

LETTERS TO THE EDITOR

Irish Journal of Psychological Medicine 1995 June; 12 (2): 81-82

The psychiatrist in primary care: let's look before we leap.

Sir – Dr Phelan in her review “The psychiatrist in primary care: let's look before we leap”¹ mentions three models of collaboration between general practitioners and psychiatrists. I would like to draw your attention to a variant as practised in the Scottish Borders over 25 years where 100% of psychiatrists collaborate with GPs.

Dingleton Hospital serves a mostly rural, scattered population of 104,000, there is no “Out-Patient Department” and all referrals go to the multidisciplinary sector team. Patients are seen for the most part in their own homes, preferably with a co-therapist. Only very small numbers are seen in the psychiatric hospital or GP surgery. Liaison takes place at regular meetings between the teams and GPs at the local health centres.

This home assessment and home treatment service has many advantages. There is improved access to care, with Dingleton's failure to attend rate running at about 5%. Patients often perceive it to be less stigmatising to be seen in the privacy of their own homes and patients' transport difficulties in this rural area are also overcome in this way. Home assessment with a co-therapist facilitates a holistic approach with improved opportunities for investigation and intervention in psycho-social and family factors. The GPs have access to the multidisciplinary team on a regular basis, with opportunities for collaboration, education, training and support.

Some of the disadvantages to collaboration models cited in Dr Phelan's article do not apply in the Scottish Borders. Because the system has evolved in this way over a period of time, administration and record keeping is not an issue. Accommodation problems are overcome by seeing people in their homes. Time spent in transit can be kept to a minimum by careful planning of visits. Hand free dictaphones and mobile phones can be used while travelling and time between joint work can be used to review cases.

Although the service at Dingleton has not yet been evaluated in the Scottish Borders, Burns et al^{2,3} evaluated the Dingleton model in an urban setting. They found no differences in clinical or social functioning outcome but a substantial reduction in inpatient care and reduced total cost for a home based service. They believe that adequate investment in funding expanded teams able to operate in a truly multidisciplinary manner, would prove cost effective in the medium term.

Thus while appreciating Dr Phelan's caution about widespread introduction of

psychiatric services in primary care settings, I would suggest the Dingleton model as one that could be extended to Ireland in both rural and urban areas. Given the Irish system where there are many single-handed GPs it is unlikely to be cost effective for multidisciplinary teams to visit every small practice on a regular basis. However, a negotiated time limited attachment may be applicable.

Eleanor Halloran, MRCPsych, MRCGP, MICGP,
Registrar in Psychiatry,
Dingleton Hospital,
Melrose,
Scotland.

References

1. Phelan D. The psychiatrist in primary care: let's look before we leap. *Ir J Psychol Med* 1995; 1: 17-21.
1. Burns T, Beardsmore A, Bhat AV, et al. A controlled trial of home based acute psychiatric services. I: Clinical and social outcome. *Br J Psychiatry* 1993; 163: 49-54.
2. Burns T, Raferty J, Beardsmore A, et al. A controlled trial of home based acute psychiatric services. II: Treatment patterns and cost. *Br J Psychiatry* 1993; 163: 55-6.

Author's reply to 'Commentary on A sceptical reflection on the diagnosis of multiple personality disorder'

Sir – I am grateful that Dr Putnam, one of North America's most eminent contributors to the multiple personality disorder (MPD) debate, should give me the opportunity to elaborate on my earlier paper in the journal.^{1,2} Dr. Putnam begins by noting that multiple personality disorder (MPD) has a long history. So, of course, have demonic possession and exorcism, but these are also chimera.

Mistakenly, he says that I note a continuity between early 17th. century reports and modern cases. This is not the case. Modern cases are quite different. For example, earlier reports describe cases where the number of personalities were relatively few (often “dual personalities”) and a reported history of childhood sexual abuse rarely reported.

