CHAPTER 1

Concepts of Eating Disorders

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The nosology of mental disorders inevitably dithers between the wish to delineate useful categories and the hope of discovering natural kinds. It would be good to achieve both but each aspiration alone is elusive enough. Indeed, some would reckon the second hope to be forlorn and there has been a tendency to emphasise the pragmatic and the descriptive. The current classifications—ICD-10 and DSM-IV—are the offspring of this tendency (WHO, 1992; APA, 1994). Yet there is a nagging feeling that there are 'real' disorders out there to be discovered rather than merely defined.

Within the field of eating disorders, anorexia nervosa crystallised out as a separate and distinct disorder over the course of the last century (Mount Sinai, 1965). It had the advantage of one criterion that was both undisputed and easy to measure, namely low weight. However, it was the description of the characterising beliefs and behaviours that led to the disorder being separated off from other states with weight loss. Furthermore, it was the description of similar beliefs and behaviours in people of unremarkable weight that led to the definition of bulimia nervosa and its relatives. However, it is arguably when the definition of mental disorder relies upon the mental state—as it almost inevitably should—that classification becomes more difficult. Can we really measure people’s thoughts and feelings reliably and is it reasonable to expect that they should fit neatly into categories? Even classifying behaviour is problematic enough. However, if we do observe that people come to suffer in similar ways and with similar beliefs then this may give clues not only about sociological generalisations but also, perhaps especially, about innate and probably biological mechanisms which may underpin their disorder.

People may come to be more similar when they are stuck within a morbid process than when they are well because the range of their behaviour and experience is at least in part constrained by potentially definable processes in which such biological mechanisms are playing some limiting part. Tolstoy wrote, ‘all happy families resemble one another, but each unhappy family is unhappy in its own way’. This is questionable even with regard to families and unhappiness, but with individuals and disorder it seems likely that the reverse is true. The range of what is morbid is narrower than the range of the non-morbid. Anti-psychiatrists tend to emphasise the prescriptive nature of ‘normality’ and to portray the person who is ‘labelled’ mentally disordered as something of a free spirit. However, the
psychiatric perspective is different. The patient suffering from a mental disorder is seen as
constrained and trapped by forces that are outwith his or her control. It is the sufferer who
is the tram compared with the normal person who resembles the bus in having much more
freedom. Both the bus and the tram are limited by their physical attributes but the tram is
additionally constrained by the rails. Study of the patterns of disorder could give clues as
to the nature of these ‘rails’.

So what is the status of our current attempts at classification? What patterns can we discern
in people with eating disorders? How well do our conventional diagnoses map these patterns?
And do any of these patterns suggest the presence of plausible mechanisms of aetiological
significance? Do our categories promise to be more than convenient pigeonholes? Are there
‘real’ disorders out there?

What follows is a clinician’s view of our present classifications and some speculation
about what mechanisms and natural kinds might lurk beneath the surface of their syndromes
and diagnostic criteria.

**CURRENT CLASSIFICATION**

An ideal classification should consist of categories that are mutually exclusive and collect-
ively exhaustive. Its entities should be discreet and together they should cover the ground.
The classification of eating disorders measures up to these standards rather poorly. The
canon contains only two major categories—anorexia nervosa (AN) and bulimia nervosa
(BN). Anorexia nervosa has low weight as an essential criterion. Bulimia nervosa has binge
eating as a necessary criterion. The two disorders share the criterion of what in broad terms
might be described as an over-concern about body weight and size although some would
see a major difference in degree or emphasis in the typical ideas held by sufferers from AN
and BN. In DSM-IV, AN takes precedence over BN in the sense that the presence of the
former bars the diagnosis of the latter. In contrast in the earlier version, DSM-III-R, it was
possible to make the dual diagnosis of both AN and Bulimia Nervosa (APA, 1987). There is
in DSM-IV, however, a new subclassification of AN into binge–purging and pure restricting
subtypes. The rules in both of these sets of criteria represent different responses to the
fact that low weight and bingeing occur together commonly and that, hence, the cardinal
features of AN and BN are closely related even in cross-section. When longitudinal course
over time is considered then the overlap becomes even more striking. In many series, a
substantial minority of BN sufferers have a past history of AN. The reverse transition from
BN to AN is less common, but does occur. Thus, AN and BN are far from being entirely
discrete disorders and can be made to seem so only by dint of a certain sophistry. However,
if the classification of the eating disorders fails to meet fully the ideal of providing discreet
entities, it fails even more in respect of the second criterion, that of covering the ground.
Many people present with eating disorders that fulfil criteria for neither of the two main
disorders. How are these to be classified?

