

Commentary: Aggression and impulsivity after head injury

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The most taxing behavioural complications of severe head injury for relatives include irritability and aggression, as illustrated by J. B. Longitudinal studies show an increase over time in the frequency and severity of temper outbursts (Brooks *et al*, 1986; Hall *et al*, 1994), which take the form of explosive or impulsive aggression. I will comment on the nature and treatment of impulsive aggression as illustrated in case J. B., emphasising the interaction of neurobiology with motivational, situational and personality factors.

There is no satisfactory nosology of human aggression. Some theorists focus on overt aggressive acts; some on combinations of personality and EEG abnormalities (Monroe, 1978); and others on traits – a person high in activity, emotionality and impulsivity is likely to be aggressive (Buss & Plomin, 1975); while Eichelman & Hartwig (1990) have developed a database-generated multi-axial approach, encompassing DSM diagnosis and psychological, biological and social axes. Monahan & Steadman (1994) focus on the risk of harm to others. They argue for the inclusion of subject factors (including dispositional, historical and clinical aspects), together with victim and situational factors, with special note of the interaction or conditional effects between them.

Plutchik & van Praag (1995) present a processing model of aggression, in which triggers (threat, challenge, insult) lead to an aggressive impulse which then interacts with subject's current constellation of risk factors ('amplifiers') and modulators ('attenuators') (Table 1) to generate the probability of action. Sufficiently probable action will lead to overt behaviour, which in turn generates a risk of violence or of self-harm, which feeds back to the original trigger. Before the head injury, J.B. had five risk factors for the translation of aggressive impulse to action, and he had a history of at least two subtypes of aggression – premeditated and impulsive.

Aggressive outbursts that result from organic brain dysfunction are thought to have characteristics sufficiently typical to suggest an 'organic aggressive syndrome' (Silver & Yudofsky, 1995) (Box 1).

Table 1. Amplifiers and attenuators of aggression (adapted from Plutchik & van Praag, 1995)

Amplifiers	Attenuators
Learning difficulties	Timidity
Childhood abuse	High self-control
Conduct disorder	Victim behaviour, e.g.
Access to weapons	appeasement
Mistrust	Social support
Trait impulsivity	Situational factors, e.g.
Abnormal mental state	limit-setting
Brain damage	Compliance with
High arousal	treatment or care
Alcohol and drugs	
Victim behaviour	
Situational factors	

These features of aggression are similar to those of Elliot's (1992) 'episodic dyscontrol syndrome', a behaviour of doubtful syndromal validity (Lucas, 1994). Episodic dyscontrol behaviour arises not only in organic syndromes, but also in psychoses

Box 1. Features of organic aggression (adapted from Silver & Yudofsky, 1995)

Reactive – triggered by modest or trivial stimuli

Severity – out of proportion to precipitating stress or provocation

Non-purposeful – no obvious goal served

Explosive – build-up is not gradual; hair-trigger response to provocation; tends to 'feed on itself'

Response to incoming stimuli during outburst – none or irrational

Periodic – the agent lacks self-control to refrain from repetition (after an interim of relative calm)

Ego-dystonic – remorse after outbursts

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and personality disorders (Mungas, 1983). Barratt (1992) described the same phenomena, which he labelled 'impulsive aggression', in violent prisoners without known organic disorders. Moreover, it is not only impulsive aggression in organic disorders that may respond to carbamazepine, but also impulsive aggression in personality and psychotic disorders, often independently of EEG and neuropsychological status (Mattes, 1984; Gardner & Cowdry, 1986).

Impulsivity

Psychologists suggest two broad bipolar components to the dimension of impulsivity: resisting urges *v.* giving in to urges, and responding immediately to a stimulus *v.* planning before making a move (Plutchik & Van Praag, 1995). Psychiatrists, on the other hand, emphasise impulsive behaviours (e.g. bulimia, pyromania, the addictions) or components of disorders (e.g. frontal lobe syndrome). Impulsivity interacts with other personality traits: thus a person who is high on impulsivity and low on control often reveals erratic and impulsive behaviour, while another who scores highly on both impulsivity and control measures might suffer severe conflict that is not often overtly expressed.

Impulsive aggression appears to require a minimal trigger, to be insensitive to current modulating factors, and to feed positively back to the trigger or impulse.

The relationship between impulsivity and aggression

Recent studies suggest that long-standing psychological abnormality and environmental stresses or cues may be necessary for the development of violent impulses. When such impulses occur, their control may relate to trait impulsivity (Barratt, 1993), a history in boys with childhood hyperactivity, impulsivity, inattention (Farrington, 1995), or brain dysfunction (through organic disorder or substance abuse or both).

About 30% of the variance of 'pure' aggression measures is accounted for by 'pure' impulsivity indices (Stein *et al.*, 1993).

Common to both impulsivity and selected forms of aggression are: genetic predisposition; acting without thinking; poor attentional fixity; low serotonin levels; an inability to control selected behaviours despite a vow to do so; frontal lobe dysfunction; and a possible response to anti-convulsants. The associations are likely to differ

for impulsivity and aggression (Plutchik & van Praag, 1995). Psychophysiological studies reveal altered evoked response potentials in certain impulsive aggressive subjects compared with controls, but the findings are heterogeneous in aggressive subjects (Stein *et al.*, 1995).

