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Tsunami lung: a necrotising pneumonia in survivors of the Asian tsunami

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TO THE EDITOR: The disastrous events of Boxing Day, 2004 left hundreds of thousands dead, injured or homeless across large parts of Asia. Many aid teams dispatched to affected areas are grappling with the aftermath of this catastrophe. Here, I present one of many clinical observations of what we encountered in the field. It is a clinical anecdote, but one worth sharing, as it may guide future teams in similar situations.

A 62-year-old woman was admitted to hospital with a history of vague ill-health for 12 months, and a subacute illness over the 4 weeks since immersion in the tsunami, with persistent cough, dyspnoea and weakness to the point of being largely bed-bound. She was cachectic, had a fever of 37.5°C and scattered crackles in both lower lung fields. Radiology facilities were not available and she was not producing sputum. She was treated empirically with antituberculous chemotherapy, as well as broad-spectrum antibiotics in the form of amoxycillin and ciprofloxacin orally, but her condition did not improve. X-ray facilities became available soon thereafter, and a chest x-ray showed changes more in keeping with a necrotising pneumonia than tuberculosis (Box [a]). Her treatment was changed to intravenous meropenem (1 g

every 8 hours) and her condition was slowly improving when we left.

During our 2-week posting in Banda Aceh, we saw about 6–10 patients at three hospitals presenting about a month after their immersion, with fluctuating fever; chronic, non-productive cough; and radiological evidence of bilateral, asymmetric, necrotising pneumonia with cavitation. Some patients developed empyemas and pneumothoraces (Box [b]). They failed to respond to broad-spectrum antibiotics including ampicillin/gentamicin/metronidazole and ticarcillin-clavulanate/cotrimoxazole. *Burkholderia pseudomallei* was cultured from the pleural fluid of two of these patients, and *Nocardia* sp. from the sputum of another. A notable feature of these patients was their subacute presentation weeks after immersion in the tsunami, the persistence of symptoms despite other broad spectrum antibiotic therapy, and the development of radiological and clinical manifestations of necrosis with pleural involvement.

Many of our patients described the area as being “black”. In view of the immersion in muddy water in a tropical environment, *B. pseudomallei* is likely to have been one of the causative organisms in many of these cases. However, it has not been possible to culture *B. pseudomallei* from all patients, and it is likely that their infections were polymicrobial given the circumstances of their injuries. A variety of bacterial organisms, as well as fungi, have been recognised in other such situations.^{1–3}

When we were there, Fakinah hospital provided one of the few laboratory services in Banda Aceh, and the availability of these facilities were limited as they were focused on public health surveillance. Thus, collection of specimens for culture was not routine. Furthermore, when we arrived

there were no nurses, no medical records and no medication or observation charts. While the situation improved rapidly during our stay, these limitations meant that recognition of emerging clinical patterns was important. Many of the antibiotics initially used for patients with immersion injuries were ineffective in this setting, and the use of carbapenems became our first-line, or early second-line, antibiotic in post-immersion respiratory infections in Banda Aceh.

- 1 Ender PT, Dolan MJ. Pneumonia associated with near-drowning. *Clin Infect Dis* 1997; 25: 896-907.
- 2 van Dam AP, Puijm MT, Harinck BI, et al. Pneumonia involving *Aspergillus* and *Rhizopus* spp. after a near-drowning incident with subsequent *Nocardia cyriacigeorgici* and *N. farcinica* coinfection as a late complication. *Eur J Clin Microbiol Infect Dis* 2005; 24: 61-64.
- 3 Chaney S, Gopalan R, Berggren RE. Pulmonary *Pseudallescheria boydii* infection with cutaneous zygomycosis after near drowning. *South Med J* 2004; 97: 683-687. □

