
# Forget community care - reinstitutionalisation is here

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# Correspondence

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## Forget community care – reinstitutionalisation is here

Although we agree that care in the community is perceived as a failure within the public domain and definitively as portrayed by the media, there are a number of issues around deinstitutionalisation that have not been addressed by Professor Leff (2001). Certainly, the apparent invisibility of community teams, the muddling of schizophrenia with personality disorder, 'split mind' and homicides, and the modern prevalence of homelessness are all factors. More important, perhaps, is that we do not really know why community care developed during the second half of the 20th century, and why it is now returning to what a 19th-century editor called 'bricks and mortar humanity' (Wynter, 1859).

It may be that the studies following the planned resettlement of asylum populations quoted by Leff show no subsequent homelessness in the discharge populations, but this ignores the new long-stay problem. That is to say younger patients, who have never been through the asylum system, and who go in and out of in-patient units on the revolving-door circuit. Leff's experience of 20% of patients being homeless is out of date, current levels being nearer 40% or even 50% in our east London wards, for example. Another third are people readmitted from hostels, and now no longer accepted by these because of 'risk management', drug use or other 'difficult' behaviours.

This leads on to concerns about violent crime, in that there has been a decrease in the proportion of violent crimes committed by people with mental illness, but not a decrease in the numbers. This may reflect generally rising crime rates, but we remain ambivalent, in psychiatric circles, about the relationship between schizophrenia and criminality, and there seems to be a tendency to try to gloss over it. This also has an impact on what Leff calls the 'mixed economy of care', and a 'complex network of interlinked facilities and professionals'. In fact this is a Gormenghast-like labyrinth, with voluntary agencies, privately run hostels, and forensic units carefully trying to ward off all difficult comers and, in the case of the latter, usually being full. The proportion of time spent on interface issues (e.g. meetings, letters, telephone conversations) compared with patient care is rising remorselessly.

Whether you call something a continuing care unit, a 24-hour nursing staffed hostel or a medium secure rehabilitation unit does not matter, since essentially you are reproducing the asylum. The fact of the matter is that we are now entering a period of reinstitutionalisation, in both the UK and other parts of the world, for reasons that we do not really understand. Deinstitutionalisation occurred in all Western industrialised countries, at a different pace and linked to very distinct national events such as the Psychiatry-Enquete in Germany, the Law 180 in Italy or Powell's 'water tower' speech in this country.

Now, there seems to be a similar underlying pattern across various countries. This time, it is reinstitutionalisation with a rising number of forensic beds, new-style institutions in the form of supported housing, and an increasing frequency of compulsory treatment. It is not just a matter of perceptions, but rather a notion of public safety. Thus, we see a rising tide of individualist preference over communal support (e.g. the car *v.* the train), a widening gap between stronger and weaker groups in society (e.g. the rich and the poor), and a medico-legal climate of blame and risk attribution. There is probably a realistic balance between what community care can do and what might benefit from old or new kinds of institutions, but such balanced acceptability needs more careful research.

**Leff, J. (2001)** Why is care in the community perceived as a failure? *British Journal of Psychiatry*, **179**, 381–383.

**Wynter, A. (1859)** Editorial: Non-restraint in the treatment of the insane. *BMJ*, 418.

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## Compensation claims after whiplash neck injury

Although the effort in the study by Mayou & Bryant (2002) is substantial, we find that there is a tendency in such studies to fail to account for at least two important confounding variables. Our own experience, in both clinical and medico-legal practice, is that asking patients about pre-accident emotional stress is too often unreliable. Interviews with family members and review of employment records and reports often uncovers a wealth of data on these patients that was otherwise not forthcoming. Yet this is a difficult task in research studies in most cases. What is less difficult, however, is seeking the opportunity to review all pre-accident medical records, which often refer to lengthy or recent histories of significant life stressors. This is important; if some of the subjects who reported a lack of pre-accident emotional difficulties in a study actually have them, they confound the comparison of emotionally vulnerable *v.* non-vulnerable accident victims. No difference will appear to exist between the two groups because in reality they are much more alike than the researchers can know. Although researchers do use methods that suggest self-reported data is still valid, if the purpose of the research is to study psychosocial variables, then short-cuts or surrogate measures are not sufficient.

