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## Necrotising pneumonia due to Panton–Valentine leukocidin-positive methicillin-sensitive *Staphylococcus aureus*

Ada S-Z Cheung, Craig A Aboltins, John R Daffy and Peter A Stanley

**TO THE EDITOR:** Panton–Valentine leukocidin (PVL) is a potent necrotising toxin, which, although produced by less than 5% of all *Staphylococcus aureus* strains, is strongly associated with pathogenic isolates that cause recurrent furunculosis and severe necrotising pneumonia.<sup>1</sup> The virulence of PVL-positive community-associated methicillin-resistant *S. aureus* (CA-MRSA) causing necrotising pneumonia was recently highlighted in the Journal.<sup>2</sup> Conversely, PVL produced by methicillin-sensitive *S. aureus* (MSSA) is uncommon.<sup>1</sup> Here, we describe a case of fulminant necrotising pneumonia caused by PVL-positive MSSA, which, to our knowledge, is the first reported case in Australia.

A previously well 33-year-old man presented to the emergency department with a 48-hour history of pleuritic chest pain, fever and productive cough. On presentation, the patient was hypotensive, and in acute renal failure and hypoxemic respiratory failure (type I). Chest x-ray showed bilateral widespread air space consolidation.

Despite treatment with intravenous fluid resuscitation and early broad-spectrum antibiotics (ceftriaxone, azithromycin, vancomycin and co-trimoxazole), the patient's condition rapidly deteriorated, requiring intubation and inotropic support.

Multiple blood and sputum cultures isolated MSSA. Bronchoscopy revealed widespread airway haemorrhage. A transoesophageal echocardiogram excluded endocarditis.

Progressive leukopenia developed. Septic shock and respiratory failure worsened, despite treatment with flucloxacillin as well as maximal inotropic and ventilatory support. The patient died 72 hours after presentation from fulminant pneumonia. Polymerase chain reaction testing subsequently identified the PVL gene in the isolated MSSA.

Rising rates of CA-MRSA causing recurrent furunculosis and severe necrotising pneumonia have been reported worldwide.<sup>3</sup> Necrotising pneumonia often affects children and young adults,<sup>1</sup> and, despite current treatments, mortality rates are over 50%.<sup>4</sup> Our patient exhibited two major fac-

tors predictive of increased lethality: leukopenia and airway bleeding.<sup>4</sup>

PVL has been well described in CA-MRSA; it is present in at least 96% of the two predominant strains in south-east Australia.<sup>3</sup> There is some evidence that PVL is the major pathogenic factor of CA-MRSA, although this remains controversial.<sup>5</sup> The precise pathogenesis of PVL has not yet been discovered; however, a severe inflammatory response secondary to PVL's cytolytic effects on polymorphonuclear leukocytes, as well as the induction of other bacterial virulence factors, are possibilities.<sup>1</sup> Therapies directed against the PVL toxin, including antibiotics such as clindamycin that target the bacterial ribosome, or intravenous immunoglobulin, have been suggested but have little supportive data.<sup>2,3,6,7</sup>

In contrast to CA-MRSA, the PVL gene is found much less frequently in MSSA, being present in only 2% of isolates in one French study.<sup>1</sup> Specific Australian prevalence data are lacking, but PVL-positive MSSA isolates have similar potential to cause severe invasive disease.<sup>1,6</sup>

Most cases of severe necrotising staphylococcal pneumonia and recurrent furunculosis are caused by CA-MRSA, and empirical therapy for these conditions should cover this organism. This case involving MSSA highlights the role that PVL may play in the pathogenicity of these conditions, and shows that the development of novel therapeutics directed at PVL may be of value.

Ada S-Z Cheung, Medical Registrar  
Craig A Aboltins, Infectious Diseases Physician  
John R Daffy, Infectious Diseases Physician  
Peter A Stanley, Infectious Diseases Physician  
Department of Infectious Diseases, Northern Hospital, Melbourne, VIC.  
ada.cheung@austin.org.au

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## NHMRC grant applications: a comparison of "track record" scores allocated by grant assessors with bibliometric analysis of publications

Michael J Davies

**TO THE EDITOR:** Predicting research quality on the basis of past research publications is clearly imprecise, as noted by Nicol et al in their recent article on National Health and Medical Research Council (NHMRC) grant applications.<sup>1</sup> They note that assessor ratings of applicants' "track records" correspond poorly with the bibliometric data for authors, and that there is vast variability between discipline panels. For immunology, the correlation between track record scores and journal impact or citations was high, at over 0.7. For public health, the correlation was actually negative.

