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ORIGINAL COMMUNICATION

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Squints and diplopia seen after brain damage

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Abstract The aim of this study was to investigate the incidence of squint after brain damage. We performed an observational study on 239 consecutive patients admitted to a specialist neurological rehabilitation unit: 129 with stroke, 84 with head injury and 26 with other conditions. Standard orthoptic measures, including visual acuity, cover test, eye movement recording and tests of binocular function were performed. Of all the patients, 89 (37%), were found to have squints, but only 32 of these (36%) experienced double vision. Brain stem lesions causing peripheral ocular motor impairment were found in a high proportion of patients after head injury (56%). Squints were found in 27 of 95 (28%) patients with cortical strokes, many with no other signs of brain stem involvement. Left was just as likely as right hemisphere damage to be associated with squint, but right-sided lesions seemed to protect against diplopia. We conclude that squint is common after brain damage, even if the brain stem is not obviously affected, but only a minority of these patients with acquired squint suffer diplopia.

Key words Brain damage · Diplopia · Squint · Hemisphere stroke

Introduction

A wide range of ocular motor impairments may follow neurological damage and clinicians often use these to make anatomical or pathological diagnoses [11]. However, little is known about the frequency or nature of such problems in patients with brain damage or about their implications for rehabilitation. Therefore we have been studying the visual status of a large number of adults admitted to a specialized neurological rehabilitation centre. Most patients had had a stroke or a head injury.

By studying every patient who was admitted we were able to assess the nature and frequency of visual problems in these neurological rehabilitation patients. Here we discuss the squints which occur after brain injury. We wanted to establish how many patients had squints (manifest deviation of the eyes) and to explore the nature and extent of their fusional difficulties, in particular how many suffered from diplopia. We were also interested in how the effects of right and left hemisphere lesions differed.

Patients and methods

All patients admitted over 3 years to the Rivermead Rehabilitation Centre, Oxford were assessed orthoptically, whether or not they complained of any visual symptoms. Most patients were seen within 2 weeks of admission (which was always more than 2 months after onset of disease) and some were reassessed as indicated.

A history of previous eye problems and current visual function was taken. Because these patients had a wide range of neurological impairments such as ataxia, weakness, sensory loss, amnesia and aphasia, their orthoptic investigation was necessarily adapted to take account of any other problems [5].

Visual acuity

Visual acuity was assessed using a Snellen chart at 6 m with glasses as necessary [13]. If the patient was unable to name the let-

ter, then tracing the letter shape or pointing to its match were acceptable responses.

Visual fields

Visual fields were assessed in all patients by confrontation testing [2]. This was often the only means available because of problems of attention, visual neglect or communication complicating the assessment. Such problems are easier to monitor in confrontation testing. Visuospatial neglect was assessed using the star cancellation test [9] given as a clinical routine.

Pupillary reactions

Pupillary reactions were elicited with a pen torch to check the brain stem visual and autonomic reflexes. Abnormalities of eyelid movement were also recorded.

Eye movements

Eye movements were assessed by observing: (1) movements to the nine cardinal positions of gaze; any limitation was recorded, and the nature of any gaze palsies noted; (2) pursuit, slow eye movements following a moving target; (3) saccadic, fast, refixation eye movements from one stationary target to another; (4) any other abnormalities, such as nystagmus (rhythmic oscillation of the eye which may be jerky, pendular or gaze evoked), oculomotor ataxia (oscillations of the eyes which often result from cerebellar lesions) or other problems with fixation [11]. Where possible the eye movements were also recorded using an infra-red recording technique [8]. Fixation stability, pursuit movements, saccades and vergence in free space were assessed in this way.

Convergence

Convergence was measured using the RAF rule [13] and also by recording the prism strength required to correct any manifest deviation.

Accommodation

Near visual acuity was assessed using the RAF rule [13] by recording the smallest print that could be seen clearly at the nearest convergence distance. Where accommodation was reduced, attempts were made to improve this using convex lenses.

The Cover Test

This test was carried out to detect the presence of a manifest squint (apparent when both eyes are open) or a latent deviation which is only seen when the two eyes are dissociated. (In latent squint the patient can still use the eyes together for binocular vision.) The direction of any deviation of the eyes for near and distance fixation was recorded, noting any alternation of fixation.

