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LETTERS

INFECTIVE ENDOCARDITIS

Time to monitor incidence after NICE guidance

In the wake of guidance on infective endocarditis from the National Institute for Health and Clinical Excellence (NICE),[1] the number of cases of endocarditis seems to have noticeably increased.[2] Specialists in my area say that such cases have increased 10-fold, and I have already seen as many cases as I would expect to see in 10 years. Although these data do not prove a causal relation and not all cases include dental treatment, we as doctors should sharpen our diagnostic acumen and examine the guidance critically.

Disappointingly, no system has been put in place to monitor changes in the incidence of endocarditis following the NICE guidance.

Dentists seem to be scrupulously following the guidance, some telling their patients that dentists could be deemed to be negligent if they did not follow the guidance. Dentists in my area have been receptive to modifying their approach, adopting the more inclusive American guidance with the support of local doctors.[3]

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Competing interests: None declared.

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CARDIOVERTER DEFIBRILLATORS

Should not be implanted in isolation after infarction

Disappointingly, Liew states that the optimal time to implant a cardioverter defibrillator after acute myocardial infarction remains "unresolved."[1] However, the timing of implantation has become enigmatic because of a too simplistic approach.

Sudden death remains a large risk soon after infarction, and its causes, not just its symptoms, must be treated early. Sudden cardiac death happens in patients with extensive heart disease, which must therefore be addressed as well as the rhythm abnormality. Death remains inevitable after cardiac rupture or left main occlusion, regardless of how many shocks an implantable cardiac defibrillator delivers. DINAMIT confirmed this simple, expected logic.[2] Sudden arrhythmic death was significantly reduced, but it was counterbalanced by an equivalent increase in other causes of death, such as heart failure and recurrent myocardial infarction.

Comorbidity has never been systematically addressed in any of the trials of implantable cardiac defibrillators after infarction. They report that a "sizeable" proportion of the subjects underwent angiography and intervention, but such intervention was random, not randomised. This is not the same as ensuring balanced study arms by making intervention part of the randomisation protocol. VALIANT has the same obvious flaw of past methods and confirms that patients with sudden cardiac death also have reinfarction, cardiac rupture, and terminal heart failure.[3] Unless these aspects of a patient's disease are addressed, an implantable cardioverter defibrillator cannot be useful. Thus VALIANT does not confirm that a cardioverter defibrillator should not be implanted within 40 days after an acute myocardial infarction, merely that it should not be implanted in isolation.

This question needs to be answered: does early implantation of cardioverter defibrillators after infarction in conjunction with appropriate revascularisation reduce mortality?

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Competing interests: None declared.

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GOUT

Clarifying some aspects of management

In response to Cayley's article,[1] we would like to clarify some important aspects of the management of gout. In acute gout, we would recommend 0.5 mg of colchicine two to three times daily for three days, in accordance with published guidelines.[2] More frequent (one to two hourly) dosing should be avoided, because of the high incidence of gastrointestinal adverse effects and potential treatment failure.[2 3] Lower doses may be needed in older patients and in those with renal impairment.

Oral prednisolone at a dose of 25-35 mg daily for five days without tapering is a reasonable acute third line treatment.[2 4] Corticosteroids may also be given intramuscularly or intra-articularly in acute gout.

Urate lowering treatment, aiming for a serum urate of less than 0.30 mmol/L, should be considered in patients experiencing two or more attacks of gout a year.[2] Allopurinol remains the treatment of choice; alternatives include the uricosuric drugs, sulfinpyrazone and benzbromarone, or the newer xanthine oxidase inhibitor, febuxostat. Prophylaxis against acute attacks, using colchicine 0.5 mg twice daily or a non-steroidal anti-inflammatory drug, is needed for up to the first six months of treatment.[5]

Lifestyle adjustments, such as reducing weight and alcohol intake, are also extremely important. The metabolic syndrome often coexists with gout, so patients require continuous monitoring for the development of comorbidities such as hypertension, hypertriglyceridaemia, and type 2 diabetes.

