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Hysteria following brain injury

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Abstract

Of 167 patients referred to a unit treating severe behaviour disorders after brain injury, 54 showed clinical features closely resembling those of gross hysteria as described by Charcot. Close correlation was found with very diffuse insults (hypoxia and hypoglycaemia), but not with severity of injury or with family or personal history of hysterical or other psychiatric disorder. The findings may have implications for the understanding of the nature of hysteria.

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"Hysteria" is a difficult term, partly because it embraces several distinct ideas. The complexity is well illustrated by Veith[1] and in two recent multi-author books.[2 3] In addition to the difficulties of characterising the central idea of hysteria, problems arise from the fact that a range of disorders is subsumed. Four semi-separable syndromes may be identified: hysterical personality disorder, Briquet's syndrome or "the stable syndrome of hysteria,"[4] the more focal presentations of dissociation still usually referred to as "conversion hysteria," and what might be called "Charcot's syndrome" or "gross hysteria." Charcot described a large number of individuals, mainly but not exclusively inpatients, who for much if not all of the time showed a wide and varying range of usually gross disturbances of function and behaviour that could not be explained on a basis of demonstrable pathology of the central nervous system and were thus considered to be "simulations."[5] It was his view, however, that these simulations were compulsive rather than voluntary, and were the result of an as yet unrecognised kind of organic brain disturbance.

In a private hospital unit specifically designed for treating patients with severe behaviour disorders following brain injury,[6] it is the routine practice to make systematic observations and ratings of behaviours,[7] and all observations are pooled and discussed regularly by the whole treatment team. As a result, a particular patient with severe hypoxic brain injury from an anaesthetic accident (see below) was ultimately recognised to be presenting a wide array of behaviours that could be explained only on a basis of gross hysteria. (It was interesting that it took several months before the team suddenly realised the nature of these disturbances. In retrospect it seemed that the emotive aspects of severe brain injury of this kind had elicited what amounted to a mental set against accurate identification of the problem.)

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Case history

Case A

At the age of 26, A, a female schoolteacher, underwent planned cholecystectomy. The operation went well, but during the immediate recovery period she became cyanosed. Urgent investigation revealed that the lines supplying nitrous oxide and oxygen had been reversed on the other side of the wall of the theatre. With appropriate treatment cardiorespiratory function soon became normal, but she remained unconscious for some days, emerging very gradually, and continuing to seem confused for many months. It became apparent that she was cortically blind, with gross disturbance of eye movements, and there was a diffuse movement disorder with dystonic quadriparesis, and later, mild choreoathetosis. She soon became disinhibited in talk and behaviour, and required constant supervision. She began to produce an almost continuous stream of "empty" phrases ("Nurse, darling, I'm hungry, I'm thirsty, I want to go to the toilet, nurse. . .").

A succession of "special" nurses all developed the habit of responding to each of these utterances in a reassuring manner. She gradually regained mobility (though this was awkward and hampered by her visual deficit) and also control of bowel and bladder. Several attempts at formal rehabilitation were blocked by her disordered behaviour.

When case A was seen some two and a half years after the injury constant utterances interfered with all activities, although she was able to cooperate with physical examination to some extent. There was no apparent response to visual stimuli, though for most of the time she kept her eyes closed. Eye movements were severely disrupted, with searching nystagmus. Speech was slightly dysarthric and often telegrammatic. There was no paresis, but most movements were dyspraxic, and there was plastic rigidity of all limbs; slight choreoathetotic movements were seen at rest and on effort. Reflexes were normal. CT had shown mild diffuse cortical atrophy and moderately enlarged ventricles, but an electroencephalogram was normal.

She was admitted to a single room in a general psychiatric unit and had one to one nursing. It was thought she had considerable potential for further rehabilitation if the behaviour disorder could be overcome. The constant

phrases were seen as the result of consistent inappropriate reinforcement by nursing staff of initially exploratory verbalisations provoked by absence of vision. The expectation was that consistent ignoring of the empty phrases, combined with positive attention for any appropriate communications, would soon lead to the end of the maladaptive behaviour. In the event, the initial response to this approach was as expected, but before the frequency of utterances reached zero it rapidly increased again. Over several months the frequency rose and fell in rhythmic fashion, despite carefully consistent responses from staff. At this point she was transferred to the behavioural unit and full reassessments were made in all areas of functioning. An assessment meeting of all staff six weeks after transfer revealed many deviant behaviours. It became obvious that she had begun to speak ingressively—that is, on inspiration only—for most of the time. However, various members of the team reported occasions, especially at night, when she would speak completely normally and hold sensible and even sophisticated conversations; one night nurse reported conversations in French after she had been talking about her French night classes. There were also reports of the sudden retrieval of small objects from the floor on the other side of the room. It became apparent that case A had long since learned her way around the unit, despite the fact that she usually went about with her eyes closed and with her hands held palms up above her head. Though any request for information previously given was met with responses suggesting severe memory disorder, case A often supplied such information accurately in casual conversation, or when listening to the discussions of other residents. Similarly, requests for specific actions or movements often produced the precisely opposite response, while normal versions of the same actions appeared naturally in the course of complex behaviours. As a general feature, there was a noticeable tendency for the performance of almost any skill to vary from occasion to occasion.

