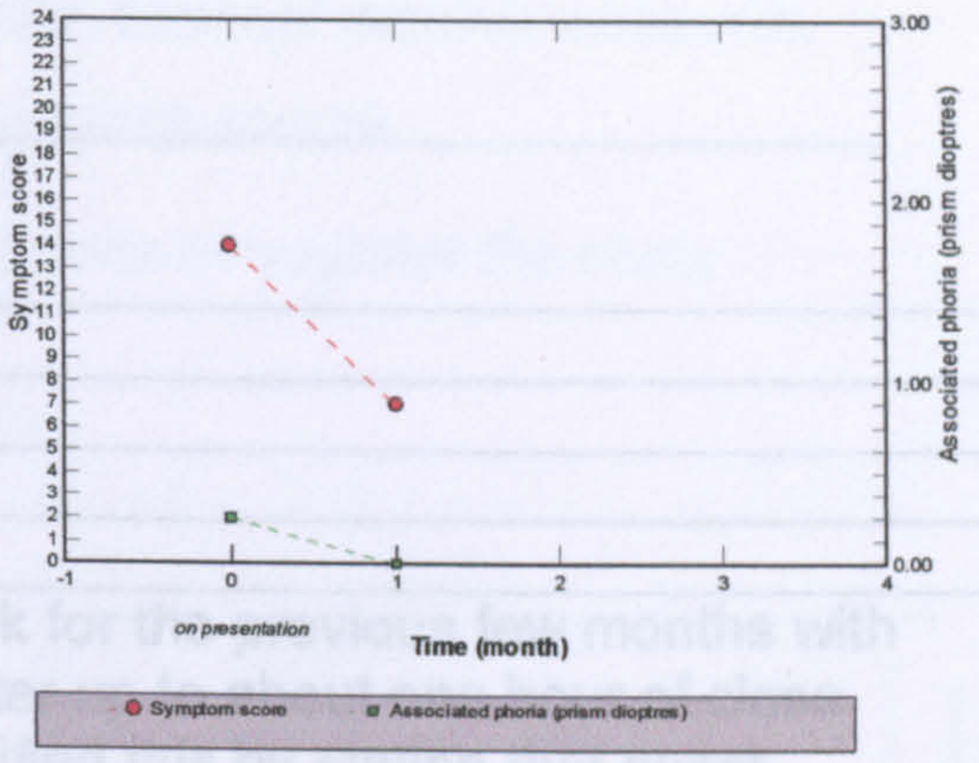


Figure 7.40. Changes in symptom score and associated phoria over the treatment period for subject kadep.

Comment

This subject showed an improvement in her symptom score, NPC and fusional reserves following one month of treatment. The recovery time following the near cover test was also shorter at the one-month review. Despite this apparent progress, the subject still found her symptoms bothersome at the one month review and the treatment cannot therefore be considered to have been successful.

VI. Case history

Subject	*neth*
Age	31 years-old
Sex	Female
Occupation	Administrator
Symptoms	Difficulty with close work for the previous few months with discomfort occurring after up to about one hour of close work although she qualified this by stating that onset depended on how tired she was. Fluorescent lighting exacerbated the discomfort which took the form of occasional headaches, aching or watering eyes, blurred vision, and double vision
Reported compliance	Full compliance
Resolution of symptoms	Unsatisfactory
Follow-up	-
Outcome	Unsuccessful

Figure 7.41 shows no improvement in NPC or symptom score after one month of treatment.

Figure 7.42 describes the slight decrease in recovery time following the near cover test at the one month review followed by an increase in at the two month review.

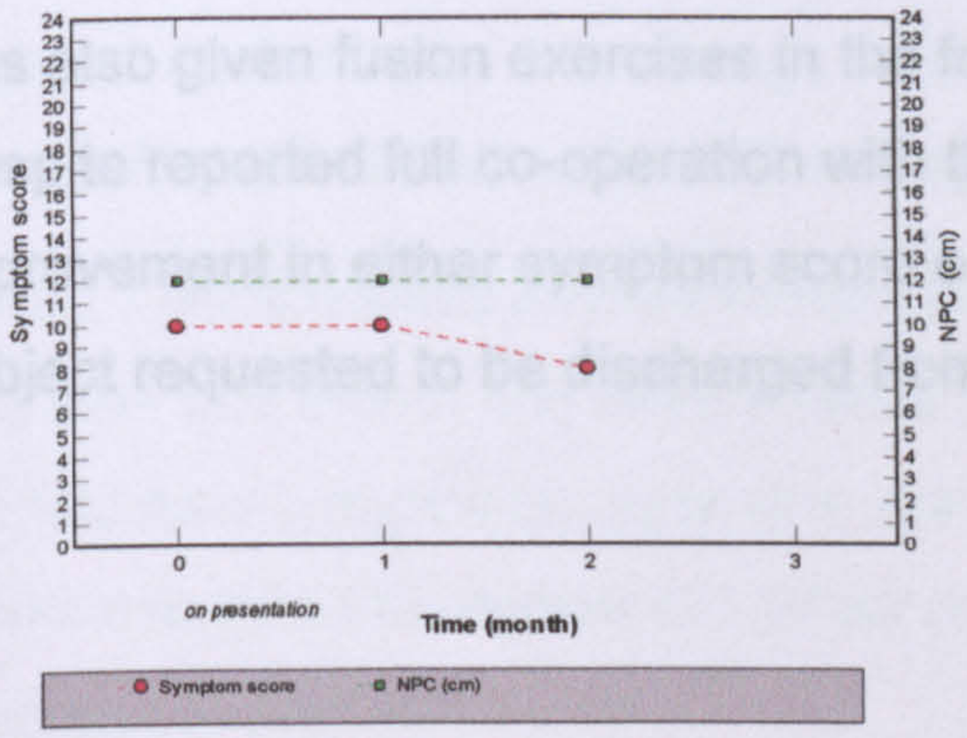


Figure 7.41. Changes in symptom score and NPC over the treatment period for subject neth.

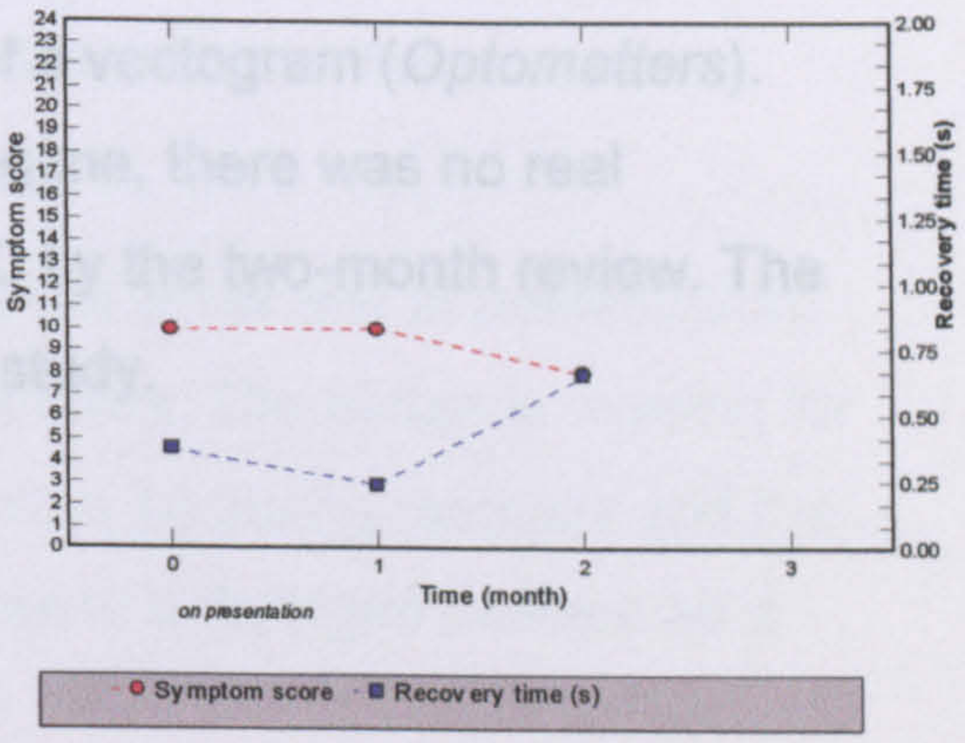


Figure 7.42. Changes in symptom score and recovery time over the treatment period for subject neth.

Figure 7.43 illustrates the lack of improvement in fusional reserve measures over the treatment period together with the symptom scores.

Figure 7.44 illustrates the lack of associated phoria throughout the study period.

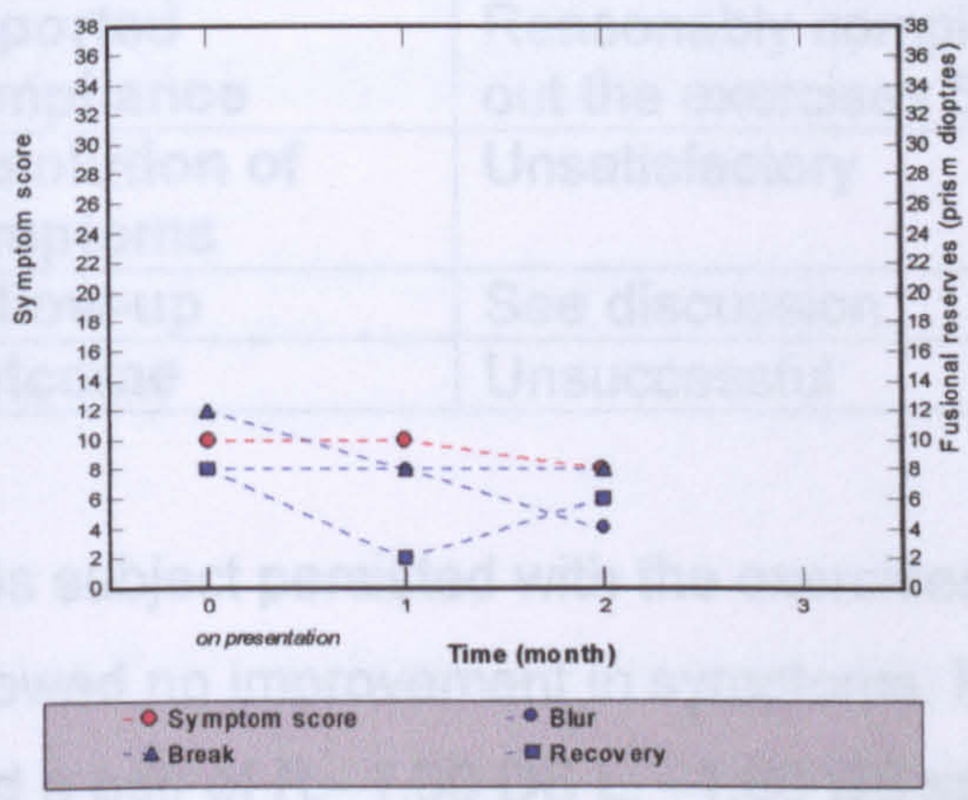


Figure 7.43. Changes in symptom score and fusional reserves over the treatment period for subject neth.

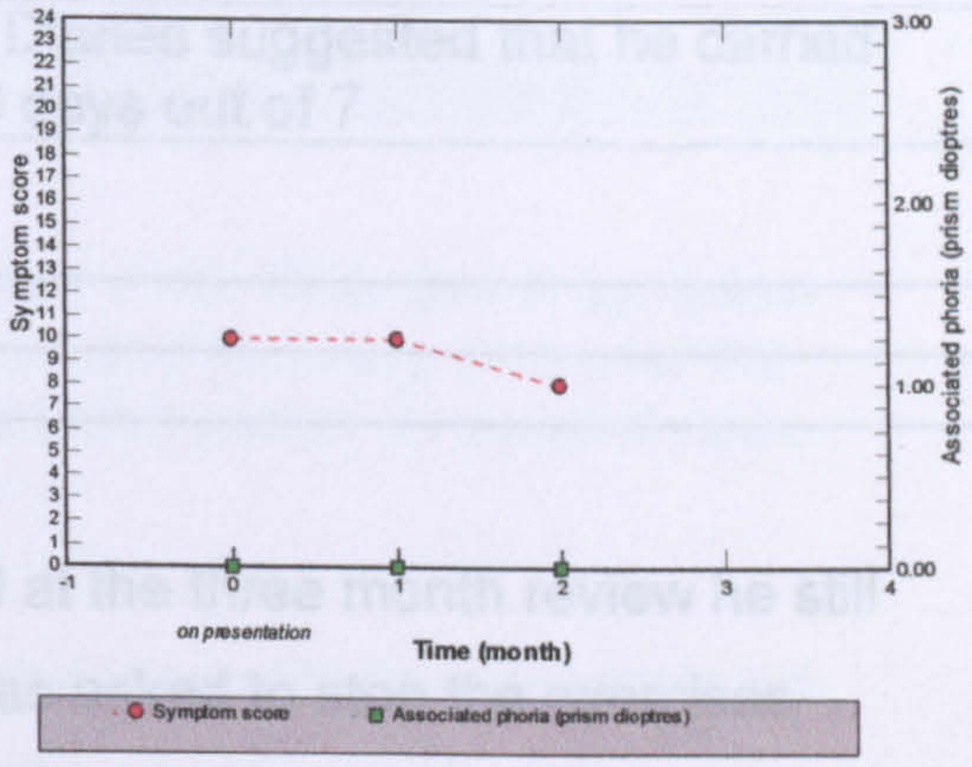


Figure 7.44. Changes in symptom score and associated phoria over the treatment period for subject neth.

Comment

At the one-month review, the subject requested an alternative intervention and was requested to continue with the previously prescribed orthoptic regime but was also given fusion exercises in the form of a vectogram (*Optomatters*). Despite reported full co-operation with this regime, there was no real improvement in either symptom score or NPC by the two-month review. The subject requested to be discharged from the study.

VII. Case history

Subject	vicox
Age	23 years-old
Sex	Male
Occupation	Optometry undergraduate
Symptoms	Discomfort, including headaches, blurred vision, diplopia and tired eyes, following two hours of close
Reported compliance	Reasonably compliant. Diaries suggested that he carried out the exercises 5 or 6 days out of 7
Resolution of symptoms	Unsatisfactory
Follow-up	See discussion
Outcome	Unsuccessful

This subject persisted with the exercises and at the three month review he still showed no improvement in symptoms. He was asked to stop the exercises and a pair of R –1.00 DS L. –1.00 DS spectacles were prescribed with the intention of utilising accommodative convergence to alleviate the associated phoria. These were to be worn for all close work.

At the review one month later the subject reported that the spectacles gave him a headache after 15 minutes of use and that he had not worn them more than three or four times. However, he added that he was uncertain whether or not a concurrent throat infection/cold was associated with the headache. The subject was discharged at this time.

Figure 7.45 illustrates little change in symptom score over the treatment period. A small reduction in NPC was apparent.

Figure 7.46 illustrates recovery time following the cover test at presentation, and after one month and three months of exercises. The datum is missing for the two month review because one eye blinked a lot during recovery and the fellow eye failed to recover but rather remained in a diverged position as a manifest strabismus.

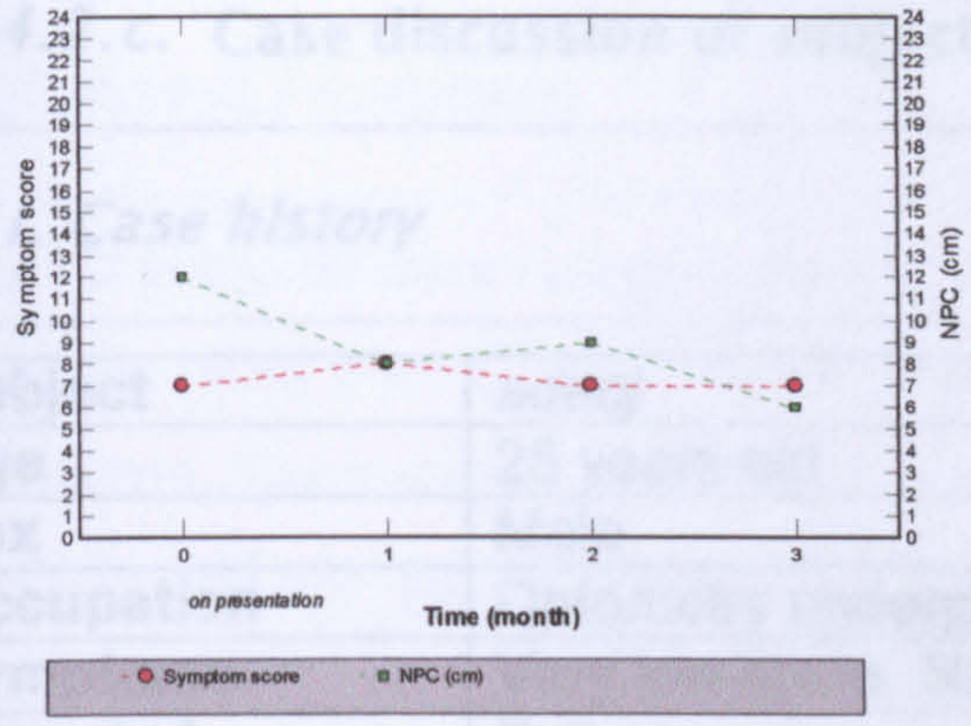


Figure 7.45. Changes in symptom score and NPC over the treatment period for subject vicox.

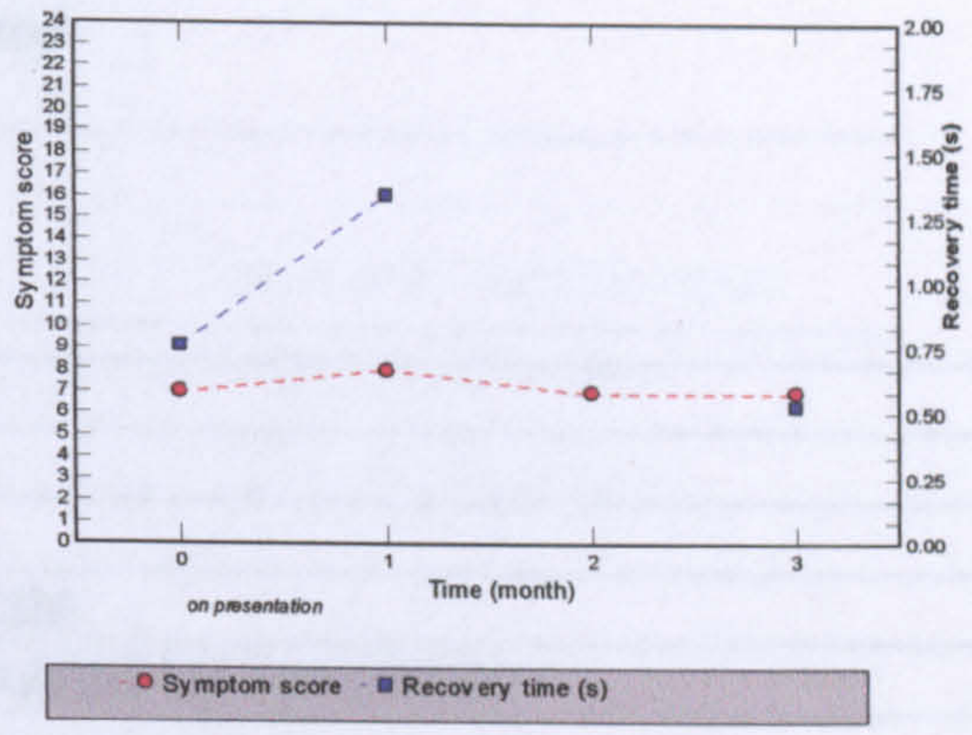


Figure 7.46. Changes in symptom score and recovery time over the treatment period for subject vicox.

Figure 7.47 illustrates changes in fusional reserve amplitudes accompanied by change in symptom score over the treatment period.

Figure 7.48 illustrates the amplitude of associated phoria that increased over the treatment period.

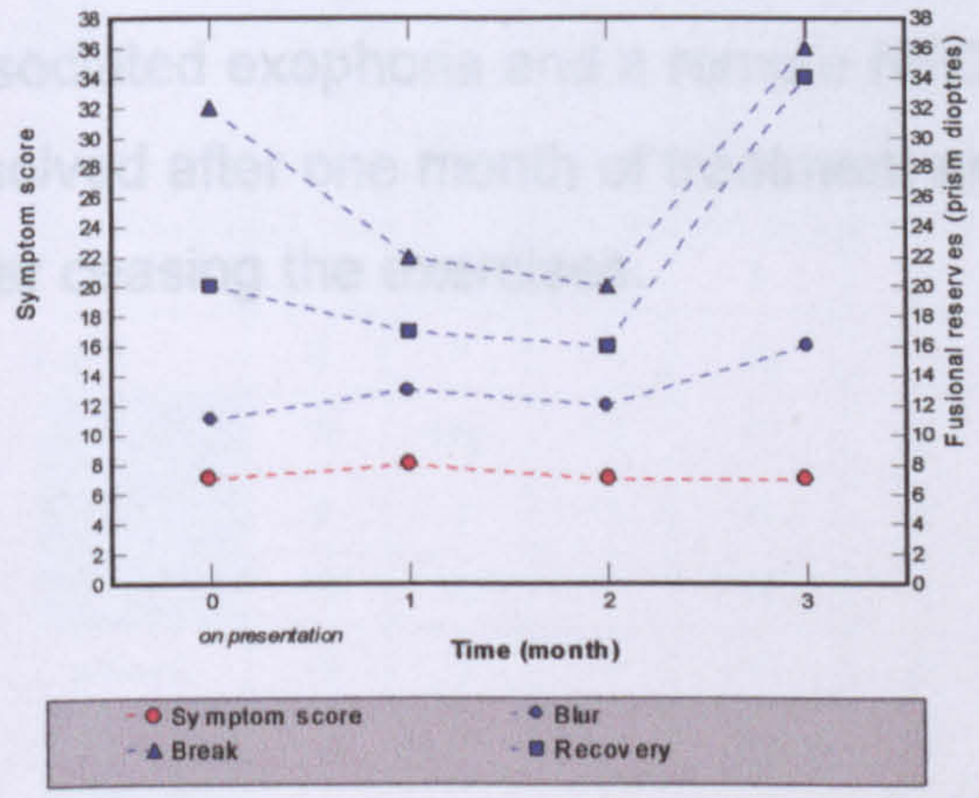


Figure 7.47. Changes in symptom score and fusional reserves over the treatment period for subject vicox.

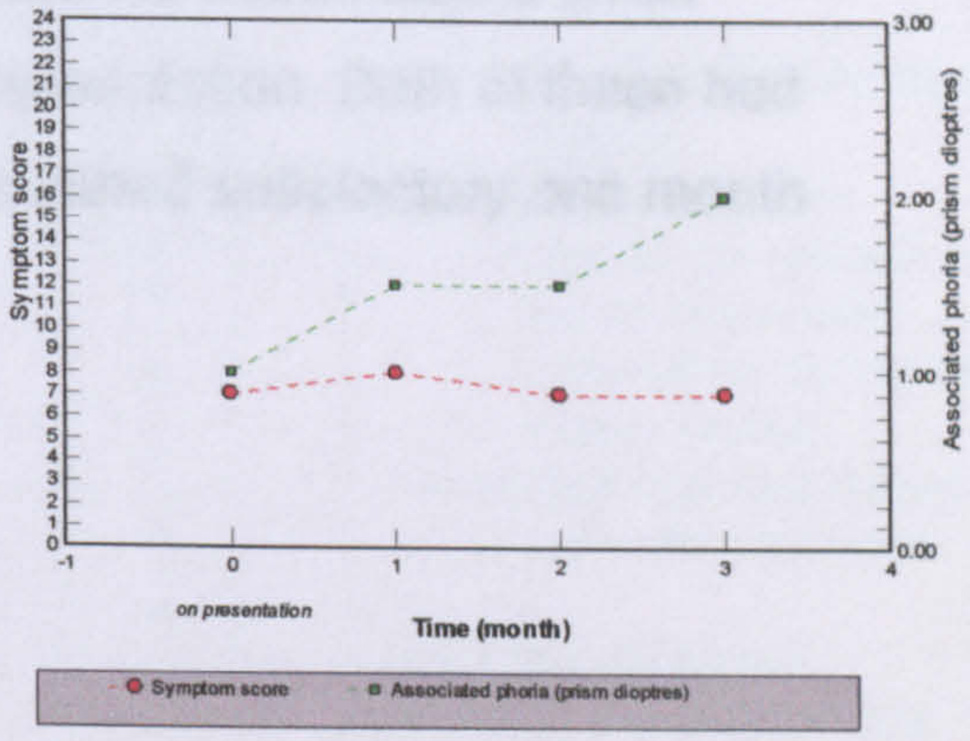


Figure 7.48. Changes in symptom score and associated phoria over the treatment period for subject vicox.

Comment

It was concluded that the regime of orthoptic exercises used in this study was not efficacious for this subject.

7.4.2.c. Case discussion of subject *adeoj*

I. Case history

Subject	*adeoj*
Age	25 years-old
Sex	Male
Occupation	Optometry undergraduate
Symptoms	Very low score. Not troubled by symptoms
Reported compliance	Full
Resolution of symptoms	Not applicable
Follow-up	See discussion
Outcome	Clinical signs satisfactory

This subject is included in isolation from the other CI subjects in this study because he had been referred to the study solely because of a known CI. As previously discussed, his symptom score was very low and he was not troubled by the symptoms. There was no apparent suppression at near as tested with the near Mallett unit binocularity test. He manifested a small associated exophoria and a remote NPC at presentation. Both of these had resolved after one month of treatment and remained satisfactory one month after ceasing the exercises.

7.4.3 Non-CI subjects

Non-CI subjects were categorised into those with a horizontal ocular motor anomaly other than CI and those with apparently normal ocular motor measures. The subjects in the latter group were included as anecdotal controls and are highlighted in Table 7.9.

7.4.3.a. Symptom score

Table 7.9 describes changes in symptom score after one month and, where applicable, two months of orthoptic treatment had been carried out. Symptom scores at one month after cessation of exercises are included, where available. One subject continued with treatment for a period longer than two months and the additional data is included in the case discussion for that subject.

