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Symptoms at one year following concussion from minor head injuries

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Summary

Of 131 patients with mild concussion, 19 (14·5 per cent) still had symptoms after 1 year. Of these 19 patients, 10 had some symptoms of which they had not complained at 6 weeks. Symptoms at 1 year were more common among women and among patients who had had positive neurological findings at 24 h. Of the 19 patients who had symptoms at 1 year, 8 were involved in law suits and 6 had been suspected of malingering 6 weeks after their accident. Five of these patients were both involved in law suits and suspected of malingering.

INTRODUCTION

A WIDELY quoted study on symptoms following concussion from minor head injuries is that by Miller (1961). He suggested that the worse the head injury the fewer the symptoms. He thought that such symptoms never followed sports injuries and that the essential cause was the hope of financial gain through compensation litigation. Kelly (1972) has pointed out that Miller's cases were referred to him by insurance companies on average 15 months after the accident and suggested that it was because his patients were selected in this way that he came to his conclusions. Kelly studied cases referred to him for investigation and treatment. On average they were referred 3 months after the accident. He showed that symptoms arose in patients without litigation, that patients with sports injuries were similarly affected, that there was no inverse relationship to the severity of the accident and that the settlement of a claim did not necessarily lead to resolution of the symptoms.

Lidvall et al. (1974) followed up 85 cases of mild head injuries for a period of 90 days after the accident. Of their patients 16 per cent had less than 1 min of amnesia and 50 per cent had less than 5 min. Repeated and extensive examinations were done. They postulated a multifactorial aetiology of postconcussion symptoms, but were unable to confirm this.

In our previous study (Rutherford et al., 1977) we reported that at 6 weeks after sustaining a mild head injury, 51 per cent of patients had at least 1 symptom. We gave evidence that the causes of the symptoms were both organic and psychological. We now report on the symptoms of the same patients 1 year after injury.

PATIENTS AND METHODS

Our previous study described 145 patients who had a period of complete amnesia due to head injury, were admitted to the observation ward for 1–2 days, and were re-examined 6 weeks after injury. At 1 year, 13 of these patients were untraceable and 1 had died from causes unconnected with his head injury, leaving 131 patients who form the basis of this study. The 14 untraceable or dead patients were compared with the remaining 131 for age, sex, number of symptoms at 24 h, number with each individual symptom at 24 h, number of symptoms at 6 weeks and number with each individual symptom at 6 weeks. No significant difference ($P < 0{\cdot}05$) was

discovered between the two groups for any of these variables. The absence of significant difference could be due in part to the small numbers involved.

We wrote to all the patients. Nineteen patients reported symptoms and were asked to attend for examination and all of them attended. Another 99 reported that they had no symptoms. Of these, 22 came for a final check in hospital, while 77 did not come for any examination. Those who did not reply were written to again. If still no reply was received, the general practitioner was asked if the patient had attended for any symptoms which could have been connected with his head injury in the year following the accident. There were 13 patients in this category. At 6 weeks, 9 of them had reported no symptoms, 3 had reported 1 symptom and 1 had reported 4 symptoms. In no case did the general practitioner report that any of them attended for symptoms. We have assumed that at 1 year they were free from symptoms.

The methods of examining, recording and sorting of data were the same as those used in our previous paper (Rutherford et al., 1977). The form on which symptoms at 1 year were recorded was very similar to that used for recording symptoms at 6 weeks. We also recorded on it whether it was known that the patient was making a legal claim for compensation and whether their work capability or personal relationships had been altered by the head injury.

As the numbers were small χ^2 tests would only have been appropriate in one or two cases. We have therefore used Fisher's exact probability test throughout, giving values to three places beyond the decimal point. A level of significance of $P < 0{\cdot}05$ has been used throughout.

RESULTS

The results are set out in *Tables I–IV*. The total number of patients with symptoms was 19 (14·5 per cent of the total cases). Of these 6 had only 1 symptom, 7 had 2 symptoms and 6 had between 4 and 9 symptoms. Headache was the most common symptom followed by irritability and dizziness (*Table I*). Patients over 40 years of age seemed somewhat more likely to have symptoms, although this trend did not reach the level of significance (*Table II*). Women were more prone to symptoms than men. The apparent trend for social classes 4 and 5 to have more symptoms was not significant.

