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Annotation

Anger and Irritability

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Although there have been many studies of violent behaviour, anger has been neglected as a subject of scientific and clinical investigation. Anger can be defined as an affective state experienced as the motivation to act in ways that warn, intimidate or attack those who are perceived as challenging or threatening. Anger is associated with sensitivity to the perception of challenges or heightened awareness of threats. Anger is likely to be closely associated with fear. Although anger is common in mental illnesses it is largely neglected, perhaps because of the social stigma attached to its expression.

A place for the description of the physiology and pathology of anger has always existed. Seneca counselled against both hasty anger "to bite him that bites" and "indignation, which proceeds from an overgreat suspicion". He regarded anger as "the canker of human nature" (Clode, 1888, pp. 189–199). Galen described four humours which included the choleric temperament, while in the 16th century Bright, Downame and Burton (Hunter & Macalpine, 1963, pp. 36–41, 55–59, 99–100) accorded anger a separate place under the rubric of melancholia. Specht (1901) described a clinical entity consisting of the morbid affect of anger, which was often accompanied by the delusions of paranoia but sometimes occurred without delusions. He believed this to be a third affective illness, distinct from mania and depression, although he subsequently modified his theory to hold that morbid anger or irritability was an intermediate state between mania and depression (Specht, 1908).

Should anger have a place in scientific and clinical nosology, or does it deserve only the status of folk psychology (Harrison, 1991) or as a subject for literary and artistic description (Stanford, 1983, pp. 164–168)?

The place of anger in theories of mood

The earliest attempts at a scientific theory of mood tended to move away from Galen's four humours. Heinroth (1818, p. 16), influenced by the English physicians Cullen and Brown, divided all 'passions' into desires and fears. Wundt (1863) used introspective research methods to describe a basic emotional state or 'common feeling' (p. 217) which is elaborated by physiological and psychological processes first into positive and negative 'affective processes', and then are connected to ideas and become 'emotions'. He particularly emphasised the link from emotion, through impulses and desires, to the motivation to act (pp. 228–230). This theme of emotion as motivation has been taken up by contemporary researchers (Panksepp, 1986).

James (1891) is credited with describing, at the same time as Lange, what might be the predecessor of behavioural theories of emotional acts. He hypothesised that external cues elicited reflex vegetative physiological reactions. These responses were perceived and subjectively interpreted according to circumstances ('I am frightened because I run away') (James, 1891, vol. 2, pp. 449–454). It followed that there would be as many 'emotions' as language permitted, and no special brain centres for emotion (vol. 2, pp. 472–474). This was refuted by Cannon (1915), who agreed with James that fear and anger were called forth by the same stimuli but pointed out that they were accompanied by identical somatic changes. He went on to show that emotion could proceed even in the absence of somatic changes, which were merely secondary and adaptive, "to increase power in the attack and in the defense or flight" (pp. 267–284; and Cannon, 1927).

More recent theories of emotion emphasise semantic analysis. Johnson-Laird & Oatley (1989) and Oatley & Johnson-Laird (1990) proposed that there are five 'basic' emotions, by which they mean emotions that are linguistic primitives, irreducible to more simple concepts and therefore likely to have some biological basis. These correspond to happiness, sadness, fear, anger, and disgust. Ortony & Clore (1989) questioned many of the suppositions of the work of Johnson-Laird & Oatley, favouring instead a linguistic analysis that points, like James' theory, to an infinite number of 'basic' emotions, with only positive and negative dimensions of emotion as truly irreducible. Using a different but related technique, McNair *et al* (1971) performed factor analysis on the self-reported mood states of large numbers of normal individuals using a mood-adjective check-list. They found separable 'mood

factors' for depression–dejection, tension–anxiety, anger–hostility, fatigue and vigour, as well as less distinct factors for confusion and friendliness.

It is worth noting briefly that psychodynamic theories of emotion centre on 'instinct', which is possibly a mistranslation of Freud's term '*treibe*', which Lacan (1977, p. 49) said would have been better translated as 'drive', a product of desire and its objects. Lacan favoured a broad division into positive and negative 'drives'. Anger is usually seen in psychodynamic terms as an aspect of a 'death wish' or as inseparable from 'bad objects'.

All 'two emotion' theories assign anger to the dysphoric group, but this is at odds with the commonly described experience of relief and satisfaction associated with feeling and expressing anger, even if these feelings are mixed with guilt or regret.

To summarise, classifications of emotions fall into two groups: theoretically derived classifications tend to postulate only two states, while classifications based on research usually name a greater number and usually include anger. Although the 'two state' theories appear rigorous, their roots are likely to be in metaphysics rather than logic (Runciman, 1955, pp. 171–180).

Anger and descriptive psychopathology

Psychiatric practice does not currently look for morbid moods of anger, nor for fear other than the specific symptoms of anxiety. Indeed, both the medicalisation and politicisation of anger and violent behaviour have provoked sharp controversy (Breggin, 1975; Fields & Sweet, 1975).

Modern attempts to systematise clinical psychopathology have taken a number of approaches to anger. The Present State Examination (PSE; Wing *et al*, 1974) rates irritability as present even if only subjective and never expressed, but no definition of subjective irritability is given. Irritability is not rated as present if anger or violent behaviour has been noted by others and yet is denied by the subject. The Schedule for Affective Disorders and Schizophrenia (Spitzer & Endicott, 1979) uses the word 'anger' for the subjective state and 'irritability' for overt behaviour, which can also be associated with other dysphoric moods such as depression.

