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Lecture

Brain, Mind and Behaviour
Some Medico-legal Aspects

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The advent of new imaging techniques is broadening our understanding of the major psychiatric illnesses. The increased knowledge of brain function will have consequences for the expert medical witness who has to give evidence in court. Both the insanity defence and the defence of automatism depend on disorders of the mind. Psychiatry is now able in many cases to produce evidence that these are consequent upon disorders of the brain. In presenting evidence in court there is an apparent conflict between 'brain words' and 'mind words'.

Psychiatry is changing in a significant way, as new imaging techniques are helping to answer questions about the relationship of brain to mind, consciousness and behaviour. Knowledge of the neurotransmitter changes which underpin pathological brain states is increasing, and it seems possible that before the end of the century much of the pathology of the major psychiatric illnesses will be understood. This will lead to a clearer understanding of the relationship between brain pathology and the impulsive behaviour that leads to violent crime, which will have important implications for the development of the law.

'Mens rea'

It is a fundamental tenet of English law that, providing the mind is sane and the person not under duress, everybody of the age of discretion is presumed responsible for his actions. *Actus non facit reum nisi mens sit rea* 'the deed does not make a man guilty unless his mind is guilty'. Unless the offence is a statutory one which carries an absolute liability (e.g. driving with a raised blood–alcohol level), the presence of a guilty mind can be negated by three major considerations (as well as the defences of duress and provocation). The first defence is that the mind is innocent, and does not know the difference between right and wrong. Mental subnormality can fall into this category, but is seldom used as a defence as there are mental health statutes which are preferable.

The second defence is that the mind is diseased. This is the defence of insanity, and is dependent on the McNaughton rules: that is, "at the time of the committing of the act the party accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing, or if he did know it, that he did not know he was doing what was wrong".

The third defence is that the mind is absent – 'automatism'. Here the law enters a quagmire into which, it has been said, only a man in desperate need of some sort of defence will venture. There are two types of automatism in law: sane and insane. Sane automatisms are due to external factors – a bee-sting on the arm causing a reflex action, concussion after a blow on the head impairing mental function, or hypoglycaemic confusion and bizarre behaviour following injected insulin, are all examples of automatisms due to external factors. Insane automatisms are due to an inherent internal cause. Cerebral tumour, arteriosclerosis, an islet-cell tumour of the pancreas, or epilepsy, are all internal factors which may lead to automatic behaviour in a confusional state. A defendant found not guilty due to sane automatism walks free from court, whereas a verdict of not guilty due to insane automatism, until recently, led to a mandatory referral to hospital, often a secure hospital (Fenwick, 1990*a*). However, since the Criminal Procedure (Insanity and Unfitness to Plead) Act 1991, there is no longer a mandatory requirement that a person found to be insane be admitted to hospital, as the judge can decide on disposal.

The distinction between sane and insane automatisms makes little medical sense. It is difficult to argue, for example, that the confusional state from the insulin secreted by an islet-cell tumour is any different from the confusional state produced by injected insulin. The reason for the distinction is clear. The lawyers are concerned with the protection of society, and therefore with the likelihood of recurrence of a violent act. The medical profession, however, is also concerned with the possible treatment of the patient so as to avoid recurrence,

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so the aetiology and pathology of the confusional episode are all-important.

Brain, mind and automatism

To reach its present position on insanity and automatism the law has had to refine the difference between brain and mind. It was easy to argue from the McNaughton rules that schizophrenic or depressed patients who were deluded or hallucinated did not know the nature and quality of their actions or, if they did, that they did not know what they were doing was wrong. It was clear that when violence was committed in these circumstances, the offender should be treated in a secure hospital. However, if violence were due to an organic process which temporarily distorted the functioning of the mind, the position was less clear. The case of R. v. Charlson (1955) illustrates this. The defendant was standing at a window of his house which overlooked a river. He called to his young son and pointed out to him a rat sitting on a stone. He then picked up a wooden mallet, struck his son several times on the head, and threw him through the window. Charlson then wandered out of the house and, when detained a few minutes later by a policeman, said he felt he had done something awful but could not remember what it was. Charlson was found to have a cerebral tumour, and was acquitted on the grounds of sane automatism; cerebral tumour is not a disease of the mind.