DSM-IV does provide two additional diagnoses, namely binge eating disorder (BED)
and eating disorder not otherwise specified (EDNOS). Binge eating disorder is included
only as a provisional category ‘for further study’. It is strictly a variety of EDNOS within
DSM-IV although, in practice, it has come already to be accorded the status of a diagnosis
in its own right. However in general, EDNOS is defined essentially by exclusion, that is as
being any clinical eating disorder that does not fulfil criteria for AN or BN.
THE PROBLEM OF EDNOS

The classification of the eating disorders achieves the standard of being collectively exhaustive only through having the ‘rag bag’ or residual category of EDNOS. The EDNOS category has only one positive criterion and one negative criterion. The positive criterion is that the individual being thus diagnosed should be deemed to have an eating disorder of clinical severity—a disorder that matters. The negative criterion is that the disorder should not fulfil criteria for AN or BN.

The EDNOS category thus defined is common. In many clinical series of people presenting to eating disorders services it is the single most common diagnosis and in some forms the majority of cases. Furthermore, as with AN and BN, the longitudinal perspective is illuminating but complicating. Many cases of the two main disorders change their characteristics over time so that those who have suffered from either at one time come later to suffer from neither but continue to have a clinically significant eating disorder (Sullivan et al., 1998; Fairburn et al., 2000). They can then be diagnosed only as being in a state of EDNOS. It is less clear whether people commonly move from a time of sustained EDNOS into one of the classic disorders.

A weakness of the EDNOS category resides in the limitations of its two criteria. The positive criterion is not defined. Where is the line to be drawn that defines a state as an eating disorder and of clinical significance? This is a matter of judgement. For instance, someone who is eating little and has lost a great deal of weight through severe major depression or because of delusions of poisoning would clearly have a disorder of clinical significance but would still not be diagnosed as EDNOS. The diagnosis is not appropriate because the state is not construed as an eating disorder. There is an implicit further criterion operating here; that is, that EDNOS should be diagnosed only if no non-eating disorder diagnosis is adequate. The positive criterion is further tested when there is uncertainty about whether an individual with eating disorder symptoms, such as maladaptive weight concern or self-induced vomiting, is affected to an extent that constitutes a disorder of clinical significance. Interestingly the judgement may sometimes depend upon the degree not only of the eating disorder symptoms but also of the associated non-specific symptoms. Thus, if the person has important associated anxiety and depressive symptoms or major problems of self-esteem—albeit not amounting to diagnosable syndromes in their own right—this may contribute to the decision that a diagnosis of EDNOS is appropriate. However, DSM-IV does not set out how these judgements should be made.

The negative criterion is also questionable when an individual fails narrowly to fulfil just one criterion for one of the major disorders. For instance, amenorrhoea is a difficult symptom to evaluate and yet some criteria demand that it should be present for the diagnosis of AN in females. The use of an oral contraceptive pill can complicate the issue and, furthermore, there is evidence to suggest that the presence or absence of this symptom makes little difference. Should someone who shows an otherwise typical picture of AN really be denied the diagnosis because of continuing menstruation? The ICD-10 system makes the sensible provision for a diagnosis of so-called ‘Atypical AN’ (or indeed ‘Atypical BN’) in cases where an individual narrowly misses fully meeting criteria but is clearly in a state very closely akin to one of these main disorders. However, once again these categories are not really defined and the atypical categories merely provide a buffer zone between the full disorders and others. There is still disputed territory at the other margin. In epidemiological work, the term ‘partial syndrome’ is often used to describe these sorts of states. The decision about
whether to count a subject as a ‘case’ in a survey may require a different kind of judgement to that of the clinician who must decide whether a patient fits a diagnosis. In the former case, the decision may affect aetiological inference; in the latter, the decision may influence the nature of the treatment offered. Sometimes whether or not treatment will be offered at all may be at stake. These things can be important.

