clozapine?) or does he regularly show 'simple entertainment' videos to families?

I am not surprised that many of the families 'love it'; presumably when it confirms improvement and affirms hope for the future. What of those for whom clozapine has proved less successful?

I, too, welcome hope for people with schizophrenia and their families and recognise the important contribution of clozapine. I am not sure that pointing out one or two biases and a dislike of sentimentality need totally dismiss hope and wonder at the sensitivity of those who see them as such.


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Routine blood monitoring in epileptic patients with learning disability

Sir: The audit of Amaladoss & Arumainayagam concerned epilepsy in this predisposed and vulnerable group (Psychiatric Bulletin, 1994, 18, 680–682). Their assertion that there is little justification for routine annual blood monitoring for epileptic patients is questionable, especially as only 75 cases were surveyed. Most clinicians are familiar with the argument that routine blood monitoring may identify unsuspected subtherapeutic or toxic drug levels in a patient with major communication difficulties. Furthermore, although routine measurements will not predict acute idiosyncratic drug reactions, subclinical biochemical or haematological deterioration may be discovered. This is especially important with valproate-associated hepatotoxicity – hepatic enzymes may be raised on routine monitoring when the hepatotoxicity can be reversed but this is unlikely to be the case by the time overt clinical signs develop. Careful clinical judgement is required to decide when to reduce or withdraw valproate in the light of borderline abnormalities (Wyllie & Wyllie, 1991). In one series, 36 of 37 cases of fatal valproate-associated hepatic failure had learning disability, developmental delay or congenital anomalies (Dreifuss et al, 1987). Therefore, although such fatalities are rare, we should be particularly vigilant in the learning disability population – especially as those with a lesional basis for their difficult to control seizures may well receive multiple antiepileptic drugs which is itself a risk factor for drug-associated hepatotoxicity. In this regard, it is debatable whether best clinical practice can be defined using small audit projects aimed at measuring current practice rather than clinical research per se. Similar considerations suggest that routine haematological monitoring may identify clinically unsuspected cases of significant valproate-induced thrombocytopenia as well as chronic leucopenia associated with carbamazepine or ethosuximide.

Routine blood monitoring also provides an opportunity to ensure that other appropriate tests are done, for example, thyroid function and lithium levels, in a group of patients whose access to primary care services is at times tenuous. We are currently undertaking an audit of epilepsy care in learning disability in the Oxford region. Participating consultants expressed a variety of views on the need for routine blood monitoring but the consensus opinion was in favour of annual measures of: antiepileptic drug levels, routine biochemistry and haematology and folate levels in the few taking phenytoin or phenobarbitone.


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Sirs: Amaladoss & Arumainayagam's audit concerned epilepsy in an in-patient mentally handicapped population (Psychiatric Bulletin, 1994, 18, 680–682). We conducted a similar audit on 154 in-patients within Phoenix Trust in June 1992. We do not agree with Amaladoss & Arumainayagam that there is little justification for any annual blood monitoring in the mentally handicapped who are on anticonvulsant medication.
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