'What is Anxiety and how should we treat it?'

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This paper provides an overview of this special issue on anxiety and attempts some integration. It concludes that a generally acceptable definition of 'anxiety' is lacking but that, even in the absence of such a definition, a coherent picture of both normal and pathological anxiety is emerging. The key problem in the past has been dissentangling a number of dialectically linked entities. It is argued that panic, obsessive compulsive disorder and eating disorders should all be viewed as intrinsically separate from anxiety - but that each can, on occasion, be both a cause and an effect of anxiety. Equally, even if it were possible to isolate a pure anxiety, uncontaminated by these other reactions, it is argued that this anxiety will include separate cognitive, autonomic, expressive and skeletal reactions each of which can arise for independent causes, and each of which tends to interact dialectically with the other. It is suggested that if account is taken of the dialectical interactions, between the components of anxiety and between anxiety and related emotions, it may be possible in the future to develop tests which can separate superficially similar symptomatologies into different cases requiring specific combinations of therapies.

It is now over 100 years since William James (1884) set the philosophical cat among the psychological pigeons with the question 'What is an emotion?', and even now we lack a definite answer to the more specific question 'What is anxiety?'. For emotion in general (McNaughton 1989a) and anxiety in particular (McNaughton, 1989b), I have argued that it is a mistake to try and answer such questions directly. First, they encourage us to try and answer philosophically questions which should be addressed via experimental data. Second, they encourage us to sift the experimental data for unitary entities. Neither of these is a fruitful approach to any emotion, as evidenced by a century of confusion. Indeed, for some time, 'emotion' developed such a bad odour within psychology that it dissappeared as a term from undergraduate textbooks - an amazing situation when many of us would see our emotional life as being the only truly interesting aspect of our psychology.

The contents of this issue allow us to take a somewhat different approach. Let us look at the wide variety of opinions and data dished up when people were asked to write a paper on a particular aspect of 'anxiety' - with no requirement that they should first address the issue of 'What is anxiety?' or agree on the answer to this question. From these data and opinions, I will in this paper try and extract ideas which can help us in the academic pursuit of the understanding of 'anxiety' and the clinical pursuit of helping clients with 'anxiety disorders'. It is my contention that once we have looked at the papers in this issue as a whole we can arrive at a view (if not definition) of anxiety and can extract some coherent principles (if not a complete theory).

Let us first summarize some key points from the different papers which will allow us to address the issue of 'what is anxiety?'.

Theoretical approaches

Strongman (this issue) suggests that theories of anxiety can be categorised as 'psychoanalytic, learning/behavioural, physiological, phenomenological/existential, cognitive, and those concerned with uncertainty'. Although Strongman does not emphasise this, it is clear that at the phenomenological level these categories are not mutually exclusive and, hence, that a major problem with theories of anxiety has been their tendency to focus on only one part of the problem. The result has been as incoherent as was the case in the parable of the different blind people each describing an elephant as they touched different parts of it.

It is also clear that each of the theories categorised by Strongman overlaps the boundary of its nominal category. For example, 'Freud's theory of anxiety ... can be characterised as based on the adaptive functions of anxiety and as being dependent on the cognitive processes that are a part of individual learning and appraisal'. This is not far from

the fundamentals of both the modern ethoexperimental analysis of anxiety or the modern cognitive analysis. Learning/behavioural analysts such as Eysenck clearly have a tendency to equate aspects of their models with specific biological processes and, although Strongman does not comment on this, the modern breed of behaviour analyst (e.g. Dickinson, 1980) sees themselves as not simply classifying S-R relationships but as determining, via behavioural observation, changes in the cognitive structure of an animal. (For example, a manipulation which changes behaviour at a later date is evidence of an internal change at the time of presentation even if there is no initial change in observed behaviour.) Likewise, it is worth noting that Gray's (1982) highly physiological theory of anxiety has its origin quite specifically in two-process learning theory - with the physiological work following on from learning theoretic analysis as an explicit attempt to uncover mechanism. The modern 'cognitive' theories emphasise 'the importance of taking into account the cognitive system as well as the physiological and behavioural' - but note that Strongman says 'as well as' rather than 'instead of'. In his treatment of uncertainty theorists, Strongman notes explicitly that they 'cut across the more traditional divisions'.

In this context the category of physiological psychology is most interesting. Strongman notes that 'physiologically based theories rely on a model of human psychology' and, as noted above, particularly in the case of theories such as Gray's you can not have a physiological psychology theory until you have at least some psychology to be physiological about. It follows also that for all substantive, testable suggestions made by any type of theorist (psychoanalytic, behavioural, existential, cognitive, uncertainty) there will be neural and physiological corollaries which can be tested and can aid in testing the psychological aspects of the theories.

McNaughton's (this issue) description of 'Brain mechanisms of anxiety', then, depends on a prior, at least approximate, psychological foundation. There is the DSM-IIIR definition of anxiety, which is actually insufficient to allow a purely biological analysis - and there is the refinement of this via ethoexperimental analysis and pharmacology. Although McNaughton does not make this explicit, his analysis of brain mechanisms implies the conflation of the different approaches to anxiety which we have mentioned above.

First, certain stimuli elicit behaviour reactions which can be categorised by ethoexperimental methods and which appear to be primarily controlled by specific brain areas. However, these stimuli elicit, and the brain areas control, activity in the autonomic and hormonal systems as well as the skeletal. These are less visible to the psychologist but there is good reason (McNaughton, 1989a) to view them as the fundamental basis of the 'feeling component' of emotions in general and anxiety in particular.

Second, both ethological and pharmacological approaches indicate that 'anxiety' can be induced by both innate adaptive mechanisms (linking back to some of Freud's ideas) and as a result of conditioning (linking back to two process, and other, learning theories). What is convenient about the analysis by Blanchard and Blanchard (1990), referred to by both Strongman and McNaughton, is that it allows us to categorise the difference between fear and anxiety - and see the relation between innate and conditioned forms of them. Fear, as paradigmatically the set of skeletal, autonomic and cognitive responses to a predator, has the evolutionary function of *removing* the animal from a dangerous situation. Anxiety, as paradigmatically the set of skeletal, autonomic and cognitive responses to a conflict between the possibility of a predator and the desire to approach some appetitive goal, has the function (via risk analysis) of ultimately *introducing* the animal into the potentially dangerous situation. Situations can clearly be assessed as 'dangerous' on the one hand and 'potentially dangerous' on the other either because of innate mechanims (e.g. the rat's innate anxiety response to the odour of a cat) or because of learning.