Several behaviours were described, some recurringly, which had the quality of the Ganser symptom,[8] or "approximate answers." (The essence of this symptom is that a response is given which, though incorrect, is wrong in a way that implicitly shows knowledge of the correct response. Although most commonly described in terms of verbal responses, it clearly has its counterpart in non-verbal behaviours.) A recurring example was that, on the way to the swimming pool she would walk into each of the nine trees beside the path, without ever missing one. The most impressive example, however, and the one that led to the sudden dropping of scales from eyes, as it were, was reported by an occupational therapy student. She had been told (though the news had not reached the rest of the team) that some weeks before A had accurately identified the colours of a series of cards, but only on some days. The student decided to try this again and was accompanied in the session by a nurse who knew A well. The first card presented was red. A offered twenty guesses without mentioning red at all. By this point the nurse had lost patience and said, "For goodness' sake, A, what are the colours of the rainbow?" A's immediate response was: "Orange, yellow, green, blue, indigo, and violet." When producing opposite or otherwise aberrant responses, A would often show behavioural evidence of playfulness, commonly a "knowing grin."

The members of the team suddenly saw a whole range of behaviours in a new light, including the habit of walking around with eyes closed, which had not previously received open comment, but which is far from typical of the blind. All felt embarrassed at not having had the idea before, but most initially experienced a feeling of guilt at thinking of her problems as hysterical, an interesting but not uncommon reflection on the general perception of the nature of hysteria.

There were two useful effects of this discovery. Firstly, it led to a great deal of reading and general inquiry on the topic of hysteria so that the team became much more effective in managing her behaviour. Indeed, it marked the turning point after which her behaviour and performances began to improve, in some instances dramatically. One specific example was that, quite out of the blue, she began to walk around the unit bent double with her palms down, almost at floor level. Recent reading of Hurst made it possible to recognise this as "camptocormia" (from the Greek, "bent tree trunk"), a frequent hysterical manifestation in soldiers in the trenches during the first world war.[9] (Interestingly, a recent case has been reported from Nicaragua in a soldier with head injuries.[10]) This had not been discussed on the unit so there seemed no possibility that specific suggestion was involved in its appearance. Hurst's recommended method of treatment was to reassure the patient by pointing out the high degree of motor skill and balance required for this feat. The method was applied each time the behaviour appeared, and it ceased within two days. This was in contrast to her ingressive speech, which had by then persisted for some months. It was eventually "cured" by a similar method using the services of a particularly charismatic colleague posing as a "speech expert"—again an approach much favoured by Hurst.

Secondly, it focused attention on the idea that her underlying deficits, neurological and neuropsychological, could be more accurately appraised. This was achieved both by stitching together a patchwork of all best performances and by deliberately exploiting her tendency to produce opposite and approximate answers.

An important observation at this point was that many of the hysterical behaviours appeared spontaneously, rather than in response to requests or questions. This seems to be a characteristic of hysteria, and reasonably attracts the description of "playing games" or "manipulativeness." The term manipulativeness causes much difficulty. As Chodoff and Lyons point out, manipulative behaviour is universally human.[11] However, this is true only in the sense of manipulation of

Table 1 Numbers (percentages) of patients in each group showing different features of hysteria

Hysterical feature	*Total (n = 167)*	*H (n = 54)*	*Non-H (n = 113)*
Game playing	55 (33)	49 (91)	6 (5)
Approximate answers	42 (25)	42 (78)	0
Neurological conversion	26 (16)	26 (48)	0
Psychological conversion	51 (31)	51 (94)	0
Opposite effort	53 (32)	50 (93)	3 (3)
Ideomotor dyspraxia	30 (18)	29 (54)	1 (1)
Unreinforcibility	60 (36)	51 (94)	9 (8)

Table 2 Numbers of hysterical features in patients, by sex

Hysterical feature	*Male (n = 109)*	*Female (n = 58)*	*Total (n = 167)*
0	88	17	105
⩾1	21	41	62
⩾2	20	35	55
⩾3	20	34	54
⩾4	18	23	41
⩾5	14	16	30
6	8	8	16

others in pursuit of a specific goal. What seems to characterise patients with hysteria is, as Charcot put it so eloquently,[5] that they tend to "simulate for no other reason than art for the sake of art."

Methods

Having observed and discussed this range of hysterical behaviours, against a background of recently acquired knowledge of the disorder, the team members were now able to recognise similar problems in other patients currently in the unit and in those referred for day or inpatient assessment. It also became possible, in taking the history from relatives or other carers, to frame non-leading questions that would reveal the presence of hysterical features. Based on experiences with case A, seven features were identified. The standard behaviour rating scale used in the unit was modified to allow them to be identified more accurately,[7] and their presence or absence was added to the database kept for each patient referred.

