LEADERSHIP AND MEDICAL TRIBALISM

The recent Royal College of Physicians (RCP) report examining professionalism, identified leadership as an essential prerequisite for our profession in the new millennium. Leadership was also featured in the latest RACP News (magazine of the Royal Australasian College of Physicians), which highlighted leaders among the College's Fellows: a state governor, two vice-chancellors of leading universities, 10 deans of medical schools, and the last three chief medical officers of the Australian Government. This is an impressive line-up, but is reaching the pinnacles of the establishment, academia or the bureaucracy synonymous with leadership?

The Concise Oxford Dictionary defines a leader as “n. 1 a. a person or thing that leads. b. a person followed by others.” But, as noted by a US academic, there is another dimension: “... leadership must be intimately connected to the process of change. The leader expresses not what the group is but what it might be.”

And yet, doctors are not followers, and are wary of change. New medical bodies appear regularly, as John Green, former chief executive of the Royal Society of Medicine and observer of doctors, opines: “There is no kingdom too small for a doctor to be king of.” Medical tribalism appears to be endemic, and is not without its consequences.

Sir Donald Irvine, past president of the United Kingdom General Medical Council, notes: “Tribalism has a profound impact on the profession — we are a dysfunctional profession at that level right through the system, and it is hurting us.” In fact, Dame Janet Smith, Chairman of the Shipman Inquiry, echoes such sentiments: “Tribalism causes doctors as a group to protect themselves, rather than acting collectively.”

The RCP report strongly recommends a “common forum” that speaks “with a unified voice”. With more than 30 medical tribes making up Australian medicine, is this imperative possible?

Martin B Van Der Weyden
Decline in meningitis admissions in young children: vaccines make a difference

Hannah C Moore and Deborah Lehmann

TO THE EDITOR: Meningitis is one of the most serious infections in young children. The annual incidence of Haemophilus influenzae type b (Hib) meningitis between 1984 and 1988 was 150 per 100,000 population in Aboriginal children and 27 per 100,000 in non-Aboriginal children younger than 5 years.1 A conjugate Hib vaccination program was introduced in Western Australia in January 1993, before a nationwide program commenced in July 1993. Subsequent marked declines in incidence of Hib meningitis have been reported.2-4 However, there are no declines in incidence of Hib meningitis in young children.2-4

The WA Data Linkage System (WADLS) encompasses statewide population-based record linkage of the statutory birth and death registries, midwives’ notification system, and hospital morbidity database,5 and is one of few such resources worldwide. As part of a larger study to determine the burden of infection in a cohort of births between 1990 and 2000 using the WADLS, we investigated hospitalisation for all-cause meningitis (International classification of diseases, 9th revision, diagnosis codes 003.21, 036.0, 047, 049.0, 054.72, 320-322) in 17,296 Aboriginal and 252,775 non-Aboriginal children younger than five years of age in Western Australia.7-8

In Aboriginal infants (<12 months), the meningitis rate fell by 41% between 1992 and 1993–1994 and by a further 54% in 1995–1996, and has remained stable since (Box). In Aboriginal children aged 12–23 months, rates declined by 44% between 1993–1994 and 1995–1996 and again by 50% in 1997–1998, and no meningitis admissions were reported in 1999–2000.

In non-Aboriginal infants, meningitis rates declined by 36%, from 1.8 per 1000 child-years in 1992 to 1.2 per 1000 child-years in 1993–1994, with a further 50% decline in 1997–1998, since when rates have remained stable. Rates declined by 57% between 1992 and 1993–1994 in non-Aboriginal children aged 12–23 months, declined a further 47% in 1995–1996, and have since remained stable at about 0.2 per 1000 child-years.

With the decline in meningitis admissions, the disparity between Aboriginal and non-Aboriginal children has narrowed: the relative rate (RR) of Aboriginal to non-Aboriginal meningitis admissions fell from 7.3 in 1992 to 5.0 in 1999–2000 in infants, while in children aged 12–23 months, the RR was >7.0 in 1992–1993, fell to 3.0 in 1997–1998, and was indefinite in 1999–2000 (Box). In the absence of other relevant interventions, we attribute declines in meningitis admissions to the introduction of Hib vaccine. This is supported by other studies showing a reduction in Hib meningitis following vaccination.2-4

Retrospective data provide an opportunity to assess overall trends in admissions. Future linkages with immunisation and laboratory data will allow us to investigate pathogen-specific admissions and evaluate vaccination programs.

