are enjoying a second vogue as a means of controlling symptoms quickly during the period between starting and responding to a disease-modifying drug. The long-term use of glucocorticoids needs to be weighed against their serious adverse effects, such as their propensity to cause hypertension. When prescribing combination therapy for rheumatoid arthritis, it is important to make dosage adjustments and substitutions in order to achieve objective evidence of disease suppression and to accommodate intolerance to individual drugs or drug-related adverse events. Disease control is of paramount importance in order to reduce cumulative joint damage and the increased cardiovascular mortality[3], both of which have been shown to correlate with unsuppressed disease activity.

There is evidence that fish oil in anti-inflammatory doses can reduce symptoms in rheumatoid arthritis[4] and that fish oil (and other interventions which increase dietary intake of omega-3 fatty acids) generally reduces cardiovascular mortality.[5] Fish oil has also been shown to reduce discretionary NSAID use in rheumatoid arthritis.[4] It also has a number of favourable effects on cardiovascular physiology, including a modest reduction in blood pressure and reduced arterial stiffness.

The important practical point is that a 'need' for NSAIDs in rheumatoid arthritis can be used as a prompt for more intensive application of other therapies. This approach is especially important in patients for whom treatment of other health problems, such as hypertension and heart failure, may be compromised by NSAIDs.

Situations do arise in which it is decided that a patient with cardiovascular disease requires treatment with an NSAID when alternative approaches have failed. If possible the NSAID should be used for second-line analgesia in as small a dose and for as short a period as needed to control symptoms.[6] A short-acting drug is preferable to one with a long half-life. It is important to realise that NSAIDs can perturb cardiovascular homeostasis in ways that run counter, directly or indirectly, to the beneficial actions of drugs used to manage hypertension and cardiac failure. The combination of ACE inhibitors and diuretics with NSAIDs may be especially problematic and should be avoided, if possible. While there are no absolute contraindications to using NSAIDs with cardiovascular drugs, it needs to be recognised that NSAIDs can compromise treatments for cardiovascular disease.

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Professor Cleland and Dr James have a research interest in the health benefits of fish oil. The Royal Adelaide Hospital Preventive Care Centre distributes bottled fish oil for patient use.

Letters

Letters, which may not necessarily be published in full, should be restricted to not more than 250 words. When relevant, comment on the letter is sought from the author. Due to production schedules, it is normally not possible to publish letters received in response to material appearing in a particular issue earlier than the second or third subsequent issue.

Pre-eclampsia

Editor, – I read with interest the article on biochemical tests in pregnancy (Aust Prescr 2006;29:48–52) and wish to comment on the discussion of pre-eclampsia. The author maintains that the diagnosis is based on a triad of hypertension, proteinuria and oedema, yet the Australasian Society for the Study of Hypertension in Pregnancy has issued a consensus statement which asserts otherwise.[1] While hypertension is a requirement, proteinuria (as one of a range of possible end organ effects) is not mandatory to make the diagnosis. Oedema is specifically excluded unless its onset is rapid and generalised. This is important to appreciate as severe forms of pre-eclampsia (and indeed eclampsia) can occur in the absence of the 'triad'. Furthermore, 'routine' urinalysis at each visit in low-risk pregnancies has been discontinued in many centres due to its limited value.

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Associate Professor HA Tran, author of the article, comments:

In pre-eclampsia the detection of hypertension is of utmost importance and blood pressure needs to be rigorously controlled. The presence of proteinuria and oedema is less critical but will further assist in arriving at the correct diagnosis. Although eclampsia can occur in the absence of the 'triad', alternative differential neurological diagnoses need to be considered.

While the clinical utility of 'routine' urinalysis may not be as important in the diagnosis of pre-eclampsia, it is sometimes useful in detecting asymptomatic bacteriuria and glycosuria. Bacteriuria confers an increased risk of pyelonephritis and prematurity[1], and glycosuria may identify unsuspected diabetes other than gestational diabetes. Interventions for both of these conditions can result in better outcomes.[1,2] It is of additional interest that the current British guideline for antenatal care recommends that 'whenever blood pressure is measured in pregnancy a urine sample should be tested at the same time for proteinuria'.[3] The decision to discontinue this practice in low-risk patients is then probably a function of cost versus benefit.