Sensory fusion

Sensory fusion (i.e. the ability to fuse images centrally) was assessed with the aid of prisms to correct the deviation [14]. The patient viewed a small light through glasses with one red and one green lens. Sensory fusion was present if the patient could fuse the two images with prisms and this was maintained over 2 dioptres of vergence. In some cases, the two images just "float" over each other and never fuse together; this is known as superimposition and indicates loss of fusion capacity. If fusion could be maintained over a larger range of vergence, then the patient was also said to have *motor fusion*. This is the ability to maintain fusion of the images of both eyes during large convergent eye movements.

In many patients the eyes were divergent for near fixation (within 1 m). Although sensory fusion could be shown beyond this distance, no convergence could be demonstrated for near targets, a condition known as "convergence paralysis" [7]. These cases were recorded as having loss of motor fusion only.

Diplopia

The patients were asked whether they experienced double vision (diplopia). This was established non-verbally in patients with aphasia, either by using their fingers or drawing. Diplopia was reported more often by left hemisphere patients, suggesting that we had probably been able to communicate with them successfully.

Classification of acquired squints

In order to use both eyes together to see a single image, both eyes must be able to point to the same point in space. Thus, damage to ocular motor neurons or their axons or to higher eye movement control centres can all cause squint. The images seen by the two eyes must be of roughly similar clarity, size, shape and stability. Therefore reduced acuity, and upset of the mechanisms which control fixation, can also cause squint. Finally central binocular fusional mechanisms must have developed in early childhood, if the patients are to use their eyes together to give a single image. Thus squint may arise for a number of reasons, and patients were therefore classified according to the following criteria, applied in the order given.

1. Had the patient had a squint since childhood?
2. Did the patient have visual acuity below 6/60 in one eye?
3. Was there any evidence of ocular palsy? Head injuries can affect all three ocular motor nerves. The sixth is said to be the most vulnerable because of its long course over the apex of the petrous temporal bone, where it can be involved in fractures at the base of the skull. However, even slight head injury can damage the fourth nerve, and severe head injuries often damage it on both sides as it exits from the dorsal surface of the brain stem and sweeps around the midbrain. Severe head injuries are also a common cause of third nerve palsies, and these lead to double vision, ptosis and blurred vision. Thus all three ocular motor nerves were affected in our patients, and in every combination. Detailed MRI studies would have been required to determine whether their palsies were the result of brain stem injury rather than damage to the ocular motor nerves themselves. Since this was not the focus of our study, we did not pursue this issue further.
4. Was there evidence of skew deviation? In these bizarre squints, which are usually vertical and torsional, but can involve any combination of directions, the movement of the ocular muscles is intact; so they should not be confused with nerve palsies, where the movement of the eyes is restricted. The cause is thought to be prenuclear dysfunction of vestibular circuitry in the medulla [3, 11].
5. Was there Parinaud's dorsal midbrain syndrome? These patients show bilateral loss of elevation and sometimes depression as well. They suffer convergence retraction nystagmus when trying to look up, which causes giddiness and nausea. In addition they often have reduction or even paralysis of convergence, and reduced accomodation, due to involvement of visual, vestibular and ocular motor pathways in the midbrain [1].

6. Was the squint convergent or divergent?
7. Applying these criteria left a final miscellaneous group of eight patients with extensive impairments causing combinations of the above types of squint.

Results

A total of 239 consecutively admitted patients were seen. Damage was localised on clinical grounds supplemented by CT or MR images where indicated. Of the patients, 129 (54%) had had strokes: 44 of these had left and 62 had right hemisphere damage; 16 had bilateral hemispheric and 7 had brain stem lesions. Eighty-four (35%) patients had had head injuries; 26 (11%) had a miscellany of neurological diagnoses, such as encephalitis, brain tumours or cerebral anoxia. The head injuries and miscellaneous pathologies had often led to bilateral hemisphere and brain stem damage.

Squints were found in a surprisingly large number (89) of these patients (37%) using the Cover Test: in 37 (56%) of patients with head injury, usually as a result of ocular palsy; 8 (31%) of those with anoxia, encephalitis etc.; and in 34 (26%) of those with strokes. However, of the latter only 7, all of whom had squints, had other signs of brain stem involvement. Unexpectedly, squints were equally common after left- (25%) and right-sided (23%) hemisphere lesions.

Probably the reason why such a high incidence of squint has not been previously reported in neurological rehabilitation patients is that a surprisingly large proportion of the patients with squints (57–64%) failed to complain of diplopia. Another unexpected finding for our squint sample was that far more patients with left- than right-sided lesions complained of diplopia. Only 1 of 14 (7%) of those with right-sided lesions experienced double vision, whereas 10 of 11 (91%) patients with left hemisphere damage did so, a highly significant difference (chi-square = 17.5, $P < 0.0001$).

