

**National Judicial College of Australia  
Conference on the Australian Justice System in 2020  
Sydney, Saturday 25 October 2008**

**Criminal law as it pertains to 'mentally incompetent defendants':  
an M'Naghten Rule in the light of cognitive neuroscience**

**By**

**Maxwell Bennett AO**

**Professor of Neuroscience**

**University Chair**

**Scientific Director, *Brain and Mind Research Institute*,**

**University of Sydney**

## 1. The M’Naghten Rules and cognitive neuroscience

### 1.1 The M’Naghten Rules

Daniel M’Naghten shot the secretary of the Prime Minister of England Sir Robert Peel in 1843. The trial was held before Lord Justice Nicholas Tindal (1776-1846) and the advocate briefed on behalf of the assassin was Sir Alexander Cockburn (1802-1880), later Lord Chief Justice of England<sup>1</sup>. Cockburn made effective and extensive use of the recently published (1838) work *A Treatise on the Medical Jurisprudence of Insanity*<sup>2</sup> by the celebrated American psychiatrist and founder of mental health institutions, Isaac Ray. Cockburn was then able to shift the plea of insanity made on the basis of the defendant being unable to tell right from wrong to other considerations given in Ray’s treatise. The treatise is based on the thought that:

*‘In all of this, however, there is nothing different from what occurs in many, if not the greater proportion of chronic diseases. That the intermissions of mania are ever so complete that the mind is restored to its original integrity, would seem scarcely probable, from the fact, that the very seat of the pathological changes is the material organ on which the manifestations of the mental phenomena depend. For if the mind be rendered as sound as before the attack, it necessarily follows that the brain is equally restored, since in point of health they stand to each other in the relation of cause and effect ([2]; p.321).’*

Cockburn took the following stance in his defence of M’Naghten<sup>3</sup>:

*‘It was now placed beyond doubt that madness was a disease of the body, the result of morbid organization, and that its nature was to be precisely and accurately ascertained by those only who had made this disease and its pathology the object of long reflection and diligent investigation. The discoveries of modern science had thrown much light upon this subject, and many of the positions laid down by Lord Hale and the other authorities of former times were left to very great objection and doubt ([3]; p.2).’*

Cockburn undertook to prove to the jury that the prisoner:

*‘was the creature of delusion and uncontrollable impulse, which took away from him the character of a responsible being ([3]; p.2)’*

After M’Naghten was found not guilty on the grounds of insanity the House of Lords began an enquiry into considerations of murder evidence in insanity. In particular the Lords put a series of questions to a panel of judges headed by Lord Tindal concerning the course to take when a defendant claimed to have committed the crimes whilst insane. Lord Tindal’s report, the foundation of the law of insanity throughout the English common law world, is indebted to Isaac Ray, as is a later addition that received the enthusiastic support of Lord Cockburn.

The report states<sup>4</sup> (the designations in square brackets are mine):

*it must be clearly proved that, 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,*

*[T1] as not to know the nature and quality of the act he was doing;*

*[T2] or, if he did know it, that he did not know he was doing what was wrong.*

To which he added a further consideration:

*[T3] that he labours under such partial delusion only, and is not in other respects insane, we think he must be considered in the same situation as to responsibility as if the facts with respect to which the delusion exists were real.*

Later Cockburn, when Lord Chief Justice of England, sent a memorandum to the chair of the select committee of the House of Commons on the homicide law amendment bill which reinforced a further far-reaching consideration in relation to the law of insanity, and one which was used in the defence of M'Naghten, as follows:

*[C] there are forms of mental disease in which, though the patient is aware he is about to do wrong, the will becomes overpowered by the force of irresistible impulse, the power of self-control, when destroyed or suspended by mental disease, becomes (I think) an essential element of responsibility.'*

Isaac Ray's ideas form part of the foundation on which the Tindal/Cockburn rules were later formulated. In relation to [T1], Ray had suggested that:

*'Intellectual mania is characterized by certain hallucinations in which the patient is impressed with the reality of facts or events that have never occurred, and acts more or less in accordance with such beliefs; or having adopted some notion not all together unfounded, carries it to an extravagant and absurd extent. It may be general, involving all or the most of the operations of the understanding; or partial, being confined to a particular idea, or train of ideas ([2]; p.152).' (for) the senses have no share; the imagination alone is exalted; the brain is exclusively the seat of the disturbance; the patient mistaking creations of his imagination, for objects actually present to his senses ([2]; p.154).'*

In relation to the issue raised in [T3], Ray had previously commented that:

*'When a man suffers under a partial derangement of intellect, and on one point only, it would be unjust to invalidate acts which were totally distinct from, and uninfluenced by this so-limited insanity; but if the act done bears a strict and evident reference to the existing mental delusion, we cannot see why the law should not also interpose a limited protection, and still less why courts of equity, which in their ordinary jurisdiction relieve against mistake, should deny their aid in such cases([2]; p. 245).'*

And in relation to [C] Ray had made the point that:

*'The various forms of homicidal insanity have thus been illustrated, by selecting a few cases only, from a mass that would fill a considerable volume. Now, however these cases may differ from one another; whether the individual has succumbed to the propensity to kill, after a long struggle with his better nature, or has yielded to it at once and instantaneously; whether harassed by previous disease of body or despondency of mind, or apparently in sound health and with a cheerful disposition; whether his passions have been tamed by the discipline of a good education, or allowed to seek their gratification without restraint; they all possess one feature in common, the irresistible, motiveless impulse to destroy life ([2]; p.229).'*

The Tindal/Cockburn guide to the law of insanity has been adopted and extended to different degrees in the United States of America and Australia in more recent times, but with very little if any reference to the progress made in our understanding of the workings of the human brain since Ray's thesis that so influenced the formulation of the guide.