The authors consider some possible reasons for the wide discrepancies, such as poor coverage of public health publications in the journals captured by Institute for Scientific Information citation indexes. Nevertheless, they are at a loss to explain why the variation is quite so great, and conclude by suggesting that the time is right for an automated approach to assessing quality.

We need to consider the implications of this suggestion carefully. Track record within NHMRC project grants is assessed relative to opportunity, with regard to factors such as legitimate career interruptions, administrative and teaching load, and typical publication rates for the field in question. In the fellowship or program grants schemes, there appears to be less emphasis on relativity, which may explain some of the closer correspondence between actual and expected citation rates.

We need to be clear that the use of an "automated" system that uses surrogate measures of research quality will disadvantage individuals who experience a period of illness, take maternity leave, change their research area, or carry a period of heavy administrative or teaching load, as well as those who publish books, book chapters or

government publications. It will also disadvantage teams in which feasibility requires fieldwork collaborators whose applied work does not readily translate into peer-reviewed journal publications. For instance, much public health research is based in the community or takes advantage of data collections in the public health system. Collaborators working in this context often have relatively limited opportunities for peer-reviewed publication. Nevertheless, their active collaboration is often critical for achieving a feasible research plan. Also disadvantaged would be teams with a new or junior investigator, particularly if the new team member was the first named investigator.

On the other hand, a move to an automated system of quality assessment would further advantage grant applicants who work in research-dedicated institutes, those engaged in basic research, and those who do not require external collaboration.

Given these reservations, I suggest further investigation, by discipline, of what makes a “good” track record, before recommending a single assessment formula.

**Competing interests:** I hold an NHMRC Fellowship; am currently a Chief Investigator on NHMRC project, strategic, and program grants; and am a grant reviewer for the NHMRC. I also hold two postgraduate qualifications in public health.

**Michael J Davies**, Senior Research Fellow  
Department of Obstetrics and Gynaecology,  
University of Adelaide, Adelaide, SA.  
michael.davies@adelaide.edu.au

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## Tako-tsubo cardiomyopathy: how stress can mimic acute coronary occlusion

Laven Padayachee

**TO THE EDITOR:** Abdulla and Ward's excellent article on tako-tsubo cardiomyopathy (TTC)<sup>1</sup> raises two important issues.

The first issue is the diagnostic dilemma faced by emergency physicians and cardiologists in differentiating TTC from ST-elevation myocardial infarction (STEMI) in centres that lack coronary angiogram capabilities.

In patients presenting with chest pain and ST elevation on electrocardiography, the diagnosis of TTC might be suspected on recognition of risk factors and the common psychological, physical and emotional stressors that precipitate TTC.<sup>1</sup> Supporting evi-

dence can be obtained by demonstration of basal hyperkinesis and apical or midventricular hypokinesis on transthoracic echocardiography. This modality is now available in many centres without coronary angiography.

However, if the diagnosis is incorrectly made as STEMI rather than TTC, the patient runs the risk of unnecessary thrombolysis. Alternatively, after risk-benefit analysis, the clinicians may transfer the patient to a facility with coronary angiography to confirm TTC.

The second issue is the therapeutic dilemma facing intensivists treating TTC-related shock with adrenergic inotropes. Although cardiogenic shock in TTC is uncommon, it can still occur (4.2%).<sup>2</sup> As increased endogenous catecholamines are thought to be central to the pathophysiology of TTC,<sup>3</sup> treating shock with inotropes puts the clinician in a quandary.

Agents such as adrenaline, dobutamine, dopamine, milrinone and noradrenaline increase cyclic AMP within the myocardial cell, and are commonly used to restore blood pressure and cardiac output. However, in TTC, inotropes may theoretically delay resolution of the apical ballooning. A recent echocardiographic study showed no improvement in apical and midventricular akinesis with the use of low-dose dobutamine.<sup>4</sup>

Levosimendan is a calcium sensitiser that has been used successfully to stabilise shock secondary to TTC (with and without use of an intra-aortic balloon pump).<sup>5</sup> Levosimendan is non-adrenergic and allows earlier introduction of  $\beta$ -blockers than would be possible with adrenergic inotropes.

