'Depression by any other name . . .'

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Summary

There is a popular conviction that psychological disturbance is invariably a reaction to environmental events. This approach is associated with total rejection of the so-called 'medical model'. However, allowance must be made for the fact that contemporary knowledge of psychiatric illness is well behind what is now known about general medicine, and current ideas about psychiatric aetiology may be emotionally determined to give satisfying concepts which are often almost impossible to refute.

By tracing the progress of some hypotheses in general medicine, as they developed in the past, there are similarities with some current observations about the methods of action of physical treatments in psychiatry, treatments which have been discovered empirically.

There is a widespread assumption that psychological responses relate precisely to the degree of evoking stress or the patient's personal attitudes to the stress. However, emotional responsiveness may show an intrinsic individual variation so that many patients who over-respond to stress may do so because of enhanced responsiveness rather than necessarily because psychological events have brought about a state of vulnerability.

The exclusively dynamic approach to psychiatry has been perhaps unnecessarily encouraged by naming an illness 'endogenous depression' which could well be a primarily physical metabolic disturbance in the central nervous system but which happens to present with prominent depression, as well as other abnormalities. The emphasis on the component of depressed mood artificially delineates the illness and tends to impede consideration of its wider implications.

To suggest that our knowledge of psychiatry lags perhaps a century behind that of general medicine would be unlikely to occasion much dissent. However, the treatment of psychiatric conditions has been showing signs of significant advance while the understanding of aetiologies is coming along more slowly and painfully, the controversies engendered in the process producing a sense of déjà vu when placed in historical context. Not surprisingly, many

of the issues have been tossed about before in other branches of medicine (with the same concurrent aggression no doubt) although not everyone caught up in the contemporary fray appears to be able to recognize this and obtain profit from it.

Whilst deductions and hypotheses in medicine over the ages have subsequently been revealed as of highly variable quality, sometimes to the point of absurdity, observations of clinical phenomena have usually been remarkably accurate even from earliest times. The former, whilst presenting illusions of objectivity, can easily become entangled with emotional attitudes and social fashions, while this is less likely of the latter.

The starting point of medicine has usually tended to be from symptoms and signs, which can be complained of, seen and described. But the journey from perceiving presentations to understanding aetiology have been strewn with errors and are likely to prove even more ridden by obstacles in psychiatry where clinical signs so far remain relatively inconspicuous.

Let us take an historical example from general medicine. Dropsy has always been an easily recognizable condition, described for centuries. But the great Thomas Sydenham (1624-89) was able to offer only vague ideas about its causes. He wrote in his 'Treatise on Dropsy' (1683), 'however, that there are secret passages thro' which the waters are conveyed from the cavity of the belly to the intestines is manifest; for daily experience shows that hydrogogues carry off as much water downwards, as if it were originally contained in the intestines themselves'. 'Is manifest', indeed! And one wonders just what 'daily experience' showed hydrogogues carrying off water. Of course, he was not in possession of sufficient information and his proposition was therefore insecurely based—a readily recognizable situation from the viewpoint of contemporary psychiatry. This is an early example of the academic hypocrisy of making hypotheses appear as established facts.

William Withering (1741-99) would have been able to do better. He learned in 1775 from an old lady in Shropshire that an extract of the purple foxglove was an effective treatment for dropsy and

he found that it was of value in cases of heart disease but it did not cure all patients with dropsy (Clendening, 1942). This early association between some cardiac conditions and oedema began the transition from a symptom-orientated concept to an understanding of aetiology via the empirical discovery of effective medication, which was further advanced by the classical report of Richard Bright (1789–1858) implicating renal disease in some cases of dropsy (Singer and Ashworth Underwood, 1962).

A progression becomes apparent. First, a physical sign is regarded as a disease-entity because it is the only means of recognition, then by the discovery of treatment (in which the foxglove cures some but not all cases of dropsy) the original disease-entity is no longer tenable. Later, by more searching observation, other associations of the physical sign may be found, from which a potential differential diagnostic classification becomes possible (clinical differentiation between cardiac and renal oedema). Subsequently, detailed scientific study produces precise physiological information with the discovery of the fundamental processes involved (the physiology and pathology of oedema become understood) (Fig. 1).