Subject	Symptom score at presentation (A)	Symptom score after 1 month (B)	Symptom difference (B – A)	Symptom score after 2 months of exercises (C)	Symptom difference (C – A)	Symptom score 1 month after ceasing exercises (D)	Regression of symptoms (D – B or C)
airey	6	7	+1	3	-3	na	na
arif	8	7	-1	na	na	na	na
jadoo	12	14ψ	+2	9	-3	na	na
mayli	5	9	+4	8	+3	5	-3
nazmo	15	15	0	10	-5	na	na
shmeh	10	9	-1	11	+1	na	na
sselb	10	17	+7	na	na	na	na
vcurt	9	6	-3	0 m♠	-9	0	0
Vicup	16.5	18 *	+1.5	na	na	na	na

Na = not applicable for this subject
ψ = reported exercise regime not carried out but no close-work undertaken during the interim period
* = subject reported failure to carry out exercises

m= 3 month review
♠ = subject reported that fluorescent lights at work have been removed
name = anecdotal control subject

Table 7.9. Symptom profiles of the non-CI subjects over time.

7.4.3.b. Recovery time

Table 7.10 lists recovery time (RT), following the automated cover test, of non-CI subjects.

Subject	RE RT at presentation	LE RT at presentation	RE RT after 1 month	LE RT after 1 month	RE RT after 2 months of exercises	LE RT after 2 months	RE RT 1 month after ceasing	LE RT 1 month after ceasing
airey	0.68	1.21	1.04	1.12	2.05	0.70	na	na
arif	0.70	0.75	0.38	0.50	na	na	na	na
jadoo	0.84	nd	nd	nd	0.87	0.59	na	na
mayli	0.92	0.53	nd	0.52	nd	fc	nd	nd
nazmo	0.50	0.47	0.37	⊗	0.78	0.36	na	na
shmeh	0.71	0.69	0.66	0.43	0.56	0.36	na	na
sselb	0.59	5.52	0.57	4.69	na	na	na	na
vcurt	0.36	nd	fc	nd	0.65 m	0.34 m	fc	nd
vicup	0.43	0.83	1.03	2.85	na	na	na	na

nd = no data
na = not applicable to this subject
fc = failed to make a recovery
⊗ = phoria amplitude too small to measure recovery time
m = 3 month review
↓ = subject refused eye movement recording

Table7.10. Recovery times(s) of non-CI subjects following the automated near cover test over time.

Figure 7.49 illustrates the changes in average (right and left) recovery times following the cover test for the non-CI subjects. If bilateral data were not available, monocular data was used for the purposes of this figure.

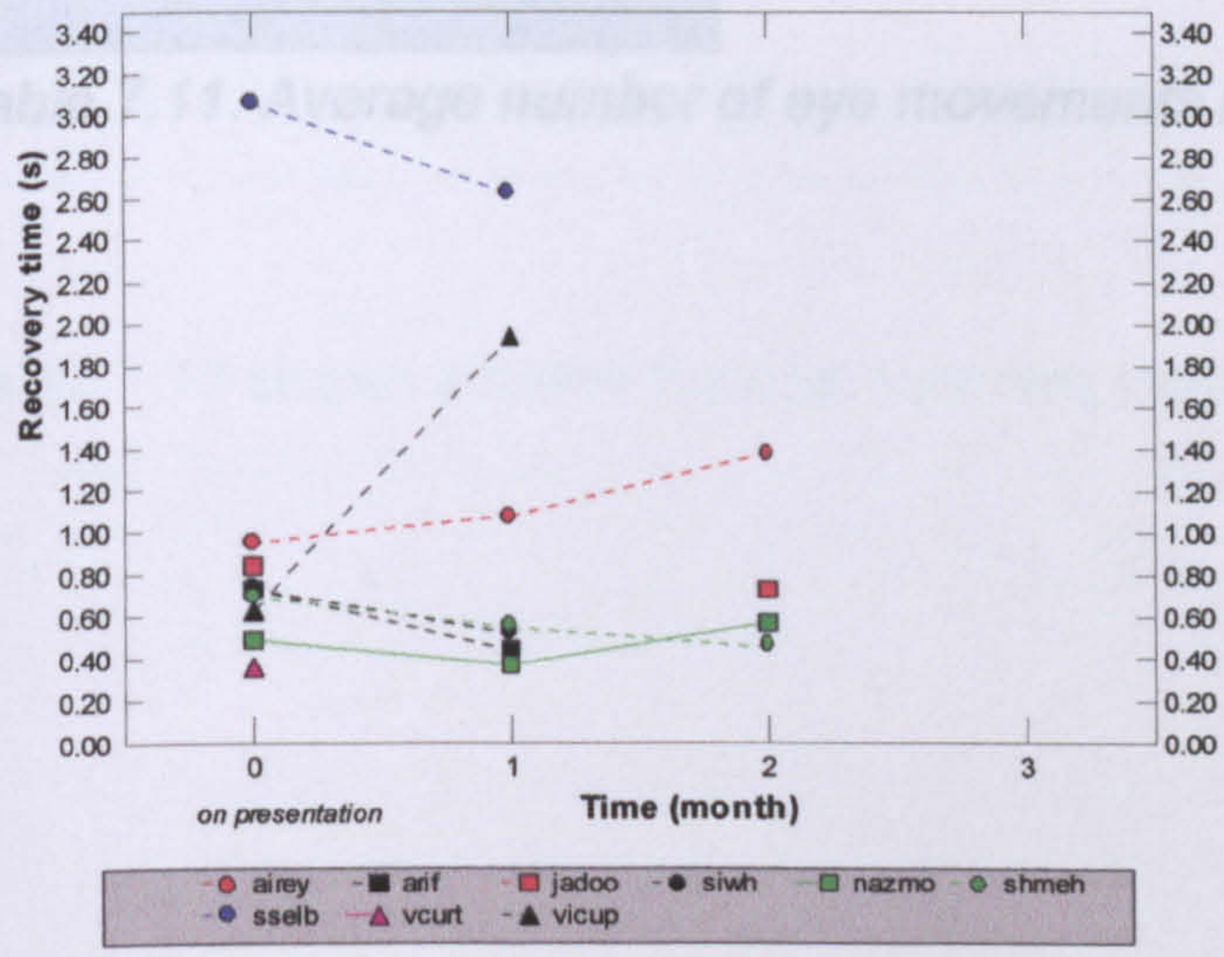


Figure 7.49. Scatter plot showing average recovery time (right and left eyes) for subjects within the non-CI group.

7.4.3.c. Number of recovery movements

Table 7.11 lists the average (of right and left eye) number of recovery eye movements made by each subject following the automated cover test. Where bilateral data was not available, the available monocular data is included in brackets. Some data are missing due to difficulty in interpreting traces due to blinks.

Subject	At presentation	After 1 month exercises	After 2 months exercises	After 1 month ceasing exercises
airey	5.5	5.5	4.5	na
arif	4	3.5	na	na
jadoo	(3)	nd	2	na
mayli	5	(1)	(fc)	nd
nazmo	2.5	(1) ⊗	4	na
shmeh	5	3.5	2	na
sselb	3	2.5	na	na
vcurt	(2)	fc	2 m	fc
Vicup	2	6.5	na	Na

na = not applicable for this subject

nd = no or inconclusive data

fc = failed to recover

↓ = subject refused eye movement recording

⊗ = left phoria amplitude too small to determine characteristics

m = 3 month review

Table 7.11. Average number of eye movements made to achieve recovery

7.4.3.d. NPC

Table 7.12 shows changes in NPC measures over time during the period of orthoptic intervention.

Subject	NPC at presentation (A)	NPC after 1 month (B)	NPC difference (B – A)	NPC after 2 months of exercises (C)	NPC difference (C – A)	NPC 1 month after ceasing exercises (D)	Regression of NPC (D – B, or D – C where applicable)
airey	3	3	0	6	+3	na	na
arif	6	6	0	na	na	na	na
jadoo	6	8 ψ	+2	8	+2	na	na
mayli	6	6	0	6	0	6	0
nazmo	8	8	0	6	-2	na	na
shmeh	8	8	0	8	0	na	na
sselb	6	6	0	na	na	na	na
vcurt	6	6	0	6 m	0	7	+1
vicup	6	5	-1	na	na	na	na

na = not applicable to this subject

ψ = reported exercise regime not carried out

m = 3 month review

Table7.12. NPC measures (cm) of non-CI over time.

7.4.3.e. Positive fusional reserves

Table 7.13 shows positive fusional reserves (blur/break/recovery) over time.

Subject	Positive fusional reserves (blur/break/recovery) (Δ)						
	At presentation (A) (A1/A2/A3)	After 1 month (B)	Change (B – A)	After 2 months of exercises (C) (C1/C2/C3)	Change (C- A)	After 1 month of ceasing exercises (D) (D1/D2/D3)	Regression (D – B) (or D –C when applicable)
airey	26/28/16	16/36/18	-10/08/02	20/32/18	-6/4/2	na	na
arif	12/12/11	14/20/16	02/08/05	na	na	na	na
jadoo	16/32/24	16/28/26ψ	00/04/02	34/36/12	18/08/14	na	na
mayli	# /12/11	# /16/12	-/04/01	# /18/16	-/06/05	# /20/18	-/02/02
nazmo	10/12/10	12/16/14	02/04/04	#/16/14	-/00/00	na	na
shmeh	04/04/01	12/16/14	08/12/13	08/12/10	04/08/09	na	na
sselb	24/36/12	12/16/14	-12/-20/02	na	na	na	na
vcurt	10/28/12	08/20/12	02/-08/0	12/22/20 m	02/-06/08	12/18/12	00/10/00
vicup	36/44/12	16/24/22	na	na	na	na	na

= blur point not observed by subject

na = not applicable to this subject

ψ = *reported exercise regime not carried out*

m = *3 month review*

Table 7.13. Positive fusional reserves (blur/break/recovery) of non-CI subjects over time.

7.4.3.f. Associated phoria

Table 7.14 shows associated phoria measurements over time.

Subject	At presentation	After 1 month exercises	After 2 months exercises	After 1 month ceasing exercises
Airey	0	½ R xo	1 L xo	na
Arif	0	0	na	na
Jadoo	0	½ R so	0	na
Mayli	½ R xo	0	0	0
nazmo	½ L so	0	0	na
shmeh	0	0	0	na
sselb	0	0	na	na
vcurt	0	0	0 m	½ R so
vicup	0	0 u	na	na

na = not applicable to this subject

u = unstable

m = 3 month review

Table 7.14. Changes in associated phoria over time.

7.4.3.g. Discussion of individual case histories

3 subjects manifested anomalies of their ocular motor system that were not defined as CI.

I. Case history

Subject	*airey*
Age	30 years-old
Sex	Male
Occupation	Banker
Symptoms	Headaches, aching or watering eyes, blurred vision, diplopia, and tired eyes after about two hours of close work
Reported compliance	Full compliance. Very motivated
Resolution of symptoms	Unsatisfactory
Follow-up	See discussion
Outcome	Partly successful

This subject was a 30 year-old male banker who was referred by the Eye Clinic because of his symptoms and an amplitude of exophoria for both distance and near vision which had raised the index of suspicion of the referring practitioner.

The subject manifested a low degree of myopia that was borderline for inclusion in this study. His refraction was R. –0.50/-0.25 x 180 L. –0.50/-0.25 x 180.

He manifested nearly 10^{Δ} exophoria at near and although this appeared fully compensated on presentation and the NPC and fusional reserves appeared normal, the level of stereoacuity (120") raised the index of suspicion that there was some embarrassment to binocular vision.

The subject had been prescribed spectacles but had expressed resistance against advice that he should wear them for any critical vision. He was adamant that he wished to pursue a course of exercises to alleviate his

symptoms and attended for a total of four review appointments over a period of five months. His diaries suggested he was compliant with the regime.

Despite the symptom score reducing to a relatively low level by two months, the subject maintained that the symptoms were still troublesome. Following the review at three months, the subject was advised to use his spectacles for all close work and continue with the exercises. At five months the symptom score appeared to be stable and the subject requested discharge from the study.

Figure 7.50 describes the subject's symptom score over the five months of treatment accompanied by the NPC. It is of interest to observe that there was a slight increase in NPC at two months accompanied by a reduction in symptom score both of which were sustained at the three and five month reviews.

Figure 7.51 illustrates an increase in recovery time following the near cover test at two and three months followed by a decrease at three months and a further increase at five months.

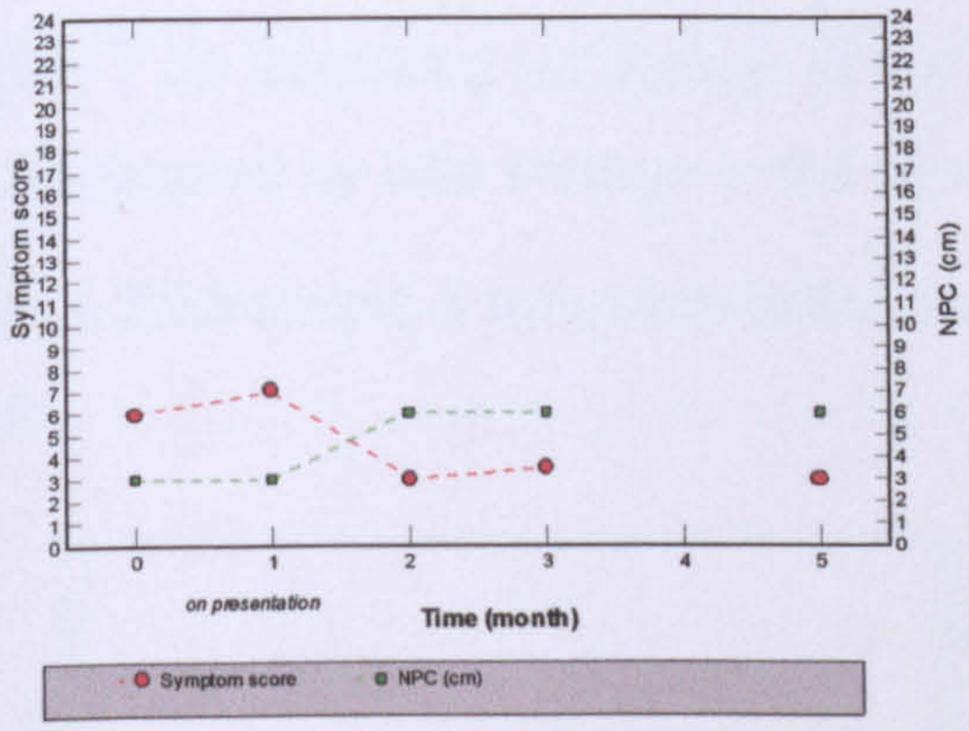


Figure 7.50. Changes in symptom score and NPC over the treatment period for subject airey.

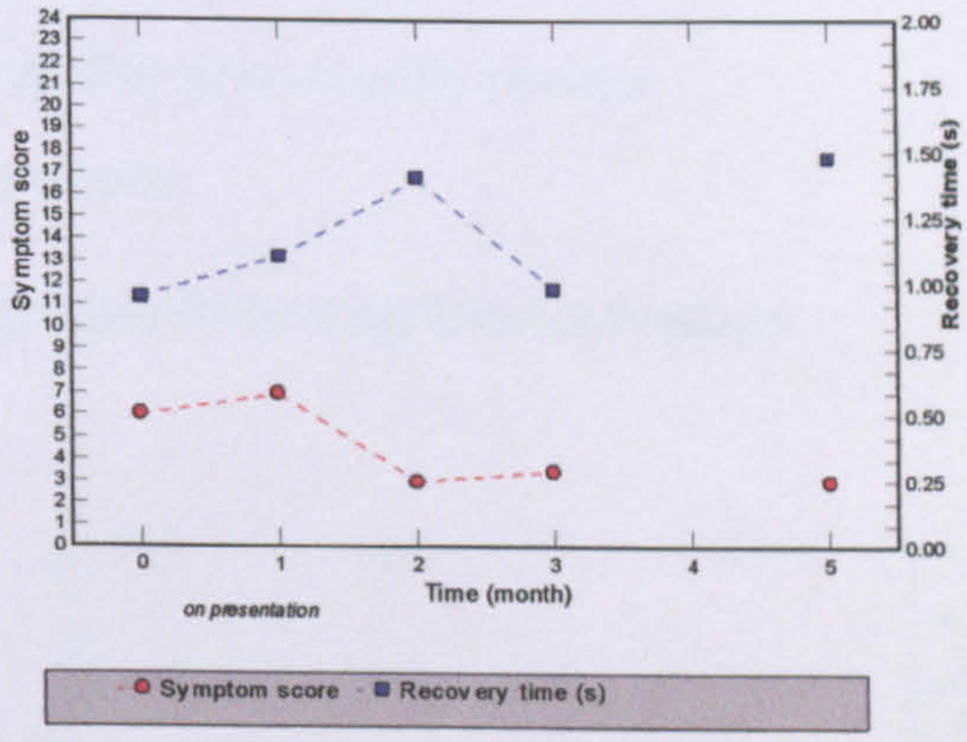


Figure 7.51. Changes in symptom score and recovery time over the treatment period for subject airey.

Figure 7.52 illustrates changes in fusional reserves over the treatment period.

Figure 7.53 shows a manifestation of associated phoria at one and two months with an apparent resolution at three and five months.

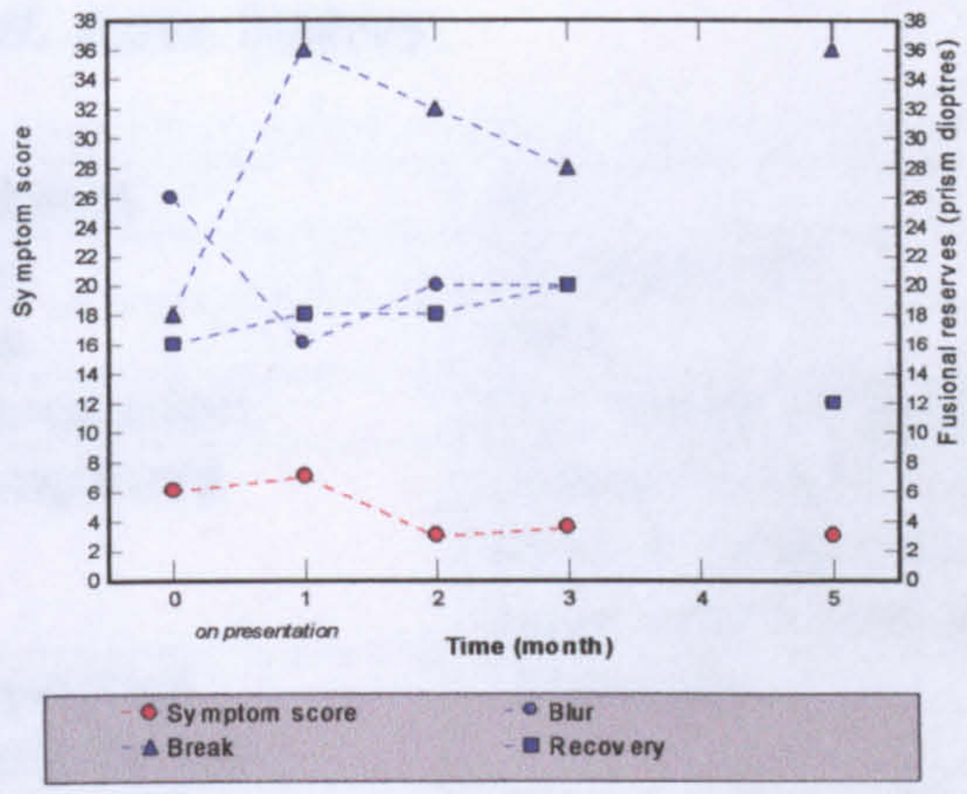


Figure 7.52. Changes in symptom score and fusional reserves over the treatment period for subject airey.

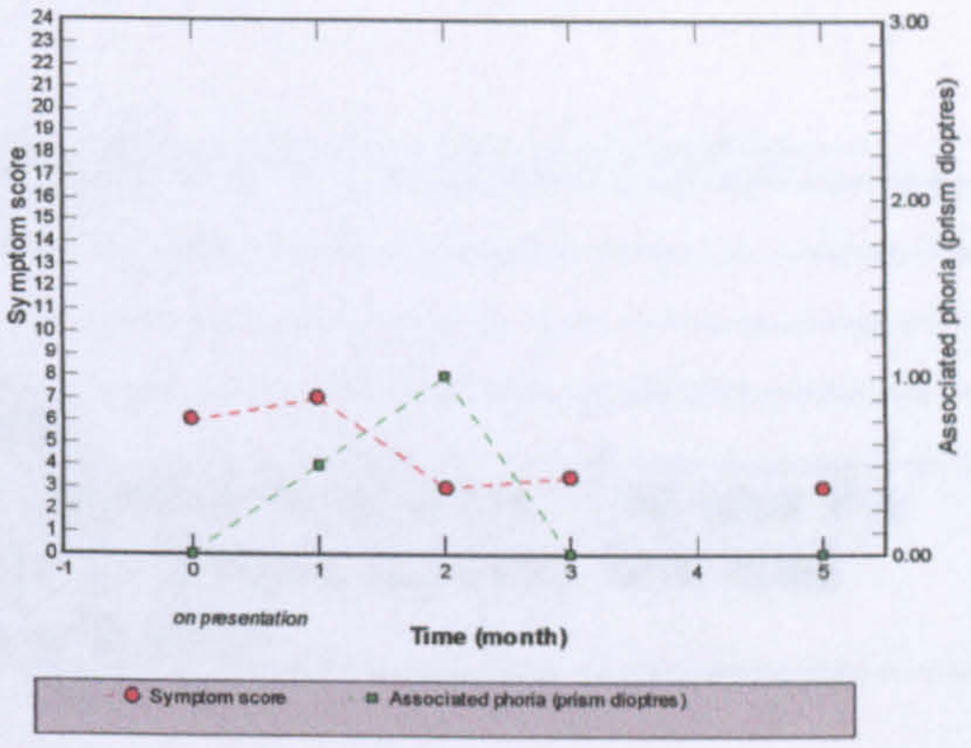


Figure 7.53. Changes in symptom score and associated phoria over the treatment period for subject airey.

Comment

This subject appeared to have a low tolerance of symptoms. At the end of the course of exercises he had come to terms with having to wear his spectacles more than he had wished but wished to continue with the exercises.

II. Case history

Subject	*Arif*
Age	23 years-old
Sex	Male
Occupation	Optometry undergraduate
Symptoms	Discomfort after up to 2 hours of close work. This took the form of headaches, aching or watering eyes, and tired eyes and/or loss of concentration
Reported compliance	Uncertain
Resolution of symptoms	Little change
Follow-up	See discussion
Outcome	Unsuccessful

This subject had referred himself to the study because of his symptoms and the knowledge that he had an exophoria at near.

He presented with a normal NPC and no associated phoria. However, positive fusional reserves were somewhat low relative to the amplitude of near exophoria.

Figure 7.54 illustrates no change in the NPC at the one-month review accompanied by little change in the symptom score.

Figure 7.55 shows a reduction in the recovery time following the near cover test.

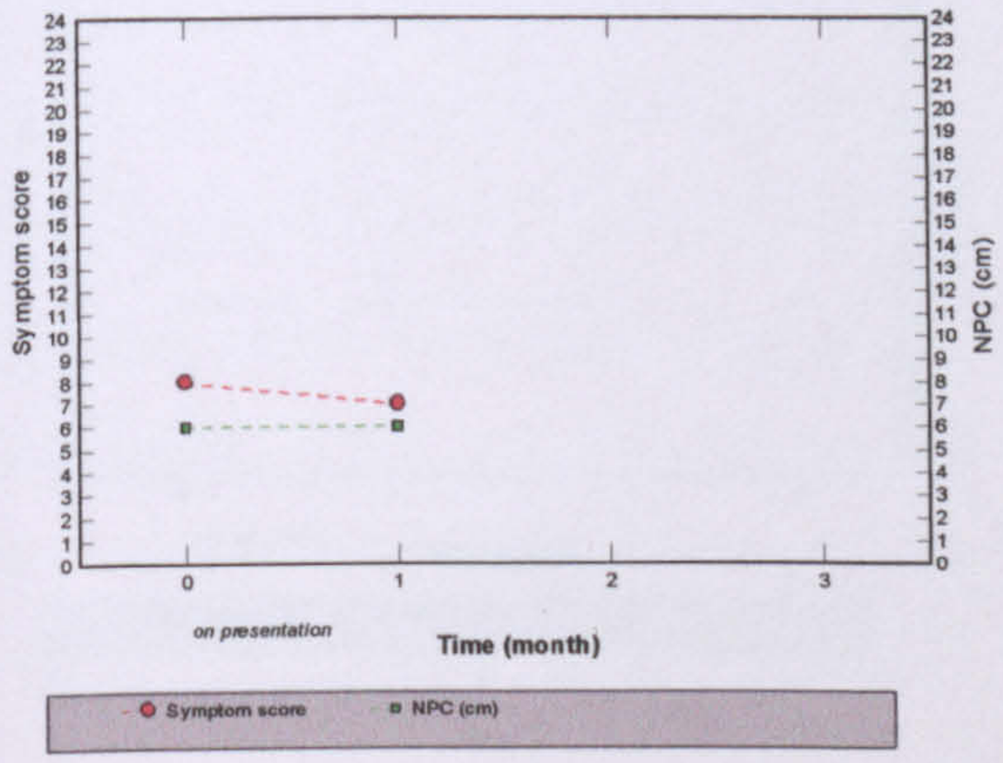


Figure 7.54. Changes in symptom score and NPC over the treatment period for subject arif.

