LETTERS

Multicentre research: negotiating the ethics approval obstacle course

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TO THE EDITOR: The obstacles presented by Human Research Ethics Committees (HRECs) have caused a significant delay in commencing a valuable research project.

We are currently conducting a multicentre study investigating the outcomes of hypertensive pregnancies in a cohort of 1620 women. It is a retrospective review of medical records and does not entail any participation of the women. Ethics approval was sought and gained from the New South Wales Health Department and one other NSW area health service (AHS) involved in the study. The bulk of the medical records (85%) are held by this AHS and a smaller proportion by eight other AHSs in NSW. Despite these prior approvals, the process of gaining ethics approval from the eight AHSs was fraught with obstacles at every stage. After 8 months' work, we have received approval from the HREC of each of the AHSs.

Our experience has revealed many inconsistencies in the requirements of the HRECs in the different AHSs, as summarised in the Box. These inconsistencies highlight discordances with the guidelines to support researchers and HRECs drawn up by the National Health and Medical Research Council (NHMRC).

The NHMRC's National Statement on Ethical Conduct in Research Involving Humans[1] clearly outlines that, once approval has been gained from one HREC, other sites should accept that approval. Unfortunately, it seems that the HRECs involved in giving approval for our study did not follow the guidelines relating to multicentre projects. Other researchers have reported similar problems.[2-4] Breen and Hacker[2] suggest that HRECs are slow to adopt a simplified review process because this interferes with traditional practices of each committee making its own assessment.

It is indisputable that ethics considerations are a vital component when undertaking human research. It is also crucial to have a reliable and trustworthy process that evaluates research proposals in order to protect participants from physical and psychological harm. However, it has taken the research midwife (who is on a 1-year non-renewable grant) 8 months to secure ethical approval at all sites. This process is cumbersome and counterintuitive to the principles and guidelines for multicentre research in this country.

1. National Health and Medical Research Council. National statement on Ethical Conduct in Research Involving Humans. Canberra: NHMRC, 1999.
2. Breen KJ, Hacker SM. Privacy legislation and research [comment]. *Med J Aust* 2002; 177: 523-524.
3. Whitmen D, Webb P, Green A. National ethics committee urgently needed [letter]. *Med J Aust* 2003; 178: 187.
4. Jamrozik K, Kolybaba M. Are ethics committees retarding the improvement of health services in Australia? *Med J Aust* 1999; 170: 26-28. ❑

Summary, by area health service (AHS, coded S to Z), of different requirements for gaining ethics approval for a multicentre study

Area health service	S	T	U	V	W	X	Y	Z
No. of pages of application form	19	20	19	20	12	23	2	11
No. of copies of form required	1	1	17	15	20	16	1	14
No. of hospitals in AHS covered by approval	2	2	4	1	3	5	3	5
Approval covered private hospitals in AHS also	na	na	Yes	No	No	na	No	na
No. of contacts made (phone/letter/email) to gain approval	20	15	20	15	20	30	10	20
Time taken to gain approval	3 months	5 months	4.5 months	8 weeks	6 weeks	6 weeks	1 week	3 weeks
Special requests during approval process	A	F	A, B, C, E	A	A, D	A		G, H
Approved after first submission	Yes	No	No	Yes	Yes	Yes	Yes	Yes

na = not applicable.
A = Asked for local researcher to be a contact person for the study.
B = Charged a $33 fee to submit application.
C = Requested scientific protocol with references.
D = Requested budget form.
E = Reviewed by scientific advisory committee before human research ethics committee (HREC).
F = Requested consent and subject information forms.
G = University HREC's approval as well as approval of area health service HREC required.
H = Final approval required from chief executive officer of major hospital in that AHS.