Our findings show that substantial improvements can be achieved given government commitment to implement appropriate preventive measures. Adequate funding and continued commitment is needed to ensure these measures are accessible to all WA children.

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Increase in caesarean section rates among low-risk women in Queensland, 1990–2004

Trisha C Johnston and Michael D Coory

TO THE EDITOR: The current rate of caesarean sections in Australia (29% of all live births) is higher than the rate in other similarly affluent countries.1 In addition, the rate is continuing to increase; for example, it was less than 20% in 1993.2

Some commentators have suggested that this increase is partly a result of caesarean sections undertaken for non-medical reasons, such as patient demand.2,3 We examined trends in the rates of caesarean section for low-risk women using population-based perinatal data for Queensland over 15 years between 1990 and 2004. Our aim was to assess whether caesarean sections were becoming more common among women with no obvious medical indication for the procedure.

The increase in caesarean sections among low-risk women was most dramatic in the private health care sector, where the percentage increased from 10% to 19% (Box). This represents an average annual increase of 6.6% (95% CI, 4.3%–5.0%). In the public health care sector, the increase was less — from 6% to 8% — an average annual increase of 2.4% (95% CI, 2.0%–2.7%). The increase in the private sector in Queensland was similar to the increase reported in the United States.4

The appropriate use of caesarean section, as for any medical intervention, should be based on evidence about the benefits and harm, with doctors, women and their families choosing a method of delivery after considering balanced information on poten-
nERAL outcomes of each method. There is continuing debate about the feasibility of randomised trials to clarify the benefits and harm of caesarean deliveries among low-risk women.\(^1\) Opposition to such trials is based mainly on ethical concerns about inflicting a surgical procedure on healthy women based only on randomisation.

Non-randomised studies have compared outcomes of caesarean section versus vaginal delivery. However, their results are inconclusive because of the difficulty of distinguishing the effects of factors that influence the selection of delivery method from the effects of the delivery method itself (confounding by indication).\(^3,5\)

In the absence of randomised trials, non-randomised studies that remove this potential bias by restricting the sample to women who remain at low risk throughout the pregnancy and delivery, according to clearly defined criteria, may provide useful information. They would need to assess both short-term and long-term outcomes. Until such better evidence is available, it is impossible to judge whether or not the current increase in caesarean section rates among low-risk women is desirable.

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Nephrotic-range proteinuria in the obese patient
Andy K H Lim

**TO THE EDITOR:** The incidence of obesity is rising, and physicians are likely to face the problem of obesity-related glomerulopathy (ORG) recently illustrated by Tran.\(^1\) But how can the clinician distinguish ORG from primary (idiopathic) focal segmental glomerulosclerosis (FSGS)? Both may present with nephrotic-range proteinuria, but the prognosis and choice of treatment may differ.

To date, the largest published study comparing ORG with primary FSGS is one by Kambham et al.\(^2\) In an analysis of 6818 renal biopsies, 71 patients with ORG were identified and compared with a control group of 50 patients with classic FSGS. The study showed that ORG less frequently progressed to end-stage kidney failure, with a 5-year renal survival rate of almost 90% (compared with about 50% in primary FSGS).\(^2\) While weight loss can reduce hyperfiltration and albuminuria in ORG,\(^3\) spontaneous remission is uncommon in primary FSGS.\(^4,5\) Does every obese patient with nephrotic-range proteinuria have ORG and an "indolent" course?

The degree of weight loss reported in the case described by Tran may not be achievable or sustainable in most obese patients. Do we have the luxury of waiting to assess the impact of weight loss on proteinuria? In about 50% of patients with primary FSGS, the serum creatinine level doubles after an average of 39 months.\(^2\) Furthermore, patients with primary FSGS and nephrotic-range proteinuria who do not achieve remission have a 5-year renal survival of only 50%, compared with almost 100% for those who attain remission.\(^5\) In addition, patients treated with corticosteroids (with or without cyclosporin or cyclophosphamide) have higher remission rates (30%–63%) than untreated patients (11%–14%).\(^3,5\) Therefore, a delay in introduction of specific therapy is not ideal.