Advances in childhood leukaemia

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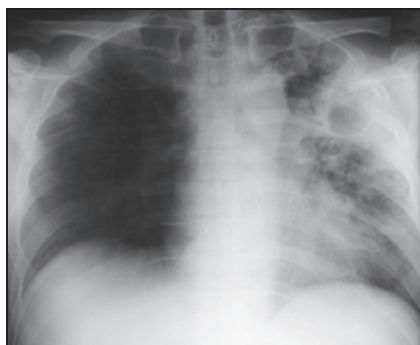
TO THE EDITOR: When discussing causes of childhood leukaemia, Ziegler et al stated, “Exposure to electromagnetic fields has been ruled out as playing any significant role”.¹ They cited one large study² in support of this statement, but overlooked two independent pooled analyses that showed the opposite. Greenland et al analysed 12 studies involving 2656 patients and 7084 controls,³ and Ahlbom et al analysed nine studies involving 3247 patients and 10 400 controls.⁴ Each analysis found an association with a doubling of risk of childhood leukaemia at levels of household exposure at and over 0.4 microtesla (4 milligauss). Confounders and sources of bias to explain these findings have been sought without success. Consequently, in 2002, the International Agency for Research on Cancer classified 50 and 60 Hz magnetic fields as a “possible carcinogen” (Group 2b)⁵ even though the mechanism of an effect is not clear.

The role of magnetic fields in childhood leukaemia cannot be “ruled out”, given the substantial epidemiological evidence, the international classification of magnetic fields as a possible carcinogen, and the subtlety of gene-environment interactions. Moreover, although exposures to magnetic fields are low within most households, there is opportunity to easily prevent or treat the uncom-

Chest x-rays of patients with subacute necrotising pneumonia



(a) Bilateral consolidation with scarring and early cavitation in the lower lung fields



(b) Bilateral necrotising pneumonia complicated by right pneumothorax

mon situations where household exposures exceed 0.4 microtesla by means of electrical engineering, household wiring and town planning.

- 1 Ziegler DS, Pozza LD, Waters KD, Marshall GM. Advances in childhood leukaemia: successful clinical-trials research leads to individualised therapy. *Med J Aust* 2005; 182: 78-81.
- 2 UK Childhood Cancer Study Investigators. Childhood cancer and residential proximity to power lines. *Br J Cancer* 2000; 83: 1573-1580.
- 3 Greenland S, Sheppard AR, Kaune WT, et al. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Childhood Leukemia-EMF Study Group. *Epidemiology* 2000; 11: 624-634.
- 4 Ahlbom A, Day N, Feychting M, et al. A pooled analysis of magnetic fields and childhood leukemia. *Br J Cancer* 2000; 83: 692-698.
- 5 International Agency for Research on Cancer. IARC Monographs Volume 80. Static and extremely low frequency electric and magnetic fields. Lyon: IARC, 2002. Available at: <http://monographs.iarc.fr/htdocs/monographs/vol80/80.html> (accessed Feb 2005).

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IN REPLY: Hocking states that electromagnetic fields cannot be ruled out as a cause of childhood leukaemia. However, several large studies have all failed to find any association between childhood exposure to electromagnetic radiation and leukaemia.¹⁻⁴ The two pooled meta-analyses Hocking refers to both found no increased incidence of leukaemia with exposure to electromagnetic fields of <0.4 microtesla.^{5,6} Although there was an increased risk of leukaemia with exposure to ≥0.4 microtesla, 99.2% of children with leukaemia had not received such a high level of exposure.⁵ In addition, both studies acknowledged the potential for selection bias. As such, for the overwhelming majority of children with leukaemia, exposure to electromagnetic fields does not play any significant causative role. Although we agree its effect cannot be ruled out for the remaining <1% of patients, it should not be given undue epidemiological weight.

- 1 Linet MS, Hatch EE, Kleinerman RA, et al. Residential exposure to magnetic fields and acute lymphoblastic leukemia in children. *N Engl J Med* 1997; 337: 1-7.
- 2 Dockerty JD, Elwood JM, Skegg DC, Herbison GP. Electromagnetic field exposures and childhood leukaemia in New Zealand. *Lancet* 1999; 354: 1967-1968.
- 3 McBride ML, Gallagher RP, Theirault G, et al. Power-frequency electric and magnetic fields and risk of childhood leukemia in Canada. *Am J Epidemiol* 1999; 149: 831-842.