The length of retrograde or post-traumatic amnesia was not shown to affect the proportion of patients with symptoms. Headache, anosmia and diplopia at 24 h, which were associated with higher symptom rates at 6 weeks, did not affect the symptom rate at 1 year. However, the presence of positive neurological findings (other than anosmia and diplopia) at 24 h was associated with a higher symptom rate at 1 year (*Table III*).

Table I. Symptoms at 1 year following injury

Symptom	*No. of patients*	*% patients*
Headache	11	8·4
Irritability	7	5·3
Anxiety	5	3·8
Depression	2	1·5
Insomnia	3	2·3
Fatigue	3	2·3
Loss of concentration	4	3·1
Loss of memory	5	3·8
Anosmia	2	1·5
Diplopia	1	0·8
Visual defect	3	2·3
Hearing defect	2	1·5
Dizziness	6	4·6
Epilepsy	0	0
Sensitivity to alcohol	0	0
Others	1	0·8

At 6 weeks accidents caused by falls were associated with a higher symptom rate, but this was not so at 1 year (*Table IV*). At 6 weeks, patients who blamed their employer or a large impersonal body were more likely to have symptoms. At 1 year a trend in this direction was still evident, but it did not reach significance. No patient who was symptom free at 6 weeks complained of symptoms at 1 year; 37·8 per cent of patients with 3 or more symptoms at 6 weeks had symptoms at 1 year (*Table IV*).

Eight persons were involved in litigation, and all of these had symptoms at 1 year. Of the 19 patients with symptoms at 1 year, 9 had the same symptoms of which they had complained at 6 weeks. In the other 10, at least 1 of the symptoms was new. Of the 10 patients with new symptoms at a year, 60 per cent were involved in litigation, whereas of the 9 who had symptoms which were not new, only 2 (22 per cent) were involved in litigation. This association is not significant, probably because of the small numbers ($P = 0{\cdot}170$).

Of 15 patients thought to be malingering at 6 weeks, 5 (33 per cent) were involved in litigation. Of 103 not thought to be malingering, only 3 (2·9 per cent) were involved in litigation ($P = 0{\cdot}008$). Of 126 people who thought their work capability was unchanged, 4 (3·2 per cent)

Table II. Symptoms at 1 year by age, sex and social class

	No symptoms (%)	*Symptoms* (%)	*P value*
Age			
<20 yr	37 (86·8)	6 (13·2)	
20–39 yr	44 (93·6)	3 (6·4)	0·062
>40 yr	31 (75·6)	10 (24·3)	
Sex			
M	75 (90·4)	8 (9·6)	0·044
F	37 (77·1)	11 (22·9)	
*Social class**			
1 and 2	20 (95·2)	1 (4·8)	
3	64 (85·3)	11 (14·7)	0·247
4 and 5	24 (77·4)	7 (22·6)	

*Information missing for 4 patients.

Table III. Symptoms at 1 year by amnesia, symptoms and signs at 24 h

Condition		*No symptoms* (%)	*Symptoms* (%)	*P value*
Retrograde amnesia	Less than 15 min	106 (84·8)	19 (15·2)	0·592
	Over 15 min	6 (100)	0	
Post-traumatic amnesia	Less than 15 min	37 (90·2)	4 (9·8)	0·100
	15 min–1 h	35 (76·1)	11 (23·9)	
	Over 1 h	40 (90·9)	4 (9·1)	
Headache at 24 h	Absent	32 (94·1)	2 (5·9)	0·155
	Present	80 (82·5)	17 (17·5)	
Diplopia at 24 h	Absent	102 (85·7)	17 (14·3)	0·686
	Present	10 (83·3)	2 (16·7)	
Anosmia at 24 h	Absent	98 (86·0)	16 (14·0)	0·713
	Present	14 (82·4)	3 (17·6)	
Other positive CNS signs at 24 h	Absent	84 (90·3)	9 (9·7)	0·026
	Present	28 (73·7)	10 (26·3)	

Table IV. Symptoms at 1 year by external cause, motivation, symptoms at 6 weeks and malingering