The features are listed in table 1, and the appendix gives the operational definitions on which identification was based.

The features include a failure to respond to reinforcement. This was necessarily more accurately assessable in those who spent more than a day under close observation. It seems to be relevant and of interest but was not included in the criteria for identification of the hysterical behavioural pattern.

The rating scales and neurological, psychiatric, and neuropsychological assessments of all patients referred over the next seven years were examined for the presence of the six other features, and the group was divided into two subgroups: those with three or more features present were called the "H" group and those with fewer than three, the "non-H" group. These two groups were then compared with respect to age at injury, sex, severity of injury, interval from injury to assessment, type of injury, and various other aspects of their neurological state and personal history.

All patients had extensive neuropsychological assessments, which were repeated several times in those who underwent rehabilitation in the unit. Usually the results of formal assessments were at considerable variance with the observed skills of the individual, and it has to be concluded that the assessments did not provide reliable evidence of the pattern of brain damage. Most patients had CT very soon after injury; attempts at late scanning were invariably undermined by a lack of sufficient cooperation.

Results

There were 54 patients (32·3%) with three or more primary features and 113 with fewer than three (table 2). (That a criterion of two features adds only one individual (32·9%) and that of just one feature only eight (36·5%) suggests that any of these features represents a qualitative difference.)

Table 3 shows that the non-H group was typical of people with head injuries in age and sex. The H group, on the other hand, was slightly older (Mann-Whitney U test, p = 0·018). More remarkably, the male to female ratio for this group is 0·6:1, despite the fact that the majority suffered traumatic head

Table 3 Details of patients referred for brain injury

	Total			*H*			*Non-H*		
	Male (n = 109)	*Female (n = 58)*	*Total (n = 167)*	*Male (n = 20)*	*Female (n = 34)*	*Total (n = 54)*	*Male (n = 89)*	*Female (n = 24)*	*Total (n = 113)*
Age at injury* (years):									
Mean (SD)	28·5 (14·0)	30·4 (13·7)	29·2 (13·9)	30·6 (14·4)	33·8 (13·6)	32·6 (13·9)	28·0 (13·9)	25·7 (12·6)	27·5 (13·7)
Range	8–65	9–62	8–65	14–61	15–62	14–62	8–65	9–50	8–65
Months from injury*:									
Mean (SD)	36·6 (31·8)	38·8 (38·9)	37·3 (34·3)	31·9 (27·6)	30·7 (27·4)	31·1 (27·2)	37·6 (32·7)	50·3 (49·4)	40·3 (37·0)
Range	1–201	1–197	1–201	3–117	1–96	1–117	1–201	4–197	1–201
Coma duration (weeks)*:									
n	94	47	141	20	30	50	74	17	91
Mean (SD)	4·9 (5·9)	7·5 (10·0)	5·8 (7·6)	3·5 (3·3)	3·8 (4·0)	3·7 (3·7)	5·3 (6·4)	14·1 (13·8)	6·9 (8·9)
Range	0–25	0–52	0–52	0–12	0–18	0–18	0–25	14–52	0–52
Physcial disability score**:									
Mean (SD)	5·10 (4·48)	5·04 (5·04)	5·08 (4·66)	6·30 (3·96)	4·18 (4·86)	4·96 (4·63)	4·83 (4·56)	6·30 (5·12)	5·14 (4·70)
Range	0–16	0–16	0–16	0–16	0–16	0–16	0–16	0–16	0–15
M:F ratio			1·9:1			0·6:1			3·7:1

*Only significant differences—coma duration: females, H *v* non-H, p = 0·016 (*t* test), p = 0·008 (U test); non-H, males *v* females, p = 0·008 (*t* test), p = 0·008 (U test).
**Bond's neurophysical scale[12]—*t* test: p > 0·1 between all groups; Mann-Whitney: p > 0·08 between all groups.

Table 4 Distribution of coma severity

Coma duration	*< 6 h*	*>6 h– 1 day*	*>1 day– 1 week*	*>1– 2 weeks*	*>2– 3 weeks*	*>3– 4 weeks*	*>4– 6 weeks*	*>6– 12 weeks*	*>12 weeks*
Total No of patients	6	1	13	23	23	14	17	18	26
No in H group (n = 50)	3	0	7	5	11	5	6	9	4
% of total	50	0	54	22	48	36	35	50	15
% of H group	6	0	14	10	22	10	12	18	8

Table 5 Primary injury in patients in both groups. Values are numbers (percentages) of patients