There are many self-report questionnaires that seem to emphasise behaviour rather than mood, and all conceptualise 'irritability', 'aggression' and 'anger' in different ways, some emphasising contextual cues (Novaco, 1975, 1985), some separating scores for aggression towards others and towards self (Caine *et al*, 1967), others focusing on arbitrary types of hostility (Buss & Durkee, 1957). It is questionable whether any of these scales primarily reflects angry mood rather than other factors mediating between social interactions and violent or aggressive reactions.

Grounds (1987) cited the philosophy of mind of Wittgenstein as evidence that it is valid to infer subjective states from external signs: "an inner process is in need of outward criteria". Indeed, to deny this inference leads to a needless metaphysical dualism. There are many self-reported and semantic instruments used by psychologists to measure aggressive or irritable behaviour, but psychiatrists are often reluctant to infer anger from a mental state examination that includes external evidence (e.g. the PSE definition above). Stanford (1983, pp. 21–48) notes that actors simulating emotions such as anger can induce real emotional states in the audience, and this is evidence of the audience's ability to perceive emotion through context and form as well as content.

Although violent or aggressive behaviour is not proof of anger, and anger is not necessary for violence or aggression (Gunn, 1973, pp. 56–57), such behaviour should at least raise the possibility of an angry mood.

Neurophysiological theories of anger

Aggressive and hostile behaviour is readily elicited by stimulation of the brain in cats, rats, and other animals. Similarly, the subjective affect of fear is readily evoked by selective brain stimulation in man in areas of the limbic system, but anger is evoked in this way less commonly than other emotions (Gloor *et al*, 1982); this may suggest that in man, as distinct from rats, anger is more subtly related to situational and cognitive modification. Heath (1986) describes a link in man between rage and stimulation of the mesencephalic tegmentum, often in association with memories of past rages. Heath (1986) suggests that in man the neural system for emotion and that for memory have much in common and may be one and the same.

Prodromal irritability, often with suspiciousness and sensitivity to imagined wrongs is a recognised symptom of temporal lobe epilepsy. It is distinct from and usually of longer duration than the auras that immediately precede a partial or grand mal fit. The association of such phenomena with the psychoses of epilepsy and the possible link with discharges in the temporal lobe and septal structures thought to be involved with emotion is of interest (Blanchet & Frommer, 1986; Heath, 1986; Lishman, 1987). There may also be a relationship between specific mood states such as anger and evoked or psychogenic seizures (Fenwick, 1981). Anger, as well as fear and 'paranoia', is also recognised as part of the aura immediately preceding epileptic seizures (MacLean, 1986).

There is a pharmacological overlap between mood-stabilising and anticonvulsant medications which has given rise to speculation concerning the linkage of 'uncontrolled rage outbursts' or 'episodic dyscontrol' and minor neurological dysfunction (Bach-y-Rita *et al*, 1971), although this view has been contested (Mattes, 1986). Coccaro (1989) found evidence of dysfunctional brain 5-hydroxytryptamine systems in those with 'impulsive aggression' who have personality disorders (mostly borderline personality disorder) but not bipolar affective disorder. Unfortunately the literature he reviews is weakened by lack of psychopathological description or clear definitions of the behaviour involved. It remains unclear whether low levels of 5-hydroxytryptamine in cerebrospinal fluid are more directly related to 'overt aggression' or poor impulse control (Van Praag, 1991).

Adams (1979) described three related systems of aggressive behaviour based on ethology and neurophysiology in animals: offence (dominance, the behaviour towards an intruder in the individual's territory), defence (fight or flight according to context), and submission. He noted a specific neurophysiological mechanism for rapidly switching between defensive aggression and submission, according to context.

Gray (1991) has formulated a theory of affective states as motivational states, based on experimental data from rats and man, drawing on learning theory, neurophysiology and pharmacology. Gray has described three 'separable brain subsystems' for affect or motivation. The first of these is the 'fight–flight' system for responses to painful stimuli generally, including frustrative non-reward. This might tentatively be equated with fear and anger in humans, although clearly there are many differences between observed behaviour in animals and subjective human experience.

The theories of Adams and Gray are readily compatible, and suggest that there might be not one but two possible neurophysiological mechanisms for anger. However, efforts to distinguish between 'offensive' and 'defensive' aggressive behaviour in humans have been notably unsuccessful. Pulkkinen (1987) found a single dimension of aggression in school children unless mutually exclusive definitions were used. A study of situational contexts of aggression in a non-psychiatric offender population (Henderson, 1986) indicated that affects, particularly fear and 'excitement', are important elements of certain types of violent behaviour.

