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This Issue in the Journal

The Safe-T-Sleep® device: safety and efficacy in maintaining infant sleeping position

T de Chalain

This study aimed to test the safety and efficacy of a commercially supplied infant sleep-wrap device in selecting and maintaining infant sleeping position. Thirty one babies were observed for nearly 400 hours. With correct use, there were no adverse incidents and selected body position was maintained in over 92%, and head position in over 85% of observations. We advocate the use of the STS device as an adjunctive measure in the treatment of babies referred with plagiocephaly without synostosis (ie, flattening of the skull induced by persistent sleeping in one position).

Resuscitation teaching in New Zealand schools

C Lafferty, P Larsen, D Galletly

We surveyed every primary and secondary school in New Zealand to determine the extent to which resuscitation is taught in New Zealand. Our results indicate that the majority of primary schools are not teaching resuscitation, and the majority of secondary schools are treating resuscitation as an optional subject, taught only to a small proportion of students. Resuscitation needs to become a compulsory rather than an optional component of the curriculum, with corresponding levels of funding, if we are to achieve widespread community knowledge of how to save lives.

Current practice for anticoagulation prophylaxis in inguinal hernia surgery: a questionnaire survey

S Anwar, P Scott

The formation of blood clots in the legs and their subsequent propagation to the lungs following surgery is a well known and serious complication. Incorrect use of current prophylactic measures to avoid this complication can result in under or over treatment with associated side effects. Prophylactic treatment should be instituted after due consideration has been given to the patient's medical condition and the type of surgery to be performed.

Correction of deformational auricular anomalies by moulding – results of a fast-track service

S Tan, A Wright, A Hemphill, K Ashton, J Evans

Ear anomalies are conventionally treated with surgery. However, the majority of ear anomalies are deformational and can be treated cheaply and non-surgically with a simple splint. For this to be effective, treatment should be initiated within the first three months of life. Paediatricians, obstetricians, family doctors and midwives should

be encouraged to manage these anomalies through moulding so that the use of the technique becomes widespread.



Quality improvement: time for radical thought and measurable action

Louise Thornley, Robert Logan and Ashley Bloomfield

Last week's 3rd Asia Pacific Forum on Quality Improvement in Health Care, which attracted leading overseas commentators and around 900 participants, is evidence of the momentum building around healthcare quality in New Zealand. Specific initiatives in recent years include the Health and Disability Services (Safety) Act 2001, professional-led credentialling of senior medical staff,¹ and sentinel events reporting.²

The momentum received a further boost during the conference with the release of the report 'Improving quality: a systems approach for the New Zealand health and disability sector'.³ Health professionals should not underestimate the importance of this report. If you are going to read one 'policy' document this year – or at least the executive summary – make it this one.

The 'Improving quality' (IQ) report is the result of a robust process, led by the Ministry of Health. A working group with wide representation provided guidance and a draft was circulated to the sector for comment. The report builds on advice to the Minister of Health from the National Health Committee (NHC),⁴ and a discussion paper on quality improvement in hospitals.⁵ The NHC advice drew on a review of international experience,⁶ discussions from two national workshops,⁷ consultation with a wide range of health and disability providers, input from consumers and Maori organisations, and submissions on a discussion document.⁸

The IQ report identifies key dimensions of quality: people-centred, access and equity, safety, effectiveness and efficiency (Figure 1). The report also advocates a systems approach to healthcare quality, not as an end in itself but as a means to enhance services for people. Thus, quality must encompass all levels of the system – individuals, teams, organisations, subsystems – as well as interactions between different levels. Quality is the responsibility of all people working in healthcare, but these people must be supported by a system that places a high priority on safety and enables ongoing quality improvement.

Figure 1. Quality dimensions for the New Zealand health and disability system



The language of quality can be confusing, but quality activities fall into two broad camps: quality assurance and quality improvement. Quality assurance is about setting expectations (standards), their implementation, and measurement of performance against them. Quality assurance is essential and we need to do it well to maintain the safety of our healthcare system. However, international evidence increasingly suggests that a sole focus on quality assurance is not enough.³

Quality improvement is where we need to head – combining quality assurance activities with an explicit concern for quality and continuous improvement. Quality improvement is underpinned by incremental change where all individuals, teams and organisations (from small providers to the Ministry of Health) critically evaluate their practice, incorporate new learning into their work and, importantly, share their learning with others. This is not a simple linear process with a beginning and an end, but an ongoing cycle of reflection and action. A quality-improvement approach calls for us to examine not just what we are doing (outputs), but also how we are doing it and what we are getting – the outcomes.⁹ This implies monitoring the total process of care, and measurement of outcomes rather than outputs.

In addition to incremental changes in practice, more radical change is required, and herein lies the challenge. There needs to be a fundamental shift in our attitude towards quality improvement. As stated unequivocally by the NHC, quality improvement should be the prime focus of healthcare delivery if we are to achieve the best possible outcomes. Quality must no longer be seen as of interest just to those people with ‘quality’ in their job title, but needs to become the responsibility of everyone.

Not surprisingly, most doctors and other health professionals maintain that they already have a focus on quality as part of their work. We do always strive to do the best for patients and families, and there are many existing activities that demonstrate a commitment to quality.

But in reality how often do we follow routine audit with decisive action and ongoing evaluation? Do we analyse the accessibility of our services? Do we assess our competence in working with people from cultures other than our own? What processes do we use to critically assess emerging evidence to ensure new

interventions are safe and actually improve outcomes for patients at a reasonable cost – both to the public purse and to individuals? Do we measure and analyse the outcomes of our practice and, even more importantly, present this information to the people seeking our advice?

International quality expert Dr Donald Berwick, who spoke at the Auckland conference, has written movingly on his wife's personal experience within the health system to reflect the crisis facing healthcare around the world, and to suggest ways to address this crisis and improve quality of care.¹⁰ The crisis he describes is not one of funding or unlimited demand, but a crisis in the way that healthcare systems deliver care.

Berwick argues that to improve quality we need a new approach that faces the reality of the current problems and involves leadership, teamwork, integration and good communication. It calls for innovative approaches that may not necessarily use the 'tools' that we are used to. Berwick challenges us to think outside the square, and to question whether our traditional thinking and practices are helping us to improve healthcare.

While Berwick's experience lies within the US healthcare system, any clinician reading his account will instantly recognise many of the small and large failures in the care received by his wife. The errors were not rare; they were a daily occurrence. If they are chillingly familiar to us, they must be more so for the numerous people worldwide whose care is affected by such failures. However, even more alarming is the obvious conclusion that many of these failures are easily preventable and that their prevention would actually save rather than cost money.

Key to a change in thinking must be a strong emphasis on being 'people-centred'. The genuine placement of people at the centre of healthcare decisions at all levels is a prerequisite for improving the outcomes of healthcare. In particular, cultural competence at all levels of the system is crucial. Cultural competence in healthcare requires us to understand social and cultural factors that influence patients, and devise interventions that take these factors into account.¹¹ Cultural competence is a necessary skill for health professionals and an essential part of effective care.¹² In New Zealand we have a particular obligation to understand relevant Maori cultural issues and to apply that understanding in practice.

As health professionals we have an important part to play in improving quality – in particular as leaders and role models – but we need to be clear on what this means in practice. The challenge to improve quality demands that we revolutionise our thinking about quality, moving from a focus on quality assurance to a model of quality improvement that routinely involves different professionals and people who use our services. On a practical level, we can begin now by making incremental, evidence-based changes for better outcomes. Both radical thought and measurable action are called for.

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References:

1. Ministry of Health. Towards clinical excellence – framework for the credentialling of senior medical officers in New Zealand. Wellington: Ministry of Health; 2001. Available online. URL: <http://www.moh.govt.nz/moh.nsf/c7ad5e032528c34c4c2566690076db9b/85a5c6ade26c60fac256a10000a5d68?OpenDocument> Accessed September 2003.
2. Ministry of Health. Towards clinical excellence: learning from experience. A report to the Director-General of Health from the Sentinel Events Project Working Party. Wellington: Ministry of Health; 2001. Available online. URL: <http://www.moh.govt.nz/moh.nsf/7004be0c19a98f8a4c25692e007bf833/008deb2fa836ba68cc256ad000804456?OpenDocument> Accessed September 2003
3. Ministry of Health. Improving quality (IQ): A systems approach for the New Zealand Health and Disability Sector. Wellington: Ministry of Health; 2003. Available online. URL: <http://www.moh.govt.nz/moh.nsf/7004be0c19a98f8a4c25692e007bf833/f9eb9f14e7626b8ccc256d96007f6b4e?OpenDocument> Accessed September 2003.
4. National Health Committee. Safe systems supporting safe care: final report on health care quality improvement in New Zealand. Wellington: National Health Committee; 2002. Available online. URL: <http://www.nhc.govt.nz/publications/SafeSystemsSupportingSafeCare.pdf> Accessed September 2003.
5. Ad hoc group established by the Associate Minister of Health Hon Ruth Dyson. Discussion paper: quality improvement strategy for public hospitals. Wellington: Ministry of Health; 2001. Available online. URL: <http://www.moh.govt.nz/moh.nsf/7004be0c19a98f8a4c25692e007bf833/addbf3afa1174214cc256ad40014ba26?OpenDocument&Highlight=0.dyson> Accessed September 2003
6. Coster G, Buetow S. Quality in the New Zealand health system. Background paper to the National Health Committee. Wellington: National Health Committee; 2001. Available online. URL: <http://www.nhc.govt.nz/publications/qualityreport/qualityreport.html> Accessed September 2003.
7. National Health Committee. Summary of National Health Committee Quality Workshop 21 February 2001. Wellington: National Health Committee; 2001. Available online. URL: <http://www.nhc.govt.nz/wrkprg/QualityWorkshopReportFeb2001.pdf> Accessed September 2003.
8. National Health Committee. Safe systems supporting safe care: a discussion document on quality improvement in health care. Wellington: National Health Committee; 2001. Available online. URL: <http://www.nhc.govt.nz/publications/qualityreport/discussion.html> Accessed September 2003.
9. Bloomfield A, Logan R. Quality improvement perspective and healthcare funding decisions. *BMJ* 2003;327:439–43.
10. Berwick D. Escape fire: lessons for the future of health care. New York: Commonwealth Fund; 2002. Available online. URL: http://www.cmwf.org/programs/quality/berwick_escapefire_563.pdf Accessed September 2003.
11. Betancourt JR, Green AR, Carrillo JE, Ananeh-Firempong O. Defining cultural competence: a practical framework for addressing racial/ethnic disparities in health and health care. *Public Health Rep* 2003;118:293–302.

12. Durie M. Cultural competence and medical practice in New Zealand. Address to the Australian and New Zealand Boards and Council Conference. 2001 Nov 22; Wellington, New Zealand.



Plagiocephaly – more questions than answers

Ed Mitchell and Lynne Hutchison

There has been a striking increase in referrals for plagiocephaly without synostosis (PWS) to neurosurgical and plastic surgical units in New Zealand and overseas.^{1,2} It appears that this increase is related to the increasing acceptance of the SIDS-protective supine sleeping position, although greater awareness and better recognition of the problem also play a part. Prior to 1991, when the National SIDS Prevention Campaign in New Zealand first promoted non-prone sleeping, and at a time when most Western babies were sleeping on their tummies or sides, 'plagiocephaly' meant frontal head flattening; now, there is an almost universal association with occipital flattening. The effects of gravity on a soft, rapidly growing infant cranium lying on a flat surface are such that moulding can occur if the resting position is constantly the same. And, if that is combined with a neck-muscle dysfunction that impairs head rotation, the stage is set for the typical cephalic configurations of a parallelogram-shaped head or brachycephaly or both.

The prevalence of PWS is unclear and has historically been clouded by misclassified lambdoid synostosis.^{1,3,4} A population-based Dutch study found a prevalence of 9.9% in infants under the age of six months; however, the deformity was visually assessed and not quantified.⁵ Head shape varies between the perfectly symmetrical to the severely abnormal, and thus prevalence will vary depending upon the criteria used to define normal and abnormal. However, there are difficulties in quantifying PWS. Although a subjective visual assessment is important, a quick, reliable, objective and noninvasive measure of severity would be useful in order to quantify the abnormality and to follow the deformity over time.³ Methods described in the literature include callipers,^{6,7} 3-D CT scans,^{8,9} photographs,¹⁰ articulated rulers,¹¹ and manual tracings made from flexible strips pressed around the circumference of the infant's head.¹² The fact that there are many different methods suggests that none is ideal.

The natural history of PWS is unknown; few adults have such a deformity, suggesting that it is self-correcting or masked by hair growth. Probably most cases routinely improve with time, but there are no good long-term studies that address the issue of how much time. The effects of true non-treatment are unknown and difficult to evaluate because most parents will try to correct the condition using counter-positioning. There seems to be a group of infants with persistent and severe PWS, but as yet we do not know how to identify these children early in order to institute early treatment strategies.

Primary prevention advice includes gently varying the head position at each sleep until the infant can do it alone, changing the cot environment to encourage looking around, and giving supervised tummy time for play from an early age to encourage upper-body strength.¹³ Although we have conducted a case-control study that gives some support to these recommendations, a larger prospective study is needed to confirm the integrity of this advice.¹⁴

The two fundamental principles underlying treatment of PWS are early recognition and keeping the infant from lying on the flat spot. Positioning advice for the treatment of PWS has ranged from simply staying off the flattened occiput whilst asleep,² to 'active counter-positioning',¹² which not only advises keeping off the flat spot but actively applies pressure to prominent areas by positioning the infant on the bossed side of the occiput. Upright time and supervised tummy time also help to avoid pressure on the occiput. Some authors¹⁵ argue that repositioning is effective only before four months of age, because after that infants tend to reposition themselves, and because longer trials of repositioning only serve to delay treatment, thus making positive outcomes more difficult. Treatment options for severe cases include helmets, which constrain the bossed areas and provide room for growth over the flat areas. More invasive treatment options are cranial surgery and botulinum toxin treatment for severe torticollis contributing to head deformity.

The plagiocephaly literature contains a few references to devices designed to keep the infant positioned off the flat part of the occiput. One author has advocated tying a large knot in a stocking cap, to be positioned over the flat area,¹⁶ others have suggested 'positioning rolls',² sandbags¹⁷ or foam wedges;⁶ none appears to have been assessed for safety. Soft sleeping helmets with a block or cone attached to the flattened side have also been used to keep the plagiocephalic infant from turning to a favoured postural position. To our knowledge, there are three commercially available positioning devices for sale in New Zealand at present. They are a foam-wedge system called the Sleep-Ez™; a sloping foam block with a saucer-shaped indentation for the head to rest in called the Occ-Block; and a fabric sleep wrap known as the Safe-T-Sleep®. The lower portion of the Safe-T-Sleep® device is fastened around the mattress while the upper portion wraps around the infant's chest and abdomen to maintain sleep position.

If a positioning device is to be used it needs to not only maintain the position but be absolutely safe. De Chalain has attempted to address the difficulty of recommending a safe and effective system of keeping infants off a flat occiput by testing the Safe-T-Sleep® device in the monitored environment of a hospital setting. His findings are published in this issue of the NZMJ.¹⁸ The infants tested were sick children. In this study, the head position was maintained in the desired position for 85% of the observed hours. Would the results be any different in healthy and possibly more active infants at home in the care of busy parents? It is not clear how often the nursing staff elected to use the supine position and the semi-supine position, and whether this position was changed during the night; one would imagine that maintenance of head position would be easier in the semi-supine posture. In addition, it would be interesting to know whether the clothing was always pinned to the Safe-T-Sleep®. We are of the opinion that fastening the Safe-T-Sleep® to the infant's clothing would be the only way to prevent an active, strong-minded baby from rolling to prone were they determined to do so.

It is uncertain from this report as to the seriousness of the adverse events encountered. Safety would be difficult to assess in such a study, as rare events would not be detected. The two adverse events in this trial involving 31 babies are summarised as 'Device too loose – unwanted movement'. This suggests a very real possibility that untrained caregivers could have the same problem were the device not secured correctly *every time*. However, the manufacturer reports that no deaths or serious

adverse effects have been reported with over 70 000 units sold (personal communication, M Rutherford, 2003).

Early awareness and recognition are vital for the management of plagiocephaly. The authors are now recommending the Safe-T-Sleep® for the treatment of plagiocephaly, but have not subjected this to any formal trial. Although they have found that counter-positioning is as effective as treatment with a helmet, this might indicate that neither changes the natural history of the disorder. There is a need for a randomised controlled trial of sleep-positioning devices versus an education programme for the treatment of mild to moderate PWS.

Unfortunately, there are more questions than answers. There is no quick fix for a misshapen infant head, and the old maxim 'prevention is better than cure' is very relevant. Because of parental and health professional concern about plagiocephaly, recommendations need to be made, even though the evidence for these recommendations is limited. Prevention programmes should raise awareness that not just sleeping, but holding and playing positions and the use of car seats may be important. Watching for a preferential head orientation and encouraging turning of the head both ways are prudent. Perhaps we could do away with the expense of and reliance upon positioning aids except in the more severe cases.

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References:

1. David DJ, Menard RM. Occipital plagiocephaly. *Br J Plast Surg* 2000; 53:367–77.
2. Argenta LC, David LR, Wilson JA, Bell WO. An increase in infant cranial deformity with supine sleeping position. *J Craniofac Surg* 1996;7:5–11.
3. Rekeate HL. Occipital plagiocephaly: a critical review of the literature. *J Neurosurg* 1998;89:24–30.
4. Huang MH, Gruss JS, Clarren SK, et al. The differential diagnosis of posterior plagiocephaly: true lambdoid synostosis versus positional molding. *Plast Reconstr Surg* 1996;98:765–76.
5. Boere-Boonekamp MM, van der Linden-Kuiper LT. Positional preference: prevalence in infants and follow-up after two years. *Pediatrics* 2001;107:339–43.
6. Mulliken JB, Vander Woude DL, Hansen M, et al. Analysis of posterior plagiocephaly: deformational versus synostotic. *Plast Reconstr Surg* 1999;103:371–80.
7. Kelly KM, Littlefield TR, Pomatto JK, et al. Cranial growth unrestricted during treatment of deformational plagiocephaly. *Pediatr Neurosurg* 1999;30:193–9.
8. Glat PM, Freund RM, Spector JA, et al. A classification of plagiocephaly utilizing a three-dimensional computer analysis of cranial base landmarks. *Ann Plast Surg* 1996;36:469–74.
9. Lo LJ, Marsh JL, Pilgram TK, Vannier MW. Plagiocephaly: differential diagnosis based on endocranial morphology. *Plast Reconstr Surg* 1996;97:282–91.

10. Clarren SK. Plagiocephaly and torticollis: etiology, natural history, and helmet treatment. *J Pediatr* 1981;98:92–5.
11. Watson GH. Relation between side of plagiocephaly, dislocation of hip, scoliosis, bat ears, and sternomastoid tumours. *Arch Dis Child* 1971;46:203–10.
12. Loveday BP, de Chalain TB. Active counterpositioning or orthotic device to treat positional plagiocephaly? *J Craniofac Surg* 2001;12:308–13.
13. Education for Change. Protecting your baby's head shape. Christchurch: Education for Change; 2000. Web site: <http://www.efc.co.nz/> Accessed September 2003.
14. Hutchison BL, Thompson JM, Mitchell E. The determinants of nonsynostotic plagiocephaly: a case-control study. *Pediatrics* 2003 (In press)
15. Littlefield TR, Pomatto JK, Kelly KM. Dynamic orthotic cranioplasmy: treatment of the older infant. Report of four cases. *Neurosurg Focus* 2000;9:e5.
16. Graham JM Jr. Alterations in head shape as a consequence of fetal head constraint. *Semin Perinatol* 1983;7:257–69.
17. Pollack IF, Losken HW, Fasick P. Diagnosis and management of posterior plagiocephaly. *Pediatrics* 1997;99:180–5.
18. De Chalain T. The Safe-T-Sleep[®] device: safety and efficacy in maintaining infant sleeping position. *NZMJ* 2003;116(1181). URL: <http://www.nzma.org.nz/journal/116-1181/581/>



The Safe-T-Sleep[®] device: safety and efficacy in maintaining infant sleeping position

Tristan de Chalain

Abstract

Aims The issue of infant sleeping position has socio-political ramifications. Current recommendations endorse supine sleeping as an aid to reducing the risk of sudden infant death syndrome (SIDS). Persistent sleeping of a newborn infant in the same position may induce plagiocephaly without synostosis (PWS). Parents in our craniofacial clinic, whose children present with PWS, often feel torn between apparently conflicting goals – avoiding SIDS and avoiding PWS. The Safe-T-Sleep[®] device, a form of infant sleep wrap, purportedly allows safe semi-supine positioning, thus ameliorating PWS (by preventing the infant from lying on the cranial ‘flat spot’) while not increasing the risk of SIDS. Before recommending the device to parents in our plagiocephaly clinics, we designed a prospective, hospital-based trial to assess the safety and efficacy of the device in maintaining selected sleeping positions. This was not a trial of the efficacy of the Safe-T-Sleep[®] device in treating plagiocephaly.