Surveillance for Barrett's oesophagus: if you do it, do it properly

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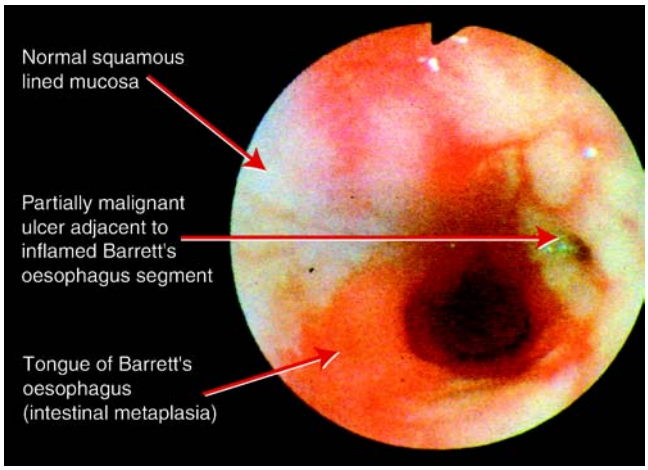
TO THE EDITOR: Reflux oesophagitis is an increasingly common medical condition, and up to 10% of all patients with reflux oesophagitis have associated columnar-lined oesophagus, or Barrett's oesophagus (Box).[1] This is associated with an increased risk of adenocarcinoma in Australian data of 1 in 176 patient-years.[2] Current surveillance guidelines for Barrett's oesophagus recommend second-yearly endoscopy with quadrantic biopsies at 2–3 cm,[3,4] although recent reviews have suggested that the time interval can be extended.[1] No prospective study has demonstrated that screening for Barrett's oesophagus improves survival in the screened population. All the evidence is based on retrospective reviews. If, however, screening is to be of benefit, then adequate tissue sampling is required, other-

wise the screening test provides false reassurance and is a poor use of endoscopy resources.

We retrospectively audited the endoscopies performed for Barrett's oesophagus (with intestinal metaplasia) surveillance at our institution over 5 years (1996–2001). In this period, 253 endoscopies were performed as surveillance procedures for Barrett's oesophagus. We reviewed the endoscopy and histopathology reports to determine whether an adequate number of biopsies had been taken.

Barrett's oesophagus



We found that quadrantic biopsies (defined as four biopsies per 2 cm or less) at every 2–3 cm were performed in 27 of 72 (38%) short-segment, 27 of 150 (25%) long-segment (3–10 cm), and 1 of 13 (8%) extensive (> 10 cm) Barrett's oesophagus. An acceptable number of biopsies were taken from 40% of patients. The number of biopsies taken per centimetre of Barrett's oesophagus was inversely proportional to the length of Barrett's oesophagus.

The median interval between surveillance procedures was 12 months. In most surveillance procedures (137/217; 63%) the endoscopy was performed following medical review in the outpatient department rather than because of planned call-back, although in only a few cases did it appear to be due to alarm symptoms such as dysphagia or weight loss. There was little consistency in recommendations among the medical staff. Only one cancer was identified through surveillance in this period, with another presenting in a patient previously on the call-back system who had been lost to follow-up. Three high-grade dysplasias were found.

Our retrospective audit revealed that the endoscopists were not following biopsy guidelines, and revealed significant variances in practice. Previously, we found a similar picture with post-polypectomy surveillance; with re-education and development of a prospective review of all cases, we have been able to greatly improve this aspect of practice.[5]

We were surprised at the result of our audit, and invite other endoscopy units to perform a similar audit of their own practice. It has encouraged us to develop a process similar to the one we have adopted to improve post-polypectomy surveillance. This should improve our practice and enable more efficient utilisation of the endoscopy facility.

1. Spechler S. Clinical practice. Barrett's esophagus. *N Engl J Med* 2002; 346: 836-842.
2. Hillman LC, Chiragakis L, Clarke AC, et al. Barrett's esophagus: macroscopic markers and the prediction of dysplasia and adenocarcinoma. *J Gastroenterol Hepatol* 2003; 18: 526-533.
3. Sampliner RE. Practice guidelines on the diagnosis, surveillance, and therapy of Barrett's esophagus. The Practice Parameters Committee of the American College of Gastroenterology. *Am J Gastroenterol* 1998; 93: 1028-1032.
4. Digestive Health Foundation. Gastro-oesophageal reflux in adults — a guideline for clinicians. 3rd ed. Sydney: Gastroenterological Society of Australia, 2001: 18-19.
5. Bampton PA, Sandford JJ, Young GP. Applying evidence-based medicine improves use of colonoscopy resources in patients with a moderate risk of colorectal neoplasia. *Med J Aust* 2002; 176: 155-157. ❑

