Correspondence

EDITED BY LOUISE HOWARD

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Authorship of clinical trial reports
I was most interested to read the report by Baldwin et al (1999), particularly as I am listed as a co-author, although I had never seen the text before and did not know it was being submitted for publication! How did such a situation arise and how can its repetition be avoided in future?

In many countries, particularly the USA, it is editorial policy to obtain consent to submission that includes all of those who participated directly in the work described; often to the extent of obtaining individual signatures. This is to be applauded. Working at the Feighner Research Institute in London, I personally treated 27 patients who were included in the above trial and informed the company concerned, on more than one occasion, that I wished to see the text of any manuscript submitted for publication prior to the event. But I received no reply.

Individual investigators should be named with their centres, rather than lumped together in an unspecified ‘study group’. It would also be helpful, in the context of any statistical conclusions reached, if the number of cases contributed from each individual centre were to be recorded in published reports.

Paroxetine in social phobia/social anxiety disorder: Randomised, double-blind, placebo-controlled study.
British Journal of Psychiatry. 175, 120–126.

D. Wheatley Feighner Research Institute, Charter Chelsea Clinic, 7 Radnor Walk, London SW1 4PB

Authors’ reply: We are grateful to Dr Wheatley for his comments on the publication of the treatment study with paroxetine, and would like to acknowledge publicly the contribution made by Dr Wheatley and his colleagues within the Feighner Research Institute. We thank all those principal investigators, their research teams and the patients who kindly took part in this investigation.

It is difficult to acknowledge all the clinicians who participate in large multi-centre treatment studies, and naturally some investigators will be disappointed when their contribution is not credited as much as that of their colleagues. Ideally, publication plans should be discussed at meetings with potential investigators prior to the start of a study, and then reviewed during the course of the investigation. This is now the policy at SmithKline Beecham, but this study with paroxetine was started before the policy was in place. Due to a change in personnel within SmithKline Beecham, the communication between the sponsoring company and all the participating investigators has been less than optimal, for which we apologise.

In the future, SmithKline Beecham will try to ensure that all the participating investigators are happy with the publication plan before the start of any treatment study. However, the question of contribution to full authorship will remain problematical, for collaborative study groups and journals alike.

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Editor’s response: The Journal does enforce a policy of obtaining the signatures of all authors before accepting a manuscript for publication. In this instance, confusion has arisen because of uncertainty over the authorship status of members of a study group. This issue has now been clarified in our ‘Instructions to authors’ as follows:

The Journal does not consider to be authors people thanked in the Acknowledgements or listed as members of a study group on whose behalf a paper is submitted, but whose names do not appear as authors on the title page of the manuscript, or whose signed agreement to the manuscript submission has not been obtained. It is the responsibility of the corresponding author to ensure that authorship is agreed among the study’s workers, contributors of additional data and other interested parties, before submission of the manuscript.

Full text of these Instructions is located on the Royal College of Psychiatrists’ web-site (http://www.rcpsych.ac.uk/pub/bip_ita.htm) and is next scheduled for paper publication in the July issue of the Journal.

Psychological model of post-stroke major depression
Gainotti et al (1999) concluded that post-stroke major depression may fit with a psychological model rather than with a neurological model based on their findings that post-stroke patients with major depression displayed more reactive symptoms (emotionalism, catastrophic reaction, anxiety) and fewer endogenous symptoms than patients with endogenous depression.

There are some concerns regarding the conception and the methodology of the study. The major drawback in the methodology is the bias in selecting the control group. It is not surprising that patients with endogenous depression will have more endogenous symptoms than patients with post-stroke major depression, as major depression can be diagnosed using DSM-III-R (American Psychiatric Association, 1987) operational criteria in the absence of endogenous or melancholic symptoms. Further, it is also expected that affective symptoms related to brain damage, such as emotionalism and catastrophic reaction, will be more prevalent in post-stroke major depression than in patients with endogenous depression.