This was a clear statement that brain (tumour) and mind are separate. If this view had held, it would be theoretically possible to argue in court that automatisms arising from diseases of the brain were sane, and a sufficient defence. The advances in imaging would make such a defence not only easier but also, probably, popular. For example, it would be quite logical now for schizophrenia to be put into the category of 'diseases of the brain' rather that 'diseases of the mind'; if this were upheld in court, schizophrenic people committing violent acts would automatically have to be released.

Even more contentious would be the use of abnormal magnetic resonance imaging (MRI) scans to provide a defence of sane automatism in patients with minimal brain damage who showed impulsive and potentially dangerous behaviour. Clearly, as the ligands used in positron emission tomography (PET) scanning become more refined, receptor function will be more clearly demonstrated. As the mechanisms of behaviour become better understood, it may become apparent that many impulsive acts could depend on receptor changes and therefore result from disease of the brain. This would then allow the defence of sane automatism, leading to automatic acquittal. A recent article points to the dangers of over-interpreting imaging data in court (Mayberg, 1992).

The case of Kemp

This danger was foreseen in the case of R. v. Kemp (1957, 1956). Kemp seriously injured his wife, with whom he had an excellent relationship, in a motiveless attack with a hammer. It was accepted that when he attacked her he was in a confusional state due to arteriosclerosis, a disease of the brain. Kemp thus wished to plead sane automatism. Lord Justice Devlin disallowed this, saying that the proper plea was insane automatism. He said:

> "The law is not concerned with the brain but with the mind, in the sense that mind is ordinarily used, the mental faculties of reason, memory and understanding. If one read for 'disease of the mind' 'disease of the brain', it would follow that in many cases pleas of insanity would not be established because it could not be proved that the brain had been affected in any way, either by the degeneration of the cells or in any other way. In my judgement, the condition of the brain is irrelevant and so is the question of whether the condition of the mind is curable or incurable, transitory or permanent. There is no warranty for introducing these considerations into the definition in the McNaughton rules. Temporary insanity is sufficient to satisfy them. It does not matter whether it is incurable and permanent, or not."

This case raised two interesting points: the idea of treatability, and the question of length of time of impairment. Justice Devlin was very clear that both time and treatability were irrelevant, and did not affect the application of the McNaughton rules. Kemp was duly found insane.

The question of time was raised again in the case of Bratty v. A. G. for Northern Ireland (1963), and subsequently in R v. Sullivan (1983, 1984). Bratty, who strangled a girl and put her in the boot of his car, claimed that a 'terrible blackness' had come over him. The rather questionable defence of epilepsy was put forward, and the question of sane and insane automatism was again raised. At the trial Lord Denning said:

> "It seems to me that any mental disorder which has manifested itself in violence, is prone to recur, is a disease of the mind. At any rate, it is the sort of disease for which a person should be detained in hospital rather than given an unqualified acquittal."

In the case of Sullivan, who assaulted an 80-year-old man while he was in a confusional state during an epileptic seizure, a plea of sane automatism was disallowed. He appealed on the grounds that epilepsy

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was not a disease of the mind, and the appeal was taken to the House of Lords. In a definitive judgement they confirmed that epilepsy was a disease of the mind and that it did not matter how short the episode was; if the McNaughton rules were satisfied, then the plea must be one of insane automatism.

Time and mental state

How brief can a disturbance of brain function be if the transient alteration of behaviour which it causes is to be classified as an automatism within the McNaughton rules? In the field of epilepsy, it is possible to see gradations in the length of mental disturbance ranging from the postictal psychoses lasting for weeks, the postictal confusional states and the prodromal changes lasting for hours, the peri-ictal and ictal automatisms lasting for minutes, and the aura lasting for seconds. It is not difficult to accept that all these would be long enough to satisfy the House of Lords' ruling in the case of Sullivan. However, it is now possible to record cognitive changes which occur with each epileptic sharp wave (spike) by electroencephalography (EEG), even though that spike may only last between 70 and 250 milliseconds.