In 1972 about half of the United States adhered to a rule, The Model Penal Code<sup>5</sup>:  
The legally insane is so damaged that he lacks 'substantial capacity either  
*[T1] to appreciate the criminality of her conduct or*  
*[C] to conform her conduct to the requirements of the law'.*

A Federal Rule<sup>6</sup> was developed in 1984 which states that a defendant is required to prove,  
by 'clear and convincing evidence' that 'at the time of the commission of the acts constituting the offence, the defendant, as a result of a severe mental disease or defect, was unable to appreciate the  
*[T1] nature and quality or*  
*[T2] the wrongfulness of the acts.'*

In Australia The Law Reform Commission of Western Australia reported that the commission accepts the 'question whether a person suffering from mental disorder is to be held criminally responsible for an offence is governed by section 27 of the Criminal Code, which provides that<sup>7</sup>:  
A person is not criminally responsible for an act or omission if at the time of doing the act or making the omission he is in such a state of mental disease or natural mental infirmity  
*[T1] as to deprive him of capacity to understand what he is doing,*  
*[C] or of capacity to control his actions,*  
*[T2] or of capacity to know that he ought not to do the act or make the omission.'*  
*[T3] A person whose mind, at the time of his doing or omitting to do an act, is affected by delusions on some specific matter or matters, but who is not otherwise entitled to the benefit of the foregoing provisions of this section, is criminally responsible for the act or omission to the same extent as if the real state of things had been such as he was induced by the delusions to believe to exist.'*

They go on to comment that: 'Section 27 of the Criminal Code provides three independent tests for the defence of insanity (2.14):

- 1.*[T1] lack of capacity to understand what one is doing;*
- 2.*[T2] lack of capacity to know that what one is doing is wrong; and*
- 3.*[C] lack of capacity to control one's actions.'*

More recently (2005) Justice David Kirby of the NSW Supreme Court explained the mental illness defence as given by the M'Naghten Rules, in relation to the case involving Trent Jennings, who had taken methamphetamine and committed a murder, as<sup>8</sup>:

' The McNaghten rule has two limbs. The accused must demonstrate either that he was labouring under such a defect or reason, from a disease of the mind,

*[T1] that he did not know the nature and quality of his act*

*[T2] or, alternatively, if he did know, then he did not know that what he was doing was wrong.*

The first limb, in lay terms, requires that the accused did not know what he was doing, whereas the second deals with his appreciation of the morality of what he was doing, that is, whether it was right or wrong.' Here [T1] and [T2] are precisely the words used by Lord Tindal 162 years earlier.

## **1.2 Consideration of one of the M'Naghten's Rules in the light of cognitive neuroscience**

The lack of understanding of what one is doing [T1], arises under the condition of a delusion or of a hallucination, which I have recently discussed in relation to the associated neural mechanisms<sup>9</sup>. This is not considered further here. The lack of capacity to know that what one is doing is wrong [T2] and the extent of criminality under a delusion [T3], are not considered here either. It is to [C] that this short essay is principally addressed and in doing so, I hope, makes obvious that much work needs to be done concerning the extent to which contemporary cognitive neuroscience bears on issues in the 'Medical Jurisprudence of Insanity'. For as Ray comments in his great work of that name 170 years ago ([2]; see, Preliminary Views, p.3):

*'It would seem, therefore, an almost self-evident proposition, that a certain knowledge of the mind in its healthy state is an essential preliminary, to the attainment of correct ideas concerning its diseased manifestations. If, in addition to this, it is considered, that opinions on the nature of insanity, viewed solely in the light of a disease – of a derangement of the physical structure, - have been constantly changing for better, it follows of course, that its legal relations, which should be determined in some measure by our views of its nature, ought to be modified by the progress of our knowledge. That much of jurisprudence of insanity, in times past, should bear marks of the crude and imperfect notions, that have been entertained of its pathological character, is not to be wondered at; but, it is a matter of surprise, that it should be adhered to, as if consecrated by age, long after it has ceased to be supported by the results of more extensive and better conducted inquiries. It is to be feared, that principles, laid down on this subject by legal authorities, have received too much of that reverence, which is naturally felt for the opinions and practices of our ancestors; and that innovations have been too much regarded, rather as the offspring of new fangled theories, than of the steady advancement of medical science.'*

## **2. Thinking about a future act but unable to inhibit an ongoing act and so initiate the new act**

### **2.1 Apparent lack of self-control and the pre-supplementary motor area of the brain.**

Patients, with lesions located by non-invasive magnetic resonance imaging in the pre-supplementary motor area (pSMA) of the brain, may display behaviour in which they have great difficulty in resisting picking up and

using the nearest object to them, showing an apparent lack of self-control in these instances<sup>10</sup>, a condition called Utilization Behaviour (for a review, see [11]). Such behaviour is manifest when, for instance, the patient who is already wearing glasses picks up another pair of glasses nearest to them on a table and proceeds to put these on over the existing pair. The absurdity of the situation is apparent to the patient; nevertheless they find it very difficult to resist utilizing the second pair of glasses.