Third, both the emphasis of the biological analysis on 'risk assessment' as being fundamental to anxiety and the neural analysis implicating the frontal and cingulate cortices in anxiety indicate that, *in addition to* very simple stimulus-response relationships, anxiety can result from highly complex cognitive analysis - indeed any form of mental activity which results in the organism perceiving a source of potential danger. Here it should be emphasised that even in the simplest conditioning experiments, the rat does not 'learn the response'. If a rat is faced with a shock its immediate reaction is to vocalise and escape. Presented with a stimulus which predicts the shock it goes silent and still. Thus the conditional stimulus is not producing Pavlovian substitution - the moving forward in time of the response to the unconditional stimulus. The conditional stimulus elicits the expectation of shock - and the rat responds appropriately to this expectation. Thus, even in the simplest cases anxiety is best viewed as a consquence of stimulus evaluation and assessment. The fact that the cognitive evaluation involved is not complex does not stop it from being cognitive.

Here, we must clear up a possible misconception. Strongman states in his conclusion that 'anxiety is characterised by its genesis being ... uncertain to the individual'. The rat facing the smell of the cat is far from uncertain about the genesis of its anxiety. Likewise, the rat facing a conflict between the approach to food and the avoidance of a shock is well informed about the nature, location and contingencies of the relevant stimuli. What is uncertain is the best course of action to be taken. It is, then, the conflict between incompatible goals which creates uncertainty as to action tendency, not uncertainty about the nature of the source of the potential danger.

A final important message to take from these theoretical approaches, and particularly from the physiological, is that 'anxiety', whatever we mean by the term, encompasses skeletal, autonomic, hormonal and cognitive reactions. Further, the available neurology suggests that 'anxiety' as defined by DSM-IIIR may encompass a number of discrete but clinically overlapping entities with dialectically interacting control mechanisms. In what follows, then, we must be ready for a variety of different perspectives on what may, or may not, be a variety of different neural systems.

Origins of anxiety

Anxiety, as we have viewed it so far, is an adaptive reaction

to potentially dangerous situations. However, as a clinical entity, anxiety is maladaptive by definition. The clinical goal with an anxious patient is not, therefore, to eliminate anxiety (which could have quite lethal consequences in some situations), but to reduce proneness to anxiety or the amount of anxiety. Thus, the origins of clinical anxiety as a problem lie less in the situations faced by an individual than in that individual's predispositions.

It is the business of epidemiology to elucidate the underlying factors which give rise to morbidity and, hopefully, to provide means of controlling them. In the process, epidemiologists can provide us with interesting clues as to the nature and control of 'normal' anxiety.

Probably the most important feature of the data presented by Oakley-Browne (this issue) is that they give us no reason to believe that New Zealand populations are greatly different from overseas populations in relation to the epidemiology of anxiety (although on some measures different studies report markedly different rates). Further (and justifying post-hoc our efforts in producing this issue) prevalence rates are high, as is the number of health dollars consumed by persons suffering from anxiety disorders.

While different anxiety disorders tend to have different ages of onset, there is high co-morbidity between them and also between anxiety disorders and depression, mania and somatoform disorder. Oakley-Browne notes that 'when persons with anxiety disorders are found to have another anxiety disorder or other psychiatric disorder, at rates greater than expected by chance, doubts are cast on the discreteness of the disorders. There has been a strong argument put forward by some authors [for] ... a "general neurotic syndrome"'. This conclusion is at variance with the biological analysis summarised above - something we will attempt to resolve at the end of this paper.

While anxiety itself should be highly episodic (depending on the availability of threatening stimuli) it is interesting to note that anxiety disorders are frequently chronic with symptoms lasting over many years. This suggests some overiding endogenous factor giving rise to, or at least promoting, the disorder (or disorders). Nonetheless, there are some cases such as Posttraumatic Stress Disorder where a clear precipitating incident is part of the definition - and in the case of PTSD symptoms are as persistent as in other types of anxiety disorder. It could be (although Oakley-Browne does not suggest this) that the relatively early onset of phobic disorders and the high prevalence of social phobia in adolescents are the result of precipitating environmental factors. If this is so it dilutes somewhat the notion of a 'general neurotic syndrome' without reducing the possibility that there is some single underlying factor which predisposes to neurotic disorders.

The need for such dilution is also suggested by the epidemological data on children's anxiety disorders reviewed by Lodge and Tripp (this issue). It is true that certain 'childhood anxiety disorders' do appear to have a chronic course. These include overanxious disorder (generalised anxiety disorder in DSM-IV) which has a relatively late onset and obsessive-compulsive disorder. However, other disorders do not appear to follow such a chronic course, especially if they are treated early.

It seems likely from all these epidemiological data, then, that events can precipitate anxiety disorders, particularly in individuals who have been rendered prone to them - and, further, that chronic experience of anxiety could itself be a pre-disposing factor to the experience of subsequent anxiety. Given the early onset of many anxiety disorders, and the suggestion from PTSD that life events can predispose to chronic anxiety, it is clearly important to assess how far early childhood events are an important aetiological factor. At present there are no good data on this. Lodge and Tripp review data which suggest that 'increased negative self-talk and, to a lesser extent, decreased positive self-talk, is associated with heightened levels of situational anxiety in children'. Given the view that it is the evaluation of stimuli as potentially threatening which leads to anxiety, this opens the possibility that some cognitive bias in childhood can give rise to a tendency to anxiety disorder subsequently.