Binnie *et al* (1991) have carried out an elegant series of studies on patients with epilepsy showing such disruptions of consciousness during epileptic spikes, but in the absence of epileptic 'fits'. Videotape case studies carried out in Scandinavia have shown that this phenomenon is common, and is responsible for the disruption in learning in many children with epilepsy. These children have been shown to miss cues presented to them; even though they are not having epileptic seizures in the accepted sense of the word, their cognition is disturbed by single epileptic spikes.

Spikes on EEG are extremely common in a wide range of clinical conditions. They are seen, for example, in behaviourally disturbed children, patients with some brain damage, and patients with both depressive and schizophrenic illness, even at a time when their mental states are normal (Struve *et al*, 1977; Small, 1970). Work on the cognitive effects of these single EEG sharp waves has been limited mainly to epileptic populations; it is not yet known whether the spikes would be linked with cognitive change in other diagnostic groups, but it is safe to assume that they might.

Split-second insanity

The possibility that such momentary disruption of consciousness could affect behaviour and might lead to an offence presents an interesting legal dilemma. An experiment in Holland has demonstrated that such EEG spikes can cause a driver to swerve, though the driver may be quite unaware that they are occurring (Binnie *et al*, 1991). Should an accident occur in this situation, a defence of automatism would be appropriate. However, the driver feels only that he must have driven carelessly, but does not understand why. It might not occur to anybody concerned that he could have acted automatically and the McNaughton rules might be satisfied. As spikes, possibly with cognitive impairment, occur widely in different patient groups (Shagass, 1977), offences which arise owing to apparent deficits of attention may in fact be taking place during this 'split-second insanity' and may occur more widely than is realised.

Changing view of the spike

The concept underlying cortical spikes has also changed. Evidence from several fields suggests that they may be part of a generalised brain inhibitory mechanism and that they themselves are under strict regulation by different systems in other parts of the brain: for example, the nigral hippocampal system. Stimulation of the substantia nigra in animals reduces hippocampal firing. Accepted opinion is that cortical spikes are often a non-specific finding of little clinical importance. There is now good evidence that they may be very important (Binnie *et al*, 1991; Engel *et al*, 1991).

In the future forensic psychiatrists may well be expected to produce evidence in court on the effect of such spikes on brain activity. The infant science of magnetoencephalography could provide just such evidence. The advantage of measuring magnetic rather than electrical activity is that magnetic waves pass through the brain, cerebrospinal fluid (CSF), skull and scalp without attenuation, and thus theoretically enable deep source activity to be measured more accurately than by EEG. Current models of the EEG and its co-registration with other structural data are not yet sufficiently developed to allow precise calculation of activity in the depth of the brain. Magnetoencephalographic techniques can now provide precise information of where the spikes arise in the depth of the brain, and their surface fields. This information can suggest whether or not the spikes are likely to affect cognition, and if so, to what extent (Ricci, 1990; Sutherling & Bath, 1990; Vieth, 1990; Fenwick, 1990*b*).

The case of the absent-minded solicitor

A patient of the author, a solicitor, witnessed and signed an affidavit purporting to have been written

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by a man who did not actually exist. This was discovered, and the solicitor was charged with fraud. He was, however, known to have epilepsy. There was good reason to suspect that at the time the document was signed, transient cognitive impairment due to increased spike activity had led to a disturbance of consciousness. It was fortunate that he had that afternoon attended a physician for final confirmation that he had epilepsy, and thus his clinical state, with the presence of spikes recorded on an EEG, had been noted. The presence of split-second insanity was not tested in court despite good evidence to support it, as an MRI scan showed the man to have a left temporal indolent glioma, and this was put forward as the cause of his insanity. The jury failed to reach a verdict and the case was dropped. His epilepsy was subsequently controlled by drugs.