A related phenomenon is Anarchic Limb, which occurs in patients with lesions encompassing pSMA as determined by magnetic resonance imaging (for a review, see [11]). In this case, with a lesion on one side of the brain, the patient will tend to firmly grasp objects and even people with the opposite hand, experiencing great difficulty in releasing them<sup>12</sup>. Again the patient generally realizes the inappropriateness of the act but is unable to resist carrying it out, feeling 'magnetically drawn' to the object or person.

Thus the propensity of some subjects to fail in restraining themselves from inappropriate acts and to initiate appropriate acts, even though recognizing what is appropriate and attempting to act on such thoughts, can be correlated with brain lesions, particularly those that involve the pSMA. This brain area has therefore been singled out for detailed investigations, which we now turn to (§2.2 and §2.3) before considering further effects of lesions in pSMA (§2.4) and loss of synaptic connections in pSMA consequent on a disease such as schizophrenia (§2.5).

## **2.2 In normal subjects the pre-supplementary area functions during preparation to carry out an act.**

A normal subject is instructed, on hearing one of two tones (each at a different frequency), to either extend or not to extend their middle finger on hearing a further tone some 2 seconds later (at a third frequency). In the intervening 2 seconds, at about 0.1 seconds immediately following the instruction, considerable electrical activity is recorded in the pSMA peaking at about 0.6 seconds. There is however very little activity in the adjacent cortical area<sup>13</sup>. Electrical activity is not evident at all if the experiment is reversed and the initial tone warns the subject that 2 seconds later they will be presented with one of two tones indicating that they should move or not move their middle finger. These observations suggest that pSMA is concerned with supporting our capacity to select an appropriate action set, that is, a set of neurons that are necessary to carry out an action during the preparatory period before the act is implemented<sup>13</sup>.

Such an interpretation is supported by functional magnetic resonance imaging (fMRI) studies of pSMA in normal subjects executing finger movements. The subject is asked to either execute a finger movement at 0 seconds or to think of executing the movement without doing so while counting down from 10 to 0 seconds and then up to 5 seconds. The fMRI shows greatly increased activity in the pSMA during the 10 seconds before executing the act, but only a relatively small or no increase in the adjacent cortex; similar results are obtained when the act is not executed, but simply imagined (see [14]).

Taken together, these observations indicate that pSMA is involved during the preparatory phase of carrying out an act, that is in engaging the appropriate neurons required in order for the act to be subsequently implemented.

### **2.3 In normal subjects pre-supplementary area functions during switching from one act to another.**

Giving a normal subject the task of moving, on instruction, their eyes in a particular direction (a saccade), and then subsequently instructing them to reverse the direction of the saccade before it has occurred, gives rise to heightened activity in pSMA, closest to the front of the brain as determined using fMRI<sup>15</sup>. Such heightened activity does not occur unless the original instruction is reversed<sup>15</sup>. It seems then that rostral pSMA is active when a subject is involved in resolving a conflict of actions.

Electrical observations support the conclusion that pSMA is involved in inhibiting one act and establishing another. Nerve cells can be recorded from in pSMA that fire impulses at high frequency when instructions require a sudden switch from one kind of activity to another<sup>16</sup>. For example, a screen might be viewed with two different coloured squares on it, one to the left and the other to the right of a central fixation point, the instruction being to make a saccade to one or other of the colour squares, depending on a matching colour to one of them presented at the fixation point. Following a series of trials during which the requested colour matching occurs at random to the left or right of the fixation point a sudden switch is made in the colour at the fixation point so requiring that the other colour should be matched. Nerve cells in the pSMA begin heightened firing about 0.015 seconds later, reaching a maximum at 0.25 seconds and falling to low levels at 4 seconds, which is about 0.1 seconds before the correct saccade commences<sup>16</sup>. If a mistake in the directions of the saccade is made, the pSMA neurons only shows a low and delayed increase in activity before the saccade commences, with no increase in firing occurring at all during trials when no switch to a new colour to be matched occurs<sup>16</sup>. Clearly, the activity of these neurons reflects a successful switch in the saccade before this commences as well as the failure of a correct saccade at switching. It is therefore consistent with attributing to pSMA a function related to a subject being able to quickly and successfully resolve changes in required action.

### **2.4 Subjects with lesions in pre-supplementary area have significantly decreased ability to inhibit one act and switch to another**

A small injury to the pSMA leads to an incapacity to carry out an appropriate action. This area of the brain must function normally in order for a set of nerve cells (the action set) to be chosen over that of another action set in order to efficaciously fulfil a particular act at a subsequent time (see [17] for a short review). This is revealed in patients with highly localized lesions to their pSMA. If such patients are given instructions to carry out an action, such as pushing a button with the left hand, and then told to countermand this and to push it with the right hand, they have difficulty in doing so and although eventually successful, the act is clearly slow and somewhat laborious<sup>18</sup>.

Injury to pSMA leads to a high probability of failure to change direction in a saccade when instructed to do so shortly after the original saccade is begun<sup>19</sup>. If transcranial magnetic stimulation, in which the field from the magnetic coils is used to interrupt electrical activity in the cortex, is applied over the pSMA, then switching is disturbed between cued right- and left-handed responses but does not interfere with on-going responses that do not require switching, nor does it affect switching when the field is applied elsewhere over the frontal cortex<sup>20</sup>.