Also, post-accident stressors unrelated to an accident (e.g. death of a friend or family member, or moving house) have been shown, albeit in a small group of subjects, to be important predictors of whiplash outcome (Karlsborg *et al*, 1997). In research, to obtain this information, one need merely ask the subjects to check off what may seem like a list of not uncommon life events. We have found in clinical and medico-legal practice that patients tend to be more forthright about reporting these events, although we are impressed at how frequently people

manage to cope and keep working after many stressful life events, and yet have work disability and develop post-traumatic stress disorder after minor motor vehicle collisions. Perhaps it is as Sir John Collie remarked long ago:

> 'In short, the essential quality of a thing is its worth to the individual, and its value to him is its power to serve his private ends. On one occasion, when examining a working-man for an injury to his thumb, he observed me examining the terminal phalanx of one of his fingers, which had been partially removed, obviously as the result of a former accident. "That," said he, "is of no importance; it was done at home"!' (Collie, 1917).

**Collie, J. (1917)** *Malingering and Feigned Sickness* (2nd edn), p. 15. London: Edward Arnold.

**Karlsborg, M., Smed, A., Jespersen, H., *et al* (1997)** A prospective study of 39 patients with whiplash injury. *Acta Neurologica Scandinavica*, **95**, 65–72.

**Mayou, R. & Bryant, B. (2002)** Psychiatry of whiplash neck injury. *British Journal of Psychiatry*, **180**, 441–448.

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Regarding Mayou & Bryant's study (2002), it is interesting that the predictors of pain at 1 year are 'feeling not to blame for the accident', claiming compensation and 'anger cognitions'. With multivariate analysis, only claiming compensation at 3 months is a predictor of pain at 1 year. This means that feeling not to blame for the accident, initial anger or anger cognitions are predictors of pain only in claimants, otherwise not. Thus, of two patients, both not-at-fault, and both equally angry, it is the one who chooses litigation that will have the worse outcome. Why?

Does litigation/claims create a psychosomatic phenomenon that allows anger and victimisation to express itself as pain? Or are litigants more likely to be compelled to focus on all sources of aches and pains in their life (even pre-accident sources) by keeping pain diaries more often and by being instructed to see more physicians and therapists, to withdraw from more activities that hurt, to take more medications, to develop poor physical fitness, postural problems, medication adverse effects and anxiety?

It is further interesting that 14% of accident victims with no injury had bodily pain at 3 months! How does this happen? Is it a manifestation of psychological distress, or perhaps does pain occur as part of life, even if not injured (or, for that matter, even if not involved in an accident)? The percentage of accident victims with pain at 1 year in the 'no injury' group is half that of whiplash injury victims with pain at 1 year (27%). As one does not expect whiplash injury to create an immunity from whatever is affecting the 'no injury group', half of the whiplash injury group was going to have pain at 1 year, even if they had had no injury, or had fully recovered from their injury, because the 'no injury' group gets pain anyway. Not all of the pain at 1 year in whiplash victims can thus be due to physical effects of the initial injury, since then there would be at least some additional burden of pain from whatever factors also cause pain in the 'no injury' group as well. Statistically, half of the chronic pain that exists in whiplash patients is independent of having had an initial physical injury.

The findings of this study also suggest that when a physician encounters a patient who is not to blame for an accident and who is feeling angry, the physician should very clearly advise that entering a claim will adversely affect the patient's health and is more likely to lead to chronic pain.

**Mayou, R. & Bryant, B. (2002)** Psychiatry of whiplash neck injury. *British Journal of Psychiatry*, **180**, 441–448.

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**Authors' reply:** Drs Kwan and Friel make familiar general points about the interpretation of prospective research. However, they underestimate the practical, methodological and ethical difficulties of obtaining and using medical records and of qualifying information about life events. It is also worth noting that in medico-legal practice it is very common for medical experts and lawyers to disagree about the significance of medical histories and of life events following the identified trauma.