Now, dare we substitute 'depression' for 'dropsy'? There are protests at once. The mind is not the body, with psychological phenomena you cannot be dogmatic, you cannot employ organic concepts, each personality is unique, emotional experience is not amenable to classification. The 'medical model' is inappropriate for the affairs of the human mind (a sentiment which recalls nothing so much as religious resistance to early tentative scientific advances). Implicit in the contemporary insistence that diagnosis in psychiatry is a sterile exercise is either that all psychiatric patients need all types of treatments, only one, or else none. The process of diagnosis can be seen simply in terms of an attempt to decide which therapeutic approach is most appropriate.

One wonders why there is such a widespread need to believe that the mind reacts only to the environ-

ment and, unlike all other physiological systems, cannot be vulnerable to internal metabolic processes as well. How else can the mind function except by physiological processes of some kind—for all that they themselves may be modified by experiences? Contrary to increasingly noisy contemporary protestations, psychiatry may have much to learn from the theories of general medicine.

There is a further difficulty with regard to depression, a semantic one. We use the word subjectively to include all kinds of psychological states, including disappointment, boredom, feeling fed up, unhappy or miserable as well as for extreme despair. Sandison (1972) pertinently suggests that depression is 'a thin word, declaring a position of hopelessness and despair. It contrasts with . . . melancholy, the black humour, for, as a 16th century physician put it, "melancholy occupies the mind and *changes the temperature of it*"' (P.K.B.'s italics). So, when various emotional states are described as depression, how much have they got in common? Can 'depression' describe, as often suggested, a totally pathological emotional experience (endogenous), an enhanced degree of the emotion beyond the normal range (neurotic) as well as an adaptive and normal, if uncomfortable, emotional state (e.g. grief)?

Whether there are essentially different types of experiences called depression or whether there is a continuum of depressive emotion would be hard to resolve in the present state of knowledge, but this aspect probably matters less for the moment than a consideration of possible causes. After all, breathlessness might be considered either as a continuum or as consisting of several separate experiences of respiratory difficulty, but there are nonetheless all kinds of different implications in the breathlessness of a fit person after strenuous exercise, of someone with cardiac insufficiency after mild exertion, and of a patient with pneumothorax at rest. In none of these circumstances would a disease-concept called 'dyspnoea' be of much clinical value, although it

FIG. 1.

could be postulated that the first was obviously 'reactive', the second due to an inability to cope with a specific stress and the third 'endogenous'!

By committing ourselves to calling a syndrome 'depression' we equate the symptoms with the abnormality and confine the entity only to those cases presenting with prominent depression. Returning to the original example, it is similar to the situation in which, because oedema is so readily apparent, heart disease is only diagnosed if oedema is present, and those cases without detectable oedema will be excluded. With increasing knowledge, it becomes clear that oedema is not the *essence* of cardiac disease but is only one secondary aspect. Analogously, perhaps depressed mood is a frequent but not invariable presentation of an uncharted illness not necessarily associated only with emotional disturbance. It can be argued that if depression is absent how can a depressive illness be diagnosed? This is just how we are committed by a name. There are cases, increasingly recognized now, which present primarily with, for example, pain, anxiety or anorexia and which respond to similar physical therapies as do cases with obvious depression; treatments such as electroconvulsive therapy (ECT) or anti-depressant medication. It is at least likely, therefore, that they are all presentations of a similar but as yet unknown basic pathology, clues about the nature of which are becoming available.

As with the use of foxglove for dropsy, physical treatments for depression may be the next stage in elucidation of the origins of the symptom. They offer at least one piece of objective information, available since the 1930s but relatively little emphasized, that some cases of depression respond decisively to, for example, ECT while others do not. The apparent therapeutic specificity of the anti-depressants, unlike the non-specific stimulation of the amphetamines, points in a similar direction. Just as there was a 'foxglove-responsive' oedema which subsequently proved to be of cardiac origin, there appears to be a form of depression readily responsive to physical treatments, suggesting a specific cause.