		H			*Non-H*		
	Total (n = 167)	*Male (n = 20)*	*Female (n = 34)*	*Total (n = 54)*	*Male (n = 89)*	*Female (n = 24)*	*Total (n = 113)*
Head injury	130 (78)	15 (75)	16 (47)	31 (57)	78 (88)	21 (88)	99 (88)
Brain tumour	2 (1)	1 (5)	0	1 (2)	1 (1)	0	1 (1)
Subarachnoid haemorrhage	10 (6)	0	2 (6)	2 (4)	5 (6)	3 (13)	8 (7)
Stroke	3 (2)	1 (5)	2 (6)	3 (6)	0	0	0
Encephalitis	8 (5)	2 (10)	3 (9)	5 (9)	3 (3)	0	3 (3)
Anoxia	12 (7)	1 (5)	9 (26)	10 (19)	2 (2)	0	2 (2)
Hypoglycaemia	2 (1)	0	2 (6)	2 (4)	0	0	0

injury (see table 5). In terms of rehabilitation referrals, the whole group was unusual, having a mean time from injury to referral of just over three years; the unit is in a private hospital and the threshold of management difficulty is not surprisingly quite high before funding is likely to be made available. There was a trend for the H group, especially females (t test, p = 0·1), to be referred earlier than the others. These were referrals to a unit designed for those with very severe behaviour disorders,[13] and this difference probably reflects the fact that patients with the characteristics of the H group tend to present greater management problems than do the majority of patients with straightforward brain injuries.

Table 3 also shows that the whole group had suffered extremely severe injuries. Accurate estimates of coma duration were available for 141 of the 167 referrals (50 of the 54 in the H group). Estimates of post-traumatic amnesia, the other leading index of severity, were available in only 65 (16 of the H group). The data have therefore not been further analysed on the basis of this measure. Although the significance was not great (*t* test, p = 0·07), the non-H group sustained more severe injuries; most of the difference was accounted for by the females (p = 0·006). The females were also much more severely injured than the males, but only in the non-H group. Table 4 suggests that there was no straightforward relation between severity and the appearance of the "H syndrome," though it was perhaps least common among patients with most severe injuries. The three members of the H group with coma of less than six hours were all stroke victims. Table 3 shows also that physical disability had no close correlation with hysterical features, though on the whole slightly fewer in the H group had severe or very severe disability.

Table 1 shows not only that the primary features are all much more common in the H group but that failure of abnormal behaviours to be consistently modified by reinforcement was also typical; interestingly, those in the non-H group who failed to respond in this way all showed evidence of pre-existing psychopathic traits.

Table 5 suggests that anoxic and hypoglycaemic insults were almost exclusive to the H group. When other forms of primary injury were examined more closely (table 6), associated factors that add obvious ischaemic or hypoxic hypoxia to the primary insult were very much more common in the H than in the non-H group. Indeed, very diffuse insults were almost a necessary feature of the H group; the only exceptions were patients who suffered strokes.

Though the incidence of post-traumatic epilepsy was not greatly different between the total groups (table 7), the males of the H group had more than twice the incidence of other subgroups. Thus the overall incidence was 18%; in males in the H group it was 40%, but in those in the non-H group, only 15%. Conversely only 12% of the females in the H group developed epilepsy, compared with 21% of the non-H females. This might suggest that additional hypoxic insult sustained in the course of seizures had contributed to the development of the behavioural syndrome.

Table 6 Causes of very diffuse insults (*). Values are numbers (percentages) of patients

Main cause	Plus	*H (n = 54)*	*Non-H (n = 113)*	*Total (n = 167)*
Head injury	Nil	1 (2)	81 (72)	82 (49)
	Swelling*	20 (37)	6 (5)	26 (16)
	Shock*	4 (7)	2 (2)	6 (4)
	Asphyxia*	2 (4)	2 (2)	4 (2)
	Arrest*	1 (2)	0	1 (1)
	Hydrocephalus*	3 (6)	6 (5)	9 (5)
	Fat embolism*	0	2 (2)	2 (1)
Subarachnoid haemorrhage	Nil	0	7 (6)	7 (4)
	Swelling*	2 (4)	1 (1)	3 (2)
Stroke		3 (6)	0	3 (2)
Tumour	Nil	0	1 (1)	1 (1)
	Surgical shock*	1 (2)	0	1 (1)
Encephalitis	Nil	0	3 (3)	3 (2)
	Swelling*	5 (9)	0	5 (3)
Anaesthesia	Ischaemia*	5 (9)	0	5 (3)
	Anoxia*	2 (4)	0	2 (1)
Carbon monoxide poisoning*		1 (2)	2 (2)	3 (2)
Hypoglycaemia*		2 (4)	0	2 (1)
Asphyxia*		1 (2)	0	1 (1)
Cardiac arrest*		1 (2)	0	1 (1)
All diffuse insults*		50 (93)	19 (17)	69 (41)

Table 7 Numbers (percentages) of patients in both groups with post-traumatic epilepsy

Total			H			Non-H		
Male (n = 109)	Female (n = 58)	Total (n = 167)	Male (n = 20)	Female (n = 34)	Total (n = 54)	Male (n = 89)	Female (n = 24)	Total (n = 113)
21 (19)	9 (16)	30 (18)	8 (40)	4 (12)	12 (22)	13 (15)	5 (21)	18 (16)