Dr Ferrari's first paragraph over-interprets multivariate analysis dependent on statistical significance in concluding that initial anger or anger cognitions are early predictors of pain in claimants. Although there are some differences between claimants and non-claimants, our overall experience in this study, and in a previous paper which followed up claimants for 6 years, is that the two groups are very similar (Bryant *et al*, 1997). The research findings, together with clinical experience, indicate that litigation is one of a number of reminders of the accident which do result in subjects focusing on their aches and pains. Further accidents, continuing medical complications and persistent financial difficulties are probably other important factors acting in a similar manner.

Fourteen per cent of accident victims with no recorded injury in the emergency department had pain at 3 months which was attributed to the accident. Perhaps the most likely explanation is that these people suffered minor musculo-skeletal injuries but that the symptoms did not become significant for hours or days after the accident. This is well described in relation to whiplash neck injury. It is therefore incorrect for Dr Ferrari to use our evidence to draw conclusions about the extent to which pain reported by whiplash patients may be independent of physical injury.

I also strongly disagree with Dr Ferrari's final conclusion that patients who are not to blame but angry should be advised not to enter a claim. The financial and other losses may be considerable and compensation desirable and even necessary. The more appropriate conclusion is that medical and legal procedures should take account of the patient's reactions and beliefs, avoid increasing distress and attempt to provide a sympathetic and rapid resolution of both the medical and the legal issues.

**Bryant, B., Mayou, R. & Lloyd-Bostock, S. (1997)** Compensation claims following road accidents: a six-year follow-up study. *Medicine, Science and the Law*, **37**, 326–336.

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## Regional selectivity of novel antipsychotics

Xiberas *et al* (2001) measured $D_2$ receptor occupancy in striatum, thalamus and temporal cortex in patients treated with haloperidol, risperidone, amisulpride, clozapine and olanzapine. On the basis of their findings, they conclude that in the striatum and in the thalamus atypical antipsychotics induce a significantly lower $D_2$ binding index than haloperidol does. Their results are consistent with previous studies showing only small differences between striatal and temporal cortex blockade by traditional compounds and relatively selective $D_2$

**Table 1** $D_2$ dopamine receptor binding indices in striatum, thalamus and temporal cortex, and the ratios of temporal/striatal (temporo-striatal) and thalamic/striatal (thalamo-striatal) binding indices in patients taking traditional and atypical antipsychotics (data from Xiberas *et al*, 2001)

| Drug | Binding index (%) | | | Temporo-striatal index | Thalamo-striatal index |
|---|---|---|---|---|---|
| | Striatum | Thalamus | Temporal cortex | | |
| Haloperidol 3 mg | 66.6 | 91.2 | 88.3 | 1.33 | 1.37 |
| Risperidone 6 mg | 67 | 92.2 | 92.2 | 1.38 | 1.38 |
| Amisulpride 1000 mg | 61.5 | 69.9 | 87.8 | 1.43 | 1.14 |
| Olanzapine 20 mg | 69.6 | 91.9 | 91.8 | 1.32 | 1.32 |
| Clozapine 200 mg | 45.9 | 79 | 90.1 | 1.96 | 1.72 |

blockade in temporal cortex caused by atypical antipsychotics (Pilowsky *et al*, 1997; Bigliani *et al*, 2000).

Looking at the data from Xiberas *et al* (2001), we came to different conclusions. Using equipotent doses of antipsychotics (doses which lead to the same occupation of $D_2$ receptors in the striatum), no differences in thalamo-striatal and temporo-striatal indices between typical and atypical antipsychotics could be shown (Table 1). We suggest that atypical antipsychotics do not exert special temporal lobe or limbic selectivity. The selectivity depends more on the dose than on the type of antipsychotic (typical *v*. atypical). This is in agreement with Nyberg & Farde (2000) and Geddes *et al* (2000), who argue that non-equipotent doses can partly explain differences between classical and novel antipsychotics.

