Integrating critical psychiatry into psychiatric training

D B Double

There is an orthodox medical approach to the problems of interpreting and treating mental disorders. The argument of this chapter is that any challenge to this orthodoxy is suppressed by mainstream psychiatry. Over recent years, criticism of psychiatry has been marginalised as "anti-psychiatry". Voices against the harmful and inhumane tendencies within psychiatry are seen as disreputable. This chapter comments on this remarkable situation and argues that critics of psychiatry should be heard with more understanding and empathy. Self-questioning is required in psychiatric training.

Some psychiatrists are honest enough to acknowledge a rift in practice. For example, a psychiatrist, known for his support of user groups and critical approaches, was a visiting speaker at one of the regular fortnightly Tuesday evening meetings in Sheffield, which took place over many years under the auspices of Professor Alec Jenner of the Sheffield University Department of Psychiatry. Despite the overt radicalism of the speaker's talk, he confessed that as an academic psychiatrist responsible for the training of medical students and psychiatrists he adopted a required conventional mode in his teaching. He did not, however, believe in its content. In particular he thought that he had to teach certain requirements to enable trainees to prepare for the examination of the Membership of the Royal College of Psychiatrists. The unfortunate fact is that once many psychiatrists have passed their professional test, they still do not adopt a properly alert practice. Their sensitivities have been eroded by their training.

Similarly, a senior registrar in psychiatry, who had not long managed to obtain her first consultant psychiatrist appointment, once ruefully remarked to me that she had become "irretrievably biological" in her approach to psychiatry (Double, 2001). Although this is regarded as an acceptable outcome of her training, she was not able to deal with any criticism of psychiatry. It is reasonable to expect psychiatric trainees to be able to consider the ideological implications of their practice.

Biological bias in psychiatry

Psychiatry is in danger of being defined by neurobiology. As an illustration, Nurnberger, in a review of *The Neurobiology of Mental Illness* by Charney, et al, (1999), noted that neurobiology was only represented in a few sections of the 1959 *American Handbook of Psychiatry*. As he remarks:

> *Few fields in medicine have experienced such a change in the underlying model of illness and treatment as did clinical psychiatry in the last part of the 20th century. Primary reasons for this development included the following: the overwhelming evidence of the efficacy of pharmacologic treatments, a growing appreciation of the heritability of psychiatric disorders, the standard use of objective, criterion-based diagnoses, and the ability to examine the structure and function of the brain directly.*

The primacy of these developments needs to be challenged. Nurnberger concludes by making grandiose claims for a textbook of neurobiology:

> *It should find a place on the shelf of practitioners in mental health-related fields, since it will help them interpret the reports they will see in the future on genetic and neuroanatomical abnormalities in their patients. It will be of interest to physicians in other specialties who may wonder about the rationale for the use of anticonvulsants in patients with mood disorders, the interpretation of abnormalities on magnetic resonance imaging scans in patients with schizophrenia, or the use of the sleep laboratory in differential diagnosis. Teachers of clinical and basic neuroscience will use this book to convey the relevance of laboratory-derived concepts and methods to public health and human behavior. The discipline described here is a far cry from the psychiatry we grew up with. Sigmund Freud, a neurobiologist by training, would have been proud.*

Sigmund Freud, of course, created psychoanalysis. Freud met Charcot, the leading neurologist of his time, in Paris after he received a scholarship for research abroad in 1885. The importance of Charcot for Freud is obvious in that a lithograph of the painting "Une lecon du Docteur Charcot a la Salpetriere" hung on the wall of his consulting room (Engelman, 1998). Moreover, Freud initially had a *Project for a Scientific Psychology* (Freud, 1895), and revealed in his letters to Fliess his motivation to conquer the mind (Masson, 1985). He had a notion of psychological determinism, but was confused (Rieff, 1966). But all this scarcely provides sufficient grounds for accreting his heritage to neurobiology.