Case J. B. exhibited multiple risk factors for aggression before his head injury, and is likely to have continued his aggressive behaviour in any event. If he had had no risk factors for aggression, but become purely impulsive after his head injury, he might have shown bulimia, sexual disinhibition, overspending or heavy drinking but not particularly aggression. The marked susceptibility of the frontal and temporal lobes to traumatic brain injury reveals a neurological substrate for acquired impulsivity (as well as reduced planning, self-control and abstract reasoning). In J. B.'s case this has combined with trait impulsivity (reflected in his learning difficulties and conduct disorder) to lower further his threshold for aggression. Alcohol is a notorious facilitator of aggression after head injury, increasing risk of arrest and contributing to poor outcome.

Management

Management begins with a risk assessment (Grounds, 1995; Moore, 1995). J. B.'s brief aggression towards nurses, his aggressive 'catastrophic' reaction to psychological testing, and his assaults on an old lady were observations made by different professionals and had different time courses; hence the importance of a multi-disciplinary assessment of risk.

Treatment consists of general and behavioural measures and consideration of drugs. Before embarking on treatment for aggressive behaviour, a behavioural analysis is required to elicit specific cues or inadvertent rewards of the behaviour. The antecedents, behaviours and consequences of J.B.'s aggression were documented. If there is no improvement or behavioural analysis is too difficult, pharmacotherapy may then be required.

Drugs are widely used (and commonly misused) in the management of patients with aggression after head injury. The use of drugs should be considered in two categories: the treatment of any underlying clinical disorder (such as depression in J. B.); and the treatment of aggression. There is no satisfactory classification of aggression which can predict which patient is most likely to respond to the principal classes of drugs listed as beneficial in Table 2. In the treatment of chronic or impulsive aggression, the choice of medication is often guided by the underlying hypothesised mechanism

Table 2. Psychopharmacology of aggression after head injury (adapted from Ratey & Gordon, 1993; Dolan & Coid, 1993; Silver & Yudofsky, 1995)

Agent	Indications	Comments
Antipsychotics	Psychotic symptoms	Over-sedation and akathisia may increase aggression
SSRIs	Depressive symptoms; irritability; emotional lability; impulsivity	May respond to subnormal doses
Anticonvulsants – carbamazepine; valproate	Associated seizures; impulsive aggression; 'episodic dyscontrol'	Sedation; hepatotoxicity
Lithium	Manic symptoms; emotional lability; learning disability	Neurotoxicity and confusion
Benzodiazepines	Anxiety symptoms	Risk of paradoxical rage
Beta-blockers	Chronic or recurrent aggression; high arousal	Latency of 4–6 weeks; effective in placebo-controlled trials
Buspirone (5-HT _{1A} agonist)	Anxiety or depressive symptoms	Delayed onset of action
Psychostimulants – methylphenidate; pemoline	If history of attention deficit/hyperactivity disorder	
Anti-androgens	Reduce sexual aggression in certain males	

of action (i.e. effects on serotonin; kindling, arousal etc.), or by associated symptoms (e.g. antidepressants for depression, or antipsychotics in those with paranoid misinterpretation). A partial response after a specific drug should lead to consideration of adjunctive treatment with a drug that has a different mechanism of action.

Behavioural treatment and other measures

The general management of aggressive behaviour begins with the identification of warning signals and documenting its antecedents, behaviours and consequences. The psychosocial aspects of the treatment of aggression after head injury are crucial and may be a prerequisite for beneficial pharmacological effect. Family support and training are essential. Behavioural treatment of aggression includes aggression replacement techniques, such as differential reinforcement of other or incompatible behaviours, communication skills training and the programming of activities. Aggression decelerative techniques include calming pre-violent patients; and time-out, which is effective in violent patients immediately after incidents (see Corrigan *et al*, 1993). Therapeutic community approaches, although sometimes useful with violent offenders, have not been examined in organic aggressive patients. Box 2 summarises helpful behavioural and general measures.

The multifactorial aetiology of J. B.'s aggression suggests that he may respond to anticonvulsants, SSRIs (both for impulsive aggression and depression), abstinence from alcohol, structuring of activities, and multi-agency care. While the case for a care manager in relatively uncomplicated head injury is unproven, it would seem to be essential for patients like J. B. The rational

Box 2. Behavioural and general measures in the treatment of organic aggression

Cognitive-behavioural approaches – aggression replacement by differential reinforcement; assertiveness training; activity programming; communication skills training; promotion of competence in other areas; token economy

Aggression decelerative techniques – social extinction; time out; other measures

Family support – control of alcohol consumption; support and educate relatives and carers; service delivery and coordination; care manager

Measures to decrease specific vulnerability to – stress; loss of support; lack of structure; symptomatic or cognitive decompensation

management of aggression after head injury benefits from multi-axial evaluation and will almost certainly be multi-disciplinary and collaborative. The continuation of family therapy, drug treatment, behavioural measures and social support is often required into the long term.

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Multiple choice questions

- With regard to episodic dyscontrol behaviour:
 - alteration of consciousness during dyscontrol occurs in over two-thirds of cases
 - EEG abnormalities are specific
 - carbamazepine reduces outbursts mainly in patients with EEG abnormalities
 - aggression is triggered by significant stimuli
 - beta-blockers have been shown to be effective in placebo-controlled trials.
- Impulsivity:
 - is associated with slow cognitive tempo
 - accounts for over 50% of aggressive behaviour in the general population
 - has a high heritability
 - is reduced by SSRIs
 - is reduced by 5-HT_{1A} agonists.
- Aggression after head injury:
 - usually has a gradual build-up
 - does not respond to time-out procedures
 - rarely depends on situational factors
 - responds to carbamazepine only in patients with associated seizures
 - is facilitated by higher levels of alcohol than those consumed before the injury.

MCQ answers

1	2	3
a F	a F	a F
b F	b F	b F
c F	c T	c F
d F	d T	d F
e T	e F	e F