Attack on a painting

Paroxysmal discharges in the brain are often ignored, but a recent case again raises the question of their significance in impulsive crimes. Another patient of the author had a history of impulsive, aggressive behaviour. After a quarrel with his wife he entered hospital and was given 40–50 mg daily doses of diazepam. He discharged himself after a few days, and the following day bought a knife, and went to the local art gallery where he slashed a painting. He pleaded guilty and was sent to a medium secure unit for treatment.

An MRI scan of his brain showed him to have an enlarged right temporal horn, and the EEG showed frequent discharges of generalised spike and wave. Brain topography showed the spike and wave to be arising in his frontal poles. There is evidence that spike and wave activity can arise from focal lesions of the cingulate gyrus, and thus one can postulate that these discharges were interfering with his impulse control (Williamson *et al*, 1987).

The patient said that for a number of years he had contemplated slashing a painting to draw attention to his plight. So why did he choose this particular time? It could be postulated that spike and wave arising from the right frontal region (probably the cingulate area) were involved in his impulse disinhibition, and that this was augmented by the diazepam withdrawal.

Brain damage and neurochemical change

If it is accepted that the McNaughton rules apply at time-scales of 100 ms or so, then the fine structure of brain function – for example, what is happening at the synaptic level – may then become available as a basis for a defence. It takes some time for mind (i.e. consciousness) to arise, in the order of 400–500 ms (Libet, 1993). It could, therefore, be argued that at these shorter time scales mind is no longer involved, as the act would have taken place before it reached consciousness. If it can be shown that abnormal bursts of electrical activity precede violent acts, then it could be argued that the person should not be held responsible, and that a defence of insanity or of insane automatism should be allowed. Judges have long rejected the concept of the irresistible impulse; the time may be approaching, however, when a defence of insanity due to an 'unconscious' (and therefore irresistible) impulse is again respectable.

Sleepwalking and disease of the mind

A recent extension of the McNaughton rules introduces sleepwalking into the area of insanity. Until July 1989, sleepwalking was not held to be a disease of the mind (Fenwick, 1986*b*, 1987). It was considered to be automatism *simpliciter*, or sane automatism. If the plea were successful this led to an acquittal, so it was an attractive and increasingly popular defence. In 1989 in Bristol Crown Court, Burgess was accused of unlawful wounding. He claimed that while asleep he had picked up a bottle and broken it over the head of his sleeping girlfriend (R. v. Burgess, 1991). Still asleep, he had then gone behind the sofa, unplugged the video machine, and hit her over the head with it, cutting her and severely denting the video. She, somewhat dazed, tried to escape, whereupon – still asleep, he claimed – he came round the sofa, caught her as she crawled away, put his knee on her chest, his hands around her throat, and attempted to strangle her. He did not stop until with great presence of mind she managed to say: "I love you, Bar".

Burgess wished to plead sane automatism, but the prosecution argued that sleepwalking had a genetic cause, arose from a specific stage of sleep (stage 4), and that violence during sleepwalking, if it occurred, tended to be recurrent. It therefore fulfilled all the criteria of an insane automatism. Judge Lewis ruled that sane automatism was therefore not available to Burgess, and the Appeal Court agreed and dismissed the appeal. They argued that on the medical evidence presented, sleepwalking was a disease of the mind (R. v. Burgess, 1991).

It is interesting that Canadian law takes a different view. In the case of R. v. Parker (1992) it was ruled at appeal that sleepwalking is a sane automatism and, in brief, the decision turned on the point at which the *mens rea* is lost. In British law the *mens rea* is

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lost with the onset of the sleepwalking episode – which is an abnormal event. The Canadian view is that *mens rea* is lost with the loss of consciousness at sleep onset – which is a normal event. Thus, the rather tortuous Canadian legal view is that sleepwalking is normal and a sane automatism.

Diminished responsibility

The defence of diminished responsibility, which allows a verdict of manslaughter, has implications for neuropsychiatrists. In order to satisfy the legal requirements for diminished responsibility, the Homicide Act 1957 states that at the time the crime was committed the defendant was suffering from "such abnormality of mind (whether arising from a condition of arrested or retarded development of mind or any inherent causes, or induced by disease or injury) as substantially impaired his mental responsibility for his acts and omissions in doing or being party to the killing". Here again, the law is quite specific. It is the mind that has to be disordered rather than the brain. The Act might also apply to an offence committed during a spike discharge (as in split-second insanity) which apparently does not involve the mind but does involve the brain.