## **2.5 Subjects with decreased gray matter (synapses) in pre-supplementary area have significantly decreased capacity for switching to another act**

It is not necessary for a small lesion to disturb the normal functioning of pSMA as a decrease in gray matter from the normal can also lead to errors in switching. The relative lack of self-control in subjects with different schizophrenia-related personality disorders and the volume of gray matter in the pSMA as determined by magnetic resonance imaging are negatively correlated<sup>21</sup>. The extent of disorganized behaviour in chronic schizophrenia patients is also negatively correlated with gray matter volume in areas next to pSMA<sup>22</sup>. Given the substantial evidence now available that changes in gray matter volume during development are most likely due to changes in numbers of synaptic connections (reviewed in [23]), it is very likely that the above observations indicate that loss of synapses in pSMA leads to an inability of the subjects to easily switch between motor acts and avoid socially unacceptable behaviour.

We have considered in this section (§2) a spectrum of behaviours ranging from overtly bizarre acts as in Utilization Behaviour through to difficulties in switching hand and eye movements, associated with lesions of different sizes in pSMA, to conditions in which there are no lesions but a likely loss of synaptic connections with resulting behavioural difficulties. These various observations indicate a need to determine the normal extent of synaptic connections in pSMA so as to be able to evaluate the capacity of an individual to control their actions. Most importantly, these considerations lead to more subtle observations of behaviour, often only revealed under experimental conditions in which both behaviour and non-invasive brain imaging occur, so as to be able to assess the extent to which the subject is able to express normal psychological capacities related to self-control.

## **3. Failure to think about a future act and therefore acting only to satiate an immediate desire**

### **3.1 Impulsivity, delay aversion and the orbitofrontal cortex of the brain**

Impulsive behaviour is that characterized by acting without thought, i.e. on the impulse. Such behaviours are inappropriate for the context, premature, poorly planned with often adverse consequences. The Barratt Impulsiveness Scale is the self-report method that is most often used to assess the extent of impulsivity and includes items for measuring attention, and perseverance. Very interestingly, healthy subjects show a negative correlation between their degree of impulsivity and the volume of gray matter in their orbitofrontal cortex (OFC)<sup>24</sup>, a part of the brain that is involved in satisfying appetites concerned with olfaction, sexual arousal, as well as the

ingestion of food (for a full description, see [25]). Indeed, fMRI shows a positive correlation between the capacity to successfully maintain restraint and activity in the OFC<sup>26</sup>.

Delay aversion behaviour refers to the inability to weigh short-term minor rewards against long-term major rewards, so that decreased delay aversion indicates an increased inability to postpone the reward. Delayed aversion then gives a measure of the extent of acting to immediately satiate a desire. This behaviour is also dependent on the functioning of the OFC<sup>27</sup>.

Thus impulsivity and failure of delay aversion, acting without prior thought as to the consequences of one's actions, are associated with abnormal activity in the OFC. This part of the brain, together with adjacent cortical and sub-cortical areas receives a dense set of synapses from dopamine releasing neurons located in the midbrain, so forming part of the neural circuits that are thought to be involved in cravings to satisfy appetites and in addiction (for a review, see [28]). The importance of the OFC is highlighted below because of its role in impulsivity and delay aversion (§3.2 to §3.4), and the changes in the OFC that accompany drug addiction (§3.5), particularly those due to abnormalities in dopamine release (§3.6) and the loss of synaptic connections (§3.7).

### **3.2 Impulsivity arises after lesions to the orbitofrontal cortex**

Lesions and neurodegeneration in the OFC give rise to disinhibition and so impulsive and socially inappropriate behaviour<sup>29-32</sup>.

### **3.3 Impulsivity is characteristic of a number of psychiatric conditions that involve decreases of activity in orbitofrontal cortex**

Impulsivity is a major characteristic of Borderline Personality Disorder in which a subject also has paranoid ideas, difficulties with relationships, outbursts of anger as well as violent behaviour that often leads to criminality. The risk of suicide is very high, up to 5000 times that of the general population. Non-invasive brain imaging with positron emission tomography (PET) shows that subjects with this disorder have decreased activity in their OFC compared with normal subjects<sup>33</sup>, an observation confirmed using fMRI<sup>34</sup>.

### **3.4 Delay aversion is dependent on the functioning of orbitofrontal cortex**

The ability to weigh short-term minor rewards against long-term major rewards, that is delay aversion, is critically dependent on the normal functioning of the OFC<sup>35,36</sup>. Using the Iowa Gambling Task as a measure of appropriate decision-making in reward-related behaviour shows that the appropriateness decreases with a decrease in activity in OFC<sup>36</sup>.

### **3.5 Changes in impulsivity and delay aversion in drug addicts correlates with changes in the orbitofrontal cortex**

Drugs of addiction that lead to increased impulsivity and decreases in delay aversion, such as methamphetamine and cocaine<sup>37-40</sup>, are associated in drug-addicted subjects with decreases in activity of the OFC as measured with non-invasive brain imaging techniques<sup>39-41</sup> (for a review see [42]).

### **3.6 Decreases in activity in the orbitofrontal cortex in drug addiction involve changes in the release of the transmitter dopamine**

Amphetamine, even in non-addictive subjects, causes immediate release of dopamine from nerve terminals and inhibits removal of dopamine after its release<sup>43,44</sup>, thereby giving a transient high increase in extracellular dopamine followed by a very low capacity for release<sup>45,46</sup>. Cocaine also increases extracellular dopamine, but in this case by just inhibiting the removal of dopamine from the extracellular space once it is released<sup>47,48</sup>.

