LETTERS TO THE EDITOR

The neuropsychology of hanging: an historical perspective

Medalia, Merriam and Ehrenreich¹ reported two cases of attempted hanging with subsequent isolated memory defects. Their report is particularly interesting, as hanging is still one of the most common methods of committing suicide. How death occurs during hanging and how, when and why the brain's activity is altered by strangulation, has been the subject of much debate. Medalia *et al* ignore this literature, and instead incorrectly emphasise in the first sentence of their abstract that "only one report on the neuropsychological sequelae of attempted hanging exists in the English literature".¹

In 1983 a report by Mengech² described "Strangulation as a cause of Korsakoff psychosis". Other English-language reports on the neuropsychological consequences of attempted hanging stem from Whiteley³ and Stengel.⁴

Furthermore, there is a considerable amount of early German-language literature on the neurological and neuropsychological consequences of unsuccessful attempts at hanging.⁵⁻⁹ Already in 1896 Boediker⁷ described attempted hanging as usually resulting in retrograde amnesia. Two of the first detailed reports of such cases were given by Wagner^{8,9} in Southeastern Austria. In his article from 1889⁸ he referred to 17 previous cases which had been described in the years between 1826-87. Wagner⁹ also mentioned Leidesdorf,¹⁰ who had given examples of cases related to hanging. Leidesdorf¹⁰ for example wrote: "The rope which the Siberian Shaman allows to be laid around his throat, until, shortly before choking, he goes into ecstasies, . . ."

Of principal importance are Wagner's publications and a related report of Pick (which is cited in Wagner⁹) as both emphasised the time point of the onset of retrograde amnesia before hanging.

Wagner⁹ noted that attempted hanging usually resulted in severe memory disturbances which he characterised as "dementia acuta", but that these amnesic states were usually only temporary; in a few cases he noted that the reverse pattern might even follow, namely an improvement of amnesic functions or the cure of a psychosis.

A few years later, Wollenberg¹¹ described a 39 year old carpenter, who hanged himself during a stay in a psychiatric clinic, to which he had been admitted with paranoia and because he had tried to hang himself eight days earlier. Wollenberg assumed that a preceding loss of consciousness was an important requirement for retrograde amnesia to occur, though unconsciousness did not necessarily lead to retrograde amnesia.^{12,13}

Some years later, Wachsmuth¹⁴ reported a 23 year old merchant who had a shot-wound in his head of uncertain origin; an attempted suicide was assumed to have occurred. The patient made a second attempt by hanging himself during his admission to hospital,

which resulted in profound retrograde amnesia. At the time of Wagner and Wollenberg there was still a serious discussion on whether retrograde amnesia after hanging was organic or psychic in origin, and this was reviewed by Schneider.¹⁵

The question was also central to Sommer's report¹⁶ on "the amnesic disturbances after attempts by hanging". On the basis of his two cases he confirmed the observation of other scientists that following strangulation there is a retrograde amnesia for a few days and usually also an anterograde one for one or two days.

In the ensuing debate over the views of Möbius (that amnesia following strangulation is of hysterical nature) and Wagner (that it is organic), Sommer was strongly inclined to follow Wagner's opinion (Paul,¹⁷ had argued vehemently against Möbius's opinion, and also Meyer¹⁸ considered Wagner's view the most plausible one). Sommer assumed that complete blockage of the carotids and the interruption of the venous discharge resulted in temporary but still severe nutritional disturbance of the nervous tissue with functional detriment to the neurons but without damaging them permanently. The controversial views of Möbius were published in 1922.¹⁸

Meyer¹⁹ combined a large number of psychological and neurological tests to determine the status of his patient. This 24 year old man attempted to hang himself and was found and rescued early in the morning. His skill memory was preserved, and he remembered how to salute, while his episodic memory was disturbed. Meyer compared the patient's behaviour over several days, asking daily the same questions and tabulating the respective answers. He concluded that the patient's behaviour is best described as Korsakoff's syndrome, and that strangulation will typically result in Korsakoff's symptoms.

Fraenkel²⁰ in 1911 reported the case of a 20 year old woman who after strangulation had massive disturbances in anterograde and retrograde memory and was disoriented to time and place. The patient remained in an amnesic condition even after four months, but had not been psychiatrically normal before the attempted hanging. Fraenkel discussed this case as resembling the symptoms of Korsakoff's psychosis.