Methods The devices were trialed on 31 babies, between birth and 11 months of age. A total of 396 hours of observations were recorded.

Results The device maintained the selected body position in 94% of recorded observations and head position in 87%. There were no significant adverse events or complications associated with the use of the Safe-T-Sleep[®] device.

Conclusions The device appears to be safe and effective. It is now being advocated in our clinic as an aid to active counter-positioning strategies to passively correct incipient or established positional plagiocephaly in younger babies.

New Zealand has experienced something of an epidemic of sudden infant death syndrome (SIDS) or cot deaths over recent years. As a result, there has been considerable national debate about the issue and research into causal factors is examined with great interest by a wide range of interested parties. Current recommendations from experts in the field strongly advocate supine positioning for sleeping infants, citing a sixfold reduction in the risk of SIDS when compared with a prone sleeping position, and a threefold reduction when compared with side-lying.^{1,2,3}

Concomitant with the reduction in SIDS rates that these recommendations have produced, is a marked increase in the number of babies in New Zealand referred for advice regarding plagiocephaly without synostosis (PWS) or ‘flat heads’.⁴ It is axiomatic that if a neonate, and more especially a premature neonate, is positioned consistently in the same attitude, be it supine, supine with the head turned to one side, or side-lying, the cranium will flatten, simply due to the effects of gravity pressing a soft skull against a relatively unyielding surface (the mattress). A comparable rise in the numbers of PWS referrals has been seen in the USA in the years subsequent to the 1992 recommendation from the American Academy of Paediatrics that supine

sleeping was best for babies.^{5,6} It is hard to escape the conclusion that supine sleeping, with a single, maintained attitude, may be causally related to flat-head presentations.

Since most cases of PWS can be prevented, or, if picked up early, resolved by active counter-positioning (ie, never allowing the infant to sleep on the cranial 'flat spot'), it would be useful to be able to offer parents some practical means of applying the principle that the child must not be allowed to sleep on the flattened plane of a deformed skull. That is, some way of being able to select and maintain a sleeping position that is both safe and effective. Too often children of six months or older are referred for consultation regarding a well-established case of PWS; the trouble is that, by this time, not only is the deformity well developed and obvious, but the child is also old enough to have very firm ideas about what is his or her favoured sleeping position and will actively resist parental attempts to modify this. It is much more comfortable for the baby to lie with the flat spot down on the mattress than try to sleep balancing the skull on the adjacent high spot. It is precisely because of this difficulty that some resort to orthotic devices such as the DOC band[®] and cranial moulding helmets.⁵ Not only are such devices costly and difficult to make, but they need regular adjustment to remain effective as the head shape alters. In many cases they are simply abandoned by parents frustrated by the difficulties of finding a comfortable, efficacious, device that the infant will tolerate wearing. Certainly, PWS is easier to prevent than resolve; while most children, given sufficient time and attention to restricting pressure on the flat spot, will revert towards normal craniofacial symmetry, some are left with significant asymmetry, which even exuberant hair growth cannot fully hide. In the USA, long-term follow-up studies have shown that about 4% of cases remain sufficiently deformed to warrant consideration for surgical correction of the plagiocephaly.⁷

Consequently, when the commercially available Safe-T-Sleep[®] (STS) device came to our attention, it was felt that it might offer a means whereby an infant, presenting with incipient or established PWS, might safely sleep in the semi-supine position, but with the head turned away from the flat spot and mattress pressure confined to the high-spot area. In other words, it might offer a means of maintaining control of sleeping position and therefore be useful as an adjunct in the treatment of the burgeoning number of babies presenting with flat heads. Before recommending the device to parents, however, we needed to be certain that it was both safe and effective in maintaining sleeping position. To this end, a prospective, hospital-based trial was designed to assess the STS device. It should be noted that this was **not** a trial of the device's efficacy in the treatment of established PWS or 'flat head', since we have already established that active counter-positioning (ie, prevention of the infant lying on the flat spot), by whatever means achieved, is very effective at correcting plagiocephaly.⁴ If it could be shown that the STS device was both safe and effective at maintaining a selected sleeping position, we would be able to recommend it to parents of plagiocephalic infants as a useful adjunct in achieving active counter-positioning.

Methods

A prospective trial was designed in which a number of STS devices were purchased and trialled, according to the manufacturer's instructions, in the wards and special care units of Middlemore Hospital, a large, regional healthcare facility located in suburban South Auckland. The manufacturers of the device had no input into the design of the study or its outcome, and no financial or material benefit accrues to the hospital or the authors as a result of this study. Those involved in the design and execution of the study have no connection whatsoever with the manufacturers of the STS device.

After obtaining ethical approval to proceed with the study, and obtaining informed consent from individual parents or caregivers of each baby entered in the trial, the following design was applied.

All trial entrants were categorised according to age interval: 0–3 months, 3–6 months, 6–9 months and 9–12 months. Babies were eligible for entry into the trial providing they were not afflicted with PWS and they were spending at least one night in the hospital in the medical or surgical wards. Neonates in the special care baby unit (SCBU) for observation or treatment of a relatively minor complaint, which did not impact significantly on their overall mobility or strength, were also eligible. The primary care physician in charge of each patient, who was not part of the study team, assessed their patient as being suitable for trial entry and parents reserved the right to remove their baby from the trial at any time.

Each entrant was placed in an STS device when being put down for a night's sleep and the selected head and body position noted. (See appendix for demonstration of how STS device is fitted.) The starting position was semi-supine or supine, with the head turned to the left or to the right. The immediate care-giving nurse had freedom to select the initial sleeping position, according to the infant's perceived needs. When semi-supine was chosen as the start position, a rolled towel was placed behind the raised shoulder. Every hour thereafter, the observed head and body positions were noted and recorded. Variances from the positions selected were documented, as were difficulties like restlessness, 'escape' from the device, and possible dangerous positioning, such as facial obstruction by bedclothes, soft toys and so on. The data were recorded by night-staff nurses at the bedside, who were otherwise not involved in the study. Data were recorded as hours' observation points, and form the basis of the analyses that follow.

Results

The results of our observations are presented in Tables 1, 2 and 3.

Table 1. Hours of observation of infants wearing STS devices

Age group	Number of babies	Hours of observation
0–3 months	17	240
3–6 months	2	27
6–9 months	10	108
9–12 months	2	21
Total	31	396

Table 2. Proportional maintenance of selected sleeping position

Age group	% body position maintained	% head position maintained
0–3 months	93 (16/240)*	91 (21/240)
3–6 months	95 (1/27)	85 (4/27)
6–9 months	92 (9/108)	80 (22/108)
9–12 months	95 (1/21)	90 (2/21)
Mean	94	87

*numbers in brackets indicate ratio of hours of observation in which position not maintained

Table 3. Adverse events summary

Age group	Incidence of adverse events	Details
0–3 months	2	Device too loose* – unwanted movement
3–6 months	0	
6–9 months	1	Child was febrile – overheated
	1	Very active child – required frequent device adjustment
9–12 months	1	Unwanted head movement

*In no instance was a child actually able to turn into the prone position. However, failure to apply the STS sleep wrap according to the manufacturer's instructions makes this a theoretical risk.

Discussion

In assessing the results of this simple data-collection exercise, several useful points can be made.

In the first instance, it would appear that the younger the baby the easier it is to select and maintain a sleeping position with the STS device. This is probably true of any similar behaviour the parents may be trying to teach the child and probably relates to tolerance; in the neonatal period, novelty is more readily accepted. As the babies become older, they become stronger, louder and less likely to passively accept such impositions as a relatively restrictive positioning device.

Overall, there were very few untoward experiences with the STS device. In no single instance was a child ever at physical risk. However, in two babies in the 0–3 month age group the device was applied too loosely. This allowed a deal of unwanted movement that could conceivably have resulted in an unmonitored child being able to turn over within the device sufficiently to place itself at risk. However, when the device was firmly applied and pinned to the overpants, as recommended by the manufacturer, this did not occur.

Not surprisingly, the babies' heads were free to move more than were their bodies. Nevertheless, for this sample of 31 babies undergoing nearly 400 hours of monitored observation, the STS device was at least 85% (mean 87%) successful in maintaining selected head position, and at least 92% (mean 94%) successful in maintaining body position, across all age groups.

Although the major flaw of very low patient numbers in some of the groups precludes extrapolation to the wider population, it nevertheless appears that the STS device may well be helpful to those wishing to maintain a selected sleeping position in babies. As such it might prove a useful addition to a therapeutic programme of active counter-positioning as treatment for cranial moulding or plagiocephaly. Not surprisingly, it is most readily accepted when introduced early in the baby's life; indeed, its use in the treatment of positional plagiocephaly may be obviated by the early introduction of intelligent advice regarding sleeping position. We tell our families to practise supine sleeping with the head turned to the left on night one, to the right on night two and so on. It is in helping such families to achieve this goal, especially as a training aid in the younger babies, or as a behaviour modifier in the slightly older baby, that such a device would seem to be most useful. Its efficacy in specifically altering the plagiocephalic deformation that is being seen much more commonly today remains to be established (although, anecdotally, it would indeed seem to be very effective in this regard). At the least, however, our data have enabled us to recommend the device to our patients' families as being effective and safe in maintaining a selected sleeping position in babies less than a year of age.

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References:

1. Dwyer T, Ponsonby AL, Newman NM, Gibbons LE. Prospective cohort study of prone sleeping position and sudden infant death syndrome. *Lancet* 1991;337:1244–7.
2. Fleming P, Gilbert R, Azaz Y, et al. Interaction between bedding and sleeping position in the sudden infant death syndrome: a population based case-control study. *BMJ* 1990;301:85–9.
3. Gunn AJ, Gunn TR, Mitchell EA. Is changing the sleep environment enough? Current recommendations for SIDS. *Sleep Medicine Reviews* 2000;4:453–69
4. Loveday BP, de Chalain TB. Active counterpositioning or orthotic device to treat positional plagiocephaly? *J Craniofac Surg* 1994;12:308–13.
5. Ripley CE, Pomatto J, Beals SP, et al. Treatment of positional plagiocephaly with dynamic orthotic cranioplasty. *J Craniofac Surg* 1994;5:150–9.
6. Turk AE, McCarthy JG, Thorne CH, Wisoff JH. The “back to sleep campaign” and deformational plagiocephaly: is there cause for concern? *J Craniofac Surg* 1996;7:12–8.
7. Argenta LC, David LR, Wilson JA, Bell WO. An increase in infant cranial deformity with supine sleeping position. *J Craniofac Surg* 1996;7:5–11.

Appendix

Figure 1. The main component of the Safe-T-Sleep[®] device wraps around and fastens to the mattress. The central portion, which is sewn to the main mattress-wrap portion is spread out to receive the baby.



Figure 2. The baby is placed supine on the central portion and the velcro-backed straps placed snugly around the central chest and abdomen.



Figure 3. The overpants are pulled up over the lower margin of the central wrap and safety-pinned in place. This prevents the baby wriggling out of the device. To select a semi-supine position, a rolled towel can be placed behind the shoulder (not illustrated).



Figure 4. Detail showing how the overpants are pinned to the central section of the sleep wrap.





Resuscitation teaching in New Zealand schools

Christiana Lafferty, Peter Larsen and Duncan Galletly

Abstract

Aims Resuscitation skills such as cardiopulmonary resuscitation (CPR) are taught as an optional component of the New Zealand school curriculum. This study was conducted to determine the frequency of, and factors influencing, CPR teaching in New Zealand primary and secondary schools.

Methods At the end of the 2001 school year, we surveyed by questionnaire every school in New Zealand asking which schools taught CPR skills during 2001, what other resuscitation skills were taught, and what the barriers to greater teaching of resuscitation were.

Results Seven hundred and fifty four of 2205 (34.9%) primary schools and 173 of 456 (38.6%) secondary schools returned the survey. Of primary schools, 37.5% taught resuscitation skills during 2001, as did 81% of secondary schools. In secondary schools, resuscitation was most commonly taught during year 12 (pupil age 16–17 years), but then only as an elective subject to 10–30% of students. For both primary and secondary schools there was a positive correlation between school size (number of pupils) and the teaching of resuscitation ($p = 0.0001$). The most significant barriers to resuscitation teaching were identified as funding, an overfull curriculum and, in primary schools, the question of the suitability of teaching resuscitation to young children.

Conclusions This survey indicates that the majority of primary schools are not teaching CPR skills, or other life-saving first aid, and that the majority of secondary schools are treating these subjects as optional, taught only to a small proportion of students. If New Zealand is to achieve widespread community CPR knowledge, it is suggested that greater funding needs to be available to schools for resuscitation/first-aid training and the subject must become a compulsory, rather than optional, component of the school curriculum.

As in other developed countries, New Zealand has an incidence of out-of-hospital cardiac arrest of approximately one in two thousand per annum. Most arrests are associated with myocardial ischaemia, and 95% of the victims die¹ (personal communication, T Smith, St John, Northern Region, 2002 and P Roberts, Wellington Free Ambulance, 2001). New Zealand has one of the highest incidences of death associated with drowning compared with other developed countries and likewise with motor vehicle crashes.²

For most causes of sudden unexpected death, a bystander's ability and willingness to perform cardiopulmonary resuscitation (CPR) will increase the chance of the victim's survival. For out-of-hospital cardiac arrest, bystander CPR increases the likelihood of survival two to three times,³ and for drowning, CPR may be all that is required to resuscitate the victim. In order to ensure that victims of cardiac arrest and drowning have the greatest possible chance of survival, it is desirable that as many people as

possible within the community have the knowledge and skill to perform CPR. It has been suggested that in order to achieve this CPR should be taught at an early age, as part of the school curriculum to all school students.⁴⁻⁹

In 1999 the New Zealand Ministry of Education introduced a new Health and Physical Education curriculum for New Zealand schools. This curriculum comprises a set of achievement objectives expressed at eight progressive levels, each level catering for the students' development and maturity as they move from year 1 to 13 (corresponding to ages 5 to 18). Preliminary aspects of resuscitation and first aid are first *suggested* at Level 1 (years 1–5: ages 5 to 10), rescue breathing at Level 3 (years 2–8: ages 6 to 13), CPR at Level 5 (years 6–12: ages 11 to 17), and CPR repeated at Level 7 (years 9–13: ages 14 to 18). Throughout this staged introduction the topics are given as *suggested* inclusions to the curriculum and can be expanded on, or replaced by other unrelated topics, at the teacher's or school's discretion.¹⁰

Given the importance of CPR teaching to a national strategy for cardiac arrest survival, and given a non-mandatory school curriculum, this present study sought to assess the frequency of resuscitation teaching in New Zealand primary and secondary schools and to identify perceived barriers to this teaching.

Methods

In October 2001, a questionnaire with prepaid reply envelope was posted to the 'health coordinator' of every New Zealand school listed in the Ministry of Education database. Those schools not responding within four weeks were sent a reminder letter via email, and another copy of the questionnaire at the end of November 2001.

For the purposes of this study resuscitation training was defined as the formal teaching of one or more of the following: access to the emergency services (dial 111), rescue breathing, adult chest compression and CPR in children.

The questionnaire sought information based upon the 2001 school year, and included (a) whether resuscitation had been taught at the school; (b) by whom; (c) to which year groups; and (d) which skills were taught. The health coordinator was also asked to rate the importance of a number of listed factors that might limit the teaching of resuscitation and, in an open-ended question, to list any other barriers that they identified. Finally, we asked how many pupils were enrolled at the school, how many teachers were employed and how many of these held current CPR/first-aid certificates.

The returned surveys were divided into primary (years 1 to 8) and secondary schools (years 9 to 13) for separate analysis. Responses from composite schools (schools with both primary and secondary pupils) were split into primary and secondary school categories according to year.

School decile ratings were obtained from the Ministry of Education. Decile 1 schools are the 10% of schools with the highest proportion of students from low socioeconomic communities, whereas decile 10 schools are the 10% of schools with the lowest proportion of these students. Statistical analysis was performed using Statview 5.0 (Abacus Concepts, USA).

Results

Primary schools (years 1–8: ages 5 to 12) Seven hundred and fifty four of 2205 (34.9%) primary schools completed the survey, and of these 277 taught resuscitation during 2001 (37.5%). Teaching of resuscitation was most likely to occur in schools with larger school rolls ($p = 0.0001$, logistic regression). There was no relationship between a school's decile rating and the teaching of resuscitation (logistic regression).

Of the 277 schools teaching resuscitation, in 104 the resuscitation trainer was a school teacher. External training agencies supplemented, or were used instead of, teachers as

follows: Red Cross (107 schools), Order of St John (57 schools), Royal Life Saving (43 schools), Surf Life Saving (30 schools), other outside agency (34 schools).

Of those schools teaching resuscitation, the health coordinators of 146 (53%) were able to list the skills taught at the school. The health coordinator was less likely ($p < 0.05$) to do this if the teaching had been conducted by an outside agency. The number of schools teaching rescue breathing, adult chest compression and CPR in children are given in Table 1.

Table 1. Resuscitation skills (child CPR, mouth-to-mouth rescue breathing and adult chest compression) taught by year group*

Year	Child CPR	Mouth-to-mouth rescue breathing	Chest compression
1	4	5	5
2	4	5	5
3	5	6	6
4	9	18	14
5	29	55	36
6	42	86	57
7	47	88	60
8	51	93	65
9	10	17	12
10	17	27	22
11	27	24	34
12	70	75	73
13	22	27	28

*146 primary schools and 121 secondary schools answered this section of the survey

The cited barriers to teaching resuscitation in primary schools are given in Tables 2 and 3. The significant barriers identified were (a) the perception that primary children were too young to be taught resuscitation skills; (b) that resuscitation was not a mandatory part of the primary school curriculum; and (c) funding. Seventy primary schools indicated that they taught resuscitation only every second or third year, and did not teach resuscitation during 2001 for that reason. A very full curriculum was noted by 4% of schools, and 4% had never thought of teaching resuscitation.

In 734 primary schools with a total of 7042 teaching staff, 3359 (48%) teachers were identified as holders of first-aid/CPR certificates.

Table 2. Barriers to greater teaching of resuscitation in primary and secondary schools

Barriers	Primary schools*	Secondary schools*	p value [†]
Time restrictions	2.5 (1.3)	3.5 (1.4)	ns
Resuscitation is not mandatory in the school curriculum	2.2 (0.7)	4 (1.4)	0.01
The age groups in our school are not suitable to be taught resuscitation skills	2.0 (1.0)	5.0 (0)	0.001
Funding is inadequate to bring in external trainers	2.5 (1.8)	2.0 (1.4)	ns
Funding is inadequate to train teachers as instructors	2.5 (1.4)	2.0(1.3)	ns
Funding is inadequate for purchase of equipment	2.4 (1.8)	1.5 (0.7)	ns
Few staff interested in teaching resuscitation	3.6 (1.0)	4.0 (1.4)	ns

*mean (Standard Deviation) score on a scale of 1 (greatly limits teaching) to 5 (does not limit teaching at all) for how important these factors were in limiting the extent of resuscitation teaching in 2001;

[†]unpaired t test was used to compare scores for primary and secondary schools, with p <0.05 indicating statistical significance

Table 3. Other barriers to teaching resuscitation cited by health coordinators

Barriers	Primary schools (%)	Secondary schools (%)
Alternating yearly cycle, did not teach in 2001	9	3
The curriculum is too full	4	15
Student ages are inappropriate to teach resuscitation	4	-
School has not ever thought about teaching resuscitation	4	-
Resources are not available	2	3
Resuscitation is not important	1	2
It is difficult to organise resuscitation teaching	1	-
Previous bad experience with external resuscitation instructors	0.6	-
Lack of written resources in the Maori language	0.4	1

Secondary schools (years 9–13: ages 13 to 18) One hundred and seventy three of 456 secondary schools returned the survey (38.6%), and of these 140 taught resuscitation to at least some pupils during 2001 (81%). As with the primary school group, there was a significant positive correlation between the number of students on the school roll and the teaching of resuscitation (p = 0.001, logistic regression). There was no relationship between a school's decile rating and the teaching of resuscitation (logistic regression).