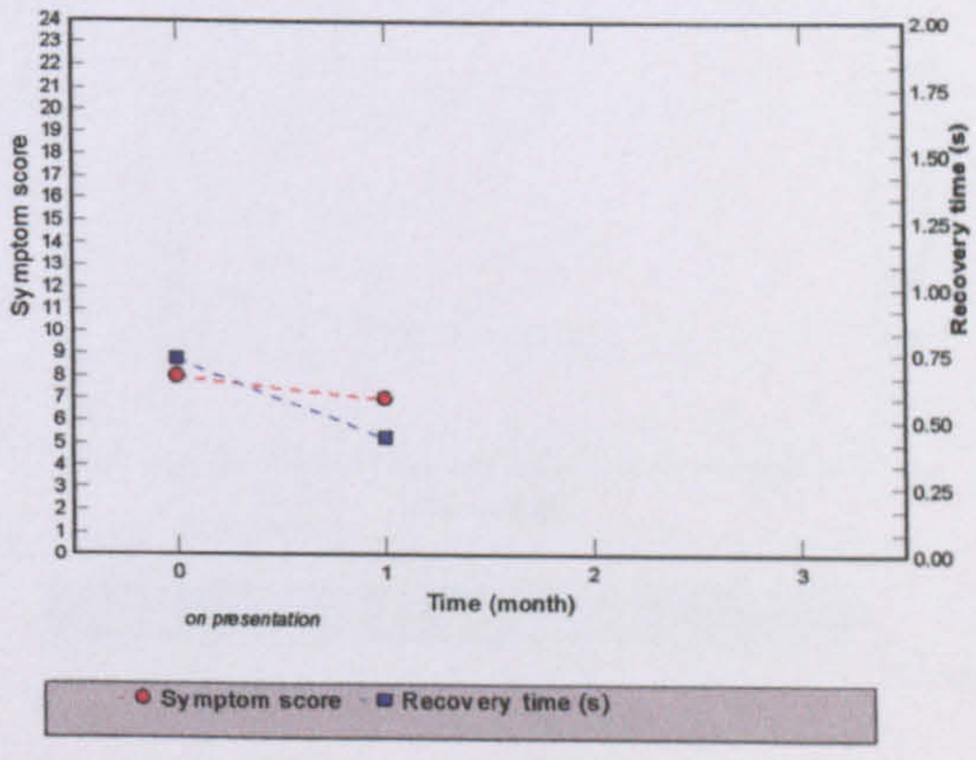


Figure 7.55. Changes in symptom score and recovery time over the treatment period for subject arif.

Figure 7.56 shows an increase in blur, break and recovery measures at the one-month review.

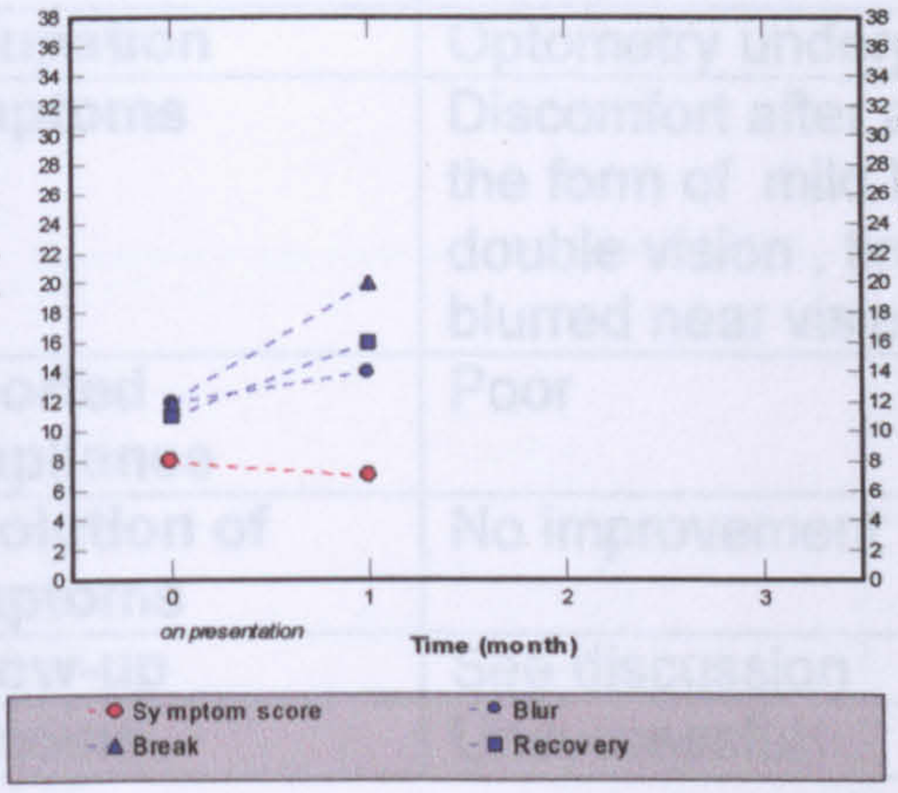


Figure 7.56. Changes in symptom score and fusional reserves over the treatment period for subject arif.

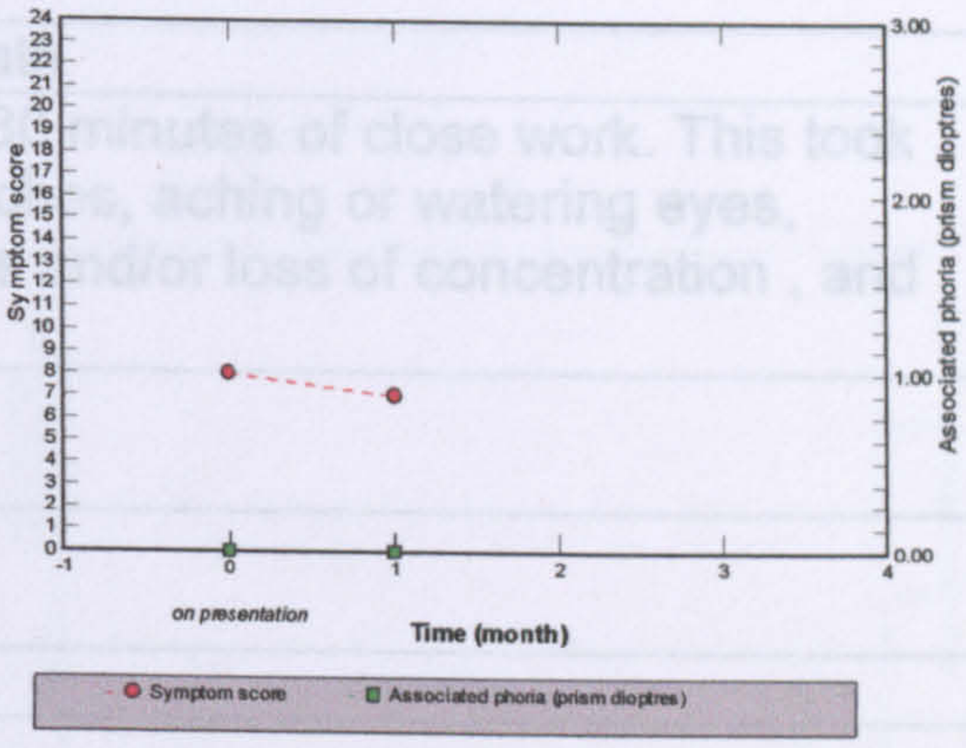


Figure 7.57. Changes in symptom score and associated phoria over the treatment period for subject arif.

The near exophoria remained fully compensated as assessed by the near Mallett unit and this is shown by the data in Figure 7.57.

Comment

This subject failed to keep his next review appointment and did not respond to further requests to arrange an appointment.

III. Case history

Subject	*shmeh*
Age	25 years-old
Sex	Female
Occupation	Optometry undergraduate
Symptoms	Discomfort after about 30 minutes of close work. This took the form of mild headaches, aching or watering eyes, double vision , tiredness and/or loss of concentration , and blurred near vision
Reported compliance	Poor
Resolution of symptoms	No improvement
Follow-up	See discussion
Outcome	Unsuccessful

Although a near exophoria was well compensated and her NPC was normal, her positive fusional reserves were poor. She reported that she carried out the exercises only "occasionally".

Figure 7.58 shows no change in NPC over the period that she should have been carrying out the exercises. The symptom score at the end of the two-month period had increased slightly on the score at presentation.

Figure 7.59 shows a reduction in recovery time following the cover test at the one and two month reviews.

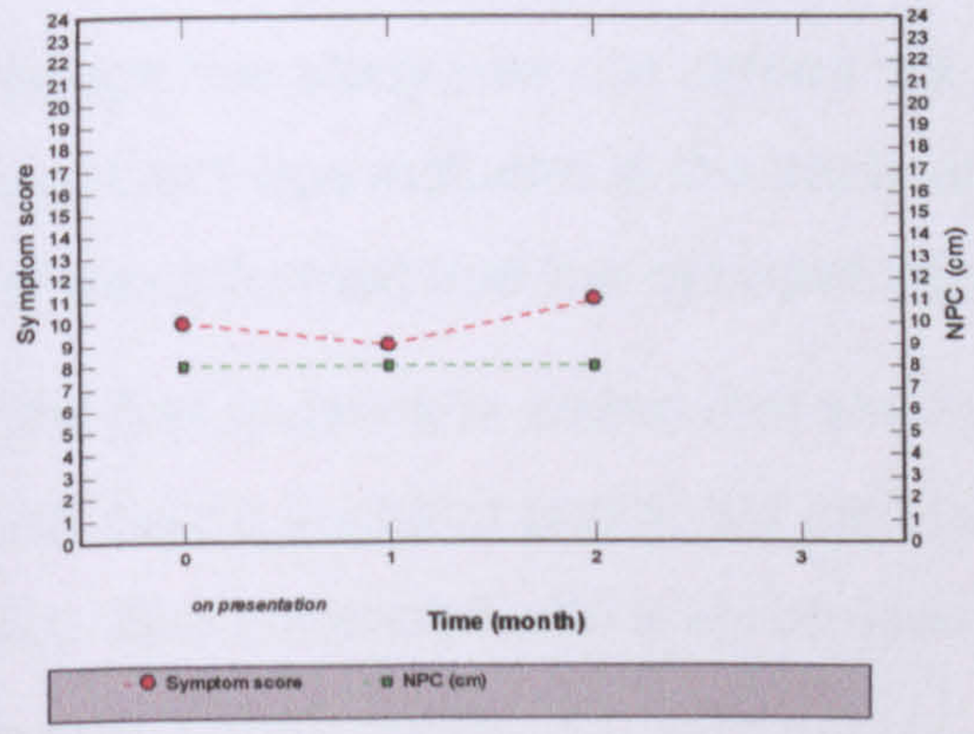


Figure 7.58. Changes in symptom score and NPC over the treatment period for subject shmeh.

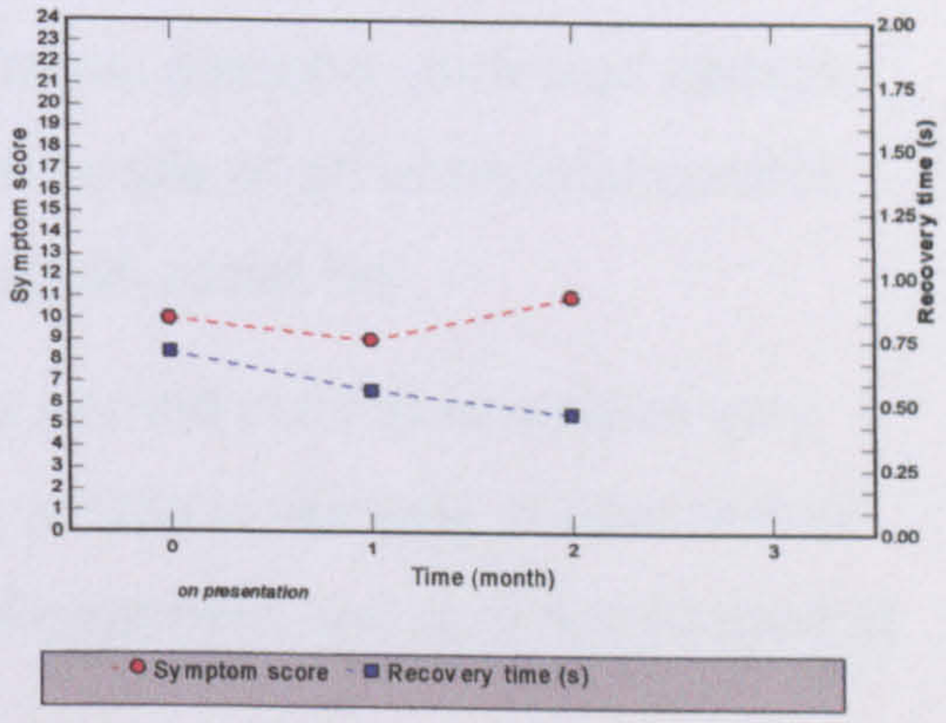


Figure 7.59. Changes in symptom score and recovery time over the treatment period for subject shmeh.

Figure 7.60 shows an improvement in fusional reserves at one month followed by regression at two months.

Figure 7.61 illustrates no the change in associated phoria which remained at zero at all presentations.

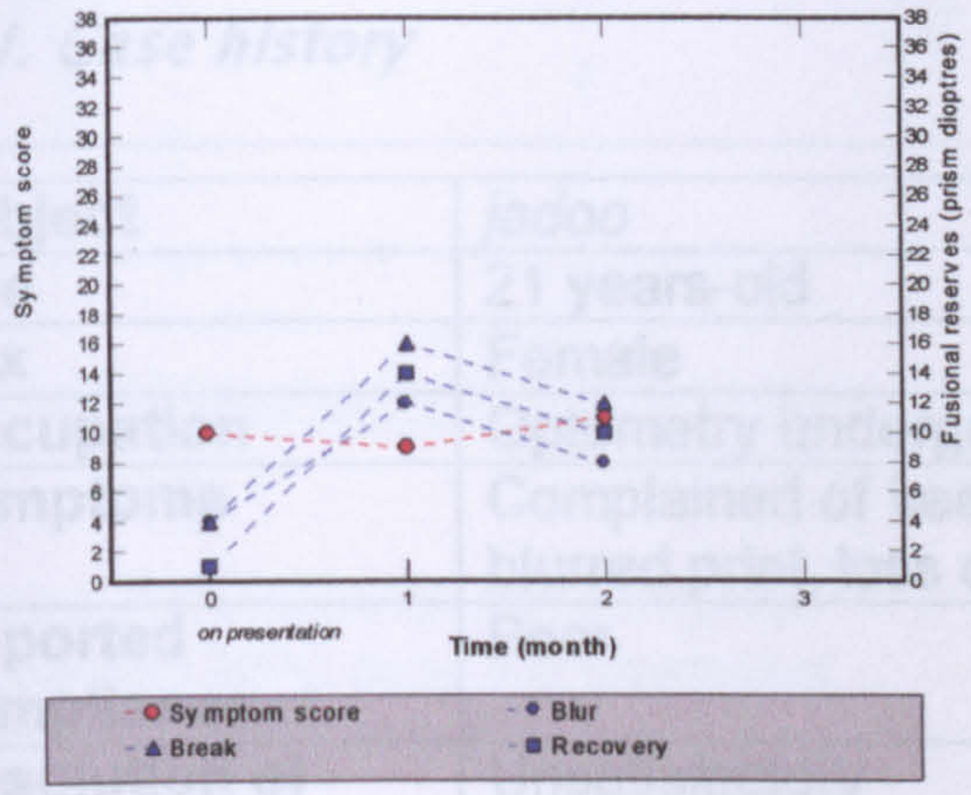


Figure 7.60. Changes in symptom score and fusional reserves over the treatment period for subject shmeh.

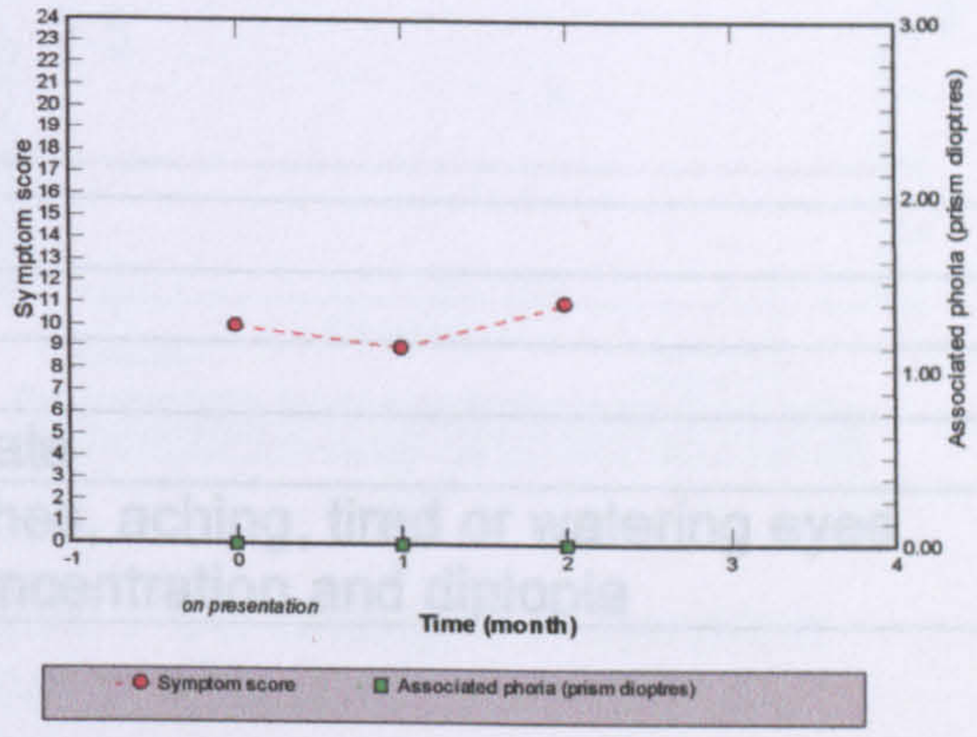


Figure 7.61. Changes in symptom score and associated phoria over the treatment period for subject shmeh.

Comment

This subject failed to keep a further monthly follow-up appointment. It was concluded that the orthoptic regime used in this study was unsatisfactory.

7.4.4 Anecdotal controls

6 subjects had no observed significant anomalies of their ocular motor system.

I. Case history

Subject	*jadoo*
Age	21 years-old
Sex	Female
Occupation	Optometry undergraduate
Symptoms	Complained of headaches, aching, tired or watering eyes, blurred print, loss of concentration and diplopia
Reported compliance	Poor
Resolution of symptoms	Unsatisfactory
Follow-up	See discussion
Outcome	Unsuccessful

This subject had referred herself to the study because of the symptoms associated with near vision.

She manifested ½ $^{\Delta}$ esophoria at near which was fully compensated. NPC and fusional reserves were well developed.

Although this study was not carried out to a formal placebo controlled design, this subject was included in the study as an example of an anecdotal control. She was informed that the orthoptic regime would assist her.

At the first review she stated that she had not carried out the exercises very much over a vacation period but also had done little in the way of near vision tasks. She continued with a second month of treatment and was discharged at the end of that month.

Figure 7.63 shows a slight worsening of the symptom score and NPC at the end of the month that the subject had not carried out the exercises and claimed to have done little demanding close work.

The datum for recovery time following the cover test at the one-month review was not available because of blinks. The recovery times on presentation and at the two-month review are shown in Figure 7.64.

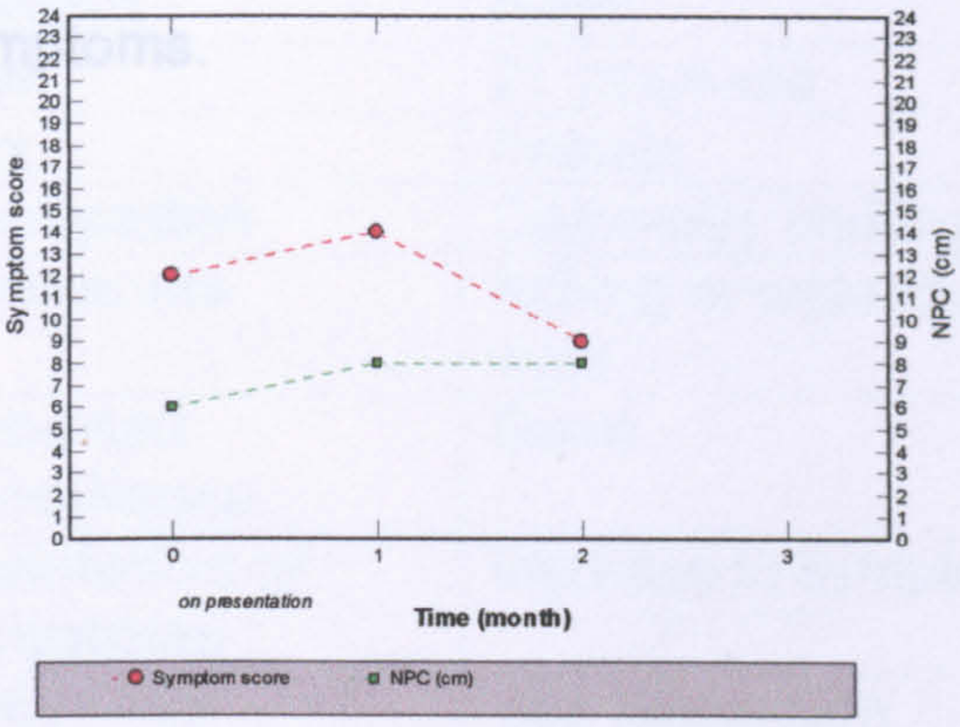


Figure 7.63. Changes in symptom score and NPC over the treatment period for subject jadoo.

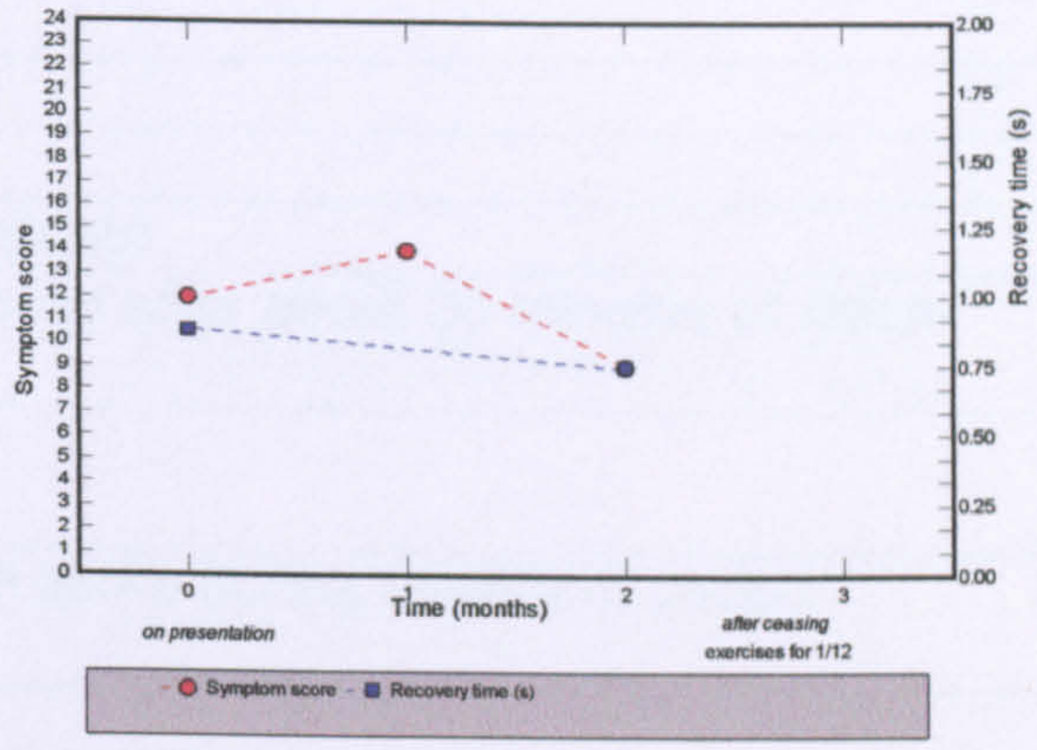


Figure 7.64. Changes in symptom score and recovery time over the treatment period for subject jadoo.

Figure 7.65 illustrates the fusional reserves measured at presentation, and after one and two months.

Figure 7.66 illustrates a manifestation of a small esophoric associated phoria at the one-month review accompanying the reported increase in symptoms. The associated phoria was not manifest at the two-month review.

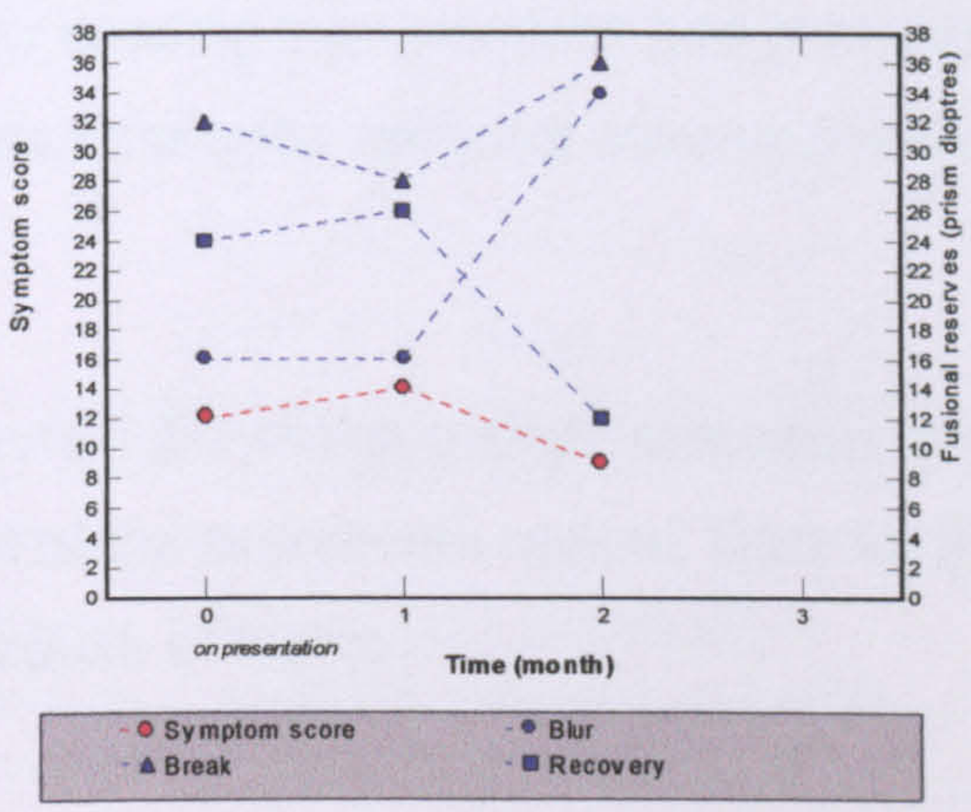


Figure 7.65. Changes in symptom score and fusional reserves over the treatment period for subject jadoo.