Abnormal imaging – abnormal behaviour

Recent data from PET and single photon emission computerised tomography (SPECT) confirm our previous neurophysiological understanding of the brain. Scanning has shown increased blood flow in those brain areas where, on the basis of both animal and human work, this would be predicted.

There is accumulating evidence that changes resulting from pathology demonstrated by both the SPECT/PET and the MRI can affect cognitive functioning. The evidence also suggests that it is possible to infer, from the location of the lesion, the quality and extent of the cognitive change. It is beyond the scope of this article to review this field in detail, but three recent studies should be mentioned. First, studies show that SPECT scanning in patients with cerebral pathology demonstrates areas of hypoperfusion which correlate highly with the established neuropsychometric deficit (Besson *et al*, 1990; Miller *et al*, 1991). Second, a study by Eisenberg & Levin (1989) showed that mild head injury is more easily visualised by MRI scans than computerised tomography (CT) scans, and also that there was a correlation between neuropsychological deficit and MRI scan pathology. The third is a recent PET scan study which has shown that patients who have medial lesions in the thalamus on CT scanning are likely to show frontal hypoperfusion. The hypoperfusion is usually ipsilateral, but is greatest and bifrontal in the case of bilateral thalamic lesions (Szelies *et al*, 1991).

These three studies indicate that lesions in the MRI and SPECT/PET scans should be taken seriously and not ignored as being 'non-specific'. Lesions in the thalamus, frontal or temporal region may be expected to contribute to some extent to impulsive criminal behaviour, and MRI, CT and SPECT/PET scans should be examined in detail. A normal report does not necessarily mean that the scan is normal. A dilated temporal horn which is within normal neuroradiological variation may be highly significant when combined with the clinical, neuropsychometric, and EEG data which all point to damage to that temporal lobe.

Abnormal chemicals – abnormal behaviour

Virkkunen (1992) supports the view that low serotonin levels lead to increased impulsiveness in man. He has shown, with others in his group, that low CSF levels of the metabolite of serotonin, 5-hydroxyindoleacetic acid (5-HIAA), are found in offenders who have carried out violent crimes, some of whom show excessive sensitivity to alcohol.

Depressive illness, which is known to produce impulsive behaviour, leads to a reduction in the turnover of serotonin (5-HT) in the brain. In animal models, 5-HT is known to be involved with aggression (Olivier & Moss, 1990). Low 5-HT in rats will lead to an increase in mouse-killing behaviour (Eichelman, 1987). In man, there is evidence from several studies that low CSF 5-HT levels are correlated with increased aggressiveness (Eichelman, 1990). There is also some evidence that brain 5-HT levels are lower in people with depressive illnesses who are self-violent and commit suicide (Traskman-Bendz, 1990).

The relationship between low serotonin turnover and impulsive and aggressive behaviour must raise questions about our current concepts of responsibility.

Impulsive behaviour – a guilty brain or a guilty mind?

When dealing with abnormalities of brain structure and function it is almost impossible to draw a distinction between brain and mind. The following cases illustrate this. In each, the defendant had had no previous history of violence before the assault, which in every case was frenzied, violent, and quite out of character. In each case the explanation

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probably lay more in the abnormal physiology of a damaged brain than in the wilful mind of the defendant.

The case of the gentle schoolboy

J was an adolescent boy of good character, who made a vicious and unprovoked attack on the 18-year-old daughter of a neighbour. Shortly before the attack he had fallen over, felt faint, and leaned against a gate. He had a penknife with an open blade in his back pocket. Just as the girl was about to walk past him he was aware that his mind had "gone out of control". He was unable to think straight, and was impelled to run towards the girl while filled with an intense feeling of anger. He caught and held her, stabbing her several times in the neck and chest. She cried out: "J, stop it, stop it!" He pushed her to the ground, raised her head and then hit it on the ground. At this point his mind "suddenly snapped back" and he realised what he had done. He helped his victim to the side of the road, made her comfortable, then ran to her parents' home to summon help. She was taken to hospital and fortunately suffered no lasting harm.