Resuscitation was taught by school teachers at 100 schools, Red Cross at 47, Order of St John at 40, Royal Life Saving at 8 and Surf Life Saving at 10 schools. Other external instructors were used at 17 schools.

Of the 140 responding secondary schools teaching resuscitation during 2001, the health coordinator indicated which skills were taught to each age group in 121 (86%). The health coordinator was less likely (p <0.001) to indicate the skills taught if

resuscitation had been taught by an external agency. The number of schools teaching rescue breathing, adult chest compression and CPR in children are given in Table 1.

Resuscitation was most commonly taught (95 schools) in year 12 (corresponding to age 17 years), where most schools (71%) indicated that they treated it as an optional subject taught to between 10% and 30% of the year group. Only two secondary schools taught resuscitation to a portion of students within each year group, 42% taught resuscitation to students within only one year group, and a further 33% within two year groups. On the basis of the proportion of students taught in each year group, we estimate that 45% of secondary school students are not taught resuscitation, 20% are taught once, 22% twice and 13% more than twice during their five years at secondary school.

The cited barriers to teaching resuscitation in secondary schools are given in Tables 2 and 3. The most important barriers identified by the schools were funding, and a curriculum that was too full.

In 165 secondary schools with a total of 6888 teachers, 1689 (25%) teachers were identified as holders of first-aid/CPR certificates.

Discussion

In New Zealand cities, the likelihood that a victim of out-of-hospital cardiac arrest (OHCA) receives *any* attempt at CPR is approximately 50%; the proportion of these receiving *effective* CPR is not known, but is thought to be approximately 30%. In the remaining 50%, bystanders are either unable or unwilling to provide CPR. With an OHCA survival rate of 5–13% in New Zealand, and a known two- to threefold increase in cardiac arrest survival with bystander CPR, the overall number of lives lost as a result of failure to provide CPR is likely to be significant¹ (personal communication, T Smith, St John, Northern Region, 2002 and P Roberts, Wellington Free Ambulance, 2001). Community CPR skills and education are therefore important issues for public health education.

The school years have the potential to provide guaranteed exposure of future adults to CPR skills. Thereafter, the learning of CPR will involve cost, self-motivation or legislation. A New Zealand adult's exposure to CPR is largely determined by (a) workplace first-aid regulations; (b) voluntary, paid attendance at commercial CPR training courses; and (c) exposure to depictions of CPR in the media (which are infrequent and often inaccurate).

The teaching of resuscitation skills to school children was introduced in Norway as early as 1961. Subsequent international experience has shown that school-age children are more likely to accept CPR training than older people,¹¹ are motivated to learn, and do so quickly and easily.^{5,6,12,13} The European Resuscitation Council, the American Heart Association and the American Academy of Paediatrics have all recommended that resuscitation be taught to all school children.^{6,8,9}

Resuscitation is not a mandatory component of the New Zealand school curriculum, and from the present survey we would estimate that only approximately 55% of secondary school pupils are exposed to CPR teaching during those school years. Students at different schools receive widely disparate exposure and, despite the staged introduction described by the curriculum, current teaching lacks continuity, with CPR being taught, if at all, at the end of both primary and secondary years. This lack of

continuity is coupled with confusion as to *what* should be taught *when*. Further, where training is provided by outside agencies, schools may have little knowledge as to what is actually being taught.

The status of resuscitation in the curriculum means that it is ultimately up to schools to decide whether to allocate funds and manpower to its teaching. The reasons for a school choosing to teach or not to teach CPR are therefore important. The most common barriers to CPR teaching cited by schools relate to funding, appropriateness of teaching to young children, the non-mandatory curriculum, and an overfull curriculum.

Health coordinators noted inadequate funding for training aids, purchase of training from external training agencies, and for training teachers in order to conduct in-house teaching. Although training aids such as manikins are certainly necessary, the most important CPR and first-aid skills can be learnt quickly by people of average intelligence, and do not require extensive training or clinical experience. Given a teacher's educational background, and their ability to adhere to a defined curriculum, it is likely that schools already possess much of the manpower necessary to deliver CPR training. Although external agencies would provide useful additional exposure for students, the presence of skilled and interested teachers would allow skills to be taught and revised whenever timetabling allowed, could provide a role model and would promote a school as being committed to producing caring adults. Several authors have also reported successful use of peer training,¹⁴ where selected older pupils are used to teach younger pupils alongside a fully trained teacher. The advantages of peer training are that it can reduce costs, provide positive role models for younger children and reinforce the skills of the older pupils. Further reductions in cost could also come about from the use of innovative teaching methods such as video training,¹⁵ where students use video instruction coupled with training manikins to learn CPR skills.

An important consideration for primary schools is the suitability of teaching resuscitation to young children of differing age, size and intellectual maturity. For the delivery of effective chest compressions rescuers must be of a suitable size and strength. For New Zealand primary schools there was considerable confusion in this regard. Schools taught a widely disparate range of skills and a number of primary schools reported that instructors from external agencies had informed them that it was inappropriate to teach any resuscitation skills in primary school. In contrast, other schools reported that at least one of these agencies was teaching children as young as five years how to perform chest compressions and expired-air rescue breathing. The literature clearly indicates that children from the age of 10–11 years are capable of learning how to perform CPR^{6,12,13} and, prior to this age, how to access emergency medical services (dial 111) and provide other simple forms of first aid. We believe that these observations indicate that the school curriculum must contain explicit national guidelines on *what* should be taught *when*, rather than leaving this to the interpretation of individual schools or training agencies.

One of the most important problems in resuscitation education is the rapid fall off in skills and knowledge following initial training.¹⁶ For this reason, those within the health professions are often required to repeat CPR tuition, and certification, on an annual basis. Repetition of learning, as well as over-training to a higher than expected level, increases the likelihood of long-term basic skill retention.⁴ The school years

would provide an ideal setting within which to deliver high-quality, structured annual tuition from year 6 onwards.

Since CPR can be taught in training sessions taking little more than one hour, annual training would therefore require five hours of the entire secondary school curriculum. If an overfull curriculum is a significant barrier to teaching CPR, and this truly cannot be accommodated as part of the present curriculum, we would argue that the subject must displace other components. CPR, simple first aid and actions to take at the scene of an accident are critical life skills that may need to be employed at any time, without notice and without reference to books or consultation with others. This is in contrast to the bulk of the curriculum, for which there is no life-threatening urgency requiring the possession of immediate 'off-the-cuff' knowledge.

A limitation of the current study was the low response rate, but this is not unusual for postal surveys. Some recently published reports of postal studies investigating resuscitation teaching show response rates of 43% from European medical schools,¹⁷ and 32% from cardiac-arrest survivors,¹⁸ where it could be expected that these groups would have a high interest in the teaching of resuscitation. We cannot accurately determine whether the non-responders are more or less likely to be teaching resuscitation than those schools that did respond to the survey, and therefore caution must be used in extrapolation of the data in the current study to all schools.

Despite the view of international resuscitation councils that the teaching of resuscitation in schools should be regarded as the primary educational strategy to achieve widespread learning of CPR,⁹ and the *suggested* inclusion of resuscitation in the New Zealand school curriculum, the present study indicates that only 55% of secondary school pupils receive exposure to CPR teaching. In order to achieve widespread, effective community resuscitation knowledge we believe that the teaching of simple emergency care must become a fully funded, mandatory part of the school curriculum provided annually to all children, according to a clearly defined progressive curriculum. Given cost restraints, we believe this can best be accomplished by internal training provided by school teachers, perhaps supplemented by appropriate and experienced external health professional trainers. Simple skills for managing cardiac arrest, road trauma and drowning must be promoted to children as important life skills to be possessed by all responsible adults.

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References:

1. Crone PD. Auckland Ambulance Service cardiac arrest data 1991–3. *NZ Med J* 1995;108:297–9.
2. European Resuscitation Council. Part 5: new guidelines for first aid. *Resus* 2000;46:93–102.

3. Eisenberg MS, Mengert TJ. Cardiac resuscitation. *N Engl J Med* 2001;344:1304–13.
4. Lester CA, Weaton CF, Donnelly PD, et al. The need for wider dissemination of CPR skills: are schools the answer? *Resuscitation* 1994;28:233–7.
5. Goldberg RJ, Gore JM, Love DG, et al. Layperson CPR – are we training the right people? *Ann Emerg Med* 1984;13:701–4.
6. Eisenburger P, Safar P. Life supporting first aid training of the public – review and recommendations. *Resuscitation* 1999;41:3–18.
7. Lewis RM, Fulstow R, Smith GB. The teaching of cardiopulmonary resuscitation in schools in Hampshire. *Resuscitation* 1997;35:27–31.
8. American Academy of Pediatrics Committee on School Health. Basic life support training school. *Pediatrics* 1993;91:158–9.
9. European Resuscitation Council. Part 1: introduction to the international guidelines 2000 for CPR and ECC. *Resuscitation* 2000;46:3–15.
10. Ministry of Education. Health and physical education in the New Zealand curriculum. Wellington: Learning Media; 1999.
11. Lejeune PO, Delooz HH. Why did persons invited to train in cardiopulmonary resuscitation not do so? *Eur Heart J* 1987;8:224–8.
12. Plotnikoff R, Moore PJ. Retention of cardiopulmonary resuscitation knowledge and skills by 11- and 12-year-old children. *Med J Aust* 1989;150:296–302.
13. Corne L, Rydant L, Lauwaert D, Bruynseels P. Teaching cardiopulmonary resuscitation basic life support to school-children. *Acta Anaesthesiol Belg* 1984;35 Suppl:107–13.
14. Lester C, Donnelly P, Weston C. Is peer tutoring beneficial in the context of school resuscitation training? *Health Educ Res* 1997;12:347–54.
15. Todd KH, Braslow A, Brennan RT, et al. Randomized, controlled trial of video self-instruction versus traditional CPR training. *Ann Emerg Med* 1998;31:364–9.
16. Chehardy P, Doherty A, Dracup K, et al. Cardiopulmonary resuscitation and emergency cardiovascular care. Education. *Ann Emerg Med* 2001;37 (4 Suppl):S49–59.
17. Garcia-Barbero M, Caturla-Such J. What are we doing in cardiopulmonary resuscitation training in Europe? An analysis of a survey. *Resuscitation* 1999;41:225–36.
18. Kliegel A, Scheinecker W, Sterz F, et al. The attitudes of cardiac arrest survivors and their family members towards CPR courses. *Resuscitation* 2000;47:147–54.



Current practice for anticoagulation prophylaxis in inguinal hernia surgery: a questionnaire survey

Suhail Anwar and Patrick Scott

Abstract

Aims The incidence of deep vein thrombosis (DVT) and pulmonary embolism (PE) is well documented in patients undergoing surgery involving general anaesthesia. A large number of trials have been conducted establishing the efficacy of prophylactic measures against deep vein thrombosis, yet there remains wide practice variation amongst surgeons regarding the use of anticoagulation measures. The main aims of our study were to survey the use of DVT prophylaxis for inguinal hernia repairs in the UK, and to establish any variations amongst British surgeons in their use of anticoagulation measures for repair of inguinal hernias.

Methods We conducted a questionnaire survey amongst surgeons of the Association of Endoscopic Surgeons of Great Britain and Ireland (AESGBI). Two hundred and fifty questionnaires were sent with a response rate of 72%.

Results Our results have shown wide variation amongst British surgeons in the use of anticoagulation measures. Furthermore, only 10% of the surgeons in the laparoscopic and 14% in the open group risk stratify their patients; 10% of the surgeons do not use any DVT prophylaxis at all.

Conclusions Although the incidence of DVT in inguinal hernia repair is very low this is a very commonly performed procedure. Both over and under treatment with thromboprophylaxis can have implications in terms of side effects and costs. One possible way to avoid problems is to risk stratify patients before thromboprophylaxis is instituted.

Thromboembolic disease has long been recognised as a major cause of post-operative mortality and morbidity. The incidence of deep vein thrombosis (DVT) in patients having general surgical procedures is quoted to be about 25%;¹ these figures, however, have been confounded by the different selection criteria used by various large studies in this field. The THRIFT II consensus group, for instance, quoted a DVT incidence of 10–80% in general, urological and gynaecological patients.^{2,3} Despite the fact that many risk factors for DVT are known and the efficacy of prophylaxis is well established,^{4,5} there are no fixed guidelines for the use of DVT prophylaxis in various general surgical operations, and practice varies from surgeon to surgeon. The inconsistent use of DVT prophylaxis can lead to under or over treatment; the former putting the patient at risk of post-operative thromboembolic disease,¹ and the latter resulting in undesirable side effects,⁶ not to mention cost implications.⁷

Methods

The majority of the surgeons practising laparoscopic hernia repairs in the UK are also involved in open hernia surgery (100% in our study). We therefore used the members of the Association of Endoscopic

Surgeons of Great Britain and Ireland (AESGBI) as a representative sample for our questionnaire survey. This would give us the additional benefit of highlighting any difference in practice of anticoagulation prophylaxis between open and laparoscopic hernia repairs. Figure 1 is the questionnaire that was posted subsequently to 250 consultant surgeons.

Figure 1. Questionnaire survey sent to surgeons of the Association of Endoscopic Surgeons of Great Britain and Ireland (AESGBI)

<p>Q1) Do you carry out laparoscopic repair of inguinal hernias?</p> <p>Q2) Which of the following measures do you undertake to prevent DVTs in a patient having laparoscopic inguinal hernia repair? (Tick one or more) Anticoagulants (heparin/Clexane[®] etc). Please mention dose and timing. Stockings Intermittent pneumatic compression Others</p> <p>Q3) Which of the following measures do you undertake to prevent DVTs in a patient having open inguinal hernia repair? (Tick one or more) Anticoagulants (heparin/Clexane[®] etc). Please mention dose and timing. Stockings Intermittent pneumatic compression Others</p> <p>Q4) What thromboprophylactic measures, if any, do you take for inguinal hernia repairs done under local anaesthetic?</p>
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Results

In total, 250 questionnaires were posted. We received 185 replies out of which three surgeons were already retired and two could not be located at their particular address. We have therefore worked out our calculations from a figure of 180 completed replies (72% response rate).

Ninety nine surgeons (55%) perform laparoscopic repairs for inguinal hernias and all of them do open hernia repairs as well, whereas 81 surgeons (45%) repair hernias by an open technique only. Out of the 99 that do laparoscopic hernias, 75 take identical measures for DVT prophylaxis for laparoscopic and open hernias, whereas 24 differ between prophylaxis for open and laparoscopic surgery. There is no consistent pattern, but out of these 24, 17 surgeons take slightly more measures for their laparoscopic hernias and 7, for their open hernias.

Tables 1 and 2 show the methods used for prophylaxis in laparoscopic and open hernia repairs respectively. As clearly demonstrated by these figures, there is no predominant method used for prophylaxis, instead the distribution is random and seems rather empirical. Ten per cent in the laparoscopic group and 14% in the open group said that the only time they use pharmacological prophylaxis, with or without any additional measures, is when the patient is at a high risk of thromboembolic disease post-operatively. High-risk factors were defined as age over 40 years, previous DVT and obesity.

Table 1. Prophylactic anticoagulation measures for laparoscopic repair of inguinal hernia

Type of prophylaxis	Number of surgeons (%)
Heparin and stockings	22 (22.2)
Stockings and IPC	12 (12.1)
No measures	12 (12.1)
Stockings	12 (12.1)
High risk only*	10 (10.1)
IPC	10 (10.1)
Heparin, stockings and IPC	9 (9.1)
Heparin and IPC	6 (6.1)
Heparin	6 (6.1)

IPC = intermittent pneumatic compression

*these patients received heparin, with or without mechanical prophylaxis, only if stratified in high-risk group (heparin could be unfractionated heparin or low-molecular-weight heparin)

Table 2. Prophylactic anticoagulation measures for open repair of inguinal hernia

Type of prophylaxis	Number of surgeons (%)
Heparin and stockings	35 (19.4)
Stockings	26 (14.4)
High risk only*	25 (13.9)
Heparin, stockings and IPC	25 (13.9)
No measures	19 (10.6)
Stockings and IPC	18 (10.0)
IPC	17 (9.4)
Heparin	9 (5.0)
Heparin and IPC	6 (3.3)

IPC = intermittent pneumatic compression

*these patients received heparin, with or without mechanical prophylaxis, only if stratified in high-risk group (heparin could be unfractionated heparin or low-molecular-weight heparin)

We also included a question about the use of DVT prophylaxis in hernia repairs under local anaesthetic. The results (Table 3) yet again showed no consistent pattern, with different groups practising various techniques. Twelve surgeons do not perform hernia surgery under local anaesthetic. Again 10 (5.6%) use pharmacological prophylaxis only if the patient is in a high-risk group.

As far as the use of heparins was concerned, in the open group a total of 100 surgeons use heparins with or without additional measures, out of which 44 use low-dose unfractionated heparins (UFH) and 56 use low-molecular-weight heparins (LMWH). These figures were nearly the same for the laparoscopic hernia repairs.

Table 3. Prophylactic anticoagulation measures for open repair of inguinal hernia performed under local anaesthetic

Type of prophylaxis	Number of surgeons (%)
No measures	46 (25.6)
Stockings	31 (17.2)
Heparin and stockings	24 (13.3)
No reply	14 (7.8)
Heparin, stockings and IPC	12 (6.7)
Do not perform surgery under local anaesthetic	12 (6.7)
Stockings and IPC	11 (6.1)
High risk only*	10 (5.6)
IPC	8 (4.4)
Heparin	8 (4.4)
Heparin and IPC	4 (2.2)

IPC = intermittent pneumatic compression

*these patients received heparin, with or without mechanical prophylaxis, only if stratified in high-risk group (heparin could be unfractionated heparin or low-molecular-weight heparin)

Discussion

Surveys conducted in the United States⁸ and England⁹ have shown wide practice variation in the use of DVT prophylaxis. In 1993 the estimated cost to the National Health Service of DVT and PE was over £200 million. If all the patients at high risk of developing post-surgical DVT had received prophylaxis, the NHS would have saved between £30 million and £80 million.¹⁰ On the other hand, use of heparin (UFH and LMWH) has been reported to be associated with wound complications and haematomas,^{6,11,12} resulting in morbidity with its accompanying financial implications. The rational application of DVT prophylaxis demands knowledge of risk factors. All surgical patients admitted to the hospital should be assessed for their risk of DVT with respect to their medical history, clinical signs, existing conditions and the result of blood tests. They should then be categorised according to their level of risk and appropriate prophylaxis given.

The incidence of DVT and PE after inguinal hernia repair is very low. Dudda and Schunk have quoted a rate of 0.9% for PE and 0.7% for DVT in their series of 1202 inguinal hernia operations.¹³ Kark et al reported one case of DVT in a review of 1098 hernia repairs under local anaesthetic without any prophylaxis.¹⁴ Begin has reported one case of DVT from a series of 200 laparoscopic, extraperitoneal inguinal hernia repairs in France.¹⁵ Kopanski et al have shown a statistically significant difference in the incidence of thrombotic complications after laparoscopic and open operations with the former being much lower than the latter.¹⁶ Finally, there is evidence to support the preferred use of LMWH over UFH in general surgery with respect to better prophylactic efficacy, once-daily injections and cost-saving implications,^{17,18} but even this topic is subject to debate and controversy.

Our study is a very simple one, highlighting the variance and inconsistency in the use of anticoagulation measures amongst British surgeons. Although most surgeons are aware of the associated risks and are using some form of prophylaxis, the pattern is random and inconsistent.

The wide variation shown in our survey reflects the lack of research on which to base good practice. Some might argue that, due to the very low rate of DVT in inguinal hernia repair, 'type' of prophylactic measure is not important. We, however, point out that this is a very commonly performed procedure and lack of risk stratification before instituting prophylaxis can potentially cause over or under treatment of patients with its associated cost implications.