After the Second World War, Jacob and Pyrkosch²¹ carried out a detailed analysis of the consequences of hanging on the anatomy of the brain. They based their investigation on the investigation of three out of 20 brains from cases who died after hanging. It is interesting that, as in other cases with hypoxic or anoxic brain damage, the hippocampal formation was consistently affected. The subicular cortex, and Ammon's horn especially, showed degenerated neurons. Other areas of cell damage were in the claustrum, the pallidum, and the thalamus. It cannot be determined, however, if the damage was primarily caused by the reduced oxygen consumption or by convulsions which may occur during hanging.

Together, these early examples may show that there has been a wide discussion of the neurological and neuropsychological sequelae of hanging during the past 100 years and both the mechanisms and brain centres discussed by Medalia *et al*¹ as acting critically on or affecting cognition, had already been similarly debated by earlier authors.

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A neurological audit in Vitoria, Spain

In response to your invitation we are sending this brief account of a neurology outpatient audit in Vitoria, Spain. This is a health district with a population of 300 000 served by two neurology units based in two hospitals, totalling eight neurologists (one neurologist per 37 500 population). The data are based on the 987 new patients seen in 1989 by the three neurologists in one of the hospitals.

The objectives of our audit were somewhat different from the ones in the English audit reported in your journal,¹ reflecting the different systems they study. In Spain there are proportionally more neurologists than in the UK and general practitioners tend to refer all patients whose complaints they suspect to be neurological. This results in the number of referrals being too large to be seen in hospital, so that two types of neurologist are evolving: those working in primary care

centres who see all new cases and filter them to the hospital if necessary, and the more traditional hospital-based ones. Vitoria is unusual in that its comparatively large number of neurologists and hospital facilities offers a fully hospital-based neurology service.

We did not study the waiting time: the aim was stated at the beginning of the audit that no patient should wait for more than four weeks and this was checked at the appointment office. This aim was easily achieved and in fact for most of the year the waiting time was around two weeks. No classification as "urgent" or "routine" cases was considered necessary. (In the past, with longer waiting times, we had attempted that classification on the basis of the referring doctor's notes and found it unreliable; we had also tried to briefly interview each patient but it proved impractical). Occasional patients were sent as urgent and seen the same day. Seventy seven per cent of all cases were referred by their general practitioner.

The table shows the main preliminary diagnoses. Sixty six per cent of all new outpatients had conditions that could be termed "non-organic" and 34% had organic problems. The most common diagnoses were headaches (27%), anxiety-depression (19%), dizziness-faints (11%), epilepsy (5%), tremor and other movements (4%), vascular disease (4%).

Five hundred and seventy one patients (58%) were discharged to the referral source; 10 (1%) were admitted to hospital; 139 (14%) were given a further appointment without ordering any tests; 267 (27%) received outpatient investigation, for example, 100 had a CT brain scan, 89 an EEG

and 27 an EMG.

When both outpatient clinics are compared it seems apparent that they are similar. There is a greater percentage of non-organic problems in the Spanish referrals (66%) compared with the English ones (50%) as would be expected from the different settings. But dealing mainly with outpatients who do not have serious disease is probably a universal neurological (medical?) problem which applies even in the restrictive British system.

We would also like to make the following observations:

1) Are scarce resources being wasted with irrelevant problems? The question of who should attend neurological clinics cannot be answered from theoretical principles. We believe it is the system which decides: users' expectations, general practitioners' skills, working conditions and traditions and available resources together produce a net result in the patients we see. It is not clear which variables could be modified to change the pattern: lectures and "educative" discharge letters to general practitioners do not seem to have much effect and increasing or reducing outpatient hours in one English district only changed the number of referrals of the same unselected mix of patients.²

2) Wood *et al*¹ claim that waiting time was "unacceptably long". Here again we feel that acceptability is a relative concept. It would be unacceptable to waste precious time in which an early intervention would have made a prognostic difference, but that hardly seems to be the case in the type of patients seen, as indeed in most non-acute neurology. Once more, acceptability becomes "social acceptability". The British system seems to accept longer waiting times than ours but it would be difficult to prove that your patients are any the worse for it. As an example of the relativity of acceptance, it is often those patients who find waiting for two weeks unacceptable who present themselves with the most variegated arrays of functional symptoms.