Psychometric testing showed J to be of above-average intelligence with no clear evidence of a focal cognitive deficit. Physical examination revealed a raised right eye (indicating a small right temporal fossa and thus prenatal or early-childhood damage to the right temporal lobe). A CT scan showed an enlarged right temporal horn, suggestive of mesial temporal damage. (MRI scan was not then available.) EEG examination showed abnormal activity over the right hemisphere, which was maximal in the right temporal leads. Sleep recording and methohexitone activation with sphenoidal leads confirmed these abnormalities but added no new information. No epileptic spikes were seen. Psychiatric examination showed J to be a normal adolescent with no history of aggressive behaviour and no abnormalities in his mental state or in his fantasy life.

J pleaded guilty and the judge ruled that he should be put under medical care. With hindsight, a defence of 'not guilty due to insane automatism' might perhaps have been considered. It is probable that at the time of the attack the cerebral activity within J's right limbic system was disordered and this caused a behavioural change. If so, the inference is that J should have come within the McNaughton rules at the time of his act: a guilty brain rather than a guilty mind (Fenwick, 1986*a*).

The case of the respectable housewife

Another example is that of Mrs D, a respectable, middle-aged housewife without a forensic history, who enjoyed gambling with a friend. The friend was one day found dead after a frenzied attack. Her skull had been fractured. She also had 30 stab wounds from a kitchen knife and a scarf tied tightly round her neck. Her killer evidently wished to leave nothing to chance. Mrs D said she was out shopping at the time of the murder and could remember nothing. However, the circumstantial evidence against her was overwhelming and she was charged with murder.

Clinical examination showed Mrs D to have a raised right eye. MRI scan revealed a mildly dilated right temporal horn, some changes in the right frontal lobe, and possibly increased atrophy of the cortical sulci frontally. Neuropsychological deficits pointed to right hemisphere dysfunction; the routine and sleep EEGs were normal. A sphenoidal examination showed spike discharges at both sphenoidal leads, and foramen ovale electrodes showed abnormal discharges arising from the right foramen ovale lead. (Foramen ovale leads are electrodes which are passed through the foramen ovale into the cranial cavity. They come to lie alongside the medial surface of the temporal lobe in close proximity to the amygdala and hippocampus. This is a standard technique for monitoring the activity of the limbic structures in patients being investigated for possible temporal lobe surgery. It carries a very low morbidity in experienced units (Wieser *et al*, 1985).) This abnormal activity was arising from the area of the right medial temporal structures. There was thus clear evidence of a brain lesion in the very structures which control aggression. Her plea of diminished responsibility was accepted by the Crown.

Three years after the offence Mrs D was beginning to show abnormalities of frontal lobe functioning. A SPECT scan showed a marked reduction in flow in the right parietal region. Taking all the evidence together, she clearly had right hemisphere and right limbic damage at the time of the murder. Presumably this damage led to impaired impulse control, and triggered the vicious attack. Again it seems that this is a case of a guilty brain rather than a guilty mind – but without brain imaging and implanted electrodes her brain damage would never have been discovered, and a plea of diminished responsibility could not have been considered.

The case of the miserable teenager

It is argued by neurologists and by neurophysiologists that spikes, both on the cortex and in the depth, may have no pathological significance. Although this statement is unobjectionable, one must be aware of double standards, for both the neurologist and the neurophysiologist will use the presence of these very same spikes to confirm pathology.

M, an 18-year-old youth with a blameless background and personality, was eating fish and chips in a club he frequently visited. When the barmaid asked him not to, he set about her in a vicious and uncontrolled way, hit her repeatedly on the head, and strangled her. Everyone who examined and interviewed him agreed that the provocation was minimal and that the attack was impulsive and frenzied.

For three months before the attack M had been under stress; he was being persecuted by a local gang of youths, who had repeatedly threatened and on one occasion chased him. He became depressed and suicidal.