**Bigliani, V., Mulligan, R. S., Acton, P. D., *et al* (2000)** Striatal and temporal cortical D2/D3 receptor occupancy by olanzapine and sertindole *in vivo*: a [123I]epidepride single photon emission tomography (SPET) study. *Psychopharmacology*, **150**, 132–140.

**Geddes, J., Freemantle, N., Harrison, P., *et al* (2000)** Atypical antipsychotics in the treatment of schizophrenia: systematic overview and meta-regression analysis. *BMJ*, **321**, 1371–1376.

**Nyberg, S. & Farde, L. (2000)** Non-equipotent doses partly explain differences among antipsychotics – implications of PET studies. *Psychopharmacology*, **148**, 22–23.

**Pilowsky, L. S., Mulligan, R. S., Acton, P. D., *et al* (1997)** Limbic selectivity of clozapine. *Lancet*, **350**, 490–491.

**Xiberas, X., Martinot, J. L., Mallet, L., *et al* (2001)** Extrastriatal and striatal $D_2$ dopamine receptor blockade with haloperidol or new antipsychotic drugs in patients with schizophrenia. *British Journal of Psychiatry*, **179**, 503–508.

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**Authors' reply:** We thank Dr Kopeček *et al* for their interest in our paper (Xiberas *et al*, 2001*b*). They conclude that atypical antipsychotics do not exert special temporal or limbic selectivity, which depends instead on drug dosages. First, we believe that generalisations drawn from data obtained from five patients, each one treated with a different antipsychotic drug, are not sound, because of intersubject variability. For instance, should Dr Kopeček *et al* have considered plasma drug concentrations and patient H2 of our article, their conclusion would have been modified. In our article, we drew conclusions from the statistical comparisons of [$^{76}$Br]-FLB457 measures obtained with positron emission tomography (PET) in subgroups of patients, receiving the usual dosage recommended by the pharmaceutical firms for each antipsychotic drug, for treating psychotic episodes.

Second, we have already reported the importance of dosage when interpreting neuroimaging measures of regional $D_2$ dopamine receptor blockade by antipsychotic drugs (Xiberas *et al*, 2001*a*). Inspection of the table that Kopeček *et al* draw from our article suggests that for a striatal $D_2$ receptor binding index approaching 65–70%, the atypical antipsychotics induce extrastriatal/striatal indices comparable with that induced by the lowest oral dosage of haloperidol reported. This is consistent with our previous publication (Xiberas *et al*, 2001*a*) where we specifically highlighted the dose-dependence of extrastriatal/striatal $D_2$ blockade, from a study in a larger sample of patients treated with an atypical antipsychotic. We demonstrated that plasma concentrations were more accurately related than daily oral doses to the different regional binding profiles determined with PET. Clearly, two binding profiles could be distinguished depending on the plasma concentration of the drug: low striatal binding associated with marked extrastriatal binding for low plasma concentrations, or marked binding in both striatal and extrastriatal regions for higher plasma concentrations. This may be applicable to both atypical and typical compounds, if very low doses of typical neuroleptics (i.e. below the recommended therapeutic dose range) are considered, but this is a speculation. Therefore, having previously highlighted the effect of dosage (Xiberas *et al*, 2001*a*), we chose to highlight in our second article (Xiberas *et al*, 2001*b*) that, at plasma concentrations obtained in actual clinical practice, and compared with haloperidol, various atypical antipsychotic drugs have a regional binding profile that is higher in mesocorticolimbic regions than in striatum.

## Declaration of interest

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**Xiberas, X., Martinot, J. L., Mallet L., *et al* (2001*a*)** *In vivo* extrastriatal and striatal $D_2$ dopamine receptor blockade by amisulpride in schizophrenia. *Journal of Clinical Psychopharmacology*, **21**, 207–214.

**—, —, —, *et al* (2001*b*)** Extrastriatal and striatal $D_2$ dopamine receptor blockade with haloperidol or new antipsychotic drugs in patients with schizophrenia. *British Journal of Psychiatry*, **179**, 503–508.