Lessons from early large-scale adoption of celecoxib and rofecoxib by Australian general practitioners

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TO THE EDITOR: The lessons from the introduction of COX-2-selective non-steroidal anti-inflammatory drugs (NSAIDs), related by Kerr et al,[1] have a corollary in the introduction of angiotensin-II-receptor antagonists 2 years previously. Both cases involved common conditions (osteoarthritis and hypertension), with extensive prescribing of newly developed and marketed agents, which blocked an enzyme further down the cascade of reactions to avoid adverse outcomes — the gastrointestinal upset and bleeding associated with non-selective NSAIDs, and the cough and angioedema caused by angiotensin-converting enzyme inhibitors. In both conditions, off-patent, low-cost alternative drug therapies were available — paracetamol and acetylsalicylic acid, and thiazide diuretics and β-blockers, respectively.

My quantitative investigation of general practitioners' perceptions of newer versus older antihypertensive agents suggested that they thought newer agents were more efficacious, and were safer in the short term and long term, but were more expensive.[2] These beliefs were held despite the lack of long-term safety data. Younger doctors were more likely to hold these beliefs. It is possible that the experience of using older medications permitted older doctors to maintain a healthy scepticism towards the marketing claims of medical representatives. It may be interesting for Kerr and colleagues to look at the demographics of GPs in the General Practice Research Network to see if these findings hold true for this cohort.

1. Kerr SJ, Mant A, Horn FE, et al. Lessons from early large-scale adoption of celecoxib and rofecoxib by Australian general practitioners. *Med J Aust* 2003; 179: 403-407.
2. Nelson MR, Reid CM, Krum H, McNeil JJ. Factors influencing family physician adherence to hypertension treatment guideline recommendations on the initiation of pharmacotherapy. *Am J Cardiovasc Drugs*. In press. ❑

Effect of computerised prescribing on use of antibiotics

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TO THE EDITOR: We would like to add our perspective to the discussion on computerised prescribing.[1] Electronic

prescribing with appropriate decision support is recognised by the Medication Safety Taskforce of the Australian Council for Safety and Quality in Health Care as a key initiative to prevent patient harm.[2] Such systems reduce the opportunity for prescribing errors and improve patient safety.[3]

The widely espoused benefit of the clarity and legibility associated with electronic prescribing appears to have led many to assume that electronic prescribing is an essential component of medication safety systems whether or not it includes a decision support system.

In fact, electronic prescribing without decision support has been associated with an increase in the incidence of error[4] and inappropriate use of medications.[1]

A computer-generated discharge summary was developed at a tertiary referral teaching hospital in Brisbane. With this system, a discharge prescription was generated using information entered from the database by the medical officer.

As part of standard practice, a pharmacist reviews all discharge prescriptions and compares them with the inpatient medication chart, discussing any apparent errors with the medical officer. We conducted an audit of 200 consecutive medical discharge prescriptions (100 generated by computer and 100 handwritten) in mid-2001. The errors detected are summarised in the Box. The same medical staff were responsible for both types of prescriptions. Significantly more errors in prescribing were noted for the computer-generated prescriptions than for the handwritten scripts ($P < 0.001$). The proportion of errors with potential for harm was similar in both groups.

Three specific types of error occurred more frequently with electronic prescribing. We can speculate that dosing errors occurred when previous discharges were copied and the previous dose was continued, duration errors occurred as a result of a computer default to 10 days' therapy, and the continuation of drugs not required for discharge resulted from copying previous medication records and not reviewing the current drugs prescribed.

This uncontrolled observational audit demonstrated that electronic prescribing without decision support in busy medical wards can significantly increase the risk of patient harm when compared with the handwritten system. The discharge prescription component of this system was withdrawn on the basis of this audit, and the paper-based system reinstituted until a safer alternative becomes available.