Clinical examination showed him to be left-handed with a raised left eye, and to have poor coordination with his right hand. A CT scan showed a small left temporal lobe with a dilated left temporal horn and enlarged air sinuses on the left, suggesting early brain damage, predominantly to the left hemisphere, and maximal temporally. EEG showed some cortical spiking bitemporally, but more

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frequently on the left. Neuropsychometry showed a 19-point verbal/performance deficit with reduced performance measures. The reduced growth of his left temporal lobe suggested damage to this area at an early age. It is thus likely that speech had moved from his left hemisphere to colonise his right hemisphere, with a subsequent reduction in performance IQ. There is thus evidence from several sources to indicate an early damaged left hemisphere.

The neuropsychiatric picture suggests the following probable explanation. There was evidence that in the weeks before the offence M had become increasingly violent and impulsive to himself (often hitting his own head against a wall) as his depression deepened. It seems likely that there was an interaction between his impulsiveness (low serotonin turnover) and the brain damage, which led to reduced control at the time of the attack. Presumably damage to his amygdala was one of the major factors involved. To see whether the structures that mediate aggression and impulse control were indeed damaged, the defence asked for foramen ovale electrodes to be implanted. The intracranial depth EEG recorded showed a disordered pattern from the left amygdala and hippocampus, suggesting damage in these structures. Brain damage to the left hemisphere and left limbic system interacting with his depressive illness was put forward as the basis for a defence of diminished responsibility.

The prosecution, however, took the view that M was a normal adolescent and that he was entirely responsible for the attack. Unfortunately, the jury agreed with the prosecution's view, which perhaps reflects the difficulty laymen are bound to have in understanding complex neuropsychiatric evidence.

The case of the abused wife

This final case illustrates another difficulty that is likely to become more widespread as our understanding of brain function progresses and new imaging techniques become more widely available. This difficulty concerns the differences and similarities between 'mind words' and 'brain words' and whether or not they are equivalent descriptions of brain function.

Mrs R was the middle-aged mother of two children. She had a good previous personality and no previous convictions. She had been married for 15 years to an alcoholic husband who had seldom worked, and often physically abused her. She had to provide the money for the family and was also responsible for running the house and caring for the children. In the weeks before the assault she had become depressed, with the bills mounting, the roof leaking, and her husband lying in bed all day and often drunk. On the day of the murder, he had sent her out to steal a bottle of spirits for him. She had been caught, and was mortified by the charges which were to be brought. That night he went out and did not come home for his supper. When he finally returned, he slumped in a chair, drunk. She took a hammer and brutally and violently hammered him to death.

Clinical examination of the defendant was normal, apart from a mildly raised blood pressure. The resting EEG was abnormal and in response to alcohol there was a marked increase in spiking. Neuropsychometry showed a significantly poor visual memory, suggesting right limbic dysfunction, and an overall verbal intelligence of only 76. MRI scan showed white-matter lesions (probably ischaemic) in her frontal lobes and the parietal region of the right hemisphere. There was also a large lesion in her right putamen, which raised the question of frontal hypoperfusion and the possibility of a frontal lobe syndrome. (As already mentioned, there is a close connection between thalamic damage and frontal blood flow. On the basis of current data, however, the putamen is possibly too anterior for an alteration in frontal perfusion to occur. Her frontal perfusion was not checked by SPECT scanning, which would have settled the matter.)

One report for the defence suggested a possible explanation using 'brain words'. As she had survived the marriage for 15 years, and it had not changed in any significant way, some new factors must have been involved. Her cerebral function had deteriorated, with a recently acquired abnormality of the right hemisphere, compromised right thalamic and possibly right frontal functioning. There was also evidence of right limbic damage. The abnormal cortical spiking with alcohol suggested possible diffuse vascular pathology. She had become depressed over a number of months, which led to a reduction in 5-HT levels and thus to an increase in impulsiveness. On the night in question the interaction of her reduced impulse control, her damaged brain, and the anger she felt at the events of that particular day led to an impulsive and violent killing.