This possibility underlies the proposed dichotomy between exogenous and endogenous forms of depression which has produced one of those irresolvable controversies which seem endemic in psychiatric writing (Lewis, 1971). The clinical aspects of this problem have tended to resolve round the difference between 'reactive' and 'endogenous' causation; the former term being used when there has been a sufficient apparent cause for the illness. Reactivity is probably the least reliable feature as a basis of differentiation because, apart from the difficulty in deciding upon the validity of alleged cause and effect, reactivity to circumstances is a feature of all human responses and conditions, and a

conscious acceptance or rejection of a cause, either on the part of patient or doctor may, in fact, be the real issue in the 'reactive' illnesses. Furthermore, it is possible that there is a considerable delay between the causal events of a depressive illness and its manifestation, which would obscure the association.

Does cold really produce pneumonia? Does dampness cause rheumatism? Does adversity result in depression? These questions tend to reflect traditional beliefs and expectations. After all, it is presumed that we no longer expect masturbation to result in schizophrenia, although this was a strongly held medical belief, rejection of which would have been quite heretical less than a century ago (Hare, 1962). However, to the other questions posed there may be similar answers—'sometimes yes, especially in association with other factors in the vulnerable or predisposed'. So individual response, the amplitude of which will vary, may be as important as the circumstances that produce it. The response is specific to the individual, the causative circumstances are general, relatively non-specific and frequently unavoidable anyway. But it is true that their effect may sometimes be enhanced because of special psychological significance to an individual. Experience with the treatment of the so-called psychosomatic illness has suggested that modification of physiological responses may, in many cases, be therapeutically easier and more realistic to achieve than significant stabilization of the personality.

With regard to the generality of stress it should be remembered that severe depressive illnesses can result from such disparate events as childbirth, influenza and taking reserpine, as well as from grief. This emphasizes that metabolic changes can so alter emotional responses ('change the temperature' of the mind) that the stresses of life are experienced as intolerable whereas in other circumstances, on the basis of a more stable metabolism, the individual would be able to cope. In such cases it may be impossible to distinguish between the alleged cause of the illness and its early manifestations. A middle-aged patient may become profoundly depressed which may be attributed to the fact that her children have grown up and left home. This may be true but there may be yet more truth in the possibility that she is passing through a period of neurophysiological mood instability associated with menopausal endocrinological changes which render her specially vulnerable at this time so that the emotional response to the loss of the support of her children becomes overwhelming. Of course, in these circumstances the patient needs support and help with readjusting to a new, apparently less rewarding life but this is a process that may be quite unsuccessful if her mood is not stabilized by means of antidepressant medication or ECT.

The exact contribution of early experience to the subsequent onset of depression is unclear (Birtchnell, 1970; Munro, 1966). The psychodynamic approach to psychiatric conditions traces causes from early life, quite reasonably, but makes little or no allowance for differences of individual emotional responsiveness (Bridges and Jones, 1973). For example, it is a common observation that some individuals are naturally placid and some over-excitable. It is surely worth considering that many patients show intrinsically enhanced emotional reactions to situations that others, who are constitutionally less likely to be emotionally overloaded, adapt to more readily.

It is often assumed that the emotional response is directly related to the degree of emotional stress and the intermediate variable of individual responsiveness is usually little taken into account. If the response is directly related to the stress then the stress is of fundamental importance, whereas if there is a variability in emotional responsiveness, then this may be relatively more important than the evoking stress. In the latter case the response is likely to require more therapeutic attention than its stimulus, which is a justification for emphasis on physical treatments in appropriate cases.

With their almost exclusive preoccupation with the detection of causes located in the individual's past, the psychodynamic schools have tended to ignore relative responsiveness which has led to a tendency in clinical psychiatry to become preoccupied with obsessively complete histories, while offering little guidance as to priorities and emphasis. Within their histories it is often difficult to see the wood (of that which is of clinical significance) for the trees (of historical details). One can, however, feel reassured that they contain that which is being sought—but where is it and how much is really relevant?

The difficulties experienced with the reliability of psychiatric diagnosis are often blamed upon false theories but it is even more likely that the intuitive,

descriptive methods that have to be employed at the moment are just too crude to succeed with any accuracy and should be accepted as such without too much over-defensiveness. Learning this lesson from the past would save a lot of polemics and associated autonomic wear and tear for the disputants.