3) We read that "there is a considerable shortfall in specialist neurological services in the United Kingdom".¹ Certainly the UK has fewer neurologists than many other countries and a large percentage of neurological problems are dealt with by other physicians. We do not know whether British neurologists generally believe that such problems are badly managed and in fact there seems to be a controversy on the subject.³ But it must be kept in mind that when the number of neurologists reaches a certain point, the question of which patients have to be managed by neurologists ceases to be debatable: neurologists have to deal with all patients traditionally considered neurological. That can considerably change the practice of neurology, with many neurologists becoming a "neurological general practitioner" whose activity largely involves seeing headaches and odd turns in outpatients and strokes on the ward. That can create its own problems such as professional frustration, loss of diagnostic vigilance,⁴ decline of diagnostic and therapeutic skills and a two-tier system with run-of-the-mill neurologists on one side and neurologists of excellence on the other. For many neurologists it may be the end of that seemingly enviable status in which the British neurologist can cope well with his quota of major neurological illness and is provided with "an interesting professional life in the course of which he sees several patients weekly with complex illnesses".⁵

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HLA antigens in familial Guillain-Barré syndrome

The Guillain-Barré syndrome is a sporadically-occurring acute demyelinating polyneuropathy. Its occurrence in first degree relatives is rare with only two reported examples^{1,2} one of which is doubtful.² The search for an association with histocompatibility class I and II antigens (HLA) has been largely unrewarding with no or weak associations.³⁻⁵ We report the occurrence of a Guillain-Barré syndrome in a father and son together with strikingly similar HLA types.

The father was admitted to hospital in 1981 at the age of 58 years one week after a "flu-like" illness. He had a one day history of increasing weakness in the limbs and paraesthesia in the hands, feet and tongue. The tendon reflexes were absent, vibration sense was absent in the feet and he had grade 3/5 weakness proximally in the limbs. The CSF contained protein 300 mg/l and 4 wbc/cmm: oligoclonal bands were present. Facial weakness and ventilatory failure evolved rapidly over three days and he required intermittent positive pressure respiration. He was treated with plasmapheresis. Thereafter, he gradually improved, was discharged two months later and made a complete recovery.

His son was admitted to hospital in 1990 at the age of 43 years two weeks after an upper respiratory infection. There was a five day history of paraesthesia and weakness in the limbs. The tendon reflexes were absent. The CSF protein was raised at 1945 mg/l and there were no oligoclonal bands reported. Nerve conduction studies showed slow conduction in keeping with an acute demyelinating neuropathy. He also deteriorated, was treated with plasmapheresis but did not require ventilatory support. He made a complete recovery over the next three months.

The HLA typing showed remarkably similar results in the father and son. Both had A2, A29, B5, B44, Bw4, Dr7, Drw53. The son's additional results were Dr6, Drw52 and Dqwl.

It is surprising that familial cases have not been described more frequently in a condition in which the immune responses to a variety of viral and other antigenic stimuli may be determined by genetic factors. The HLA types were identical as far as they have been analysed, and they do not include HLA-DR3 or HLA-DR2, both of which have been reported to show weak associations with the

Table New outpatient referrals in 1989

Diagnosis	Number
ORGANIC ILLNESS	337 (34%)
Mental disorders	9
Post-traumatic syndrome	6
Alcoholism	5
Endocrinopathies	4
Sleep disorders	4
Transient global amnesia	2
Degenerative and hereditary	
Tremor and abnormal movements	44
Parkinson's disease	33
Dementia	15
Mental retardation	15
Other degenerative conditions	11
Motor neuron disease	1
Intracranial masses	
Tumour	4
Hydrocephalus	3
Chronic subdural haematoma	1
Other disorders of central nervous system	
Epilepsy	52
Spinal cord disease	6
Multiple sclerosis	1
Disorders of peripheral nervous system	
Bell's palsy	21
Carpal tunnel and other mononeuropathies	20
Root lesions	19
Disorders of other cranial nerves	10
Neuropathy	8
Myopathy	2
Vascular disease	
Stroke	20
Transient ischaemic attack	20
Temporal arteritis	1
NON-ORGANIC ILLNESS	650 (66%)
Headache	265
Anxiety-depression	191
Dizziness-odd turns	56
Syncope	53
Other	69
No disorder	16