Thus the EDNOS category inevitably includes some less severe cases that nevertheless pass the test of being of clinical significance. Many of these will be ‘partial syndromes’ of a kind that just miss out on fulfilling criteria for one of the main disorders. They will often do so in ways which may be quantitative—the bulimic who does not binge quite often enough—or qualitative, that is, their difference does not seem to threaten the essence of the disorder—e.g. the previously cited case of the female ‘anorectic’ whose periods persist surprisingly despite important weight loss. However, there will also be people who have disorders which are diagnosed as EDNOS but who seem to be caught up in patterns of difficulty that are qualitatively different in ways which do seem to be significant.

**ATYPICAL BEHAVIOURS**

Some unusual cases differ in terms of the behaviour that they show. A not uncommon clinical picture is that of the person who is at an unremarkable weight and does not binge. She is thereby barred by definition from being diagnosed as having either AN or BN. She nevertheless induces vomiting after almost every meal. This pattern is one of the examples of EDNOS cited in the DSM-IV manual. A similar behavioural variant would be the person who eats nothing at all because of fear of weight gain but sustains a fair body weight entirely through the consumption of calorific fluids. Another important condition is eating disorder associated with insulin-dependent diabetes mellitus which may sometimes be severe without involving either weight loss or bingeing. Omitting or using insulin erratically in the service of weight control may constitute a clear eating disorder and be life threatening without even approximating to either classic AN or BN (Peveler, 1995). All such states are truly atypical but nevertheless they seem to be sufficiently akin to the typical eating disorders that it does not offend our clinical sensibilities to include them as interesting variants. We seem to feel that in essence they are the same. Is this because we feel that the essence of the eating disorders lies in the beliefs and ideas of the sufferer? But what of people who are atypical in their ideas?

**ATYPICAL IDEAS**

Controversies about details notwithstanding, both AN and BN include among their necessary criteria the issue of what, for the sake of brevity, might be called ‘weight concern’. The different systems use different words but they all clearly refer to ideas which are at least similar. Furthermore, these ideas are held to be the central psychopathology of the disorders. They are deemed to be of the essence and to provide the motivation for the eating restraint which seems to be a key to the pathogenesis of AN and probably of BN too. And yet, there seem to be eating disordered people who do not have them or at least do not talk about them. Every clinician has come across many sufferers who initially deny concern
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about body weight and shape. Some later reveal that they had had such ideas but that they had been wary and kept quiet about them. Others continue to deny having such weight concerns. Some convince some clinicians that this is truly the case. But is it possible to have, say, anorexia nervosa without weight concern? The diagnostic criteria would say not. However, the clinicians who first described anorexia nervosa in the nineteenth century did not emphasise weight concern. Indeed, the early accounts by Gull and Lasegue do not mention it even though their clinical descriptions are in other respects both vivid and thorough (Mount Sinai, 1965). Likewise, colleagues working in China describe many young women who otherwise seem to have anorexia nervosa but who lack evident weight concern (Lee, Ho & Hsu, 1993). So sure are they that these are cases of ‘anorexia nervosa’ that they are so designated in the papers which describe them. So much for diagnostic criteria. But then, surely, Lee and his colleagues are following what most would regard as clinical common sense. Perhaps, such common sense rests upon an as yet unmentioned third attribute of a good classification—along with discreet entities and covering the ground—namely that of utility in practice. The Chinese patients without weight concern probably need to be managed in much the same way as their more typical equivalents. But what does such pragmatism do to ideas about the essence of eating disorders?

MOTIVATED EATING RESTRAINT

It is possible to make only a modest change to the diagnostic criteria for the eating disorders and thereby encompass some of the non-weight concerned sufferers. If eating restraint is promoted to be a central or even necessary component of the mechanism of the eating disorders, then weight concern may be seen as one motivation for such restraint among many that are possible (Palmer, 1993). For instance, restraint may be motivated by religious ideas, ideas of fitness, ideas of asceticism and so on. Many clinicians will recognise some patients for whom such ideas seem to occupy the same position as ideas of weight concern in more typical cases. They reflect the same ‘entanglement’ between ideas of weight and eating control and wider personal issues such as self-esteem and emotional control. Such atypical ideas may be more common in atypical sufferers such as males.