Gainotti et al seek to create an impression that there is no association between endogenous depression and psychological stressors, and that post-stroke major depression with symptoms such as anxiety and hyperemotionalism are the representation of a psychological reaction to stressful situations. The available literature fails to support the validity of dichotomous endogenous/reactive and endogenous/non-endogenous classifications (Farmer & McGuinn, 1989). Further, emotionalism observed in post-stroke patients is often
precipitated by non-specific stimuli, and crying or tearfulness observed in emotionalism may not be associated with alterations in mood (Poeck, 1969). No attempt was made to examine whether the symptoms assumed to be stress-related, in stroke patients with major depression, were correlated with the severity of functional impairment or with subjective perception of stressful situations.

It would be of interest to investigate the differences in prevalence of endogenous or melancholic symptoms between post-stroke major depression and major depressive disorder. Moreover, using the same classification procedure in subtyping post-stroke depression and depressive disorders may cause limitations and constraints when phenomenological comparisons are made between these two disorders. To overcome this problem, classification of post-stroke depressive disorders could be approached from the bottom up, with the identification of depressive symptoms in stroke patients. By applying multivariate analysis, these symptoms can be grouped into clusters or syndromes, which can then be validated.


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Author’s reply: Dr Ramasubbu argues against our suggestion that psychological rather than neurological factors mostly account for post-stroke depression (PSD). His/her main argument is that our results could be due to a bias in selecting a control group of patients with endogenous depression. He/she remarks that it is not surprising that endogenous symptoms were more frequent in this control group, whereas affective symptoms related to brain damage prevailed in patients with major PSD. This argument could certainly be appropriate if the aim of our study consisted in matching the main symptoms of patients with major PSD and with major endogenous depression, but this was clearly not the scope of our paper. Patients with major endogenous depression were, in fact, the group against which we matched various subgroups of patients with major PSD observed at various time intervals from stroke. The scope of these comparisons consisted in assessing whether patients with endogenous depression are more similar to patients with major PSD observed immediately after the stroke than to those observed in more chronic post-stroke periods. A similar strategy had been used in previous studies of this field conducted by our research group, since the distinction between various forms of PSD plays a crucial role in the model of PSD proposed by Robinson et al (see Starkstein & Robinson, 1989, for review). This model is, in fact, substantially based on the distinction between two forms of PSD: (a) the major form, due to a lesion encroaching upon the left frontal lobes and indistinguishable from the clinical and pathophysiological points of view from the major forms of endogenous depression; and (b) the minor form, considered as a form of reactive (dysthymic) depression and having no specific anatomical substrate. In a previous paper (Gainotti et al, 1997) aiming to test this original version of the Robinson et al model, the symptomatological profiles of patients affected by endogenous depression were matched with those in three groups of stroke patients, on the basis of DSM–III–R criteria, as having no depression, minor PSD, or major PSD. The following predictions were made: (a) if major PSD is indistinguishable from endogenous depression, whereas minor PSD is a reactive form of depression, then the symptomatological profile of patients with a major form of PSD should be more similar to that of patients with endogenous depression than to that of patients with a form of minor PSD; (b) if, on the contrary, no qualitative difference exists between major and minor forms of PSD, then patients with major PSD should be more similar to those with a minor form of PSD than to those with a form of endogenous depression. Our results clearly supported the second prediction showing that a continuum exists between major and minor forms of PSD.

To account for these and other data also inconsistent with the Robinson et al model, Herrmann & Wallesch (1993) proposed a restricted version of the model which assumes that only the forms of major PSD observed in the acute post-stroke periods are very similar to endogenous depression, whereas those observed in more chronic stages must be considered as reactive forms, mainly due to psychosocial factors.

To test this new version of the Robinson et al model with a strategy similar to that used in our previous paper, we matched the symptomatological profiles of patients with major PSD observed at various time intervals from stroke with those of patients with endogenous depression. Since the profiles of patients with major PSD observed at various time intervals from stroke were very similar, and were very different from those in patients with endogenous depression, we concluded that even the restricted version of the Robinson et al model is inconsistent with our data.