Cognitive and behavioural theories of anger

Dollard *et al* (1939) marshalled a wealth of sociological, anthropological, and even economic evidence for their basic postulate that "aggression is always a consequence of frustration". They gave a purely behavioural definition for frustration (interference with an instigated reinforcing goal response), but a definition of aggression that encompassed any behaviour which follows frustration and reduced the instigation to frustration, or an act whose goal response is injury to an organism (p. 8). However, they also listed eight nouns (including 'anger') and ten verbs (including 'hurt' and 'humiliate') which in their view "carry something of the meaning of the concept". Since aggression is not necessarily associated with anger and can include such diverse qualities as competitiveness, dominance, coerciveness and violence, the emphasis on aggression can be confusing. Bandura's (1973, pp. 53–59) social learning theory proposed that any state of emotional arousal would increase the probability of aggression when the context predisposed to aggression. Beck (1976, pp. 64–75) recognised anger as an emotion distinct from violent or aggressive behaviour and discussed it in terms of provocation and the appraisal of threat. The strength of the angry response is determined by such intellectual or cognitive factors as value judgements, self-esteem, and expectations, which are all regarded as enduring dispositions. A link between anger and 'paranoid' ideas or attributional styles was also noted (pp. 87, 98).

Averill (1983) replicated earlier community surveys of the 'everyday experience of anger', and formed a theory of anger as a socially constituted syndrome, a social role governed by social rules. Averill emphasised that anger can have positive functions within interpersonal relationships, although it could be debated whether by this he meant assertiveness and some types of aggression rather than anger. Beck holds that anger is best controlled rather than expressed in relationships (1988, p. 147). Averill's rule-governed syndrome is compatible with the frequent rule-breaking of angry behaviour, since this behaviour has the function of defining self-image and the image one presents to others.

Zillmann's (1988) most recent revision of his theory of emotions (1979) advances a 'two factor' theory linking appraisal processes and a single arousal state. These processes are seen as interactive in anger rather than either having primacy. Novaco (1986) pointed out that such theories do not give enough weight to the role of perceptual biases which determine what receives attention.

Cognitive and behavioural models of affect, and in particular anger, emphasise enduring dispositions and attributional styles but neglect the mood-dependent variation of sensitivity thresholds for perception and response to stimuli. Examples of

mood-dependent variation in such functions include the influence of mood on ease of recollection of pleasant and unpleasant memories (Lishman, 1974; Master *et al*, 1983), or violent events being remembered in a state of arousal (Taylor & Kopelman, 1984). Likewise, variations in emotional arousal lead to altered selectivity of attention (Easterbrook, 1959). Novaco's (1989) theory of anger emphasises the interaction of cognitive dispositions and mood states, and the way in which they can be mutually reinforcing and propagating through rehearsal of grievances and intrusive violent imagery.

Ciompi (1991) has developed a theory of 'affect-logic', according to which memories are stored and recalled, and integrated with their attendant emotions, just as perception is inseparably connected to emotion. This theory accommodates phenomena relevant to anger such as affective variation of memory, attention, and other processes, and gives greater weight to affects as motivators and movers of thought processes generally.

Sociological theories of anger and violence

There is a substantial literature explaining levels of violence at a societal level in terms of such social constructs as anomie, frustrated aspirations, and conflict. These theories do not necessarily reflect causes of anger; indeed, they demonstrate that anger is neither necessary nor sufficient for violence. They often underestimate the role of anger as an amplifier and a uniting factor in collective violence (Novaco, 1986). Novaco (1985) linked the disadvantaged status of the habitually violent to the neglect of anger as a problem.

Weissman *et al* (1971) demonstrated that in depressed women irritability was often intense but confined to certain interpersonal contexts. Kopper & Everson (1991) found that men and women did not differ in scores on a variety of self-report measures of anger and its expression. Breaking down the groups according to a measure of sex-role identity appeared to produce lower anger scores for those classed as feminine rather than masculine (with a third, intermediate group). This may be due to circularity in the definition of sex roles, since the instrument used (Bem, 1974) weighs aggression and assertiveness on the 'masculine' dimension.

Clearly, social constraints on the expression of anger are strong, but it may also be that social factors modulate the perception of cues to anger. This may be relevant to the finding that many homicides are committed by people intimately related to the victim (Hafner & Boker, 1982, pp. 232–240).

Alcohol and anger

The linkage of alcohol and violence bridges issues of psychopathology regarding angry mood and the social context of provocation and angry behaviour. The link between alcohol abuse and dysphoric mood is confused by issues of causality: which comes first, the alcohol or the mood disorder? It would appear that secondary depression and anxiety are no more common among in-patients whose primary diagnosis is alcohol abuse than among other psychiatric in-patients (Bernadt & Murray, 1986). The same study however found a high prevalence of situational anxiety among alcoholics (74% when Research Diagnostic Criteria were used).

There are many studies of the linkage between violence and alcohol, although few have considered a link specifically with anger. Blum (1981) considered the many possible confounding factors other than anger intervening between alcohol and violence, such as situation (e.g. crowded, male-dominated pubs), cultural sanctions, and group interactions.

Pathological intoxication (Coid, 1979) and episodic dyscontrol (Bach-y-Rita *et al*, 1971) are described as syndromes in which some organic deficit renders the subject intoxicated more easily than normal, or results in abnormal mental states during intoxication. These usually involve anger or aggressive behaviour. Pernanen (1976) noted that prolonged excessive use of alcohol may give rise to changes in predisposition which increase the likelihood of violence, whether alcohol has been recently consumed or not. These changes may include altered reality-orientation functions as suggested above, such as narrowing of perceptual field, and rigidity of cognitive set. This is thought to lead to an inability to change course by either backing down or accepting appeasement once the subject thinks a hostile interaction has commenced. Collins (1981) reviewed studies of the effect of alcohol on mood, and noted that many studies indicate an increase in anger, at least when drinking in company. Alcoholics respond differently to non-alcoholics, with more dysphoric feelings and less accurate prediction while sober of their own aggression when drunk.