The reduction of OFC activity associated with impulsivity and shorter delay aversion in chronic amphetamine users, involves the reduction of dopamine release at nerve terminals<sup>49,50</sup>. The eventual decrease in dopamine transmission in the OFC with amphetamine is correlated with decreased delay aversion in both animals and humans<sup>51-55</sup> (for a review, see [56]). It is likely that the failure of dopamine release from nerve terminals in OFC of addicts leads to impulsivity and shorter delay aversion that reinforces the addiction. Evidence for changes in the genes for the synthesis of dopamine predisposes a subject to addiction<sup>57</sup> through a consequent decrease in OFC activity with subsequent impulsivity and failure of delay aversion (for a review see [56]).

### **3.7 Decreases in gray matter (synapses) of orbitofrontal cortex in schizophrenia and drug addiction**

Addictive drugs not only modulate transmission in the OFC, but also lead to a loss of synapses, exacerbating the abnormal behavioural state characterized by impulsivity and failure of delay aversion. Cocaine users show a 5% to 11% decrease in the gray matter of OFC<sup>58</sup> and there is a loss of synaptic connections in OFC following amphetamine use<sup>59</sup>.

Early studies of structural changes in the OFC of those suffering from schizophrenia, using magnetic resonance imaging, could not find any loss of gray matter<sup>60-62</sup> although more recent studies do<sup>63,64</sup>. The most recent research on this topic has identified the technical basis for these differences and shows a correlation between the extent of loss of OFC gray matter and the extent of expression of core components of the schizophrenia syndrome<sup>65</sup>. This decrease of gray matter is most likely due to a decrease in the loss of synaptic connections, as there are no changes in the density of nerve cells in the OFC of patients with schizophrenia<sup>66</sup>.

## **4. The need for reconsideration of the M'Naghten Rules**

### **4.1 The definition of 'mental illness'**

There have been recent attempts in Australia to bring some clarity to the plea of 'mental illness' under the M'Naghten Rules. Shea suggests replacing "imprecise phrases such as 'mental illness' and 'mental defect' (and associated terms such as 'abnormality of mind', 'mental disturbance', 'mental dysfunction' and 'mental disorder) [67; p.357]" with a few symptoms that only need consideration in the defence of mental illness, namely 'delusions, hallucinations, severe mood disturbances (depression or elevation) and severe impairment of intellect [67; p.358]'. More recently The Victorian Law Reform Commission has drawn up its own 'categories of mental

illness/impairment' namely psychotic illness, depression or depression related illness, IQ related illness, personality disorder and alcohol/drug abuse<sup>68</sup>. The first three of these are allied to the Shea list of symptoms, namely delusions/hallucinations, severe mood disorder and severe impairment of intellect. The statutory definition covering impaired mental functioning in the ACT (pre 2003) is especially interesting, stating that such an impairment amounts to '[A] disturbance or defect to a substantially disabling degree, of perceptual interpretation, comprehension, reasoning, learning, judgment, memory, motivation or emotion<sup>69</sup>'. A number of these psychological categories are open to investigation by both behavioral and neuroscientific means<sup>70</sup>. Unless one subscribes to the view that in determining mental impairment 'the mere fact of organic damage alone is insufficient. It is the fact of disorder which is decisive, not the cause of it [71; p.7]' then the contributions of both psychology and neuroscience might be illuminating. Indeed the ACT definition could be paraphrased as 'A disturbance of psychological capacities' for such capacities include perceiving, thinking, remembering and feeling. These considerations need to be developed much more fully, perhaps by the NSW Law Reform Commission that is at present reviewing the criminal law relating to people with cognitive and mental health impairments.

#### **4.2 A M'Naghten Rule and cognitive neuroscience**

To what extent do the considerations of this short essay on normal and abnormal activity in pSMA and OFC impact on Cockburn's suggestion that '[C] *there are forms of mental disease in which, though the patient is aware he is about to do wrong, the will becomes overpowered by the force of irresistible impulse, the power of self-control, when destroyed or suspended by mental disease, becomes (I think) an essential element of responsibility.*'

We humans, like other animals, can act in the world only by contracting muscles, and therefore initiating limb movements and vocalization, or excreting sweat, tears and urine. I have focused on a small group of these behaviours that require normal working of the pSMA or the OFC in order for us to be able to execute the behaviour appropriately. It is very likely that the disruption of synaptic connections between nerve cells in these areas of the brain restricts our capacity for appropriate behaviour within the activities considered here, and that the more extensive the synapse loss the greater is the incapacity. So large lesions in pSMA, due for example to benign tumor growths or localized failure of the vasculature, are associated with Utilization Behaviour and Anarchic Limbs; small lesions, requiring fMRI to be detected, may be associated with difficulties in switching behaviour from one pattern to another; and other changes in synaptic connectivity, again requiring fMRI for detection, may be associated with some core symptoms of behaviour manifest in certain psychiatric diseases. Cockburn would probably have regarded this class of behaviours as indicating 'the power of self-control'.