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Deleterious cognitive effects of antimuscarinic drugs

Editor, – I suggest that the article 'Anticholinergic drugs for overactive bladder' (Aust Prescr 2006;29:22–4) gives insufficient prominence to the inevitable occurrence of cognitive impairment from antimuscarinic drugs. There is overwhelming evidence that all antimuscarinic drugs cause cognitive impairment even in healthy people[1], and this is frequently clinically significant in older people.[2,3,4] Any possibility that a treatment will worsen patients' mental function has profound implications and must be regarded with the utmost seriousness.

The therapeutic margin is narrow, or non-existent, and individual variations in blood concentrations and response mean that in practice it will be difficult to achieve a consistently favourable therapeutic effect. Many other commonly used drugs also have antimuscarinic effects so interactions are likely to be frequent. I suggest the average practitioner has insufficient knowledge of these interactions to successfully avoid them.[5]

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Associate Professor KH Moore, one of the authors of the article, comments:

Dr Gillman makes an important point regarding the potential for anticholinergic drugs to induce or exacerbate cognitive impairment, especially in the elderly. However, he paints a rather black picture with very broad strokes so an examination of the evidence is needed.

Reference 1 describes a very precise psychometric analysis of scopolamine administration in 24 people, that showed significant decline in performance on spatial and pattern memory tests. This is hardly 'overwhelming evidence that all antimuscarinic drugs cause cognitive impairment'.

Similarly, references 2 and 3 describe studies with 30 and 16 users of a wide range of anticholinergic drugs. The first study showed a 19% attributable risk of mild cognitive impairment for these drugs. In the second paper, all patients were receiving the cholinesterase inhibitor donepezil for Alzheimer's disease. Not surprisingly, donepezil was less

effective for preventing cognitive decline in those on anticholinergic drugs (at two years, but not at one year).

Reference 4 is an interesting review article about the role of anticholinergic drugs in delirium but also discusses studies that included small numbers of patients (n = 15–34).

Reference 5 is a detailed review of the pharmacokinetics of a range of bladder-active anticholinergics. It is very informative but does not appear to support the suggestion that they should be avoided.

Nevertheless, our article could have made greater mention of the risks of anticholinergic therapy in exacerbating or precipitating cognitive impairment, especially in the elderly. These drugs should only be given in conjunction with bladder training at the lowest dose possible to achieve reduced frequency, urgency or urge incontinence, and for the shortest duration possible.

Confessions of a biased reader

Editor, – I wonder how many other people share my obsession about checking declarations of conflicts of interest before they read any letter or article?

I note many well-credentialled academics seem very committed to evidence-based medicine when presenting their arguments. For me, all this effort becomes completely neutralised when I realise that they have received sponsorship associated with the very products they are arguing for. Unfortunately, my bias is so compelling that I cannot take their well presented discussion seriously. How many other people suffer from this problem?

Chris Commens
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Book review

Pocket guide to ECGs. 2nd edition. Duncan Guy.

Sydney: McGraw-Hill; 2006. 162 pp. Price $37.95

Maros Elsik, Cardiology Fellow, Department of Epidemiology and Preventive Medicine, Monash University, The Alfred Hospital, Melbourne

This book is aimed at general practitioners, medical students, hospital residents and nursing staff. It is now in its second edition so it has clearly found a market. Having read numerous similar books, though not the first edition of this guide, I found it to be useful and practical.

The book is divided into four sections. The first section is devoted to the normal ECG. This describes the usual 'normal' parameters, but also includes a section on the so often ignored but commonly encountered sources of artefact (and misdiagnosis) such as calibration, tremor and lead reversal. The second section describes common abnormalities seen in clinical practice, and provides pathophysiological causes for them. The format is consistent throughout, easy to follow and interspersed with practical and relevant 'clinical tips'. Section three, the so-called quick reference guide to common cardiac disorders, is logically ordered and sufficiently detailed. In the era of increasing use of devices, it was refreshing to see section four on pacemakers and pacemaker ECGs, stating that significant abnormalities can be detected with a standard ECG, rather than interrogating the device first. The use of real rather than digitally enhanced ECGs throughout the text is of much practical benefit.

A few minor shortcomings of the text include the absence of an index (despite a detailed list of contents), only a brief description of early repolarisation (often a source of confusion), and although not entirely specific, the criteria for differentiating ventricular tachycardia from less serious broad complex tachycardias are not listed. It would have also been useful to include a few examples of commonly encountered and potentially serious electrolyte disturbances as well as digitalis effect and toxicity.

The ECG rulers on the back cover, and the accompanying CD-ROM with self-test ECGs, are additional useful extras, although the CD did not work on my computer.

I found this book easy to follow and packed with useful information. I would recommend it to readers of *Australian Prescriber* as a useful guide and a quick reference.