

The clinician may notice this non-adherence as missed appointments, incomplete self-monitoring data and in test results that reflect poor control. The clinician at this point, under a treat-to-target approach, may feel obliged to intensify the therapy. This carries the unintended consequence of increasing the treatment workload, further overflowing the patient's capacity to execute the plan, with ongoing deterioration not only of disease control but also of the patient-clinician relationship.

Our research group is exploring how best to respond to this form of non-adherence, which reflects the constraints of many competing demands that patients face. What can clinicians do in the meantime?

While these are early days in our journey, I would think clinicians should consider rejecting treat-to-target as not being consistent with evidence-based medicine. Why? Because the targets are not always based on high quality evidence and may be promoted and enforced without consideration of patient context and goals. We should redefine targets, prioritising goals that patients value, and involve patients with this prioritisation. Treatment burden should be favourably balanced by treatment value expressed in the answer to questions, such as, will this

treatment or procedure (for example checking your glucose daily) increase the odds that you will live longer, feel better, or live unhindered by complications of disease or treatment? These are the new targets and not many treatments achieve these goals. Let us focus on treating to these patient goals and make healthcare fit the lives of our patients. That is the basis for minimally disruptive medicine.⁵

References

1. Montori VM, Fernandez-Balsells M. Glycemic control in type 2 diabetes: time for an evidence-based about-face? *Ann Intern Med* 2009;150:803-8.
2. Hayward RA, Krumholz HM, Zulman DM, Timbie JW, Vijan S. Optimizing statin treatment for primary prevention of coronary artery disease. *Ann Intern Med* 2010;152:69-77.
3. Nilsson PM. ACCORD and risk-factor control in type 2 diabetes. *N Engl J Med* 2010;362:1628-30.
4. Russell LB, Suh DC, Safford MA. Time requirements for diabetes self-management: too much for many? *J Fam Pract* 2005;54:52-6.
5. May C, Montori VM, Mair FS. We need minimally disruptive medicine. *BMJ* 2009;339:b2803.

Conflict of interest: none declared

Letters

The Editorial Executive Committee welcomes letters, which should be less than 250 words. Before a decision to publish is made, letters which refer to a published article may be sent to the author for a response. Any letter may be sent to an expert for comment. Letters are usually published together with their responses or comments in the same issue. The Editorial Executive Committee screens out discourteous, inaccurate or libellous statements and sub-edits letters before publication. The Committee's decision on publication is final.

Drug-induced hyponatraemia

Editor, – I have read Dr Shannon's article (*Aust Prescr* 2011;34:42-5) and the article in *Medicines Safety Update* (April 2011), both of which are excellent, simply written summaries on hyponatraemia. However, I have two objections to the traditional advice of stopping the medication that causes hyponatraemia and then giving other treatments as necessary.

Firstly, it is sometimes impossible to stop an antidepressant or antipsychotic which is necessary. Also, it is unlikely that any other psychotropic drug will be better as they can all cause hyponatraemia due to the syndrome of inappropriate antidiuretic hormone secretion.¹ Secondly, the situation can be remedied by fluid restriction, either on an inpatient or outpatient basis, provided adequate explanation is given to the patient.²

The mechanism of hyponatraemia with psychotropics is probably a combination of increased fluid intake^{3,4} and stimulation of central serotonergic and alpha₁ adrenergic receptors to release antidiuretic hormone.⁵

Antidepressant-induced hyponatraemia can spontaneously remit in spite of continuing treatment,⁶ although it is safer if there is fluid restriction of 800 ml/day with gradual liberalising of the restriction as the serum sodium rises. This approach successfully raised the serum sodium in all patients in our study, and maintained levels over a six-month follow-up period.² It seems to re-set the hypothalamic osmostat and there is rarely need for sodium replacement.

To detect hyponatraemia, I assess urea and electrolyte concentrations three days after starting an antidepressant in all patients over 65 years old. If present, I treat with modest fluid restriction and monitor the patient.