There are two major problems which need to be dealt with here. First, and this is the subject of much of Lodge and Tripp's paper, it will be important to develop means of accurately assessing the cognitions of children. There are major problems for self-report in adults which are greatly exacerbated in studies with young children. It may be that a useful transfer can develop here between learning theoretic approaches (in their modern guise of assessing cognitive structures in non-verbal animals) and conventional cognitive approaches (which face severe problems assessing pre- and para- verbal children). Second, is the chicken and egg problem. Extensive longitudinal studies of mind-boggling difficulty will be required if we are to tease out the relative contribution of, on the one hand, negative life events (or other incidents) which may increase the probability of negative cognitions and, on the other hand, the simple occurrence of negative cognitions themselves and the occurrence (consequential or otherwise) of autonomic changes; and if we are to separate such situationally-induced factors from any genetically or perinatally determined predisposition to later anxiety.

Cognition and anxiety

While it is clear that cognitive biases, if they are present, should alter the liklihood of anxiety, it is also known that both bodily state and expressive behaviour can impact on cognitive assessment (see e.g. McNaughton 1989a). Tripp, Tan and Milne (this issue) review a number of studies which show a correlation between anxiety disorders and negative cognitive biases - or more strictly between anxiety disorders and the lack of the positive cognitive biases present in non-anxious people. They also showed an increased tendency to attend to and search out threatening stimuli. That such biases can also be detected in non-clinical subjects in anxiety-provoking situations suggests that in at least some cases anxiety is producing the bias directly. There is a clear basis here for a dialectical, positive-feedback, interaction.

As noted by Tripp et al, 'elevated risk perception was also observed in the depressed subjects, suggesting heightened risk perception is associated with disturbed mood

generally, not just anxiety'. To analyse this issue further they developed the Risk Perception Questionnaire - a critical feature of which was the presence of two subscales 'Life Events' and 'Daily Stressors and Uplifts'. These separated out subjects' judgements of the liklihood of major infrequent and minor frequent events, respectively. Non-clinical subjects had an unrealistically optimistic view of the probability of occurrence of Life Events but, interestingly, had a realistic view of Daily Events. Despite this realistic overall assessment of the probability of occurrence, the subjects demonstrated significant (albeit modest) correlations between state anxiety and pessimistic expectations of daily risk and between trait anxiety and pessimistic expectations of both daily risk and Life Events.

By contrast, in a comparison of anxious patients, 'non-anxious' patients and community control subjects, they found that anxious patients had higher perceptions of both the liklihood of occurrence of negative events and the severity of threat than did community controls. However, the 'non-anxious' patients tended to show a similar pattern, if not to the same extent. Tripp et al say 'these data suggest that increased risk and threat perception for negative life events is associated with psychological stress generally ... [but] overestimating the probability of common annoying events may be unique to the anxiety disorders'.

To understand these results, it is worth returning for a moment to the ethological literature. As emphasised by the Blanchards, risk assessment behaviour is a critical feature of anxiety. However, this is not to say that the conclusions resulting from that risk assessment will be unique to anxiety. Let us consider the apparent relation between anxiety and depression in non-human animals. Perhaps the simplest example is the development of separation anxiety (which also has the advantage of showing considerable detailed homology with human separation anxiety). Removal of the parent clearly increases the risks faced by the young organism. It should logically lead to an increased perception of threat. This in turn should lead to behaviours (including risk assessment) which will determine the true level of threat and, if possible, reduce it. The initial reaction, therefore, is one of agitation and vocalization. If this agitated behaviour does not resolve the situation, the adaptive equation changes. Agitation uses energy and increases the probability that a predator will appear. In the longer term, therefore, (provided that the parent will not return soon) the most appropriate response is a behavioural and metabolic depression. A biphasic pattern of agitation followed by depression is, indeed, observed in a variety of species (e.g. Kaufman and Rosenblum 1969).

Provided that we can treat separation anxiety/depression as equivalent to anxiety/depression in general, it follows that in many cases of depression there will be increased perception of threat since anxiety and depression are both means of coping with potential threat and differ only with respect to the animal's perception of whether it can do anything to reduce the perceived level of threat.

While anxiety should, and does, produce changes in the perception of threat, it also produces more general changes in cognitive processes. As McDowall and Allison (this issue) note 'there is evidence suggesting that subjects who score highly on various measures of anxiety perform poorly on certain types of memory and learning task. ... For example, highly anxious subjects demonstrate less clustering in their recall of categorised lists, are poorer at the digit span task, and are slower to retrieve task relevant material'. They investigated the hypothesis that this result is due to a specific impairment of explicit as opposed to implicit learning - linking this specifically to the idea that explicit tasks are more sensitive than implicit ones to lesions of areas such as the hippocampal formation which result in an amnesic syndrome.

They found that normal subjects made anxious by the threat of a shock were significantly less explicitly aware of the pattern of a set of stimuli in their learning task. Overall, the anxious subjects showed no significant difference in the *rate* of implicit learning as assessed by reaction time although they showed generally slower performance. When only those subjects who were unaware of the pattern were analysed, a transfer test showed no evidence of any difference in the amount of implicit learning in anxious as compared to non-anxious subjects. These data tend to confirm others in the literature which suggest that even mild acute anxiety can affect cognitive processes which have no obvious emotional content - presumably because of a competition for limited intellectual resources.

Consequences of anxiety

Modification of cognitive processes can be viewed as an immediate consequence of anxiety (although it is probably better to think of it as simply a part of anxiety). However, in this section, we will consider longer-term consequences which can be thought of as resulting from, or being triggered by, anxiety - especially when the anxiety itself is of long duration.

We have already considered one possible psychodynamic consequence of continued anxiety (whether of pathological or normal origin) - depression. Here, the argument goes, the continuance of perceived threat should, and does, lead to an inhibition of active attempts to assess or remove the source of threat. Thus, all other things being equal, we would expect pathological anxiety to result in at least some depression.