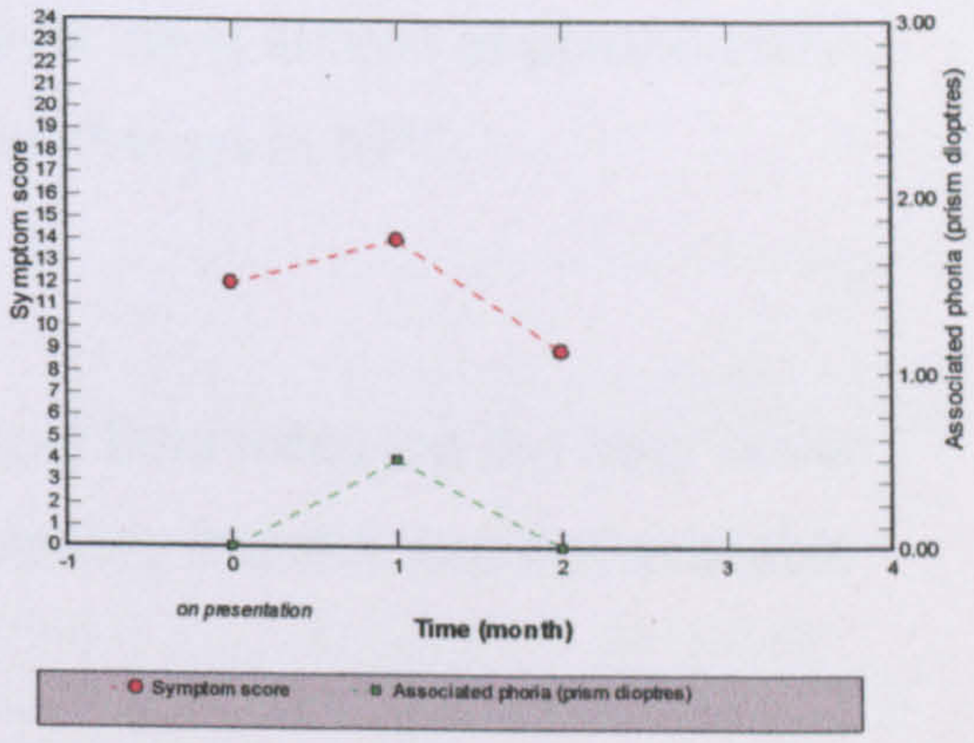


Figure 7.66. Changes in symptom score and associated phoria over the treatment period for subject jadoo.

This subject was discharged following the two-month review.

Comment

The regime of exercises did not coincide with a satisfactory resolution of symptoms.

II. Case history

Subject	*mayli*
Age	21 years-old
Sex	Female
Occupation	Optometry undergraduate
Symptoms	Aching or watering eyes after about 30 minutes of close-work
Reported compliance	Good
Resolution of symptoms	Increase in symptom score during treatment period
Follow-up	See discussion
Outcome	Unsuccessful

This subject was referred to the study by the Departmental Eye Clinic because of symptoms associated with near vision.

She was orthophoric at near with a normal NPC. On presentation to the study she manifested a small (½ $^{\Delta}$) exophoric associated phoria at near. This subject was included in the study as an example of an anecdotal control. She was informed that the orthoptic regime would assist her.

Figure 7.67 shows an increase in symptom score after one month of treatment. This had improved slightly at the two-month review and one month after ceasing the exercises had returned to level initial scored at presentation. These changes were not accompanied by any change in NPC.

Figure 7.68 shows a slight reduction in recovery time following the near cover test at the one-month review. Data for the following months was not available because of blinks.

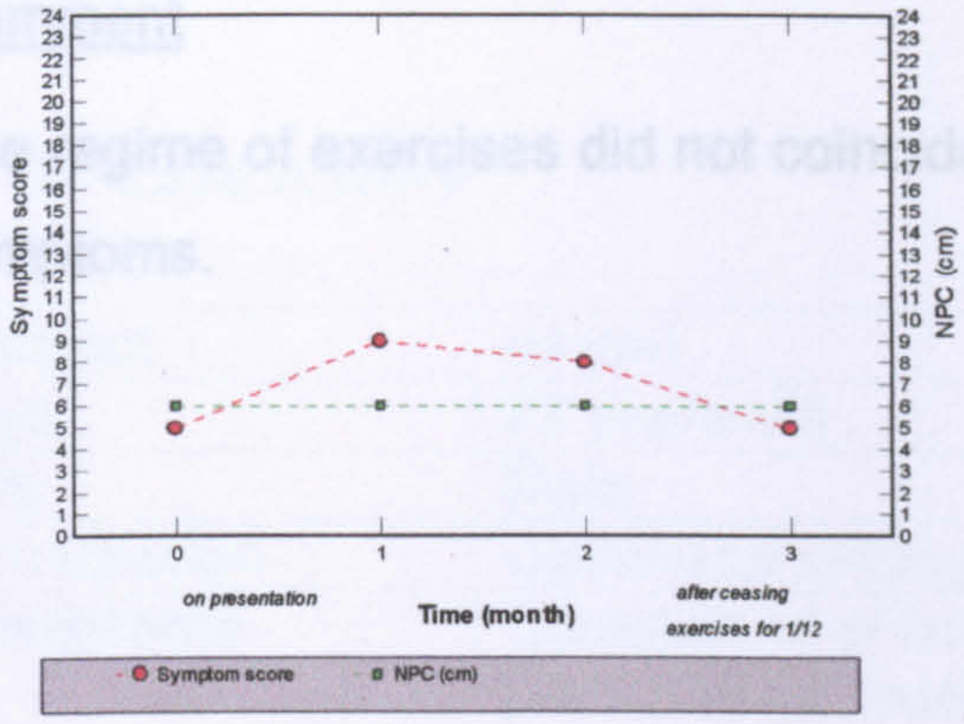


Figure 7.67. Changes in symptom score and NPC over the treatment period for subject mayli.

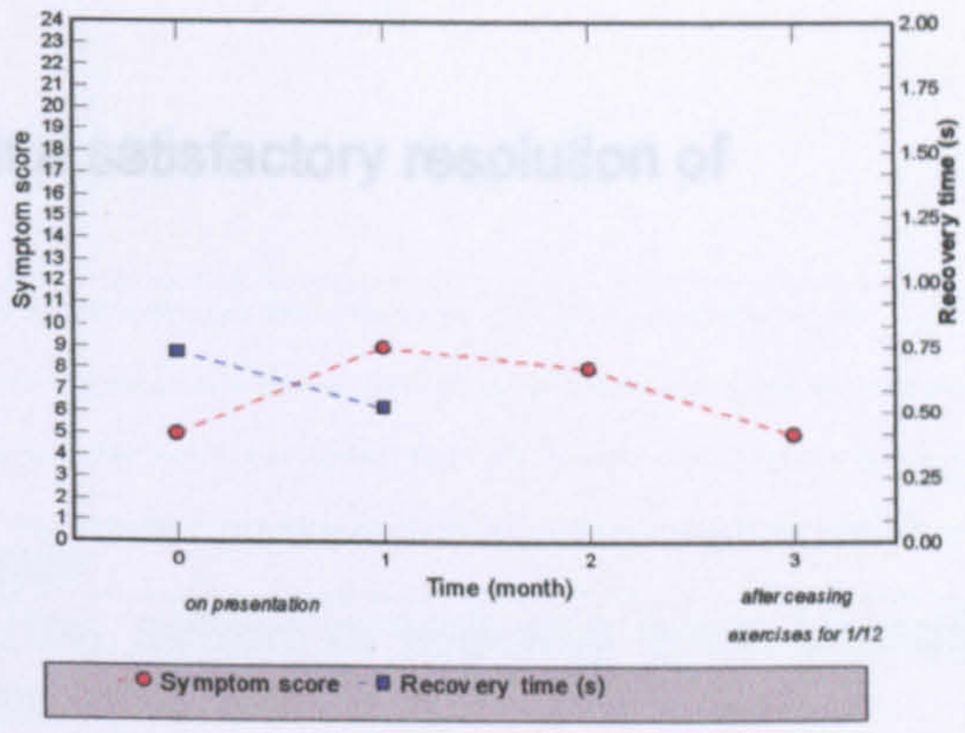


Figure 7.68. Changes in symptom score and recovery time over the treatment period for subject mayli.

Figure 7.69 shows an increase in fusional reserves over the period of treatment and after ceasing the exercises. The subject was unable to perceive *blur* during measurement of the fusional reserves.

Figure 7.70 shows an initial associated exophoria which was not observed at subsequent reviews.

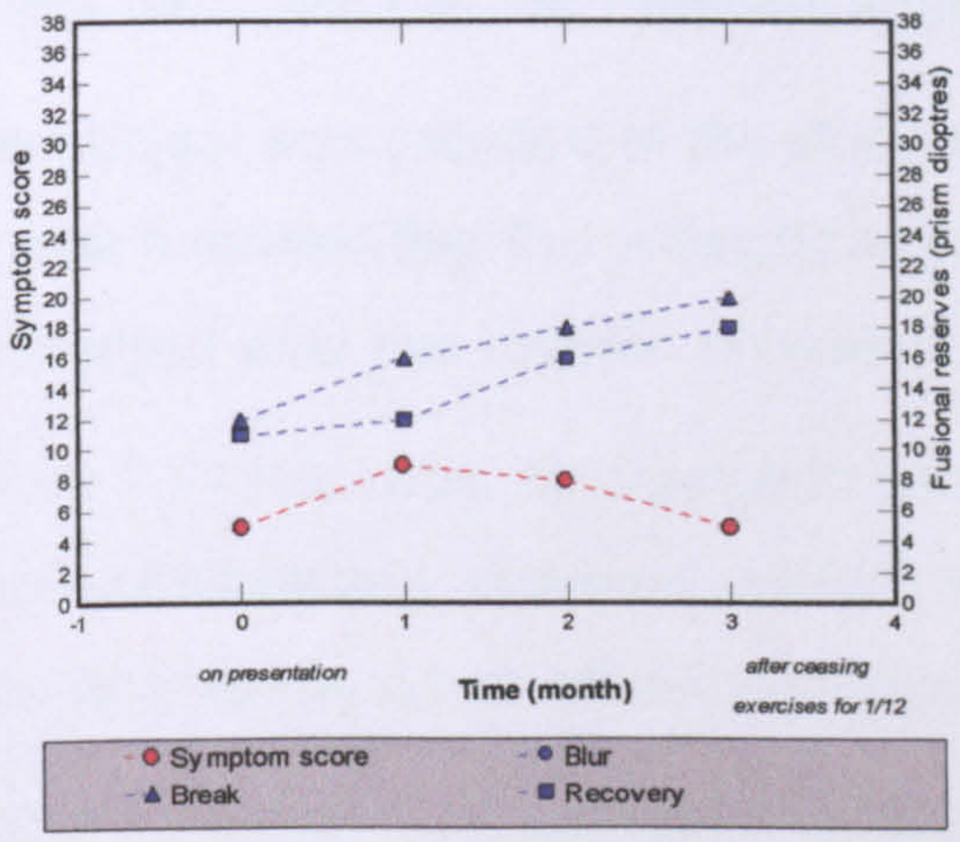


Figure 7.69. Changes in symptom score and fusional reserves over the treatment period for subject mayli.

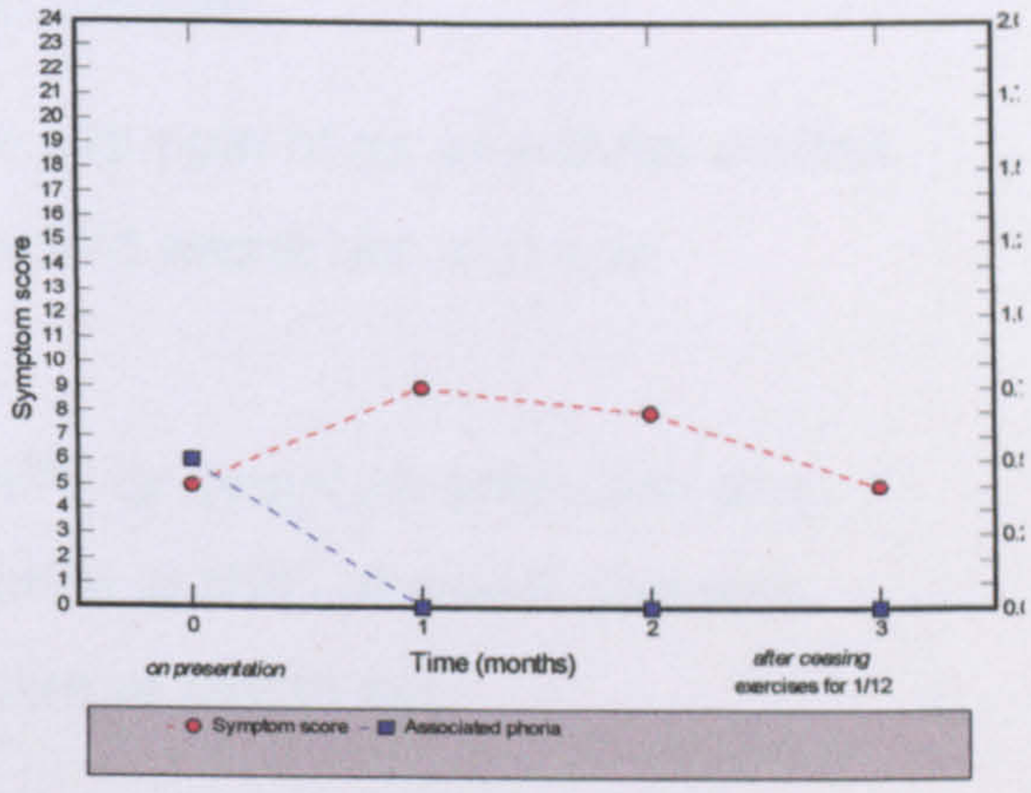


Figure 7.70. Changes in symptom score and associated phoria over the treatment period for subject mayli.

At her last appointment she did agree to attend for a further review two months after ceasing the exercises. She did not keep this appointment.

Comment

The regime of exercises did not coincide with a satisfactory resolution of symptoms.

III. Case history

Subject	*nazmo*
Age	23 years-old
Sex	Male
Occupation	Optometry undergraduate
Symptoms	Complained of headaches, aching or watering eyes, blurred print, double vision, tired eyes and loss of concentration after about one hour of close work
Reported compliance	Full compliance
Resolution of symptoms	Reduced score after two months of treatment
Follow-up	Discharged after two months of treatment
Outcome	Unsuccessful

This subject had referred himself to the study because of symptoms associated with near vision work.

He manifested 1^{Δ} of near exophoria (automated cover test) and on presentation showed a small amplitude of 'paradoxical' esophoric associated phoria. NPC and fusional reserves appeared 'normal'.

This subject was included in the study as an example of an anecdotal control. He was informed that the orthoptic regime would assist him and was discharged after two months of treatment.

Figure 7.71 illustrates no change in either NPC or symptom score are one month of treatment. However, a slight reduction in NPC at month two was accompanied by a reduction in symptom score at month two.

Figure 7.72 shows an increased recovery time at the review at which the symptom score was lowest.

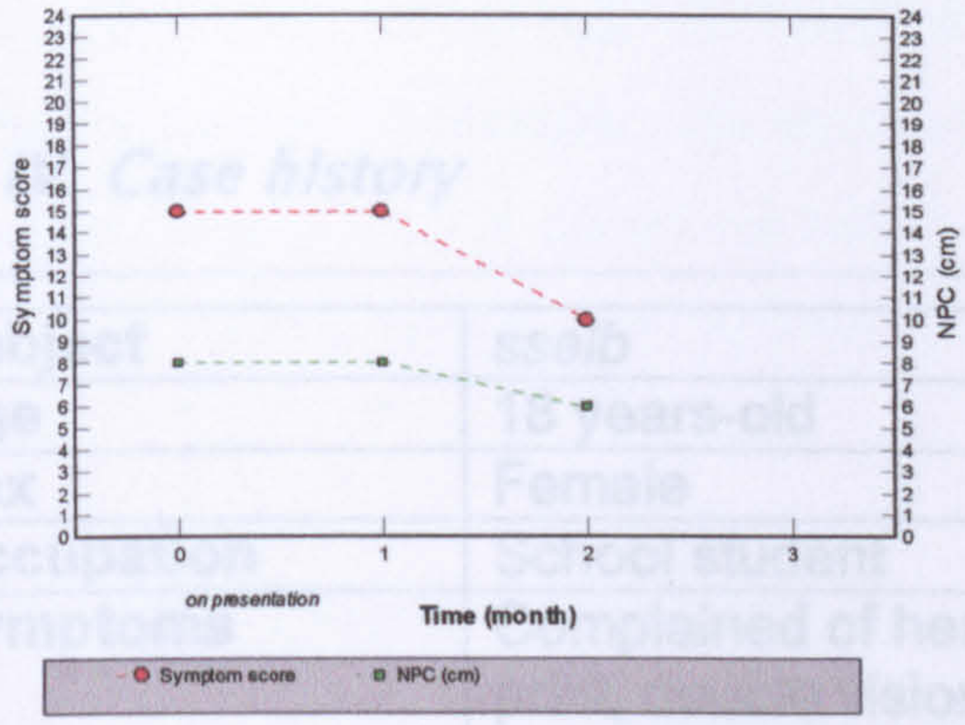


Figure 7.71. Changes in symptom score and NPC over the treatment period for subject nazmo.

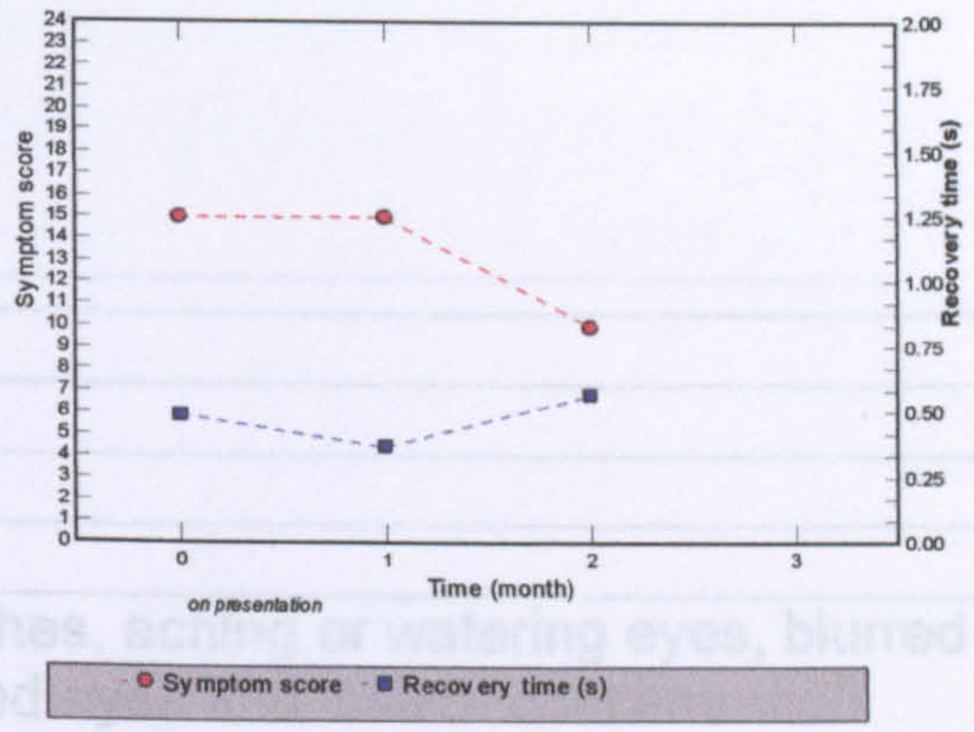


Figure 7.72. Changes in symptom score and recovery time over the treatment period for subject nazmo.

Figure 7.73 shows little change in fusional reserves over the treatment period.

Figure 7.74 shows an alleviation of the small associated phoria at the first review, which was sustained thereafter.

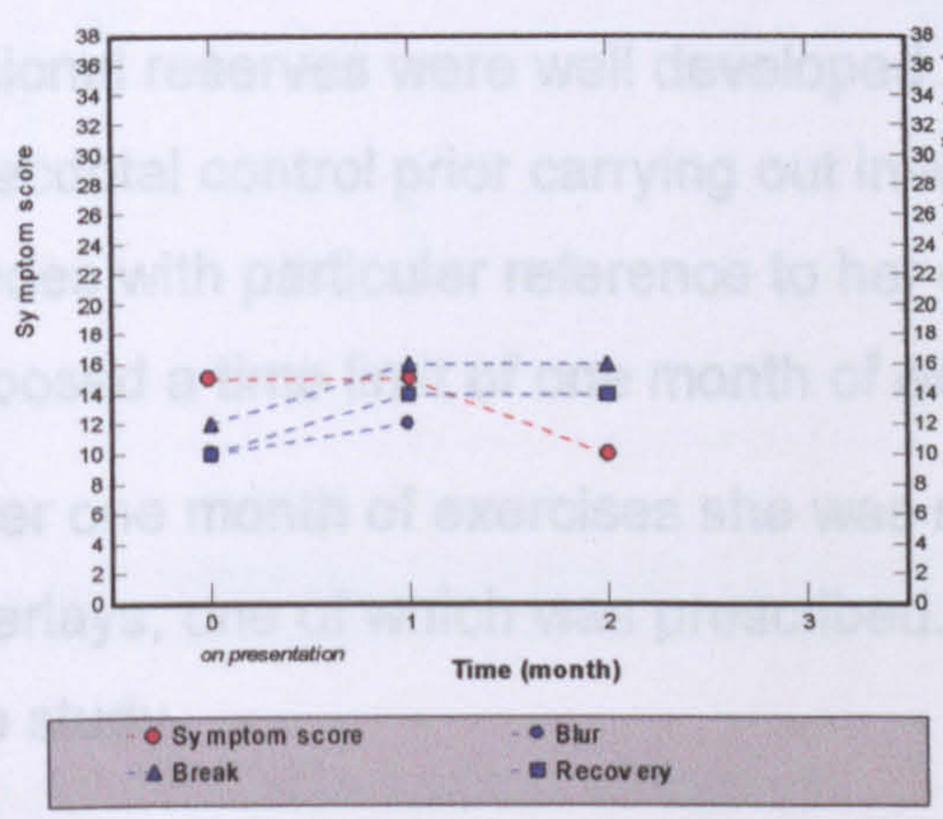


Figure 7.73. Changes in symptom score and fusional reserves over the treatment period for subject nazmo.

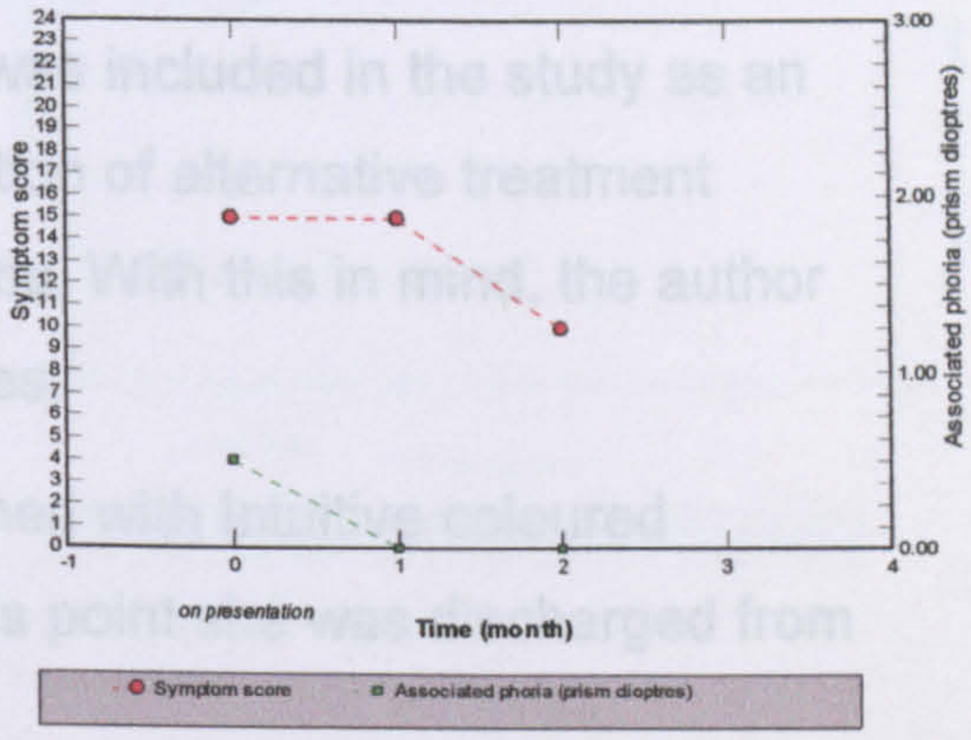


Figure 7.74. Changes in symptom score and associated phoria over the treatment period for subject nazmo.

Comment

The regime of exercises did not coincide with a satisfactory resolution of symptoms.

IV. Case history

Subject	*sselb*
Age	18 years-old
Sex	Female
Occupation	School student
Symptoms	Complained of headaches, aching or watering eyes, blurred print, double vision, tired eyes and loss of concentration after about 30 minutes close work
Reported compliance	Good
Resolution of symptoms	Increase in symptom score during treatment period
Follow-up	See discussion
Outcome	Unsuccessful

This subject was dyslexic and was referred to the student by the Departmental Eye Clinic on the basis of her near vision symptoms. Although she manifested a small exophoria at near, this was well compensated and her NPC and fusional reserves were well developed. She was included in the study as an anecdotal control prior carrying out investigation of alternative treatment modes with particular reference to her dyslexia. With this in mind, the author imposed a time limit of one month of exercises.

After one month of exercises she was screened with Intuitive coloured overlays, one of which was prescribed. At this point she was discharged from the study.

Figure 7.75 shows no change in NPC at the month review. This was accompanied by a reduction in symptom score.

Figure 7.76 illustrates the slow recovery times following the cover test at both presentation and the one-month review. As explained previously, these data were the averages of right and left eye. It was of interest to note that the recovery time for the right eye was approximately 0.6 s on both occasions in comparison to the left eye of approximately 5 s on both occasions.