However, progression to more valid concepts about depression could follow as in Fig. 2. This takes into account the possible specific alleviation of some forms of depression by anti-depressant medication, which has been mentioned. It proposes that there are other forms of depression with other causes and associations, and there is likely to be considerable overlap which cannot as yet be resolved as our methods of delineation are too crude. One may as well try to differentiate anxiety states from thyrotoxicoses, for example, without means of measuring thyroid function. By careful observation, it may be possible in some cases, but most of the others will be in doubt.

Another example of the fallacies attendant on preoccupation with symptom-syndromes is to be found in the relationship between anxiety and depression. Traditionally these two symptoms have been regarded as essentially separate although they may overlap, and each had its particular methods of treatment. However, clinical experience now suggests some cases of both anxiety and also of depression have a common aetiology on the basis that anxiety can respond to the same physical treatments as do some forms of depression. On this proposition the original concept of two clinical entities, synonymous with symptoms, then becomes rearranged so that the new postulated syndrome cuts across the previous classification (Fig. 3).

The implications of a name for a syndrome or disease became increasingly apparent. It can artificially narrow the clinical concept. 'Endogenous depression' may not primarily be about depression at all just as asthma is less to do with breathlessness

FIG. 2.

FIG. 3.

fundamentally than with abnormal autonomic activity, although its presentation focuses the mind on respiration. Endogenous depression seems to be a condition with genetic associations which may involve a cerebral biochemical vulnerability (Medical Research Council, 1972). But because it commonly presents with depressed mood, which is a readily understood experience, there has been a tendency to assume that it is exclusively a psychological illness with psychological causes. We expect to feel unhappy when we are lonely, bereaved or disappointed, therefore all patients who complain of unhappy feelings must inevitably be lonely, bereaved or disappointed, and in the absence of acceptable environmental causes, we set about finding the 'deep' reason for the unhappiness which will accord with our expectations. The associated symptoms and signs in fact suggest that the illness could involve hypothalamic malfunction (Pollitt, 1965; Davies, Carroll and Mowbray, 1972). A new, neutral name would help in reconsidering the illness with less prejudice.

'Endogenous depression', as we must call it at the moment, could be a psychosomatic condition in which a primary disturbance of organic function may be enhanced, but is not necessarily primarily caused, by psychological stress. The relevance of psychiatric factors to the group of so-called psychosomatic illnesses is now being seen in better perspective. Psychological stresses are just one example of the range of stresses that disrupt vulnerable metabolism in patients with these conditions. While psychiatric treatment, both psychotherapeutic and psychopharmacological, can often reduce the effects of stress, to

try to conceive a life without stress and conflict is unrealistic. It should be remembered that intense pleasure may be as stressful, in physiological terms, as adversity. So one aim should be towards identifying and modifying vulnerable areas of metabolism to avoid, as far as possible, the patient being overwhelmed by adverse circumstances which may be inevitable.

These ideas are not meant to have relevance to the whole field of psychiatry and psychology. They are an attempt to suggest that the medical model has importance for some aspects of mental health and is not to be lightly discarded in a wave of sociological enthusiasm for the idea that we are only the products of our environment with an exclusive need for self-expression and revolution rather than phenothiazines and behaviour therapy. Furthermore, it must be pointed out that the tremendous advances in the treatment of psychiatric patients that have occurred in the past 25 years or so have depended much more upon the discovery of new drugs than upon the exploitation of psychodynamic concepts, old or new.

Thus from the hypotheses and methods which can be learnt from general medicine, more specific psychiatric treatments may be discovered and, just as important, methods of more accurate diagnosis may be found by which the most effective type of therapy can be selected in relation to individual cases. For some patients physical treatments will be decided upon with confidence as to their relevance and chance of success. This leaves other areas of emotional distress and social maladjustment to be dealt with by different means. Certainly a few patients will require

a combination of different methods—psychological, behavioural and physical. But the allotment of resources and their relative priorities should depend upon as careful a clinical assessment as can be achieved in our present state of knowledge. Everyone does not require the same type of treatment but neither, alternatively, should every patient have a little of all therapies. Resources and research will be most effectively employed in attempting, first to break down problems into disparate aspects, and then to investigate appropriate specific solutions. Psychiatry has now matured beyond a need for the security of a single system of beliefs which purport to explain everything, absolutely everything (Slater, 1975).

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