M Roxanas
Department of Psychiatry
University of Sydney
Concord Hospital, Sydney

References

1. Spigset O, Hedenmalm K. Hyponatremia in relation to treatment with antidepressants: a survey of reports in the

World Health Organization data base for spontaneous reporting of adverse drug reactions. *Pharmacotherapy* 1997;17:348-52.

2. Roxanas M, Hibbert E, Field M. Venlafaxine hyponatraemia: incidence, mechanism and management. *Aust N Z J Psychiatry* 2007;41:411-8.
3. Fabian TJ, Amico JA, Kroboth PD, Mulsant BH, Corey SE, Begley AE, et al. Paroxetine-induced hyponatremia in older adults: a 12-week prospective study. *Arch Intern Med* 2004;164:327-32.
4. Marar IE, Amico JA. Vasopressin, oxytocin, corticotrophin-releasing factor, and sodium responses during fluoxetine administration in the rat. *Endocrine* 1998;8:13-8.
5. Anderson IK, Martin GR, Ramage AG. Central administration of 5-HT activates 5-HT_{1A} receptors to cause sympathoexcitation and 5-HT₂/5-HT_{1C} receptors to release vasopressin in anaesthetized rats. *Br J Pharmacol* 1992;107:1020-8.
6. Strachan J, Shepherd J. Hyponatraemia associated with the use of selective serotonin re-uptake inhibitors. *Aust N Z J Psychiatry* 1998;32:295-8.

Dr G Shannon, author of the article, comments:

I thank Dr Roxanas for his comments. My article specifically looked at the recognition and management of severe hyponatraemia, rather than the milder forms. In severe hyponatraemia, particularly if the patient is symptomatic, I think stopping of any medications known to be associated with hyponatraemia (e.g. selective serotonin reuptake inhibitors) is an essential part of the emergency management of this life-threatening condition. Consultation with the patient's psychiatric team about ongoing management of their psychiatric condition is important in the management plan.

In an asymptomatic patient with non-severe hyponatraemia, the possibility of continuing their selective serotonin reuptake inhibitor would depend on the availability of close monitoring and perceived compliance with fluid restriction, and should only be considered in consultation with the treating psychiatrist.

Book review

Therapeutic Guidelines: Rheumatology Version 2 (2010)

Casey Maddren, Academic general practice registrar, Department of General Practice, The University of Sydney, Westmead Hospital, Sydney

When looking at a resource my first question is, do I need this? The title *Therapeutic Guidelines: Rheumatology* is music to my ears, a guidebook for often difficult to manage, chronic complaints.

The guidelines have undergone rigorous assessment and reassessment with feedback from practitioners to editors. The results of this 'closing the loop' are obvious in the text.

This edition has multiple new features. These are summarised in the electronic *Therapeutic Guidelines* (eTG) and as an insert in the book format. All these additions are clinically relevant.

I foresee this guide will be immensely useful within my own practice with potential application as a reference guide for diagnostic dilemmas, patient information handouts, red flags and drugs in pregnancy (including for men planning

to conceive with their partner). Of particular note are clinical boxes throughout the text which provide an easily accessible guide to interpretation of results, comparison of presentation of arthropathies, doses of injectable steroid, joint aspiration and other common situations when a quick answer is needed. The electronic contents page improves accessibility.

A strength of the text is its holistic approach, reflecting the needs of general practice and including nonpharmacological methods, exercises (with pdf files for printing from eTG) and recommendations for ongoing monitoring of disease. Not only does the guide provide the means but also the evidence that this approach benefits patients.

Sound rheumatological management hinges on the doctor-patient relationship, with an emphasis on clear understandable information being provided by the practitioner. This text is succinct and comprehensible, enabling its use as a resource for such discussions.

Overall, *Therapeutic Guidelines: Rheumatology* is a useful resource for practitioners, students and allied health professionals.