There has always been a conflict between somatic and psychological approaches in psychiatry. The tenets of biological psychiatry are not new, even if they have been reinforced by the introduction of a plethora of psychotropic medications over recent years. For example, Haslam in 1817 concluded that insanity is "a corporeal disease". The professional implications for him were clear, because it then made mental illness "the peculiar and exclusive province of the *medical* practitioner" (his emphasis). He also naively admitted: "[F]rom the limited nature of my powers, I have never been able to conceive . . . *a disease of the mind*." (Haslam, 1798, again his emphasis)

Psychological approaches were also disparaged later in the nineteenth century (Clark, 1981). The insistence on somatic explanations of madness produced a resistance to overt psychologising. As anticipated by Henry Maudsley: "The explanation, when it comes, will not come from the mental, but from the physical side." (Maudsley, 1874)

At this basic level, modern mainstream psychiatry does not differ from Haslam and Maudsley in its views. Even though the essential conflict of the relation between mind and body remains as it was, the context is, of course, now different. Nurnberger describes four of the important recent developments in the first quote above and a critique of each of these conceptualisations will be provided in turn, viz. (1) The overwhelming evidence (or otherwise) of the efficacy of pharmacologic treatments (2) A growing appreciation (or otherwise) of the heritability of psychiatric disorders (3) The standard use (or otherwise) of objective, criterion-based diagnoses (4) The ability (or otherwise) to examine the structure and function of the brain directly.

(1) Bias in clinical trials

Randomised controlled trials have replaced uncontrolled studies and become accepted as the scientific "gold-standard". However, randomised controlled trials are too often assumed to produce unbiased evidence (Chalmers, 1998). The most methodically rigorous trials are associated with less treatment benefit than poor quality trials (Moher, et al, 1998). Even the best quality trials may still not completely eliminate bias because of difficulties is sustaining the two key elements of the method: that the trial is conducted "double-blind", which means that neither subjects nor experimenters are aware of their group allocation so that their expectancies do not bias outcome; and that any inherent differences between groups are equalised by randomisation.

Randomisation can be confounded because of drop-outs from the trial, leading to bias because of exclusions after randomisation.

One history of medicine is of doctors prescribing medication which is subsequently regarded as useless and often dangerous (Shapiro and Shapiro, 1997). The question is how much this situation continues into the present. Two examples will be considered (i) the efficacy of antidepressants, including the problem of antidepressant discontinuation and (ii) the efficacy of lithium

(i) *The efficacy of antidepressants*

Reviewing the research on antidepressant trials is a massive task. The first attempts to systematise the data such as Morris and Beck (1974) looked at those trials published between 1958 and 1972 and found that tricyclic antidepressants were significantly more effective than placebo in 61 out of 93 group comparisons. Rogers and Clay (1975) concluded that the evidence for imipramine, the original antidepressant, was so strong that further trials of this drug were not justified in endogenous depression in non-institutionalised patients. Since these two reviews, many new antidepressants have been introduced, particularly the class of SSRIs (serotonin specific reuptake inhibitors).

However, *New Scientist* could still report in 1998 that the benefits of antidepressant medication could be "mostly in the mind" (Day, 1998). At least one third of the published clinical trials of approved antidepressants are negative for efficacy (Thase, 1999). The US Agency for Health Care Policy and Research (1999) produced a conservative lower limit to treatment efficacy for newer antidepressants using an intention to treat analysis of response rates of 50% for antidepressants and 32% for placebo in major depression. Antidepressants are not always effective and there is a considerable placebo response.

The double-blind methodology is inadequate in many antidepressant trials (Even, et al, 2000). Patients and doctors may be cued in to whether patients are taking active or placebo medication by a variety of means. In fact if treatment is clearly superior to placebo, this should be obvious to raters in the trial making it not technically blind. Patients in clinical trials are naturally curious to ascertain whether they are in the active or placebo group, and may for example notice that placebo tablets they have been taking taste differently from medication to which they have previously become accustomed. Active medication may produce side effects which distinguishes it from inert medication. The difference between drug and placebo effect may be a true pharmacological effect, but the possibility that it is an enhanced placebo effect cannot be excluded (Kirsch and Sapirstein, 1998).