- 4 UK Childhood Cancer Study Investigators. Childhood cancer and residential proximity to power lines. *Br J Cancer* 2000; 83: 1573-1580.
- 5 Ahlbom A, Day N, Feychting M, et al. A pooled analysis of magnetic fields and childhood leukemia. *Br J Cancer* 2000; 83: 692-698.
- 6 Greenland S, Sheppard AR, Kaune WT, et al. A pooled analysis of magnetic fields, wire codes, and childhood leukemia. Childhood Leukemia-EMF Study Group. *Epidemiology* 2000; 11: 624-634.

Warfarin reversal: consensus guidelines, on behalf of the Australasian Society of Thrombosis and Haemostasis

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TO THE EDITOR: The recent position statement by the Warfarin Reversal Consensus Group provides clear and concise guidelines for a number of clinical scenarios related to the use of warfarin.¹ Unfortunately, it makes the general statement about the periprocedural management of warfarin in patients with atrial fibrillation (AF), "clinical experience suggests that bridging therapy is not required" [page 496]. Clinicians caring for patients with large ischaemic stroke in these circumstances may beg to differ.

Although studies of bridging therapy in patients with AF in the periprocedural period are lacking, there are data which suggest that there is a considerably higher risk of thromboembolism during this period than would be expected by simply calculating the risk for several days off anticoagulation therapy.²⁻⁴ My own study of such patients undergoing endoscopy found a stroke risk of up to 3% in those at high risk.² Many of these strokes were severe. The prothrombotic periprocedural environment may be a factor here, although advanced age and vascular risk factors may also contribute. The outstanding risk factor, however, is a previous history of stroke,² and this is also a major risk factor for perioperative stroke in patients without AF.⁵ I would suggest careful, individualised assessment of all patients, and judicious bridging therapy where possible for patients with AF who have a past history of stroke.

- 1 Baker RI, Coughlin PB, Gallus AS, et al. Warfarin reversal: consensus guidelines, on behalf of the Australasian Society of Thrombosis and Haemostasis. *Med J Aust* 2004; 181: 492-497.
- 2 Blacker DJ, Wijedicks EF, McClelland RL. Stroke risk in anticoagulated patients with atrial fibrillation

undergoing endoscopy. *Neurology* 2003; 61: 964-968.

- 3 Dunn A, Turpie A. Perioperative management of patients receiving oral anticoagulants: a systematic review. *Arch Intern Med* 2003; 163: 901-908.
- 4 Kearon C, Hirsh J. Current concepts: management of anticoagulation before and after elective surgery. *N Engl J Med* 1997; 336: 1506-1511.
- 5 Blacker DJ, Flemming KD, Link MJ, Brown RD. The preoperative cerebrovascular consultation: common cerebrovascular questions before general or cardiac surgery. *Mayo Clin Proc* 2004; 79: 223-229.

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TO THE EDITOR: The recent position statement from the Warfarin Reversal Consensus Group provides a comprehensive, coherent and practical approach to warfarin reversal management.¹

In reviewing the position statement, and with particular reference to the paragraph about modifiers of warfarin response, we noted that the contribution of cytochrome P450 2C9 (CYP2C9) genotype to the response to warfarin was not addressed. There is debate in the current literature about the clinical utility of evaluating CYP 2C9 genotype in patients already taking or about to start warfarin therapy. Nonetheless, a significant body of evidence supports the contribution of CYP2C9 genetic variants as modifiers of response to warfarin therapy.

CYP2C9 is the enzyme principally involved in metabolising warfarin.² Several studies have identified the presence of single nucleotide polymorphisms in the CYP2C9 gene resulting in the expression of two allelic variants of CYP2C9 (CYP2C9*2 and CYP2C9*3) that are associated with reduced enzymatic activity, impaired metabolism of and increased sensitivity to standard warfarin doses.^{2,3} The allelic frequency of the mutant genotypes is in excess of 21% in the white population² (they occur at reduced frequencies in African American populations and are rare in Asian populations⁴). The presence of allelic variants (CYP2C9*2 and CYP2C9*3) with reduced enzymatic activity is closely correlated with increased bleeding complications.^{2,3} Thus, there is potential for a considerable clinical impact given the large number of patients taking warfarin.