Table 8 Numbers (percentages) of patients in both groups with extrapyramidal disorders

	H (n = 54)	Non-H (n = 113)
None	15 (28)	105 (93)
Chorea	8 (15)	0
Dystonia:		
Diffuse	8 (15)	4 (4)
Activated	9 (17)	2 (2)
Action	1 (2)	1 (1)
Parkinsonian features	12 (22)	0
Buccofacial dyskinesia	1 (2)	0
Red nucleus tremor	0	1 (1)
Total	39 (72)	8 (7)

Extrapyramidal disorders (table 8) were very much more common in the H group, being present in 72% compared with only 7% in the non-H group. This is hardly surprising, however, as the basal ganglia are among the brain structures most sensitive to hypoxic or hypoglycaemic insult.[14–17]

There tends to be a general assumption that emotional and behavioural disorders after brain injury represent either an exaggeration of pre-existing problems or a reaction to the awareness of deficits. As almost 40% of the group showing grossly hysterical features were male and hysterical disorders in general psychiatric practice are very much more common in females, this assumption seems unlikely. Moreover, severity of injury was if anything greater in the non-H group, who had as many physical and cognitive deficits as the H group, so to argue for greater emotional reaction would require evidence of some predisposition. Table 9 shows no significant difference between the H and non-H groups so far as family history of hysteria (2% and 1% respectively) and personality disorder (22% and 19%) are concerned. Table 10, however, shows a higher than expected incidence of previous psychiatric disorder in the males of the H group. Allowing for the fact that hysterical traits and episodes are generally rare in males, and considering therefore the total incidence of any previous psychiatric history, the H group had considerable excess over the non-H group, though curiously the highest incidence of all was in the females in the non-H group. As behavioural characteristics related to psychiatric disorder may predispose to the acquisition of various kinds of brain injury, the history of each individual was scrutinised for evidence of a direct causal link between the previous history and the form of the insult. When this was taken into account, there was no significant difference between the two groups in the incidence of previous psychiatric disorder, which seems to be a determinant of brain injury, rather than of the H group features.

Discussion

These findings suggest that very diffuse forms of brain insult (hypoglycaemia and the various types of hypoxia) are able to cause a pervasive disorder marked by behaviours that closely resemble those described by Charcot.[5] Analysis of the most common features suggests an even closer resemblance to the childhood pathological demand avoidance syndrome recently described by Newson.[18] A case conference illustrating the same clinical picture, though complicated by pre-existing severe affective disorder, has recently been published from the Institute of Psychiatry.[19] The evidence does not support the view that this clinical picture represents a disorder reactive to the psychological stresses of severe brain injury and the personal and functional losses it produces: these were at least as pronounced in subjects who did not show the picture. Neither does family history nor personal psychiatric

Table 9 Numbers (percentages) of patients with a family history of hysteria and personality disorder

	H			Non-H		
	Male (n = 20)	Female (n = 34)	Total (n = 54)	Male (n = 89)	Female (n = 24)	Total (n = 113)
Hysteria	0	1 (3)	1 (2)	1 (1)	0	1 (1)
Personality disorder	4 (20)	8 (24)	12 (22)	14 (16)	8 (33)	22 (19)

Table 10 Psychiatric history in patients in both groups. Values are numbers (percentages) of patients

	H			Non-H		
	Male (n = 20)	Female (n = 34)	Total (n = 54)	Male (n = 89)	Female (n = 24)	Total (n = 113)
Hysterical:						
Traits	0	3 (9)	3 (6)	0	4 (17)	4 (4)
Episodes	0	4 (12)	4 (7)	1 (1)	2 (8)	3 (3)
Other psychiatric disorders	7 (35)	4 (12)	11 (20)	12 (13)	4 (17)	16 (14)
Any psychiatric history	7 (35)	8 (24)	15 (28)	12 (13)	7 (29)	19 (17)
Linked to injury	4 (20)	4 (12)	8 (15)	1 (1)	3 (13)	4 (4)
Not linked	3 (15)	4 (12)	7 (13)	11 (12)	4 (17)	15 (13)

history suggest a specific vulnerability; the male: female ratio of 0·6:1 also argues against this. There were a few patients in the H group in whom the clinical features clearly did derive from pre-existing personality disorder, but they were a very small minority.

The aetiology of hysteria has always been mysterious. There have been those who have believed it to result from organic brain disorder,[2 5 20–35] and those who have seen it as a purely psychologically determined disorder.[36–44] Most writers make room for multifactorial interaction, and there is at least a measure of agreement that those who develop hysterical disorders are in some way predisposed or vulnerable through some anomaly of the nervous system or its functioning. The nature of any such anomaly is far from clear, however.