Putnam says that my paper is in the tradition of prior critiques, perhaps encouraging a yawn, but the reality is that there are few, developed, sceptical critiques of MPD, hardly enough to warrant the categorisation “tradition”. A recent and distinguished exception is to be found in North *et al.*³

He takes me to task for basing my speculations about the incidence and prevalence of MPD on straw-polls (letters published in the *Bulletins of the Royal College of Psychiatrists* and the *British Psychological Society*, circulated to 6000 psychiatrists and psychologists) and correspondence with colleagues. However, by his own admission there are, as yet, no published data to resolve arguments

Epilim® Prescribing Information (Eire)

Presentation: 1. Epilim 200 Enteric Coated. A lilac-coloured, enteric coated tablet containing 200mg Sodium Valproate Ph Eur. 2. Epilim 500 Enteric Coated. A lilac-coloured, enteric coated tablet containing 500mg Sodium Valproate Ph Eur. 3. Epilim 100mg Crushable Tablets. A white, scored tablet containing 100mg Sodium Valproate Ph Eur. 4. Epilim Syrup. A red, cherry-flavoured syrup containing 200mg Sodium Valproate Ph Eur. per 5 ml. 5. Epilim Liquid. A red, cherry-flavoured, sugar-free liquid containing 200mg Sodium Valproate Ph Eur. per 5 ml. 6. Epilim Intravenous. Off white sterile, freeze dried Sodium Valproate Ph Eur. 400 mg in a clear glass vial supplied with an ampoule of 4 ml of solvent (Water for Injections). **Indications:** In the treatment of generalised, partial or other epilepsies. Epilim Intravenous may be used for short term therapy, where oral treatment is temporarily not possible. **Dosage and Administration:** Daily dosage requirements vary according to age and bodyweight. *Oral:* To be taken with or after food. Epilim may be given twice daily. Enteric coated tablets should be swallowed whole. *I.V.* Epilim *I.V.* should be reconstituted immediately prior to use and infusion solutions containing it used within 24 hours. Any unused portion should be discarded. Epilim Intravenous may be given by direct slow intravenous injection or by infusion using a separate intravenous line in normal saline, dextrose 5%, or dextrose saline. **Monotherapy:** *Oral:* Adults: Start at 600 mg daily increasing by 200mg at 3-day intervals until control is achieved. (Maximum dose 2500 mg per day). Children over 20 kg: Initially 400 mg/day with spaced increases until control is achieved. (Usually within the range 20-30mg/kg bodyweight per day). Children under 20 kg: 20 mg/kg body weight per day; in severe cases may be increased but only when plasma valproic acid levels can be monitored. Above 40 mg/kg per day monitor clinical chemistry and haematological parameters. *I.V.:* Patients already satisfactorily treated with Epilim may be continued at their current dosage using continuous or repeated infusion: other patients may be given a slow intravenous injection over 3 - 5 minutes, usually 400 - 800 mg depending on bodyweight (up to 10 mg/kg) followed by continuous or repeated infusion up to a maximum of 2500 mg/day. Epilim Intravenous should be replaced by oral therapy as soon as practicable. Daily requirement for children is usually in the range 20 - 30mg/kg/day and method of administration is as above. **Combined Therapy:** It may be necessary to raise the dose when used with anticonvulsants which induce liver enzyme activity. Dosage of barbiturates should be reduced if sedation is observed. Optimum dosage is mainly determined by seizure control and routine measurement of plasma levels is unnecessary. **Contra-indications, Warnings, Etc.