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Competing interests: ALH and KMJ are trustees of the UK Gout Society. KMJ has received educational sponsorship from A Menarini Pharma UK, the manufacturers of febuxostat. TGR has no conflicts of interest.

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Management in primary care

Although welcome, Cayley's article focuses exclusively on acute gout and omits several important aspects of diagnosis and management that are particularly relevant to general practice.[1] The article rightly points out that hyperuricaemia does not necessarily mean that the patient has gout. However, conversely, serum uric acid concentrations may be normal during an acute attack and then rise as acute inflammation settles.[2] Hence, the absence of hyperuricaemia does not exclude the diagnosis of gout, and—if normal during an acute attack—serum uric acid should be rechecked once the attack has settled.

Gout has a strong association with the metabolic syndrome and traditional cardiovascular risk factors, and it is an important risk factor for coronary heart disease.[3] Cayley advises assessing the patient for risk factors for gout including cardiovascular disease, hypertension, and diabetes. However, the importance of assessing traditional cardiovascular risk factors such as hypertension, hyperlipidaemia, and diabetes relates to reducing cardiovascular risk in these high risk patients. Traditional cardiovascular risk factors are checked in only 26% of patients presenting to primary care with acute gout.[4] Glucose and lipids should be checked in addition to the investigations suggested by Cayley and managed appropriately if abnormal.[5]

Finally, a review should be scheduled after the acute attack has resolved to discuss continuing management including modification of adverse lifestyle factors, detailed review of drugs including diuretics, consideration of urate lowering drugs such as allopurinol for long term management, and a management plan to facilitate self care in case of further acute attacks.

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Competing interests: ER has received an honorarium from Ipsen. CDM has no competing interests.

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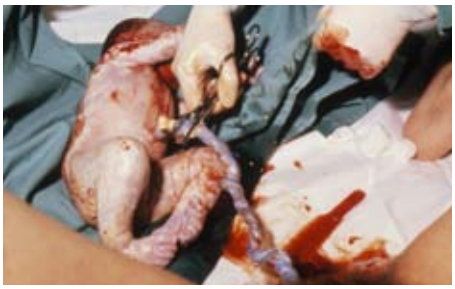
RUSHING TO CLAMP UMBILICAL CORD

More evidence is needed to inform practice


JOHN HESELTINE/SPL

The Royal College of Obstetricians and Gynaecologists' Scientific Advisory Committee did not advise that there is "no evidence that the timing of cord clamping affected postpartum bleeding."[1] Rather, it said there is "no statistically significant difference" for postpartum haemorrhage or severe postpartum haemorrhage, and therefore the optimal timing for cord clamping is "unclear."[2] Lack of evidence of an effect is not the same as evidence of lack of effect.

We agree that immediate cord clamping should have been rigorously evaluated decades ago. We disagree that the current evidence supports "a rush to change." Trials of immediate versus deferred cord clamping for term and preterm births have not reported data for all important outcomes, have been underpowered for serious adverse effects, and lack adequate long term follow-up of the women or children.[3 4] This is particularly important for very preterm infants, for whom the risk of disability is greatest.

Recommendations about timing of cord clamping are conflicting because the evidence on substantive clinical outcomes is unclear. For example, although the World Health Organization has removed the need for immediate cord clamping as part of active management of the third stage of labour, this is described as "weak recommendation, low quality evidence."[5] We are unlikely to have consistency in recommendations for practice until we have strong high quality evidence on which to base them. Further randomised trials are needed to resolve the uncertainties. Opinion leaders and influential organisations have an important role in encouraging such trials.[2 5]

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Competing interests: All the authors are co-applicants or collaborators on a programme of work that includes developing strategies for providing initial care with the cord intact and a pilot randomised controlled trial of timing of cord clamping for preterm births. LD contributed to developing the RCOG Scientific Advisory Committee opinion paper on clamping of the umbilical cord. DT was a member of the Guideline Development Group for the NICE intrapartum care guideline. WT-M is principal investigator of the Australian Placental Transfusion Study (ACTRN12610000633088).