Studies linking the heritability of criminality and alcoholism give conflicting results. Mednick *et al* (1988) concluded that, unlike a propensity for property crime, violent crime is not heritable. Others have shown a link between the heritability of alcoholism and criminality (Bohman, 1978), including alcohol-related violence. These studies however can

be criticised for using alcohol-related offences as one index of criminality, thus introducing circularity into measures which ought to be independent if a relationship is to be inferred.

It would appear that no consistent, direct relationship between alcohol and angry mood can be found, although social and psychological aspects of alcohol abuse may predispose to both anger and violent behaviour. Likewise, genetic studies on the relationship between property-related crime and violence are inconclusive. Alcohol abuse, like criminality, is associated with violence, but the nature of the relationship, and its possible mediation via anger, is unclear.

Anger and illness

The place of anger in a variety of physical diseases has attracted more attention than it has in psychopathology.

Irritability and hyperphagia are among the features of the obesity–hyperventilation or 'Pickwickian' syndrome, with a favourable 'personality change' on recovery (Sullivan *et al*, 1983). Alcohol is thought to have a significant role as an initiating factor, although it is not necessary as a continuing factor (Issa & Sullivan, 1982).

Anger is recognised as being associated with a risk of developing coronary heart disease (Friedman & Booth-Kewley, 1987), although the association with objective evidence of coronary artery disease has been questioned by Stone & Costa (1990), who also raise the need for better differentiation between 'neurotic' anger, in which resentment and impulsivity are said to be important, and 'antagonistic hostility', in which callous, controlled, and unfriendly dispositions are at work.

Anger, irritability, tantrums and violence are listed among diagnostic criteria for paranoid, antisocial borderline, histrionic, and narcissistic personality disorders, the 'dramatic' or 'flamboyant' cluster of personality disorders in DSM–III–R (American Psychiatric Association, 1987). Tyrer & Alexander (1988), using an instrument which excludes direct measures of subjective distress, have described a similar cluster of sociopathic, impulsive, explosive, sensitive/paranoid-aggressive, and histrionic personality disorders, which is united by the prominence of callousness, aggression, impulsiveness, irritability, and irresponsibility. It is worth noting that in practice such patients are often angry, often for prolonged periods, but they are seldom continuously subject to one affect. They have periods of euthymic mood and of depression, and anxiety also.

Both cyclothymic (Akiskal *et al*, 1977) and dysthymic disorders (Akiskal *et al*, 1983) have been considered either as mental illnesses or personality disorders, prompting the question, when does a recurrent or persistent affective state become a relapsing or chronic affective disorder rather than a personality disorder (Tyrer *et al*, 1983)?

Neenan *et al* (1986) describe the evolution in two male patients of schizoid, avoidant, and paranoid personality traits, secondary to a long history of panic attacks. Noyes *et al* (1991), using an accepted personality inventory, found that apparent personality traits such as dependency, lack of self-confidence, emotional instability, and sensitivity to criticism distinguished a large series of patients with panic disorder from matched controls, but at follow-up those whose panic symptoms had improved also improved in these 'personality traits'. These reports illustrate the way in which the presentation of apparent personality disorder can mask underlying affective disorders. Reports by psychologists (e.g. Deffenbacher *et al*, 1986) using questionnaires such as the State–Trait Anger Scale (Spielberger *et al*, 1983) often emphasise anger or irritability as a lifetime trait or situational state without considering whether an episodic disorder, a process, reaction, development, or derailment is at work.

There are descriptions of mood states other than depression, mania, and anxiety, such as '*Verstimmung*' or 'ill-humoured mood state'. '*Verstimmung*' has tended to be conflated with depression by English-speaking psychiatrists (Fish, 1985, p. 74). Irritability has found its way into the diagnostic criteria for depression, dysthymia, and mania (American Psychiatric Association, 1987; World Health Organization, 1987), despite lack of consensus on the meaning of terms such as 'dysphoria' and 'irritability' (Snaith & Taylor, 1985; Gabriel, 1987).

Specht's (1901) proposal that anger could be a third affective illness was not entirely lost, although many subsequently tried to show that paranoia was a form of 'masked depression' rather than anger. Gaupp (1914), however, agreed with Specht, and emphasised the feelings of shame, fear, and anger in paranoia. Gaupp's pupil Kretschmer (1927) developed the concept of paranoid disposition or 'sensitive reaction type'. He characterised this as a propensity for strong emotional reactions coupled with the conscious retention of affect-laden groups of ideas. Specht's observations regarding anger in paranoia (delusional disorder) have received support from the 'Vienna school' of psychiatry, where Berner *et al* (1982), Gabriel (1987) and Chanda & Gabriel (1988) have published observations supporting his theory.

Recent reports have described a similarity between 'anger attacks' and panic attacks, the anger attacks

apparently responding to antidepressant medication (Fava *et al*, 1990). The same workers describe 'hostility' as a prolonged phase in the recovery from major depressive illness that responded to amitriptyline (Fava *et al*, 1986).