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References:

1. Clagett GP, Reisch JS. Prevention of venous thromboembolism in general surgical patients. Results of meta-analysis. *Ann Surg* 1988;208:227-40.
2. Second Thromboembolic Risk Factors (THRIFT II) Consensus Group. Risk of and prophylaxis for venous thromboembolism in hospital patients. *Phlebology* 1998;13: 87-97.
3. Thromboembolic Risk Factors (THRIFT) Consensus Group. Risk of and prophylaxis for venous thromboembolism in hospital patients. *Br Med J* 1992; 305:567-74.
4. Collins R, Scrimgeour A, Yusuf S, Peto R. Reduction in fatal pulmonary embolism and venous thrombosis by perioperative administration of subcutaneous heparin. Overview of results of randomized controlled trials in general, orthopedic and urologic surgery. *N Engl J Med* 1988;318:1162-73.
5. Prevention of fatal postoperative pulmonary embolism by low doses of heparin. An international multicentre trial. *Lancet* 1975;2:45-51.
6. Beerlage HP, de la Rosette JJ, Frima AJ. Association between haematoma after inguinal hernia repair and site of heparin injection. *Eur J Surg* 1992;158:287-8.
7. Oster G, Tuden RL, Colditz GA. A cost-effective analysis of prophylaxis against deep-vein thrombosis in major orthopedic surgery. *JAMA* 1987;257:203-8.
8. Conti S, Daschbach M. Venous thromboembolism prophylaxis: a survey of its use in the United States. *Arch Surg* 1982;117:1036-40.
9. Morris GK. Prevention of venous thromboembolism. A survey of methods used by orthopaedic and general surgeons. *Lancet* 1980;2:572-4.
10. Office of Health Economics/Health Economic Evaluation Database. Publication briefing, October 1996. Web site: <http://www.oh-eheed.com/Default.htm> Accessed September 2003.
11. van Ooijen B. Subcutaneous heparin and postoperative wound hematomas. A prospective, double-blind, randomized study. *Arch Surg* 1986;121:937-40.
12. Goris RJ, Van Dongen LM, Deleu HW, Doesburg DW. Low dose subcutaneous heparin and wound hematoma formation in inguinal hernia operations – a double-blind randomized clinical trial. *Neth J Surg* 1981;33:19-22.
13. Dudda W, Schunk R. Lotheissen-McVay repair of hernia. Late follow-up analysis after 1202 operations for inguinal and femoral hernias. *Langenbecks Arch Chir* 1990;375:351-8.
14. Kark AE, Kurzer M, Waters KJ. Tension-free mesh hernia repair: review of 1098 cases using local anaesthesia in a day unit. *Ann R Coll Surg Engl* 1995;77:299-304.
15. Begin GF. Laparoscopic extraperitoneal treatment of inguinal hernias in adults. A series of 200 cases. *Endosc Surg Allied Technol* 1993;1:204-6.

16. Kopanski Z, Cienciala A, Ulatowski Z, Micherdzinski J. Comparison of thrombosis rate after laparoscopic and conventional interventions with the I(125) fibrinogen test. *Wien Klin Wochenschr* 1996;108:105–10.
17. Jorgensen LN, Wille-Jorgensen P, Hauch O. Prophylaxis of postoperative thromboembolism with low molecular weight heparins. *Br J Surg* 1993;80:689–704.
18. Haas S. European consensus statement on the prevention of venous thromboembolism. European Consensus Conference, Windsor, UK, November, 1991. *Blood Coagul Fibrinolysis* 1993;4 Suppl 1:S5–8.



Correction of deformational auricular anomalies by moulding – results of a fast-track service

Swee Tan, Anna Wright, Anna Hemphill, Kari Ashton and Joan Evans

Abstract

Aim To report on the result of a fast-track referral service in treating deformational auricular anomalies using moulding therapy, by employing nurses who were familiar with the indications and technique, working in close liaison with plastic surgeons.

Methods A deformational auricular anomaly is defined as an ear having normal chondrocutaneous components but an abnormal architecture; therefore, it can be manipulated digitally to a normal shape. Having demonstrated the value of auricular moulding therapy to our neonatal practitioners, we established a fast-track referral and treatment protocol for infants with deformational auricular anomalies. Treatment was initiated promptly by one of four nurses. The type and severity of the auricular anomaly were documented both clinically and photographically before and three months following cessation of treatment. Assessment of the results was made by comparing the pre- and post-treatment photographs and by a postal questionnaire, which was dispatched to the parents of the patients three months after treatment was discontinued.

Results Sixteen male and 14 female patients, aged between one day and 15 weeks (mean 24 days) with 44 deformational auricular anomalies, underwent moulding therapy. Complete correction or marked improvement was achieved in 26 patients (87%) with 38 ears (86%) while slight or no improvement occurred in 4 patients (6 ears), following one to 14 (mean 7) weeks of moulding. Questionnaires were returned by the parents of 24 patients (80%). According to the parents' assessment, complete correction or marked improvement occurred in 29 of 35 anomalous ears (83%) in 20 of these 24 patients (83%). All parents felt that auricular moulding was worthwhile.

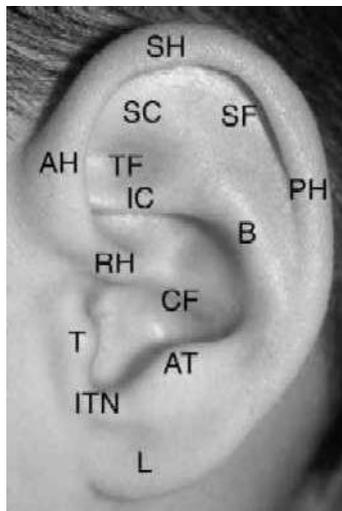
Conclusions Deformational auricular anomalies should be treated non-surgically with moulding therapy. For this treatment to be effective, it should be initiated in the first three months of life. Parental persistence with the treatment is essential for a satisfactory outcome. A fast-track referral service, employing nurses who form the first point of contact and work in close association with a plastic surgery service, is an effective treatment strategy that will largely negate the need for surgical correction of deformational auricular anomalies.

The ear or the auricle consists of a complex, convoluted, yellow elastic cartilage framework that is covered with hairless skin. The convoluted prominences and concavities of the underlying cartilage give rise to the characteristic topographic anatomy of the human ear (Figure 1).¹

The helix is the most external portion of the auricle. It consists of the root, anterior, superior and posterior portions. The lobule consists of fibro-fatty tissue and does not contain cartilage. The antihelix, which separates the concha from the helix, is a Y-shaped structure consisting of a body inferiorly and two crura superiorly. The body of

the antihelix blends into the antitragus, which is sited superior to the lobule. The antitragus is separated from the tragus by the intertragic notch. The tragus is located anterior and lateral to the external auditory canal, thus partly obscuring it. The scaphoid fossa lies between the superior crus and the body of the antihelix anteriorly and the helix posteriorly. The crura of the antihelix and anterior part of the helix form the boundaries of the triangular fossa.¹

Figure 1. The topographic anatomy of the human ear (RH = root of the helix; CF = conchal fossa; AH = anterior portion of the helix; IC = inferior crus of the antihelix; SC = superior crus of the antihelix; SH = superior portion of the helix; PH = posterior portion of the helix; L = lobule; B = body of the antihelix; AT = antitragus; T = tragus; ITN = intertragic notch; SF = scaphoid fossa; TF = triangular fossa)



There are at least 40 descriptive and eponymous terms used to categorise congenital auricular anomalies.¹ This heterogeneous group of anomalies should be classified as either malformational (eg, auricular tags and sinuses, anotia, microtia and cleft ear) or deformational (eg, lop, cup, Stahl's, kinked and prominent ear).^{1,2} Malformations are structural abnormalities that result from abnormal embryological development. Deformation is caused by abnormal physical forces applied to a normal structure, which may occur in utero or following birth. Similarly, a malformed ear may be subjected to deformational forces.¹ The majority of congenital auricular anomalies are deformational in nature.¹

A deformational auricular anomaly can be simply defined as an ear having normal chondrocutaneous components but with an abnormal architecture; therefore, it can be manipulated digitally to a normal shape.^{1,2} These auricular anomalies usually affect the helix and antihelix, although occasionally the deformation is confined to the conchal fossa.²

Conventionally, both malformational and deformational auricular anomalies are corrected surgically when the ear has adequately grown,² although with variable

results^{3,4} and significant complication rates.² Moulding therapy is effective for deformational^{2,5-8} and certain malformational⁹ auricular anomalies. Although this simple, effective and inexpensive technique has been available for over 20 years, its wider acceptance in Western countries has occurred only relatively recently.^{7,8}

For a satisfactory outcome, moulding therapy should be initiated within the first three months of life.^{2,7} However, children with congenital auricular anomalies are normally referred 'routinely' to the plastic surgery service and the narrow 'window of golden opportunity' for moulding is often missed. Having demonstrated the value of auricular moulding therapy to our neonatal paediatricians, family doctors, and midwives, we have established a fast-track referral and treatment protocol for infants with deformational auricular anomalies. This report presents, prospectively, the results of our approach.

Methods

Protocol The protocol aimed at identifying newborn infants with deformational auricular anomalies with a view to early correction with moulding therapy. These patients were referred by our neonatal paediatricians, general practitioners, and community midwives through a fast-track referral service established at the Department of Plastic & Reconstructive Surgery, Radcliffe Infirmary, Oxford, England and the Wellington Regional Plastic, Maxillofacial & Burns Unit, Wellington, New Zealand. During the study period, several children with malformational auricular anomalies were referred for treatment. These children were excluded from this study.

The obstetric, perinatal and family history and associated anomalies, if present, were recorded and managed appropriately. The type and severity of the auricular anomaly were documented both clinically and photographically. Moulding therapy was offered to these children and was initiated promptly by one of four nurses who were familiar with the indications and technique of this treatment.

Technique Various materials have been used for auricular moulding.^{2,5,7-10} We utilised a simple splint fabricated by inserting a piece of lead-free solder (Multicore Solders Ltd, UK) within an 8 French polyethylene suction catheter (Figure 2).^{2,7} The splint was customised for each ear with its length determined by the extent of the deformity. It was applied to the eave of the helical rim and secured to the ear with Steri-Strips (3M, St Paul, MN) to maintain the ear in the corrected position (Figure 3). In addition, the ear was taped to the mastoid scalp if it was protruding (Figure 4). The parents of the infants under treatment were taught the moulding technique and were given an information sheet outlining the treatment protocol. They were instructed to inspect and reshape the splint and replace the adhesive tapes if necessary after each feed. The splint was replaced weekly and the parents were asked to observe for skin irritation or ulceration. Treatment was maintained until satisfactory correction was achieved and continued for a further week, or discontinued if no improvement was observed after four weeks of continuous moulding.

The infant was reviewed by a nurse weekly for two weeks, at one month and then monthly for up to three months. The nurses provided the point of contact for the parents and arranged for a routine plastic surgical review whenever necessary.

Figure 2. A simple splint consisting of a lead-free soldering wire and an 8 French suction catheter. The length of this device is dependent on the extent of the auricular deformity being corrected, a short splint being used to correct a localised deformity, and a longer splint required for a more extensive anomaly.

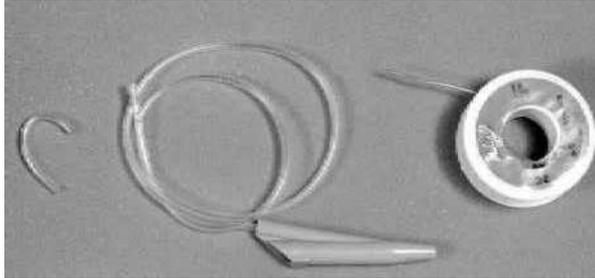


Figure 3. Bilateral cup ear anomaly in a one-week-old boy. (A) The more severely affected right ear was corrected (B) with a long splint. (C) Improvement after three months of moulding (result = ‘marked improvement’).

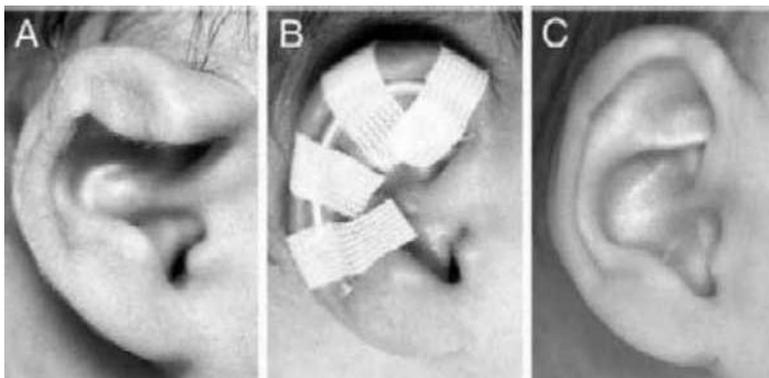
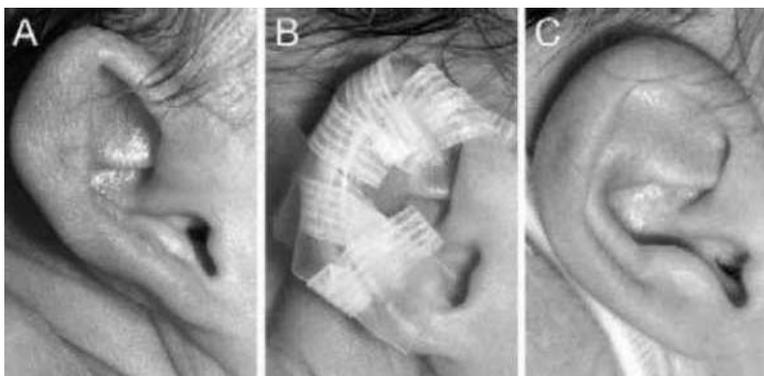


Figure 4. (A) Right-sided prominent ear with absent antihelical fold in a two-week-old child born five weeks prematurely (B) treated with a splint and taping of the ear to the mastoid scalp. (C) Excellent result (with creation of antihelical fold and reduction of distance between the helical rim and scalp from 3.0 to 1.2 cm) after 16 weeks of treatment (result = ‘marked improvement’).



Assessment Clinical and photographic documentation of the auricular anomaly was obtained prior to the commencement of treatment and three months following completion of moulding therapy. The result of treatment was assessed by comparing pre- and post-treatment photographs and graded as: 1 = complete correction; 2 = marked improvement; 3 = slight or no improvement. A postal questionnaire was also dispatched to the parents of the patients three months following cessation of therapy. The parents were asked to score the results using the grading described above. They were also asked to indicate whether the moulding technique was 'easy', 'difficult', or 'very difficult'. To help us evaluate parents' satisfaction with the therapy, they were asked if the treatment was worthwhile and whether they would recommend it to other children with a similar auricular anomaly.

Results

Thirty consecutive children (16 boys and 14 girls) with 44 deformational auricular anomalies were the subject of this report. The auricular anomalies affected the right side, left side and both sides in 9, 7 and 14 children respectively. There were 17 lop ears (11 patients), 14 prominent ears (10 patients), 8 cup ears (6 patients), and 5 kinked ears (3 patients).

Nineteen children were full term, 2 were one week overdue and 9 children were born prematurely (11, 5, 3 and 1 weeks premature in 1, 3, 2 and 3 infants respectively). Ten children had associated obstetric problems (mild gestational hypertension in 4, oligohydramnios in 1, gestational diabetes in 1, breech delivery in 2, and birth by Caesarean section in 2). Associated anomalies included distal hypospadias in one child and respiratory distress syndrome in another. Five of the 10 children with prominent ear(s) had a positive family history of the anomaly.

Moulding therapy was initiated between one day and 15 weeks (mean 24 days) after birth and treatment was maintained for one to 14 (mean 7) weeks. The children were followed up for 5 to 11 (mean 8) months. An excellent result was achieved in 13 children (19 ears) and marked improvement occurred in 13 children (19 ears) (Figures 3–6). Slight or no improvement occurred in 4 patients (6 ears). In the last group, treatment was interrupted by systemic illness in one child and was associated with poor compliance in the remainder. In two of these patients, treatment was initiated at 15 and 10 weeks respectively and these children removed the splints repeatedly.

Figure 5. (A) Left-sided lop ear in a three-day-old child moulded for three weeks (B) with improvement (result = 'marked improvement').

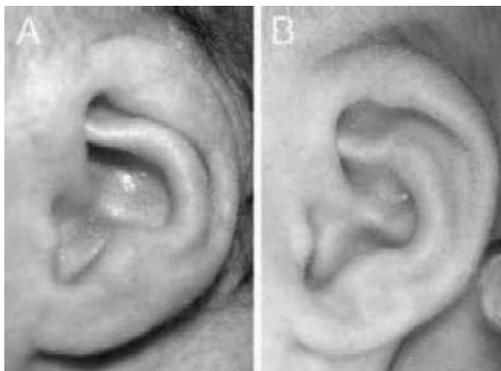
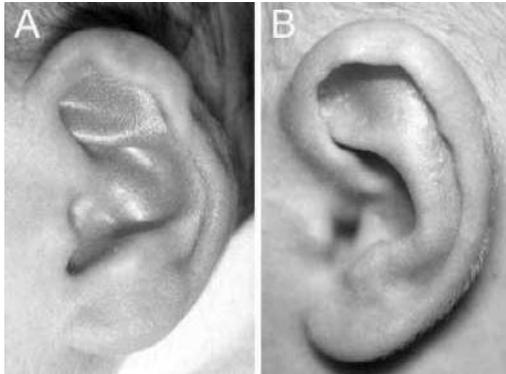


Figure 6. (A) The more severely affected left side of a three-day-old child with bilateral kinked ear deformity splinted for three weeks (B) with improvement (result = ‘marked improvement’).



Complications included skin irritation in 4 children (4 ears) requiring temporary cessation of moulding therapy. No skin ulceration occurred and no relapse of the anomaly was observed during the follow-up period.

Twenty four of the 30 questionnaires (80%) were returned by the parents of the children treated. According to the parents' assessment, 'excellent result' was achieved in 10 patients (14 ears), 'marked improvement' occurred in 10 patients (15 ears) and 'slight or no improvement' in 4 patients (6 ears). The parents of 19 children felt that the moulding technique was 'easy'; 4 found it 'difficult' while one indicated that the technique was 'very difficult'. All the parents of these 24 children felt that moulding therapy was worthwhile and would recommended the treatment for other children with similar auricular anomalies.

Discussion

Splinting has been successfully used for correction of congenital dislocation of the hip,⁷ club foot,¹¹ and cleft lip nasal deformity.¹² The neonatal auricular cartilage is very pliable and lacks elasticity immediately after birth. However, within a few days the ears become more elastic and firm,¹⁰ a fact that has been attributed to a decreasing circulating (maternal) oestrogen level in the neonate.¹³ The levels of circulating free oestradiol are highest during the first 72 hours after birth and decrease rapidly thereafter, reaching the levels similar to those of older children by six weeks of age.¹⁴ Cartilage elasticity is dependent upon its proteoglycan concentration.¹⁵ Hyaluronic acid, an important constituent, is increased by oestrogen and is responsible for the malleable nature of the neonatal ear.¹⁶ It is during this earlier neonatal period that deformational and certain malformational auricular anomalies can be moulded, so early treatment is critical for success.^{2,7,10,12,16} We have noted that children who are breast-fed require an extended period of treatment, due to the ear cartilage remaining pliable for longer,² presumably because of persistent elevated levels of oestrogen.

Although pleasing results can be obtained with surgical correction of congenital auricular anomalies, significant complications and morbidity may occur including pain and emotional trauma associated with the anomaly and its surgical correction.⁷ Many auricular deformities (such as lop ear, Stahl's ear, cryptotia, and kinked ear) are

difficult to correct surgically, and results are often disappointing.⁷ Auricular moulding is the treatment of choice for deformational auricular anomalies.¹ However, the narrow window of opportunity for treatment is often missed because of late referral and hence this technique has not become widely accepted in Western countries.⁷

One of the reasons for late referral may be the impression that auricular anomalies in neonates correct spontaneously with age. Although there has been a Japanese longitudinal study showing that the incidence of some auricular anomalies decreases whilst that of some others increases with age, it was not possible to predict which of the anomalies would resolve spontaneously.⁶ It is also not clear if very minor anomalies were included in the study. The authors of the present study have not observed complete spontaneous correction of auricular anomalies amongst the cohort of patients referred to the service. With our current knowledge, it seems reasonable to carry out non-surgical correction on all deformities that are significant and treatable.⁷

Parental persistence with the treatment is essential for a satisfactory outcome as shown in our series. Good results can be obtained, often within a short period if moulding is initiated soon after birth.^{2,6-8,10} However, a longer duration of treatment is required for more complex anomalies or if the treatment is delayed.^{2,6-8,10}

Although auricular moulding has been shown to be effective until up to six months of age in the one Japanese study,⁶ our experience generally shows that it is largely ineffective if initiated after three months of age.⁷ This is partly related to the less pliable nature of the auricular cartilage in older children and also partly because it is more difficult to apply and maintain the splint adequately in these children because of poor cooperation, as seen in two of our patients.