We have identified an allelic frequency of CYP2C9*2 and CYP2C9*3 genotypes in an Australian population of patients attending an anticoagulant clinic comparable to that

reported in the literature.^{2,3} We also identified international normalised ratios in excess of the target range in patients with the CYP2C9*2 or CYP2C9*3 genotype undergoing induction warfarin therapy with standard dosing regimens, relative to those who did not have these genotypes (personal, unpublished data, presented as: *Cytochrome P450 CYP2C9 genotyping and warfarin induction therapy*, presented at the 2004 Annual Scientific Meeting of the Haematology Society of Australia and New Zealand [Oct 17–20, Melbourne, Australia]).

Recent reports suggest that CYP2C9 genotyping before inducing warfarin therapy may avert bleeding complications.⁵ However, CYP2C9 genotyping is currently only available within research institutes and larger corporations with research and development facilities and does not attract a Medicare rebate. While simple and inexpensive, genetic CYP2C9 screening has yet to be proven cost effective. However, genotyping may be of benefit in averting over-anticoagulation in certain clinical scenarios. These include commencing warfarin therapy in “high risk” elderly patients; those in whom low-dose, long-term, low-testing-frequency warfarin regimens are being contemplated; and in other “high risk” patients, such as those with conditions affecting warfarin metabolism, including liver disease, and in those taking medications known to interact with the hepatic metabolism of warfarin.

1 Baker RI, Coughlin PB, Gallus AS, et al. Warfarin reversal: consensus guidelines, on behalf of the Australasian Society of Thrombosis and Haemostasis. *Med J Aust* 2004; 181: 492-497.

2 Freeman BD, Zehnauer BA, McGrath S, et al. Cytochrome P450 polymorphisms are associated with reduced warfarin dose. *Surgery* 2000; 128: 281-285.

3 Aithal GP, Day CP, Kesteven PJ, et al. Association of polymorphisms in the cytochrome P450 CYP2C9 with warfarin dose requirement and risk of bleeding complications. *Lancet* 1999; 353: 717-719.

4 Sullivan-Klose TH, Ghanayem BI, Bell DA, Zhang ZY. The role of the CYP2C9-Leu359 allelic variant in the tolbutamide polymorphism. *Pharmacogenetics* 1996; 6: 341-349.

5 You JH, Chan FW, Wong RS, et al. The potential clinical and economic outcomes of pharmacogenetics-orientated management of warfarin therapy — a decision analysis. *Thromb Haemost* 2004; 92: 590-597. □

Serge Lubicz

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TO THE EDITOR: The article by Baker et al was a timely review of managing anticoagulation therapy and balancing the risks of

thrombosis and bleeding.¹ However, in managing anticoagulation therapy before non-cardiac surgery in patients with mechanical cardiac valve prostheses, the suggested 5-day cessation of warfarin therapy, with only subcutaneous heparin cover, is not appropriate. I have had three patients with mechanical bileaflet mitral prostheses develop valve thrombosis while under this protocol, two with a fatal outcome. I have also had one patient with a mechanical bileaflet aortic valve develop a popliteal arterial embolus requiring thrombectomy, despite being treated according to the protocol.

The consequences of valve thrombosis and thromboembolism far outweigh the lesser complications of increased bruising or bleeding associated with non-cardiac surgery. To avoid the potentially devastating complications of valve thromboembolism associated with the routine cessation of warfarin therapy 5 days before surgery, warfarin ought to be continued to maintain an INR (international normalised ratio) of around 2.0, supplemented with subcutaneous heparin. Alternatively, full intravenous heparinisation can be used while ceasing warfarin treatment, and continued postoperatively until the INR is restored to the therapeutic level. Warfarin should never be reversed with vitamin K, except in cases of life-threatening haemorrhage.