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## Measuring amygdala volume

Chance *et al* (2002) described volumetric measurement of the amygdala and found few differences between normal and schizophrenia post-mortem samples. This fails to confirm published magnetic resonance imaging (MRI) data on hundreds of individuals which have been systematically reviewed and analysed (Wright *et al*, 2000). Chance *et al* (2002) report mean absolute volumes (643 $mm^3$ for nine men and 612 $mm^3$ for nine women) that are much smaller than those reported in MRI studies. They go on to speculate on the reasons for this discrepancy and point

to 'limitations' in both MRI and meta-analysis. The authors are right to highlight the problem of anatomical definition of the amygdala *in vivo* and how other imaging parameters may obscure (or reveal) laterality effects and differences between subject groups. However, they are wrong to blame meta-analysis. Systematic review and meta-analysis of MRI data is a powerful means of quantifying the precise effects that are the subject of speculation by Chance and colleagues.

We have recently carried out just such a review of the normal human amygdala (Brierley *et al*, 2002). Some 39 studies and 51 data-sets met our inclusion criteria, allowing comparison of 1491 amygdala pairs. The weighted mean volumes (95% CI) for the left and right amygdala were 1726.7 $mm^3$ (35.1) and 1691.7 $mm^3$ (37.2), respectively. The range was from 1050 to 3880 $mm^3$. This variance is clearly worrying. We were able to examine systematically some of the causes of this and found that most imaging parameters, such as slice thickness and plane of orientation, were not particularly influential. Study size had a weak but significant effect, with larger studies tending to give smaller volumes. Anatomical definition was the most important variable. Studies which employed the Watson criteria (Watson *et al*, 1992) gave significantly larger volumes than the remainder. Gender differences persisted (male greater than female) even in studies which attempted to control for intracranial volume. Laterality effects were not significant.

The ease of obtaining high-resolution anatomical brain images afforded by modern MRI on large samples of individuals across the life span means that MRI should be taken as the gold standard on regional volumetrics rather than post-mortem samples with all their attendant deficiencies. However, in order to exploit the advantages of MRI, researchers must pay particular attention to reliability of anatomical definitions. We have proposed that Watson's criteria be adopted generally and have recommended some minor improvements (Brierley *et al*, 2002).

**Brierley, B., Shaw, P. & David, A. S. (2002)** The human amygdala: a systematic review and meta-analysis of volumetric MRI. *Brain Research Reviews*, in press.

**Chance, S. A., Esiri, M. M. & Crow, T. J. (2002)** Amygdala volume in schizophrenia: post-mortem study and review of magnetic resonance imaging findings. *British Journal of Psychiatry*, **180**, 331–338.

**Watson, C., Andermann, F., Gloor, P., *et al* (1992)** Anatomic basis of amygdaloid and hippocampal volume measurement by magnetic resonance imaging. *Neurology*, **42**, 1743–1750.

**Wright, I. C., Rabe-Hesketh, S., Woodruff, P. W. R., *et al* (2000)** Meta-analysis of regional brain volumes in schizophrenia. *American Journal of Psychiatry*, **157**, 16–25.

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**Authors' reply:** We agree with David *et al* that the key problem, which we have highlighted, in *in vivo* MRI studies of the amygdala, is anatomical definition. The ability to define anatomical boundaries at the cellular level means that post-mortem samples set the gold standard for anatomical delineation. Indeed, the generally smaller volumes of the amygdala (uncorrected for tissue shrinkage in Chance *et al*, 2002) reported in post-mortem studies are indicative of more conservative estimates when the precise boundaries can be seen. This is consistent with Brierley *et al*'s (2002) conclusion in their meta-analysis that anatomical definition is the most prominent contributor to variance in MRI volume estimates of the amygdala.

Our criticism is not of meta-analysis *per se*, but of the inclusion of some studies, which owing to low scan resolution use only very approximate anatomical definitions. Particularly problematic in schizophrenia is the use of landmarks, which may be systematically shifted with respect to the boundary of the amygdala, owing to other changes in the temporal lobe. While MRI studies have the obvious advantages of an *in vivo* assessment and larger sample size, post-mortem studies are also important as a check on the possibility of systematic bias which may enter the MRI literature (Walker *et al*, 2002).