Comparison of error rates with electronic and handwritten discharge prescribing systems

Examples of prescribing errors		Computer	Handwritten
Number of prescriptions		100	100
Number of drugs		700	605
Omissions	Warfarin and irbesartan omitted	12	16
Duplications	Spironolactone and atorvastatin duplicated	2	0
Dosing errors	Prednisolone 50 mg in the morning for 10 days ordered: should have been reducing by 10 mg every second day	25	5
Drug errors	Diltiazem oral 60 mg three times a day ordered: should have been diltiazem slow release 180 mg in the morning	4	4
Drug name unclear	Fluticasone inhaler: no strength	6	0
Duration error	Antibiotics intended for 3 or 5 days: ordered for 10 days (default quantity)	13	1
Drug not required on discharge	Frusemide 80 mg twice daily was continued: the drug had been stopped during admission	15	4
Route error	Glyceryl trinitrate 5 mg oral ordered: patch was the intended form of drug	3	0
Frequency error	Carvedilol ordered for mornings: had been twice daily in hospital	1	0
Total number of errors		81	30
Error rate per item		11.6%	5.0%

1. Newby DA, Fryer JL, Henry DA. Effect of computerised prescribing on use of antibiotics. *Med J Aust* 2003; 178: 210-213.
2. Australian Council for Safety and Quality in Health Care. Second national report on patient safety — Improving medication safety. Canberra: Australian Council for Safety and Quality in Health Care, 2002: 39-46.
3. Bates DW, Teich JM, Lee J, et al. The impact of computerized physician order entry on medication error prevention. *J Am Med Inform Assoc* 1999; 6: 313-321.
4. Shojania KG, Duncan BW, McDonald KM, et al, editors. A critical analysis of patient safety practices. Evidence report/Technology assessment No. 43. Rockville, Md: Agency for Healthcare Research and Quality, 2001. (AHRQ Publication No. 01-E058.) ❑

Palliative care: new guidelines for psychosocial care

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TO THE EDITOR: The Supplement on palliative care (15 Sep 2003) provided a welcome overview of this critical area of clinical practice.[1] Many of the articles emphasised the generalist nature of palliative care and the importance of multidisciplinary care and teamwork.

Recently published guidelines[2] now provide evidence-based information on psychosocial aspects of care for health professionals working in the field, as well as surgeons, radiation oncologists, medical oncologists, general practitioners, nurses, social workers, psychologists, psychiatrists, physiotherapists and occupational therapists. These are the world's first comprehensive, evidence-based guidelines on the social, psychological and economic impacts of cancer and how these can be better prevented, managed and treated by health professionals. They cover the whole spectrum of cancer care, from diagnosis through to treatment and palliation.

The guidelines are based on comprehensive and systematic reviews of the international research literature and an extensive consultative process to ensure their clinical relevance. Developed by the National Breast Cancer Centre and

the National Cancer Control Initiative (NCCI) and funded by the Federal Government, the guidelines were approved by the National Health and Medical Research Council (NHMRC) in April 2003. The guidelines cover the most commonly occurring cancers: colorectal, breast, gynaecological, head-and-neck, lung, pancreatic, prostate and urogenital cancers, and melanoma and non-Hodgkin's lymphoma. Recommendations for clinical practice are rated according to NHMRC levels of evidence.[3]

Four main areas are addressed by the guidelines:

- understanding the challenges of cancer and how people react;
- provision of care by the treatment team to all patients with cancer;
- referral for specialised care;
- issues requiring special consideration — culture, age, geography and sexual orientation.

The guidelines were developed to assist health professionals in supporting adult cancer patients. This includes providing information and choice to patients, helping them deal with procedures and treatments, providing emotional and social support, ensuring continuity of care, and dealing with specific concerns that may arise, including anxiety and depression.

The guidelines can be accessed at the NCCI's website (www.ncci.org.au), or hard copies can be obtained from the National Breast Cancer Centre.

1. Maddocks I, O'Connor M, Dunne P, Barnes T, editors. Palliative care: a new dimension in healthcare. *Med J Aust* 2003; 179 (6 Suppl): S1-S48.
2. National Breast Cancer Centre and National Cancer Control Initiative. Clinical practice guidelines for the psychosocial care of adults with cancer. Sydney: National Breast Cancer Centre, 2003. Available at: www.ncci.org.au (accessed Nov 2003).
3. National Health and Medical Research Council. A guide to the development, implementation and evaluation of clinical practice guidelines. Canberra: NHMRC, 2000. ❑

Tissue plasminogen activator (tPA) for acute ischaemic stroke: why so much has been made of so little

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TO THE EDITOR: I respect the opinion of Hoffman[1] and others who have recently expressed concerns about the use of tissue plasminogen activator (tPA) in patients with ischaemic stroke. However, it is critical that these views do not dampen the enthusiasm for better stroke treatments. The Therapeutic Goods Administration recently approved tPA for ischaemic stroke patients with symptoms of less than 3 hours' duration. This approval has catalysed collaboration between stroke units around Australia and may provide benefits to patients who do not receive tPA, by improving clinical pathways, as well as a more coordinated approach to treatment.