Bulik (this volume) considers a second type of possible linkage - between anxiety disorder and eating disorders. She concludes that 'clinical and epidemiological evidence converge to suggest that a sizeable portion of women with anorexia and bulimia nervosa present with comorbid anxiety and that the anxiety disorder often occurred first'. Whether the pre-existing anxiety can cause or precipitate the eating disorders or shares a dependence on some common underlying risk factor is not clear. However, it seems likely that anxiety by itself is not the sole determinant of the observed linkage and Bulik suggests, for example, that only certain types of childhood anxiety (and indeed only one type of childhood social anxiety) will predispose to eating disorder. In other words, anxiety in the simplest sense needs to be coupled with some other factor to create the risk of eating disorder. Equally, for some cases of eating disorder prior anxiety may play no part in the aetiology at all.

Bulik notes the similarities between eating disorders and obsessive-compulsive disorder but points out that an 'important difference between bulimic and OCD behaviour is that the individual with OCD actively avoids situations in which they would be likely to encounter the feared stimulus. The bulimic woman, in contrast, via cravings or urges to binge, actively engages in binge-eating which results in the anxious state of fullness which she then counteracts by purging'. There also appears to be a 'genetic association between bulimia nervosa, panic and phobias'. Thus, OCD, panic/agoraphobia, anorexia nervosa and bulimia nervosa could all share a common general *pattern* of aetiology (with perhaps some overlap in specific aetiological factors). It is worth noting here that many researchers on the normal control of eating would now take the view 'that humans and other animals are not driven to eat by internal energy deficits; they are drawn to eat by the anticipated pleasure-producing effects of food ... The neural systems that control eating and drinking act to correct deficits, but that is not their usual function' (Pinel, 1993, p 322-323, 343).

Thus, compulsions, panic and eating can all be elicited by stimuli in the external environment. In each case, therefore, we can envisage a scenario where the occurrence of appropriate stimuli can elicit cognitions which then interact with a high level of state or trait anxiety to produce an unacceptably large response. A reasonable explanation of this interaction (in the case of pathological reactions) comes from the fact that anxiety (viewed as an adaptive responsive to the perception of potential threat) would be expected to increase compulsions (as in the case of a parent continually checking their child in a novel situation), to increase panic (which can be viewed as resulting from the confirmation that the potential threat is, in fact, actual) and to increase eating (due to the release of corticosterone by stimuli which predict threat). As Bulik notes, once reactions are occurring there could be further exacerbation of the symptoms by conditioning. For example, if a panic attack occurs for purely physiological reasons (Klein, 1980) in some place such as a supermarket this could result in anxiety on a subsequent occasion because of the association of the place with the prior aversive event. The anxiety could then increase the probability of the panic attack and a vicious cycle would be set up.

From this we can conclude that high levels of anxiety resulting from some behavioural or brain pathology could produce compulsive behaviour, panic or eating as a normal consequence and the latter can, given the right conditions, exacerbate the anxiety. Equally, high levels of obsessional behaviour, panic (in the most basic physiological sense) or eating which themselves result from some behavioural or brain pathology could produce anxiety as a normal consequence and the latter would then be expected to exacerbate the original pathological condition.

The implied dialectical interactions we have discussed make it very difficult to determine in any particular case whether the OCD, panic or eating disorder is a cause or an effect of anxiety (and the essence of true dialectics is that cause and effect cannot be distinguished from each other even at the theoretical level). However, there are some cases of anxiety pathology where cause and effect can be more easily determined - with post-traumatic stress disorder probably being the prime example.

As reviewed by MacDonald, Chamberlain and Long (this issue), PTSD involves 'the development of characteristic symptoms following exposure to extreme trauma ...[these] include persistent re-experiencing of the traumatic event, persistent avoidance of stimuli associated with the trauma, general numbing of responsiveness, and persistent symptoms of increased arousal'. All of these symptoms can be viewed as responses to perceived potential threat with the re-experiencing of the event being viewed as the cognitive/memorial equivalent of the risk assessment behaviour seen in animals faced with a 'potential predator' and the numbing of responsiveness being viewed as the equivalent of the behavioural inhibition. While some pre-disposing factors can be identified as increasing sensitivity to PTSD, MacDonald et al attribute the occurrence of PTSD largely to the extent of combat stress experienced by veterans.

What, then, are the consequences of this situationally-induced anxiety? Before we answer this question explicitly we should notice that, as with our discussion of OCD, panic and eating disorders, in PTSD we are looking not only at the effects of some anxiety-eliciting event but the long-term consequences of a failure to resolve the induced anxiety. This is because it is only those cases who cannot resolve the anxiety who are classified as having the disorder.

Not surprisingly, 'veterans with PTSD frequently experience ... major depression or manic disorder, other anxiety disorders and substance abuse'. The occurrence of major depression fits with the idea that depression is a likely consequence of a failure to resolve anxiety (of whatever origin) and the occurrence of other anxiety disorders fits with the suggestion made above that anxiety can predispose to obsession, panic and eating disorders (if these are latent), which can, in turn, exacerbate the general levels of anxiety.

'Individuals with PTSD are also likely to have poor physical health'. This fits with the fact that long-term anxiety and depression will both be associated with high levels of stress hormones which will, in turn, depress the immune system. There are likely to be other factors at work here too (and, of course, ill health may interact with environmental factors such as employability to increase the probability of anxiety or depression).

Overall, then, we can see that both acute and chronic anxiety can, in interaction with other factors, predispose to a variety of conditions, many of which can, in turn, feedback positively to produce further anxiety in a vicious cycle.

Pharmacology of anxiety

There are two separate ways in which we can approach the pharmacology of anxiety, academic and clinical. In this section we will look, from an academic point of view, at the light that pharmacology can throw on the normal control of anxiety. We will look later (and more briefly) at the clinical aspects of the pharmacology.

There are also two separate ways in which we can view the results of pharmacological research. Where compounds (such as the benzodiazepines and possibly caffeine) appear

to act in a neuromodulatory manner, they give us reason to suppose there may be endogenous compounds the circulating level of which exerts a hormonal control of anxiety. It seems likely that there will be both anxiolytic and anxiogenic agents of this sort. Where compounds of different chemical types have different effects on different symptoms of anxiety, they provide evidence for the presence in the nervous system of separate neural systems controlling those symptoms. This can then give us insight into the possible relations between those symptoms, each other, and anxiety.