There are some clinicopathological differences between ORG and primary FSGS that may help distinguish the two entities (Box). However, Kambham et al found that only two parameters were independently significant: serum albumin level and age.\(^2\) Although their study was based on a US population, it serves to demonstrate the principle that the major distinguishing feature between ORG and primary FSGS is the presence of full-blown nephrotic syndrome in primary FSGS (as demonstrated by the severity of hypoalbuminaemia).

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Clinicopathological differences between ORG and primary FSGS*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ORG</th>
<th>Primary FSGS</th>
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<tbody>
<tr>
<td>Mean age at presentation (years)(^1)</td>
<td>42.9</td>
<td>32.6</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White (%)</td>
<td>74</td>
<td>52</td>
</tr>
<tr>
<td>African American (%)</td>
<td>21</td>
<td>22</td>
</tr>
<tr>
<td>Nephrotic syndrome (%)</td>
<td>5.4</td>
<td>54</td>
</tr>
<tr>
<td>Mean 24-hour protein excretion (g)</td>
<td>4.1</td>
<td>6.9</td>
</tr>
<tr>
<td>Mean serum albumin level (g/L)(^1)</td>
<td>39</td>
<td>29</td>
</tr>
<tr>
<td>Mean serum cholesterol level (mmol/L)</td>
<td>5.9</td>
<td>8.6</td>
</tr>
<tr>
<td>Presence of pedal oedema (%)</td>
<td>35</td>
<td>68</td>
</tr>
<tr>
<td>Mean degree of segmental sclerosis (%)</td>
<td>10</td>
<td>39</td>
</tr>
<tr>
<td>Proportion of cases with glomerulomegaly (%)</td>
<td>100</td>
<td>10</td>
</tr>
<tr>
<td>Mean arteriosclerosis score (range, 0–3)</td>
<td>1.34</td>
<td>0.98</td>
</tr>
<tr>
<td>Mean degree of glomerular podocyte foot process fusion (%)</td>
<td>40</td>
<td>75</td>
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</tbody>
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ORG = obesity-related glomerulopathy. FSGS = focal segmental glomerulosclerosis. *Adapted from Kambham et al.\(^2\) † Independently significant.

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LETTERS
Obese patients have a similar risk of developing primary FSGS to people in the general population, and patients with nephrotic syndrome (particularly older adults) should not be presumed to have ORG and treated with weight loss alone. Certain pathological findings in a renal biopsy are helpful, but not definitive, in distinguishing ORG from primary FSGS. A biopsy would also exclude other treatable causes, such as minimal change disease. In addition to treatment with angiotensin-converting enzyme inhibitors, immunotherapy should be considered for obese, nephrotic patients, after discussing the potential risks and benefits with a nephrologist.

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Birth centre trials are unreliable
Kathleen M Fahy and Sally Tracy

To the Editor: The 2005 Cochrane review *Home-like versus conventional institutional settings for birth* has been cited in the public media to claim that birth centres are less safe than labour wards as there was an increased risk of a baby dying during or immediately after childbirth.1

This “headline-grabbing” statement is false. Firstly, this finding from the systematic review did not reach statistical significance.1 Secondly, the outcomes reviewed were related to the allocated place of birth, not the care provided. This fact is critically important, as 48% of women who were booked to have their baby in a birth centre did not give birth there.2 This is a predictable effect of the intention-to-treat principle. However, such high rates of “treatment contamination” negatively affect confidence in the study results.3 Additionally, the vast majority of baby deaths examined in the Cochrane review happened before labour and thus had nothing to do with care during childbirth.

One might wonder whether there was a real increased perinatal mortality rate resulting from delayed transfers from birth centres.4 The analysis found 41 deaths in total, but only six that occurred in normally formed babies who reached term (these are the only babies who are eligible to be born in a birth centre). Three of these deaths were associated with birth centre care, and three with standard labour care.