Snaith & Taylor (1985), using a self-report checklist, found irritability in many patients with postnatal depression, anxiety disorders, obsessive–compulsive disorder, or depression. Craig *et al* (1985) used interview rating scales and found that in male schizophrenics there were separable dimensions of mood for hostility–suspiciousness as well as anxiety–depression, retardation–affective blunting, and thought disturbance.

Ciompi (1989), in keeping with his theory of 'affect-logic', outlines a theory for the onset of schizophrenia in which vulnerable individuals under stress may over-react in an 'aggressive emotional and/or confused' way, leading to further social tensions and escalation to some critical point of emotion or arousal where the system/mental state shifts to psychotic functioning.

The mentally ill are over-represented among those convicted of violent offences (Taylor & Gunn, 1984). Mowat (1966, pp. 111–117) drew attention to the frequency of paranoid illnesses, particularly delusions of infidelity, among mentally disordered perpetrators of homicide. Kennedy *et al* (1992) have observed that prolonged and pervasive mood states of fear and anger occur together in patients with delusional (paranoid) disorder who act violently, and that delusions, mood, and actions are congruent in such cases. Although one cannot infer from such clinical observations whether the mood or delusions are primary, prolonged moods of anger deserve more attention.

A clinical definition of anger

There are reasonable grounds to suppose that anger is closely associated with fear, ranging from animal and human physiological evidence for a fight–flight system through to social-psychology theories concerning the situational appraisal of environmental threats, as well as clinical observations (e.g. in paranoia).

It is reasonable to recognise anger as a mood, not only from verbal accounts of subjective mental state but also from inference based on the recognition of anger in other (unspoken) aspects of the mental state.

> Anger is an affective state experienced as the motivation to act in ways that warn, intimidate or attack those who are perceived as challenging or threatening. Anger is coupled to and is inseparable from a sensitivity to the perception of challenges or a heightened awareness of threats (irritability). This affective motivation and sensitivity can be subjectively experienced even if no external action occurs.

This definition requires little alteration to describe fear. The sensitivity to challenge and threat would be the same, but the affective motivation experienced would instead be to escape, appease, or avoid.

Anger may be a transient state of variable frequency and variable intensity. Where anger is prolonged (often through rehearsal of grievances and intrusion of violent images and reveries), it may be judged intense if there are frequent external expressions of anger, if minimal provocation evokes such external expressions, or if the extent of preoccupation and rumination is such as to interfere with normal activities. Since the expression of anger is more unacceptable in society than expression of any other affective state, and since anger is commonly destructive of social relationships, it may constitute a disability even if work and activities of daily living are unimpaired.

While excessive lability of mood, frequent episodes of violent behaviour, and intense or prolonged experiences of anger may all be problems, there is as yet no evidence for a distinct mental illness characterised only by a mood of anger. It is probably best to define a disease state in terms of 'stable derailment', that is, continuous subjective anger, lasting longer than some arbitrary length of time, resulting in some dysfunction in interpersonal relationships or social role. However, a disorder of anger might also be defined by analogy with panic disorder, in which frequency of 'rage attacks' could be related to psychiatric morbidity and disability.

Conclusions

Whereas the 'two emotion' theories depend heavily on a dualistic distinction between mental processes (cognition, will, superego) and physiological processes (arousal, instinct, drive), the empirical theories usually define anger as a separate emotion with a number of others, are compatible with phenomenological and philosophical theories of mind, and allow for the possible identification of separable brain subsystems specific for emotions, and for the possibility of continuity between observation and experiment in animals and man.

Anger has been virtually forgotten in the psychiatric assessment of mental state, despite the long history of medical interest in anger and its general acceptance as a problem in most currently recognised mental illnesses and in its own right. Whether a distinct affective disorder – a morbid state of anger – can be described remains to be seen, but merits further research. Possible neurochemical mechanisms involving 5-hydroxytryptamine in impulsivity and anger with labile mood (panic rages) emphasise the need

for studies to aid better clinical description and definition of anger. In the same way, the recognition of anger in paranoia may lead to the recognition of prolonged anger in those who are 'paranoid' although not deluded, and a greater realisation of the role of anger in many patients currently classed as personality disordered. If an anger disorder exists, it is likely to be found not in primary care or psychiatric out-patient clinics, but in prisons and other places where the marginalised and rejected congregate.

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References

ADAMS, D. B. (1979) Brain mechanisms for offence, defence and submission. *Behavioural and Brain Sciences*, **2**, 201–241.

AKISKAL, H. S., DJENDEREDJIAN, A. H., ROSENTHAL, T. L., *et al* (1977) Cyclothymic disorder: validating criteria for inclusion in the bipolar affective group. *American Journal of Psychiatry*, **134**, 1227–1233.

———, HIRSCHFELD, R. M. A. & YERVANIAN, B. I. (1983) The relationship of personality to affective disorders: a critical review. *Archives of General Psychiatry*, **40**, 801–810.

AMERICAN PSYCHIATRIC ASSOCIATION (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R). Washington, DC: APA.

AVERILL, J. R. (1983) Studies on anger and aggression: implications for theories of emotion. *American Psychologist*, **38**, 1145–1160.