The findings of this study raise the possibility that some aspect of the physical damage to the brain is responsible for the appearance of the clinical features. If this is so, what structures might be involved? The cluster of features seems to depend on the nature of the injury, rather than on its severity, whether this is measured in terms of coma duration or the extent of neurophysical deficits. The structures most likely to be damaged by any degree of hypoxia or hypoglycaemia are those of the basal ganglia and diencephalon,[14–17] and the high incidence of extrapyramidal disorders in the H group is evidence that some of these structures were indeed damaged. It is instructive also to look more closely at the three patients with stroke. There were no patients with stroke in the non-H group, reflecting the fact that severe disorders of social behaviour are rare after stroke. The patient who had suffered a middle cerebral artery stroke was a man with an unequivocal history of psychopathic personality disorder who had always been described as both aggressive and manipulative. The two others were women without previous psychiatric disorders who had suffered multiple discrete small infarcts in the basal ganglia and thalamus, again implicating the same general brain area in the generation of the clinical features.

It may be possible to speculate on the mechanisms whereby damage to these areas might produce behavioural changes of these kinds. Although each of the hysterical features is distinct in terms of the operational definitions embodied in the behavioural rating scale, they can be construed as being closely related as they all have an element of simulation or playfulness. An alternative interpretation might be in terms of attempts to dominate others. Both playfulness and dominance have strongly biological bases, and indeed they seem to be closely linked patterns of behaviour.[45 46] Little seems to be known of their neurophysiological bases, but evidence implicates diencephalic (thalamocingulate) and other parts of the limbic structures.[47]

The patients seem to share at least one other characteristics, namely dissociation. For example, the game playing, approximate answers, dissociative memory disorders and opposite effort are often quite transparent, in a way that would be embarrassing if the individual were fully aware of his or her performance. Moreover, the neurological "conversions" were classic and included several examples of hysterical contracture—as Charcot often demonstrated, there is dissociation at an autonomic and automatic level in such disorders.[5] With many, however, the main dissociation seems to be simply that of the awareness of the act of simulation. Several attempts have been made to explain dissociation on a basis of thalamocortical gating mechanisms.[33 34 48]

In their responses to demands and in the behaviours produced for the apparent purpose of avoiding any kind of compliance these patients showed a lack of concern about pleasure or pain amounting to a loss of hedonic responsiveness. This would also be one possible explanation of the patients' lack of response to behaviour modification techniques, which are designed to exploit operant conditioning through the control of positive reinforcement.[49] There is much evidence to implicate diencephalic structures,[50–52] largely hypothalamic and septal (the latter being said to be represented in man by the nucleus accumbens), in these functions.

For most of the patients their behaviours, including their apparent inability to use appropriately the skills of which they showed intermittent but unequivocal evidence, had led them into settings of care and treatment that were palpably uncongenial. Despite this, they seemed unable to deploy their skills in ways that would rapidly have earned them more pleasant, less stressful surroundings, and in many cases a return to their own homes and families. This suggests a further factor, in addition to their anhedonia. On the one hand, they seemed to be driven to behave as they did; on the other hand, they seemed unable not to. This aspect amounts to Paget's "I cannot will"[20] transposed from the level of simple action to that of complex behaviour. Defects of will are certainly common in extrapyramidal disorders and have been described in hypoxic insults to the basal ganglia.[53] The caudate nucleus, and the nucleus accumbens in particular, have been proposed on the basis of animal neurophysiological research as the interface for the selection of outputs—that is, behaviours—between those relevant to cognitive or to affective motivations.[54]

The possibility that a particular constellation of areas of brain damage, produced perhaps by chance because of the pattern of insults resulting from hypoxia or hypoglycaemia, may create a clinical picture of gross hysteria de novo has wider implications. Although these patients present the gross disturbances of Charcot's form of hysteria, the core features appear, in various mixtures of degrees, in the whole range of "spontaneous" hysterical disorders. These disorders may result from abnormalities of the same set of brain systems, determined by neurodevelopmental anomalies (so called migration disorders) or by perinatal insult. This suggests directions for further research, including a search for hysterical syndromes in

those known to have other developmental anomalies (learning disabilities, the "clumsy" syndrome, and so on) or to have suffered perinatal hypoxia.

This study was begun while I was working in the Kemsley Unit at St Andrew's Hospital, Northampton. I thank the staff of that unit, whose skills of observation made the study possible.

Appendix

Operational definitions

The following are attempts at concise definitions of the features and are therefore somewhat pedantic. In staff training, simpler but much lengthier descriptions were given, with regular repetition, to ensure that the rating of relevant behaviours was standardised. In this study the requirement was simply the recurring presence of the feature, in whatever degree, and this was assessed by group discussion and consensus about pooled descriptions of behaviours. For each definition below the sources of information used are indicated in brackets by B (behavioural observations in everyday settings), N (neurological and mental state examinations), and P (formal neuropsychological assessment).