We therefore think that no methodological defect exists in this or in our previous study and that our data allow us to conclude that no qualitative difference exists either between minor and major forms of PSD or between forms of major PSD that arise at various time intervals from stroke.

Let us pass now to our suggestion that psychological factors, rather than neurological factors, mostly account for PSD. This suggestion was mostly due to the distinction that we have more analytically described in our previous study (Gainotti et al, 1997) between motivated and unmotivated affective patterns. The term ‘motivated reactions’ refers not only to the reactive symptoms of anxiety, emotionalism and catastrophic reactions, whose prevalence in patients with PSD could be due (as Dr Ramasubbu suggests) to a bias in the selection of the control group, but also to a qualitative analysis of the responses given by patients in sections of the Post-Stroke Depression Rating Scale (PDSRS) devised to evaluate ‘depressed mood’, ‘guilt feelings’ and ‘thoughts of death and/or suicide’. In these sections, patients were requested to qualify their response by saying whether their bad mood, guilt feelings and thoughts of death were related to their actual condition or were independent from it. Patients with major or minor forms of PSD usually attributed depression to the consequences of stroke, felt guilty because they considered their previous lifestyle as partly responsible for their actual disease, and had stroke-related death worries, whereas patients with endogenous depression attributed guilt feelings to their moral
worthlessness and had active suicidal tendencies, rather than death worries. Furthermore, in both of our studies, diurnal mood variations were motivated (i.e. linked to situations stressing handicaps and disabilities) in patients with major PSD, but unmotivated (with a prevalence of depression in the early morning) in patients with endogenous depression.

In conclusion, even if we share with Dr Ramasubbu some doubts about the validity of dichotomous endogenous/reactive classifications, we would stress two points: (a) our criticism was addressed to an influential model based on such dichotomy: and (b) our tentative hypothesis that psychological factors play an important role in PSD seems, at least in part, justified.


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Psychiatry and civil unrest in Northern Ireland

In his editorial, Daly (1999) is critical of the research that has been done on the psychological impact of the civil unrest in Northern Ireland, both in terms of its quality and quantity. He states that opportunities for valuable research have probably been missed, and those studies that have been carried out, he weighs in the balance and finds wanting. I consider his article a potentially misleading reflection on psychiatry in Northern Ireland over the past 30 years.

Much of the research he reviews emerges as speculative and inconclusive. He is critical of Lyons’ (1971) concept of “normal anxiety”. The work of Cairns & Wilson (1984) is, Daly believes, of limited usefulness as the populations studied were rural, whereas violence is largely an urban phenomenon. Curran (1988) is exposed as mistaken in his view that individuals habituate to trauma. These authors published their findings 29, 16 and 12 years ago, respectively. It is all too easy to find fault today.

Daly concludes that lack of trust in the authorities and a fear of breaches of confidentiality have resulted in treatment avoidance and exacerbation of symptoms. He gives no evidence for these conclusions. Psychiatrists in Northern Ireland have struggled to avoid opportunism and prejudice, and to maintain impartiality. It would be a matter of concern if this was not the public perception.

Daly widens the concept of victim to include “terrorists incarcerated for paramilitary crimes”. If offenders are to be viewed thus, there is a risk of widening the concept of victim to the point where it becomes meaningless. The research to which he refers in his next sentence (Lyons & Harbison, 1986) related to one crime only, that of murder. Political murderers were found to be a more stable group than non-political murderers. That paper had no comment to make on the victim status of prisoners or on political crimes in general, contrary to the impression conveyed by Daly. His subsequent reference to a report in a local newspaper (Belfast Telegraph, 26 September 1998), in the context of psychological problems consequent on imprisonment, is speculative.

Finally, Daly has overlooked a crucial consideration in his editorial. It is no exaggeration to say that the political situation in Northern Ireland has made it difficult, if not at times hazardous, to carry out research on offenders and victims. On occasions where research has been done, it has not been feasible to publish it. Psychiatrists practising in Northern Ireland over the past 30 years have laboured under difficulties not experienced by colleagues elsewhere in the UK. Daly should not victimise them.