Using active drugs without apparent specific treatment effects as controls generally reduces the effect size of antidepressant treatment, maybe because bias is less likely to be introduced because of the detection of active effects in the control drug (Thomson, 1982). However, the adequacy of these active placebo trials can be questioned (Quitkin, et al, 2000).

The importance of the placebo response is relevant to problems in discontinuing medication. People may form attachments to their medications more because of what they mean to them than what they do. Psychiatric patients often stay on medications, maybe several at once, even though their actual benefit is questionable. Any change threatens an equilibrium related to a complex set of meanings that their medications have acquired. These issues of dependence should not be minimised, yet commonly treatment is reinforced by emphasising that antidepressants are not addictive. For example, the Defeat Depression Campaign of the Royal College of Psychiatrists criticised the general public for generally taking this view (Priest, et al, 1996). The general public might reasonably expect psychiatrists specialising in disorders of the mind to recognise psychological dependence, base their advice on clinical experience, and use their common sense (Double, 1997).

A sceptical view about the value of antidepressant medication is commonly rejected because it is regarded as undermining people's faith in their treatment. The issue is really about the scientific validity of claims for the efficacy of antidepressants. There is more uncertainty about this issue that many seem prepared to accept.

(ii) *The efficacy of lithium*

The reasoning behind the introduction of lithium into clinical practice was fallacious. Cade (1949) thought that mania may be caused by urea circulating in excess in the body. In an attempt to test this hypothesis he tested the effects of lithium urate, as this compound is the most soluble urate available. To determine whether lithium salts by themselves had any discernible effects, lithium carbonate was injected into guinea pigs and the animals became extremely lethargic and unresponsive to stimuli before once again becoming normally active after a few hours. This effect was almost certainly due to lithium toxicity. Even if what was being observed suggested that lithium may be of benefit in treating manic excitement, the association of ideas was illogical and serendipitous.

Unsurprisingly then, such an observation was not taken forward at the time, although understandably the toxic effects of lithium were of concern. Also, lithium is the exception to the pharmaceutical industry-led introduction of psychotropic medication (Tansey, 1998). However, as noted by Kline in 1968:

> *Lithium, the 20-year old Cinderella of psychopharmacology, is at last receiving her sovereign due. Just plain old lithium - widely discredited by early abuse in the treatment of gout, rheumatism, kidney stone, uraemia; briefly but dramatically misused as a salt substitute for precisely the type of patient in cardiac or renal failure for whom it should not ordinarily be used - the modest proclamation of its use for manic and other excitement states in a journal of limited circulation in a remote country was to pass almost unnoticed. Only 9 papers or letters were published in the first 5 years reporting new cases and very few more in each of the next two 5-year periods, in contrast to some 10,000 papers on chlorpromazine in its first 15 years and more than 2,000 on LSD. This year, however, will probably witness the publication of more papers on the subject than in all the previous 19 years.*

Schou, et al, (1954) tried lithium in several manic patients and were convinced of its value. Apparently, they took it more or less for granted that a drug that had a therapeutic effect during an ongoing manic episode must also prevent further episodes of mania (Schou, 1999). Cade (1949) had observed that lithium had no apparent beneficial effect on a small number of depressed patients. When it was reported that lithium ameliorated or prevented recurrences of depression the way was paved for the acceptance of lithium as a prophylactic agent for manic-depressive illness.

Moncrieff (1995, 1997) reviewed the controlled trial evidence of lithium and concluded that its efficacy has never been satisfactorily demonstrated. Jefferson (1998) has attempted to right a more conventional balance about the value of lithium. A meta-analysis of more recent placebo controlled lithium discontinuation studies did not find as great an effect of lithium as previous reviews (Baker, 1994). Non-specific factors do seem to be important as evidenced by the finding that rapid withdrawal is associated with higher relapse than gradual withdrawal. Furthermore, the largest recent trial to date has found no significant difference between lithium and placebo as measured by time to recurrence during maintenance treatment (Bowden, et al, 2000).