BACH-Y-RITA, G., LION, J. R., CLIMENT, C. E., *et al* (1971) Episodic dyscontrol: a study of 130 violent patients. *American Journal of Psychiatry*, **127**, 1473–1478.

BANDURA, A. (1973) *Aggression: a Social Learning Analysis*. New York: Prentice Hall.

BECK, A. T. (1976) *Cognitive Therapy and the Emotional Disorders*. New York: International Universities Press.

——— (1988) *Love Is Never Enough*. London: Penguin.

BEM, S. L. (1974) The measurement of psychological androgyny. *Journal of Consulting and Clinical Psychology*, **42**, 155–162.

BERNADT, M. W. & MURRAY, R. M. (1986) Psychiatric disorder, drinking and alcoholism: what are the links? *British Journal of Psychiatry*, **148**, 393–400.

BERNER, P. & KUFFERLE, B. (1982) British phenomenological and psychopathological concepts: a comparative review. *British Journal of Psychiatry*, **140**, 558–565.

BLANCHET, P. & FROMMER, G. P. (1986) Mood change preceding epileptic seizures. *Journal of Nervous and Mental Disease*, **174**, 471–476.

BLUM, R. H. (1981) Violence, alcohol and setting: an unexplored nexus. In *Drinking and Crime* (ed. J. J. Collins), pp. 110–142. New York: Guilford Press.

BOHMAN, M. (1978) Some genetic aspects of alcoholism and criminality. *Archives of General Psychiatry*, **35**, 269–276.

BREGGIN, P. R. (1975) Psychosurgery for the control of violence: a critical review. In *Neural Bases of Violence and Aggression* (eds. W. S. Fields & W. H. Sweet). St Louis, Missouri: Green.

BUSS, A. H. & DURKEE, A. (1957) An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology*, **21**, 343–349.

CAINE, T. M., FOULDS, G. A. & HOPE, K. (1967) *Manual of the Hostility and Direction of Hostility Questionnaire*. London: University of London Press.

CANNON, W. B. (1915) *Bodily Changes in Pain, Fear, Hunger and Rage. An Account of Recent Researches into the Function of Emotional Excitement*. New York: Appleton.

——— (1927) The James–Lange theory of emotions: a critical examination and an alternative. *American Journal of Psychology*, **39**, 132–143.

CHANDA, H. & GABRIEL, E. (1988) Position of affective symptomatology in the course of delusional psychoses. *Psychopathology*, **21**, 1–11.

CIOMPI, L. (1989) The dynamics of complex psychosocial systems: four fundamental psycho-biological mediators in the long-term evolution of schizophrenia. *British Journal of Psychiatry*, **155** (suppl. 5), 15–21.

——— (1991) Affects as central organising and integrating factors: a new psychosocial/biological model of the psyche. *British Journal of Psychiatry*, **159**, 97–105.

CLODE, W. (1888) *The Morals of Seneca: A Selection of His Prose*. London: Walter Scott.

COCCARO, E. F. (1989) Central serotonin and impulsive aggression. *British Journal of Psychiatry*, **155** (suppl. 8), 52–62.

COID, J. (1979) Mania a potu: a critical review of pathological intoxication. *Psychological Medicine*, **9**, 709–719.

COLLINS, J. J. (1981) Alcohol careers and criminal careers. In *Drinking and Crime* (ed. J. J. Collins), pp. 152–206. New York: Guilford Press.

CRAIG, T. J., RICHARDSON, M. A., PASS, R., *et al* (1985) Measurement of mood and affect in schizophrenic in-patients. *American Journal of Psychiatry*, **142**, 1272–1277.

DEFFENBACHER, J. L., DEMM, P. M. & BRANDON, A. D. (1986) High general anger: correlates and treatment. *Behavioural Research and Treatment*, **24**, 481–489.

DOLLARD, J., MILLER, N. E., DOOB, L. W., *et al* (1939) *Frustration and Aggression* (reprinted 1944). London: Kegan, Paul, Trench, Trubner.

EASTERBROOK, J. A. (1959) The effect of emotion on cue utilisation and the organisation of behaviour. *Psychological Reviews*, **66**, 183–201.

FAVA, G. A., KELLNER, R. LISANSKY, J., *et al* (1986) Hostility and recovery from melancholia. *Journal of Nervous and Mental Disease*, **174**, 414–417.

———, ANDERSON, K. & ROSENBAUM, J. F. (1990) "Anger attacks": possible variants of panic and major depressive disorders. *American Journal of Psychiatry*, **147**, 867–870.

FENWICK, P. (1981) Precipitation and inhibition of seizures. In *Epilepsy and Psychiatry* (eds E. H. Reynolds & M. R. Trimble). ch. 22. London: Churchill Livingstone.

FIELDS, W. S. & SWEET, W. H. (1975) Prologue. In *Neural Bases of Violence and Aggression* (eds W. S. Fields & W. H. Sweet). St Louis, Missouri: Green.

FISH, F. (1985) *Fish's Clinical Psychopathology* (2nd edn) (ed. M. Hamilton). Bristol: Wright.

FRIEDMAN, H. S. & BOOTH-KEWLEY, S. (1987) The 'disease-prone personality': a meta-analytic view of the construct. *American Psychologist*, **42**, 539–555.