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Author’s reply: Dr Harbison criticises my recent editorial in a rather defensive manner but does not provide any evidence to refute my opinion that opportunities for valuable research have been missed over the past 30 years or so.

He/she seems unhappy with my review of some of the research carried out, commenting that “it is all too easy to find fault today”. In my editorial I commented that “at the time Lyons (1971) was carrying out his research the field of traumatology was in its infancy” and, in relation to Curran (1988), that “more recent research in the field suggests” a different view to that of Curran regarding habituation to trauma. The whole purpose of a literature review is to examine previous research critically in the light of further developments.

In the Social Services Inspectorate document referred to in my paper (Department of Health and Social Services, 1998), the issue of confidentiality was addressed; for example, “Another G.P. noted that ‘the individuals that are most affected in our area are of a predominantly nationalist viewpoint. There is a fundamental distrust of Government agencies [and] distrust and fear of leakage of sensitive information’”. Information received from the project leader in the Social Services Inspectorate has confirmed a minority, but consistent, viewpoint, mainly from those of a nationalist background, that the authorities, including those working in health and social services, are not to be trusted (J. Park, personal communication, 1999). As Dr Harbison writes, this indeed should be a matter of concern.

Dr Harbison is critical of me for commenting that “some people would consider terrorists incarcerated for paramilitary crimes to be victims”. It has been reported that a number of people who subsequently become involved in terrorist crime have themselves previously been victimised (Smyth, 1998). A study looking at 80 perpetrators of homicide found that 52% met criteria for current post-traumatic stress disorder (Pollock, 1999). It would seem unethical to exclude anyone from being considered a victim, and therefore a potential candidate for treatment, on the basis of having been involved in criminal activity. Dr Harbison has commented that on occasions it has not been feasible to publish research carried out. I find it difficult to understand why properly structured and anonymised research could not have been published. In order to ensure that psychiatrists maintain impartiality, it is important that such research should be published whatever the results, provided the findings are clinically relevant.
Dr Harbinson’s final comment about my victimising other psychiatrists is rather emotive. He/she is correct regarding the speculative nature of some aspects of my paper. This has been necessary because of the relative lack of research. However, I am certainly not intending that criticism be directed at colleagues, a number of whom have carried out valuable research while also carrying heavy clinical workloads. It remains my view that there is a need for a detailed epidemiological community study which specifically addresses the issue of trauma.


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Studying grief in adults with learning disabilities

I read with interest the article by Bonell-Pascal et al (1999) and would agree that recognition of, and service provision for, the bereaved adult learning disability population is markedly deficient. However, their conclusion that learning disability is a significant predictor of future mental health problems following bereavement is not substantiated and should be interpreted with caution in view of the flawed research methodology.

The original cohort reported short-term psychopathological and behavioural outcomes following bereavement (Hollins & Esterhuyzen, 1997). This case-controlled study found an increase in aberrant behaviours and psychopathology in those bereaved compared with controls. Bonell-Pascal et al aimed to investigate whether the same outcomes had changed over a longer term (six to eight years). Unfortunately, the control cohort was not followed up, thereby making meaningful inferences impossible, as confounding life events are not controlled for. Also, more than half the study population had additional medical disorders of likely prognostic significance. The authors recognise the limitations of their small sample size, but this is further compounded by incomplete follow-up of the original cohort, with greater than 15% of the original cohort excluded. Furthermore, follow-up data are missing from the analysis of psychopathology and aberrant behaviour.