Moulding therapy can also be useful in certain malformational auricular anomalies that have been subjected to deformational forces,¹ although the treatment is unlikely to completely correct the anomaly. Nevertheless, it forms a useful adjunct that may minimise the extent of future surgery.

Different materials have been used to achieve auricular moulding.^{2,5,7-10} The simple device used in this study is malleable and its large calibre reduces the risk of pressure necrosis. Skin irritation occurred in four children requiring temporary cessation of moulding. Once taught, the parents were happy to replace the splint and the majority of the parents managed the technique satisfactorily.

Congenital auricular anomalies may be associated with other abnormalities.⁷ The auricular anomaly should not be treated in isolation and any associated anomalies should be evaluated and managed appropriately.

Auricular moulding is simple, non-invasive, effective, and it can be done without anaesthetic and at low cost. Deformational auricular anomaly should be treated non-surgically during the early neonatal period, long before the child becomes aware of the anomaly. It is hoped that the emotional disturbance¹⁷ that may occur in these children and the need for surgical correction can be largely avoided in the future.

Deformational auricular anomalies are not a surgical problem but rather a paediatric public health issue.¹ Our neonatal paediatricians, obstetricians, family doctors and midwives should be encouraged to manage these anomalies so that the use of this technique will become widespread.¹

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References:

1. Porter CJW, Tan ST. A classification of congenital auricular anomalies based on embryology: implications in treatment strategies. (Submitted).
2. Tan ST, Abramson DL, MacDonald DM, Mulliken JB. Molding therapy for infants with deformational auricular anomalies. *Ann Plast Surg* 1997;38:263–8.
3. Noguchi M, Matsuo K, Imai Y, Furuta S. Simple surgical correction of Stahl's ear. *Br J Plast Surg* 1994;47:570–2.
4. Calder JC, Naasan A. Morbidity of otoplasty: review of 562 consecutive cases. *Br J Plast Surg* 1994;47:170–4.
5. Nakajima T, Yoshimura Y, Kami T. Surgical and conservative repair of Stahl's ear. *Aesthetic Plast Surg* 1984;8:101–7.
6. Matsuo K, Hayashi R, Kiyono M, et al. Nonsurgical correction of congenital auricular deformities. *Clin Plast Surg* 1990;17:383–95.
7. Tan ST, Shibu M, Gault DT. A splint for correction of congenital ear deformities. *Br J Plast Surg* 1994;47:575–8.
8. Ullmann Y, Blazer S, Ramon Y, et al. Early nonsurgical correction of congenital auricular deformities. *Plast Reconstr Surg* 2002;109:907–15.
9. Matsuo K, Hirose T. A splint for non surgical correction of cryptotia. *Eur J Plast Surg* 1989;12:187–8.
10. Matsuo K, Hirose T, Tomono T, et al. Nonsurgical correction of congenital auricular deformities in the early neonate: a preliminary report. *Plast Reconstr Surg* 1984;73:38–51.
11. Kite JH. Some suggestions on the treatment of club foot by casts. *J Bone Joint Surg* 1963;45A:406–12.
12. Matsuo K, Hirose T. Preoperative non-surgical over-correction of cleft lip nasal deformity. *Br J Plast Surg* 1991;44:5–11.
13. Kenny FM, Angsusingha K, Stinson D, Hotchkiss J. Unconjugated estrogens in the perinatal period. *Pediatr Res* 1973;7:826–31.
14. Hung W, August GP, Glasgow AM. The ovary. In: Hung W, August GP, Glasgow AM, editors. *Pediatric endocrinology*. New York: Medical Examination Publishing Co; 1978.
15. Fry H, Robertson W. Interlocked stresses in cartilage. *Nature* 1967;215:53–4.
16. Sliff M, Burn HF. The effect of intravenous oestrogens on ground substance. *Arch Otolaryngol* 1961;73:43–5.
17. Bradbury ET, Hewison J, Timmons MJ. Psychological and social outcome of prominent ear correction in children. *Br J Plast Surg* 1992;45:97–100.



Spontaneous coronary artery dissection: a report of two cases occurring during menstruation

Robert Slight, Ali Asgar Behranwala, Onyekwelu Nzewi, Rajesh Sivaprakasam, Edward Brackenbury and Pankaj Mankad

Since its first description by Pretty in 1931, many reports of spontaneous coronary artery dissection (SCAD) have been published.¹ Despite this, SCAD remains a rarity.^{1,2} The condition carries a high mortality, with the diagnosis of many cases being made post-mortem.¹ Eighty per cent of cases of SCAD occur in women, with a third of these occurring late in pregnancy or in the early puerperal period.² The majority of women who present with SCAD are pre-menopausal with a mean age of 35 to 40 years.²

We report two cases of SCAD that presented at our institution. In both instances no cardiac risk factors were identified and angiography did not demonstrate any evidence of coronary artery disease. An important observation was the menstrual status of both patients. We propose a potential pathogenesis for SCAD in pre-menopausal women.

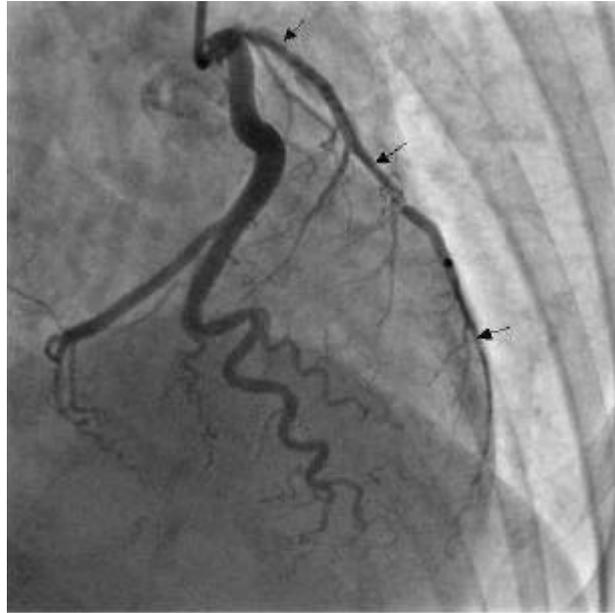
Case reports

Case 1

A 49-year-old female GP was admitted with a one-hour history of central, crushing chest pain. There was no history of cardiovascular disease or identifiable coronary risk factors. The patient was noted to be at the beginning of her menstrual cycle. ECG on admission revealed antero-lateral ischaemic changes and the patient subsequently underwent thrombolysis with streptokinase. Due to the persistence of chest pain and the ECG changes, urgent angiography was performed, which demonstrated dissection of the proximal two thirds of the left anterior descending (LAD) artery (Figure 1). No additional coronary disease was identified. Surgical intervention was felt to be the most appropriate course of action in order to preserve a large first diagonal branch. The patient was haemodynamically stable prior to surgery.

The presence of intra-mural haematoma, compressing the lumen of the LAD, was confirmed at operation. Two saphenous-vein coronary artery bypass grafts (CABG) were performed to the LAD and the first diagonal branch with the aid of standard cardiopulmonary bypass (CPB). Elective intra-aortic balloon pump (IABP) support was used to discontinue CPB. Re-exploration was performed on the first post-operative day for continuing blood loss and a vein side branch was ligated. Echocardiography on the fourth post-operative day demonstrated antero-apical akinesis with normal valvular function consistent with a trans-mural myocardial infarction. Further recovery was uneventful and the patient was well enough to be discharged home on the eighth post-operative day.

Figure 1. Left coronary angiography displaying diffusely narrowed LAD (arrows). Dissection was confirmed by the presence of intra-mural haematoma at the time of operation.



Case 2

A 43-year-old female nurse was referred for urgent CABG. She had experienced increasingly severe central chest pain throughout the previous day. There was no history of coronary artery disease or identifiable coronary risk factors. The patient was at the beginning of her menstrual cycle. ECG on admission demonstrated lateral ischaemic changes in keeping with a diagnosis of acute myocardial infarction. Despite thrombolysis, the patient's condition deteriorated and she became hypotensive and oliguric. Echocardiography showed postero-lateral akinesis. Urgent angiography was performed and revealed a left main stem dissection that was occluded with thrombus (Figure 2).

On arrival at our unit the patient was in established cardiogenic shock despite IABP support. Emergency saphenous vein grafts were performed to the LAD and the obtuse marginal arteries with the aid of CPB. Re-exploration was performed on the day of operation for excessive blood loss. Initially, reasonable pressures were maintained, although overnight, despite maximal inotropic and mechanical support, the patient's pressures began to fall and a low cardiac output state developed resulting in peripheral ischaemia and evolving renal failure. The situation was irretrievable and the patient died on the first post-operative day. No post-mortem examination was performed.

Figure 2. Left coronary angiography with visible dissection flap within the lumen of the left main stem (arrows)



Discussion

In order to arrive at a diagnosis of primary or spontaneous coronary artery dissection, several potential causes must be excluded. These include Marfan's syndrome and other connective tissue disorders, chest trauma, vasculitis and traumatic cardiac catheterisation.^{1,2} The majority of cases of SCAD are found in patients with underlying atherosclerotic coronary artery disease, in post-partum females, or have no obvious cause.²

The two cases reported raise interest in that both women were found to be menstruating at the time of spontaneous dissection. During menstruation the circulating levels of oestrogen and progesterone are known to be at their lowest. This raises the possibility of hormonally mediated changes in the wall of the coronary arteries.^{1,2} A high incidence of SCAD is also reported under the similar hormonal conditions encountered during the post-partum period.² Recent work has focussed on arterial flow patterns at different stages of the menstrual cycle.³ Coronary ischaemia is more likely to present at a time when female circulating sex hormones are low.³ The suppressive effects of female sex hormones, in particular oestrogen, on vascular smooth muscle cell activity are well described.⁴

Histopathological changes identified in the spontaneous dissection of muscular arteries include smooth muscle cell proliferation and vacuolar degeneration, fibrosis, collagen degeneration, elastin fragmentation and peri-vascular inflammatory infiltrate.^{1,2,5} It has been suggested that these changes occur in relation to an increase in vascular smooth muscle cell activity.⁵ SCAD in post-partum and pre-menopausal women occurs at a time when circulating sex hormones have dropped to a low level

after a peak in serum concentration. We suggest that the loss of hormonal vascular smooth muscle cell suppression at the time of menstruation, as encountered in our patients, may lead to an increase in smooth muscle activity with resultant weakness in the coronary media. No cyclical change in the presentation of spontaneous dissection affecting the remainder of the arterial tree has been identified in the literature. This selectivity for the coronary vasculature may relate, in part, to the additional shearing forces placed on the coronary vessels during myocardial contraction.

Primary dissection is thought to occur due to haemorrhage into the outer media of the vessel resulting in compression of the true vessel lumen. This finding may or may not be accompanied by the visualisation of an intimal flap.^{1,2} In cases where this is absent it has been theorised that bleeding from the vasa vasorum may be responsible.^{1,2} An intimal flap was visualised in Case 2 (Figure 2); however, it is more likely that Case 1 (Figure 1) was attributable to bleeding from the vasa vasorum.

SCAD is a rare although life-threatening event. We believe that a direct hormonal link outwith pregnancy could exist in pre-menopausal women for SCAD. We feel that in patients presenting with symptoms of ischaemic heart disease during menstruation the diagnosis of SCAD should be considered and early angiography instituted.

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References:

1. Jorgensen MB, Aharonian V, Mansukhani P, Mahrer PR. Spontaneous coronary dissection: a cluster of cases with this rare finding. *Am Heart J* 1994;127:1382–7.
2. Dhawan R, Singh G, Fesniak H. Spontaneous coronary artery dissection: the clinical spectrum. *Angiology* 2002;53:89–93.
3. Charney P. Coronary artery disease in young women: the menstrual cycle and other risk factors. *Ann Intern Med* 2001;135:1002–4.
4. Okubo T, Urabe M, Tsuchiya H, et al. Effect of oestrogen and progesterone on gene expression of growth regulatory molecules and proto-oncogene in vascular smooth muscle cells. *Endocr J* 2000;47:205–14.
5. Hartman JD, Eftychiadis AS. Medial smooth-muscle cell lesions and dissection of the aorta and muscular arteries. *Arch Pathol Lab Med* 1990;114:50–61.

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Drotrecogin alfa (recombinant human activated protein C) in severe sepsis – a New Zealand viewpoint

Janet Liang, Stephen Streat, John Torrance, Jamie Sleigh, Ross Freebairn and Mace Ramsay

Abstract

Aim Sepsis is a serious and increasing worldwide intensive care problem. In response to intensivists' concerns over the benefits, risks and financial implications of the use of drotrecogin alfa (recombinant human activated protein C), the first adjunctive therapy for sepsis licensed in New Zealand, the New Zealand Region of the Australian and New Zealand Intensive Care Society (ANZICS) requested an advisory statement from a working party of New Zealand intensivists.

Methods We reviewed (a) the PROWESS study of drotrecogin alfa recombinant; (b) the submission made by the sponsoring company to the FDA; (c) recent discussions and an economic evaluation of the use of the agent; (d) Australian pharmaceutical benefits scheme positive recommendations; (e) guidelines produced by the Eli Lilly Australian Advisory Board; (f) Australian hospital decisions on availability; and (g) New Zealand pricing and payment arrangements. We then formulated suggested New Zealand guidelines.

Results We recommend that hospital pharmacy and therapeutics committees review the agent now. If the agent is made available for use, we recommend that: (a) specialists prescribing the agent be required to contribute clinical data to a national register of patients, (b) patients considered for treatment should first be treated with all appropriate surgical and medical therapy for severe sepsis with high illness severity in an intensive care unit by an intensivist; (c) patients considered for treatment should not have severe comorbidity or predetermined treatment limitations or contraindications to the agent in the original phase III study; and (d) patients should be considered only if seen to be not clearly improving after a six-hour period of intensive treatment. Once a decision has been made to treat with drotrecogin alfa, treatment should commence as soon as possible and within 24 hours of meeting criteria. Although the agent is recommended for use in adults, it may be reasonable to treat some older children.

Conclusions Despite high cost and moderate benefit, it may be reasonable to treat highly selected New Zealand patients with drotrecogin alfa.

Sepsis is a serious intensive care problem worldwide.¹ The incidence of sepsis in intensive care units is reported to be increasing² and New Zealand experience is in keeping with this.³ Most recent intensive care unit (ICU) sepsis studies report mortality of around 25–50% but many factors, including the extent of comorbidity, the nature and site of infection, adequacy of surgical and antimicrobial therapy, and the severity of the acute illness, influence the outcome.¹ Sepsis was the reason for ICU admission in 349 of all 1404 ICU deaths (25%) in New Zealand ICUs in one year.⁴

The accepted general principles of the treatment of severe sepsis are to support oxygen transport,⁵ to identify and if possible remove the septic source,^{6,7} and to provide appropriate antimicrobial therapy.^{3,8-11} A firm consensus on the place of adjunctive therapies is not yet established despite considerable research and recent promising reports, including the PROWESS study of drotrecogin alfa (recombinant human activated protein C).¹² This agent (XigrisTM, Eli Lilly) is the first adjunctive therapy for sepsis to be licensed in many countries including the US, UK, Europe, Australia and, most recently, New Zealand. The agent is expensive and intensivists have expressed concerns about the benefits, risks, and financial implications of its use. These concerns led the New Zealand Region of the Australian and New Zealand Intensive Care Society to request an advisory statement on the agent from a working party of New Zealand intensivists – the authors of this paper.

Methods

We reviewed the original (PROWESS) study of drotrecogin alfa recombinant in sepsis,¹² the submission made by the sponsoring company to the (US) FDA,¹³ more recent discussions in the *New England Journal of Medicine*,¹⁴⁻¹⁶ a Canadian economic evaluation of the use of the agent,¹⁷ Australian pharmaceutical benefits scheme positive recommendations,¹⁸ guidelines for the use of drotrecogin alfa in sepsis produced by the Eli Lilly Australian Advisory Board (personal communication, M Fisher, 2002), positive (personal communications, M O'Leary, J Reeves, Y Shehabi, G Dobb, G Skowronski, R Herkes, J Lipman, D Stephens, JW Mulder, D Cook, 2002), negative (personal communications, M Fisher, JF Cade, P Harrigan, J Santamaria, 200) and still pending (personal communications, B Richards, M Parr, D Milliss, 2002) Australian hospital-pharmacy-committee decisions on availability and New Zealand pricing and payment arrangements. We then formulated consensus guidelines for the use of this agent in New Zealand.

Results

Review of relevant information The PROWESS study (a multicentre controlled trial in 1690 randomised adult patients with severe sepsis, published in March 2001) showed a 6.1% absolute reduction (30.8% to 24.7%, $p = 0.005$) in all-cause 28-day mortality from a 96-hour infusion of drotrecogin alfa at 24 ug/kg body weight/hour, despite a possibly increased risk of serious bleeding (3.5% versus 2.0%, $p = 0.06$) in the drotrecogin alfa group.¹² The company that sponsored the study (Eli Lilly) sought product registration in the United States in September 2001 and provided the FDA with extensive documentation¹³ but only one phase III randomised controlled trial. Despite approving the agent for use for 'insert indication', the FDA Anti-infective Drug Advisory Committee was split 10 to 10 as to whether the agent is safe and efficacious.¹⁶ The 'key matters of concern' for the FDA were changes made during the trial, the use of APACHE II scores,¹⁹ and the risk of serious bleeding. The decision to approve the agent despite these concerns has recently been discussed and defended by a senior FDA member.¹⁵ Concern over inconsistency of the efficacy of the agent throughout the trial (with possible implications that the mid-trial protocol amendment or changes in the formulation of the agent were responsible) has led to a recently expressed view¹⁶ that the data at present do not provide sufficient evidence for the use of the agent to become 'the standard of care'. In subsequent correspondence¹⁴ the PROWESS authors discuss these concerns (but do not refute them directly) and suggest that 'clinicians can already incorporate level I evidence from PROWESS into their practice to obtain life-saving benefit for their patients'. A recent Canadian economic analysis suggested that the cost per life-year gained by treatment with the agent was US\$27 936 if all eligible ICU patients are treated, and

US\$24 484 if only patients with APACHE II scores of 25 or more are treated.¹⁷ Furthermore, if patients with such high APACHE II scores are treated, the cost per life-year gained was related to the age of the patient (US\$16 309 aged over 40, US\$28 100 aged 80 or more). The Australian PBAC recommendations¹⁸ were that the agent be 'Recommended for listing for 'Adult patients with severe sepsis who have a high risk of death as determined by acute organ dysfunction in at least two organs or modified APACHE II score of at least 25' on the basis of acceptable cost-effectiveness', and be 'restricted to patients with two or more failed organs to prevent use in less severely ill patients where the risks may outweigh the benefits'. The Eli Lilly Australian Clinical Advisory Board recommendations were in keeping with the PBAC recommendations but included a recommendation that assessment of progress be made after four hours' full resuscitation in an intensive care unit, including surgical therapy and antibiotics and that 'if objective improvement in organ function occurs' that administration 'be delayed'. This Board also recommended that, for the purposes of defining respiratory 'organ dysfunction', this should be due to 'lung injury/ARDS secondary to sepsis' and that any patient given the agent would be expected to be receiving ventilatory support. Several tertiary Australian hospitals have not approved the use of the agent because of the 'extreme financial implications'. The New Zealand price of the agent (to a hospital pharmacy) is currently \$1909 plus GST per 20 mg vial (personal communication, Eli Lilly, 2002), which would result in a cost of \$17 181 (including GST) for the treatment (total 160 mg) of a 70 kg patient. Hospitals wishing to use the agent will have to find this cost from within existing budgets.

Suggested guidelines for the use of drotrecogin alfa in New Zealand If the agent is made available for use, we recommend that specialists prescribing it be required to contribute clinical data to a national register of patients, and we recommend the following guidelines for its use:

1. Patients should be in an intensive care unit and being treated by an intensivist (or other specialist with experience in intensive care medicine capable of providing comprehensive support of patients with severe sepsis at a high risk of death).
2. Patients should have severe sepsis, as defined in the PROWESS study,¹² and not have the exclusion criteria indicated in the study (eg, trauma patients with increased risk of life-threatening bleeding, pregnant patients, those with recent haemorrhagic stroke or with an epidural catheter in situ).
3. Patients in whom PROWESS exclusion criteria¹² are not present but who are otherwise at increased risk of serious bleeding should be carefully considered as to the balance of risk and possible benefit.
4. Patients should have two or more organ failures as defined in the PROWESS study.¹²
5. Patients with severe comorbidity likely to strongly adversely affect their outcome from severe sepsis (eg, severe congestive heart failure), those suffering from terminal disease or those in whom a decision has already been made to limit other intensive therapies (eg, dialysis) should not receive the agent.