Game playing—The repeated and varying exhibition of spontaneous behaviours that invite staff responses which are inappropriate to the resident's practical needs or outcome goals or to the prevailing circumstances and which, if made, lead to progressive deviation from the explicit requirements of the moment. (B)

Approximate answers—Responses, either verbal or behavioural, that are incorrect or inappropriate, but whose nature implicitly reveals knowledge of the correct or appropriate response. (The context and the resident's affect must make it clear that the intention is not simply one of humour or wit.) (B, N, P)

Neurological or psychological conversion—Apparent physical or cognitive deficits that vary qualitatively and quantitatively in different contexts (B) and are associated with positive evidence of hysterical nature (N or P)

Opposite effort—Responses that are consistently the opposite of what is required or requested, this being known to be within the subject's ability. (B, N, P)

Ideomotor dyspraxia—Requests for complex motor acts or behavioural performances consistently result in a wide variety of inaccurate or inappropriate responses, but never the appropriate one, which can, however, be observed in circumstances where its performance is incidental to some wider act or behaviour. (N, B)

Unreinforcibility—Initially this was assessed from the overall response to the token economy ("earnings"), changes in specific behaviours that were targets of special behavioural programmes, and subjective judgments about the individual's overt affective responses to positive reinforcement. Later, specific "trials of conditioning" were set up, based on very simple forms of behaviour subjected to consistent positive reinforcement in regular sessions over a limited period.[7 55 56] Failure to show a typical learning curve was the criterion for unreinforcibility.

1 Veith I. *Hysteria: the history of a disease.* Chicago: University of Chicago Press, 1965.
2 Merskey H. *The analysis of hysteria.* London: Baillière Tindall, 1979.
3 Roy A, ed. *Hysteria.* Chichester: Wiley, 1982.
4 Perley MJ, Guze SB. Hysteria—the stability and usefulness of clinical criteria: a quantitative study based on a follow-up period of six to eight years in 39 patients. *N Engl J Med* 1962;**266**:421–6.
5 Charcot J-M. *Clinical lectures on diseases of the nervous system,* Vol III. London: New Sydenham Society, 1889. (Translated by T Savill.)
6 Eames P, Wood R. Rehabilitation after severe brain injury: a special-unit approach to behaviour disorders. *International Rehabilitation Medicine* 1985;7:130–3.
7 Wood RL. *Brain injury rehabilitation: a neurobehavioural approach.* London: Croom Helm, 1987.
8 Whitlock FA. The Ganser syndrome and hysterical pseudodementia. In: Roy A, ed. *Hysteria.* Chichester: Wiley, 1982:185–209.
9 Hurst AF. *Medical diseases of war.* London: Edward Arnold, 1940.
10 Perez-Sales P. Camptocormia. *Br J Psychiatry* 1990;**157**: 765–7.
11 Chodoff P, Lyons H. Hysteria, the hysterical personality and "hysterical" conversion. *Am J Psychiatry* 1958; **114**:734–40.
12 Bond MR. Assessment of the psychosocial outcome after severe head injury. In: Porter R, Fitzsimons DW, eds. *Outcome of severe damage to the central nervous system.* Amsterdam: Elsevier-Excerpta Medica, 1975. (Ciba Foundation symposium 34 (new series).)
13 Eames P, Wood R. Rehabilitation after severe brain injury: a follow-up study of a behaviour modification approach. *J Neurol Neurosurg Psychiatry* 1985;**48**:613–9.
14 Levy DE, Brierley JB, Silverman DG, Plum F. Brief hypoxia-ischemia initially damages cerebral neurons. *Arch Neurol* 1975;**32**:450–6.
15 Adams JH, Brierley JB, Connor RC, Treip CS. The effects of systemic hypotension upon the human brain. Clinical and neuropathological observations in 11 cases. *Brain* 1966;**89**:235–68.
16 Brierley JB, Brown AW, Meldrum BS. The nature and time course of the neuronal alterations resulting from oligaemia and hypoglycaemia in the brain of Macaca mulatta. *Brain Res* 1971;**25**:483–99.
17 Brierley JB, Meldrum BS, Brown AW. The threshold and neuropathology of cerebral "anoxic-ischemic" cell change. *Arch Neurol* 1973;**29**:367–74.
18 Newson E. *Pathological demand avoidance syndrome: diagnostic criteria and relationship to autism and other developmental coding disorders.* Nottingham: Child Development Research Unit, University of Nottingham, 1989.
19 Collins MN, Jacobson RR. Changing interactions between bipolar affective disorder and anoxic brain damage. *Br J Psychiatry* 1990;**156**:736–40.
20 Paget J. Nervous mimicry of organic diseases. Lecture I. *Lancet* 1873;ii:511–3.
21 Janet P. *L'état mental des hysteriques.* Paris, Rueff, 1894.
22 Janet P. *The major symptoms of hysteria.* New York, MacMillan, 1907.
23 Credner L. Klinische und soziale Auswirkingen von Hirnschaedigungen. *Zeitschrift für Neurologie und Psychiatrie* 1930;**126**:721—57.
24 Schilder P. Neurosis following head and brain injuries. In: Brock S, ed. *Injuries of the skull, brain and spinal cord.* Philadelphia: Williams and Wilkins, 1940.
25 Brodie BC. *Lectures illustrative of certain nervous affections.* London: Longman, 1837.
26 Kennedy A, Neville J. Sudden loss of memory. *BMJ* 1957;ii:428–33.
27 Raskin M, Talbot J, Meyerson A. Diagnosed conversion reaction: predictive value of psychiatric criteria. *JAMA* 1966;**197**:530–4.
28 Weinstein EA, Lyerly OG. Conversion hysteria following brain injury. *Arch Neurol* 1966;**15**:545–8.
29 Weinstein EA. Patterns of reduplication in organic brain disease. In: Vinken PJ, Bruyn GW, eds. *Handbook of clinical neurology,* Vol 3. Amsterdam: North Holland, 1969:251–7.
30 Whitlock F. The aetiology of hysteria. *Acta Psychiatr Scand* 1967;**43**:144–62.
31 Tyrer PJ. Measurement of abnormal personality: a review. *J R Soc Med* 1987;**80**:637–9.
32 Levine J, Ludwig AM. Alterations in consciousness produced by combinations of LSD, hypnosis and psychotherapy. *Psychopharmacologia* 1965;7:123–37.
33 Ludwig AM. The psychobiological functions of dissociation. *American Journal of Medical Hypnosis* 1983;**26**:93–9.
34 Ludwig AM. Hysteria: a neurobiological theory. *Arch Gen Psychiatry* 1972;**27**:771–7.
35 Bendefeldt F, Miller LL, Ludwig AM. Cognitive performance in conversion hysteria. *Arch Gen Psychiatry* 1976;**33**:1250–4.
36 Sydenham T. Discourse concerning hysterical and hypochondriacal distempers. In: *Dr Sydenham's complete method of curing almost all diseases, and description of their symptoms to which are now added five discourses of the same author concerning the pleurisy, gout, hysterical passion, dropsy, and rheumatism.* 3rd ed. London: Newman and Parker, 1697:149.
37 Schilder P. The concept of hysteria. *Am J Psychiatry* 1939;**98**:1389–413.
38 Kraepelin E. Hysterical insanity. In: *Lectures on clinical psychiatry.* New York: William Wood, 1904. (Translated by T. Johnstone.)
39 Zangwill OL. The Grunthal-Storring case of amnesic syndrome. *Br J Psychiatry* 1967;**113**:113–28.
40 Ey H. History and analysis of the concept. In: Roy A, ed. *Hysteria.* Chichester: Wiley, 1982:3–19.
41 Slater E. Diagnosis of "hysteria." *BMJ* 1965;i:1395–9.
42 Slater ETO, Glithero E. A follow-up of patients diagnosed as suffering from "hysteria." *J Psychosom Res* 1965; **9**:9–13.
43 Kendell RE. A new look at hysteria. *Medicine* 1972; **30**:1780–3.
44 Roy A. Cerebral disease and hysteria. *Compr Psychiatry* 1977;**18**:607–9.
45 Fagen R. *Animal play behavior.* New York: Oxford University Press, 1981.
46 Symons D. The questions of function: dominance and play. In: Smith EO, ed. *Social play in primates.* New York: Academic Press, 1978:193–230.