Internationally acclaimed Australian stroke experts have already rebutted the arguments of opponents of the National Institute of Neurological Disorders and Stroke (NINDS) trial,[2,3] and an independent reanalysis of the NINDS data showed tPA to be more effective than originally reported.[4] It is time to stop bickering about the NINDS trial[5] and move forward, by focusing on a collaborative effort between emergency departments and stroke teams. In the near future, there will surely be better data on new-generation thrombolytics and other agents; all will operate on similar "time is brain" protocols, as did the NINDS trial.

Obviously, more data would be useful, but when clinicians are faced with patients with acute stroke *today* they must give the patients and their families the facts about tPA, regardless of their personal opinion. These are best expressed in terms of the absolute risk reduction for disability seen in the NINDs trial. It is interesting to note the widespread discomfort with the 6.4% risk of intracerebral haemorrhage (half of which were fatal) in this trial of a "medical" therapy. Surgeons quote similar morbidity and mortality risks every day to patients undergoing procedures such as coronary artery bypass grafting. Additionally, many surgical procedures became established on much less solid evidence than the NINDs trial, and yet there is no outcry. For example, many thousands of carotid endarterectomies were performed before the publication of controlled-trial data.

In properly selected patients in expert hands, we now have a treatment that works. Its risks should not be underestimated, but should also be placed into context when compared with other powerful therapies for serious illnesses. Patients presenting with acute ischaemic stroke in Australia today have the chance to benefit from tPA, but future patients will benefit even more, partly because of the process that is being undertaken to institute pathways for using this drug.

1. Hoffman J. Tissue plasminogen activator (tPA) for acute ischaemic stroke: why so much has been made of so little. *Med J Aust* 2003; 179: 333-334.
2. Donnan G, David S, Levi C. Thrombolysis for acute ischaemic stroke: revisiting the evidence. *Med J Aust* 2003; 179: 386-389.
3. David S, Parsons M, Butcher K, Szoeke C. Thrombolysis for acute ischaemic stroke: revisting the evidence. *Med J Aust* 2003; 179: 386-389.
4. Ingall T, O'Fallon W, Louise T, et al. Initial findings of the rt-PA acute stroke treatment review panel. *Cerebrovasc Dis* 2003; 16 Suppl 4: S1-S125.
5. Tissue plasminogen activator for acute ischaemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. *N Engl J Med* 1995; 333: 1581-1587. ❑

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IN REPLY: I agree with Blacker that enthusiasm generated by thrombolytic therapy could lead to benefit for stroke patients, independent of whether the therapy is actually useful, or even whether they receive it. However, I believe there are better ways to encourage rational stroke care.

Correspondents

We prefer to receive letters by email (editorial@ampco.com.au). Letters must be no longer than 400 words and must include a word count. All letters are subject to editing. Proofs will not normally be supplied. There should be no more than 4 authors per letter. An "Article Submission Form" (www.mja.com.au/public/information/instruc.html) must be completed and attached to every letter.

There should be no more than 5 references. The reference list should not include anything that has not been published or accepted for publication. Reference details must be complete, including: names and initials for up to 4 authors, or 3 authors et al if there are more than 4 (see mja.com.au/public/information/uniform.html#refs for how to cite references other than journal articles).

I also acknowledge that some experts believe thrombolysis is proven to be beneficial if used in accordance with NINDS (National Institute of Neurological Disorders and Stroke) guidelines. Others believe the question is not yet settled and are concerned that widespread adoption of the guidelines will lead to more harm than good. It is one thing to enthuse about *possible* benefits of a treatment, particularly for a devastating disease for which we have traditionally had little to offer. It is entirely different to embrace this therapy, even though it may be harmful overall, simply out of this frustration.