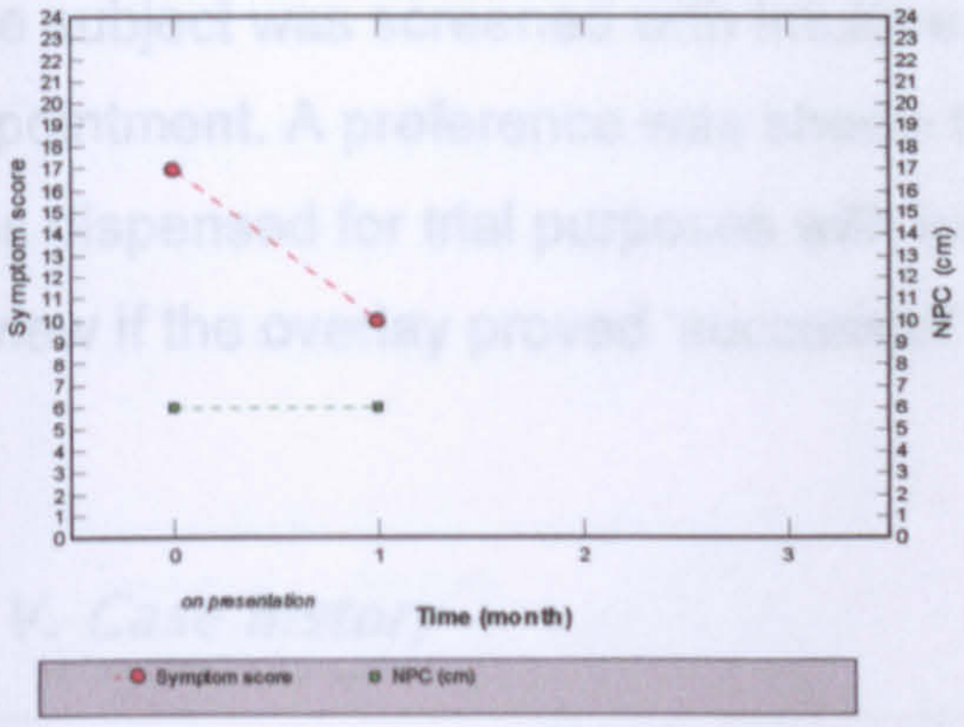


Figure 7.75. Changes in symptom score and NPC over the treatment period for subject sselb.

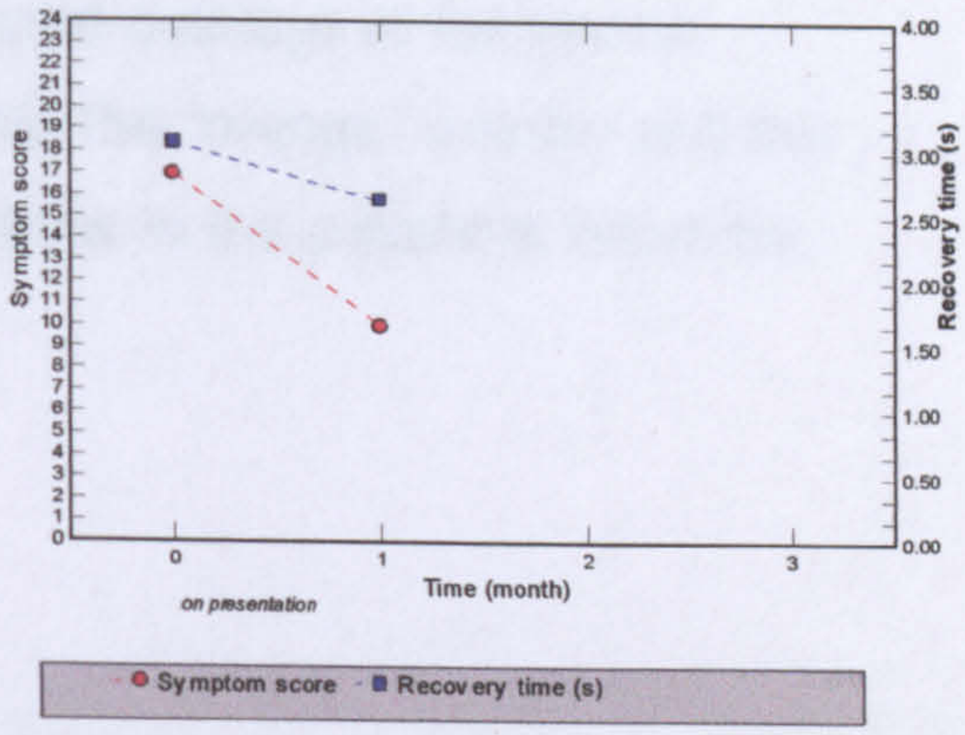


Figure 7.76. Changes in symptom score and recovery time over the treatment period for subject sselb.

Figure 7.77 shows an apparent reduction in positive fusional reserves at the one-month review. Figure 7.78 shows a zero measure of associated phoria at both presentations.

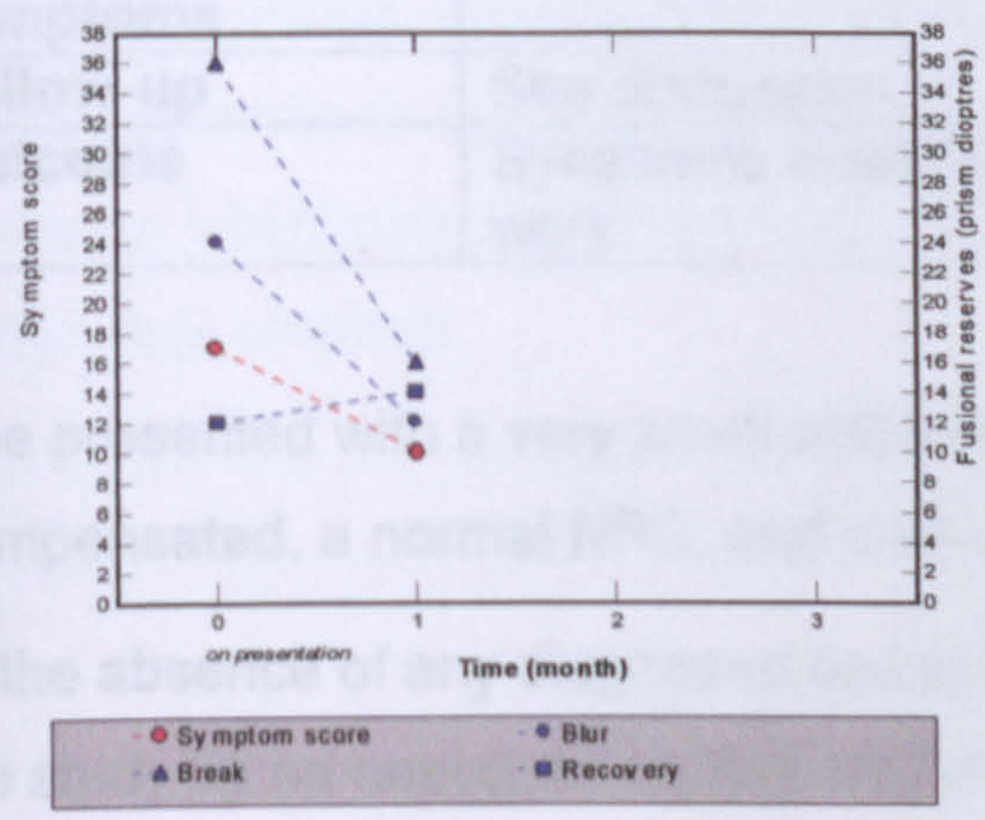


Figure 7.77. Changes in symptom score and fusional reserves over the treatment period for subject sselb.

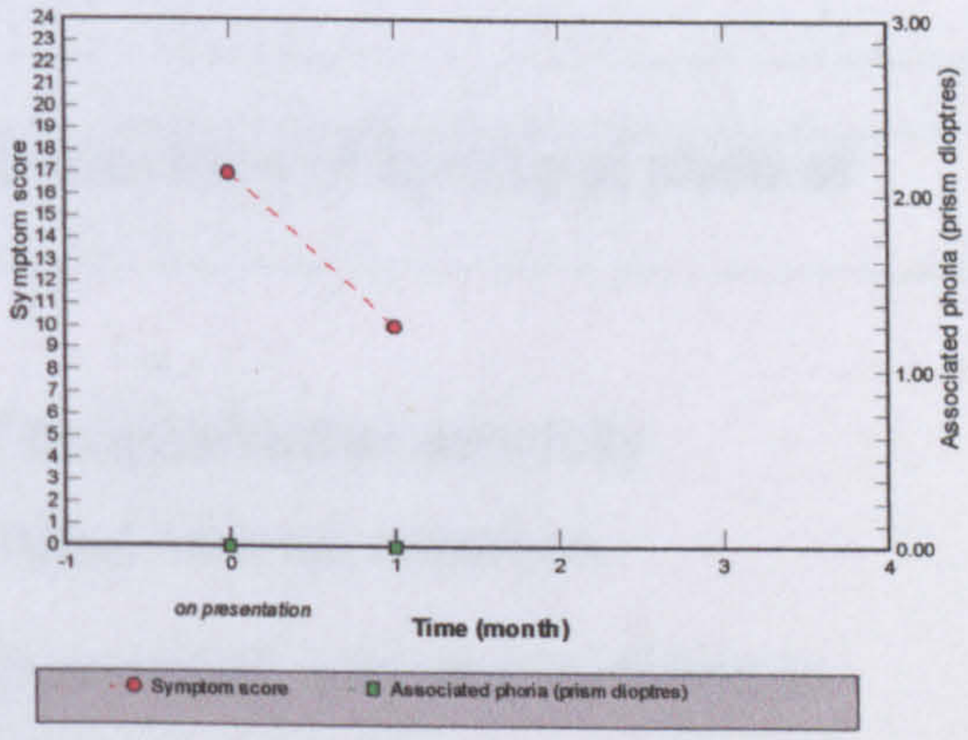


Figure 7.78. Changes in symptom score and associated phoria over the treatment period for subject sselb.

<u>Comment</u>

It was of interest to observe the slow recovery of the left eye following the near cover test despite a normal NPC, good fusional reserves and no associated phoria. Although this is simply an anecdotal observation, the author does suggest that a comparison between non-dyslexic and dyslexic recovery times following the cover test might provide an interesting area of study.

The subject was screened with Intuitive coloured overlays at the review appointment. A preference was shown towards the "orange" overlay and this was dispensed for trial purposes with instructions to the patient to return for review if the overlay proved 'successful'.

V. Case history

Subject	*Vcurt*
Age	32 years-old
Sex	Female
Occupation	Administrator
Symptoms	Complained of headaches, aching or watering eyes, blurred print, double vision, tired eyes and loss of concentration after about two hours of close work
Reported compliance	Full compliance
Resolution of symptoms	Unaffected by treatment
Follow-up	See discussion
Outcome	Symptoms resolved with alteration of lighting at place of work

She presented with a very small amplitude of exophoria that was fully compensated, a normal NPC, and well-developed fusional reserves.

In the absence of any diagnosed ocular motor anomaly, she was included in the study as an anecdotal control and informed that the exercises would help her.

Following the initial assessment, the subject returned after one month for a review. The next review was two months later at which she reported a sudden and full resolution of her symptoms following the replacement of fluorescent lights at her place of work. She was asked to cease the exercises and returned for a further review one month later at which time she was discharged from the study.

For subject *vcurt,* there were no available data for recovery times at review appointments because of blinks.

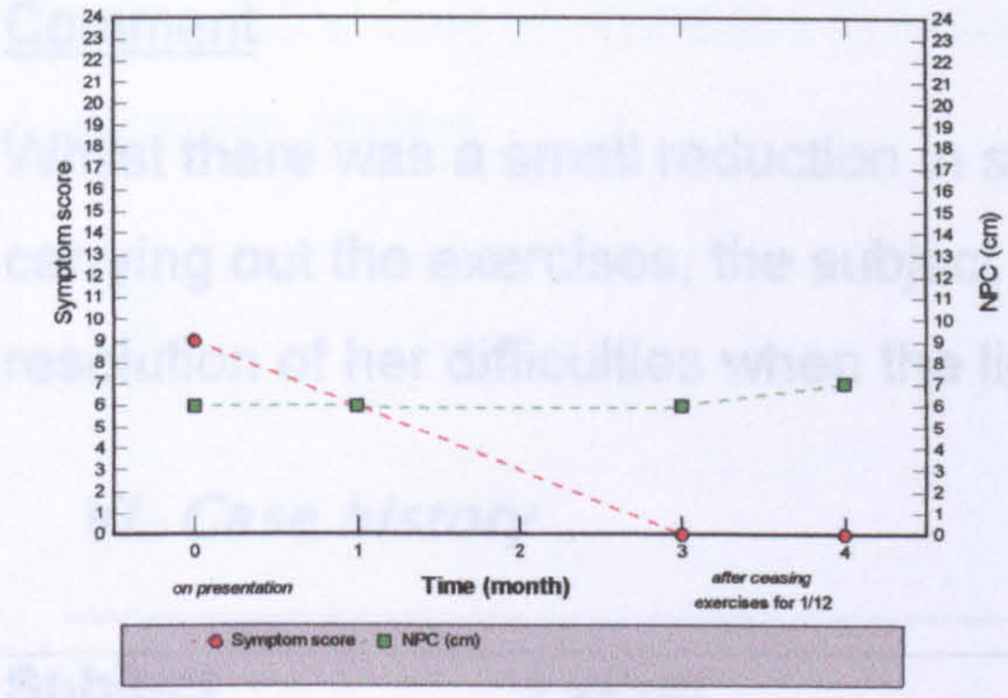


Figure 7.79. Changes in symptom score and NPC over the treatment period for subject vcurt.

Figure 7.79 shows the constant NPC measure across the period of study accompanied by the full resolution of symptoms which was associated with a change of lighting at work and reported at her third visit. Figure 7.80 shows fusional reserve measures over the treatment period. Figure 7.81 illustrates associated phoria amplitudes over the treatment period. It is of interest to note the appearance of an associated phoria ($\frac{1}{2}^{\Delta}$ right esophoric slip) at the last review at which time the patient reported that the symptoms were still completely resolved.

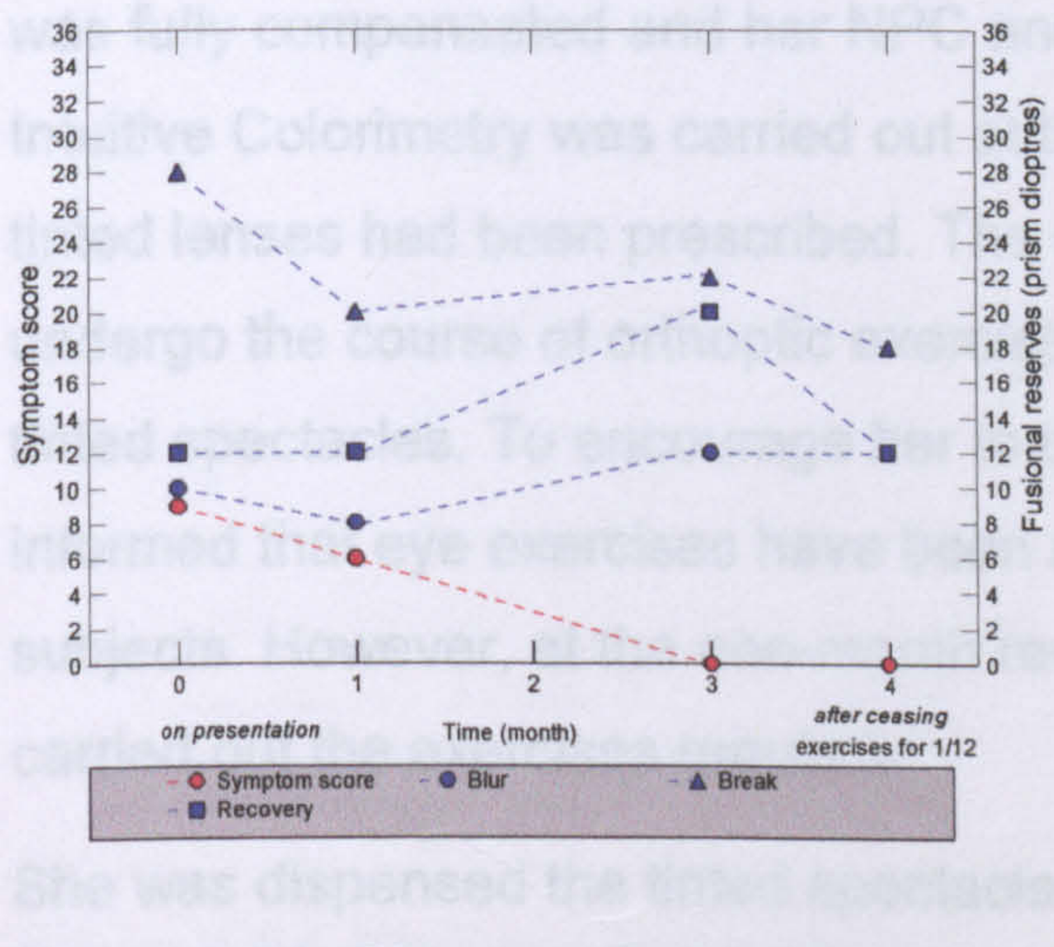


Figure 7.80. Changes in symptom score and fusional reserves over the treatment period for subject vcurt.

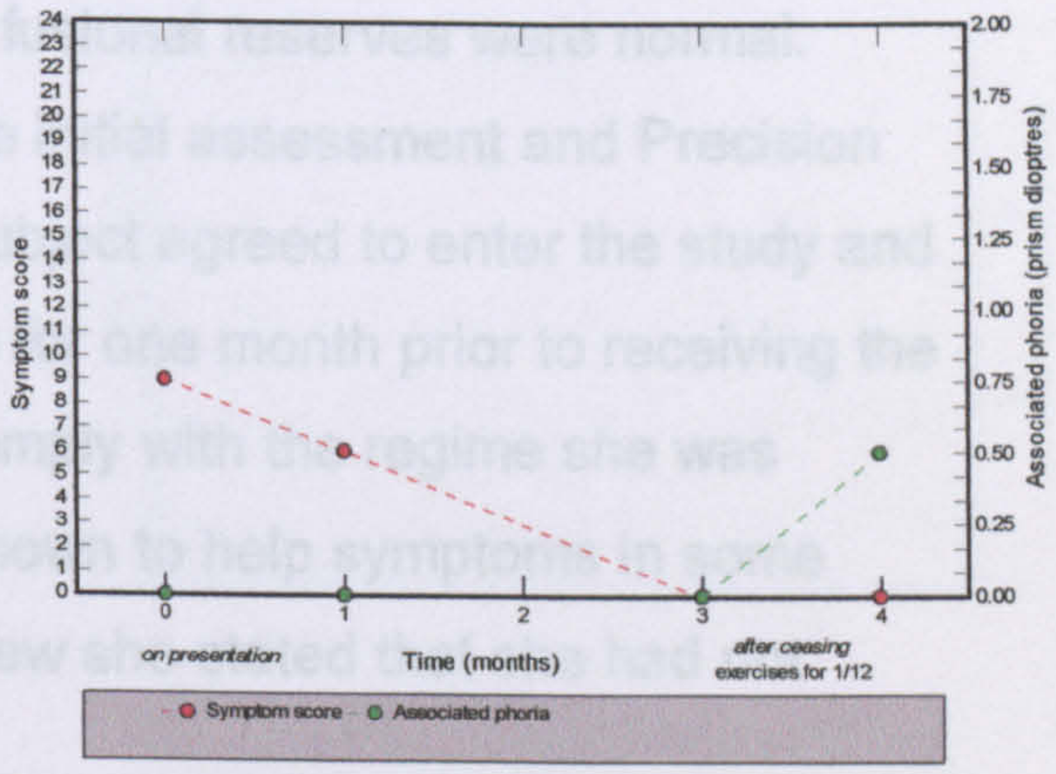


Figure 7.81. Changes in symptom score and associated phoria over the treatment period for subject vcurt.

Comment

Whilst there was a small reduction in symptoms reported after one month of carrying out the exercises, the subject recognised a sudden and complete resolution of her difficulties when the lighting was changed in her office.

VI. Case history

Subject	*vicup*
Age	28 years-old
Sex	Female
Occupation	Film producer
Symptoms	Complained of aching eyes, blurred vision, double vision, tired eyes and loss of concentration after about one hour of close work
Reported compliance	Poor
Resolution of symptoms	Unresolved
Follow-up	See discussion
Outcome	Unsuccessful

This subject was referred to the author for a visual assessment with particular reference to her dyslexia. Although she manifested an exophoria at near, this was fully compensated and her NPC and fusional reserves were normal. Intuitive Colorimetry was carried out at the initial assessment and Precision tinted lenses had been prescribed. The subject agreed to enter the study and undergo the course of orthoptic exercises for one month prior to receiving the tinted spectacles. To encourage her to comply with the regime she was informed that eye exercises have been shown to help symptoms in some subjects. However, at the one-month review she stated that she had not carried out the exercises regularly.

She was dispensed the tinted spectacles at the one-month review appointment. She wished to cease the exercises as she felt she was "too busy" to do these and was asked to return for review in six months.

Figure 7.82 shows a slight increase in the symptom score accompanied by a slight decrease in NPC at the one-month review.

Figure 7.83 shows an increase in recovery time following the cover test at the one-month review.

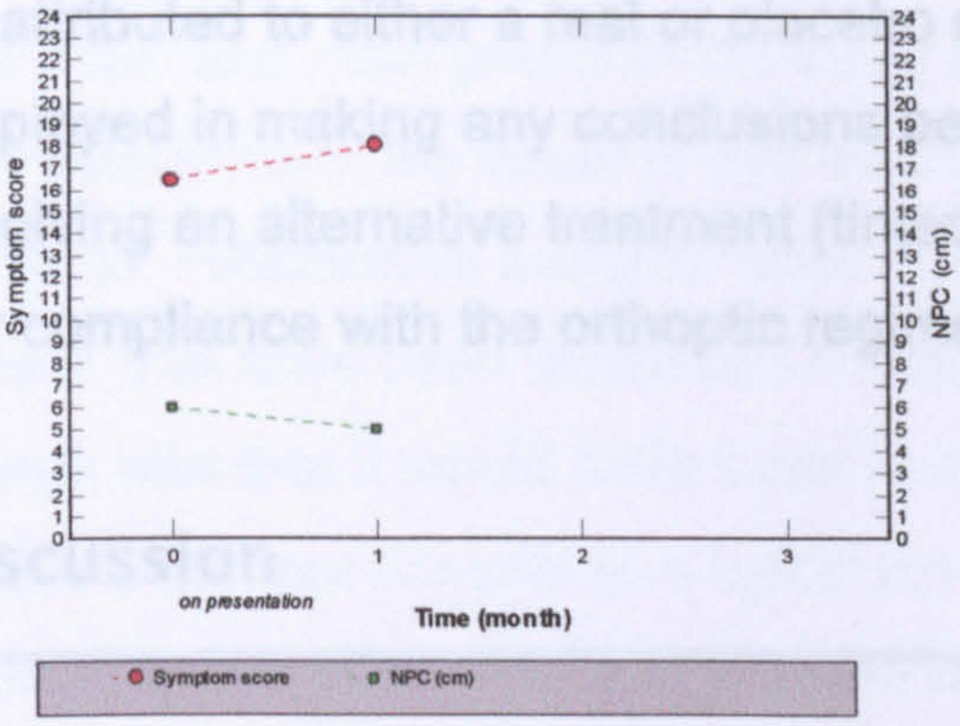


Figure 7.82. Changes in symptom score and NPC over the treatment period for subject vicup.

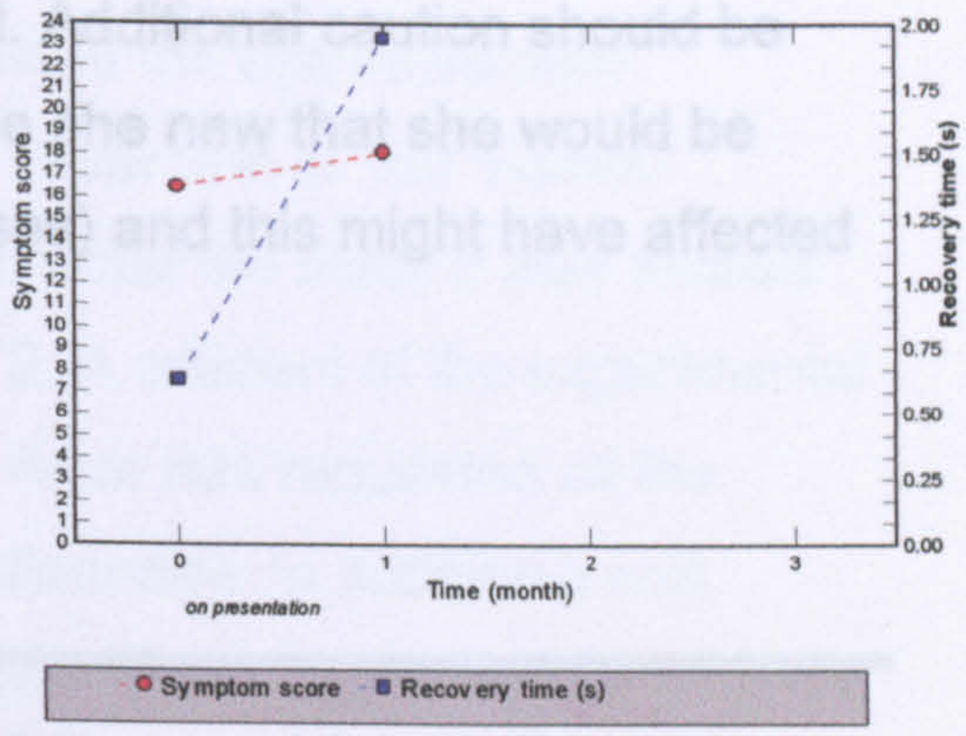


Figure 7.83. Changes in symptom score and recovery time over the treatment period for subject vicup.

Figure 7.84 shows a reduction in blur and break fusional reserve measures at the one-month review with an increase in the recovery measures. Although there was no observed associated phoria at either visit (Figure 7.85), the subject did report the monocular markers on the near Mallett unit to be unstable, at the one-month review.