GABRIEL, E. (1987) Dysphoric mood in paranoid psychoses. *Psychopathology*, **20**, 101–106.

GAUPP, R. (1914) The scientific significance of the case of Ernst Wagner (trans. H. Marshall). In *Themes and Variations in European Psychiatry* (eds S. R. Hirsch & M. Shepherd, 1974), pp. 121–133, 134–152. Bristol: Wright.

GLOOR, P., OLIVIER, A., QUESNEY, L. F., *et al* (1982) The role of the limbic system in experiential phenomena of temporal lobe epilepsy. *Annals of Neurology*, **12**, 129–144.

GRAY, J. A. (1991) Neural systems of motivation, emotion and affect. In *Neurobiology of Learning, Emotion and Affect* (ed. J. Madden). New York: Raven Press.

GRIESINGER, W. (1845) *Mental Pathology and Therapeutics* (trans. C. L. Robertson & J. Rutherford, 1867), pp. 44–114. London: New Sydenham Society.

GROUNDS, A. (1987) On describing mental states. *British Journal of Medical Psychology*, **60**, 305–311.

GUNN, J. (1973) *Violence in Human Society*. Newton Abbot: David & Charles.

HAFNER, H. & BOKER, W. (1982) *Crimes of Violence by Mentally Abnormal Offenders. The Psychiatric and Epidemiological Survey in the Federal German Republic* (trans. H. Marshall), pp. 232–240. Cambridge: Cambridge University Press.

HARRISON, P. J. (1991) Are mental states a useful concept? Neurophilosophical influences on phenomenology and psychopathology. *Journal of Nervous and Mental Disease*, **179**, 309–319.

HEATH, R. G. (1986) The neural substrate for emotion. In *Emotion: Theory, Research and Experience, Volume 3, Biological Foundations of Emotion* (eds R. Plutchik & H. Kellerman), pp. 3–36. Orlando, Florida: Academic Press.

HEINROTH, J. C. (1818) *Textbook of Disturbances of Mental Life or Disturbances of the Soul and their Treatment* (2 vols, trans. J. Schmorak, 1975). Baltimore: Johns Hopkins University Press.

HENDERSON, M. (1986) An empirical typology of violent incidents reported by prison inmates with convictions for violence. *Aggressive Behaviour*, **14**, 21–32.

HUNTER, R. & MACALPINE, I. (1963) *Three Hundred Years of Psychiatry*. London: Oxford University Press.

JAMES, W. (1891) *Principles of Psychology* (2 vols). London: MacMillan.

ISSA, F. Q. & SULLIVAN, C. E. (1982) Alcohol, snoring and sleep apnoea. *Journal of Neurology, Neurosurgery and Psychiatry*, **45**, 353–359.

JOHNSON-LAIRD, P. N. & OATLEY, K. (1989) The language of emotions: an analysis of a semantic field. *Cognition and Emotion*, **3**, 81–123.

KENNEDY, H. G., KEMP, L. I. & DYER, D. E. (1992) Fear and anger in delusional (paranoid) disorder: the association with violence. *British Journal of Psychiatry*, **160**, 488–492.

KOPPER, B. A. & EVERSON, D. L. (1991) Women and anger: sex and sex-role comparisons in the expression of anger. *Psychology of Women Quarterly*, **15**, 7–14.

KRETSCHMER, E. (1927) The sensitive delusion of reference (trans. J. Candy). In *Themes and Variations in European Psychiatry* (eds S. R. Hirsch & M. Shepherd, 1974), pp. 153–196. Bristol: Wright.

LACAN, J. (1977) *The Four Fundamental Concepts of Psychoanalysis* (ed. J-A. Miller, trans. A. Sheridan). London: Hogarth Press & Institute of Psych Analysis.

LISHMAN, W. A. (1974) The speed of recall of pleasant and unpleasant experiences. *Psychological Medicine*, **4**, 212–218.

—— (1987) *Organic Psychiatry: The Psychological Consequences of Cerebral Disorder* (2nd edn). London: Blackwell.

MACLEAN, P. D. (1986) Ictal symptoms relating to the nature of affects and their cerebral substrate. In *Emotion: Theory Research and Experience, Volume 3, Biological Foundations of Emotion* (eds R. Plutchik & H. Kellerman), pp. 61–90. Orlando, Florida: Academic Press.

MASTER, D., LISHMAN, W. A. & SMITH, A. (1983) Speed of recall in relation to affective tone and intensity of experience. *Psychological Medicine*, **13**, 325–331.

MATTES, J. A. (1986) Psychopharmacology of temper outbursts, a review. *Journal of Nervous and Mental Disease*, **174**, 464–470.

MCNAIR, D. M., LORR, M. & DROPPLEMAN, L. F. (1971) *Profile of Mood States*. San Diego, California: Educational and Industrial Testing Service.

MEDNICK, R. R. (1966) *Morbid Jealousy and Murder*. London: Tavistock.

NEENAN, P., FELKNER, J. & REICH, J. (1986) Schizoid personality traits developing secondary to panic disorder. *Journal of Nervous and Mental Disease*, **174**, 483.

NOVACO, R. W. (1975) *Anger Control: The Development and Evaluation of an Experimental Treatment*. Lexington, Massachusetts: D. C. Heath.