Of the 13 right hemisphere patients with squint who were not troubled by diplopia, 11 had left neglect, but 2 did not; nor did the single right hemisphere patient who did complain of double vision. The majority of those with left hemisphere lesions with squint who complained of diplopia (6/10) had, however, right neglect. So whereas left neglect may have helped to protect most from diplopia, right neglect did not.

Table 1 shows the numbers of patients who had each of the types of squint described earlier.

Childhood squint ($n = 12$)

Onset of squint from birth or early childhood results in diplopia, which is rapidly suppressed; and this leads to amblyopia and sometimes abnormal compensatory changes in projection and fusion. Squint acquired after this time usually leads to diplopia which cannot be suppressed or ignored. About 5% of all the patients had had childhood squints; this is similar to the 4% of the normal population found to squint [6]. All our patients with squint since childhood showed suppression or abnormal peripheral fusion without diplopia, as would be expected. It has been suggested that acquired brain damage can sometimes reverse long-standing suppression and thus cause diplopia [4], but we found no instance of this.

Reduced acuity ($n = 6$)

Reduced vision in one eye may cause divergence of that eye due to unbalanced binocular input. Three of our patients had cataracts which had had this effect and clearly were not related to their stroke. In the remaining patients the loss of vision was due to head injury and was not refractive in nature; two were completely blind in the affected eye with no perception of light and one had minimal vision limited to counting fingers. These patients had worse problems if they also had neglect or field defects. The worst situation occurred when neglect and a field defect were on the same side as the eye with the only useful vision.

Table 1 Type of squint by diagnosis

	Left hemisphere	Right hemisphere	Bilateral stroke	Brain stem stroke	Head injury	Other	Total
Childhood acuity	2	5	0	0	5	0	12
Reduced acuity	1	2	0	0	3	0	6
Ocular palsy	0	0	0	4	17	1	22
Skew deviation	0	0	0	1	4	2	7
Parinaud's syndrome	0	0	0	2	4	1	7
Convergent squint	1	0	0	0	1	1	3
Divergent squint	6	5	2	0	11	0	24
Other	1	2	0	0	2	3	8
Total with squint	11	14	2	7	47	8	89
No squint	33	48	14	0	37	18	150
Total no. of patients	44	62	16	7	84	26	239

Nerve Palsies (n = 22)

Fifteen of the patients with nerve plasies had diplopia, as would be expected; but 7 did not, perhaps because additional cortical damage, particularly on the right "protected" them from being troubled by the second image. In spite of the severity of their injuries the majority of the patients (18) retained enough cortical function to preserve sensory fusion. Of these, 8 also had motor fusion, but 4 patients had unfortunately lost all central mechanisms for fusion. This was associated with extensive brain stem or cerebellar damage causing nystagmus and unstable fixation. In the fourth nerve plasies torsion was an added obstacle to fusion. Such loss of central fusion cannot be treated if it fails to recover spontaneously and only occlusion of the squinting eye will overcome the double vision.

Skew deviations (n = 7)

These patients had few visual symptoms, and only one complained of diplopia. However, their appearance is strange, and they worried about this. Four skew deviations were due to head injury, one to brain stem stroke, one to tumour and one to anoxic brain damage. The last-mentioned was the only patient who noticed diplopia for a short period of time.

Parinaud's syndrome (n = 7)

Four of these patients had had head injuries, two, brain stem strokes and one, anoxic damage. There was other evidence of involvement of the upper brain stem and basal ganglia in all the patients. All complained of diplopia and retained some fusion. They were helped by means of prisms to correct their divergence at the near point, and with convex lenses to improve clarity of their near vision.

Convergent squint (n = 3)

All these patients had evidence of left hemisphere damage with aphasia; they also had unstable fixation and inattention. The convergent squint was small – less than 10° in amplitude. The attentional problems were those of difficulty in suppressing distractions around them, rather than of hemispatial neglect. These patients had good acuity and full movements of their eyes. Possibly the failure of their binocular vision was due to instability of fixation. All had very poor stereopsis, again presumably due to the inability to keep their eyes still and attend to the target. None of these patients had diplopia.