There are a variety of chemical agents which can precipitate anxiety. This is most obvious with panic attacks which, as noted by Hughes (this issue), 'can be precipitated to a greater or lesser extent by injections of lactic acid and its salts or inhalation of carbon dioxide'. In the case of carbon dioxide, it seems likely that what is being administered is simply the natural stimulus which would normally elicit an adaptive panic reaction (i.e. when there is a real risk of suffocation). Lactic acid and other fairly specific panicogenic agents are probably acting more or less directly on the same basic mechanism. The main lesson to draw from this is that 'spontaneous panic' in the absence of prior anxiety, and with anxiety as a consequence, is something for which we must make allowance.

A number of stimulant drugs appear to be 'anxiogenic', at least in laboratory animal tests. In some cases it is likely that this effect is due to activation of the sympathetic nervous system. As Hughes notes 'if anxiety involves an unpleasant aroused state characterised by high levels of sympathetic activity in response to some perceived or anticipated threat, then drugs that increase sympathetic arousal might also be expected to at least contribute to outcomes resembling effects of anxiogenic stimuli'. There is evidence that such peripheral activation can, indeed, feedback on emotional state. There is considerable variation in the reported effects of peripherally administered adrenaline but these differences 'are explicable in retrospect by the presence or absence of anxiety-provoking environmental cues. In a neutral setting the injection of epinephrine (adrenaline) produced no emotional changes, "cold emotion", or "as if" emotion ... In an anxiety-provoking setting, intentionally or accidentally created by the experimenters, subjects showed more evidence of anxiety and in a minority of cases panic attacks were elicited' (Lader and Tyrer, 1975). The main lesson to draw from this is that cognitive and autonomic reactions can proceed, to some extent, independently of each other and that, just as the induction of certain cognitions can result in autonomic changes, the induction of autonomic changes can induce cognitions.

In some cases the actions of the stimulant drugs are likely to be as antagonists of endogenous anxiolytic compounds acting at the benzodiazepine receptor. The specific case of caffeine is discussed in this regard by Hughes. He makes the interesting point that, while high doses of caffeine probably do have a benzodiazepine antagonist action, 'acute doses that produce anxiety in otherwise non-medicated subjects can be significantly lower than the minimum required for benzodiazepine receptor blockade'. We will discuss the functional significance of benzodiazepine receptor occupancy when we discuss anxiolytic drugs.

Hughes considers the specific case of caffeine in considerable detail. Unlike many of the other potentially 'anxiogenic' drugs it is widely used and available and also, while being to some extent panicogenic, offers a 'fairly convincing model of generalised anxiety disorder' (Lader and Bruce, 1986, p 258 cited by Hughes) and 'regular consumption of high doses of caffeine can lead to a constellation of symptoms, referred to as "caffeinism", that are virtually indistinguishable from severe, chronic anxiety' although it is categorised as separate from anxiety by DSM-III and DSMIV.

It should be noted that caffeinism is the result of relatively high doses of caffeine. Hughes points out that within non-anxious psychiatric populations and in nonclinical populations there is little evidence for a relationship between caffeine consumption and anxiety. There is slightly better evidence for an anxiogenic effect of acute administration of modest doses of caffeine and even better evidence for an interaction between caffeine and concurrent sources of anxiety, including probably generalised anxiety disorder. These latter results are reminiscent of the peripheral effects of adrenaline discussed above and raise the possibility that caffeine is having an indirect effect on anxiety via modification of sympathetic reactions.

Hughes notes that the 'favoured interpretation for anxiogenesis (as well as caffeine's other behavioural effects) now involves blockade of the sedative (anxiolytic?) neuromodulator, adenosine. This proposed mechanism is particularly relevant to panic disorder sufferers whose sensitivity to caffeine may be mediated by supersensitivity of inhibitory adenosine receptors in response to chronic hyperactivity of excitatory neurotransmitters'. This opens up the possibility that caffeine's actions are multiple with direct neural actions precipitating panic and direct effects on the central control of the autonomic nervous system changing somatic state and hence indirectly producing a feedback effect on anxiety. The main lesson to be drawn from caffeine, then, is that there are multiple ways in which anxiety can be generated and a single substance (including, of course, endogenous hormones) can act through more than one route at a time.

We will now turn to the compounds which can alleviate anxiety. Silverstone (this volume) reviews 'drugs in the treatment of anxiety' and we will consider, here, the academic lessons to be drawn from such treatment. However, it should be noted that all the drugs used to treat clinical anxiety in humans also have homologous effects on behavioural tests in animals. This extends, with drugs such as buspirone, even to the requirement for long-term administration if a benzodiazepine-like effect is to be produced (Zhu and McNaughton, 1995). The drugs must, therefore, be viewed as simply reducing anxiety or one of its symptoms rather than reversing the pathology which is the primary source of the anxiety.

Silverstone notes that 'clinical studies reveal that there are four categories of anxiety-related disorders, each of which responds differently to centrally acting drugs. These are 1) simple phobias; 2) complex phobias and panic disorder; 3)

generalised anxiety disorder (GAD); 4) obsessive compulsive disorder (OCD)'. The simple phobias do not generally require drug treatment; agoraphobia (which usually presents concurrently with panic disorder) is treatable in the same way as panic disorder, i.e. with tricyclic or MAOI antidepressants (but *not* buspirone, despite its efficacy in generalized anxiety and unipolar depression); GAD is treatable with classic anxiolytics, with novel anxiolytics such as buspirone and with classic antidepressants such as imipramine; and OCD with 5HT re-uptake inhibitors such as clomipramine.

Of particular interest for the picture of anxiety we have been building so far, Silverstone notes that 'the subjective experience of anxiety is usually accompanied by widespread sympathetic discharge together with a steep rise in circulating catecholamines. These autonomic responses can reinforce the central awareness of anxiety and thus compound the symptoms' severity'. Particularly in cases of performance anxiety, peripheral blockade of the autonomic symptoms with a drug such as propranolol produces significant relief. This is the inverse of effect we ascribed to caffeine earlier. Although Silverstone does not mention this, the use of centrally acting anxiolytics is inappropriate here not only because of the risk of addiction with drugs such as the benzodiazepines but also because of the likely reduction in the quality of the performance. This is one of a number of cases where the arousal resulting from anxiety is largely adaptive but where some other aspect (in this case peripheral tremor and tachycardia) is excessive. The effects of the beta-blockers not only provide additional evidence that autonomic state can feed back positively onto cognitive state, but also suggest that at least part of the therapeutic effect of drugs such as the benzodiazepines could result from their peripheral muscle relaxant actions supplementing their central (possibly slower developing) anxiolytic actions.