The interpretation of this Cochrane review raises questions about the validity of the underlying randomised controlled trials. In this experimental design, researcher control should ensure that people receive the specific treatment that was planned for them (treatment fidelity).4 The Cochrane handbook gives no guidance as to how to evaluate either the quality of the researchers’ definition of the planned treatments, or the fidelity between the treatments provided and the researchers’ plan.5 Most of the trials that formed the basis of the Cochrane review did not adequately define their treatments, nor adequately control the treatments provided to either group. It is not clear how the birth centre trials could sensibly be considered to have been scientifically controlled. The reviewers attempted to deal with this critical point by claiming that they were looking only at the effect of the “setting”, but their question clearly states that they were examining the effect of “care within a setting”.1

We conclude that the Cochrane review of the setting for birth is unreliable because of the weaknesses of the underlying trials. Rather than using questionable research to attack birth centres, it would be more constructive to engage in rigorously designed research that could provide robust evidence on the safety of all forms of maternity care, including standard medical care.

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Andrew F Pesce

In reply: Fahy and Tracy highlight the lack of high-level evidence about the relative safety of different models of maternity care. But in criticising the Cochrane review, it is important not to “shoot the messenger”. There is no doubt that the Cochrane review is not ideal but, like it or not, it remains the best evidence we have. The review of 8677 women in six randomised trials found a relative risk (RR) of perinatal death of 1.83 (95% CI, 0.99–3.38) in birth centres versus conventional institutional settings. In the 3332 pregnancies assigned to continuity of care by midwives who did not also work in conventional delivery suites, the RR was 2.38 (95% CI, 1.05–5.41).1

It would be fair to say that such findings should lead to real concerns about lack of safety rather than reassure the unbiased observer.

Other published evidence has raised similar concerns. A retrospective review of over 183 000 low-risk births in Stockholm, Sweden, found a statistically significant fourfold increase in intrapartum fetal mortality in women planning birth centre care compared with those planning standard care (three intrapartum deaths in 3256 babies of women planning birth centre care versus 36 deaths in 180 380 babies of those planning standard care).2 The increase in intrapartum mortality was almost sevenfold for primigravidae. These findings led to evidence-based changes in the organisation of the birth centre involved to minimise the identified risks.

To paraphrase Fahy and Tracy, rather than criticising the best available evidence reviewing birth centre outcomes, it would be more constructive to engage in rigorously designed research to assess how risk might be minimised in all forms of maternity care.

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Research is needed before GPs can engage in “positive” family planning

Angela M Cooney

To the Editor: I was very concerned to read the letter from Mazza et al1 regarding “positive” family planning and feel I must make a comment. The authors are well known for their work in the area of women’s sexual and reproductive health, but I would like to challenge some of the points they have made.

The first point: whether intervention by general practitioners would be appreciated by younger women not yet interested in motherhood. I believe it is part of the role of doctors to inform, even when a person may not be ready for the information. Telling 20-a-day smokers that they are not doing their body any favours doesn’t go down well with some people, but even this brief intervention can change behaviour and save lives.

The second point: whether GPs can respect patients’ autonomy. Every day I speak to women who have been offended and upset by doctors who have said something awkwardly, or imposed their own values, or been downright offensive. That won’t change, and recommending that well informed and tactful doctors wait before imparting vital information until the rest of the world lifts its game means we will all be waiting a long time.

Part of the art of medicine is judging the audience and knowing the perfect point in a consultation to speak, and how to say it. What can be more appropriate, when seeing a woman in her late 20s who has requested a repeat prescription for the contraceptive pill, than to ask casually (as one is unrolling the sphygmomanometer cloth), “So, do you think there might be any children in your future?” The usual response, as detailed in Cannold’s book,2 is an emphatic “yes”. The next question, “Have you got a time scale when you would be looking at that?”, may give the opportunity to mention such things as rubella vaccination, smoking and folate supplements. And if the woman indicates that pregnancy would be on her to-do list at age 38, then a reasonable and non-harassing response could be, “Could we talk about fertility rates at that age?”

By all means do research, but don’t ask doctors to be silent about this important issue until the sociologists have spent another 10 years on it. By that time, it will be too late for a lot more women.

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