Divergent squint (n = 24)

Divergent squint was the commonest type we found. It followed bilateral damage in 13 head injuries and strokes, but was almost equally common following lesions of just the right (5) or left (6) hemispheres. It resulted from loss of motor fusion, i.e. loss of convergence. This is most disabling because it causes loss of stereopsis close to, where it is most needed. All these patients had adequate vision, full ocular movements and no signs of internuclear ophthalmoplegia, so there was no obvious cause for their loss of convergence. In only a few (6) of the patients, all head-injury cases, were there other clinical signs to suggest that the brain stem was damaged; and in four stroke patients CT showed that the lesion involved the parietal cortex with no evidence of any brain stem damage. Nine of the patients, most with bilateral damage, had lost all sensory fusion. Only one of these complained of double vision. Even among the 15 patients who retained sensory fusion only 6, all with left-sided lesions, suffered diplopia.

Discussion

Our prospective study of squint after acquired brain damage is probably the largest to date. The patients were highly selected, as they all had severe or complex neurological disabilities requiring rehabilitation, but they were not specifically selected because of oculomotor problems. Therefore the findings should generalise to other settings, but they will need replication. We probably detected so many patients with squint but without diplopia because we tested all admitted patients, and not just those complaining of visual symptoms. Also, in some cases, the deviation was slight or intermittent and might not have been noticed. This was particularly true of divergent squints for near targets, which result in disabling loss of convergence and stereopsis.

Our results concerning childhood squint, and squints associated with grossly reduced visual acuity, nerve palsies, skew and Parinaud's syndrome are unsurprising, although of interest because they give an idea of the incidence of these conditions in a rehabilitation setting. Our more interesting findings are: (1) squint, particularly failure of convergence for near vision, is much more common in patients with brain damage than is generally recognised, probably because two-thirds of such patients do not complain of diplopia; (2) left-sided lesions are almost as likely to cause squint as right-sided ones, but left lesions are more likely to cause diplopia; (3) brain stem damage does not seem to be essential; lesions confined to the cerebral hemispheres can probably cause squint.

1. The surprising lack of diplopia in patients with squint of recent onset has not been noted before. Aphasia did not explain failure to complain because diplopia was reported more often after left- (91%) than right-sided lesions caus-

ing squint (6%). Initially we thought that their not noticing it could be entirely explained by hemispatial neglect. However, although there were no right hemisphere patients with left neglect who suffered diplopia, two right-sided cases without diplopia also did not have neglect, and diplopia was actually slightly commoner in left hemisphere patients with right neglect than in those without it. So although left neglect may partially explain the lack of diplopia in right hemisphere patients, right neglect does not seem to protect from diplopia in left hemisphere patients. We also looked at which eye was squinting as a function of side of lesion and presence or absence of diplopia. In left-sided lesions the left or right eye was equally likely to squint and both were associated with diplopia. After right hemisphere lesions alternating squints were the most common type, but diplopia was rare. So we do not really have a complete explanation why so few patients suffered diplopia.

2. We were surprised to find that squints were as common in patients with left as in right hemisphere lesions. This has not been suggested before, and the general view has arisen that visuospatial problems to which squints may contribute are usually associated only with right-sided lesions. It is clear, however, that both left and right hemispheres contribute to ocular motor control, though probably in subtly different ways.

3. The posterior parietal cortex is probably involved in the voluntary control not only of saccadic, but also of pursuit and vergence eye movements [10, 16, 17]. We believe therefore that some of the divergent squints (group 7) that we saw may have resulted from parietal damage alone, without brain stem involvement. In four such patients CT showed parietal lesions with no evidence of brain stem damage either in the scans or on clinical grounds. But small brain stem lesions may not show up in CT scans, so it is impossible to be absolutely sure that these patients did not have one. In 14 other patients with cortical damage and failure of convergence there was no other clinical evidence of brain stem damage such as pupillary involvement, ocular palsy or ophthalmoplegia. Although we did not have CT scans of them we think it likely that their squint was also due to cortical rather than brainstem damage. Such squints were equally common after left or right hemisphere lesions. Obtsuka et al. [15] have previously described such loss of convergence and defective accommodation following isolated cortical lesions. Their case had a left middle cerebral artery embolism in which CT showed low-density areas in the left parietal cortex without any evidence of brain stem involvement. The patient had convergence failure with reduced accommodation. As in our patients the pupillary light reflex and eye movements were normal, supporting the view that the brain stem was indeed unaffected.

Our study has thus raised many more questions than it has answered, but it is clear that squints, double vision and disturbed sensory and motor fusion are common in patients needing rehabilitation. The physiological explanation for these problems and their practical consequences both need further investigation.

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