Although Cockburn conflated 'irresistible impulse' with 'the power of self-control', cognitive neuroscience distinguishes between them and shows that different brain regions support them. Neurodegeneration in the OFC which leads to loss of synaptic connections in this brain structure, gives rise to actions made without prior thought – and so poorly planned, premature, inappropriate for the context and therefore often with adverse consequences. This impulsivity is greater the larger the decrease in gray matter, of synaptic connections, in OFC. It can be a

particularly dangerous condition, often leading to criminality and suicide. Another consequence of the failure of synaptic connections in OFC is an incapacity to restrain a drive to satiate appetites. A principal means of destroying the synaptic connections in OFC is chronic use of methamphetamine or cocaine, which because of the breakdown in the capacity to delay aversion, of being able to weigh the short-term minor reward of the drug-induced 'high' against the long-term major reward of a healthy life, reinforces the addiction.

I hope that these examples of behaviour, as they depend on the functioning of pSMA and OFC, point up how study of the normal function of a particular part of the brain has forced cognitive neuroscientists into ever more subtle degrees of analysis of human behaviour, of the expression of our psychological capacities, and of what has gone awry with these when synaptic connections fail to form or function. Given this increased knowledge, in the tradition of Isaac Ray's work, it is of course up to the Cockburns and Tindals of the judiciary to determine how such understanding assists the 'criminal law as it pertains to mentally incompetent defendants'.

1. M'Naghten v The Queen (1843) 4 St. TR. (N.S.) 847.
2. Isaac Ray. *A Treatise on the Medical Jurisprudence of Insanity*. Boston: C. Little and J. Brown, 1838.
3. Bucknill JC. The late Lord Chief Justice of England on Lunacy. *Brain* 1881; 4: 1-26
4. UKHL (1843) J16.
5. United States v Brawner (1972) US. App. D.C. 1, 471F.2d 969.
6. The Federal Rule: Comprehensive Crime Control Act (1984) U.S.C. section 7.
7. The Criminal Process and Persons Suffering from Mental Disorder Project of The Law Reform Commission of Western Australia 1991 (No. 69), 2.14.
8. R v Jennings (2005) NSWSC 789.
9. Bennett MR. Consciousness and hallucinations in schizophrenia: the role of synapse regression. *Australian and New Zealand Journal of Psychiatry* 2008; Dec. In press.
10. Boccardi E, Della Sala S, Motto C, Spinnler H. Utilisation behaviour consequent to bilateral SMA softening. *Cortex* 2002; 38:289-308.
11. Sumner P, Husain M. At the Edge of Consciousness: Automatic Motor Activation and Voluntary Control. *Neuroscientist* 2008; Mar 20 {Epub ahead of print}
12. Biran I, Chatterjee A. Alien hand syndrome. *Archives of Neurology* 2004; 61:292-294.
13. Ikeda A, Yazawa S, Kunieda T, Ohara S, Terada K, Mikuni N, Nagamine T, Taki W, Kimura J, Shibasaki H. Cognitive motor control in human pre-supplementary motor area studied by subdural recording of discrimination/selection-related potentials. *Brain* 1999; 122:915-931.

14. Brass M, von Cramon DY. The role of the frontal cortex in task preparation. *Cerebral Cortex* 2002; 12: 908-914.
15. Nachev P, Rees G, Parton A, Kennard C, Husain M. Volition and conflict In human medial frontal cortex. *Current Biology* 2005; 15:122-128.
16. Isoda M, Hikosaka O. Switching from automatic to controlled action by monkey medial frontal cortex. *Nature Neuroscience* 2007; 10:240-248.
17. Rushworth MF, Walton ME, Kennerley SW, Bannerman DM. Action sets and decisions in the medial frontal cortex. *Trends in Cognitive Science* 2004; 8:410-417.
18. Nachev P, Wydell H., O'Neill K, Husain M, Kennard C. The role of the pre-supplementary motor area in the control of action. *Neuroimage* 2007; 36 Suppl.2:T155-T163.
19. Husain M, Parton A, Hodgson TL, Mort D, Rees G. Self control during response conflict by human supplementary eye field. *Nature Neuroscience* 2003; 6:117-118.
20. Rushworth MF, Walton ME, Kennerley SW, Bannerman DM. Action sets and decisions in the medial frontal cortex. *Trends in Cognitive. Science* 2004; 8:410-417.
21. Matsui M, Yoneyama E, Sumiوشي T, Noguchi K, Nohara S, Suzuki M, Kawasaki Y, Seto H, Kurachi M. Lack of self-control as assessed by a personality inventory is related to reduced volume of supplementary motor area. *Psychiatry Research* 2002; 116:53-61.
22. Lopez-Garcia P, Aizenstein HJ, Snitz BE, Walter RP, Carter CS. Automated ROI-based brain parcellation analysis of frontal and temporai brain volumes in schizophrenia. *Psychiatry Research* 2006; 147:153-161.
23. Bennett MR. Dual constraints on synapse formation and regression in schizophrenia: neuregulin, neuroligin, dysbindin, DISC1, MuSK and agrin. *Australian and New Zealand Journal of Psychiatry* 2008; 42:662-677.
24. Matsui K, Nicoletti M, Nemoto K, Hatch JP, Peluso MA, Nery FG, Soares JC. A voxel-based morphometry study of frontal gray matter correlates of impulsivity. *Human Brain Mapping* 2008; May 8, [Epub ahead of print].

