Guidelines are not directives

Sir,

Mr McDonald's editorial title seeks to comfort: the ensuing text does the opposite (Vol 64(9), 2003, p. 510). Doctors used to describe themselves as independent professionals. They could give independent advice consistent with their own education, experience and opinion. Innovation was limited by fear of poor outcomes. Now we fear lawyers as medical litigation spirals out of control. NHS and even private sector managers are frightened of the economic cost of innovation.

Not surprisingly both doctors and managers would like to hide behind the apparent protection that guidelines might give. The problem is that very little in medicine is clearly evidence-based. When do guidelines become protocols? Financial and legal pressures will try and paralyse any novel thinking and push us into protocols. It is always difficult to move the profession towards even slightly unorthodox ideas – look at the *Helicobacter pylori* peptic ulcer saga.

Guidelines can be useful in an emergency, especially for the inexperienced, but not if they are wrong or out of date. They need endorsement from local experienced practitioners to be followed with any confidence. Ill-conceived unwieldy protocols, such as the prescriptive cancer referral procedures from the Department of Health, can be actively detrimental.

Medical progress could be frozen by protocols. As the next generation is to be given dramatically shorter training there will little stimulus for innovative thought and research. No independent thought, no common-sense, no individual, inventive input, fear of ill-informed censure, no medical advances and no point doing the job other than as a well-controlled technician; is this what we and the patients really want?

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Difficult hyponatraemia in an elderly woman

Sir,

Hyponatraemia is common, and is associated with increased mortality (Gill and Leese, 1998). Causes include thiazide diuretics (Sterns, 1987) and the 'syndrome of inappropriate antidiuretic hormone secretion' (SIADH) (Hirshberg and Ben-Yehuda, 1997). Correct ascertainment of cause is important if appropriate therapy is to be given (Gill and Leese, 1998) but there is frequently confusion and error in its investigation and management (Crook et al, 1999). We report an elderly patient with complex hyponatraemia.

An 89-year-old woman was admitted with recent malaise and dizziness. She was hypertensive, had agitated depression, and lived alone. She was taking thyroxine, thioridazine and citalopram. A formal mental assessment suggested borderline dementia. Haemoglobin, plasma glucose, liver, thyroid and renal profiles were normal. Plasma sodium, however, was 119 mmol/litre, plasma osmolality 258 mOsmol/kg and urine osmolality 543 mOsmol/kg. She had a normal short synacthen test (baseline cortisol 597 nmol/litre, 30 minute post-stimulation level 708 nmol/litre).

Hyponatraemia was felt to be a result of SIADH secondary to either citalopram or thioridazine. Both drugs were withdrawn and 2 weeks later her plasma sodium had risen to 134 mmol/litre, although her malaise and dizziness remained. After a further 2 weeks, however, her plasma sodium had fallen to 127 mmol/litre, and a week later it was 121 mmol/litre. Osmolality studies showed a plasma osmolality of 274 mOsmol/kg and urine osmolality 275 mOsmol/kg.

It was now reported that she was drinking over four jugs of water daily (the patient said she did this mainly 'because she was bored'). Dilutional hyponatraemia caused by primary polydipsia was diagnosed, and her fluid intake restricted. Her plasma sodium rose gradually to 133 mmol/litre. She was later discharged with her symptoms unchanged but plasma sodium normal.

SIADH-type hyponatraemia as a result of selective serotonin reuptake inhibitor (SSRI) treatment is well recognized, and is an increasing problem, despite SSRIs' otherwise safe side-effect profile (Woo and Smythe, 1997). SIADH is relatively common in elderly hyponatraemic patients (Hirshberg and Ben-Yehuda, 1997), and SSRI drugs are a significant cause (Hirshberg and Ben-Yehuda, 1997; Woo and Smythe, 1997). An important feature of SIADH is that investigational abnormalities may mimic hypoadrenalism and thiazide-induced hyponatraemia. These causes must be excluded before SIADH is confidently diagnosed. Following withdrawal of psychoactive drugs, this patient's hyponatraemia improved, but then later recurred. At this time she was drinking considerably more fluids and her urine osmolality was appropriately dilute, consistent with primary polydipsia. Hyponatraemia resolved with fluid restriction.

This case demonstrates that logical simple investigations can lead to the accurate assessment of hyponatraemia, and the institution of appropriate treatment which is not 'blind' or over-aggressive (Gill and Leese, 1998; Crook et al, 1999). The case also shows that so-called 'hyponatraemic symptoms' are often vague, and dubiously related to hyponatraemia itself.

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