Apropos of the therapeutic INR ranges generally recommended for mechanical cardiac valve replacements, the current generation of prostheses does not require the anticoagulation intensity of the older style prostheses.^{2,3} Lower intensity anticoagulation is sufficient to prevent thromboembolism at decreased risk of haemorrhagic complications.⁴ My personal practice for patients with bileaflet mechanical prostheses is to maintain an INR of 2.0–2.5 for aortic valves, and 2.5–3.0 for mitral valves. The higher intensity for mitral prostheses relates to potential increased thrombogenicity because of lower leaflet opening pressures, as well as the common association of left atrial dilatation and atrial fibrillation.

1 Baker RI, Coughlin PB, Gallus AS, et al. Warfarin reversal: consensus guidelines, on behalf of the Australasian Society of Thrombosis and Haemostasis. *Med J Aust* 2004; 181: 492-497.

2 Andersen PV, Aagaard J. Low-dose warfarin in patients with carbomedics heart valve prostheses. *Asian Cardiovasc Thorac Ann* 2000; 8: 11-14.

3 Van Nooten GJ, Van Belleghem Y, Caes F et al. Lower intensity anticoagulation for mechanical heart valves: a new concept with the ATS bileaflet aortic valve. *J Heart Valve Dis* 2003; 12: 495-502.

4 Acar J, Jung B, Boissel JP, et al. Multicentre randomised comparison of low-dose versus standard-dose anticoagulation in patients with mechanical heart valves. *Circulation* 1996; 94: 2107-2116. □

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TO THE EDITOR: A middle-aged woman with atrial fibrillation had her warfarin therapy stopped for 2 days before dental extraction. She had a catastrophic stroke and is now a plaintiff. I was asked if her medical management accorded with common practice.

At the October 2004 Annual Conference of the Royal Australian College of General Practitioners, I conducted a straw poll of 20 experienced GPs, of whom 18 said they would stop warfarin for between 2 and 4 days before a dental extraction. Some of these GPs regarded a dental extraction as elective surgery and pointed me to authoritative (but slightly ambiguous) sources to back up their view.^{1,2} However, a review of the medical and dental literature shows that this is an example of common practice lagging behind clinical evidence.

The first controlled trial of dental extraction in patients on warfarin therapy was conducted in 1983.³ It showed that it was not necessary to cease warfarin prophylaxis for patients whose international normalised ratio (INR) was within the normal therapeutic range. Since then, two major literature reviews have confirmed these conclusions.^{4,5} A recent Australian review on warfarin reversal expresses a similar point of view.⁶ The incidence of a serious embolic complication from stopping therapy with warfarin is 1%, and this is three times more likely to occur than bleeding complications in patients whose warfarin therapy was continued.⁴ Furthermore, a stroke is a catastrophic event, while a bleeding tooth socket is simply messy and usually easily controlled.

An authoritative review and position statement on warfarin therapy and dental procedures from the Australasian Society of Thrombosis and Haemostasis may be the catalyst required to align common practice with clinical evidence.

1 Australian Medicines Handbook. Adelaide: Hyde Park Press, 2004: 310.

2 Hankey GJ. Non-valvular atrial fibrillation and stroke prevention. National Blood Pressure Advisory Committee of the National Heart Foundation. *Med J Aust* 2001; 174: 234-239.

3 Bailey BM, Fordyce AM. Complications of dental extraction in patients receiving warfarin anti-coagulant therapy. A controlled trial. *Br Dent J* 1983; 155: 308-310.

4 Wahl MJ. Myths of dental surgery in patients receiving anticoagulant therapy [review]. *J Am Dent Assoc* 2000; 131: 77-81.

5 Carter G, Goss AN, Lloyd J, Tocchetti R. Current concepts in the management of dental extractions

for patients taking warfarin. *Aust Dent J* 2003; 48: 89-96.