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Cite this as: *BMJ* 2011;342:d122

Don't just do something, stand there

In my editorial quoted by Hutchon,[1 2] I recommended that it was better not to rush into clamping the cord. I pointed out that early cord clamping is not as benign an intervention as it may seem, and that there was increasing evidence of harm, especially in premature babies. It seems to me straightforward, therefore, that because of serious safety concerns about the intervention, it should be withdrawn. For any other intervention this would not even be debated. If there were a longstanding non-evidence based practice of giving routine antiepileptics to neonates "just in case they fitted," and serious safety concerns were raised—what would happen? Would everyone say, "we need to keep giving the intervention until there is definite proof of long term harm to neonates"? Of course not. The drug would be withdrawn until it became clear that it was both safe and effective.

The same should be the case for early cord clamping. The onus is on those who believe that routine prophylactic red cell depletion in the neonate is necessary to provide proof of its safety and benefit. Until then, I continue to advise that it is better not to rush.

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Competing interests: ADW was part of the team that developed the mini-resuscitator, which we have called the Bedside Assessment, Stabilisation and Initial Circulatory Support (BASICS) trolley. This is being developed in the University of Liverpool, but all inventors have agreed that any profits will be donated to charity.

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TOWARDS AN END TO STILLBIRTHS

Joint action is the key

Heazell's article about the neglect of stillbirth highlights the contrast with cot death.[1] The massive fall in unexpected infant deaths is the result of a joint effort by parents and professionals to stimulate and fund research, to formulate a strategy, and to pressurise the government into action. The key to progress is joint action.

I was a member of the Stillbirth Study Group, which wrote a leaflet on help for parents after stillbirth over 30 years ago. The Stillbirth and Neonatal Death Society (SANDS), which has provided enormous support to bereaved families, was formed soon after. In contrast to the experience of the Foundation for the Study of Infant Death, professionals were not ready to join with parents in a combined attack on the problem, and the stillbirth rate has hardly changed. SANDS attempted to learn about practice in maternity units in 2009; of 377 units contacted, 235 failed to reply.[2] Heazell reported that he had enquired about practices in postpartum management of still birth; 1036 of the 1134 obstetricians and 9317 of the 10 000 midwives whom he contacted did not respond.[3]

Paediatric expertise can complement that of the neonatal pathologist and the obstetrician. I have spoken to the parents of hundreds of stillborn infants.[4] I am confident that the staff of maternity units do care, and that together with parents (and paediatricians) they could instigate research and strategies to reduce the number of stillbirths and give professional care to individual families.

Let us do so without any further delay.

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Competing interests: RGW is a former adviser to Surrey SANDS Trustee Foundation for the Study of Infant Death.

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ORPHAN DRUGS

Authors' reply to BioMarin Europe

BioMarin Europe feels that our letter on the costs of orphan drugs such as amifampridine (Firdapse) for Lambert Eaton myasthenic syndrome (LEMS) and the safety of existing unlicensed treatments, such as 3,4-diaminopyridine base (3,4-DAP), contained inaccuracies.[1 2] The Department of Health has confirmed that the additional cost of amifampridine in England is £9 768 668 (€11 400 035; $15 294 803) (excluding VAT).[3] We will leave it to readers to decide whether our figure for the UK (£10m) is more accurate than the £6m cited by BioMarin.[1] Furthermore, Biomarin states that up to 18% of patients on 3,4-DAP had adverse events.[1] This is a misrepresentation of a retrospective study performed in a multiple sclerosis clinic. Only three of the 669 patients given 3,4-DAP had LEMS. Of the 18.2% of patients who had adverse events, the most common was paraesthesia (36%, hardly surprising in these patients), and only six patients had a serious event, which the authors state was "rare and similar to those seen in published reports."[4] Because no comparative data of amifampridine versus 3,4-DAP have been published, there is no evidence that the more expensive new drug is more efficacious or safer than the older drug that clinicians have more than 20 years' experience with. The dramatic difference in cost needs to be met from savings in other areas, thus disadvantaging other patients, with little or no perceivable clinical benefit.