We agree with the importance of consensus criteria for anatomical definitions which take full advantage of the improvements in up-to-date MRI visualisation. Our paper concludes with some references to studies attempting to tackle this issue for the amygdala, to which the paper of Brierley *et al* (2002) should be added.

**Brierley, B., Shaw, P. & David, A. S. (2002)** The human amygdala: a systematic review and meta-analysis of volumetric MRI. *Brain Research Reviews*, in press.

**Chance, S. A., Esiri, M. M. & Crow, T. J. (2002)** Amygdala volume in schizophrenia: post-mortem study and review of magnetic resonance imaging findings. *British Journal of Psychiatry*, **180**, 331–338.

**Walker, M. A., Highley, J. R., Esiri, M. M., *et al* (2002)** Estimated neuronal populations and volumes of the hippocampus and its subfields in schizophrenia. *American Journal of Psychiatry*, **159**, 821–828.

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## Phenomenology of acute confusional states

I read with great interest the paper by Dr Fleminger (2002) on delirium, and the relevant controversy raised by Dr Philpott regarding to whom should be attributed the first description of hypoactive delirious states (Philpott, 2002). May I suggest that this initial description was made around one century earlier than mentioned by both authors. In fact, as early as 1892 the French alienist Philippe Chaslin borrowed the term of '*confusion mentale primitive*' from a previous description proposed by Delasiauve during the 1850s. He was probably one of the first authors who gathered under a unified entity what was previously described under separate clinical features as psychosis post-influenza, post-acute diseases, post-fever and epilepsy (Chaslin, 1892). He also clearly noticed its similarity with what Lasegue had described earlier as *delirium tremens*, in which perceptual disturbances were considered as a dream-like experience (Lasegue, 1881). In his later monograph, Chaslin describes the acute confusional state as 'an acute brain disorder, consecutive to an organic significant disease, with cognitive impairment associated with delusions, hallucinations, psychomotor agitation, or reciprocally, with psychomotor retardation and inertia' (Chaslin, 1895). Despite this very early description of what has since been called hyperactive and hypoactive subtypes of delirium, there have been very few attempts to test the validity and the relevance of these subtypes. To our knowledge, at this time only one empirical exploration of what are the constitutive symptoms of each dimension has been proposed (Camus *et al*, 2000). We would like to add, concerning what Fleminger cites as possible psychological consequences of confusional experience, that another French alienist described 'permanent ideations' and 'chronic delusional states' following the post-dream-like confusional experience (Regis, 1911). We agree with

Fleminger's assumption that hyperactive subtypes are among the most stressful confusional experiences because of the possible persistence of memories of perceptual disturbances beyond the full recovery of consciousness and arousal, and beyond the normalisation of the sleep–wake cycle. But it remains unclear what factors are associated with such persistent difficulties in overcoming the dream-like experience. We hypothesise that they could be related to the implication of some specific neurobiological pathways, but their potential relationship with some premorbid personality traits should also be explored. Finally, as long as the pathophysiology of delirium is poorly understood, research into biological markers such as cerebrospinal fluid levels of neuropeptides (Broadhurst & Wilson, 2001) should be correlated to all different aspects of delirium phenomenology.

**Broadhurst, C. & Wilson, K. (2001)** Immunology of delirium: new opportunities for treatment and research. *British Journal of Psychiatry*, **179**, 288–289.

**Camus, V., Burtin, B., Simeone, I., *et al* (2000)** Factor analysis supports the evidence of existing hyperactive and hypoactive subtypes of delirium. *International Journal of Geriatric Psychiatry*, **15**, 313–316.

**Chaslin, P. (1892)** La confusion mentale primitive. *Annales Medico-Psychologiques*, **16**, 225–273.

**— (1895)** *La Confusion Mentale Primitive. Stupidité, Démence, Aiguë, Stupeur Primitive*. Paris: Asselin et Houzeau.

**Fleminger, S. (2002)** Remembering delirium. *British Journal of Psychiatry*, **180**, 4–5.

**Lasegue, C. (1881)** Le délire alcoolique n'est pas un délire mais un rêve. *Archives Générales de Médecine*, **80**, 5–28.