47 MacLean PD. Culminating developments in the evolution of the limbic system: the thalamocingulate division. In: Doane BK, Livingston KE, eds. *The limbic system: functional organization and clinical disorders.* New York: Raven Press, 1986:1–28.
48 Silverman J. A paradigm for the study of altered states of consciousness. *Br J Psychiatry* 1968;**114**:1201–18.
49 Wood R, Eames P. Application of behaviour modification in the treatment of traumatically brain-injured adults. In: Davey G, ed. *Applications of conditioning theory.* London: Methuen, 1981:81–101.
50 Rolls ET. *The brain and reward.* Oxford: Pergamon, 1975.
51 Stellar E. Brain mechanisms and hedonic processes. *Acta Neurobiol Exp (Warsz)* 1980;**40**:313–24.
52 Doty BA, Forkner MR. Alterations in pain thresholds and avoidance conditioning in rats with septal lesions. *Neuropsychologia* 1971;**9**:325–30.
53 Laplane D, Baulac M, Widlocher D, Dubois B. Pure psychic akinesia with bilateral lesions of basal ganglia. *J Neurol Neurosurg Psychiatry* 1984;**47**:377–85.
54 Mogenson GJ, Jones DL, Yim CY. From motivation to action: functional interface between the limbic system and the motor system. *Prog Neurobiol* 1980;**14**:69–97.
55 Goldstein LH, Oakley DA. Expected and actual behavioural capacity after diffuse reduction in cerebral cortex: a review and suggestions for rehabilitative techniques with the mentally handicapped and head injured. *Br J Clin Psychol* 1985;**24**:13–24.
56 Goldstein LH. Learning ability in mental handicap and after head injury. *Nursing Times* 1984;**80(3)**:54–7.