6. Prior to administration of the agent, a trial of all other appropriate therapy (including surgery or other drainage of infection, appropriate antibiotics, fluid therapy, ventilatory and inotropic support) should have been given.
7. Patients seen to be clearly improving after a period (perhaps six hours) of such intensive treatments are at reduced risk of death and should not receive the agent.
8. Patients with high illness severity (eg, APACHE II score of 25 or more)¹⁹ not clearly improving at this time should be considered for treatment.
9. Although the PROWESS study¹² enrolled patients aged 18 years and over, it may be reasonable to treat 'older' children.
10. Once a decision has been made to treat with drotrecogin alfa, treatment should commence as soon as possible, and within 24 hours of meeting treatment criteria, bearing in mind the need for secure haemostasis to be ensured if surgery has just been performed. (A period of 12 hours of post-operative haemostasis was required before commencing treatment with the agent in the PROWESS study.¹²)

Discussion

It is likely that there is an overall beneficial therapeutic effect (net reduction in 28-day mortality) from the use of drotrecogin alfa recombinant in selected intensive care patients with severe sepsis but this is debated.¹⁶ The size of this effect (and thereby the number of patients needed to treat) is debatable in the New Zealand clinical context. The PROWESS data suggested an NNT of 16.4 (95% confidence limits 9.6–52.6)¹² to result in one additional 28-day survivor. The increased risk of bleeding in the study was small in patients selected not to have high risk of bleeding.

Of particular concern in the New Zealand (and Australian) context is the problem of young patients with severe meningococcal disease, many of whom have at least moderate coagulopathy and would thereby have been excluded from the PROWESS study.¹² Although these patients may benefit from the agent, they are almost certainly at higher than usual risk of bleeding and a cautious approach to treatment is advised. To date, there have been only five reported cases (age 18–41, median 22) where drotrecogin alfa was used in meningococcal purpura fulminans.^{20,21} Profound thrombocytopenia before treatment with drotrecogin alfa was present in two patients and these were given platelet transfusions 'to maintain platelet count above 30×10^9 per litre'. No adverse bleeding events were reported. Children under 18 were excluded from the PROWESS study and there are no published randomised controlled trials in children with severe sepsis, although one is underway.²² The median time till death in a small series of children dying of meningococcal disease in Auckland was four hours and thrombocytopenic cerebral haemorrhage was a significant cause of late deaths (personal communication, J Beca, 2003). In view of the strong association of profound thrombocytopenia with mortality in young children with meningococcal disease,²³ we recommend a cautious approach to the use of drotrecogin alfa in such patients.

The issue of very high cost, moderately effective treatment is not just one for intensive care. The price of drotrecogin alfa is large and the resultant cost per life-year gained is of similar order to that of a small number of other treatments (eg, imatinib

(Glivec[®]) for liver transplantation, iloprost for pulmonary hypertension) that in New Zealand are subject to rationing. Drotrecogin alfa was given provisional consent in New Zealand on 19 September 2002.²⁴

Although New Zealand ICU practice^{25,26} probably differs from Canadian practice^{17,27} in casemix and approach, the relative cost-benefit implications of age and illness severity will remain relevant. Possible suggested strategies that might increase the cost effectiveness of this agent include restricting it to patients who are not 'clearly improving' after six hours of 'full intensive care therapies' in an ICU and restricting its use to younger patients with high severity of illness¹⁷ (eg, APACHE II over 25) who do not have limiting non-septic comorbidity. We support these strategies in our recommendations for use.

The responsibility for providing access to this agent is that of individual hospitals and this decision is expected to fall on hospital pharmacy committees. We recommend that individual area health-board pharmacy and therapeutics committees review the agent now and decide whether or not they will support its purchase and use.

Finally, we note that timing and appropriateness of surgical⁶ and antibiotic⁸⁻¹¹ therapy and resuscitation of oxygen delivery⁵ are powerful determinants of outcome in severe sepsis and suggest that all hospitals would be well advised to formally establish systems that ensure these factors of treatment are provided.

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References:

1. Angus DC, Wax RS. Epidemiology of sepsis: an update. *Crit Care Med* 2001;29(7 Suppl):S109-16.
2. Angus DC, Linde-Zwirble WT, Lidicker J, et al. Epidemiology of severe sepsis in the United States: analysis of incidence, outcome, and associated costs of care. *Crit Care Med* 2001;29:1303-10.
3. Thomas MG, Streat SJ. Infections in intensive care patients. In: Finch RG, Greenwood D, Norrby SR, Whitley RJ, editors. *Antibiotic and chemotherapy*. London: Harcourts; 2002.
4. Streat S, Judson J, Newby L, et al. Prospective audit of death, brain death and organ donation in New Zealand intensive care units. Submitted for publication.
5. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001;345:1368-77.
6. Clark MA, Plank LD, Connolly AB, et al. Effect of a chimeric antibody to tumor necrosis factor-alpha on cytokine and physiologic responses in patients with severe sepsis - a randomised, clinical trial. *Crit Care Med* 1998;26:1650-9.
7. Streat SJ, Plank LD, Hill GL. Overview of modern management of patients with critical injury and severe sepsis. *World J Surg* 2000;24:655-63.

8. Jensen AG, Wachmann CH, Espersen F, et al. Treatment and outcome of *Staphylococcus aureus* bacteremia: a prospective study of 278 cases. *Arch Intern Med* 2002;162:25–32.
9. Booy R, Habibi P, Nadel S, et al. Reduction in case fatality rate from meningococcal disease associated with improved healthcare delivery. *Arch Dis Child* 2001;85:386–90.
10. Dupont H, Mentec H, Sollet JP, Bleichner G. Impact of appropriateness of initial antibiotic therapy on the outcome of ventilator-associated pneumonia. *Intensive Care Med* 2001;27:355–62.
11. Ibrahim EH, Sherman G, Ward S, et al. The influence of inadequate antimicrobial treatment of bloodstream infections on patient outcomes in the ICU setting. *Chest* 2000;118:146–55.
12. Bernard GR, Vincent JL, Laterre PF, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. *N Engl J Med* 2001;344:699–709.
13. Eli Lilly. FDA briefing document: Anti-infective Advisory Committee. Drotrecogin alfa (activated), [recombinant human activated protein C (RhAPC)], Xigris™, BLA#125029/0. 12th September 2001. Available online: http://www.fda.gov/ohrms/dockets/ac/01/briefing/3797b1_02_FDA briefing.pdf Accessed September 2003.
14. Ely EW, Bernard GR, Vincent JL. Activated protein C for severe sepsis. *N Engl J Med* 2002;347:1035–6.
15. Siegel JP. Assessing the use of activated protein C in the treatment of severe sepsis. *N Engl J Med* 2002;347:1030–4.
16. Warren HS, Suffredini AF, Eichacker PQ, Munford RS. Risks and benefits of activated protein C treatment for severe sepsis. *N Engl J Med* 2002;347:1027–30.
17. Manns BJ, Lee H, Doig CJ, et al. An economic evaluation of activated protein C treatment for severe sepsis. *N Engl J Med* 2002;347:993–1000.
18. Australian Pharmaceutical Benefits Scheme. Positive recommendations of the Pharmaceutical Benefits Advisory Committee. June 2002. Drotrecogin Alfa (activated). Available online: <http://www.health.gov.au/pbs/general/listing/pbacrec/pbacrecjun.htm> Accessed September 2003.
19. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. *Crit Care Med* 1985;13:818–29.
20. Bachli EB, Vavricka SR, Walter RB, et al. Drotrecogin alfa (activated) for the treatment of meningococcal purpura fulminans. *Intensive Care Med* 2003;29:337.
21. Weisel G, Joyce D, Gudmundsdottir A, Shasby DM. Human recombinant activated protein C in meningococcal sepsis. *Chest* 2002;121:292–5.
22. Nimah M, Brill J. Coagulation dysfunction in sepsis and multiple organ system failure. *Crit Care Clin* 2003;19:441–58.
23. Peters MJ, Ross-Russell RI, White D, Kerr SJ, Eaton FE, Keengwe IN, Tasker RC, Wade AM, Kline NJ. Early severe neutropenia and thrombocytopenia identifies the highest risk cases of severe meningococcal disease. *Pediatr Crit Care Med* 2001;2:225–31.
24. NZ Gazette. New medicine – provisional consent. Drotrecogin alfa, activated (Xigris™). 19 September 2002, Available online: <http://www.medsafe.govt.nz/reg.htm> Accessed September 2003.
25. Streat S, Judson JA. Cost containment: the Pacific. *New Zealand. New Horiz* 1994;2:392–403.
26. Zimmerman JE, Knaus WA, Judson JA, et al. Patient selection for intensive care: a comparison of New Zealand and United States hospitals. *Crit Care Med* 1988;16:318–26.
27. Barnett R, Shustack A. Cost containment: the Americas. *Canada. New Horiz* 1994;2:332–5.

Comment:

Drotrecogin alpha (activated): a magic bullet or budget blowout?

For the first time since antibiotics were introduced for the treatment of infection, an adjunctive therapy has been shown to improve survival. In the presence of sepsis, drotrecogin alpha (recombinant human activated protein C) modulates the systemic inflammatory, procoagulant, and fibrinolytic host responses to infection.¹ The PROWESS multicentre study in which drotrecogin alpha (activated) was randomised to 1690 patients showed an absolute all-cause 28-day mortality reduction of 6.1% overall.¹ However, the benefits were most marked in more severe sepsis (Acute Physiology and Chronic Health Evaluation (APACHE) II score >25).²

This treatment is not cheap. The cost of a 96-hour treatment is NZ\$17 181.³ If one uses the PROWESS entry data, for each life saved 16 patients need to be treated. Drotrecogin alpha (activated) costs US\$160 000 (NZ\$278 000) per life saved, but as little as US\$27 400 (NZ\$48 000) per quality-adjusted life-year when limited to patients with an APACHE II score =25.²

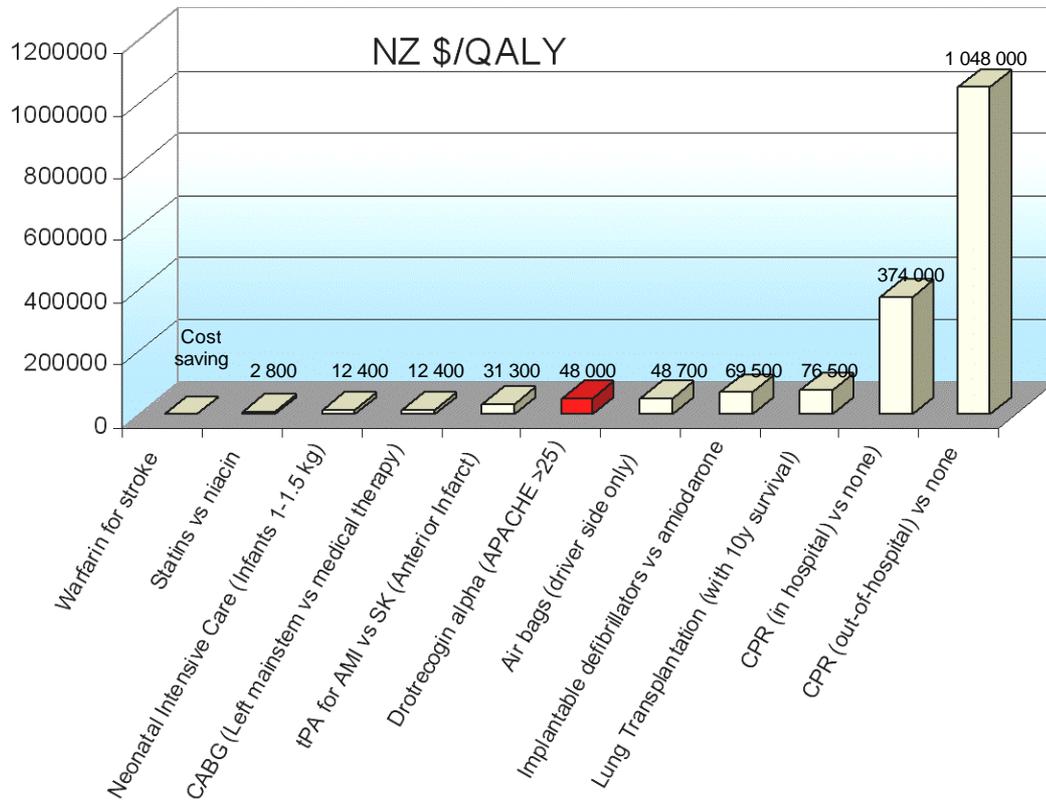
How does this stack up against other high-cost therapies? The cost effectiveness of drotrecogin alpha (activated) is comparable to most of the interventions in Figure 1 and better than that of airbags, implantable defibrillators, lung transplantation, and cardiopulmonary resuscitation.

In selected patients this therapy is clearly cost effective and beneficial. The problem is that New Zealand's public hospital system is fragmented into 21 district health boards and (unlike our Australian, UK, and US counterparts) concentrates on cost cutting as opposed to efficiency gains. In an environment where the funding for drotrecogin alpha (activated) is dependent on hospital pharmaceutical budgets it seems doubtful that all New Zealanders who may benefit from this treatment will get it. Inequity of access is at odds with Right 4 (3, 4) of the Health and Disability Code of Rights:⁴

- Every consumer has the right to have services provided in a manner consistent with his or her needs.
- Every consumer has the right to have services provided in a manner that minimises the potential harm to, and optimises the quality of life of, that consumer.

In the US, drotrecogin alpha (activated) has been granted new-technology status from the Centers for Medicare and Medicaid Services (CMS). This allows hospitals to receive additional reimbursement for treatment of Medicare patients.⁵ Similarly, New Zealand should fund drotrecogin alpha (activated) nationally, with the conditions of that funding based upon agreed guidelines, and at the same time develop a central database. This would improve equity of access and allow audit of the impact of the use of drotrecogin alpha (activated) on both patients and budgets.

Figure 1. Comparison of drotrecogin alpha with other widely used interventions, NZ\$ per quality-adjusted life-years (adapted from Figure 6, reference 2)



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References:

1. Bernard GR, Vincent JL, Laterre PF, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. *N Engl J Med* 2001;344:699–709.
2. Angus DC, Linde-Zwirble WT, Clermont G, et al; PROWESS Investigators. Cost-effectiveness of drotrecogin alfa (activated) in the treatment of severe sepsis. *Crit Care Med* 2003;31:1–11.
3. Liang J, Streat S, Torrance J, et al. Drotrecogin alfa (recombinant human activated protein C) in severe sepsis – a New Zealand viewpoint. *NZ Med J* 2003;116(1181). URL: <http://www.nzma.org.nz/journal/116-1181/586/>
4. The Health and Disability Commissioner (Code of Health and Disability Services Consumers' Rights) Regulation 1996. Available online. URL: http://www.hdc.org.nz/aboutus/the_code/TheCode.html Accessed September 2003.
5. Maggon K. Risk benefit assessment of APC. *Healthcare Management* 2003, 1–15 April. Available online. URL: <http://www.expresshealthcaregmt.com/20030415/edit2.shtml> Accessed September 2003.



Two brothers with nail cyanosis

Ellis Hon and Albert Li

Cyanosis of fingers and nails in infants and children deserves investigation and treatment of the underlying causes. It may be due to a trivial condition such as acrocyanosis of well neonates, which requires no treatment other than reassurance of the parents. On the other hand, serious cardiopulmonary conditions need to be excluded.¹ These conditions include cyanotic heart disease and chronic pulmonary conditions such as cystic fibrosis and empyema. These conditions are usually accompanied by the clubbing of fingers. Cyanosis without clubbing can also occur in Raynaud's phenomenon.¹ We report the case of two brothers with an unusual cause of 'cyanosis' of fingernails and toenails.

Case report

A sixteen-year-old boy with good past health was evaluated because his fingernails, and subsequently toenails, had become blue over a four-week period. He was a nonsmoker and denied any systemic symptoms, change of dietary habit, injury to the nails, or medication/nail polish consumption. He wore white socks and his shoes were not blue. Over the same period, his elder brother had also developed a similar blue hue in his fingernails and toenails.

Figure 1. Appearance of the fingernails and toenails of the patient who presents with nail 'cyanosis'



Physical examination revealed an apparently well teenager with uniform cyanosis of his toenails, and to a lesser extent in his fingernails (Figure 1). The pulps of his fingers and toes remained pink and the peripheral arterial pulses were normal. Blanching of the finger and toe tips did not alter the cyanosis, suggesting that the

discolouration was in the nail plate rather than the vascular nail bed. There was no central cyanosis or cardiopulmonary abnormality and his examination was unremarkable. His oxygen saturation was 99% in room air. Laboratory investigations were as follows: haemoglobin 14.0 g/dl; white blood count $7.4 \times 10^9/l$ with normal differentials; platelet count $282 \times 10^9/l$. Complement C3 was 1.31 g/l (reference range 0.62 to 1.87), and C4 was 0.27 g/l (reference range 0.20 to 0.59). Liver function and renal function tests were normal. Rheumatoid factor and anti-nuclear antibodies were negative. Urine analysis was also unremarkable.

As there was no clubbing or cardiopulmonary abnormality to explain his nail cyanosis, a nail-trim biopsy was scheduled in order to rule out abnormal pigment deposition in the nail matrix. The 'cyanosis' was accidentally removed by alcohol swab prior to nail biopsy. Further inquiry revealed that the discolouration was due to the dye from new blankets purchased and used by the two brothers in the preceding four weeks. Interestingly, the two brothers used separate blankets and slept in separate rooms.

Discussion

Nail discolouration due to dyeing has not been reported as a differential diagnosis for peripheral cyanosis. In our patient, there was no history of subungual haematoma or antimalarial use, and no evidence of Raynaud's phenomenon, central cyanosis or cardiopulmonary disease. His oxygen saturation was good. The finger and nail pulps remained pink and suggested that the problem primarily lay within the nail parenchyma. Blue nails secondary to chronic silver exposure, alcaptonuria and HIV have been reported in the literature.²⁻⁴ Poisoning with a drug or chemical should be suspected in any child who presents with sudden-onset cyanosis. A number of compounds activate haemoglobin oxidation from the ferrous to the ferric state to form methaemoglobin. These include nitrites and nitrate (contaminated water), chlorates, quinines, aniline dyes, sulfonamides, acetanilid, phenacetin bismuth subnitrate and potassium chlorate.⁵⁻⁷ In a male with completely negative clinical findings and normal oxygen saturations, we suggest that 'dyed nails' should be considered as a differential diagnosis of nail cyanosis.

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References:

1. Gawkrödger DJ. *Dermatology: an illustrated colour text*. 1st edition. Edinburgh: Churchill Livingstone; 1992. p. 61-3.
2. Plewig G, Lincke H, Wolff HH. Silver-blue nails. *Acta Derm Venereol* 1977;57:413-9.
3. Leppard B. Blue nails are a sign of HIV infection. *Int J STD AIDS* 1999;10:479-82.
4. ter Borg EJ. Clinical image: bluish discoloration of the nails in ochronosis. *Arthritis Rheum* 1998;41:1895.
5. Mansouri A, Lurie AA. Concise review: methemoglobinemia. *Am J Hematol* 1993;42:7-12.

6. Osterhoudt KC, Wiley CC, Dudley R, et al: Rebound severe methemoglobinemia from ingestion of a nitroethane artificial-fingernail remover. *J Pediatr* 1995;126:819–21.
7. Daniel III CR. Pigmentation abnormalities. In Scher RK, Daniel III CR, editors. *Nails: therapy, diagnosis, surgery*. Philadelphia: Saunders; 1990. p. 153.

Survival of the New Zealand Medical Journal

This extract is taken from a report of the British Medical Association (New Zealand Branch) Annual Meeting in Nelson, published in the New Zealand Medical Journal 1903, Volume 3 (9), p108

The President stated that at the Council meeting that afternoon the following resolution was passed: "That it be a recommendation to the general meeting that the annual subscription to the *Journal* be increased to 10s." He would call upon the Editor of the *Journal* to explain the position, financially and otherwise.