	No symptoms (%)	*Symptoms* (%)	*P value*
External cause			
Falls	26 (81·2)	6 (18·6)	0·405
Others	86 (86·9)	13 (13·1)	
*Whose Fault**			
Employer or impersonal body	15 (71·4)	6 (28·6)	0·078
All others	94 (88·7)	12 (11·3)	
Number of symptoms at 6 weeks			
None	58 (100·0)	0 (0·0)	
1–2	31 (86·1)	5 (13·9)	<0·001
3+	23 (62·2)	14 (37·8)	
Malingering†			
No	90 (87·4)	3 (12·6)	0·016
Yes	9 (60·0)	6 (40·0)	

*Information missing in 4 patients.
†23 patients did not attend hospital at 6 weeks; they were interviewed at home and no note made about malingering.

were involved in litigation. Of 5 who thought their work capability had deteriorated, 4 (80 per cent) were involved in litigation ($P = 0{\cdot}001$). Of 127 who thought their family relationships were normal, 5 (3·9 per cent) were involved in litigation. Of 4 who thought their relationships had deteriorated, 3 (75 per cent) were involved in litigation ($P < 0{\cdot}001$).

DISCUSSION

We set out to answer three basic questions about postconcussion symptoms following minor head injuries—how many patients complain, of what do they complain and why? One year after their accidents 14·5 per cent of our patients had some complaint. This figure cannot be taken as applicable to all adults with minor head injuries, regardless of the treatment they receive. Kelly (1972) has produced evidence to show that symptom rates are much higher when patients get no explanation of their symptoms, no encouragement and no treatment. We hope that our figures represent a reasonable outcome among patients who get good diagnosis, treatment and rehabilitation.

It is a weakness of the study that 13 patients were untraceable and 1 had died from unconnected causes. Many of those patients came from areas of major social upset due to the civil disorder in Northern Ireland. It was common for people to move house without notifying a change of address. We compared these 14 patients with the remaining 131, but found no difference in the composition of both groups.

Thirteen patients were recorded as having no symptoms on the basis of replies from their general practitioner. This method of collecting information was accepted only after patients had failed to reply to letters, and their houses had been found to be empty at the time of visiting. The motivation of patients to cooperate in this regard was not high, especially if they had only had symptoms for a day or two following injury. Our figures on the proportion of patients with symptoms must be understood to be the most accurate estimate we could make under the circumstances. Any error is likely to be an underestimate.

Our patients complained of 14 different symptoms. One patient complained of 9 symptoms. It is probably the multiplicity and the diversity of complaints which has given the term 'postconcussion syndrome' such widespread acceptance. However, of the 19 patients who complained, 6 had only 1 symptom and a further 7 had 2. The remaining 6 had between 4 and 9 symptoms. In a syndrome one expects groupings of inter-related symptoms. We found no such groupings either at 6 weeks or at 1 year. In the latter case, this is not surprising in view of the small numbers of patients with multiple complaints.

In our report on symptoms 6 weeks after injury, we produced evidence for both organic and psychogenic factors in the cause. The present report confirms the original finding.

Organic causes

Various factors tend to support an organic cause. Headache, anosmia and diplopia at 24 h after injury, considered separately, were not associated with a higher symptom rate at 1 year. (There were significant associations for each of these with more symptoms at 6 weeks.) With only 19 patients complaining of symptoms at 1 year, this result is not surprising. When all other positive neurological findings at 24 h were considered as a single group, then there was a significant association with symptoms at 1 year. This clearly supports the theory of an organic cause.

Although the trend towards higher symptom rates in patients over 40 years of age does not quite reach the significance level, if the cause was organic one would expect such an association. The ageing of brains and their blood vessels would presumably make them more susceptible to trauma.

In addition, the pattern of development of symptoms to some extent supports the organic theory. Symptoms following concussion can be divided into three groups. Firstly, those present at 24 h, which are not found after the first few days including nausea, vomiting and drowsiness. Secondly, those present at 24 h, which continue for weeks or months, including headache, dizziness, fatigue and epilepsy. There is a strong tendency for patients with headache and dizziness at 24 h to complain of the same symptoms at 6 weeks, although it does not reach the point of significance. Fatigue is very difficult to assess at 24 h. Many patients have either sustained accidents at night, or had hourly interruptions for taking observations. Fatigue may be felt yet not complained of, though when asked specifically many patients deny fatigue. Epilepsy has been so rare in this series that no conclusions can be drawn. Lastly, are those symptoms which are complained of after a few weeks, though not complained of originally. This includes all the remaining symptoms in *Table III*. Although patients do not mention these symptoms in the first 24 h, this does not mean that the condition

has not arisen. Symptoms like irritability, sleeplessness, failure in concentration or memory and sensitivity to alcohol are gradually recognized by the patient in the circumstances of his daily life. One would expect that by 6 weeks any of them that were present would have been noted.