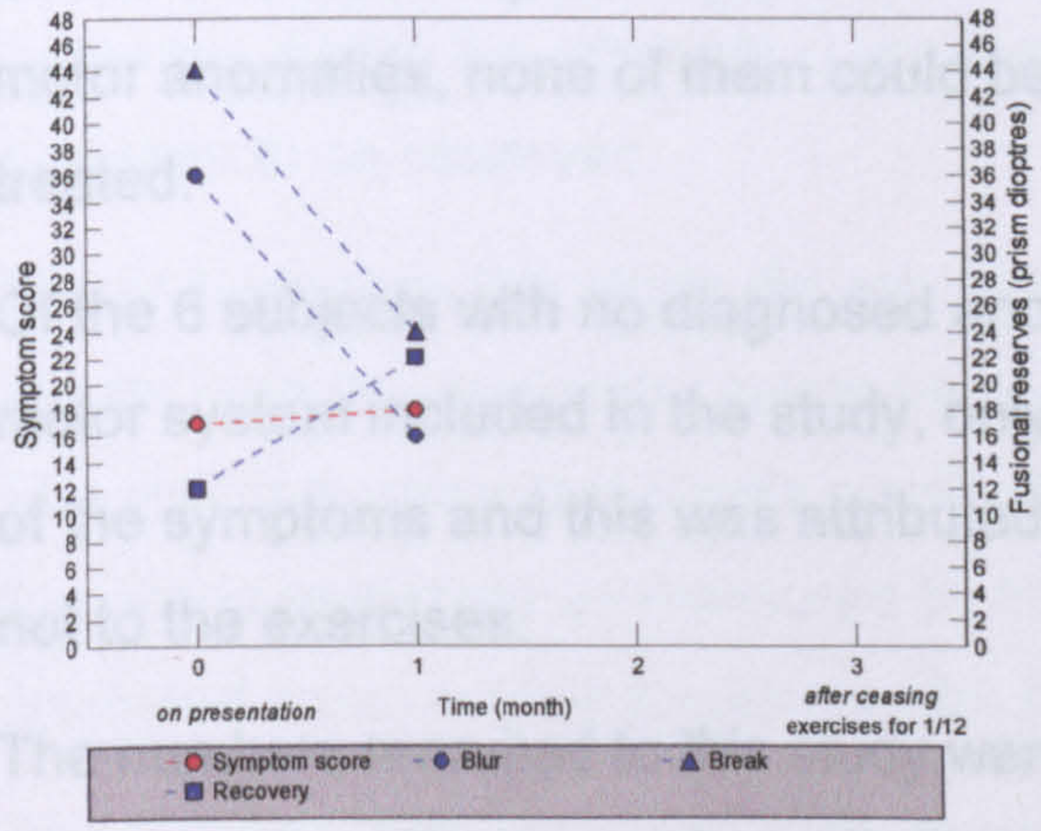


Figure 7.84. Changes in symptom score and fusional reserves over the treatment period for subject vicup.

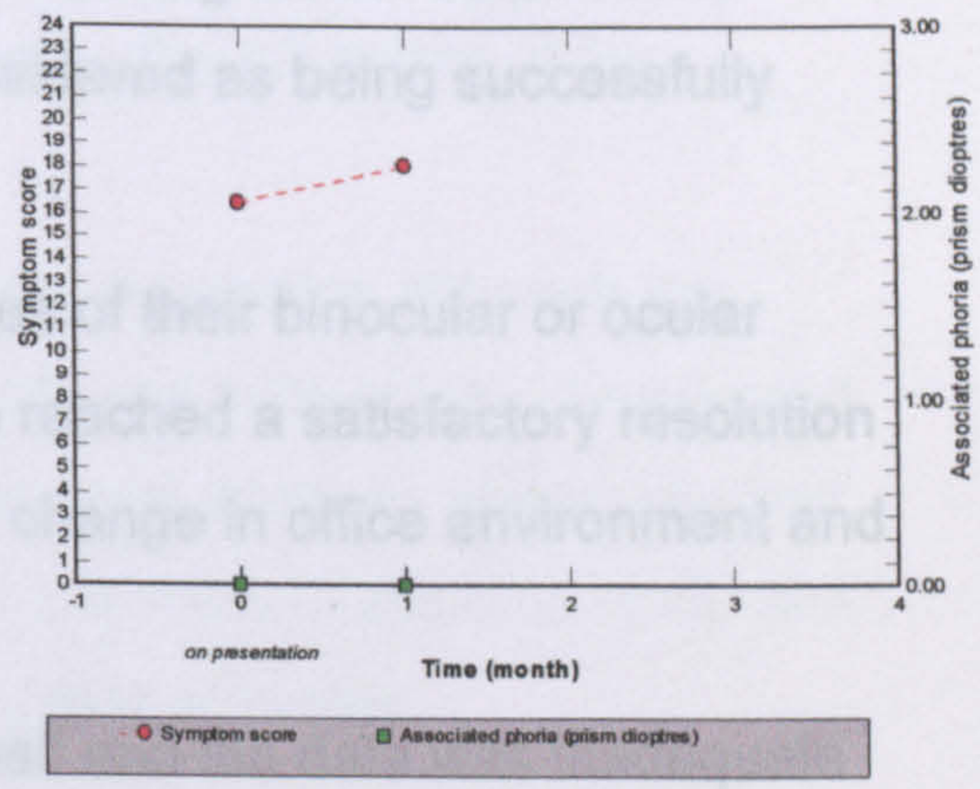


Figure 7.85. Changes in symptom score and associated phoria over the treatment period for subject vicup.

Comment

The orthoptic exercises failed to produce a reduction in symptoms that could be attributed to either a real or placebo effect. Additional caution should be employed in making any conclusions because she new that she would be receiving an alternative treatment (tinted lenses) and this might have affected her compliance with the orthoptic regime.

7.5 Discussion

12 subjects with CI attended for at least one follow-up. Of these 5 subjects obtained a satisfactory resolution of their symptoms after carrying out the orthoptic regime. This included one subject *(adeoj)* whose symptom score was minimal on presentation.

The remaining subjects were not successful. For some subjects this lack of success may have been contributed to by non- or poor compliance with the exercise regime. One subject's symptoms were subsequently satisfactorily resolved following refractive intervention.

Of the 3 non-CI subjects treated with the exercise regime for other ocular motor anomalies, none of them could be considered as being successfully treated.

Of the 6 subjects with no diagnosed anomalies of their binocular or ocular motor system included in the study, only one reached a satisfactory resolution of the symptoms and this was attributed to a change in office environment and not to the exercises.

The numbers recruited to this study were small and the data was inadequate to explore hypotheses such as whether there would be a reduction in recovery time following the cover test associated with successful resolution of binocular/ocular motor anomalies.

Chapter 8. Discussion

The initial and main phase of the study examined the eye movement characteristics of the covered eye during the cover test of 100 'normal' subjects. Prospective subjects were invited to enter the study if they fulfilled criteria that have been discussed in Chapter 2. A criticism of the experimental design was that it would have been useful to have had requested all the subjects to have completed a symptom questionnaire. In addition it was recognised, in retrospect that it would have been advantageous if all subjects had undergone a full binocular vision investigation rather than a screening to rule out the presence of a disqualifying refractive error, manifest strabismus, or amblyopia. However, at the time it was judged that some restriction on numbers needed to be made because of time constraints imposed by carrying out a full work-up on each subject.

8.1 A comparison between a 2 s, 10 s and alternate cover test

Cooper (1992) and Evans (1997) suggested that the phoria amplitude usually *increases* during the alternate cover test allowing the full extent of the deviation to be observed.

The findings from this study did not confirm this suggestion although the results must be used with caution because only one subject was investigated. The alternate cover test phoria amplitude remained relatively stable whereas the phoria amplitude as measured by the 2 s intermittent unilateral cover test produced a decreasing amplitude with the number of cycles.

The recovery time increased with repeated occlusion during the alternate cover test. The main reason for the increase appeared to be due to an increase in latency. It is uncertain why this phenomenon should arise. Possibilities may include a behavioural effect such as a loss of attention. Alternatively, the repeated alternate unilateral exposure of the fixation target may produce an enhanced *procrastination* effect. However, a further question arises as to whether the alternate cover test data truly reflect the latency of

the movement of the eye taking up fixation on removal of the cover or whether the dominant factor is the disruption of fixation of the fellow eye which previously had been fixating.

The findings of this limited pilot study suggest that a further investigation is warranted.

8.2 Eye movement characteristics of 100 'normal' subjects during the cover test

The initial phase of the study entailed the recording and analysis of the eye movement characteristics of 100 'normal subjects. The mean 10 s phoria amplitude^{RL} for distance was 0.00° of heterophoria with a range from 2.51° exophoria to 3.03° esophoria. These results are similar to the findings (Dowley, 1979; Schor & Ciuffreda, 1983). For near, analysis of 10 s *phoria amplitude*^{RL} showed the mean phoria was 1.38° of exophoria with a range from 5.62 ° exophoria to 3.48 ° esophoria. For emmetropes aged 18 to 35 years, exophoria was more common than esophoria for near fixation. It was not uncommon to find a small esophoria in the distance and exophoria at near. It was uncommon to find an exophoria in the distance and an esophoria at near.

Most eye care practitioners in clinical practice assume that the amplitude of phoria, for any patient, is normally equal in both eyes. It is usual for just one measurement to be recorded in the patient's notes. The results of this study suggest that it is indeed reasonable for practitioners to record their phoria estimations on "normal" young adult subjects making the assumption that the measure is independent of which eye is covered.

Information about the fusional decay profile can be gleaned by comparing 2 s and 10 s amplitudes. The difference between the phoria amplitude measured after 2 s occlusion and 10 s occlusion increased with increasing phoria amplitudes. However, larger amplitude phorias did not necessarily take longer to reach a position of equilibrium during dissociation when compared to

smaller phorias. In addition, for any one subject, one eye may show a different fusional decay profile compared to its fellow.

Most descriptions of the conventional cover test recommend an occlusion period of approximately 1 s to 2 s (von Noorden, 1990; Stidwill, 1990; Evans, 1997). An analysis of variance for repeated measures revealed a statistically significant difference between the 2s and 10s phoria amplitudes. This, together with the finding that the mean times to reach the 10 second amplitude were greater than to reach 2 seconds, suggests that, in many cases, the eye had not reached a state of equilibrium at 2 s.

Some eyes took at least 10 seconds to reach the maximum amplitude with a possibility that they have not even reached a position of equilibrium by that time. These results confirm the findings of Peli and McCormack (1983) who reported eyes taking up to 8 s to reach a position of equilibrium. This study has shown that the mean time for eyes to reach the amplitude of phoria measured after 10 s of occlusion was between 4 and 5 seconds. There was no significant difference in these findings between the 'normal' and the 'referred group'. These findings suggest that practitioners may wish to consider occluding each eye for longer than the 1 s or 2 s when estimating the amplitude of phoria.

Vergence latencies have previously been reported as being of the order of 0.20 s (Westheimer and Mitchell, 1956). Typical values for saccadic reaction time are of the order of 180 to 220 ms measured from target appearance (Baloh and Honrubia, 1976). In this study, the mean latency time for any type of first recovery movement was 0.29 s (range 0.07 to 6.78 s). There was a statistically significant difference ($p = <0.001$) between distance latencies (mean = 0.26 s; range 0.69 s to 0.98s) and for near (mean =0.31 s; range 0.07 s to 6.78 s). There was a statistically significant difference between distance and near convergence eye movement latency periods ($p = 0.01$).

It had been reported that convergence responses, in general, have faster dynamics than the divergence responses (Zuber and Stark, 1968; Mitchell, 1970). However, unlike previous reports, Krishnan et al (1973) found that divergence latencies tended to be shorter than convergence latencies.

Krishnan et al (1973) reported fusional vergence latencies of between 130 and 250 ms for temporally unpredictable stimuli.

The results of this study supported neither Zuber and Stark (1968) and Mitchell (1970) nor Krishnan et al (1973) as no statistically significant difference was found between convergence and divergence latencies for either distance or near.

However, for near fixation, both divergence and convergence latencies were statistically significantly shorter than saccadic latencies ($p = 0.01$ and $p < 0.001$ respectively). There was also a statistical difference between adducting and abducting saccades ($p = 0.09$).

There was a significant statistical difference between exophores and esophores for frequencies of initiating saccades and vergence eye movements ($p < 0.001$) suggesting that esophores more commonly commence recovery with a saccade and exophores with a vergence eye movement. The author is unaware of any previous reports of this finding. A possible reason why esophores more commonly commence recovery with a saccade is that divergence is relatively more passive as compared to convergence. Although neurons in the striate and prestriate cortex are capable of eliciting divergence (see Chapter 1), the evidence for active divergence is less compelling than for convergence with its tonic, proximal and accommodative components.

The median number of distinct eye movements made to achieve recovery following the distance cover test in the normal group was 2 (range = 1 to 5). There was no significant difference between exophores and esophores.

For the near cover test, there was a significant statistical difference between exophores and esophores for the number of eye movements to achieve recovery ($p = 0.001$; 95% confidence interval = 0.00 to 1.00). For near exophores, the median number of distinct eye movements made to achieve recovery in the normal group was 3 (range = 1 to 8). The median for near esophores was 2 (range = 1 to 3).

The author hypothesises that the anecdotal descriptions of 'smooth' or 'jerky', used by practitioners to describe recovery movements following the cover

test, relate to the number of eye movements made to achieve recovery. It is suggested that an increased number of distinct eye movements will produce a more 'jerky' appearance. The results alluded to above would predict that near exophores would produce more 'jerky' recoveries than near esophores.

The mean recovery time for all eyes following the distance cover test was 0.86 s (range = 0.15 s to 5.41 s). The mean recovery time for all eyes following the near cover test was 1.05 s (range 0.16 s to 7.75 s). The maximum end of the range reflects very slow recovery times and any such eye was manifesting a heterophoria that had in clinical terms, 'broken down' into a squint. These cases were of interest as the subjects concerned entered the study as 'normal', asymptomatic subjects with no known history of binocular vision anomalies.

There were very poor correlations between amplitude of phoria and recovery time for both exophores and esophores following both the distance and near cover tests. This was expected because of the variation between eyes for latency times, the type of eye movements and the number of recovery movements used to reach the recovery position.

There was also a very weak correlation between the recovery times for the right and left eyes following the distance and near cover test. This tends to confirm the observation that recovery may be rapid when the occluder is removed from one eye and slow when removed from the other eye (Lyle & Wybar, 1967). Any possible relationship with dominance was not assessed in this current study and it is suggested that this is an area for further study..

Exophoric eyes exhibiting a single eye movement to achieve recovery recovered statistically significantly faster than exophoric eyes showing 3 or more eye movements to reach the recovery position. This finding tends to support the anecdotal reports of the 'fast-smooth' recovery compared to the 'slow-jerky' recovery. These findings merit further investigation.

8.3 Comparison of the cover test with other tests of binocular function

A number of studies have compared the level of agreement of the amplitudes of horizontal phoria measured with the cover test and with other procedures (Calvin et al, 1996; Rainey et al, 1998; Schroeder et al, 1996).

In this study, the 10 s automated cover test showed a poor level of agreement with both the distance and near conventional cover test, and with the Maddox wing (near). However, it showed a level of agreement (2 SD = $\pm 1^1/_2{}^{\Delta}$) with the Maddox rod (distance) that may be described as clinically acceptable for the measurement of horizontal phoria amplitude. A possible reason for the latter finding was that both techniques cause full dissociation for enough time to allow the eye to reach a position of relative equilibrium. By contrast, the Maddox wing does not allow full dissociation, there being a small area of the back-plate that may be seen binocularly. Finally, as previously alluded to, the conventional (or ~ 2 s) cover test is less likely produce a stable position of rest when compared to the 10 s procedure.

8.4 Eye movement characteristics during the cover test of 30 referred subjects

The mean distance phoria amplitude^{RL} for the referred group at first presentation was 0.83° exophoria with a range of phoria amplitude from 7.45° exophoria to 1.89° esophoria. There was a significant statistical difference in distance phoria amplitude between the referred population and the normal group ($p = 0.03$). The mean near phoria amplitude^{RL} for the referred group at first presentation was 3.06° exophoria with a range of 7.72° exophoria to 0.31° esophoria. 28 subjects manifested exophoria at near and 2 were esophoric. There was a statistically significant difference in near phoria amplitudes between the referred population and the normal group ($p = 0.001$).

The subjects in the referred group had been selected primarily because of symptoms that had been attributed to a suspected horizontal ocular motor anomaly. Although amplitude of phoria alone should not have been a criterion for referral to the study, it was possible that larger amplitudes of near phoria might have raised the index of suspicion of referring practitioners. It is possible that this caused them to err towards referring a symptomatic patient manifesting a large amplitude of near phoria and complaining of near vision symptoms. However, it was of interest to note that the referred group also had a statistically significant difference in distance phoria amplitude.

There was no significant difference in recovery latency between normal and referred groups but there was a marginal difference ($p = 0.09$) in near recovery time of exophoric eyes between the normal and referred groups.

There was a statistically significant difference between the normal group and the referred group for the number of eye movements required to achieve recovery for both distance ($p = 0.004$; 95% confidence interval = 0 to 1) and near ($p = 0.0002$; 95% confidence interval = 0 to 1) cover tests. The 95% confidence interval of 1 eye movement is of clinical interest. It would be expected that the more distinct movements made to reach the recovery position, the 'jerkier' the appearance would be to the clinician carrying out subjective observations during the conventional cover test.

The frequencies of initiating saccades and vergence eye movements were very similar for near exophores in both the normal and referred groups with vergence being more common.

8.5 Symptom scores

A fast recovery, following the cover test, has been alluded to as being a sign of a 'well compensated phoria' (Stidwill, 1990; Evans, 1997). The presence of symptoms has been demonstrated to be correlated with associated phoria (Sheedy & Saladin, 1977; Schor, 1983; Jenkins et al, 1989).

Overall there was poor correlation between recovery times and symptoms. However, it was of interest to observe that, for the combined normal and

referred group, those subjects manifesting recovery times^{RL} falling into the interquartile range of distribution (0.64s to 0.96 s), produced significantly higher symptom scores than those subjects who showed recovery times^{RL} of less than 0.64 s or more than 0.96 s.

These findings tend to support the long held anecdote that a fast recovery movement suggests that a phoria is well compensated and by deduction, will not causing symptoms. However, what these findings also suggest is that very slow recovery movements of about 1s or greater are not associated with a high symptom score. The author hypothesises that the presence of a very slow recovery movement is an indicator of a sensory strategy, such as suppression, that operates to prevent symptoms occurring. The Mallett test of binocularity, which was employed as a test of suppression in this study, was shown to be a poor discriminator with regard to symptoms and there was a very low prevalence of subjects reporting any degree of central suppression. The author suggests that this area warrants further investigation.

Whilst there was a statistically significant difference in the number of recovery movements^{RL} between the lower quartile and interquartile groups of subjects categorised by recovery time^{RL}, there was no convincing evidence of a strong relationship between the number of recovery movements and symptom scores.

Although there was a poor linear relationship between symptom scores and NPC, there was a significant difference in symptom scores between those subjects with an NPC of < 10 cm and those with an NPC of 10 cm or greater. This suggests that the use by clinicians of 10 cm as a diagnostic cut off point is justified in terms of symptoms.

8.6 The effect of orthoptic treatment on cover test eye movements

The aim of this small study was to investigate the effect of a regime of orthoptic exercises on eye movement characteristics during the cover test.

There were some criticisms relating to the design of the study and these have been addressed in Chapter 7.

30 subjects entered the study predominantly because of symptoms associated with, or suspected of being associated with, a horizontal ocular motor anomaly. Subjects were grouped into those with CI, those with other ocular motor anomalies, and those for whom a full examination showed that an ocular motor anomaly was unlikely to be a cause of the reported symptoms. 12 subjects with CI attended for at least one follow-up. Of these 5 subjects obtained a satisfactory resolution of their symptoms after carrying out the orthoptic regime. This included one subject *(adeoj)* whose symptom score was minimal on presentation.

The remaining subjects were not successful. For some subjects this lack of success may have been contributed to by non- or poor compliance with the exercise regime. One subject's symptoms were subsequently satisfactorily resolved following refractive intervention.

Of the 3 non-CI subjects treated with the exercise regime for other ocular motor anomalies, none of them could be considered as being successfully treated.

Of the 6 subjects with no diagnosed anomalies of their binocular or ocular motor system included in the study, only one reached a satisfactory resolution of the symptoms and this was attributed to a change in office environment and not to the exercises.

A criticism of this study was that the numbers recruited to this study were disappointingly small. The data were inadequate to explore hypotheses such as whether there would be a reduction in recovery time following the cover test associated with successful resolution of binocular/ocular motor anomalies. However, with the experience gained from this study, a future, preferably double masked placebo controlled study, may be more fruitful.

8.7 Conclusion

The findings of this study suggest that an occlusion period of about 10 s would produce a position of equilibrium for the covered eye in only approximately 80% of eyes (see Figures 3.19 & 3.20). Assuming that it is desireable to reach equilibrium, it is recommended that, when carrying out the cover test, practitioners occlude an eye for longer than the 2 s that appears to be the current *modus operandi.* Although in the past longer periods of occlusion have been alluded to (Clarke, 1893; Percival, 1928), to routinely occlude an eye for longer than about 10s would make the cover test difficult to carry out routinely in the clinic setting. However, the author wishes to support a clinical pearl of wisdom on this matter received from a highly respected practitioner of a previous generation. Earnshaw (personal communication, 1998) explained the usefulness of observing an eye when removing the occluder following a monocular refraction of the fellow eye, a technique he regularly employs.

This study has shown that there is indeed a relationship between recovery time and reported symptoms. As has been alluded to anecdotally by practitioners, a fast recovery of less than approximately 0.6 s is likely to be a good sign in terms of symptoms. However, a really slow recovery time of greater than approximately 1 s is also likely to be associated with a low symptom score. It is the intermediate recovery times that are associated with symptoms. Although it is likely that practitioners would have difficulty in subjectively grading recovery times into 'fast', 'intermediate' and 'slow', it is suggested that this would be a useful further area of study.

Whereas a relationship between recovery time and symptoms was demonstrated, the relationship between the number of recovery eye movements and symptoms was weaker. Nonetheless, the referred population did make significantly more recovery eye movements than the normal population.

The cover test has become established part of the optometric examination despite limited evidence of its value or usefulness. Within the context of a clinical test, 'usefulness' is difficult to define but probably relates to the ability

of the test to predict symptoms, help the practitioner to reach a diagnosis, or guide the clinician towards a successful form of management and monitor the effect of treatment.

Is the cover test useful within this definition? The size of phoria is poorly compensated with symptoms. The anecdotal view that the 'speed' and 'smoothness' of recovery is a good predictor of symptoms is given some credence by this study although the correlation is poor. The cover test is helpful in determining a diagnosis – firstly of strabismus/heterophoria and secondly the direction and magnitude of the deviation. This information is of importance when considering the management of patients.

Other tests of binocular function are not well correlated with eye movement characteristics during the cover test, and indeed show little relationship to each other. Are any of them useful? It is perhaps unfair to consider individual tests in isolation as the clinician will generally look at the pattern of results across a variety of tests. The numbers involved in this study were insufficient to perform the multilinear regression between these variables in order to investigate co-variance. Only by performing such as study will the practitioner be able to carry out an evidence-based approach to the treatment of ocular motor anomalies.

The second question that we sought to address was what aspect of binocular function (if any) is changed by a regime of orthoptic exercises. The ideal randomised control design was thwarted by ethical considerations and the difficulties of recruiting a reasonably homogenous group of subjects. The small number of subjects recruited and their rather diverse characteristics, limited the value of this part of the study. However, by considering these subjects as individual cases it has been possible to provide some objective evidence for the efficacy of orthoptic treatment. Not all subjects respond to treatment and none of the clinical tests assessed were found to be good discriminators of subjects who were likely to benefit.

Furthermore, while several aspects of binocular function were found to parallel the amelioration of symptoms, the results were very variable.

Despite the growing body of literature relating to binocular vision and ocular motor function, there is surprisingly little information about the meaning and value of many clinical tests that are routinely carried out. This study has for the first time, provided a detailed analysis of one of the most common clinical tests – the cover test.

This work needs to be expanded to include other tests and the relationships between them. Only then can the clinician provide proper management of patients with ocular motor problems.