25. Völlm BA, de Araujo IE, Cowen PJ, Rolls ET, Kringelbach ML, Smith KA, Jezzard P, Heal RJ, Matthews PM. Methamphetamine activates reward circuitry in drug naïve human subjects. *Neuropsychopharmacology* 2004; 29:1715-1722.
26. Horn NR, Dolan M, Elliott R, Deakin JF, Woodruff, PW. Response inhibition and impulsivity: an fMRI study. *Neuropsychologia* 2003; 41:1959-1966.
27. Mobini S, Body S, Ho MY, Bradshaw CM, Szabadi E, Deakin JF, Anderson IM. Effects of lesions of the orbitofrontal cortex on sensitivity to delayed and probabilistic reinforcement. *Psychopharmacology* 2002; 160:290-298.
28. Goldstein RZ, Volkow ND. Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex. *American Journal of Psychiatry* 2002; 159:1642-1652.
29. Chow TW. Personality in frontal lobe disorders. *Current. Psychiatry Reports* 2000; 2:446-251.
30. Berlin HA, Rolls ET, Kischka U. Impulsivity, time perception, emotion and reinforcement sensitivity in patients with orbitofrontal cortex lesions. *Brain* 2004; 127:1108-1126.
31. Berlin HA, Rolls ET, Iversen SD. Borderline personality disorder, impulsivity, and the orbitofrontal cortex. *American Journal of Psychiatry* 2005; 162:2360-2373.
32. Rogers RD, Everitt BJ, Baldacchino A, Blackshaw AJ, Swanson R, Wynne K, Baker NB, Hunter J, Carthy T, Booker E, London M, Deakin JF, Sahakian BJ, Robbins TW. Dissociable deficits in the decision-making cognition of chronic amphetamine abusers, opiate abusers, patient with focal damage to prefrontal cortex, and tryptophan-depleted normal volunteers: evidence for monoaminergic mechanisms. *Neuropsychopharmacology* 1999; 20:322-339.
33. Soloff PH, Meltzer CC, Becker C, Greer PJ, Kelly TM, Constantine D. Impulsivity and prefrontal hypometabolism in borderline personality disorder. *Psychiatry Research*. 2003; 123:153-163.
34. Silbersweig D, Clarkin JF, Goldstein M, Kernberg OF, Tuescher O, Levy KN, Brendel G, Pan H, Beutel M,

- Pavony MT, Epstein J, Lenzenweger MF, Thomas KM, Posner MI, Stern E. Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. *American Journal of Psychiatry* 2007; 164:1832-1841.
35. Lubman DI, Yücel M, Pantelis C. Addiction, a condition of compulsive behaviour? Neuroimaging and neuropsychological evidence of inhibitory dysregulation. *Addiction* 2004; 99:1491-1502.
36. Bolla KI, Eldreth DA, London ED, Kiehl KA, Mouratidis M, Contoreggi C, Matochik JA, Kurian V, Cadet JL, Kimes AS, Funderburk FR, Ernst M. Orbitofrontal cortex dysfunction in abstinent cocaine abusers performing a decision-making task. *Neuroimage* 2003; 19:1085-1094.
37. Morgan MJ. Recreational use of "ecstasy" (MDMA) is associated with elevated impulsivity. *Neuropsychopharmacology* 1998; 19:252-264.
38. Hanson KL, Luciana M, Sullwold K. Reward-related decision-making deficits and elevated impulsivity among MDMA and other drug users. *Drug and Alcohol Dependence* 2008; 96: 99-110.
39. Winstanley CA, Bachtell RK, Theobald DE, Laali S, Green TA, Kumar A, Chakravarty S, Self DW, Nestler EJ. Increased Impulsivity during Withdrawal from Cocaine Self-Administration: Role for {Delta}FosB in the Orbitofrontal Cortex. *Cerebral Cortex* 2008; Jun 6. [Epub ahead of print].
40. Winstanley CA. The orbitofrontal cortex, impulsivity, and addiction: probing orbitofrontal dysfunction at the neural, neurochemical and molecular level. *Annals of the New York Academy of Sciences* 2007; 1121; 635-655.
41. Everitt BJ, Hutcherson DM, Ersche KD, Pelloux Y, Dalley JW, Robbins, TW. The orbital prefrontal cortex and drug addiction in laboratory animals and humans. *Annals of the New York Academy of Sciences* 2007; 1121:576-597.
42. Goldstein RZ, Volkow ND. Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex. *American Journal of Psychiatry* 2002; 159:1642-1652.
43. Heikkila RF, Orlansky H, Cohen G. Studies on the distinction between uptake inhibition and release of (3H)dopamine in rat brain tissue slices. *Biochemical Pharmacology* 1975, 24:847-852.