I disagree with Blacker that the best way to inform patients about thrombolysis is "in terms of the absolute risk reduction for disability seen in the NINDS trial". Regardless of concerns about the trial itself, it is always foolhardy to accept data from one small study as "the truth". Many other trials have found far worse results — it would be equally inappropriate to base all estimates on their worst outcomes. Finally, there is strong evidence that, even if NINDS-like "success" could be achieved under optimal circumstances, routine use in the community might well lead to overall harm.

I am also cautious about Blacker's comparison of thrombolysis and some surgical procedures in terms of adverse effect rates. No single adverse effect rate is or is not acceptable for all interventions — a 90% rate of intracerebral haemorrhage could be acceptable in a disease with 100% mortality, while a 2% rate would be completely unacceptable in a disease with no long-term morbidity. Furthermore, the adoption of many surgical interventions in the absence of persuasive evidence does not justify repeating this mistake.

Of course we *must* routinely make decisions without definitive evidence, based on our best estimates of benefits and harms. We should never take such decisions lightly, and should in general embrace the precautionary principle, which tells us not to adopt new therapies without reasonable evidence of their safety (especially when any benefit is likely to be extremely small).

Although some advocates support thrombolysis based on current knowledge, my reading of available evidence is far less sanguine. That is why I continue to argue that this treatment should not be introduced into routine practice until far better evidence of its benefit outweighing its harm becomes available. Given the possibility that this treatment will harm stroke patients overall, and the likelihood that any potential benefit is very limited for the stroke population as a whole, I ask once again, why is so much being made of so little? ❑

1. Campbell DJ. Heart failure: how can we prevent the epidemic? *Med J Aust* 2003; 179: 422-425.
2. Bradley TD, Floras JS. Sleep apnea and heart failure. Part I: obstructive sleep apnea. *Circulation* 2003; 107: 1671-1678.
3. Bradley TD, Floras JS. Sleep apnea and heart failure. Part II: central sleep apnea. *Circulation* 2003; 107: 1822-1826.
4. Jessup M, Brozena S. Heart failure. *N Engl J Med* 2003; 348: 2007-2018.
5. Mansfield DR, Gollogly NC, Kaye DM, et al. A randomised controlled trial of continuous positive airway pressure treatment of obstructive sleep apnoea and heart failure. *Am J Respir Crit Care Med*. In press. ❑

Heart failure: how can we prevent the epidemic?

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TO THE EDITOR: We were surprised that Campbell's recent article on heart failure[1] made no mention of sleep apnoea. This is despite the fact that about 50% of heart failure patients have sleep apnoea (either central or obstructive) and that quite a large body of literature now supports a causative relationship between obstructive sleep apnoea and congestive heart failure, supported by recent authoritative reviews.[2-4] Canine studies have shown that, in the absence of any other variable, obstructive sleep apnoea results in left ventricular systolic and diastolic dysfunction.

Importantly, the impact of continuous positive airway pressure (CPAP) in acute cardiogenic pulmonary oedema and subacute pulmonary oedema (central sleep apnoea) and heart failure in the setting of obstructive sleep apnoea are not mentioned. Identification and treatment of sleep apnoea in people with heart failure is supported by a trial we have recently conducted in which significant improvements in quality of life and objective markers of cardiac function were seen in patients treated with nasal CPAP.[5]

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IN REPLY: I am grateful to Naughton and colleagues for their contribution to the debate about how we might prevent the epidemic of heart failure.

I agree that both obstructive and central sleep apnoea are frequently associated with heart failure and that treatment of sleep apnoea can improve cardiac function. However, as indicated by its title, my article focused on how we might *prevent* heart failure. Central sleep apnoea in heart failure is usually the *consequence* of the heart failure.[1] Heart failure may contribute to obstructive sleep apnoea as well,[2] and obstructive sleep apnoea may, in turn, be an important contributor to heart failure pathogenesis.