Lithium was introduced into practice on a wave of enthusiasm. Schou was involved in an influential early double-blind lithium discontinuation study, which had remarkably favourable results for lithium (21 placebo patients relapsed, none on lithium) (Baastrup, et al, 1970). Despite the lack of evidence of methodological weaknesses, it is difficult to believe that bias did not in some way affect the results as they are so strikingly favourable to lithium.

A sceptical re-evaluation is required to counter the overfavourable interpretation of the efficacy of lithium in the literature.

(2) Overstating the genetic case

A common mistake is to implicate genetic transmission merely because mental illness runs in families. Twin studies and adoption studies are regarded as "natural experiments" which enable an estimate to be made of the extent to which traits are familial because of shared genes, shared environment or a combination of both.

The work of the geneticists has been replete with uncritical dogmatic statements and lack of scientific rigour. For example, Kallmann (1938) presented a pair of twin girls born illegitimately to a domestic servant who died of tuberculosis soon after she left them to be brought up separately by two of her brothers who lived in different cities. Before they were ten they only saw each other a few times, but later met more frequently. One of the twins had a child aged 15 and was subsequently admitted to hospital in a "catatonic stupor". Twenty months later the other twin was admitted to the same hospital as she had become "increasingly more and more helpless and emotionally indifferent". Kallmann regarded this pair of twins as proof "that definite somatogenic factors must count as dispositional determinants in the manifestation of a hereditary predisposition to schizophrenia". As noted by Laing (1976):

> *When one sees . . . [Kallmann's] . . . completely uncritical and naive approach to his data, there is no indication that his assertions about "completely different environments" are any less naive; nor that he has more grasp of the pitfalls of retrospective data, or of reports from family members that may be equally biased due to fantasies held in common. . . . One can place no more confidence in Kallmann's assessments of "concordance" of "environment" than in those of life history and diagnosis.*

Moreover, although logically separate, Kallmann's "scientific" presentation was combined with a eugenic campaign to prevent the propagation of the apparent genetic predisposition to schizophrenia. He stopped short of recommending compulsory sterilisation, except for the "incorrigibles". Yet his proposals to prevent breeding of schizophrenics are suspect.

Over recent years, a bewildering and apparently contradictory array of studies have reported links with various genes in schizophrenia (Moldin, 1997). In contrast, the identification and cloning of genes and the elucidation of chromosomal abnormalities has led to major progress in the molecular biology of genetic neuropsychiatric disorders, such as Huntington's disease, in which the abnormality of triplet repeat on chromosome four has now been demonstrated.

Multigene models of inheritance are still commonly regarded as the best model of familial transmission of schizophrenia (Tsuang, et al, 2000). There is speculation that the mapping of the human genome will help to put better treatments for psychiatric illness on the market and make discrimination a thing of the past (McGuffin and Martin, 1999). Despite the hype, accurate prediction may never be possible because of the complexity of the genetics of common disorders (Holtzman and Marteau, 2000). Scepticism about the overenthusiasm for genetic explanations is required.

(3) The validity of psychiatric diagnosis

Operationalisation of diagnostic criteria was developed specifically to respond to criticisms of psychiatric classification (Blashfield, 1984). The primary motivation of these standardised, criterion-based definitions was to make diagnosis more consistent and reliable, initially for research (Feighner, et al, 1972). Clinically, diagnostic manuals have been developed through editions of DSM-III, DSM-IIIR and DSM-IV (American

Psychiatric Association, 1994) and ICD-10 (World Health Organisation, 1992). However, the most reliable of diagnostic criteria are not necessarily valid in the sense of measuring what they are supposed to measure. Diagnostic criteria do not solve the dilemma surrounding psychiatric classification.