The action of the benzodiazepines is of particular interest in relation to the endogenous control of anxiety. For many drugs (e.g. buspirone which acts at the 5HT1A receptor) it seems likely that its useful therapeutic actions are an entirely serendipitous accident of the nature and distribution of their receptors in the brain - since the endogenous ligand of the receptor is a locally released neurotransmitter. For example, buspirone acts at both postsynaptic and autoreceptors and so will combine the functional effects of 5HT release in some areas with loss of 5HT release in other areas in a pattern which would never occur under normal physiological conditions. The benzodiazepines, however, act at a receptor which modulates the behaviour of the GABA receptor - and can modulate it in either an agonist or inverse agonist direction (both increases and decreases in the effects of GABA can be produced). Benzodiazepine receptors are widely distributed in the brain - but are not a necessary feature of GABAergic systems. The benzodiazepines, then, show every evidence of behaving in the same fashion as some endogenous hormone or set of hormones which modulate the general tendency to anxiety and which do so by acting simultaneously on many different systems within the brain.

Here, again, we are left with the impression of a possible 'general neurotic syndrome' (including neurotic depression) which is marred by a number of specificities suggesting that, for example, panic and obsessions are quite distinct from anxiety proper and that anxiety (at least as defined by benzodiazepine receptor density) is a very complex entity even when it has been distinguished from panic and obsessions.

What is anxiety?

I suggested at the beginning of this article that it is a mistake to attempt too direct an answer to this question and a brief perusal of the papers in this issue should convince you that 'anxiety' can be many things to many people (see also Davitz, 1969). The anxiety *disorders* include panic and obsessive-compulsive disorder according to the DSM classification, but anxiety itself appears to be quite distinct from panic or obsession if one takes a neurological or psychopharmacological point of view. Many (e.g. Eysenck 1992a, 1992b) will see anxiety as a primarily cognitive phenomenon (with autonomic reactions being a minor consequence of the cognitions) and yet there is good evidence that autonomic changes can precipitate anxiety. There are many who would see anxiety as a reaction to a specific external perceived threat, and yet one of the primary characteristics of the anxious patient is not the presence of threat as such but what we would deem an excessive tendency to react to threats or to behave as if they are constantly being threatened.

I believe that our confusion in this area is from trying to obtain a clear picture of some central core construct of anxiety which at the same time encompasses all we mean by the term and excludes everything else. What we should be looking for, instead, is a pattern in a network of dialectically interacting entities.

It is quite clear that if we alter our cognitions appropriately (so as to perceive a potential threat where none was before) we can produce the autonomic and skeletal reactions which we would normally associate with anxiety. Further, in most cases, it is difficult to say that someone is anxious without implying that they are anxious about some particular cogntivie entity.

It is equally clear that if we alter our autonomic responses (with caffeine on the one hand or beta blockers on the other) we can alter cognitive and skeletal reactions which we would normally associate with anxiety. In this context it is particularly important to note that, at the cognitive level, the changes produced by anxiety not only include specific cognitions of threat but also include an increase in the probability of more threatening interpretations of events (negative bias) and by altering the processing of non-threatening information (attention).

We must expect then dialectical interactions, often producing positive feedback, between the various different components of, and symptoms of, anxiety. These interactions will occur with great rapidity - possibly so fast that the person experiencing the anxiety will be unable to tell what is cause and what effect.

However, the possibility of dialectical interactions does not mean that the different interacting components are always

tightly coupled. Eysenck (1992b, p1-2) discusses the implications of there being at least 'three separate response systems involved in anxiety. These three systems are the behavioural, the physiological, and the verbal or cognitive. Situations that would be expected to produce anxiety typically have effects on all three of these response systems. However, there is plentiful evidence that the three systems often exhibit failures of concordance, or agreement, and so cannot be regarded as equivalent'. At least one obvious reason for this is the different purposes and hence different expression rules which can operate. Indeed, there is a substantial negative correlation between people's internal (auntonomic) and external (facial) expression of emotion (Buck, 1979, 1980). In essence, if you withold your facial expression, the pressure has to go somewhere and it finish up in your autonomic nervous system!

There are also longer loop possibilities. It appears that panic can occur as a *symptom* of extreme anxiety. However, as we noted above, it appears that panic can also occur 'spontaneously' (e.g. as the result of a high level of carbon dioxide or some other precipitating factor) and that this can result in the conditioning of anxiety in the form of agoraphobia, which itself could increase the incidence of panic in a vicious cycle. Obsessions and compulsive behaviour, in the purest non-pathological sense, would often be expected to occur as a result of anxiety (especially where, as with infection, there are no explicit safety signals). However, as we noted above, there seems good reason to suppose that, in at least some patients, obsessions or compulsive behaviour can predate anxiety (and indeed may be neurologically similar to motor symptoms resulting from disorders of the basal ganglia) with the generation of anxiety (and a possible worsening of the obsessional symptoms) as an indirect result when the compulsive behaviour is blocked. In the normal person also, compulsive behaviour (such as regular washing of the hands) could be expected to occur in the absence of any detectable anxiety (and often as an unconscious habit) and anxiety would only arise when the performance of the safety ritual is blocked. In each of these cases we can see, in the pathological cases, overactivity in mechanisms which would, under normal circumstances, be of adaptive advantage.