In the classification of psychopathology, each sub-scale shows varied changes with no interpretable trends over time. Psychopathology identified in the original 1997 study had resolved in over 70%, although a few new cases were identified, especially in the adjustment and anxiety disorder sub-categories. However, we cannot attribute these new cases to underlying learning disability as confounding medical and life events may have played a significant role. Aberrant behaviour patterns were reported as showing a mean increase in each sub-scale over time. The individual change in aberrant behaviour patterns was, however, in both directions in all sub-scales (i.e. an improvement and deterioration in behaviour). The mean change quoted by the authors in such a small study sample with wide ranges of behaviour is, therefore, unreliable. Similarly, the one statistically ‘significant’ result, showing in-appropriate speech to be more common, should also be treated with caution and taken in isolation is unlikely to be of clinical significance. Furthermore, the authors allude to problems with validation of the behavioural and psychopathological measures used in the significant proportion of the study population who were taking antidepressive medication.

It is now recognised that people with learning disability do understand the permanence of death and consequently grieve (Harper & Wadsworth, 1993; Read, 1996). Bonell-Pascal et al highlight the need to better understand the general and specific needs of this vulnerable group of people and provide some useful insight into effective interventions. Future studies should be aimed at addressing these issues, be carried out prospectively and have adequate control groups to allow safe and meaningful conclusions to be drawn.


C. A. Lyons Department of Psychiatry of Learning Disability, Black Country Mental Health Trust, Heath Lane Hospital, West Bromwich

Author’s reply: We have read Dr Lyons’ letter with interest, and considered his/her comments with great care. It is encouraging that the letter also highlights the need for further research in this area, and we are happy to say that we have new research in progress.

We acknowledge that the methodology of the published report has limitations, but that does not mean that it is flawed. Dr Lyons does little more than amplify the limitations spelled out in what was, after all, only a short research report. It is perfectly acceptable to use data from the first study as longitudinal data in the follow-up study. Furthermore, although follow-up of the original control group might have yielded some useful material, it is more probable that it would have been unreliable, because of the likelihood that these individuals would have experienced bereavements themselves in the intervening time between the first and second study.

Dr Lyons suggests that new ‘cases’ at follow up cannot be attributed to the underlying learning disability as confounding medical and life events may have played a part. One of the key points made in both 1997 and 1999 papers is that the effects of bereavement are compounded by the increase in life events experienced by the client group at such a time.

With regard to the size of the second sample, the shortfall is not excessive: as stated in the paper, three of the missing individuals were dead, and three untraceable, possibly also dead. The true follow-up rate could thus be more accurately described as 41 out of 44, or 93%. Furthermore, two of the remaining three carers refused to help with the follow-up interviews because bereavement was too sensitive an issue for either the relative or the person with learning disability.

Dr Lyons suggests that the results of the study are not ‘meaningful’. The original manuscript, which was cut in length at the request of the Editor, included qualitative material collected from carers at the same time as the quantitative data. This material supports the results of the quantitative data,
and includes a wide range of phenomena following bereavement, including withdrawal, tearfulness, weight loss, obsession with death, health problems, increase in fits, facal incontinence and regressive behaviour. Carers reported the continuing effects of bereavement. For example, one man, who had had a close but difficult relationship with his father, was still working through the bereavement with a psychologist, and taking psychoactive medication, some five years after his father's death. Another man, after a similar period of time, still cried out for his mother when something went wrong; Miss F is said still to break down easily, crying "My mother's dead!", illustrating the immediacy that a bereavement can have even after a number of years.

The work we reported adds to the growing body of evidence that bereavement can cause psychological distress and behavioural symptoms, which may well be overlooked or misinterpreted.

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Maternal eating disorder and mother–child conflict

Stein et al’s study (1999) provides essential reading for clinicians involved in treating anorexia nervosa or bulimia. However, in light of recent research concerning pregnancy and eating disorders (Morgan et al., 1999), three further areas of investigation seem to have been unexplored.

First, we have demonstrated that a third of women with bulimia developed postnatal depression, rising to two-thirds in women with previous anorexia nervosa. Second, Stein et al treat ‘eating disorders’ with unwarranted homogeneity; we found profound differences between pregnant women with bulimia and those with previous anorexia nervosa. The latter predicted postnatal depression, absence of breast-feeding and postpartum relapse into eating disorder. The most striking differences were apparent at a descriptive level, where women with previous anorexia nervosa appeared highly alienated from their infants. Third, the majority of pregnancies described in our study (Morgan et al., 1999) were unplanned due to mistaken beliefs regarding fertility.