—— (1985) Anger and its therapeutic regulation. In *Anger and Hostility in Cardiovascular and Behavioural Disorders* (eds M. A. Chesney & R. H. Rosenman). New York: Hemisphere.

—— (1986) Anger as a clinical and social problem. In *Advances in the Study of Aggression, Vol. 2* (eds R. J. Blanchard & D. C. Blanchard). New York: Academic Press.

—— (1989) Anger disturbances: cognitive mediation and clinical prescriptions. In *Clinical Approaches to Violence* (eds K. Howells & C. R. Hollin). Chichester: Wiley.

NOYES, R., REICH, J. H., SUELZER, M., *et al* (1991) Personality traits associated with panic disorder: change associated with treatment. *Comprehensive Psychiatry*, **32**, 283–294.

OATLEY, K. & JOHNSON-LAIRD, P. N. (1990) Semantic primitives for emotions: a reply to Ortony and Clore. *Cognition and Emotion*, **4**, 129–143.

ORTONY, A. & CLORE, G. L. (1989) Emotions, moods and conscious awareness. *Cognition and Emotion*, **3**, 125–137.

PANKSEPP, J. (1986) The anatomy of emotions. In *Emotion: Theory, Research and Experience, Volume 3, Biological Foundations of Emotion* (eds R. Plutchik & H. Kellerman), pp. 91–124. Orlando, Florida: Academic Press.

PERNANEN, K. (1976) Alcoholism and crimes of violence. In *The Biology of Alchoholism. Vol. 4: Social Aspects of Alcoholism* (eds B. Kissin & H. Begleiter). New York: Plenum.

PULKKINEN, L. (1987) Offensive and defensive aggression in humans: a longitudinal perspective. *Aggressive Behaviour*, **13**, 197–212.

RUNCIMAN, S. (1955) *The Medieval Manichee*. Cambridge: Cambridge University Press.

SNAITH, R. P. & TAYLOR, C. M. (1985) Irritability: definition, assessment and associated factors. *British Journal of Psychiatry*, **147**, 127–136.

SPECHT, G. (1901) Uber den patholoigischen Affekt in der chronischen Paranoia. In *Festschrift der Erlanger Universitat*. Leipzig: Bohme.

—— (1908) Uber die klinische kardinalfrage der paranoia. *Zentralblatt fur Nervenheilkunde und Psychiatrie*, **31**, 817–833.

SPIELBERGER, C. D., JACOBS, G. A., RUSSEL, S., *et al* (1983) Assessment of anger: the state–trait anger scale. In *Advances in Personality Assessment, Vol. 2* (eds J. N. Butcher & C. D. Spielberger). Erlbaum, Hillside, New Jersey: Erlbaum.

SPITZER, R. L. & ENDICOTT, J. (1979) *Schedule for Affective Disorders and Schizophrenia (SADS)* (3rd edn). New York: New York State Psychiatric Institute.

STANFORD, W. B. (1983) *Greek Tragedy and the Emotions*. London: Routledge & Kegan Paul.

STONE, S. V. & COSTA, P. T. (1990) Disease-prone personality or distress-prone personality? The role of neuroticism in coronary heart disease. In *Personality and Disease* (ed. H. S. Friedman), pp. 178–200. New York: Wiley.

SULLIVAN, C. E., BERTHON-JONES, M. & ISSA, F. G. (1983) Remission of severe obesity-hyperventilation syndrome after short-term treatment during sleep with nasal continuous positive airway pressure. *American Review of Respiratory Disease*, **128**, 177–181.

TAYLOR, P. J. & GUNN, J. (1984) Violence and psychosis I – Risk of violence among psychotic men. *British Medical Journal*, **288**, 1945–1949.

—— & KOPELMAN, M. D. (1984) Amnesia for criminal offences. *Psychological Medicine*, **14**, 581–588.

TYRER, P., STRAUSS, J. & CICCHETTO, D. (1983) Temporal reliability of personality in psychiatric patients. *Psychological Medicine*, **13**, 393–398.

—— & ALEXANDER, J. (1988) Personality assessment schedule. In *Personality Disorders: Diagnosis, Management and Course* (ed. P. Tyrer). London: Wright.

VAN PRAAG, H. M. (1991) Serotonergic dysfunction and aggression control. *Psychological Medicine*, **21**, 15–19.

WEISSMAN, M. M., KLERMAN, G. L. & PAYKEL, E. S. (1971) Clinical evaluation of hostility in depression. *American Journal of Psychiatry*, **128**, 261–266.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

WORLD HEALTH ORGANIZATION (1987) *Tenth Revision of the International Classification of Diseases Chapter V (F) Mental Behavioural and Developmental Disorders* (1987 draft for field trials). Geneva: WHO.

WUNDT, W. (1863) Vorlesungen uber die Menschen- und Tier-Seele (2 vols). Leipzig. (trans. J. E. Creighton & E. B. Titchener, 1986, *Human and Animal Psychology*). London: Swan Sonnenschein.

ZILLMANN, D. (1979) *Hostility and Aggression*. Hillsdale, New Jersey: Erlbaum.

—— (1988) Cognition-excitation interdependencies in aggressive behaviour. *Aggressive Behaviour*, **14**, 51–64.

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