The basic issue is about the meaning of psychiatric diagnosis. Single-word diagnoses do not necessarily help the understanding of a person's problems. The personal meaning of people's distress and the psychological and social origins of their difficulties are obscured by turning them into a simple diagnosis (Johnstone, 2000). In retrospect, the history of the development of diagnostic criteria over recent years has been little more than a professional discourse intent on justifying psychiatry. It has narrowed its scope and psychiatry needs to return to a biopsychosocial model, which was becoming more generally accepted by the 1950s before the widespread introduction of psychotropic medication (Wilson, 1993).

None of the above should be taken to mean that the reality of mental suffering is being denied. Thomas Szasz in a plethora of books since his original *The Myth of Mental Illness* (1972) has argued for the logical inconsistency of a diagnosis of mental illness, as for him the term illness necessarily implies physical pathology. Therefore psychopathology cannot amount to illness. Such a view does seem overly rigid. Similarly, suggestions that terms such as schizophrenia are without any meaning (eg. Boyle, 1990) can also seem to overstate the case. Diagnoses are no more than metaphorical use of language and there needs to be transparency about their use.

(4) Searching for the neuropathology of mental illness

Modern neuroimaging methods have enabled relatively non-invasive *in vivo* studies of brain structure and function. There is a multitude of studies which report abnormalities in shape, size and functions in multiple anatomical regions of the brain, particularly related to the so-called neuropathology of schizophrenia. The working hypothesis of most investigators of schizophrenia is that it is a disease of neural connectivity caused by multiple factors that affect brain development (Andreasen, 1999). However, structural and functional cerebral abnormalities in schizophrenia are at best subtle rather than gross. The most consistent finding of lateral ventricular enlargement is modest and there is a large overlap with the normal population (Chua and McKenna, 1995).

Theories of neurotransmitter dysfunction arose following the introduction of psychotropic drugs at a time when few neurotransmitters had been discovered. Despite the subsequent discovery of a vastly more complex neurotransmitter network, psychiatrists still use such simplistic notions in their everyday management of patients when they explain that mental illness is due to "chemical imbalance". For example, the dopamine hypothesis of schizophrenia (dopamine overactivity in schizophrenic brains) arose because neuroleptic drugs, such as chlorpromazine, appear to act via an inhibition of dopamine receptors. However, measurements of dopamine metabolites in vivo, or of the transmitter and its receptors in post-mortem brain tissue, do not provide unequivocal evidence of a hyperactivity of dopaminergic neurotransmission in the disease, if only because it is difficult to disentangle the iatrogenic effects of the drugs themselves (Reynolds, 1989). Moreover, many of the newer drugs do not appear to work by dopamine antagonism, raising the question of the nature of atypical antipsychotic medication.

Adolf Meyer (1951/2) was fond of seeing his philosophical approach to psychiatry, with its emphasis on the person, as an advance over the mechanistic philosophy of the 19th century. His work is currently largely neglected in the modern biological consensus in psychiatry. He warned against going beyond statements about the person to wishful "neurologising tautology" about the brain. Progress in understanding schizophrenia is more likely through rigorous and creative conceptualisation and evaluation of the evidence than uncritical application of neuroimaging methods. Reducing relations between people to objective connections seems to make them more manageable. Modern psychiatry has become so dominated by biologism that

acknowledgement that there is a philosophical issue about the relationship between mind and brain is rare.

The discovery that the adult brain is capable of extensive reorganisation necessitates radical revision of traditional notions of anatomical fixity (Eisenberg, 1995). Biochemically the brain modifies its own responsiveness to incoming stimuli through neuromodulators. The human brain seems to be socially constructed in the literal sense that brain cytoarchitecture itself is fashioned by input from the social environment. Although this claim is an extrapolation from the evidence, it holds out hope for an integration of theory, even providing evidence for a Meyerian psychobiology.

The training of psychiatrists

Each of the four recent developments which Nurnberger regards as crucial to progress has been examined and found wanting as represented by mainstream psychiatry. The case for an extensive, national programme of training and supervision in order to disseminate a critical perspective on psychiatry is overwhelming. This is consistent with the ethos of involving users and user-groups in decision making and accountability, as survivors of mental health services tend to be critical of their treatment.