** **Contra-indications:** Active liver disease, Family history of severe hepatic dysfunction, hypersensitivity to Sodium Valproate. **Side-effects:** Liver dysfunction including hepatic failure resulting in fatalities has occurred in patients whose treatment included valproic acid or sodium valproate. Patients most at risk are children particularly those under the age of three and those with congenital metabolic disorders, organic brain disease or severe seizure disorders associated with mental retardation. The incidents mainly occurred during the first six months of therapy and usually involved multiple anticonvulsant therapy. Monotherapy is to be preferred in this group of patients. Clinical symptoms are more helpful than laboratory investigations in the early stages of hepatic failure. Serious or fatal hepatotoxicity may be preceded by non-specific symptoms, usually of sudden onset, such as loss of seizure control, malaise, weakness, lethargy, oedema, anorexia, vomiting, abdominal pain, drowsiness, jaundice. These are an indication for immediate withdrawal of the drug. Patients should be instructed to report any such signs to the clinician for investigations should they occur. Whilst it is difficult to establish which, if any, investigation is predictive, tests which reflect protein synthesis e.g. prothrombin time may be most relevant. Routine measurement of liver function should be undertaken before therapy and periodically during the first six months especially in those who seem most at risk, and those with a prior history of liver disease. Hyperammonaemia without hepatic damage may occur; it is usually transient, but may occasionally present clinically. If so, Epilim should be discontinued. Valproic acid inhibits platelet aggregation. Thrombocytopenia has been reported. Prior to initiation of therapy and before surgery clinicians should assure themselves that there is no undue potential for bleeding complications. Spontaneous bruising or bleeding is an indication for withdrawal of medication. Pancreatitis, tremor, increased appetite, weight gain, transient hair loss, increased alertness, aggressiveness, hyperactivity, irregular periods, amenorrhoea, gynaecomastia, stupor and oedema have been reported. **Drug interactions** Epilim has significant interactions with phenytoin, lamotrigine and other anticonvulsants. Epilim may potentiate the effects of neuroleptics, MAOIs and other antidepressants, anticoagulants and salicylates. Cimetidine may inhibit the metabolism of Epilim. Epilim has appreciably less enzyme inducing effects than certain other anticonvulsants and loss of efficacy of oral contraceptive agents does not appear to be a problem. **Women of Childbearing Age:** An increased incidence of congenital abnormalities (including facial dysmorphism, neural tube defects and multiple malformations) has been demonstrated in offspring born to mothers with epilepsy both untreated and treated, including those treated with sodium valproate. The incidence of neural tube defects in women receiving valproate during the first trimester has been estimated to be in the region of 1%. Pregnancies should be carefully screened by alpha-fetoprotein measurement ultrasound and other techniques if appropriate. In all pregnancies monotherapy is to be recommended and dosage reviewed. The benefits of anti-epileptic therapy during pregnancy must be evaluated against the possible risks and patients should be informed of these and the need for screening. Folate has been shown to reduce the incidence of neural tube defects in the offspring of high risk women in general although no direct evidence exists in relation to women receiving anti-epileptic drugs. However, there is no reason to contra-indicate folic acid in these women. **Product Authorisation Numbers:** Epilim 200 Enteric Coated PA 77/113/1. Epilim 500 Enteric Coated PA 77/113/2. Epilim 100mg Crushable Tablets PA 77/113/5. Epilim Syrup PA 77/113/4. Epilim Liquid PA 77/113/6. Epilim *I.V.* PA 77/113/7. **P.O.M. Date of Preparation:** February, 1995. **Further information available on request from:** Sanofi Winthrop Ireland Ltd., Pottery Road, Dun Laoghaire, Co. Dublin. Telephone: (01) 2840315. © Registered trade mark.

References:

1. Chadwick D.W., *The Lancet*, 1990; 336: 291-295.
2. Dean J.C., Penny J.K. *Epilepsia*, 1988; 29 (2): 140-144.
3. Heller A.J., Chesterman P., Elwes R.D.C., Crawford P., Chadwick D.W., Johnson A.L., Reynolds E.W., *Epilepsia*, 1989; 30: 648.
4. Chadwick D.W., *Epilepsia*, 1987; 28 (2): S12-S17.
5. Gillham R.A., et al., *Epilepsy Res.*, 1990; 7: 219-225.