44. Sabol KE, Seiden LS. Reserpine attenuates D-amphetamine and MDMA-induced transmitter release in vivo: a consideration of dose, core temperature and dopamine synthesis. *Brain Research* 1998; 806:69-78.
45. Jones SR, Gainetdinov RR, Wightman RM, Caron MG. Mechanisms of amphetamine action revealed in mice lacking the dopamine transporter. *Journal of Neuroscience* 1998, 18:1979-1986.
46. Sulzer D, Sonders MS, Poulsen NW, Galli A. Mechanisms of neurotransmitter release by amphetamines: a review. *Progress in Neurobiology* 2005; 75:406-433.
47. Ritz MC, Cone EJ, Kuhar MJ. Cocaine inhibition of ligand binding at dopamine, norepineohrine and serotonin transporters: a structure activity study. *Life Sciences* 1990; 46:635-645.
48. Giros B, Caron MG. Molecular characterization of the dopamine transporter. *Trends in Pharmacological Sciences* 1993; 14:43-49.
49. Volkow ND, Fowler JS, Wang GJ, Ding YS, Gatley SJ. Role of dopamine in the therapeutic and reinforcing effects of methylphenidate in humans: results from imaging studies. *European Neuropsychopharmacology* 2004; 12:557-566.
50. Volkow ND, Fowler JS, Wang GJ, Swanson JM. Dopamine in drug abuse and addiction: results from imaging studies and treatment implications. *Molecular Psychiatry* 2004; 9:557-569.
51. Bizot JC, Chenault N, Houzé´B, Herpin A, David S, Pothion S, Trovero F. Methylphenidate reduces impulsive behaviour in juvenile Wistar rats, but not in adult Wistar, SHR and WKY rats. *Psychopharmacology (Berlin)* 2007; 193:215-223.
52. van Gaalen MM, Brueggeman RJ, Bronius PF, Scholfelmeer AN, Vanderschuren LJ. Behavioral disinhibition requires dopamine receptor activation. *Psychopharmacology (Berlin)* 2006; 187:73-85.
53. Winstanley CA, Theobald DE, Dalley JW, Robbins TW. Interactions between serotonin and dopamine in the control of impulsive choice of rats: therapeutic implications for impulse control disorders. *Neuropsychopharmacology* 2005; 30:669-682..

54. Winstanley CA, Eagle DM, Robbins TW. Behavioral models of impulsivity in relation to ADHD: translation between clinical and preclinical studies. *Clinical Psychology Review* 2006; 26:379-395.
55. Kheramin S, Body S, Herrera FM, Bradshaw CM, Szabadi E, Deakin JF, Anderson IM. The effect of orbital prefrontal cortex lesions on performance on a progressive ratio schedule: implications for models of inter-temporal choice. *Behavioural Brain Research* 2005; 156:145-152.
56. Patti T, Vanderschuren LJ. The neuropharmacology of impulsive behaviour. *Trends in Pharmacological Sciences* 2008; 29:192-199.
57. Kreek MJ, Bart G, Lilly C, LaForge KS, Nielsen DA. Pharmacogenetics and human molecular genetics of opiate and cocaine addictions and their treatments. *Pharmacological Reviews* 2005; 57:1-26
58. Franklin TR, Acton PD, Maldjian JA, Gray, JD. Croft JR, Dackis CA, O'Brien CP, Childress AR. Decreased gray matter concentration in the insular, orbitofrontal, cingulate, and temporal cortices of cocaine patients. *Biological Psychiatry* 2002; 51:134-142.
59. Crombag HS, Gorny G, Li Y, Kolb B, Robinson TE. Opposite effects of amphetamine self-administration experience on dendritic spines in the medial and orbital prefrontal cortex. *Cerebral Cortex* 2005; 15:341-348.
60. Crespo-Facorro B, Kim J, Andreasen NC, O'Leary DS, Magnotta V. Regional frontal abnormalities in schizophrenia: a quantitative gray matter volume and cortical surface size study. *Biological Psychiatry* 2000; 28:110-119.
61. Hoptman MJ, Volavka J, Weiss EM, Czobor P, Szeszko PR, Gerig G, Chakos M, Blocher J, Citrome LL, Lindenmayer JP, Sheitman B, Lieberman JA, Bilder RM. Quantitative MRI measures of orbitofrontal cortex in patients with chronic schizophrenia or schioaffective disorder. *Psychiatry Research* 2005; 140:133-145.
62. Lacerda AL, Hardan AY, Yorbik O, Vemulapalli M, Prasad KM, Keshaven MS. Morphology of the orbitofrontal cortex in first-episode schizophrenia relationship with negative symptomatology. *Progress in Neuropsychopharmacology and Biological Psychiatry* 2007; 31:510-516.
63. Sapara A, Cooke M, Fannon D, Francis A, Buchanan RW, Anilkumar AP, Barkataki I, Aasen I, Kuipers E,

- Kumari V. Prefrontal cortex and insight in schizophrenia: a volumetric MRI study. *Schizophrenia Research* 2007; 89:22-34.
64. Nakamura M, Nestor PG, Levitt JJ, Cohen AS, Kawashima T, Shenton ME, McCarley RW. Orbitofrontal volume deficit in schizophrenia and thought disorder. *Brain* 2008; 131:180-195.
65. Venkatasubramanian G, Jayakumar PN, Gangadhar BN, Keshavan MS. Automated MRI parcellation study of regional volume and thickness of prefrontal cortex (PFC) in antipsychotic-naïve schizophrenia. *Acta Psychiatrica Scandinavica* 2008; 117:420-431.
66. Cotter D, Hudson L, Landau S. Evidence for orbitofrontal pathology in bipolar disorder and major depression, but not in schizophrenia. *Bipolar Disorders* 2005; 7:358-369.
67. Shea P. M'Naghten Revisited – Back to the Future? (The Mental Illness Defence – A Psychiatric Perspective). *Current Issues in Criminal Justice* 2001; 12: 347-362.
68. Victorian Law Reform Commission. 'Defences to Homicide' Options Paper 2003
69. The Crimes (Amendment) Act 1994 (ACT) s 428B.
70. Bennett MR, Hacker PMS. *History of Cognitive Neuroscience*. New York: Wiley-Blackwell, 2008.
71. Yannoulidis ST. 'Mental Illness, Rationality, and Criminal Responsibility (Tropes of insanity and related defences)' (2003) 25(2) *Sydney Law Review* 189.