BLANK IN ORIGINAL

References

1. Abraham SV (1951) The nature of heterophorias. *Am J Ophthalmol, 34, 1007- 16*

2. Abraham SV (1964) The basic position of rest - convergence and divergence. *J Paed Ophthalmol, 1, 9-24*

3. Adams AJ (1978) Acute effects of alcohol and marijuana on vision. In *Frontiers in visual science.* Eds. Cool SJ, Smith EL, pp 93-105, Springer-Verlag, New York

4. Afandor AJ (1982) Auditory biofeedback and intermittent exotropia. *J Am Optom Assoc, 53, 481-483*

5. Alpern M (1969) Types of eye movement. In *The Eye.* Ed Davson H, Vol 3, 2nd edition, pp 65 -174, Academic Press, New York

6. Ames A Jr, Glidden GH (1928) Ocular measurements. *Trans Sect Ophthalmol AMA, 102 - 75*

7. Anderson M (1961) Orthoptic treatment of loss of convergence and accommodation caused by road accidents ("whiplash injury"). *Br Orthopt J, 18, 117-120*

8. Bahill AT, Brockenbrough A, Troost T (1981) Variabilty and development of a normative data base for saccadic eye movements. *Invest Ophthalmol Vis Sci, 21, 116 -125*

9. Bahill AT, Clark MR, Stark L (1975a) The main sequence, a tool for studying human eye movements. *Math Biosci, 24, 191- 204*

10. Bahill AT, Adler D, Stark L (1975b) Most naturally occurring human saccades have magnitudes of 15 degrees or less. *Invest Ophthalmolol 14:468 - 469*

11. Bahill AT, Ciuffreda KJ, Kenyon RV, Stark L (1976) Dynamic and static violations of Hering's Law of equal innervation. *Am J Optom Physiol Optics, 53, 786 - 796*

12. Bahill AT, Stark L, (1975) Overlapping saccades and glissades are produced by fatigue in the saccadic eye movement system. *Exp Neurol, 48, 95 -107*

13. Bahill AT, Stark L, (1979) The trajectories of saccadic eye movements. *Sci Am 240,1, 85-93*

14. Bahill AT, Troost BT (1979) Types of saccadic eye movements. *Neurology, 29, 1150-1152*

15. Baloh RW, Honrubia V (1976) Reaction time and accuracy of the saccadic eye movements of normal subjects in a moving-target task. *Aviat Space Environ Med 47: 1165-1167*

16. Barnard NAS (1989) Visual conversion reaction in children. *Ophthal Physiol Opt, 9, 371-378*

17. Barnard NAS, Thomson WD (1995a) A quantitative analysis of eye movements during the cover test - a preliminary report. *Ophthal Phys Opt, 15, 5, 413-419*

18. Barnard & Thomson (1995b) idiosyncratic variations of eye movement characteristics during the cover test. *Optom Vis Sci, 72 (Suppl), 201*

19. Barr ML, Kiernan JA (1993) *The Human Nervous System. An Anatomical Viewpoint.* 6th Ed. JB Lipincott Co, Philadelphia

20. Becker W (1972) The control of eye movements in the saccadic system. *Bibl. Ophthal, 82, 233-243* (Karger, Basel).

21. Becker W, Fuchs AF (1969) Further properties of the human saccadic system: Eye movements and correction saccades with and without visual fixation points. *Vision Res, 9, 1247-1258*

22. Becker W, Jurgens R (1979) An analysis of the saccadic system by means of double step stimuli. *Vision Res, 19, 967-983*

23. Bennett AG, Rabbetts RB (1989) *Clinical Visual Optics,* 2nd Ed, Butterworths, London, p 207

24. Berard PV (1968) Prisms - their therapeutic use in concomitant strabismus with normal retinal correspondence. In *Proceedings of the 1st International Congress of Orthoptics.* CV Mosby, St Louis

25. Bergmanson JPG (1995) *Clinical Ocular Anatomy and Physiology.* University of Houston College of Optometry

26. Berry GA (1893) *Disease of the eye,* 2nd Ed, p. 538. Pentland, Edinburgh

27. Boghen D, Troost BT, Daroff RB, Dell'Osso LF & Birkett JE (1974) Velocity characteristics of normal human saccades. *Invest Ophthalmol, 13, 619-623*

28. Borisch IM (1970) *Clinical Refraction.* 3rd Ed. The Professional Press, Chicago

29. Birnbaum MH (1993) Optometric Management of Nearpoint Vision Disorders, Butterworth-Heinemann, Boston

30. Bland JM, Altman DG (1986) Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet, 1, 307-310*

31. Braun D, Breitmeyer BG (1988) Relationship between directed visual attention and saccadic reaction times. *Exp Brain Res, 73, 546-552*

32. Brecher GA, Hartman AP, Leonard DD (1955) The effect of alcohol on binocular vision. *Am J Ophthalmol, 39, 44-52*

33. Burian HM (1939) Fusional movements: the role of peripheral retinal stimuli. Fusion Stimuli. *Arch Ophthalmol, 21, 486-491*

34. Caifa RP, Hebbard FW (1967) Involuntary eye movements occurring during fixation: effects of changes in target contrast. *Am J Optom Arch Am Acad Optom, 44, 73-90*

35. Caltrider N, Jampolsky A (1983) Overcorrecting minus lens therapy for treatment of intermittent exotropia. *Ophthalmology, 90, 10, 1160-1165*

36. Calvin H, Rupnow P, Grosvenor T (1996) How Good is the Estimated Cover Test at Predicting von Graefe Phoria Measurement ? *Optom Vis Sci, 73,11, 701-706*

37. Campbell FW, Wurtz RH (1978) Saccadic omission: why we do not see a grey-out during a saccadic eye movement. *Vision Res, 18, 1297-1303*

38. Campos EC, Catellani T (1978) Further evidence for the fusional nature of the compensation (or "eating up") of prisms in concomitant strabismus. *Int Ophthal, 1, 57-62*

39. Capobianco NM (1952) The subjective measurement of the near point of convergence and its significance in the diagnosis of convergence insufficiency. *Am Orthopt J, 2, 40-42*

40. Carroll R, Seaber JH (1974) Acute loss of fusional convergence following head trauma. *Am Orthopt J, 24, 57-59*

41. Carpenter RHS (1977) *Movements of the Eyes*, Pion Ltd, London

42. Carpenter RHS (1981) Oculomotor procrastination. In *Eye Movements: Cognition and Visual Perception*. Eds. Fisher D, Monty RA, Senders JW, 237-246, Lawrence Earlbaum, Hillsdale, NJ.

43. Carter DB (1965) Fixation disparity and heterophoria following prolonged wearing of prisms. *Am J Optom Physiol Opt, 42, 141-152*

44. Ciuffreda KJ, Tannen B (1995) *Eye Movement Basics For The Clinician*. Mosby, St Louis

45. Clarke E (1893) *Eyestrain*, pp 103-126, Churchill, London

46. Coffey B, Wick B, Cotter S, Scharre J, Horner D (1992) Treatment options in intermittent exotropia: a critical appraisal. *Optom Vis Sci, 69, 386-404*

47. Cohen AH, Soden R (1984) Effectiveness of visual therapy for convergence insufficiencies for an adult population. *J Am Optom Assoc, 55, 491-494*

48. Cohen ME, Ross LE (1978) Latency and accuracy characteristics of saccades and corrective saccades in children and adults. *J. Exp. Child Psychol, 26, 517-527*

49. Collewijn H (1999) Eye movement recording. Chapter 9 in *Vision Research. A Practical Guide to Laboratory Methods.* Eds. Carpenter RHS, Robson JG. pp 252-285. Oxford University Press, Oxford

50. Collewjin H, Erkelens CJ, Steinman RM (1988a) Binocular co-ordination of human horizontal saccadic eye movements. *J Physiol (Lond), 404, 157-182*

51. Collewijn H, Erkelens CJ, Steinman RM (1995) Voluntary binocular gaze-shifts in the plane of regard: dynamics of version and vergence. *Vision Res, 35, 3335-3358*

52. Colson Z ((1940) The effect of alcohol on vision. Experimental investigation. *J Am Med Ass, 115, 1525-1527*

53. Cooper J (1992) Clinical Implications of vergence adaptation. *Optom Vis Sci, 69, 4, 300-307*

54. Cooper J, Duckman R (1978) Convergence insufficiency: incidence diagnosis and treatment. *Am J Optom Physiol Opt, 49, 673-680*

55. Cooper J, Feldman JM, Eichler R (1992) Relative strength of central and peripheral fusion as a function of stimulus parameters. *Optom Vis Sci, 69, 966-972*

56. Cooper J, Selenow A, Ciuffreda KJ, Feldman J, Faverty J, Hokoda SC, Silver J (1983) Reduction of asthenopia in patients with convergence insufficiency after fusional reserve training. *Am J Optom Physiol Opt, 60, 982-989*

57. Cridland N (1964) The deviation behind the cover. *Br Orthopt , 21, 63-67*

58. Crone RA, Hardjowijoto S (1979) What is normal binocular vision ? *Doc Ophthalmol, 47, 163-199*

59. Daum KM (1982) The course and effect of visual training on the vergence system. *Am J Optom Physiol Opt* 59, 223-227

60. Daum KM (1983a) Analysis of seven methods of measuring the angle of deviation. *Optom Vis Sci, 60, 1, 46-51*

61. Daum KM (1983b) A comparison of the results of tonic and phasic vergence training. *Am J Optom Physiol Opt, 60, 769-765*

62. Daum K (1984a) Classification criterion for success in the treatment of convergence insufficiency. *Am J Optom Physiol Opt, 61, 10-15*

63. Daum K (1984b) Convergence insufficiency. *Am J Optom Physiol Opt, 61, 16-22*

64. Daum KM (1986a) Characteristics of exodeviations: 1. a comparison of three classes. *Am J Optom Physiol Opt, 63, 237-243*

65. Daum KM (1986b) Characteristics of exodeviations: II. changes with treatment with orthoptics. *Am J Optom Physiol Opt, 63, 244-251*

66. Daum KM (1986c) Double-blind placebo controlled examination of timing effects in the training of positive vergences. *Am J Optom Physiol Optics, 63, 807-811*

67. Daum KM (1988) Characteristics of convergence insufficiency. *Am J Optom Physiol Opt, 65, 426-438*

68. Daum KM, Rutstein RP, Eskridge JB (1987) Efficacy of computerized vergence therapy. *Am J Optom Physiol Opt, 64, 83-89*

69. Davies CE (1956) Etiology and management of convergent insufficiency. *Am Orthopt J, 6, 124-127*

70. Donders FC (1864) On the Anomalies of Accommodation and Refraction of the Eye. Translation by WD Moore. London: New Sydenham Society.

71. Dowley D (1990) Heterophoria. *Optom Vis Sci, 67, 456-460*

72. Drucker AP, Sadove MS, Unna KR (1951) Ophthalmic studies of curare and curare like drugs in man. *Am J Ophthalmol, 34, 543-553*

73. Duane A (1897) A new classification of the motor anomalies of the eyes based on physiological principles, together with their symptoms, diagnosis and treatment. *Ann Ophthalmol Otolayringol, 6, 84-130*

74. Dwyer P (1992) The prevalence of vergence accommodation disorders in a school-age population. *Clin Exp Optom, 75, 10-8*

75. Dwyer P, Wick B (1995) The influence of refractive correction upon disorders of vergence and accommodation. *Optom Vis Sci, 72, 224-232*

76. Eames TH, Cambridge M (1933) Physiological exophoria in relation to age. *Arch Ophthalmol, 9, 104-105*

77. Earnshaw R, (1998) *Personal communication.*

78. Effert R, Pflibsen K (1986) A new method to perform the cover test. *Ophthalmology, 93, 433-435*

79. Ellerbrock VJ (1950) Tonicity induced by fusional movements. *Am J Optom Arch Acad Optom, 27, 8-20*

80. Enright JT (1984) Changes in vergence mediated by saccades, *J Physiol, 350, 9-31*

81. Erkelens C J (1987) Adaptation of ocular vergence to stimulation with large disparities. *Experimental Brain Research*, 66 507-516

82. Erkelens CJ, Collewijn H, Steinman RM (1989a) Asymmetrical adaptation of human saccades to anisometropic spectacles. *Invest Ophthalmol Vis Sci, 30, 1132- 1145*

83. Erkelens C J, Steinman RM, Collewijn H (1989b) Ocular vergence under natural conditions. II. Gaze shifts between real targets differing in distance and direction. *Proceedings of the Royal Society of London B*, 236, 441-465

84. Eustace P, Wesson ME, Druby DJ (1973) The effect of illumination on intermittent divergent squint of the divergence excess type. *Trans Ophthalmol Soc UK, 93, 559-570*

85. Evans BJW (1997) *Pickwell's Binocular Vision Anomalies. Investigation & Treatment.* 3rd Ed, Butterworth Heinemann, Oxford

86. Feldman JM, Cooper J, Reinstein F, Swiatoca J (1992) Asthenopia Induced by Computer-Generated Fusional Vergence Targets *Optom Vis Sci, 69, 9, 710-716*

87. Feldman JM, Cooper J (1998) Assessing the reliability and validity of an asthenopia questionnaire. *Optom Vis Sci, 75, 12s,253*

88. Feldon SE, Burde RA (1987) The oculomotor system. Chapter 5 in *Adlers's Physiology of the Eye*. Eds. Moses & Hart, CV Mosby Co, St Louis

89. Fincham EF (1961) The accommodative reflex and its stimulus. *Br J Ophthalmol, 35, 381-393*

90. Fischer B, Boch R (1983) Saccadic eye movements after extremely short reaction times in the monkey. *Brain Res, 260, 21-26*

91. Fischer B, Breitmeyer B (1987) Mechanisms of visual attention revealed by saccadic eye movements. *Neurophysiologia, 25, 73-83*

92. Fischer B, Ramsperger E (1984) Human express saccades: extremely short reaction time of goal directed eye movements. *Exp Brain Res, 57, 191-195*

93. Fischer B, Weber H (1993) Express saccades and visual attention. *Behavioural & Brain Sciences, 16,553-610.*

94. Fisher SK, Ciuffreda KJ, Levine S, Wolf-Kelly KS (1987) Tonic adaptation in symptomatic and asymptomatic subjects. *Am J Optom Physiol Opt, 64,333- 343*

95. Fisher SK, Ciuffreda KJ, Tannen B (1988) Stability of tonic vergence. *Invest Ophthalmol Vis Sci, 29, 1577-1581*

96. Fisher SK, Ciuffreda KJ, Bird JE (1990) The effect of stimulus duration on tonic accommodation and tonic vergence. *Optom Vis Sci, 67, 441-449*

97. Flick C (1937) *Dioptric Review, 34, 124*

98. Fogt N, Jones R (1997) Comparison of the monocular occlusion and a direct method for objective measurement of fixation disparity. *Optom Vis Sci, 74, 43-50*

99. Franklin A (1997) The Cover Test. *CE Optometry, 1, 1, 8-9*

100. Freier BE, Pickwell LD (1983) Physiological exophoria. *Ophthal Physiol Opt, 3, 267-272*

101. Fry GA (1964) Fundamental variables in the relationship between accommodation and convergence. *Optom Wkly, September, 3, 21-25*

102. Georgievski Z (1994) The effects of central and peripheral visual field masking on fusional-disparity vergence. *Australian Orthoptic Journal, 30, 41-48*

103. Gibson HW (1955) *Textbook of Orthoptics,* Hatton Press, London

104. Giles GH (1943) *The Practice of Orthoptics*. 2nd Edition. Hammond, London

105. Goldrich SG (1982) Oculomotor feedback therapy for exotropia. *Am J Optom Physiol Opt, 59, 306-317*

106. Goodson RA, Rahe AJ (1981) Visual training effects on normal vision. *Am J Optom Physiol Opt, 58, 787-791*

107. Graefe E (1967) Messungen von heterophorien unter verschiedenen umfeldleuchtdichten, Dissertation, University Eye Clinic, Hamburg-Eppendorf, Hamburg

108. Green EL, Scobee RG (1951) Position of the Risley Prism in the Maddox-rod test *Am J Ophthalmol, 34, 211-217*

109. Gregerson E (1969) The polymorphous exo patient. Analysis of 231 successive cases. *Acta Ophthal (Kbh), 47, 579-590*

110. Griffin JR (1982) Binocular Anomalies: Procedures for Vision Therapy. 2nd edition, pp 140-147, Professional Press, Chicago

111. Griffin JR, Schultz WA, Vansuch JJ (1978) A comparison of two fixation disparity targets. *J Am Optom Assoc, 49, 905-907*

112. Grisham JD (1988) Visual therapy results for convergence insufficiency: a literature review. *Am J Optom Physiol Opt, 65, 448-454*

113. Grolman B (1971) Binocular refraction - fixation disparity. *Optician, 162, 16-19*

114. Grosvenor TP (1989) *Primary Care Optometry*. p. 284. 2nd Ed. Professional Press, New York

115. Haase HJ (1962) Binocular testing and distance correction with the Berlin Polatest. *J Am Optom Assoc, 34, 115*

116. Halldén U (1952) Fusional phenomena in anomalous correspondence. *Acta Ophthalmol (suppl) (Kbl), 37, 1-93*

117. Hebbard FW (1962) Comparison of subjective and objective measurements of fixation disparity. *J Opt Soc Am, 52, 706-712*

118. Henson DB, North R (1980) Adaptation to prism-induced heterophoria. *Am J Optom Physiol Opt, 57, 3, 129-137*

119. Hirsch MJ (1948) Clinical investigation of a method of testing phoria at forty centimeters. *Am J Optom Arch Am Acad Optom, 25, 492-495*

120. Hirsch MJ, Bing LB (1948) The effect of testing method on values obtained for phoria at forty centimeters. *Am J Optom Arch Am Acad Optom, 25,407-416*

121. Hoffmann FB, Bielschovsky A (1900) Uber dis der Willkue entzygenen Fusions - Bewegungen der Augen. *Arch Ges Physiol, 80, 1-40*

122. Hoffman L, Cohen AH, Feuer G (1973) Effectiveness of non-strabismic vision training in private practice. *Am J Optom Arch Am Acad Optom,50, 813-816*

123. Hogan RE, Gilmartin B ((1985) The relationship between tonic vergence and oculomotor stress induced by ethanol. *Ophthal Physiol Opt, 5, 43-51*

124. Hogan RE, Linfield PB (1983) The effects of moderate doses of ethanol on heterophoria and other aspects of binocular vision. *Ophthal Physiol Opt, 3, 1-31*

125. Hugonnier R, Clayette-Hugonnier S (1969) *Strabismus, Heterophoria and Ocular Motor Paralysis* 338-340, CV Mosby, St Louis

126. Hung GK (1992) Adaptation model of accommodation and vergence. *Ophthal Physiol Opt, 12, 319-326*

127. Hung GK, Ciuffreda KJ, Semmlow JL, Hornig JL (1994) Vergence eye movements under natural viewing conditions. *Invest Ophthal Vis Sci, 35, 3486-3492*

128. Hung GK, Ciuffreda KJ (1996) Schematic model of saccade-vergence interactions. *Med Sci Res, 24,12, 813-816*

129. Hung GK, Semmlow JL (1980) Static behaviour of accommodation and vergence: computer simulation of an interactive dual-feedback system, *IEEE Trans Biomed Eng , BME -27, 439-447*

130. Hung GK, Semmlow JL, Ciuffreda KJ (1986) A dual mode dynamic model of the vergence eye movement system. *IEEE Trans Biomed Eng, BME - 33,1021-1028*

131. Hyson MT, Julesz B, Fender DH (1983) Eye movements and neural remapping during fusion of misaligned random dot stereograms. *J Opt Soc Am, 73,1665-1673*

132. Iwasaki S (1990) Facilitation of reaction times with GAP paradigm: comparison of manual and saccadic responses. *Ergonomics, 33, 6, 833-850*

133. Jampolsky A, Ed.(1964) Ocular deviations. *Int Ophthalmol Clin, 4, 567-701*

134. Jaschinski W (1997) Fixation disparity and accommodation as a function of viewing distance and prism load. *Ophthal Physiol Opt, 17, 324-329*

135. Jaschinski-Kruza W (1994) Dark vergence in relation to fixation disparity at different luminance and blur levels. *Vision Res, 34, 1197-1204*

136. Jenkins TCA, Pickwell LD, Yekta AA (1989) Criteria for decompensation in binocular vision. *Ophthal Phys Opt, 9, 121-125*

137. Jenkins TCA, Abd-Manan F, Pardhan S, Murgatroyd RN (1994) Effect of fixation disparity on distance binocular visual acuity. *Ophthal Physiol Opt, 14, 129-131*

138. Jones R (1980) Fusional vergence: sustained and transient components. *Am J Optom Physiol Opt, 57, 9, 640-644*

139. Kenyon R V, Ciuffreda K J, Stark L (1980a) Dynamic vergence eye movements in strabismus and amblyopia: Symmetric vergence. *Invest Ophthalmol Vis Sci, 119, 60-74*

140. Kenyon R V, Ciuffreda K J, Stark L (1980b) An unexpected role for normal accommodative vergence in patients with strabismus and amblyopia. *Am J Optom Phys Opt, 57 586-594*

141. Kenyon R V, Ciuffreda K J, Stark L (1980c)Unequal saccades during vergence. *Am J Optom Phys Opt, 57,9, 586-594,*

142. Kenyon R V, Stark L (1983) Unequal saccades generated by velocity interactions in the peripheral oculomotor system. *Mathematical Biosciences, 63, 187-197*

143. Kertesz A (1982) The effectiveness of wide-angle fusional stimulation in the treatment of convergence insufficiency. *Invest Ophthalmol Vis Sci, 22, 690-693*

144. Kertesz AE, Hampton DR (1981) Fusional response to extrafoveal stimulation. *Invest Ophthalmol Vis Sci, 21, 600-605*

145. Kingstone A, Klein RM (1983) Visual offsets facilitate saccadic latency: Does predisengagement of visuospatial attention mediate this gap effect ? *J Exp Psych: Human Perception & Performance, 19, 1251-1265*

146. Klein R, Kingstone A, Pontefract A (1992) Orienting of visual attention. In *Eye movements and visual cognition: Science perception and reading.* Ed. Rayner K, p 46, Springer-Verlag, New York

147. Knapp P (1958) Treatment of divergent deviations. In *Strabismus Ophthalmic Symposium II* . p. 364-365. Ed. Allen JH, Kimpton, London

148. Kratka Z, Kratka WH (1956) Convergence insufficiency: its incidence and importance. *Am Orthoptic J, 6, 72-73*

149. Krishnan VV, Farazian F, Stark L (1973) An analysis of latencies and prediction in the fusional vergence system. *Am J Optom Arch Am Acad Optom, 50, 933-939*

150. Krzkystkowa K, Pajakowa J (1972) Retinal correspondence in divergent squint. *Klin Oczna, 42, 443-447*

151. Krishnan VV, Stark L (1977) A heuristic model for the human vergence eye movement system. *IEEE Trans Biomed Engin, BME-24, 1, 44-49*

152. Landolt E (1886) *The Refraction and Accommodation of the Eye and Their Anomalies*. Translation by Culver CM. Philadelphia: JB Lipincott.

153. Larson WL (1992) Prism adaptation without binocular vision is unlikely: a retraction of a claim to the contrary. *Optom Vis Sci, 69, 336*

154. Leigh RJ, Zee DS (1991) *The Neurology of Eye Movements*, 2nd Edition, FA Davis, Philadelphia

155. Letourneau JE, Lapierre N, Lamont A (1979) The relationship between convergence insufficiency and school achievement. *Am J Optom Physiol Opt, 56, 18-22*

156. Letourneau JE, Giroux R (1984) Using biofeedback in an exotropic aphake. *J Am Optom Assoc, 55, 909-910*

157. Letourneau JE, Ducic S (1988) Prevalence of convergence insufficiency among elementary school children. *Can J Optom, 50, 194-197*

158. Letourneau JE, Lapierre N, Lamont A (1979) The relationship between convergence insufficiency and school achievement. *Am J Optom Physiol Opt, 56, 18-22*

159. London RF, Wick B (1987) Vertical fixation disparity correction: effect on the horizontal forced-vergence fixation disparity curve. *Am J Optom*, 64, 653-655

160. Ludlam WM, Kleinman BI (1965) The long range results of orthoptic treatment on strabismus. *Am J Optom Arch Am Acad Optom, 42,647-684*

161. Ludvigh E (1949) Amount of eye movement objectively perceptible to the unaided eye. *Am J Ophthalmol, 32, 649-650*

162. Ludvigh E, McKinnon, P, Zaitzeff, L (1964) Temporal course of the relaxation of binocular duction (fusion) movements. *Arch Ophthal, 71, 389-399*

163. Ludvigh E, McKinnon, P, Zaitzeff, L (1965) Relative effectivity of foveal of foveal and parafoveal stimuli in eliciting fusion movements. *Arch Ophthal, 73, 115-121*

164. Lyle TK, Bridgeman GJO (1959) *Worth and Chavasse's Squint. The binocular reflexes and the treatment of strabismus*. 9th Ed, Ballière, Tindall and Cox, London.

165. Lyle TK, Wybar KC (1967) *Practical Orthoptics in the Treatment of Squint.* Lewis and Co, London. p. 194

166. McCormack G, Fisher SK, Wolf K (1991) Retinal eccentricity of fusion detail affects vergence adaptation. *Optom Vis Sci, 68, 711-718*

167. McCormack G, Fisher SK (1996) The source of disparity vergence innervation determines prism adaptation. *Ophthal Physiol Opt, 16, 73-82*

168. Maddox E E, (1893) *The Clinical Use of Prisms*. John Wright & Sons, Bristol

169. Maddox EE (1907) *Tests and studies of ocular muscles. 3rd Ed, pp 220-228*

170. Mahto RS (1972) Eye strain from convergence insufficiency. *Brit Med J, 2, 564-565*

171. Mallett RFJ (1964) The investigation of heterophoria at near and a new fixation disparity technique. *Optician, 148, 547-551 & 574-581*

172. Mallett RJ (1966) The investigation of ocular motor imbalance *Ophthal Opt, 6, 586 - 596 and 654 - 657*

173. Mallett RFJ (1974) Fixation disparity - its genesis in relation to asthenopia *Ophthal Opt, 14, 1159-1168*

174. Mallett RFJ (1983) A new fixation disparity test and its applications. *Optician, 186, 11-15*

175. Mallett RFJ (1988) Techniques of investigation of binocular vision anomalies. In *Optometry*, Eds. Edwards K and Llewellyn R, Butterworths, London. p 238-269

176. Manny RE, Fern KD (1997) Binocular Function. In *The Ocular Examination. Measurements and Findings.* Ed. Zadnik K, WB Saunders, Philadelphia pp157-159

177. Manson N (1962) Anaemia as an aetiological factor in convergence insufficiency. *Brit J Ophthalmol, 46, 674-677*

178. Mantyjarvi MI (1981) 'The Amblyopic Schoolgirl Syndrome'. *J Paed Ophthalmol Strabismus, 18, 30-33*

179. Margrain T (1996) *Personal communication.* City University.

180. Marlow FW (1924) *The Relative Position of Rest of the Eyes and the Prolonged Occlusion Test.* FA Davis, Philadelphia, USA

181. Marr D, Poggio T (1979) A computational theory of human stereo vision. *Proc R Soc Lond Ser B, 204, 301-328*

182. Martens TG, Ogle KN (1959) Observations on accommodative convergence, *Am J Ophthalmol, 47, 455-462*

183. Masters RL (1964) The effects of alcohol and hypoxia on the heterophorias *Aeromed Rev, 2-64*

184. Matin E, Clymer AB, Matin L (1972) Metacontrast and saccadic suppression. *Science, 178, 179-182*

185. Mayfrank L, Mobashery M, Kimmig H, Fischer B (1986) The role of fixation and visual attention in the occurrence of express saccades in man. *Eur Arch Psychiatr Neurol Sci, 235, 269-275*

186. Mays LE, Porter JD, Gamlin PDR, Tello CA (1986) Neural control of vergence eye movements: neurons controlling vergence velocity. *J Neurophysiol, 56, 1007-1021*

187. Meyers MP (1951) The position of the eyes under general anaesthesia. *Am J Ophthalmol, 34,1749-1752*

6.1 Symptom scores and recovery time

6.1.1 Average of right and left eye recovery times (recovery time^{RL})

To investigate the relationship between symptoms and recovery time the average of right and left recovery times for the combined groups were analysed. Data was excluded for those subjects for whom data for one eye was missing. The average recovery time^{RL} was 0.93 s (n = 44; SD 0.57 s). The range was from 0.32 s to 3.21 s. Figure 6.2 illustrates the frequency distribution of recovery time^{RL} with two outlying data omitted (3.21 s and 3.06 s). It should also be noted one subject who failed to make recovery movement with one eye was also omitted for all the following recovery time analyses.