It would be interesting if Stein et al were able to re-examine their data with reference to specific eating disorder diagnoses, presence of affective disorder and planning of pregnancy, all of which might generate maladaptive responses to the antecedents of conflict.


J. F. Morgan Department of General Psychiatry, St George’s Hospital Medical School, London SW17 0RE

Authors’ reply: Dr Morgan makes some interesting points in the light of his own study concerning factors which might influence the nature of the relationship between mothers with eating disorders and their infants. However, the purpose of our paper was to examine the evolution of conflict between mothers with eating disorders and their infants using detailed observations of sequences of interactions. In particular, we wanted to establish whether the way in which mothers and infants responded to different situations during mealtimes influenced development of conflict. We found that whether or not the mothers responded to the infants’ cues determined whether or not conflict arose. In addition, the infants’ behaviour also contributed to the evolution of conflict in some circumstances. The elucidation of these features of interaction, which was only possible through sequential observations and analyses, offers the potential for intervention irrespective of which background factors influence interaction. In particular, it is critical to help mothers to recognise the positive aspects of their infants’ communications and cues during mealtimes in order to facilitate this interaction.

Obviously, we are very interested in the other factors in the mother’s history or mental state which might influence the course of these interactions, such as those detailed in the Morgan et al paper cited above. However, in order to carry out the analysis of sequential interactions as we have done, and to determine the relative influence of a variety of other factors on the course of these interactions, a much larger study would be required. This would be valuable but well beyond the scope of our paper.

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Compulsory treatment in anorexia nervosa

I read with interest the paper by Ramsay et al (1999).

The matter of compulsory treatment in anorexia nervosa is clinically important. The lifetime risk of women developing this disease is 0.5%, that is half the lifetime risk of schizophrenia. The mortality rate is unacceptably high, reaching nearly 20% at 20-year follow-up. This would suggest the need for compulsory treatment in certain circumstances. However, there is disagreement between authorities about the issue, and in relation to the right of anorexic patients to receive life-saving treatment if they are unable to consent to it by reason of their mental disturbance. Various viewpoints have been presented in a recent multi-authored book (Vandereycken & Beaumont, 1999). As Ramsay et al point out, the only other empirical study attempted in this area was by Griffiths et al. (1997) in New South Wales. The situation in New South Wales at the time of the latter publication was rather different from that in the UK inasmuch as anorexia nervosa is not considered a mental illness as defined in the Mental Health Act of this State. On that ground, Mr Justice Powell of the Supreme Court of New South Wales ordered the discharge of a severely ill patient with anorexia nervosa in 1986, setting a precedent that persisted until 1999. Incidentally, the patient in question died some time after her discharge.

In the absence of suitable mental health legislation in this area, the management of severely ill patients with anorexia nervosa who refused treatment became an issue for the Guardianship Board. Unfortunately, no new provisions were inserted into the Guardianship Act to deal precisely with this responsibility. Consequently, the treatment of patients has often been severely impeded, the public guardian demanding formal requests at each stage of treatment, and hence causing a considerable delay, and sometimes refusing treatment on grounds which appear ridiculous, for example refusing a cognitive–behavioural programme because it was not ‘medical’ treatment.
As recently as 1999, the situation in New South Wales has again changed. At a hearing concerning a 19-year-old severely ill patient with anorexia nervosa, Mrs Brennan, senior member of the Mental Health Tribunal, decided that she was a mentally ill person, and hence did fall under the Act even though anorexia nervosa as such was not considered a mental illness (note not to comment on the niceties of the use of words in law, merely to report). This brings our State in line with legislation in the UK, and other states of Australia.


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Resource allocation for mental health care

We read with interest the editorial by Gyles Glover on allocations to health authorities for mental health care (Glover, 1999). It is important that the sums of money allocated to health authorities for mental health care be widely known. This will allow all interested parties to have ready access to information upon which to base any claims about the adequacy or otherwise of funds made available to health care providers.