Dr. Mason said it had been reported to him by the Secretary that, until more advertisements were secured for the *Journal*, the funds at the disposal of the management would not admit of more than two issues a year. The cost of printing and circulating the *Journal* (quarterly issues) was something over £200 a year, while the revenue amounted to only about £150 – i.e., £60 from members' subscriptions and £90 from advertisements. If, therefore, the subscription was raised from 5s. to 10s. the deficiency would be made up. Apart from all this, however, he must say that unless members were prepared to take more interest in the *Journal* it should be allowed to die. He, as Editor, found the greatest difficulty in inducing members to supply him with matter for publication, although he was aware that most practitioners were in a position to contribute interesting articles upon cases they had attended.

Dr. Collins hoped members would make up their minds to an increased subscription, as it was the best suggestion the Council could offer.

Dr. Mason pointed out that after the first year or two it might be possible to reduce the subscription, as the revenue from advertisements was gradually increasing.

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Muscular emesis

The figures below are ultrasound scans of a four-month-old child with vomiting and abdominal mass.

Figure 1 shows a transverse section through the thickened pylorus. The echogenic centre represents mucosa and the hypoechoic area represents the muscle.

Figure 2 is a sagittal section showing elongation of the pylorus.

Pylorus stenosis is usually apparent on ultrasound and responds to surgery.

Figure 1.

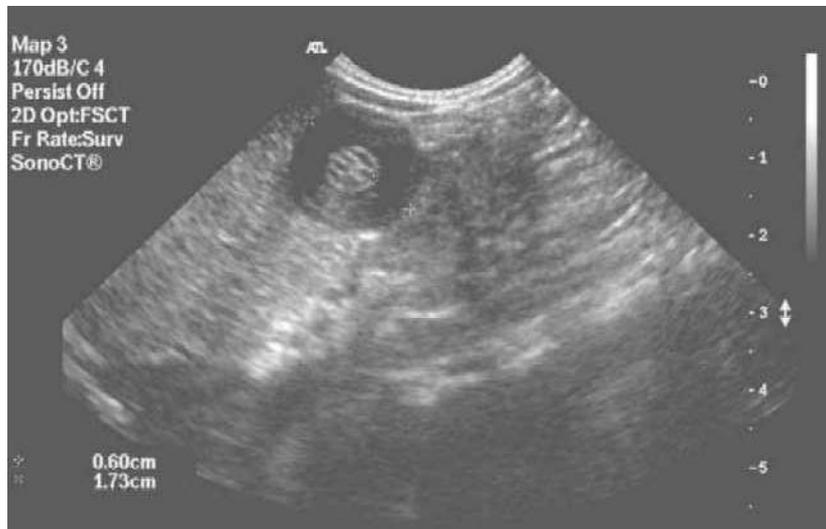
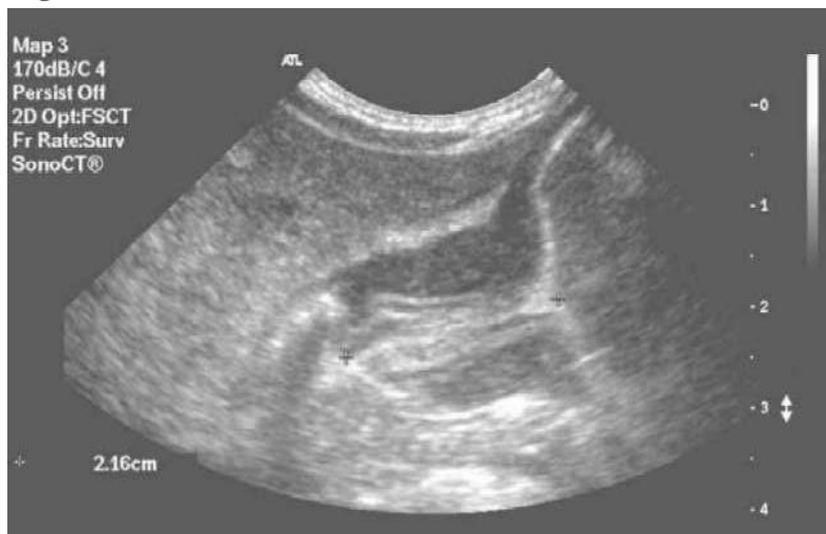


Figure 2.





Cancer clinical trials and publication bias

Failure to publish the results of large clinical trials can lead to bias in the literature and may contribute to inappropriate clinical decisions. Krzyzanowska and colleagues identified abstracts of large phase 3 clinical trials in the proceedings of the annual American Society of Clinical Oncology meetings from 1989 through 1998 and found that the probability of full publication by 5 years was significantly greater for trials with significant results than for trials with nonsignificant results. Trials with oral or plenary presentation were published sooner than those not presented, and trials with pharmaceutical sponsorship were published sooner than cooperative group trials or those for which sponsorship was not specified.

JAMA 2003;290:495-501

General practice across the Tasman (and elsewhere)?

All is not well with general practice. Australian GPs along with their international colleagues, protest that they are undervalued, overworked and no longer in control. "They feel like hamsters on a treadmill. They must run faster just to stay still...The result of the wheel going faster is not only a reduction in quality of care, but also a reduction in professional satisfaction and an increase in burnout amongst doctors." Assemble any group of Australian GPs and talk will soon turn to how recent Federal Government policies regulating general practice have reduced their fiscal autonomy, increased red tape, eroded their professional time, and diminished the quality of their clinical care.

However, a more ominous threat to the future of general practice is its increasing unattractiveness as a vocation. Junior doctors in Australia and North America are increasingly dissatisfied with general practice, and are voting with their feet.

Anecdotal reasons advanced for this discontent include the low remuneration and lack of prestige of general practice, the demands of practice that may preclude a life beyond medicine, and the advent of competing players in the delivery of primary care, such as nurse or alternative medicine practitioners.

MJA 2003;179:6-7

Kurt Semm, RIP 16/7/2003 – death of a pioneer

On 13 September 1980 gynaecologist Professor Kurt Semm performed the world's first laparoscopic appendicectomy at the University of Kiel in Germany. When Semm, director of the department of obstetrics and gynaecology at Kiel University Hospital, later told a surgical meeting what he had done, the president of the German Surgical Society called for his suspension. The scepticism was not just confined to Germany. When Semm tried to publish his paper on the first laparoscopic appendicectomy it was rejected because the technique reported was assumed to be unethical.

At the time, diagnostic laparoscopies were well accepted, but surgical attempts – apart from gynaecological sterilisation – were considered experimental and therefore unethical. It seemed unthinkable that surgeons should not have a good view of the entire operation site or have direct access and manual contact with the organs that they wished to treat, even if Semm's method might mean smaller incisions and reduced tissue damage. When Semm tried to convince his colleagues from other surgical disciplines in Kiel and elsewhere of the advantages of laparoscopic surgery – for instance, for gall bladder removal – they were mostly sceptical or apprehensive. Some of his co-workers asked him to have a brain scan, suspecting brain damage or a brain disease in someone who would attempt such an extraordinarily dangerous procedure.

Nowadays minimally invasive surgery is a scientifically established standard procedure for certain operations.

BMJ 2003;327:397

Long-term risk of breast cancer in Hodgkin Disease

Treatment of Hodgkin Disease (HD) represents one of the major medical successes of the 20th century. Fifty years ago, the typical patient survived only a few years, whereas the current 5-year relative survival rate is 85%. In the United States alone, approximately 120 000 survivors of HD are at risk for the serious late sequelae of curative therapies, including the occurrence of new primary cancers.

The leading cause of death in long-term survivors of Hodgkin Disease (HD) is second malignant neoplasms. In this case-control study of breast cancer in a cohort of female 1-year survivors of HD diagnosed at age 30 years or younger, Travis and colleagues found that treatment with radiation alone at doses of 4 Gy or more delivered to the breast was associated with a 3.2-fold increased risk of breast cancer compared with patients who received lower doses of radiation and no alkylating agents. Treatment with combined radiotherapy and alkylating agents was associated with a 1.4-fold increased risk, whereas treatment with alkylating agents alone was associated with a reduced risk of breast cancer. Ovarian damage by either radiation or chemotherapy was associated with decreased risk.

JAMA 2003;290:465–75

Deja vu – limits imposed on residents' work hours

On July 1, 2003, something came to pass in the USA that had been warmly anticipated by many medical students and residents – and dreaded by faculty and programme directors. The Accreditation Council on Graduate Medical Education (ACGME), the private body that accredits all 7800 US residency programmes, imposed new residency work hour limits.

The guidelines generally limit on-duty time to 80 hours a week and require 10 hours off between duty periods, and one continuous 24-hour period off every 7 days. The guidelines also prohibit overnight in-house call more than once in three nights, and ban residents from working more than 24 consecutive hours.

In the past residents in internal medicine programmes might work 100–110 hours a week with every third night on call, and surgical residents might work 120 hours a week with every other night on call.

Lancet 2003;362:378–9.



Ban on DTCA represents State takeover

As part of his campaign to censor pharmaceutical companies, Professor Les Toop (<http://www.nzma.org.nz/journal/116-1180/557/>)¹ submits as ‘evidence’ a bar graph showing that his gang of supporters is larger than that of Barrie Saunders. Does he really believe that a show of numerical strength constitutes proof of anything, even if followed by the Latin abbreviation QED?

A ban on direct-to-consumer advertising is another step toward a complete takeover of the medical and advertising industries by the State. The medical academics pushing this attack on free expression are funded by that very same State – and who, after all, would bite the hand that feeds?

Richard McGrath
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References:

1. Saunders B. Direct-to-consumer advertising – where does the public interest lie? NZ Med J 2003;116(1180). URL: <http://www.nzma.org.nz/journal/116-1180/557/>



Direct-to-consumer advertising – yes it can compromise patient health

Barrie Saunders has recently written in the Journal about direct-to-consumer advertising (DTCA).^{1,2} Space precludes us debating the benefits and risks of DTCA. However, we do wish to rectify omissions and misrepresentations about PHARMAC.

When PHARMAC wrote to Mr Saunders it said that there is good evidence that DTCA leads prescribers to switch to new and more expensive medicines that in many cases offer no real benefit for patients, eg, fluticasone (from beclomethasone (BDP)) and proton pump inhibitors (from H2 antagonists). Accompanying material explained the many ways that DTCA creates fiscal risk on the limited government pharmaceutical budget, including increased demand for PHARMAC to subsidise medicines that are advertised. A copy of the letter and accompanying material sent to Mr Saunders is available on the PHARMAC website, www.pharmac.govt.nz.

Mr Saunders also understates how PHARMAC manages financial risks through not funding poor investments, alongside demand-side activities, where both are based on assessment of costs and benefits.³ DTCA-promoted medicines are typically more expensive for little additional benefit. These include both products that PHARMAC has not funded (for that reason), or funded products, such as in the Flixotide switch campaign (when existing BDP is no less effective).⁴ PHARMAC does not have an 'iron grip' over availability and costs,² as the effects of the recent Flixotide DTCA campaign show.

We estimate that the 2002 Flixotide DTCA campaign caused 139 800 person-months of switching to fluticasone at the very least, costing more than \$900 000 extra compared with BDP (less costly but equally effective). This public money is no longer available to DHBs. It would, for instance, fund 43 coronary artery bypass graft operations. Or it would gain 202 quality-adjusted years of life (QALYs), along with saving \$254 000 in other DHB costs, if invested in other priority pharmaceutical areas. These are health improvements that DHBs cannot access because money had to be spent funding patients who switched from BDP to Flixotide directly because of DTCA.

We agree that there is a case for improving the uptake of statins for those with proven cardiovascular disease.⁵ PHARMAC is actively promoting improved lifestyle and access to medicines where appropriate,⁶ with its 'One heart, many lives' campaign. But most statins are cost effective.⁷ Most pharmaceutical products promoted by DTCA may not be.

Finally, Mr Saunders needs to declare his conflicts of interest here, including funding he receives from the pharmaceutical industry.

Scott Metcalfe
Wellington

Wayne McNee, Peter Moodie
PHARMAC, Wellington

Conflicts of interest: Wayne McNee is Chief Executive, Peter Moodie is Medical Director, and Scott Metcalfe (public health physician) is externally contracted to the New Zealand Pharmaceutical Management Agency (PHARMAC). PHARMAC is the crown entity responsible for funding community medicines, on behalf of district health boards. PHARMAC is currently involved in litigation with GlaxoSmithKline relating to the 2002 advertising campaign for Flixotide.

References:

1. Saunders B. Direct-to-consumer advertising - where does the public interest lie? NZ Med J 2003;116(1180). URL: <http://www.nzma.org.nz/journal/116-1180/557/>
2. Toop L, Richards D. New Zealand deserves better. Direct-to-consumer advertising (DTCA) of prescription medicines in New Zealand: for health or for profit? NZ Med J 2003;116(1180). URL: <http://www.nzma.org.nz/journal/116-1180/556/>
3. Metcalfe S, Dougherty S, Brougham M, Moodie P. PHARMAC measures savings elsewhere to the health sector. NZ Med J 2003;116(1170). URL: <http://www.nzma.org.nz/journal/116-1170/362/>
4. Adams N, Bestall JM, Jones PW. Fluticasone versus beclomethasone or budesonide for chronic asthma (Cochrane Review). In: The Cochrane Library, Issue 3, 2003. Oxford: Update Software. CD002310
5. Metcalfe S, Moodie P. More about cardiovascular disease and lipid management in New Zealand. NZ Med J 2002;115(1163). URL: <http://www.nzma.org.nz/journal/115-1163/203/>
6. Freemantle N, Hill S. Medicalisation, limits to medicine, or never enough money to go around? BMJ 2002;324:864-5. Available online. URL: <http://bmj.com/cgi/content/full/324/7342/864> Accessed September 2003.
7. Moodie P, Metcalfe S, McNee W. Response from PHARMAC: difficult choices. NZ Med J 2003;116(1170). URL: <http://www.nzma.org.nz/journal/116-1170/361/>

Editor's comment

An editorial decision was taken not to include specific conflict of interest statements with the articles relating to direct-to-consumer advertising published in the previous issue of the Journal, as the authors' interests were felt to be self evident. All authors clearly stated the extent of their personal involvement in the DTCA debate within the text of their articles.



NZ must not return to the pre-PSA era

In their recent paper 'Screening for prostate cancer: a survey of New Zealand general practitioners' (<http://www.nzma.org.nz/journal/116-1176/476/>) the authors, Durham, Low and McLeod, make several conclusions.¹

The study was widely reported in the public media as indicating that men were being put at risk by their doctors testing them for prostate cancer, and these attempts at early diagnosis should be discouraged.² Rather, men should not seek or permit early diagnosis by prostate specific antigen (PSA) and digital rectal examination (DRE) testing.

Whilst acknowledging that the authors are widely read with regard to the epidemiology of prostate cancer I suggest that the conclusions reached, and the advice given to New Zealand men and their doctors, including use of the public media, concerning the early diagnosis and management of this condition are out of step with prudent management at this time for the following reasons.

The response rate in the study was low (66.3%) and an assumption is made that there would be no difference between responders and non-responders because age and year-of-registration demographics are similar. It is possible that selection bias occurred, with those not interested in PSA testing tending not to respond. One does not have the impression that 'nearly all' offer these tests as stated. It follows that the conclusions reached with regard to proportions of GPs offering testing are suspect.

The term 'screening' is not appropriate in this context; rather what has been described in the questionnaire case vignettes is early diagnosis after informed consent in a consultation setting, an activity sanctioned by virtually all relevant peer groups, and a far cry from random population 'screening' as implied by the authors. Indeed, in two (and possibly all) of the questionnaire vignettes concern about prostate cancer was the primary reason for consultation.

The statements concerning the 19 'published reviews' and that 'New Zealand GPs support a programme of no proven benefit and the potential to cause considerable harm' are not conclusions drawn from the survey that is the subject of the paper but represent an opinion of the authors. If the 19 reviews mentioned are studied it is found that only six were published in recent years, since 1999.³ The other 13 were published early in the 'PSA era', 1997 or earlier, long before any mortality benefit could be expected, nor would their authors have considered them definitive statements in this regard. It is surprising they have been used in the context of the current paper given the lead author's statement, commenting on the Austrian study (which found a benefit for PSA testing), that 'at least 5 years, probably more than 10 years from initiation of prostate cancer screening need to pass before mortality benefit occurs.'^{3,4} Concerning the more recent six reviews it is simplistic to merely state that screening is not supported; they present more complex analysis of the issues surrounding early diagnosis. For example, the high-quality review article by Bunting presents a careful analysis of the risks and benefits of early diagnosis.⁵ While caution is advised, a return to the days of no early diagnosis is not.

The narrowness of the authors' view is perhaps best revealed by their comments on the study by Holmberg et al concerning the trial of 645 Swedish men with early, localised prostate cancer randomised to watchful waiting or radical prostatectomy reported in 2002.⁶ They do mention that no difference in overall mortality was found, but fail to offer perspective by outlining other aspects of this study: 8.9% died of prostate cancer in the watchful waiting group, versus 4.6% in the radical prostatectomy group; 27.3% developed metastases in the watchful waiting group compared with 13.4% in the prostatectomy group; 61.1% of the watchful waiting group experienced local progression of the primary, versus 19.3% of the surgical group; 24.7% of those watchful waiting required androgen deprivation therapy versus 17% of the surgical group. These differences all reached significance except for the last.

In interpreting the significance of this study it is important to realise that poorly differentiated tumours were excluded from entry; had they been included the results in the watchful waiting group would have been unacceptably worse. No responsible clinician manages these tumours by watchful waiting where the man is a candidate for potentially curative treatment.

The authors infer that localised prostate cancer has a 90% disease-specific survival at 10 years in the absence of active treatment. In a *Lancet* review of 18 238 men managed conservatively, those with Gleason 8 or more lesions had a 10-year cancer-specific survival of 45%; for Gleason 5,6 and 7 this was 77%; only for well differentiated Gleason 2–4 lesions was this figure close to the authors' at 93%.⁷ Albertson, in a study of 771 men with localised disease managed conservatively followed for 15 years, found that those with Gleason 7–10 lesions faced a high risk of death from prostate cancer; for 8–10 lesions this was from 60% to 87%.⁸

This leads to consideration of arguably the most dangerous aspect of advocating that New Zealand GPs abandon and discourage attempts at early diagnosis: it means that there is a group of men who would miss the opportunity for potentially curative treatment of an aggressive lesion. Their doctors then face the prospect of facing those men and their families later in the course of the disease. Given the commonality of prostate cancer this could be a frequent event in general practice. If an earlier request to test was refused or brushed aside there is a substantial risk of a complaint to disciplinary (and perhaps legal) authorities being successful, indeed this has already occurred in New Zealand.

Nor would it be of much use to quote the authors that 'it is unknown whether treatment for screen-detected cancer is effective.' There are many series reporting good results for surgery and radiotherapy; for example, Ohori with 90% biochemically free of disease (bned) at 10 years,⁹ Catalona with 71% bned at 10 years in another large surgical series,¹⁰ and Walsh with 70% bned at 10 years.¹¹ Many New Zealand urologists relate similar, albeit earlier, experiences; for example, my own of 123 prostatectomies, follow up one to 9 years (mean 3.8), bned 74%, cancer-specific survival 99.1%.

The authors have focussed on overall survival as their most important parameter in assessing management. This is, I suggest, the most difficult and nebulous parameter because of the comorbidity of many of these patients. Others, such as cancer-specific survival, metastases development, local progression, and bned offer more scientific

appraisal. These are ignored in the authors' approach. Patients seeking advice are primarily concerned with avoiding dying of prostate cancer, which they know to be prolonged and unpleasant. They do not expect deaths of other causes to be prevented. There is clear evidence, as outlined above, that early curative treatment prevents prostate cancer deaths.

A further problem with using overall survival as the most important parameter is that androgen deprivation therapy has a profound effect on the survival of those with metastases. If this was not the case the consequences of missing the opportunity for cure would be greater. This benefit comes with a price, high for some, in terms of mentation, body habitus and function and is usually temporary. Cure, if possible, is better. That death from prostate cancer usually occurs with elephantine slowness further reduces the usefulness of overall survival as a parameter.

The definitive investigation for a raised PSA is transrectal ultrasound scanning with biopsy (TRUS). This, while not to be embarked upon lightly, is now an acceptable, safe, office investigation thanks to the widespread use of prophylactic antibiotics, intravenous sedation and local anaesthetic, and modern equipment. Many in New Zealand have a similar experience to myself with 796 procedures performed with no major complications. It does provide information of sufficient quality to cautiously advise patients.¹² To describe it as 'potentially harmful and costly' as the authors have done, could be seen as being somewhat dismissive given the importance of the condition it is attempting to assess.