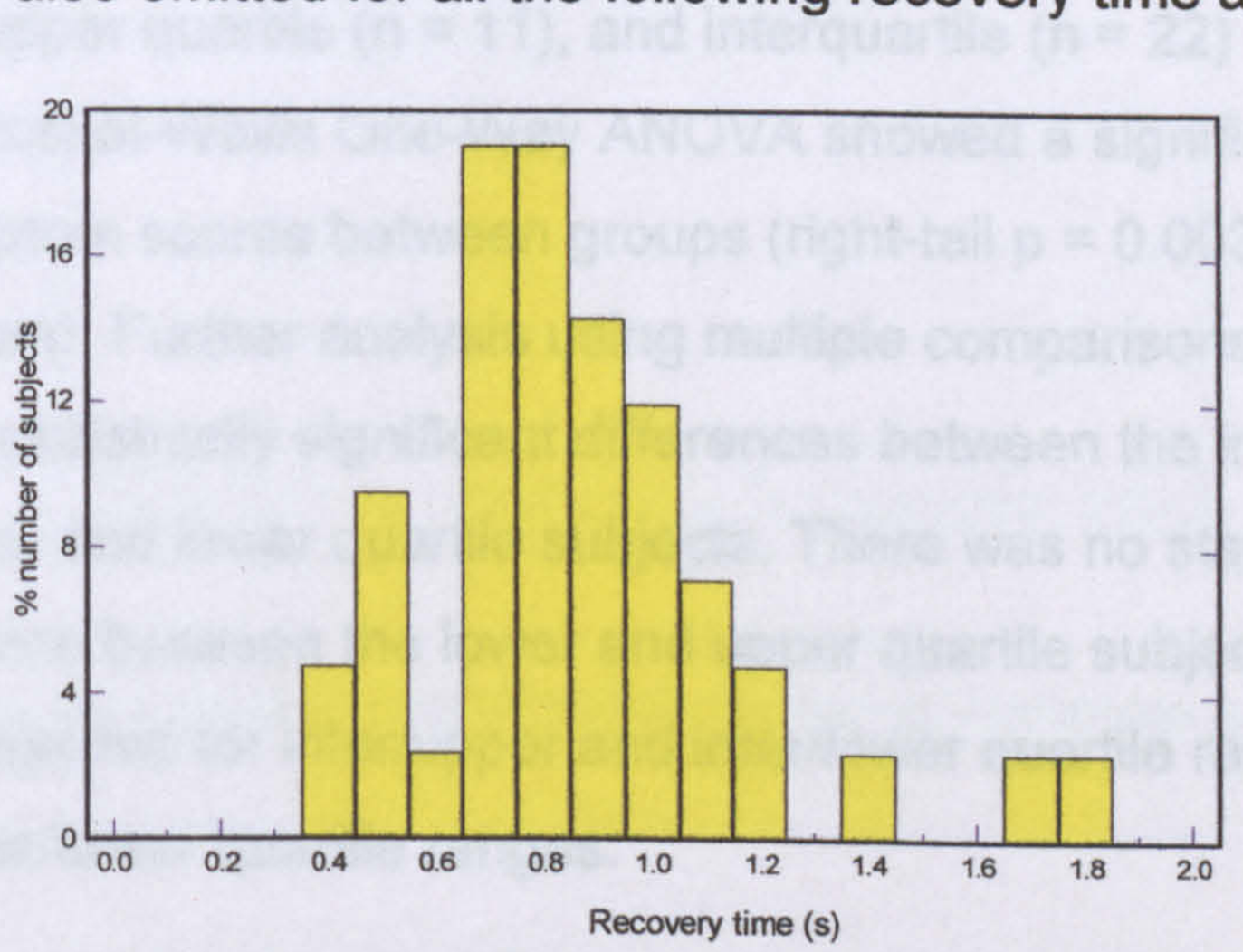


Figure 6.2. Frequency distribution of recovery time^{RL} of the combined groups (two outliers omitted) (n= 44).

There was no linear relationship between recovery time^{RL} and symptom score (Pearson R^2 = < 1%). Figure 6.3 shows symptom scores plotted against recovery times^{RL}.

203. Ogle KN, Prangen AdeH, (1953) Observations on vertical divergences and heterophorias. *Arch Ophthalmol, 49, 313-334*

204. Ogle KN, Mussey F, Prangen A (1949) Fixation disparity and the fusional processes in binocular single vision. *Am J Ophthalmol, 32, 1069-87*

205. Ono H, Nakamizo S (1978) Changing fixation in the transverse plane at eye level and Hering's law of equal innervation. *Vision Res, 18, 511-519*

206. Ono H, Nakamizo S, Steinbach MJ (1978) Nonadditivity of vergence and saccadic eye movement. *Vision Res, 18, 735-739*

207. Oohira A (1993) Vergence eye movements facilitated by saccades. *Jpn J Ophthalmol, 37, 400-413*

208. O'Shea WF, Ciuffreda KJ, Fisher SK, Tannen B, Super P (1988) Relationship between distance heterophoria and tonic vergence. *Am J Optom Physiol Opt, 65, 787-793*

209. Owens DA, Leibowitz HW (1980) Accommodation, convergence, and distance perception in low illumination. *Am J Optom Physiol Opt, 57, 9, 540-550*

210. Owens DA, Wolf-Kelly K (1987) Near work, visual fatigue, and variations of oculomotor tonus. *Invest Ophthalmol Vis Sci, 28, 743-749*

211. Pantano FM (1982) Orthoptic treatment of convergence insufficiency: two year follow-up report. *Am Orthopt J, 32, 73-80*

212. Passmore JW, MacLean F (1957) Convergence insufficiency and its management: an evaluation of 100 patients receiving a course of orthoptics. *Am J Ophthalmol, 448-456*

213. Peli E, McCormack G (1983) Dynamics of cover test eye movements *Am J Optom & Physiol Opt, 60, 712-714*

214. Percival A (1928) The Prescribing of Spectacles, Bristol, England, J. Wright & Sons

215. Pickwell LD (1972) Hering's law of equal innervation and the position of the binoculus. *Vis Res, 12, 1499-1507*

216. Pickwell LD (1973) Eye movements during the cover test. *Br J Physiol Opt, 28, 23-25*

217. Pickwell LD, Hampshire R (1981) The significance of inadequate convergence. *Ophthal Physiol Opt, 1, 13-18*

218. Pickwell LD, Jenkins TCA, Yekta AA (1987) Fixation Disparity in Binocular Stress *Ophthal Physiol Opt, 7, 1, 37-41*

219. Pickwell LD, Stephens LC (1975) Inadequate convergence. *B J Physiol Opt, 30, 34-37*

220. Pigassou-Albouy R, Jones ST (1978) The Cover Test. In Reinecke RD ed. *Strabismus. Proceedings of the Third Meeting of the International Strabismological Association.* May 10-12, Kyoto, Japan. pp 61-65

221. Porcar E, Martinez-Palomera (1997) Prevalence of general binocular dysfunctions in a population of university students. *Optom Vis Sci, 74, 111-113*

222. Powell WH Jr (1938) Ocular manifestations of alcohol and considerations of individual variations in 7 cases investigated. *J Aviat Med, 9, 97-103*

223. Purcell LR, Nuffer JS, Clements SD, Clausen LR, Schuman DO, Yolton RL (1983) The cost effectiveness of selected optometric procedures. *J Am Optom Assoc, 54, 643-647*

224. Rainey B (1997) Personal communication. Academy '97, San Antonio, Texas (American Academy of Optometry annual meeting)

225. Rainey B, Schroeder T, Goss D, Grosvenor T (1997) Inter-examiner reliability of heterophoria tests, *Optom Vis Sci, 74, 12s, 69*

226. Rainey BB, Schroeder TL, Goss DA, Grosvenor TP (1998) Inter-examiner reliability of heterophoria tests, *Optom Vis Sci, 75,10,719-726*

227. Rashbass C, Westheimer G (1961) Disjunctive eye movements. *J Physiol, 159, 2, 339-360*

228. Ravault A, Bongrand M, Bonamour G (1968) The utilization of prisms in the treatment of divergent strabismus. In *Proceedings of the 1st International Congress of Orthoptics.* CV Mosby, St Louis

229. Reading R (1988) Near point testing. In: Eds. Edwards K & Llewellyn R, *Optometry,* Chapter 10, Near point testing, 150-151

230. Reulen JPH (1984) Latency of visually evoked saccadic eye movements: temporal properties of the facilitation mechanism. *Biol Cybern, 50, 263-271*

231. Riggs LA, Niehl EW (1960) Eye movements recorded during convergence and divergence. *J Opt Soc Am, 50(a), 913-920*

232. Robinson DA (1963) A method of measuring eye movements using a search coil in a magnetic field. *IEEE Trans Bio-Med Electron, 10, 137-145*

233. Romano PE, Noorden GK von (1971) Limitations of cover test in detecting strabismus. *Am J Ophthalmol, 77, 10-12*

234. Rosenfield M (1997a) Tonic vergence and vergence adaptation, *Optom Vis Sci, 74, 303-328*

235. Rosenfield M (1997b) Prism adaptation: relevance in clinical practice. *J Optom Vision Develop, 28, 68-76*

236. Rosenfield M, Ciuffreda KJ (1990) Distance heterophoria and tonic vergence. *Optom Vis Sci, 67, 667-669*

237. Rosenfield M, Ciuffreda KJ, Ong E, Super S (1995a) Vergence adaptation and the order of clinical vergence range testing. *Optom Vis Sci, 72, 219-223*

238. Rosenfield M, Chun TW, Fischer SE (1997) Effect of prolonged dissociation on the subjective measurement of near heterophoria. *Ophthal Physiol Opt, 17, 6, 478-482*

239. Rosner J, Rosner J (1990) *Pediatric Optometry.* Butterworths, Boston

240. Ross LE, Ross SM (1980) Saccade latency and warning signals: Stimulus onset, offset, and change of warning events. *Perception & Psychophysics, 27, 3, 251-257*

241. Ross LE, Ross SM (1981) Saccade latency and warning signals: Effects of auditory and visual stimulus onset and offset. *Perception & Psychophysics, 29, 5, 429-437*

242. Rouse MW, Hyman L, Hussein M, Solan H (1998) Frequency of convergence insufficiency in optometry clinic settings. *Optom Vis Sci, 75, 88-96*

243. Ruskell G (1998) MSc course notes. City University, London

244. Sanfillipo S, Clahane AC (1970) The effectiveness of orthoptics alone in selected cases of exodeviation: the immediate results and several years later. *Am Orthopt J, 20, 104-117*

245. Saslow MG (1967) Effects of components of displacement-step stimuli upon latency for saccadic eye movement. *J. Opt. Soc. Am, 57, 1024 - 1029*

246. Scheiman MM, Peli E, Libassi D (1983) Auditory biofeedback used to enhance convergence insufficiency therapy. *J Am Optom Assoc, 54, 1001-1003*

247. Scheiman M, Gallaway M, Coulter R, Reinstein F, Ciner E, Herzberg C, Parisi M (1996) Prevalence of vision and ocular conditions in a clinical paediatric population. *J Am Optom Assoc, 67, 193-202*

248. Schiller PH, Sandell JH, Maunsell JH (1987) The effect of frontal eye field and superior colliculus lesions on saccadic latencies in the rhesus monkey. *J Neurophysiol, 57, 1033-1049*

249. Schoessler JP (1980) Disparity-induced vergence responses in normal and strabismic subjects. *Am J Optom Physiol Opt, 57, 666-675*

250. Schor CM (1979a) The influence of rapid prism adaptation upon fixation disparity *Vision Res, 19, 757-765*

251. Schor CM (1979b) The relationship between fusional vergence eye movements and fixation disparity. *Vision Res, 19, 1359-1367*

252. Schor CM (1980) Fixation disparity: A steady state error of disparity-induced vergence. *Am J Optom Physiol Opt, 57, 618-631*

253. Schor CM (1983) Fixation disparity and vergence adaptation. In *Vergence Eye Movements: Basic and Clinical Aspects.* Chapter 14. Eds. Schor CM &Ciuffreda KJ, Butterworths, Boston

254. Schor CM (1986) The Glenn Fry Award Lecture: Adaptive regulation of accommodative vergence and vergence accommodation. *Am J Optom Physiol Opt, 63, 587-609*

255. Schor CM, Ciuffreda KJ (1983) *Vergence Eye Movements: Basic and Clinical Aspects.* Butterworths, Boston

256. Schroeder TL, Rainey BB, Goss DA, Grosvenor TP (1996) Reliability of and comparisons among methods or measuring dissociated phoria. *Optom Vis Sci, 1996, 73, 6, 389-397*

257. Semmlow JL, Hung GK (1980) Binocular interactions of vergence components. *Am J Optom Physiol Opt, 57,9, 559-565*

258. Semmlow JL, Hung GK, Ciuffreda KJ (1986) Quantitative assessment of disparity vergence components. *Invest Ophthalmol Vis Sci, 27, 558-564*

259. Semmlow JL, Hung GK Hornig J-L., Ciuffreda KJ (1993) Initial control component in disparity vergence eye movements. *Ophthal. Physiol. Opt., 13, 1,48-55*

260. Semmlow JL, Venkiteswaran N (1976) Dynamic accommodative vergence components in binocular vision. *Vision Res, 16, 403-410*

261. Semmlow J, Wetzel P (1980) Dynamic contributions of the components of binocular vergence. *J Opt Soc Am, 69, 639-645*

262. Semmlow JL, Yuan W, Alvarez TL (1998) Evidence for separate control of slow version and vergence eye movements: Support for Hering's law. *Vis Res, 38, 1145-1152*

263. Sethi B (1986) Vergence adaptation: a review. *Doc Ophthalmol, 63, 247-263*

264. Sethi B, Henson DB (1984) Adaptive changes with prolonged effect of comitance and incomitant vergence disparities. *Am J Optom Physiol Opt, 61, 506-512*

265. Sethi B, North RV (1987) Vergence adaptive changes with varying magnitudes of prism-induced disparities and fusional amplitudes. *Am J Optom Physiol Opt, 64, 4 263-268*

266. Sheard C (1957) *The Sheard Volume; Selected Writings in Visual Ophthalmic Optics*, Philadelphia, Chilton Co

267. Sheedy JE, Saladin JJ (1975) Exophoria at near in presbyopia. *Am J Optom Physiol Opt, 52, 474-481*

268. Sheedy JE, Saladin JJ (1977) Phoria, vergence, and fixation disparity in oculomotor problems *Am J Optom Physiol Opt, 54, 474-478*

269. Sheedy JE, Saladin JJ (1978) Association of symptoms with measures of oculomotor deficiencies. *Am J Optom Physiol Opt, 55, 10, 670-676*

270. Sheni DD, Remole A (1982) A new method of measuring vergence limits: the rotatory grid method. *Am J Optom Physiol Opt, 59, 240-248*

271. Snydacker D, (1962) The Maddox rod test: a 10-year follow. *Trans Ophthalmol Soc Am, 60, 292-303*

272. Soderberg DC (1968) An evaluation in the use of the Maddox rod. *J Am Optom Assoc, 39, 472-478*

273. Solomons H (1978) *Binocular Vision: a programmed text*. p.264, Heinemann, London

274. Somers WW, Happel AW, Phillips JD (1984) Use of a personal microcomputer for orthoptic therapy. *J Am Optom Assoc, 55, 262-267*

275. Stark L, Kenyon RV, Krishnan VV, Ciuffreda KJ (1980) Disparity Vergence: A proposed name for a dominant component of binocular vergence eye movements. *Am J Optom Physiol Opt, 57,9, 606-609*

276. Steinman RM (1965) Effect of target size, luminance, and color on monocular fixation. *J Opt Soc Am, 55, 1158 - 1165*

277. Stella SS (1968) The association of blinks and refusion in intermittent exotropia. *Am J Optom Arch Am Acad Optom, 45, 465-471*

278. Stern A (1953) The effect of target variation and kinesthesia upon near heterophoria measurements, *Am J Optom Arch Am Acad Optom, 30, 351-365*

279. Stevens GT (1886) A system of terms relating to the conditions of the ocular muscles known as "insufficiencies." *N Y Med J, 44, 624*

280. Stidwill (1990) *Orthoptic Assessment and Management.* Blackwell Scientific Publications, London

281. Stutterheim NA (1934) The divergence of the primary position of the eyes. *Br J Ophthalmol, 18, 256-60*

282. Sullivan MJ, Kertesz AE (1979) Peripheral stimulation and human cyclofusional response. *Invest Ophthalmol Vis Sci, 18, 1287-1291*

283. Tait EF (1951) Accommodative convergence. *Am J Ophthalmol, 34, 1093-1107*

284. Tam WJ, Ono H (1994) Fixation disengagement and eye-movement latency. *Perception & Psychophysics, 56, 251-260*

285. Tam WJ, Stelmach LB (1993) Viewing behaviour: Ocular and attentional disengagement. *Perception & Psychophysics, 54, 211-222*

286. Taylor D (1990) Acquired eye movement disorders. In *Pediatric Ophthalmology*, Blackwell Science, Oxford. p. 608

287. Teitelbaum BA, Micca PJ, Jones GD, Thurn M, Lee T (1985) Differentiation of Asymptomatic Patients from Symptomatic Patients by the Slope of the Forced Vergence Fixation Disparity Curve. *Am J Optom Physiol Opt, 62,4, 282-286*

288. Toates FM (1974) Vergence eye movements. *Doc Ophthalmol, 37, 153-214*

289. Troost BT, Dell'Osso LF (1979) Fast eye movements (saccades): basic science and clinical correlations. In Thomson HS et al, Eds. *Topics in Neuro-ophthalmology,* Williams & Wilkins, Baltimore, p 246

290. Ukwade MT, Bedell HE (1993) Stability of oculomotor fixation as a function of target contrast and blur. *Optom Vis Sci, 70, 123-126*

291. Vaegan (1979) Convergence and divergence show large and sustained improvement after short isometric exercises. *Am J Optom Physiol Opt, 56, 23-33*

292. van Haeringen R, McClug P, Cameron KD (1986) Comparison of Wesson and modified Sheedy fixation disparity tests. Do fixation disparity measures relate to normal binocular status ? *Ophthal Physiol Opt, 6, 397-400.*

293. von Graefe A (1855) Über myopia in distans nebst betrachtungen über sehen jenseits der grenzen unserer accommodation. *von Graefe's Arch Ophthalmol, 2, 158*

294. von Noorden GK (1990) *Binocular Vision and Ocular Motility*. CV Mosby, St Louis

295. Walraven J (1975) Amblyopia screening with random-dot stereograms. *Am J Ophthalmol, 80, 893-900*

296. Warwick R (1953) Representation of the extra-ocular muscles in the oculomotor nuclei of the monkey. *J Comp Neurol, 98, 449-503*

297. Weale R A (1963) The Ageing Eye, HK Lewis, London

298. Westheimer G (1954) Mechanism of saccadic eye movements. *Arch Ophthalmol, 52, 710-724*

299. Westheimer G, Mitchell AM (1956) Eye movement responses to convergence stimuli. *Arch Ophthalmol, 55, 848-856*

300. Weymouth FW (1963) An experimental comparison of three common methods of measuring heterophoria. *Am J Optom Arch Am Acad Optom, 40, 497-503*

301. White JW, Brown HW (1939) Occurrence of vertical anomalies associated with convergent and divergent anomalies - a clinical study. *Arch Ophthalmol, 21, 999-1009*

302. Wick B (1977) Vision training for presbyopic nonstrabismic patients. *Am J Optom Physiol Opt, 54, 244-247*

303. Wildersoet CF, Cameron KD (1985) The effect of illumination and foveal fusion lock on clinical fixation disparity measurements with the Sheedy disparometer. *Ophthal Physiol Opt, 5, 171-178*

304. Winn B, Gilmartin B, Sculfor DL, Bamford JC (1994) Vergence adaptation and senescence. *Optom Vis Sci, 71, 797-800*

305. Wolf E, (1968) *Anatomy of the Eye and Orbit*. 6th Edition Revised by Last RJ, HK Lewis & Co, London

306. Worrell BE, Hirsch MJ, Morgan MW (1971) An evaluation of prism prescribed by Sheard's criterion *Am J Optom Arch Am Acad Optom, 48, 5, 373-376*

307. Yarbus AL (1957) Eye movements during change in the stationary points of fixation. *Biophysics, 2, 679-683*

308. Yekta AA, Jenkins T, Pickwell D (1987) The clinical assessment of binocular vision before and after a working day *Ophthal Physiol Opt, 7, 4, 349-352*

309. Yekta AA, Pickwell LD, Jenkins TCA (1989) Binocular vision, age and symptoms. *Ophthal Physiol Opt, 9, 115-20*

310. Young LR (1981)The sampled-data model and foveal dead zone for saccades. In Zuber BL, Ed, *Models of oculomotor behaviour and control.* Boca Raton, Fla, CRC

311. Young LR, Stark L (1963) Variable feedback experiments testing a sampled data model for eye tracking movements. *IEEE Trans Hum Factor Electron HFE- 4, 1, 38-51*

312. Zee DS, Optician LM, Cook JD, Robinson DA, King Engel, W (1976) Slow saccades in spinocerebellar degeneration. *Arch Neurol, 33, 243-251*

313. Zee DS, Levi L (1989) Neurological aspects of vergence eye movements. *Rev Neurol (Paris) , 145, 613-620*

314. Zhang Y, Gamlin PDR, Mays LE (1991) Antidromic identification of midbrain near response cells projecting to the oculomotor nucleus. *Exp Br Res, 84, 525-528*

315. Zuber B, Stark L (1966) Saccadic suppression: elevation by visual threshold associated with saccadic eye movements. *Exp Neurol, 16, 65-79*

316. Zuber BL, Stark L (1968) Dynamic characteristics of the fusional vergence eye- movement system. *IEEE Trans, Sys Sci Cybern, SSC-4, 1, 72-79*

Appendix 1

CITY UNIVERSITY

DEPARTMENT OF OPTOMETRY & VISUAL SCIENCE

recruit.doc

INVESTIGATION INTO EYE MOVEMENT CHARACTERISTICS DURING THE COVER TEST

Thank you for reading this information sheet. A study is being carried out to analyse the exact nature of the eye movements made during the cover test. Your assistance would be greatly appreciated.

Subjects are required who meet the following criteria:

- No symptoms during close-work (e.g. reading)
- Aged between 18 and 35 years
- Not wearing spectacles or contact lenses
- No history of eye disease
- No history of binocular vision anomalies or orthoptic treatment

If you think you fulfil the above requirements and agree to participate in this research project you will undergo a refraction and short oculomotor assessment to make sure you are indeed suitable. If you are included in the study a further appointment will be made to record your eye movements during an automated cover test. This procedure will take about an hour.

Please fill in your details below. Mr Barnard will contact you via the student pigeon hole with an initial appointment,

Full name.....

Year and group.....

Date of Birth.....

Term address.....

Holiday address.....

Tel. Term.....Holiday.....

Today's date.....

Please return this questionnaire to Mr Barnard (0504) or via staff pigeon hole on Level 2. Thank you for your co-operation.

NAS Barnard

Lecturer

APPENDIX 2

CITY UNIVERSITY

DEPARTMENT OF OPTOMETRY & VISUAL SCIENCE

INVESTIGATION INTO EYE MOVEMENT CHARACTERISTICS DURING THE COVER TEST

QUESTIONNAIRE

Thank you for agreeing to participate in this research project which is being conducted by staff from City University and Moorfields Eye Hospital. It is hoped that this study will provide more understanding about the effect that orthoptic treatment (eye muscle exercises) has on the eye muscles.

Patient Name

Mr/Mrs/Ms/Miss.....

Date of Birth.....Address .....

Tel.....

Today's date……

.PLEASE TICK ONE BOX FOR EACH QUESTION

QUESTIONS

Question 1 *How long can you do "closework (i.e. reading, writing, sewing etc.) before you note discomfort (e.g. headaches, eye ache, burning, stinging, watering, blurring, double vision, loss of concentration or tiredness) ?*

Answer	(1)	up to 15 minutes	☐
	(2)	up to 30 minutes	☐
	(3)	up to 1 hour	☐
	(4)	up to 2 hours	☐
	(5)	at least 3 hours	☐

Question 2 *How often do you get headaches when you do near work ?*

Answer	(1)	every time (100% of the time	☐
	(2)	very often (about 75% of the time)	☐
	(3)	often (about 50% of the time)	☐
	(4)	occasionally (about 25% of the time)	☐

(5) never (0% of the time) ☐

Question 3 *If you experience headaches during closework, how bothersome are these headaches (i.e. the degree to which they interfere with your normal functioning) ?*

Answer
(1) extremely bothersome ☐
(2) very bothersome ☐
(3) moderately bothersome ☐
(4) mildly bothersome ☐
(5) minimally bothersome or not at all ☐

Question 4 *Do your eyes pull, ache, or water when you do closework ?*

Answer
(1) every time I read or sew (etc.) (100% of the time) ☐
(2) very often (about 75% of the time) ☐
(3) often (about 50% of the time) ☐
(4) occasionally (about 25% of the time) ☐
(5) never (0% of the time) ☐

Question 5 *Does the reading material ever become blurry run together, or jump when you do closework ?*

Answer
(1) every time I read (100% of the time) ☐
(2) very often (about 75% of the time) ☐
(3) often (about 50% of the time) ☐
(4) occasionally (about 25% of the time) ☐
(5) never (0% of the time) ☐

Question 6 *Does the reading material ever become double when you do closework ?*

Answer
(1) every time I read (100% of the time) ☐
(2) very often (about 75% of the time) ☐
(3) often (about 50% of the time) ☐
(4) occasionally (about 25% of the time) ☐
(5) never (0% of the time) ☐

Question 7 *Do your eyes feel 'tired' and/or do you lose your concentration when you do near work ?*

Answer

(1)	every time I read (100% of the time)	☐
(2)	very often (about 75% of the time)	☐
(3)	often (about 50% of the time)	☐
(4)	occasionally (about 25% of the time)	☐
(5)	never (0% of the time)	☐

Question 8 *Please briefly describe any other problems you have when you do near work*