We are aware there is wide variation in the amounts actually spent by health authorities, as opposed to the amounts allocated. For example, in the year 1997/98 Worcester Health Authority spent £24 per head of population on mental illness (excluding mental handicap), Solihull Health Authority spent £32 per head of population, and Lambeth and Southwark spent £138 (Chartered Institute of Public Finance and Accountancy, 1998). These amounts represent 4, 5.5 and 17.4 per cent, respectively, of the overall expenditure on health care per head of population. We do not have any information on the actual amounts allocated to health authorities in this period, but are concerned at the observed variation.

We do not believe that this variation can be easily explained.

There is cause for further concern. The York formula, which is used in determining the level of resource allocation, for example, ranks Birmingham 8th in order of need but 20th in order of spend per head of population. But more worryingly, Birmingham is ranked 38th in order of spend: needs index ratio (further details available from Dr Oyebode upon request). This suggests that whatever the actual amount of money allocated, using a formula devised to take account of factors predictive of high psychiatric morbidity, health authorities may not be spending the indicative amounts allocated to them.

We believe that it is important that mental health services be adequately funded. The transparency of the arrangements for funding will become even more important as we move into an environment controlled by primary care groups or trusts. The risk is that substantial sums will be allocated but not spent on mental health services. It is clear that this is already the case but the situation could very well worsen if there is no control in the system.


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Fluoxetine-induced anaesthesia of vagina and nipples

Antidepressants drugs cause a variety of sexual side-effects. However, antidepressant-induced changes in sexual sensations are rare. We report a case of fluoxetine-induced loss of sensation of vagina and nipples.

A 48-year-old married woman with recurrent depression had good antidepressant response to fluoxetine 20 mg. However, her compliance with the medication was poor resulting in recurrences. While euthymic and on no antidepressants, her sexual function was normal. When depressed she has moderate decrease in libido. With fluoxetine 20 mg her depression remitted and her libido returned to normal. However, she developed a complete loss of sexual sensation of her nipples and vagina. Touch and pain sensations were also impaired, but only to a lesser extent. This lead to decreased satisfaction with sexual life and consequently poor compliance with the medication. Even when she became briefly hypomanic on fluoxetine, the lack of sensation persisted. We substituted her fluoxetine with trazodone 400 mg. She remained euthymic. By the fifth week her vaginal and nipple sensations returned to normal. The frequency of sexual intercourse and satisfaction improved to pre-morbid levels.

This is the first report of fluoxetine-induced loss of sensation of vagina and nipples. Fluoxetine-induced anaesthesia of penis (Neill, 1991; Measom, 1992) and vagina (King & Horowitz, 1993), which did not improve with dosage reduction or addition of cyproheptadine, but did with discontinuation of fluoxetine, have been reported. Ellison & DeLuca (1998) reported a case of genital anaesthesia caused by fluoxetine that did not improve with addition of cyproheptadine or yohimbine but responded to Ginkgo biloba. Ginkgo biloba is a Chinese herbal remedy for a variety of disorders and has diverse neurochemical effects. The mechanism of antidepressant-induced sexual anaesthesia remains elusive. The fact that the anaesthesia persisted even during the fluoxetine-induced hypomanic state confirms that this was not part of the depressive syndrome.

Sexual side-effects of antidepressant drugs cause distress, strain relationships, impair quality of life and reduce compliance with treatment. Enquiring routinely about side-effects, especially sexual side-effects of antidepressants, would help to improve compliance with treatment.


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Spontaneous orgasms – an epileptic case without structural correlate

Over 10 years ago, Vergheese (1989) reported on spontaneous orgasms, stating that in the absence of gynaecological, hormonal, or overt psychological disturbances such symptoms might be caused by epileptic seizures as part of an organic brain disease (Ruff, 1980, Remillard et al, 1983, Reading & Will, 1997). Only a few individual reports have been published on this condition, giving accounts of structural lesions in the temporal or parietal lobe. Erotic manifestations in these papers so far were described as unpleasant sensations.