It is not difficult to think that motivated eating restraint might occupy a central position in the pathogenesis of the eating disorders. Restraint in some sense is clearly involved in AN. Furthermore, it may be plausibly invoked in BN via the kind of rebound effect that has been called ‘counterregulation’ (Herman & Polivy, 1984; Palmer, 1998). However, such explanations seem to require that the sufferer is fighting her natural urges to eat. She is seen as not having lost her appetite but rather as attempting not to give in to it—‘successfully’ in the case of the AN sufferer; unsuccessfully in the case of the BN sufferer. Indeed it may be thought an advantage of accounts of eating disorders which give a central place to eating restraint that they are parsimonious in having no need to postulate some primary disorder of appetite or drive to eat. The effects of eating restraint upon individuals with an intact appetite are well documented (Herman & Polivy, 1984; Polivy & Herman, 1995). Restraint leads to distortion. Indeed, a story can be told about these effects that can be spun into a plausible account of the eating disorders. However, although parsimony of explanation may be a virtue, the simplest accounts are not always true. There could be a place for some more primary abnormality of appetite. Surprisingly there is a deal of uncertainty about appetite in eating disorders.
THE VEXED QUESTION OF APPETITE

Hunger or appetite in eating-disordered people have received rather little systematic study. There remains considerable uncertainty. This seems to be for at least three reasons. Firstly, there are inherent difficulties in measuring the subjective strength of hunger or appetite. Secondly, ratings of hunger are likely to be unreliable in people who have complex and distorting ideas about what they should be eating. The sufferer may mislead others, and perhaps even herself, when putting her subjective experiences into words or filling in a rating scale. On the other hand, for obvious reasons, what an eating-disordered individual actually eats cannot be taken as a simple behavioural indicator of the drive to eat. Lastly, clinicians and other experts may assume that they know about hunger and the like in eating-disordered subjects. However, various experts have various views. Especially with respect to AN, some claim that they ‘know’ that sufferers characteristically experience an enhanced urge to eat which is kept under tight control (see many of the present author’s writings). Others say that the drive to eat must be less than normal if the subjects are to ‘successfully’ stop themselves from eating in the face of gross self-deprivation (Pinel, Assanand & Lehman, 2000). Many are impressed—or perhaps bewildered—by the variety of accounts which their patients give to them.

With regard to the problems of measurement or even description, there are conceptual as well as technical difficulties about what hunger or appetite or drive to eat may be taken to mean as definable terms. These terms do not seem to be used consistently or reliably and may need to be thought of as far from synonymous. For instance, an eating-disordered person may say that she is never hungry but may nevertheless acknowledge a strong urge to eat. It is as if the term hunger had too positive a connotation for it to be used about such a problematic experience.

In principle, hunger or the drive to eat might be abnormal in being reduced or increased. In practice, in many cases in which hunger is reduced—i.e. where there is true anorexia—a diagnosis of an eating disorder is not seriously considered. For instance, weight loss associated with physical illness with loss of appetite or depressive illness with true anorexia is not appropriately described as anorexia nervosa. The ‘nervosa’ implies that the relationship between the person’s eating and their weight loss is more complex—more entangled with wider personal issues—than that of being simply ‘off their food’. Once again, there is some lack of clarity here. Even those who would claim that AN sufferers do have a diminished appetite would want to reserve the diagnosis for those people who seem to be not eating for broadly ‘psychological’ reasons and who have relevant and related ideas often about weight concern. For instance, a sufferer may couch her immediate aversion to eating in terms of bloating or discomfort, but also have wider ideas of guilt or whatever. At the extreme, it is certainly conceivable that a person could present at low weight who was without both ‘weight concern’ and motivated eating restraint and who seemed to have some true anorexia. Under what conditions, if any, should she (or he) be considered for a diagnosis of AN? Strictly, such a patient should be diagnosed as EDNOS if no other diagnosis fits. Such people probably do exist although they seem to be scarce (perhaps they present to other kinds of clinician). However, their apparent rarity in practice may suggest that their characteristics should not be considered as threatening refutation of hypotheses about the nature of AN itself. Perhaps they are truly different. At the other end of the dimension of appetite or urge to eat, it seems likely that those who suffer from Binge Eating Disorder (BED)
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might also have an unusual—this time increased—appetite which is not based upon the distortions of restraint.