Epidemiological evidence links obesity with hypertension and obstructive sleep apnoea.[3] Significant sleep apnoea is present in about 40% of obese people, and about 70% of people with obstructive sleep apnoea are obese.[3] Obesity and obstructive sleep apnoea may each contribute to one another, and both may contribute to hypertension. The need to prevent obstructive sleep apnoea is an argument for more effective prevention of obesity. In addition to reducing its metabolic consequences, preventing obesity is likely to reduce the incidence of hypertension and obstructive sleep apnoea, and to thereby decrease the incidence of heart failure.

For people with obstructive sleep apnoea not caused by obesity, alternative strategies will be required to prevent and treat the sleep apnoea and thus prevent its consequences.

1. Bradley TD, Floras JS. Sleep apnea and heart failure. Part II: central sleep apnea. *Circulation* 2003; 107: 1822-1826.
2. Bradley TD, Floras JS. Sleep apnea and heart failure. Part I: obstructive sleep apnea. *Circulation* 2003; 107: 1671-1678.
3. Wolk R, Shamsuzzaman AS, Somers VK. Obesity, sleep apnea, and hypertension. *Hypertension* 2003; 42: 1067-1074. ❑

Pet owners and risk factors in cardiovascular disease

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TO THE EDITOR: We refer to the recent article by Parslow and Jorm[1] and the editorial by Headey[2] on the link between pet ownership and health outcomes. There is no evidence that pet ownership *per se* confers cardiovascular benefits. Indeed, the findings of the study were that pet owners were more likely to smoke, had a higher diastolic blood pressure and a higher body mass index than the non-pet owners. The editorial points out that, based on sociological studies, it is likely that pet ownership does have a positive effect on health, but the medical data demonstrating how this is achieved are lacking.[2]

Might there be other negative effects of pet ownership on health outcomes?

A patient under our care recently demonstrated the potential risks involved in pet companionship for elderly people. An 81-year-old woman who was living independently was admitted after a fall caused by tripping over her delightful Himalayan Persian cat.

Her presenting symptom was severe back pain exacerbated by weight bearing. However, no bony abnormality was identified. She had difficulty mobilising initially, and the outcome of her fall was significant morbidity with some loss of her previous mobility, even on discharge. She stayed in hospital for 12 days. A follow-up phone call revealed that the patient was still experiencing difficulty mobilising some 2 weeks after discharge.

This case raises the possibility that the risks may outweigh the benefits of pet ownership in elderly people who are already at risk of falls. A MEDLINE search, using the terms *elderly*, *trauma*, *cat*, *pet* and *fall* (and combinations of these), did not reveal any relevant literature. A recent case report highlighted other cardiovascular issues relating to cat ownership.[3] It described a patient who had recurrent episodes of syncope whenever her cat slept on the right side of her neck. The underlying mechanism was carotid sinus hypersensitivity. She required a single-lead ventricular pacemaker and for the cat to lie on her left side.

Anecdotal evidence from colleagues highlighted the danger of "dogs taking elderly patients for walks", resulting in rotator cuff injuries. Additionally, older patients with peripheral vascular disease and fragile skin have presented with non-healing ulcers from dog scratches.

Pet ownership for the purpose of modifying cardiovascular risk factors would seem to be unwise in this population. However, the benefits of companionship and the pleasure derived from pets may outweigh the risk of falls for many elderly patients. Should we be doing more "cat scans" or "pet scans" in older patients?

1. Parslow RA, Jorm AF. Pet owners and risk factors in cardiovascular disease: another look. *Med J Aust* 2003; 179: 466-468.
2. Headey B. Pet ownership: good for health? [editorial]. *Med J Aust* 2003; 179; 460-461.
3. Singh SM, Zia MI, Fowler RA. Cat naps: an elderly woman with recurrent syncope. *CMAJ* 2003; 169; 940. ❑

Michael McDonnell

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TO THE EDITOR: Having read the article by Parslow and Jorm,[1] I dashed to my surgery — praise the Lord, my diastolic blood pressure was 70 mmHg.

So, I raced home again and reassured our labradors that they would not have to be shot.

Let's leave elderly pet owners and their blood pressures alone. Pet ownership is all about companionship, friendship, trust, care for your friend — the really important things in life — not your diastolic blood pressure!

1. Parslow RA, Jorm AF. Pet owners and risk factors in cardiovascular disease: another look. *Med J Aust* 2003; 179: 466-468. ❑

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