**Philpott, R. (2002)** Confusion. *British Journal of Psychiatry*, **180**, 467.

**Regis, E. (1911)** La phase de réveil du délire onirique. *L'Encephale*, **6**, 409–419.

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### Psychiatry in China

In response to Lyons (2001) and Kumar (2000), I have been working on the research of *Qigong*-related mental disorder and culture-bound syndrome in China for over a decade and I feel it is unfair for psychiatry in China to be represented by their remarks. I would argue that it is the misuse of *Qigong*, rather than misuse of psychiatry, that is at issue in China, according to my experience of research of *Falun Gong*-related mental disorder and culture-bound syndrome (Shan *et al*, 1987, 2000; Shan, 1999). Some of the reports about the abuse of psychiatry in China are based on political issues and lack any awareness of academic research and study in China. In fact, *Qigong* was misused in China, and the patients and practitioners who suffered with *Falun-Gong*-related mental disorders need to be treated in psychiatry. I must call for more experts in psychiatry and in the World Psychiatric Association to pay attention to the research of *Qigong*- and *Falun-Gong*-related mental disorders.

**Kumar, S. (2000)** International concern grows over psychiatric abuses in China. *Lancet*, **356**, 920.

**Lyons, D. (2001)** Soviet-style psychiatry is alive and well in the People's Republic (letter). *British Journal of Psychiatry*, **178**, 380–381.

**Shan, H. (1999)** Clinical diagnoses and Qigong-induced mental disorder (in Chinese). *Chinese Journal of Nervous and Mental Diseases*, **25**, 180–189.

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**—, Tao, M. Y. & Yu, Y. P. (2000)** *Falun Gong*-related mental disorders (in Chinese). *Journal of Clinical Psychological Medicine*, **10**, 345–347.

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## One hundred years ago

### Amaurotic family idiocy

Amaurotic family idiocy is a rare condition which has been observed in Jewish children. It was first described in 1871 by Mr. Waren Tay; since then about 68 cases have been recorded. In the *New York Medical Journal* of July 12th Dr A. Hymanson has published the following case. A male infant whose parents were Russian Jews appeared normal until the eighth month, when he ceased to take any interest in his surroundings. He would not raise his head and made purposeless movements of the limbs. The head was large, measuring 19 inches in circumference. The fontanelles were prominent and widely open, but at the age of 10 months they were gradually closing. At the age of 15 months he could not hold his head up; it was usually thrown backwards. He was very anaemic, his muscles daily became weaker and more flabby, and spontaneous movements gradually ceased. He had a vacant look and seemed to see light but did not recognise his parents. He seemed to be deaf but became frightened when anyone knocked at the door. During sleep the eyes and mouth were wide open. The pupils were slightly contracted and did not react to light. The fundi showed changes exactly similar to those described by Mr. Waren Tay. Corresponding to the macula lutea of each eye was a large bluish-white spot about twice the size of the optic disc. At its centre was a brownish-red circular dot. The optic discs were in a state of grey atrophy and the calibre of the vessels was markedly reduced. The child died at the age of 19 months. Two weeks before death there was great anorexia. He was much emaciated, could hardly move his limbs, and had gluteal bed-sores. The temperature was subnormal, 97.5° to 98° F. A necropsy was refused. Of the 68 recorded cases 40 are known to have been fatal: the result in the others is unknown. The family predisposition is shown by the fact that 28 cases occurred in 18 families. 30 cases were observed in America, 11 in England, 14 in Germany, and the remainder in other countries. The necropsies have not shown any abnormality in the form or structure of the cerebral convolutions. Thus the etiology and pathology are unknown. The chief clinical features are idiocy, weakness of all the muscles terminating in paralysis, gradual loss of sight, characteristic changes in the macula lutea, marasmus, and death at the end of the second year.

#### REFERENCE

*Lancet*, 23 August 1902, p. 519.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Epsom, Surrey