A primary increase in appetite or drive to eat might possibly be present in AN where it would trigger the restraint as a reaction. However, there seems to be little evidence for this. Such a primary increase is more plausible as a component of the mechanisms of BN and even more so for BED. In BN, the model of restraint acting upon an intact but unremarkable appetite is plausible. Most BN subjects report that the onset of attempted restraint preceded the onset of binge eating. However, this is not the case for a small but interesting minority of BN sufferers and for most of those who suffer with BED. Characteristically BED subjects either do not consistently restrain or the onset of their bingeing precedes that of restraint (Mussell et al., 1995, 1997). The different average outcome of BN and BED in terms of weight change at follow-up provides further support for a possible primary problem of increased appetite in the latter group. Fairburn et al. (2000) have shown that a community group of BED sufferers put on an average of 4.2 kg over the five-year follow-up period and that the rate of obesity rose from 22% to 39%. This weight gain occurred whether or not they continued to have BED. In contrast, BN sufferers gained on average only 3.3 kg from a lower base line and only 15% were obese at follow-up. Thus, many sufferers from BED are or become obese. Perhaps most are grappling with a drive to eat which is truly increased and which destines them for obesity, all other things being equal. Perhaps those who have a more ‘straightforward’ psychology become straightforwardly obese in the face of this increased drive to eat rather than becoming caught up in BED.

SET POINTS OR SETTLING POINTS?

Restraint-based models of eating disorder tend to go along with models of eating control which emphasise regulation of body weight. This may be seen as involving a regulation of weight around a set point or at least a set range that is variable across individuals but relatively constant for any one individual (Keesey, 1995). Some people regulate around a low weight, some around a high weight and most, by definition, around an average weight. Or so this story goes. The chief drive to eat is thought of as resulting from a biology in which even minor deprivation triggers the urge to eat in order to restore the well-fed state. Such set point models have an intuitive appeal. Furthermore, they can have an ideological utility in simplified form as the basis of a way of talking about eating disorders (Palmer, 1989). However, they have been criticised as not adequately accounting for important phenomena (Pinel, Assanand & Lehman, 2000). Especially, such models seem to overestimate the degree of the inherent stability of people’s body weight, especially with regard to the evidently widespread vulnerability to weight gain and obesity. Although there are anecdotes about Sumo wrestlers and evidence from studies that sometimes weight gain is difficult, for many people much of the time weight gain is all too easy (Sims & Horton, 1968). This seems to apply even to weight gain to levels that carry significant disadvantages for health (Pinel, Assanand & Lehman, 2000). The degree to which the bodies of many people ‘defend’ an upper limit around any set point seems to be less than the models would predict. There seems to be at least an asymmetry between the lower and upper limits. Any dieter knows this. Set point ideas seem to have merit with regard to downward deviations in weight
but are rather less good in accounting for weight gain above ‘normal’ levels. In as much as an eating disorder involves low weight and restraint, set point models may be useful. However, this may not be the case with respect to eating disorders at normal or above normal weight.

Set point ideas are often dressed up with evolutionary stories. It is suggested that regulatory mechanisms would have evolved which tended to keep an individual within a range which was optimal for survival and reproduction. However, a criticism of set point theory suggests that in the ancestral environment, where food would have been scarce, mechanisms would have been favoured that allowed an animal to eat more food when it was available than would be necessary for its immediate needs. Storage of potential energy and substance—putting on weight—would be advantageous in circumstances of erratic food supply in a way that would not be the case for strong satiety mechanisms which cut consumption when immediate needs were met. Furthermore, it is plausible that such permissive mechanisms might be more advantageous for younger females of reproductive age and, indeed, some sexual difference in satiety mechanisms can be observed (Goodwin, Fairburn & Cowen, 1987). But if restraint models are not fully adequate, what other models are available? One is that of so-called positive-incentive theory. This emphasises the rewards of eating, including its hedonic properties. Feeding is intrinsically rewarding and this is especially the case with respect of foods which might well have been valuable but scarce in the ancestral environment such as sweet foods, fatty foods and salty foods. Eating such foods was—and of course still is—especially rewarding. In the past this meant they were especially sought out despite the difficulty in finding them. Now that they are readily available, they are eaten to excess. Positive-incentive theory may hold more promise in explaining aspects of those eating disorders in which restraint seems to play little or no part and which occur at normal or high body weight. There may be complex entanglement between the hedonics of eating and emotion in people with binge eating. And less dramatically the positive incentives may be relevant to obesity.

