

## Treat the numbers or treat the patient?

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Current practice guidelines, regardless of healthcare system and country of origin, increasingly carry a similar message: treat to target. These targets are often expressed in terms of laboratory parameters which are presumed to reflect the control of a patient's condition, and by extension their health and prognosis. The assumption is that 'normalising' parameters, such as lipids, blood pressure and blood glucose in patients with type 2 diabetes, will lead to better outcomes. However, these parameters are surrogate outcomes and do not guarantee long-term clinical benefits.

Is the assumption of benefit from intense control of these parameters based on high quality evidence? Some evidence

suggests that there is no benefit, but there may be marginal harm associated with intensive control of risk factors in patients with diabetes.<sup>1–3</sup> Benefits may still accrue for younger less sick patients, but this remains speculative. Even if true, those benefits would have to offset the downside of treatment, a task made difficult by the relative good health of the patients and the necessarily prolonged course of treatment. In these younger and healthier patients, intense lifestyle modification – smoking cessation, diet, exercise, stress reduction – may be more compelling than intensive drug treatment.

Treat-to-target often requires clinicians to prescribe more drugs at higher doses. This in turn calls for more laboratory testing to determine the efficacy of these interventions on the parameters of interest and to monitor the safety of the drugs on the patient's body. Treat-to-target requires patient self-monitoring and self-management in response to the monitoring results plus more visits to nurses and physicians. Higher doses and combination therapy may also increase the likelihood of adverse effects, which in turn may require increased medical attention and reduce the patient's capacity to do patient work.

The increasing demand for treatments, investigations and visits will test the capacity of the patient and their caregivers to implement these complex programs. By some estimates, the work of being a patient with diabetes requires more than two hours every day.<sup>4</sup> Patients are expected to prioritise this 'part-time job' – to understand, plan and enrol others to help with the plan, to implement and adhere to the plan, and to reflect and value the treatment enough to keep going day after day. They have to fit this around the work of being a parent, sibling, child, spouse or relative, the work of being an employee or boss, and the work of being a citizen, a hobbyist, or a sports player.

The extent to which the patient's other 'jobs' are flexible enough to accommodate the ever-increasing work of being a patient and the ability of patients to enrol others to assist with the tasks of 'patienthood' may vary over time. Eventually, the expansion of patient workload may exceed the capacity of the patient or their caregivers to accommodate its demands. This forces the patient to prioritise, compromise and do only part of the expected patient work. They may then appear to be non-adherent to treatment.

### In this issue...

Patients, particularly those with chronic illnesses, need patience. The work of being a patient, discussed by Victor Montori, will be increased by having to manage adverse effects, such as those mentioned by Tim Lambert in his review of antipsychotic drugs. Some frustrated patients can become agitated and Gordian Fulde advises how to help them.

Atrial fibrillation is a common chronic condition. With alternatives to warfarin therapy now emerging, Himabindu Samardhi and colleagues review the treatment of the arrhythmia.

Drug treatment is delivered in different formulations. These formulations often contain several substances apart from the active ingredient. Alison Haywood and Beverley Glass briefly review the common excipients and the adverse reactions they can cause.

Some excipients can cause anaphylaxis so it is appropriate that this issue is accompanied by the latest version of the *Australian Prescriber* wallchart. This has been produced with the assistance of several specialist colleges and should enable health professionals to deal with most cases of anaphylaxis.

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.<sup>5</sup>

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## Letters

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### 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.<sup>1</sup> Secondly, the situation can be remedied by fluid restriction, either on an inpatient or outpatient basis, provided adequate explanation is given to the patient.<sup>2</sup>

The mechanism of hyponatraemia with psychotropics is probably a combination of increased fluid intake<sup>3,4</sup> and stimulation of central serotonergic and  $\alpha_1$  adrenergic receptors to release antidiuretic hormone.<sup>5</sup>

Antidepressant-induced hyponatraemia can spontaneously remit in spite of continuing treatment,<sup>6</sup> 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.<sup>2</sup> 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.

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