There are even longer term effects which must concern us. It seems clear that there are certain classes of discrete event such as exposure to extreme trauma in adults (MacDonald et al, this issue) or separation from parents in children (see Suomi, Mineka and Harlow, 1983) which can result in long term ('trait') changes in the susceptibility to anxiety. There is also reason to suppose that long-term experience of unresolved anxiety can result in depression. Both of these types of change can be understood on functional grounds. Experience of extremely noxious events should give rise to an increased sensitivity to negative stimuli since, in many cases in the evolutionary past, the noxious event will be the result of insufficient notice being taken of warning signals. Likewise, given the major adaptive disadvantages of both too high and too low levels of anxiety, it makes sense that there should be a mechanism for adjusting long-term anxiety-proneness to the appropriate amount of risk-taking behaviour to maximise profit while minimising loss. Further, as we argued earlier, a failure to resolve a source of potential threat (e.g. absence of a caregiver) changes the adaptive equations and renders depression adaptive.

This brief overview leads us to a number of specific suggestions for the way we should approach anxiety.

At the theoretical level it suggests we must be wholistic. Changes in cognition are both a cause *and* a consequence of anxiety. Learning and related behavioural factors are clearly important for the initial occurrence *and* the consequences of anxiety. Physiological changes can be both the result *and* the occasion for anxiety. Our theories of anxiety must encompass, therefore, all the different types of theory which have been offered in the past - each of which has delivered only a limited perspective.

At the epidemiological level it suggests we must be dialectical. An increased risk of anxiety disorders appears to follow from distinct traumatic events as well as from genetic predispositions. The general association of different anxiety syndromes (and the unusually broad spectrum of action of some drugs such as imipramine) has led to the view that there may be a general neurotic syndrome and that, hence, the different DSM categories reflect symptomatological variations on a single underlying disorder. However, a moment's thought will show that both genetic and environmental factors can give rise to, say, high blood pressure, and that the latter can predispose to a number of correlated physiological accidents, without there being any direct relation between the different types of accident or the best treatment for them. On this view panic, obsession or eating disorder, for example, are quite distinct from anxiety itself. Although they will each be common consequences of anxiety, they can also be consequences of other events or conditions. On occasion, therefore, they may even be causes rather than consequences of anxiety. On many occasions, it is likely that both anxiety proneness and, say, panic proneness will interact to produce a disorder combining anxiety with panic when neither anxiety nor panic would have occurred in the absence of either one of the mutually potentiating contributory factors.

If we strip away panic, obsession, depression, anorexia and the like from anxiety with what are we left? In terms of clinical patients the answer may be 'nothing'. That is, there may be no such thing as a pure anxiety patient without some other attendant symptomatology. However, a case could probably be made for generalised anxiety in the absence of comorbid depression being 'pure anxiety'. At the academic level (where the stripping away is purely in the theorist's mind and not in reality) or with normal, situationally-induced, anxiety our residuum is: the perception of potential threat; autonomic and skeletal adjustments (including, in particular, risk assessment behaviours); and the interaction of these different elements.

It is the interactions between the elements which make even this purified view of anxiety a complex one. In particular the interaction of cognition and other aspects of anxiety allows for a form of positive feedback similar to that already described for panic where a modest tendency to autonomic reactivity and a modest perception of threat could each feed on each

other. Similarly a modest negative cognitive bias and modest basal autonomic activation could each feed on the other.

What, then, is anxiety? The answer is probably best left to the ethopharmacologists: that set of reactions of the organism which result when an animal must approach a source of potential danger. This answer does not guarantee that the reactions concerned have any common central control. It does not guarantee that any particular aspect (cognition, autonomic response etc) is primary. It does not even guarantee that the different aspects of reaction are all controlled by the same elements of the eliciting situation (McNaughton 1989b) and we must certainly be prepared for cases where social rules or other circumstances selectively suppress the output from one or another of the effector systems normally involved. What provides the term 'anxiety' with coherence is the nature of the eliciting situation. From this point of view, some reactions, e.g. panic, must be viewed as borderline. As part of the reactions involved in extreme anxiety it should be included. As a reaction which is not specific to anxiety it should be excluded. Whether we include or exclude it is really a matter of choice provided we are fully aware of the nature of the reactions that do occur in potentially threatening situations and are aware of the fact that panic can occur for other reasons and can itself cause anxiety. It should always be remembered that two emotions can occur simultaneously and produce a blend (e.g. Chevalier-Skolnikoff, 1973).

How should we treat anxiety?

This issue (with the exception of Silverstone's paper) has not addressed the treatment of anxiety and clearly provides no basis for commenting on specific details of treatment. However, I think the academic picture which has emerged from it, viewed as a whole, has some general implications for diagnosis and treatment.

The treatment of anxiety with drugs is covered extensively by Silverstone (this issue) and his paper should be referred to for details. Some general points should be emphasised here. As Silverstone points out 'when a patient presents with either the psychological symptoms of anxiety ... or its somatic accompaniments ... the first step is to establish whether or not there is an appropriate environmental cause. ... Recognition of the underlying cause may be itself of considerable therapeutic benefit. ... Symptomatic relief is the next step. Attempts should first be made to effect this using psychological techniques ... However, the symptoms may be so obtrusive as to make such an approach inappropriate as the initial treatment. In such cases treatment with an antianxiety drug (anxiolytic) is indicated.' It is also now generally accepted that administration of anti-anxiety drugs should usually be for the briefest time consistent with a therapeutic effect - a matter of days only for classical anxiolytics such as the benzodiazepines.

There is an apparent paradox here. With a high level of situationally-induced anxiety it may be necessary to apply a biological treatment - an anxiolytic drug. On the other hand with panic disorder (which has potentially the most biological origins), provided the symptoms are not too obtrusive, the implication is that drugs may be unnecessary. Certainly, cognitive therapy for panic will often be very effective (Franklin, 1990). We can reconcile this apparent paradox if we remind ourselves of the highly dialectical and distributed nature of the links between the different components of anxiety. All other things being equal, a disturbance (resulting from external stimuli *or* internal chemical imbalance) in almost any part of the system should result in some increase in activity across the whole system. Likewise a therapeutically induced reduction in activity in any one part of the system should produce at least some decrease in activity in most other parts. An improvement in a patient's condition does not, therefore, mean that the treatment has directly reversed the fundamental cause of the problem.