Nor is it inevitable that all prostate cancer found will be actively treated. There is a profound awareness amongst clinicians regarding the high incidence in autopsy series of prostate cancer, especially small foci of well differentiated lesions, and the risk of over treatment. This is of major concern in view of the significant morbidity of all treatments. By carefully interpreting data from TRUS, including Gleason score, core length involvement and number of involved cores, this risk can be minimised. That this approach is successful is suggested by data from radical prostatectomy series showing that the great majority of removed prostates contain significant lesions, positive margin rates of 25–35% being common.^{9,10} My own experience is 35% positive margins, 19% poorly differentiated. It has been shown to be difficult to detect the common small foci, 97% of which have Gleason scores less than 7, by PSA and TRUS.^{5,13} More than 90% of detected lesions have been shown to be clinically significant.¹⁴ There is also strong clinical awareness that for many older men with comorbidities their prostate cancer will be of little consequence to them. It is common for these groups to be managed by surveillance or watchful waiting only. In my own practice, of 562 with prostate cancer 24% have been managed this way.

It seems the authors would have New Zealand men return to the pre-PSA era, when the mortality ratio was almost 50%, and 70% of men diagnosed, who had largely waited for symptoms as suggested by the Cancer Society and mentioned by the authors, already had metastases. Meanwhile, elsewhere in the advanced world early diagnosis is now so common that it is difficult to recruit and follow groups for no-treatment arms of trials. Half of my career has been spent working in the pre-PSA era and I have no desire to return to it. It would seem that many GPs and their patients agree.

Perhaps, when trying to help men with this disease we should learn to use the tools we have as best we can, rather than dismiss them as imperfect, leaving men to their fate.

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References:

1. Durham J, Low M, McLeod D. Screening for prostate cancer: a survey of New Zealand general practitioners. *NZ Med J* 2003;116(1176). URL: <http://www.nzma.org.nz/journal/116-1176/476/>
2. New Zealand Herald, 27 June 2003.
3. Durham J. Population screening for prostate cancer: a systematic review. Wellington: New Zealand Guidelines Group; 2002. Available online. URL: http://www.nzgg.org.nz/library/gl_complete/prostate_cancer/Prostate_Cancer_review.pdf Accessed September 2003
4. Auvinen A, Alexander FE, de Koning HJ, Miller AB. Should we start population screening for prostate cancer? Randomised trials are still needed. *Int J Cancer* 2002;97:337–8.
5. Bunting PS. Screening for prostate cancer with prostate-specific antigen: beware the biases. *Clin Chim Acta* 2002;315:71–97.
6. Holmberg L, Bill-Axelson A, Helgesen F, et al. A randomized trial comparing radical prostatectomy with watchful waiting in early prostate cancer. *N Engl J Med* 2002;347:781–9.
7. Lu-Yao GL, Yao SL. Population-based study of long-term survival in patients with clinically localised prostate cancer. *Lancet* 1997;349:906–10.
8. Albertsen PC, Hanley JA, Gleason DF, Barry MJ. Competing risk analysis of men aged 55 to 74 years at diagnosis managed conservatively for clinically localised prostate cancer. *JAMA* 1998;280:975–80.
9. Ohori M, Goad JR, Wheeler TM, et al. Can radical prostatectomy alter the progression of poorly differentiated prostate cancer? *J Urol* 1994;152:1843–8.
10. Catalona WJ, Smith DS. 5-year tumor recurrence rates after anatomical radical retropubic prostatectomy for prostate cancer. *J Urol* 1994;152:1837–42.
11. Walsh PC, Partin AW and Epstein JI. Cancer control and quality of life following anatomical radical retropubic prostatectomy: results at 10 years. *J Urol* 1994;152:1831–6.
12. Smart R. Outcomes of transrectal ultrasound scan of the prostate with sector biopsies for 323 New Zealand men with suspicion of prostate cancer. *NZ Med J* 1999;112:465–9.
13. Catalona WJ, Smith DS, Ratliff TL, Basler JW. Detection of organ-confined prostate cancer is increased through prostate-specific antigen-based screening. *JAMA* 1993;270:948–54.
14. Oesterling JE. Prostate specific antigen: its role in the diagnosis and staging of prostate cancer. *Cancer* 1995;75:1795–1804.



OOS or MUD

RSI, OOS, ME, MUD, CPS, CFS, CTD etc, are all stirred into an acronymous concoction. Are these manifestations of a disease of medical thought, which I will name malignant acronymosis (MA)?

Dr Dodwell (<http://www.nzma.org.nz/journal/116-1176/480/>)¹ attempts to resolve this terminological confusion by adding further acronyms when acronyms may be the problem. Does this stem from trying to simplify very complex problems by using telegraphic acronyms? He uses twelve in his paper.

His acronym MUD, a dirty word (DW), can mean ‘musculo-skeletal undiagnosed disorder’, ‘muddled unscientific diagnosis’ or better, I think, ‘misunderstood disorder’.

These descriptive labels condensed to initial letters masquerade as diagnoses which then tend to be read as labels for disease entities. The separate words that the letters represent are forgotten. For example, OOS consists of three words, each of which must be assessed stepwise as implied in the OSH practitioner’s guide of which I was an author:²

1. S for syndrome: there must be a recognisable symptom and sign pattern;
2. O for overuse: there must be evidence that the overuse is causative;
3. O for occupation: there must be evidence that the overuse results from work.

This stepwise logical process became buried in the acronym OOS, which was subsumed into a *disease entity* and so to the devaluation of what was a useful descriptive label. Dr Dodwell is reasonably requesting that we go the full circle and re-start doing what was originally intended. But does it help to introduce yet more acronyms? I have for ten years been involved in a large international study to define epidemiological criteria for these syndromes.³

Those afflicted by chronic pain, the majority, were then graced by another acronym: CPS (chronic pain syndrome). The same questions on overuse and work causation still have to be answered, and now the ACC act asks whether there is physical injury. CPS still lacks an agreed definition, perhaps because it is not definable, being a mix of several other syndromes. Or is it a still wider umbrella under which the OOS umbrella term can shelter? CPS could be subdivided according to extent and severity into regional (RCPS) or general (GCPS, alias fibromyalgia or chronic widespread pain (CWP)).

Some define CPS as an entity of unknown cause so that they can indulge in a tautological circle of thought enabling them to say (believe?) that this is an entity caused by a constitutional predisposition so that the evidence of work causation, no matter how strong (plausible), can be ignored. Compensation can then be denied no matter how much it may be deserved.

It has even been theorised, on remarkably little evidence, that CPS can be due to disuse (not overuse). This idea also appeals to insurers. According to this theory those

in severe pain are urged to exercise and undergo work hardening, even when it is obviously making them worse (doing harm). The catch phrases 'Use it or lose it' (UOLI) and 'Feel the pain to make the gain' (FPMG) can be disastrous in aggravating the pain and disability associated with these syndromes. There is a possible advantage in using the suggested acronyms for these mantras as they would then be rendered impotent as no-one would not know what they meant.

All this is very confusing to the layman, the review officer, the judge and even the *expert* witness and the independent medical assessor (IPA). I would suggest that the first step in resolving this verbal traffic jam is to outlaw acronyms and use plain English.

I agree that this mess needs to be cleaned up but please do not start by using the DW 'MUD' or the MUD will stick on those who desperately need our help and sympathy. The last thing that they need is derision and ridicule (DR). DR, of course, also refers to those who have sworn the Hippocratic oath (HO).

Primum non nocere. Primarily don't knock your patient (PDKYP).

I have managed to use 19 acronyms here in the hope that their overuse will lead to them being unemployed.

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References:

1. Dodwell P. OOS or MUD? Time for a cleanup. NZ Med J 2003;116(1176). URL: <http://www.nzma.org.nz/journal/116-1176/480/>
2. Wigley RD, Turner WED, Blake BL, et al. Occupational overuse syndrome. Treatment and rehabilitation. A practitioner's guide. Wellington: Department of Labour; 1992.
3. Helliwell PS, Bennett RM, Littlejohn G, et al. Towards epidemiological criteria for soft-tissue disorders of the arm. Occup Med 2003;53:313-9.



Effects of male circumcision on female arousal and orgasm

While vaginal dryness is considered an indicator for female sexual arousal disorder,^{1,2} male circumcision may exacerbate female vaginal dryness during intercourse.³

O'Hara and O'Hara reported that women who had experienced coitus with both intact and circumcised men preferred intact partners by a ratio of 8.6 to one.⁴ Most women (85.5%) in that survey reported that they were more likely to experience orgasm with a genitally intact partner: 'They [surveyed women] were also more likely to report that vaginal secretions lessened as coitus progressed with their circumcised partners (16.75, 6.88–40.77).'⁴

Presence of the movable foreskin makes a difference in foreplay, being more arousing to the female.⁴ Women reported they were about twice as likely to experience orgasm if the male partner had a foreskin.⁴ The impact of male circumcision on vaginal dryness during coitus required further investigation.

We conducted a survey of 35 female sexual partners aged 18 to 69 years who had experienced sexual intercourse with *both* circumcised and genitally intact men.

Participants completed a 35-item sexual awareness survey. Women reported they were significantly more likely to have experienced vaginal dryness during intercourse with circumcised than with genitally intact men χ^2 (df = 1, n = 20) = 5.0, p < 0.05.⁵

Women who preferred a circumcised male sexual partner averaged 27.3 years of age (SD = 8.2), while those whose stated preference was for a genitally intact partner had a mean age of 36.4 years (SD = 13.7). Thus, the role of the male foreskin in preventing loss of vaginal lubrication during intercourse may become more discernible with increasing age among women. We reported:

'During intercourse, the skin of an intact penis slides up and down the shaft, stimulating the glans and the nerves of the inner and outer foreskin. On the outstroke, the glans is partially or completely engulfed by the foreskin with more skin remaining inside the vagina than is the case with the circumcised penis. This 'valve' mechanism is thought to retain the natural lubrication provided by the female because the bunched up skin acts to block the lubrication escaping from the vagina, which results in dryness.'⁵

Our work, which supports the hypothesis of Warren and Bigelow³ and the findings of O'Hara and O'Hara⁴ about the role of the male prepuce during coitus is fully reported in Denniston et al.⁵

Research generally has not considered possible adverse effects of male circumcision upon female sexual arousal and response. While Moynihan reported that vibratory thresholds, blood flow and hormone levels were studied,¹ there was no mention of circumcision status of the male partner. Likewise, Leiblum failed to control for male circumcision status.² In light of published findings,^{4,5} this is a serious methodological omission.

Most likely, reported vaginal dryness and the related clinical designation 'female arousal disorder' is but a normal female response to coitus with a man with an iatrogenically deficient penis.⁵

It is imperative that future studies of female arousal disorder record and control the circumcision status of male sexual partners.

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References:

1. Moynihan R. The making of a disease: female sexual dysfunction. *BMJ* 2003;326:45–7.
2. Leiblum SR. Arousal disorders in women: complaints and complexities. *Med J Aust* 2003;178:638–40.
3. Warren J, Bigelow J. The case against circumcision. *Br J Sex Med* 1994;Sept/Oct:6–8.
4. O'Hara K, O'Hara J. The effect of male circumcision on the sexual enjoyment of the female partner. *BJU Int* 1999;83 Suppl 1:79–84.
5. Bensley GA, Boyle GJ. Physical, sexual, and psychological effects of male infant circumcision: an exploratory survey. In: Denniston GC, Hodges FM, Milos MF, editors. *Understanding circumcision: a multi-disciplinary approach to a multi-dimensional problem*. New York: Kluwer Academic/Plenum Publishers; 2001. p. 207–39.



Inappropriate sexual comments

Charge: A Complaints Assessment Committee charged the Doctor with disgraceful conduct in a professional respect. The particulars of the charge alleged the Doctor over the period 22 February 1995 and 28 March 1995 in the course of his management and treatment of his patient:

1. Asked questions and made comments of an inappropriate and sexual nature;
3. Performed one or more of the internal vaginal examinations in an inappropriate sexual manner; and
4. First discussed and then suggested to his patient that he should use on her a 'perineometer' which he had made himself which was inappropriate and for which there was no medical justification; and
5. When confronted by his patient on or about 28 March 1995 destroyed or sought to destroy her medical notes.

Particular 2 was withdrawn during the course of the hearing.

Background: The patient alleged the Doctor made inappropriate and sensitive remarks of a sexual nature whenever she had a consultation with him. According to the patient he asked inappropriate questions of a sexual nature and he kept trying to talk about sexual matters all the time. She alleged he made comments about her pubic hair, discussed the use of vibrators with her, spoke about how she should use vaginal fluids as a perfume and asked her questions about her sex life including questioning the size of her partner's penis. She further said he suggested she get her partner to perform oral sex on her.

The patient told the Tribunal that the Doctor tried to sexually stimulate her with a speculum. She alleged that he gave her Xylocaine gel and told her to 'massage' her clitoris to see what happened. She said he wanted her to masturbate with it and that during one consultation he was rubbing his penis through his trousers and was definitely aroused.

The patient further alleged during the course of one consultation the Doctor produced a 'gadget' which looked like a vibrator. It transpired that the device was made by the Doctor, and was called a perineometer. It was designed to test the strength of a woman's pelvic floor muscles.

The patient's evidence was that on the last day she consulted the Doctor she telephoned him and explained to him that the reason she went to him was for a few prescriptions and not to learn about sex or hear about other people's sexual problems. Later that day she went in to pick up a prescription and she was very angry. She told the Doctor that she wouldn't be coming back again so he could destroy her notes as she wouldn't be needing them. The Doctor cut up the medical file in front of her.

The Doctor vehemently denied the allegations made against him by the patient and explained that he was deeply distressed and horrified by her claims. He explained that

during his career he developed an interest in sexual education and, in particular, female sexual function and dysfunction.

The Doctor said that his interest in the pelvic floor of women related to the problems which he observed patients suffering with bladder incontinence and vaginal prolapse. He explained that he developed the perineometer for legitimate health purposes. A gynaecologist gave evidence that it was appropriate to use a perineometer to assess pelvic floor strength.

The Doctor ardently denied making any comments or asking questions which were of an inappropriate sexual nature to the patient. He said he gave the patient Xylocaine because of her complaints of soreness in the vaginal area during intercourse, as it had the effect of inducing numbness. He said he would have advised the patient to apply it externally to the area that was sore and did not tell her to apply it to her clitoris.

The Doctor told the Tribunal that the allegations he was sexually aroused and that he may have been masturbating himself when the patient was in his surgery were completely false. He suffered erectile dysfunction and provided the Tribunal with documentary evidence that showed he sought assistance for this condition long before he saw the patient.

There appeared to be some agreement between the patient and the Doctor that he requested the patient to tighten her pelvic floor muscles whilst he conducted an internal examination. The Doctor said that he would have done this for the sole purpose of assessing the strength of her pelvic floor muscles. He denied he attempted to sexually stimulate the patient with a speculum and said that he did not suggest he could use the perineometer on her.

The Doctor explained that he cut up the notes with a guillotine in front of the patient as he thought if he gave them to her, she would destroy them. He placed the notes in a rubbish bin. As soon as the patient left he retrieved the notes and placed them in an envelope. He said he realised that the better approach would have been to photocopy them and give the patient the records but retain a copy.

Finding: The Tribunal found the Doctor guilty of professional misconduct.

The Tribunal considered when the Doctor was cross examined it was very apparent that his recollection of what he said to the patient during the consultations of February and March 1995 had become blurred. The Tribunal was satisfied the Doctor no longer had an accurate recollection of what he said to the patient when he discussed these issues with her. It was equally satisfied that the patient's recall of these matters was generally accurate.

When considering Particular 1, the Tribunal noted that none of the consultation notes recorded any suggestion the patient was consulting the Doctor about issues relating to possible sexual dysfunction.

The Tribunal was in no doubt that when the patient first saw the Doctor he made assumptions about her sexual experience. He assumed that she was a prostitute and that she would be a willing listener to some of his views and theories about sex. The Tribunal was also in no doubt that the complainant did tell the Doctor that she was anorgasmic and that this fuelled his willingness to talk about sexual issues with the complainant.

The Tribunal considered the patient did not consult the Doctor about the way she might enhance her sexual life. She consulted him about specific clinical issues. It was the Doctor who pursued issues of a sexual nature with the patient believing she was interested in learning about his views and theories concerning sexual dysfunction. The Doctor appeared to have been unaware of the fact that the patient was not interested in his questions and comments concerning sexual dysfunction. It considered it was the Doctor's responsibility to appreciate the patient did not welcome his raising the sexual issues. However, it was confident the Doctor pursued these topics out of a genuine interest for the welfare of his patient and that he was not motivated by personal sexual gratification.

The Tribunal considered the Doctor's raising of sexual issues with the patient in the circumstances of this case breached the standards which the profession and the community expect of a general practitioner in the Doctor's position. Furthermore the Tribunal believed the Doctor's serious breaches of standards warranted a disciplinary finding against him. The Tribunal found that in relation to the first alleged particular of the charge the Doctor's conduct amounted to professional misconduct.

When considering Particular 3, the Tribunal was satisfied the Doctor performed four vaginal examinations, and that each of those examinations was clinically justified.

The Tribunal could readily understand the patient's suspicions and concerns about the way she believed the Doctor performed one or more of the vaginal examinations on her. The Doctor's raising of sexual topics in circumstances which caused concern and distress to the patient may easily have led her to believe that he was acting in an inappropriate way during the course of conducting vaginal examinations. However, the Tribunal was not satisfied to the requisite standard that the Doctor attempted to sexually stimulate the patient during the course of any of the vaginal examinations, or that he acted in a sexually inappropriate manner when conducting any vaginal examination.

When considering Particular 4, the Tribunal accepted the Doctor constructed the 'perineometer' for the purpose of either using it himself or allowing patients to use it to assess the strength of their pelvic floor muscles. The perineometer was not a vibrator.

The Tribunal understood why the patient was concerned and confused when the Doctor produced the perineometer. In the context of consultations in which the Doctor raised a number of sexual issues, the patient could be excused for thinking the perineometer was some form of home-made sexual device. The Doctor again completely misread his patient's concerns. There was no obvious clinical justification for the Doctor to show the patient the perineometer. The Tribunal was satisfied this was another example of the Doctor pursuing issues which interested him but which were not relevant to the reasons why he was being consulted by his patient.

While the Tribunal gave the Doctor the benefit of the doubt and concluded there was no sinister motive behind his showing the patient the perineometer, and suggesting it be used, the Tribunal was nevertheless confident there was no clinical justification for the Doctor's actions. In the circumstances of the case it was inappropriate for him to show the device to the patient in circumstances which caused her distress and concern.

When considering Particular 5, the Tribunal accepted that the patient told the Doctor that he could destroy her notes and in order to placate her he proceeded to cut her medical notes up in her presence. It further accepted that as soon as the patient left the Doctor retrieved the notes from a rubbish bin and placed them in an envelope for safekeeping.

The Tribunal considered it was not appropriate for the Doctor to cut up the patient's medical notes and his actions in cutting up the notes constituted a failure to adhere to the standards expected of a medical practitioner. However, the charge alleged the Doctor 'destroyed or sought to destroy' the patient's medical notes. The Doctor did not destroy the notes, nor did he seek to destroy them. The Tribunal was satisfied that no disciplinary finding was justified in relation to the Doctor's breaches of his duty when he cut the medical notes with his guillotine.

Penalty: The Tribunal ordered that the Doctor be censured and pay costs in the sum of \$10 000.

The Tribunal believed a lenient penalty could be imposed upon the Doctor in this case for the following reasons:

- The Doctor has practised medicine for approximately 44 years without any other complaints of a disciplinary nature being brought against him.
- The events complained of occurred a considerable time ago and there were delays in bringing this matter to the attention of the Tribunal.
- The Doctor no longer practises medicine and one of the purposes of punishment in a disciplinary forum is to discourage further offending. He has not practised medicine for some years and is never likely to practise again.

When assessing costs, the Tribunal accepted that the Doctor and his wife were not in a position to pay a substantial sum. In addition, the Doctor successfully defended a charge of disgraceful conduct and was found guilty in relation to only two particulars of the five particulars which were initially laid against him.

Addendum decision: The Tribunal ordered the Doctor be granted permanent name suppression. It further ordered publication in the New Zealand Medical Journal a summary of the Tribunal's decision.

The full decisions relating to the case can be found on the Tribunal web site at www.mpd.org.nz Reference No: 02/97C.