We describe the case of a 37-year-old female patient who experienced her first generalised tonic-clonic seizure at the age of 28. Not until five years later, when she had her second generalised seizure, did other ‘funny’ symptoms appear, which she did not recognise as epileptic manifestations. She described epigastric feelings, sensations of déjà vu, déjà vécu and depersonalisation as well as spontaneous orgasms, the latter of which was experienced as a pleasant, although embarrassing, sensation. These feelings could occur anywhere, at home, at work or on entering a bus. During those events she often was able to continue in her work or in her conversations, paleness being the only noticeable symptom. The frequency of such episodes ranged between one to six per month. Full diagnostic work-up was only performed three years later when she had had her third generalised seizure. As on the previous occasions her general, neurological and psychiatric examinations were normal. Computed tomography scan, magnetic resonance and electroencephalogram (EEG) were found free of structural lesions and focal signs, but a long-term EEG revealed a right temporal sharp-wave focus lasting five seconds, indicating that the symptoms of our patient could be identified as part of an epileptic aura pointing towards partial seizures with an origin in the right temporal lobe. This diagnosis was confirmed by the fact that all symptoms ceased after initiating antiepileptic treatment with lamotrigine.

This case suggests that neuroimaging might not always be sensitive enough to reveal structural lesions, and that long-term EEG recordings should be included in the diagnostic work-up. In doubtful cases a trial with antiepileptics can prove helpful. A thorough history-taking should always include questions about other concomitant epileptic symptoms. Whether erotic manifestations during partial epileptic seizures are experienced as being pleasant or unpleasant, they still are embarrassing and a burden for the patient and therefore warrant pharmacological treatment.


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One hundred years ago

The Metropolitan Asylums Board and its Assistant Medical Officers.

To the Editors of The Lancet

Sirs, In The Lancet of Jan. 13th there appeared an advertisement for an assistant medical officer at the Caterham Asylum of the Metropolitan Asylums Board. Having had experience of that Board I should like to point out for the benefit of intending applicants a few of the conditions under which the assistant medical officers at their asylums work. On entering upon my duties I was taken to my quarters by the steward, who apologised for the lack of accommodation offered me, explaining at the same time that such conditions were only temporary. My “quarters” consisted of one room in which I slept, the same room had also to serve me as an office, dining-room, as well as for the reception of personal visitors, and indeed for every use that it is possible to put a room to. All that was temporary as far as I was concerned consisted in the facts that on committee days a screen was taken from the room, so that visitors could see the bed and the fact that I had a knob put on the door after I had occupied my quarters for three weeks. Since I left there have been improvements at that particular asylum. The junior assistant medical officer now shares in a common dining-room and has consequently only to use his bedroom for an office and living-room generally. The medical superintendents at their asylums are not allowed to write testimonials. The Metropolitan Asylums Board, however, accept testimonials from other asylums. Such conditions prevailing one is not surprised to find that after years of service under the Board promotion is impossible elsewhere, and as the Board is not enamoured with the idea of promoting its own men the assistant medical officers find themselves in an awkward position. At other asylums it is the practice to allow the assistant medical officer the use of spare bedrooms in which to lodge a visitor, it being recognised that life in asylums is somewhat monotonous.

In my time when a visitor arrived his host laid himself down on his couch (if he had one, I had not) and with his clothes on was happy that his brother from Scotland or a medical friend from America had come to see him. They were the only visitors whilst I served the Metropolitan Asylums Board. Now, the Metropolitan Asylums Board will no longer allow any visitor to be the guest of their assistant medical officer overnight.

Would intending applicants for the post advertised kindly apply to any of the Metropolitan Asylums Board’s medical officers for information?

I am, Sirs, yours faithfully,
F.J.S.

REFERENCE

Lancet, 20 January 1900, 211.

Researchers by Henry Rallin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey
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Access the most recent version at DOI: 10.1192/bjp.176.3.299-a

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