

Moscow (2.6-3.8), and Chandigarh (1.9-5.6 in different areas of India; no prevalence study had been carried out in the Chandigarh region). Honolulu was utilised as the American centre despite the fact that it is very atypical demographically and that previous first admission rates for schizophrenia show Hawaii to rank comparatively low among the states; by contrast New Haven, Baltimore, and St Louis were recently reported to have high prevalence rates of schizophrenia (Myers *et al*, 1984).

Aarhus was used as the Scandinavian centre; previous prevalence studies (2.7 per 1000) (Nielson, 1976) and incidence studies (Munk-Jorgensen, 1986) have shown rates for schizophrenia in Denmark to be comparatively low. By contrast, most studies in Norway, Sweden, and Finland have reported high rates, up to 17.0 per 1000 in the Book *et al* (1978) study of northern Sweden. The final centre used in the WHO study was the St Loman's case register in Dublin. In 1982 schizophrenia prevalence figures from this case register were only 1.7 per 1000 (Walsh, personal communication), less than one-third the 5.9 per 1000 rate for the County Roscommon case register in western Ireland and less than one-seventh the rate of 12.6 per 1000 reported by Torrey *et al* (1984) for a suspected high prevalence pocket in western Ireland.

In summary, the WHO incidence study of schizophrenia studied seven centres for which previous prevalence studies would lead one to expect no more than a two-to-three-fold difference in incidence. That is precisely what was found. Until a multi-centre incidence study is done utilising centres from suspected high incidence (e.g. northern Scandinavia, western Ireland) and low incidence (e.g. some tropical areas) countries it would seem unwise to regard the WHO incidence study as a reflection of worldwide incidence rates.

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Psychiatrists' views on Treatment of Depression

SIR: I note Armstrong & Andrew's paper on the treatment of depression by Australian psychiatrists (*Journal*, December 1986, **149**, 742-750). This is one of many papers supported by the Royal Australian & New Zealand College of Psychiatrists under a so-called Quality Assurance Project.

The implications of this work are actually quite astounding. The modal length of treatment for each of the case vignettes is over fifteen hours per patient. Simple arithmetic would suggest that every consultant psychiatrist would have reached total saturation point with the referral of one hundred patients per annum. Quality treatment for these one hundred fortunate people would totally absorb the Quality Assured Practitioner to the exclusion of all other professional activities. This work obviously has more to do with maximising psychiatrists' incomes than with making psychiatry more available to the masses.

In Australia, I would add, this Quality Assurance Project is being held up as a model of excellence to which the College hope all psychiatrists will ultimately aspire, or should I say, conform.

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'Afternoon Radiator-Sitting Syndrome': Hypothermia and Early Diagnosis of Self-Induced Water Intoxication

SIR: We report on a patient with schizophrenia and acute self-induced water intoxication (SIWI) who presented with symptoms of hypothermia. Further investigations revealed a clinical sign which may be useful in the early diagnosis of SIWI.

Case report: A 37-year-old man, who had been an in-patient for 17 years, presented at 1600 hours with complaints of feeling unwell accompanied by uncontrollable shivering, shaking, and chills. Oral temperature was 35.8°C. Over

the next two hours he became increasingly confused and ataxic, and was incontinent of large volumes of dilute urine. Laboratory investigations were normal except for serum sodium which was markedly low at 121. Overnight he passed approximately eight litres of dilute urine (S.G. 1.001), and by 0800 the following morning was alert and symptom-free, with a temperature of 37°C and serum sodium of 136.

Close observation over the next two weeks revealed that he typically consumed 9–12 litres of water daily, and dropped his sodium from a morning value of 136 to a mid-afternoon value of 126–128, accompanied by recurrent, but less severe, symptoms of hypothermia. Regardless of ambient temperature, he wore several sweaters and jackets and spent much of the afternoon sitting beside, or on, the hot water radiators which supplied heat to the building. Interviews with nursing staff who knew the patient well revealed that this was his 'typical' behaviour pattern, which had been present for many years. With fluid restriction to 3–4 litres a day this pattern disappeared, and his afternoon sodium values returned to normal.

Seven additional patients with a similar pattern of afternoon radiator-sitting syndrome were identified in a single afternoon by a visual survey of five chronic psychiatric wards involving 210 patients. On further investigation, all seven showed polydipsia and recurrent afternoon hyponatraemia (sodium levels of 124–132). In each case, fluid restriction abolished the afternoon hyponatraemia and radiator-sitting behaviour. Four patients had no previous known history of hyponatremia or polydipsia. One later developed hyponatraemic seizures. Three had previous episodes of acute water intoxication with hyponatraemia and seizures. Chart reviews of these three revealed numerous descriptions of what appeared to be, in retrospect, afternoon hypothermia, dating as far back as 14 years prior to the onset of seizures and formal diagnosis of water intoxication.

The recurrent afternoon hypothermia and hyponatraemia associated with polydipsia in these patients may be related to volume overload with tapwater at room temperature, faster than it can be heated or cleared. Alternatively, the altered temperature regulation, increased thirst, impaired water homeostasis and circadian rhythmicity may all be secondary to increased hypothalamic dopamine activity and may represent a tardive hypothalamic syndrome.

Self-induced water intoxication typically goes unrecognised until the development of episodes of acute intoxication with severe neurological symptoms which may be life-threatening (Ferrier, 1985). Additionally, it may be related to tardive changes in hypothalamic dopamine function (Jones, 1984). Early diagnosis is therefore crucial. Screening for afternoon radiator-sitting syndrome appears to be a

rapid, inexpensive, sensitive, and, thus far, specific way of detecting patients at risk for this disorder.

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A Search for Sub-Clinical Arteriosclerotic Dementia

SIR: McDonald (1983) searched clinical records of 1432 psychogeriatric admissions for dementia plus one or more strokes or dementia plus epilepsy and found only 17 patients who might meet the criteria for the diagnosis of arteriosclerotic dementia, a subclass of multi-infarct dementia.

Warlow (1980) points out that the deterioration in cognitive ability in arteriosclerotic dementia presumed to be caused by a succession of minor vascular catastrophes tends to be "stepwise" but that not all the episodes are "necessarily obvious". That is to say, to account for cerebral-vascular pathology which would not have been predicted from the clinical history, a sub-clinical course of arteriosclerotic dementia must be postulated. We report a clinical search for minor cerebro-vascular episodes which do not amount to strokes in a female chronic psychogeriatric population in a mental hospital.

On a census day, all the psychogeriatric patients in Warlingham Park Hospital (from a total of 227) who had had more than five "turns" of any kind were identified. "Turns" included transient ischaemic attacks, Alvarez' Little Fits, drop attacks, faints, collapses, and tonic or clonic attacks. The patients thus identified were examined for the presence of severe dementia using the Kew Cognitive Map (McDonald, 1969).

Six months later, from the same population, a second group of patients was identified as being on any drug used in cardiovascular disease; this population was presumed to include those patients with diagnosed (and treated) hypertension. Again,