I agree that prospective trials are needed to guide management in this intriguing condition.

**Laven Padayachee**, Staff Intensivist  
Intensive Care, Epworth Hospital, Melbourne, VIC.  
lavenp@epworth.org.au

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Michael R Ward

**IN REPLY:** I thank Padayachee for his interest in our review.<sup>1</sup> We read Padayachee's published case series of the use of levosimendan to help recovery of left ventricular dysfunction in tako-tsubo cardiomyopathy (TTC)<sup>2</sup> after our review was published.

I agree that levosimendan is the inotropic agent of choice in this situation, and this approach has been used successfully in my hospital on two occasions. This is, in fact, what we meant by “In our experience,  $\beta$ -blockade in conjunction with non-adrenergic inotropes can prevent this vicious cycle and allow the ventricle to recover (unpublished data)”.

As Padayachee rightly points out, definitive evidence on this point would require a proper randomised trial, which would be very difficult to organise, given the low incidence of tako-tsubo cardiomyopathy and the small fraction of patients who develop cardiogenic shock requiring inotropic support. I think that it is probably better to simply state that levosimendan therapy works and makes scientific sense, so “just do it”.

However, Padayachee also surmises that, if the patient has a typical wall-motion abnormality and a typical history, it might be possible to avoid unnecessary thrombolytic therapy in cases of TTC. Unfortunately, my understanding is that this is not the case. Occlusion of the left anterior descending artery (LAD) may result in the classic TTC wall-motion abnormality if the LAD extends far beyond the apex (usually with a non-dominant right coronary). As myocardial infarction can be precipitated by stressful events, and the evolution of electrocardiographic changes in TTC is similar to that seen with an anterior infarct after thrombolysis, there is still no clear way to discriminate between the two diagnoses apart from immediate coronary angiography. Whether computed tomography (CT) angiography can accurately discriminate remains to be seen — this might be useful in centres that have CT but not a cardiac catheterisation laboratory. Until then, I believe it is probably less harmful to give TTC patients thrombolysis than to withhold thrombolysis from patients with large anterior infarcts.

Lastly, I invite Padayachee and other interested clinicians who frequently manage these patients to participate in an ongoing study of genetic predisposition to TTC for which we are currently enrolling participants.

**Michael R Ward**, Senior Staff Specialist and Senior Lecturer

Department of Cardiology, Royal North Shore Hospital and University of Sydney, Sydney, NSW.

mrward@nscchahs.health.nsw.gov.au

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## Our hearts and minds — what would it take to become the healthiest country in the world?

**Bret Hart**

**TO THE EDITOR:** It is a worthy aspiration for Australia to become the world's healthiest country, but it will take revolutionary leadership to prevent and manage the effects of obesity that will reverse the previous gains in reducing heart disease.<sup>1</sup> In addition, we have to overcome the adverse impact on the health of young people caused by fundamental changes in Australia, highlighted by Eckersley.<sup>2</sup> He also identifies medical practitioners as a potential obstacle in that we are overfocused, with government approval, "on an individual, biomedical, disease-centred approach to health at the expense of a more social, preventative model". He also calls for an increase from the current investment in prevention and public health programs, 1% of health expenditure — but that will only occur if his more radical suggestion is adopted: that governments change their focus from *wealth* to *health* creation.

It was Japan that embraced this concept, with a health creation policy developed in 1978. It led to a law ensuring that at least 5% of their compulsory health insurance expenditure is allocated to preventive activities. If we are going to achieve Ring and O'Brien's vision, we are going to have to do more than adopt Japan's healthy diet.

Other keys to their success are: good antenatal care; reinforcement of high breastfeeding rates by provision of small incentive payments; routine home visits to women during pregnancy and during the postpartum period by maternal and child health care workers; and all parents having their own maternal child health record.