An alternative report for the defence questioned the view that the pathophysiological factors found would of necessity lead to a violent act. The report was written using 'mind words', thus over the past few months Mrs R had felt sad, and become depressed. She was at the end of her tether due to her husband's beatings and her deteriorating social circumstances. She was particularly distressed and feeling guilty (due to the shoplifting episode) on the evening in question and went violently out of control when her husband came home late and drunk. This was the culmination of many years of frustration.

It is fashionable at present to produce 'mind word' descriptions in court, as if they have a validity that 'brain words' do not have. But is it fair to say that 'mind words', which describe how feelings may influence behaviour, are any more accurate and precise than 'brain words', which describe how brain pathology may influence behaviour?

In the case of Mrs R, can it be said with certainty that her feelings of sadness and guilt led to violence? Why did violence occur on this occasion when she had been abused and miserable for so many years? How can accuracy be claimed for these 'mind words'? Indeed, it could be argued that the 'brain word' description, containing as it did the relationship between structure and function, and based on scientific observation, was the more precise. There is obviously a conflict at present between 'mind words' and 'brain words'.

Guilty mind or guilty brain?

A display of uncontrolled and uncharacteristic anger following minimal provocation can be explained in

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brain words – a decrease in brain serotonin manifesting as a depression of mood and interacting with a disordered hippocampal amygdala system in a damaged brain. Or it can be explained in mind words – he felt sad and depressed, did not understand why he got so angry, and is sorry for all the trouble he caused. At first glance, brain words seem less accessible. But it can be argued that they give not only a more precise but also a more useful description of what happened, because they enable the court to judge blameworthiness.

Virkkunen (1992) examines the idea of blameworthiness and its relationship to low serotonin turnover in the brain, with consequent poor impulse control and/or disorders of aggression control (van Praag, 1991). He says:

> "These concepts can be related to free will, if by this we mean the person's ability to control himself, and consequently they could be thought of as related to the idea of diminished responsibility. It will be important for forensic psychiatry in the future to make decisions concerning, for example, diminished responsibility and dangerousness, not only on descriptive criteria, personality disorder and psychological tests, but also using biological tests."

Miller (1990) argues that forensic psychiatry has forged an essential place in our legal system, and that "forensic psychiatry is flourising". He also suggests that forensic psychiatry should be part of the general American psychiatric training programme, something which is already happening in this country. However, the next step is for forensic psychiatric training to be broadened to include neuropsychiatry. The psychiatrist should, of course, base his opinion on the clinical facts of the case, but where necessary he must now be prepared to use brain words rather than mind words. Where there is evidence of brain abnormality, it may be necessary for the forensic psychiatrist to take into court the defendant's MRI scan, SPECT scan, sleeping and waking EEG, and full neuropsychometry, as appropriate. The argument against this is usually one of cost, but in cases of homicide, where the defendant stands to receive a life sentence, detailed investigation of brain function is surely justified.

Mayberg (1992) criticises the use of these investigations in Court on the grounds that a demonstrated lesion cannot predict particular behaviours. She points out that PET and SPECT scans are used to make a diagnosis of an illness and not to predict a certain behaviour. This line of argument is an extension of the mind/brain word conflict. Who can say that feelings of depression, or deprivation in childhood will lead to particular behaviours? It is clear that the new investigations must be put in a clinical setting, and when interpreted in that situation I would suggest that brain words can be more precise than mind words.

Conclusion

The cases discussed above raise interesting questions about the nature of criminal responsibility in the light of the growth in scientific knowledge of brain function. To recapitulate: in order to be found guilty a person must have a 'guilty mind'. In other words, he must know the nature and quality of his actions and that these actions are wrong. It seems clear that, first, in the field of automatism, and second, in the area of diminished responsibility, the concept of a guilty mind belongs to a non-scientific era. There are clearly occasions when an act carried out will depend on a brain malfunction of which the person is not aware, or will depend on a brain malfunction of which he or she is aware, but cannot control (although this may not be understood at the time). As knowledge of brain functioning increases and imaging facilities become more available, it will become easier to detect minor degrees of brain malfunction, and the usefulness of the concept of *mens rea*, the guilty mind, may diminish even further.

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