Pinel, Assanand and Lehman (2000) have proposed a tentative theory of anorexia nervosa in which they suggest that the under-eating characteristic of that disorder may reflect a change of the usually positive incentive of eating towards the negative. However, it is not clear that such an interpretation fits the facts. Thus, as mentioned above, there is controversy about the nature of the subjective urge to eat in AN. Furthermore, it seems highly plausible that deprivation might well be the key drive to eating in those who are at a low weight and hungry and that the positive incentive to eat might well take over when the animal or human is well fed. Sensory specific satiety is a real phenomenon and bread and butter may well suffice when one is deprived, but it takes chocolate pudding to override that full feeling after two or three previous courses. While it may seem more parsimonious to invoke either a set point theory or a positive incentive theory, perhaps both kinds of ideas are required; the first in discussing states of deprivation and weight loss and the second in discussing the regulation of eating in times of plenty and higher weight. It is at least as easy to tell evolutionary stories around such a dual mechanism as it is around a simpler model.

The notion that there is a mechanism that regulates body weight around a set point may be contrasted with the idea that any apparent stability of body weight reflects a settling point which is the net result of two or more mechanisms that may have quite different functions. The implication for intervention may well be different, perhaps especially for the treatment of obesity where the idea of a set point that is defended even
when it is problematically high tends to promote therapeutic pessimism (Garner & Wooley, 1991).

CONCLUSIONS

Our cherished diagnoses of AN and BN are here to stay. They clearly describe many patients in the clinic and are useful. Furthermore, the use of definite diagnostic criteria has made an important contribution to research. However, an undue concentration upon individuals who fulfil diagnostic criteria may lead to a somewhat blinkered view. The testing out of new formulations such as that of BED is useful although it would be a pity if such categories invented ‘for further study’ were routinely and prematurely reified as diagnoses. We need ideas to inform our observations but to be sufficiently open-minded to be able to notice the unexpected.

The view from the clinic can potentially provide suggestions about where it might be profitable to look for more basic physiological and pathological mechanisms.

Returning to the metaphor used above, it may be possible to guess at the location of some of the ‘tramlines’ that constrain our patients. The following are some summary comments based upon the view through this particular pair of eyes.

1. In looking for mechanisms underlying the eating disorders, ideas which invoke essentially normal regulatory mechanisms which have been pushed out of kilter are to be preferred as more parsimonious if they are adequate.
2. Models based upon eating restraint seem to have merit and may even be adequate for most cases of AN and BN.
3. ‘Motivated eating restraint’ is a more inclusive and arguably better formulation than ‘weight concern’ as the criterion for the core psychopathology of most eating disorders.
4. The ‘normal mechanisms’ invoked may need to include positive-incentive ideas as well as or instead of ideas of restraint if eating disorders at normal or high weight are to be adequately explained.
5. True abnormalities of appetite or drive to eat may play a part in some cases of BN, in BED and in obesity. Likewise, some cases of restricting AN may have some primary change in appetite although this is more speculative. Such variation of appetite may be genetically determined.
6. There should be more research into the difficult topic of the phenomenology of appetite in the eating disorders.
7. Such research should go hand in hand with biological research into the complex mechanisms that are doubtless involved in normal and pathological feeding in animals and human beings.
8. Whenever practical, research should include atypical (EDNOS) cases as well as the typical.
9. A future classification may include a major divide between ‘disorders of restraint’ and ‘disorders of increased appetite’.
10. All true eating disorders—disorders with ‘nervosa’—are characterised by an ‘entanglement’ between the relevant basic weight and eating control mechanisms and the sufferer’s interpretation of the meaning of the effects of these within his or her own
individual experience. And, although some generalisations can be made, such interpretations are likely to be varied or even idiosyncratic and to defy neat classification.

REFERENCES