While these and other measures have probably contributed to Japan having the lowest infant mortality in the world, these interventions are also likely to have influenced their longevity by preventing the Barker hypothesis from being applied. This hypothesis, or developmental origins theory, was derived from observations of infants who are small at birth being at higher risk of increased blood pressure and other adverse cardiovascular endpoints later in life.<sup>3</sup>

It is interventions during the early years that have evidence of high returns on investment — whereas attempts to influence adult behaviour are difficult, and can fail.<sup>4,5</sup>

**Bret Hart**, Fellow

Australasian Faculty of Public Health Medicine, Sydney, NSW.

drbret@arach.net.au

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**Ian T Ring and John F O'Brien**

**IN REPLY:** Our paper demonstrates the considerable potential for improving Australia's already competitive international mortality ranking by focusing on several selected conditions and inequalities in their distribution among Australians.<sup>1</sup> Hart recognises the aspirational nature of the paper and proposes several challenges and opportunities to improve the health of the mothers, babies and young children of Australia.

We agree. As shown by our evidence, Australia's performance on mortality in infancy and early childhood is less than stellar. We acknowledge that our ranking on some childhood risk factors, such as obesity (which can confer lifelong health disadvantage and may affect future mortality), may well be similar or even worse. A critique of these was beyond the scope of our paper, as we confined our analysis to measures of past mortality.

There is ample evidence of effective interventions for infants, children, adolescents, adults and older people, and for various

population groups. The interventions include preventive or clinical services — as the Journal's own repository of guidelines shows.<sup>2</sup> We contend that rather than being alternatives, childhood and adulthood interventions are complementary (as are biomedical and social interventions), and we have to advance simultaneously on many fronts.

Australia has accelerated to be among the world's leaders on mortality and life expectancy, but, as Hart presages, this will not remain the case merely through a continuation of current trends. It may take a revolution, but we can at least be clear about how we compare in these areas and what we need to achieve.

**Ian T Ring**, Professorial Fellow<sup>1</sup>

**John F O'Brien**, Manager, Information Use<sup>2</sup>

1 Centre for Health Service Development, University of Wollongong, Wollongong, NSW.

2 Statistical and Library Services Centre, Queensland Health, Brisbane, QLD.

john\_o'brien@health.qld.gov.au

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## Medical staff and the hospital

**Derek H Meyers**

**TO THE EDITOR:** Van Der Weyden recently drew attention to the consequences of (mal)administration and (mis)management in public hospitals.<sup>1</sup> In 1975, the *British Medical Journal* published my article beginning:

A new disease has arisen in this part of the world. It attacks not the human body but the bodies of institutions, is a low-grade malignancy, may have effects varying from irritant to crippling, and might possibly even be fatal. The name of this disease is "administration" — with inverted commas to distinguish it from administration of benevolent type.<sup>2</sup>

In response, I received a letter from Birmingham saying, "Many congratulations on your article ... It has been received with great enthusiasm here". Similar responses came from the United States and Sri Lanka. An Australian surgeons' news magazine reprinted my article in full<sup>3</sup> — a real compliment for a physician. From North Wales, a peer of the realm wrote to me, awarding me

an "alpha plus". The new disease, it seems, was widespread.

So, what is new? Thirty-two years on, it appears that "administration" has become an even greater curse for the people who treat patients.

What can be done? Grumbling over a cup of coffee in the staff common room (if there is one) will achieve nothing. I believe clinicians of all types must get together, become active in hospital and general politics, and make their voices heard. All doctors, even those of socialist leaning, should join the Australian Medical Association, the most effective and widely representative body able to work for the good of patients and the profession.

Doctors should also take note of the findings of the Davies Inquiry into Queensland hospitals in 2005, which was prompted by the case of Bundaberg surgeon Jayant Patel. In the conclusion of his report, Commissioner Davies stated, "The view, which seems to be that of Queensland Health, that substantial adverse publicity is as serious a consequence as multiple deaths is shocking".<sup>4</sup> Among the five deficiencies contributing to the "unfortunate situations" he examined was "a culture of concealment by Government, Queensland Health administrators, and hospital administrators".<sup>4</sup> Davies was less concerned with doctors such as Patel, who took on work beyond their competence, than with administrators (including doctors) who focused on providing a service of sorts, without adequate regard for safety, and in the process failed to check the credentials of overseas-trained practitioners, allowed them to practise, and then concealed their adverse results.

It is not surprising that clinicians — who are more interested in good, safe practice than in budgets, which now seem to be the major concern in health care delivery — have been pushed out of the management of hospitals.

Derek H Meyers, Retired Physician  
Brisbane, QLD.  
d&rmeyers@acenet.net.au

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