Our patients were more likely to complain of a symptom at 1 year if they had complained of it at 6 weeks. This association was significant for 8 different symptoms. The numbers complaining of the remaining symptoms were so few that such an association became difficult to prove.

The development of symptoms after concussion, on the whole, suggests a condition which arises immediately after the injury and then gradually improves. This would support the concept of organic damage. Oppenheimer (1968) has demonstrated that neuronal damage does occur after mild head injuries with concussion. It is not surprising that symptoms result.

Psychogenic causes

It was shown in our previous study that patients who blamed their employer or some large impersonal body were more likely to have symptoms at 6 weeks than those who blamed themselves, another person or considered the accident to be an act of God. This is evidence that motivation is a factor in producing symptoms at this stage. In a similar comparison made 1 year after the accident, there is a marked trend for those blaming their employer or an impersonal body to have more symptoms. This trend does not reach significance, but this is probably due to the small number of patients involved. On the whole this evidence is suggestive of a psychological factor.

As at 6 weeks, at 1 year women have a higher symptom rate than men. Our men and women patients were compared for the length of post-traumatic amnesia, the number of symptoms at 24 h, the external cause of the accident and the person blamed for causing the accident. No significant difference was found. The sex difference does not appear to be due to extraneous factors. A similar difference was found in the series of Kelly (1972) and that of Lidvall et al. (1974). It is hard to think of a physical explanation. The different psychological reactions of men and women seems more likely. This therefore is evidence that psychological factors are involved.

We have argued that in most patients symptoms occur initially at the moment of injury or within a very short period, and from then on symptoms resolve. This is what one would expect with an organic condition. However, there are some patients whose symptoms arise later. At 1 year after injury, 10 of our 19 patients who had symptoms complained of symptoms of which they did not complain at 6 weeks. New symptoms at this stage are not conclusive evidence of psychological factors, but they are strongly suggestive. Although only 10 patients were involved, the symptoms complained of covered the full spectrum of postconcussion symptoms with the exception of fatigue, anosmia and visual defect.

Fifteen patients at 6 weeks were thought to be malingering—not unconscious victims of forces they did not understand but, at least in the early stages, deliberately exaggerating and falsifying their account of their symptoms. The grounds for judging a patient to be a malingerer were: bizarre and exaggerated descriptions of symptoms and strange responses during routine examination, most often noticed during the testing of the visual fields. The fact that 9 out of 15 patients who were thought at 6 weeks to be malingering had no symptoms at 1 year suggests that this highly subjective judgement may have been erroneous in many cases. However, 50 per cent of those judged to be malingering had symptoms at 1 year, and this strongly suggests a non-organic cause in these cases.

The question of malingering is very difficult. Some claim that the condition does not exist. Others consider that all postconcussion symptoms are an expression of malingering. Experience with medicolegal examinations outside this trial convinced us that some patients consciously exaggerate or distort their symptoms and physical signs. It seemed important to attempt to assess the relevance of such malingering in the complaints of our patients.

In spite of the limitations of our method, our results at least show that postconcussion symptoms at 1 year occur in patients not involved in litigation, and in whom signs of malingering were not found when they had deliberately been sought.

CONCLUSION

There are four different hypotheses consistent with our evidence.

1. Patients are of 2 types, those whose symptoms are caused by organic and those whose symptoms are caused by psychological factors.

2. The symptoms found after concussion are of 2 kinds, those caused by organic and those by psychological factors.

3. Symptoms after concussion are caused and

maintained by the joint action of organic and psychological factors.

4. Early symptoms after concussion are caused by organic factors. Psychological factors prevent the resolution of symptoms in some patients and stimulate the production of new symptoms in others.

It would be necessary to have a larger prospective series than our present one to assess the truth of these hypotheses. It may be that each one expresses part of the truth. On the whole the fourth hypothesis seems to offer a satisfactory explanation for most cases.

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