The lack of a whole-person way of understanding mental health problems originates in medical training. In particular, reductive causal analyses do not help to make intelligible the reasons for human action. Medical students are indoctrinated into a natural scientific perspective of basic biological processes. The issue is never raised about whether an understanding of human behaviour should take a form similar to the laws of natural science. Despite the obvious failings of positivism, its attraction is that it seems to avoid the complexities and uncertainties of psychological and social perspectives. Taken to its extreme, such an approach becomes irrational.

Once imbued with the idea that experimental natural science is an adequate discourse for a description of personal behaviour, medical students may take a mechanistic view of their treatment of patients. They are encouraged to see psychiatry as little different from medicine in this respect, although they do sense the uncertainties of mental health work and regard it as less rigorous and developed as a discipline. Still, the cause of a person's insane behaviour or other mental health problems is their "chemical imbalance" or some other brain dysfunction, even if the mechanisms underlying this pathology seems less well understood than for other physical conditions.

The difficulty of communicating with disturbed and disturbing people can create defensive practice. Concentrating on the bodily complaints of people makes sense if they have a physical origin. Emotional complaints do not have to be addressed if they are reduced to their bodily substrate. Questions are avoided about why psychiatry interfaces with the law and human rights. An ethical approach to psychiatric practice can be difficult to sustain. The aim of social control may produce a lack of compassion and feeling in the care of people with mental disorder, and the history of psychiatry confirms the common degradation of patients. The health professional's own need for self importance can override the patient's problems.

These problems are not totally unique to psychiatric training. Much of the current criticism of medical professionalism is of an arrogance created because of the difficulties in engaging with people's suffering through illness. Claims are made for medical techniques which go beyond the evidence. Patients may have needless investigations and be overtreated. In an attempt to bolster professional interests, self-questioning is discouraged in the interests of certainty.

The modern use of the term "anti-psychiatry" was introduced by David Cooper (1967). In general anti-psychiatry has been regarded as simply a passing phase in the history of psychiatry (Tantum, 1991). A more constructive approach is to place anti-psychiatry in its broader cultural context and see it in terms of its

continuities (Gijswijt-Hofstra and Porter, 1998). The most consistent use of the term "anti-psychiatry" derives from the recognition that psychiatry is not always the solution to mental illness; it may be the problem itself. The contemporary critique of psychiatry cannot dissociate itself from its history in anti-psychiatry. Its legacy may have foundered because some of its representatives were ultimately more interested in personal authenticity than carrying through their ideas in practice. Moreover, its message became confused because the movement included a wide range of different people, some of whom are essentially irreconcilable in their views. In particular, Thomas Szasz (1976) has been scathing in his censure of RD Laing. Laing never denied the reality of mental turmoil; merely contending that so-called mental illness was more understandable than generally assumed. It is the apparent denial of the reality of mental illness that allows the critique of psychiatric practice to be marginalised. Contemporary critical psychiatry needs to be clear that the concept of mental illness can be applied to psychological dysfunction (Farrell 1979). The biological viewpoint is unjustifiable, as it assumes that mental illness is real in the physical sense. Anti-psychiatry is against psychiatry's excesses not anti- "mental illness".

Psychiatrists in training need to be exposed to the ideological implications of their practice. Such a self-reflexive approach does not deny the need for the use of the Mental Health Act or the pragmatic use of medication. However, it may lead to the realisation of the importance of patient's rights as well as the assessment of risk, and the recognition of overreliance on medication.

In summary, the biomedical dominance of psychiatry needs to be challenged (Critical Psychiatry Network website). Psychiatric training needs to incorporate a critical perspective.

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Duncan Double is Consultant Psychiatrist, Norfolk Mental Health Care NHS Trust and Honorary Senior Lecturer, School of Health Policy and Practice, University of East Anglia. He is website editor of the Critical Psychiatry Network (http://www.criticalpsychiatry.co.uk/)