If the system is extensively dialectical, what are we to make of the apparent selectivities in the drug treatment literature: the fact that buspirone is effective in treating GAD, but not in treating panic; the fact that clomipramine is particularly effective in treating OCD? We account for this in terms of there being *some* patients who have a primary disorder of the periaqueductal grey (or of, e.g., brainstem sensitivity to carbon dioxide, Stein et al 1995) which results in panic and, consequentially, anxiety; and some patients who have a primary disorder of the cingulate cortex-basal ganglia loop which results in OCD and, consequentially, anxiety. In these cases, the pharmacological control of the anxiety may ameliorate the panic or OCD, but it may be insufficient to provide full symptomatic relief from the primary disorder. By contrast, a direct reduction in the primary problem will lead to a consequential reduction in anxiety. The specificities we observe, then, are not related to the core construct of anxiety which we have delineated, but of associated symptoms particularly in the cases when the 'symptoms' are in fact the primary disorders and the anxiety is a consequential symptom. Likewise, the generality of some treatments (such as imipramine) are a consequence of multiple effects of the drug - and it is generally accepted that the effects of imipramine on anxiety are unrelated to any concurrent effects it may have on depression in the same patient.

If, as we are suggesting, a range of different cause-effect patterns can all result in essentially the same symptom patterns then this could account for the fact that, even with the most careful differential diagnosis, there are many non-responders to one therapy who can be helped with some other therapy. Non-response occurs not only to specific pharmacological therapies but also to psychological treatment (which being targeted to the individual would be expected to be more generally effective).

Overall, then, our analysis suggests we must be prepared, in a clinical setting, for a number of potentially discrete disorders of which a preliminary list would be: pure panic, pure OCD, pure GAD, pure phobia. It is entirely possible, however, that pure panic and pure OCD will not generally present in the clinic since, unless they give rise to anxiety, the proband will simply adjust to the occurrence of the symptoms. The patient generally presenting with panic disorder or OCD would, on this view, have been predisposed to anxiety and it is the consequential occurrence of the latter that will result in their presentation in the clinic. (Or, they

may have a primary anxiety disorder which has released panic or OCD as symptoms.)

The predisposition to anxiety (that is, an increased susceptibility relative to some other members of the population) could obviously be inherited. However, it is clear that it can also occur as the result of stressors (the extreme case being PTSD) and it is possible (seeTripp et al this issue) that a cumulation of small, daily stressors, particularly early in life, could predispose to anxiety. If this is the case, the gradual worsening of OCD and the ultimate presentation of some panic disorder patients may be the consequence not of a predisposition to anxiety which antedated the OCD or panic, but of the gradual induction of 'neuroticism' by the cumulative effects of regular occurrence of the panic attacks or compulsions - or of the social consequences of these occurrences.

An important point about the stress induced by anxiety (which distinguishes it from many other stressors such as cold water) is that the source of the stress is in the eye of the beholder. Even in the lowly rat it is not the stimulus itself which gives rise to activation of the pituitary-adrenal axis but the animal's interpretation of the meaning of the stimulus (McNaughton, 1989a, p 58-59). It is also clear that, whatever interactions there may be between different effector systems within the body, the primary source of anxiety under normal circumstances will be the perception of some set of external events as potentially threatening. For whatever reason, then, that someone is anxious, we would expect the anxiety to be controllable if we can remove the cognitive evaluation of threat. However, we have already seen that anxiety (including situationally induced anxiety) can increase the tendency to perceive events of any kind as threatening and so it is clear that addressing specific anxieties may simply result in a transfer of anxiety to other stimuli. For a number of reasons, therefore, what is required of treatment (pharmacological or psychological) is less an alteration of anxiety itself than the production of a long term shift in the tendency to anxiety (ie the reverse effect of that achieved by traumatic events).

Overall, then, we must view pathological anxiety as potentially arising from a number of different sources. The dialectics of the system mean that we will not, in principle, be able to sort out cause and effect simply from symptomatology and, indeed, there may be many cases where no one specific cause can be identified. Rather, the typical patient may well suffer from the mutual potentiation of several different biases (cognitive or physiological) each of which, by itself, would not be problematic. This said, it seem very likely that in the near future we will be able to develop tests (e.g. Stein et al, 1995) which can separately assess specific components of the global anxiety system for their contribution to the general state of the patient and hence, perhaps, target treatments with greater precision.

Given this general picture, it appears likely that treatment will for the near future continue to involve a judicious mixture of pharmacotherapy, behavioural therapy and cognitive restructuring. The central problem, even if we have greatly improved diagnostic systems, may well be in deciding at which point in a complex interacting set of systems to intervene in any particular case - especially as the point in the system at which disorder is detected may not be the best point at which to target intervention. Given both the short-term and long-term dynamics of the systems involved a major challenge will be to determine what combinations of interventions produce cost-effective long-term improvements.

Conclusions

We are clearly some steps away from a generally acceptable definition of anxiety, far less a comprehensive theory. The complexity of the subject suggests, indeed, that a good definition will only become available once we do have a comprehensive theory. However, if we do not concern ourselves too much with definitional questions ('Is a virus alive when it has been crystallised?') and are prepared for a complex control system rather than some single control centre we can achieve a degree of understanding and, hopefully, increasing success in diagnosis and treatment.

Both academic theory and treatment must take into account:

1) dialectical interactions among the components of state anxiety - cognition, autonomic response, expression, action tendency - since each of these can increase the others;

2) dialectical interactions between anxiety itself and related emotion systems - panic, obsession/compulsion, eating - where the occurrence of one can predispose to the other, particularly as a result of conditioning;

3) dialectical interactions between state anxiety (which may predispose to longer term changes in trait anxiety) and trait anxiety (which will alter the probability of occurrence of state anxiety).

What is peculiar to anxiety and makes it difficult to dissect is that so many of these dialectical interactions involve positive rather than negative feedback, at least in the initial stages. However, it can dissected in selected laboratory cases. We must hope that it will become dissectable, and controllable, in the clinic in the future. All the papers in this issue embody attempts to move towards this ultimate goal.

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