- 6 Baker RI, Coughlin PB, Gallus AS, et al. Warfarin reversal: consensus guidelines on behalf of the Australasian Society of Thrombosis and Haemostasis. *Med J Aust* 2004; 181: 492-497. □

Hatem H Salem,* on behalf of the Warfarin Reversal Consensus group

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IN REPLY: We thank Blacker for his constructive and helpful comments. Our recommendations on bridging therapy in patients with atrial fibrillation were for patients with chronic atrial fibrillation who had not previously had a thromboembolic event.¹ We do agree with Blacker that extreme care needs to be exercised in patients with atrial fibrillation and a previous thromboembolism. These patients should be managed along the same lines as patients who are at relatively high risk of recurrent thromboembolism. We also wish to emphasise that it is extremely important to assess each individual patient carefully, and to use the con-

sensus guidelines as guiding principles, and not apply them blindly.

Dear and his colleagues correctly point out that there are several published studies that have confirmed increased warfarin sensitivity in allelic variants of the cytochrome P450 2C9 (CYP2C9) enzyme. Polymorphisms associated with reduced enzymatic activity have been reported to be associated with increased warfarin sensitivity. They suggest that determining the genotype of individuals before commencing warfarin therapy may be of benefit in reducing the incidence of over-anticoagulation in a select group of patients. We do not believe that this approach is currently practical or possible. From a practical point of view we recognise several reasons why patients become over-anticoagulated when treated with warfarin. In our article we discussed several important modifiers that contribute to an individual's sensitivity to warfarin. While we agree that polymorphisms of the CYP2C9 gene on its own have been linked with increased sensitivity to warfarin, we are not aware of any studies showing a synergistic interaction of the polymorphism with other clinically recognised causes of increased warfarin sensi-

tivity. Furthermore, we are not aware of any properly conducted studies that have attempted to address the clinical or economic viability of screening for CYP2C9 polymorphisms in patients for whom warfarin therapy is planned. Finally, the time required to obtain the results of this investigation would preclude its application in the routine management of patients who require warfarin therapy.

The letter by Lubicz highlights the difficulties encountered in bridging anticoagulant therapy in patients with prosthetic valves. As pointed out in our article, the management of these patients is controversial and mostly anecdotal.¹ We believe that the recommendations in our article are useful for most patients, but would like to emphasise the need to consult with the relevant experts in order to avoid bleeding or thrombosis. We would not recommend routine full therapeutic anticoagulation therapy with heparin immediately after surgery, or the combined use of warfarin at any international normalised ratio (INR) with subcutaneous heparin before surgery. Such approaches are more likely to cause confusion and predispose the patients to either

bleeding or the risk of thrombosis. Patients with prosthetic valves require careful handling, and involving experts in their management is critical.

Kamien's comments are important and illustrate the difficulties in changing entrenched practices. We hope that our recommendations will go some way to improving the way we manage patients on warfarin therapy who are about to undergo surgery.

1 Baker RI, Coughlin PB, Gallus AS, et al. Warfarin reversal: consensus guidelines, on behalf of the Australasian Society of Thrombosis and Haemostasis. *Med J Aust* 2004; 181: 492-497. □

X-ray machine assaults anaesthetist

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TO THE EDITOR: Incidents involving assaults on staff by medical equipment are

uncommon, but have been reported in this Journal before.¹ We report another "attack", involving an x-ray machine and an anaesthetist.

A woman was scheduled for endoscopic retrograde cholangiopancreatography in the radiology suite. During induction of general anaesthesia, the patient's foot moved against an x-ray table control knob (Box). This triggered slow, downward movement of an x-ray "C-arm", which was positioned above the head of the unsuspecting anaesthetist. Tracheal intubation was rudely interrupted when the C-arm met the anaesthetist's head and pushed it towards the patient's face. However, the radiographer in attendance quickly reversed the movement just before the anaesthetist and patient collided.

The radiology suite is often regarded as an unfriendly environment for anaesthetists.² This incident reminds us that, in some cases, it may be frankly hostile!

1 Riley RH. Anchoring an anaesthetist. *Med J Aust* 2002; 177: 687-688.

2 Alspach D, Falleroni M. Monitoring patients during procedures conducted outside the operating room. *Int Anesthesiol Clin* 200; 42: 95-111. □

A patient's foot activates a control knob on an x-ray table