However, the broader issue of the cost of orphan drugs remains unanswered, and we are amazed at the Department of Health's lack of interest given the huge sums involved. In 2007, 1% of the total NHS drugs bill—at least £110m a year—was spent on orphan drugs.[5]

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EU regulations misunderstood

Two articles on orphan drugs misunderstand the interplay between European Union and national regulations and reimbursement schemes.[1 2] They imply that under EU legislation there is a monopoly for orphan drugs, a minimal requirement for clinical data before licensing, and financial benefits before approval.

Article 8 of Regulation (EC) No 141/2000 refers to market exclusivity, wrongly interpreted in the articles as a monopoly. Market exclusivity protects an orphan product authorised for an orphan indication from similar products seeking authorisation for similar indications. This incentive can be challenged in case of lack of supply of the first product, proved clinical superiority, or agreement to share the market with the original sponsor. Exclusivity may be reduced according to Article 8(2) of the regulation. The regulation does not define any financial benefits before approval.

Orphan products are authorised on the same basis as non-orphan products. In 2007, only 44 of the 528 orphan drug designations had obtained a licence (8.5%).[3] Of these, only three obtained a licence through published data, permitted if well-established use can be proved.

Drug reimbursement is not considered in Regulation (EC) 141/2000 because it is the responsibility of member states. Concerns about establishing price and reimbursement for orphan drugs are shared by many in Europe. This makes it even more important to clearly distinguish which incentives are provided by the EU regulation and which are not.

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Competing interests: None declared.

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REFERRALS TO SECONDARY CARE

NICE "referral advice" recommendations database

McBride and colleagues conclude that the absence of explicit guidance is one reason for the inequality in primary to secondary care referrals associated with socioeconomic circumstances.[1]

Referral to a specialty service is a crucial point in a patient's management. The decision is complex, reflecting the needs and expectations of individual patients and their families, the knowledge and experience of the practitioner, and the range of services available.

In 2001, the National Institute for Health and Clinical Excellence (NICE) developed advice on the appropriate referral to specialist services in response to concerns that attempts to reduce waiting list times may affect quality of care.[2] Further referral advice was subsequently incorporated into new clinical guidelines. After a recent quality, innovation, prevention, and productivity (QIPP) workshop,[3] NICE revisited this issue and has collated all its referral guidance into a searchable database (www.nice.org.uk/usingguidance/referraladvice/index.jsp).

Uncertainty remains over the impact of guidance on reducing variation in referral rates from primary to secondary care, with the extent of its impact being dependent on the specific features of the guidance and the local cause of variation.[4] An important and recurrent theme in the literature is a need to stimulate better joint working and dialogue between primary and secondary care. Referral guidelines should not, as has been cautioned, reduce the willingness of GPs to tolerate uncertainty and increase referrals to secondary care.[5] Accordingly, NICE referral guidance should be used to encourage local health communities to discuss referral problems and develop local referral protocols. Implementing NICE guidance can help GP commissioners ensure that patients receive clinically and cost effective treatment. Following NICE guidance can also free up resources and capacity to be channelled into other services.

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Competing interests: None declared.

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EMPLOYEE OWNERSHIP IN THE NHS

Takes time to achieve

Even for a commercial company, the move to all-employee ownership, as discussed by Ham and Ellins,[1] is a major transition. Most people take years to move from a relatively passive "employee" frame of mind to that of being an active owner-partner in the business. It is this change that leads to sustained improvement in productivity, service, and innovation.

Two things are essential for success. The first is time. Public sector organisations will be going through a double transition: into a commercial world and into all-employee ownership. They need time to get to grips with those changes. Relatively long initial contracts can provide it.

The second essential is to take advice. Much has been learnt about how to structure employee ownership to be sustainable. The importance of having at least half the ownership in a trust cannot be overstated. The long lived employee owned companies, such as John Lewis (80 years) and Arup (40 years), have trust ownership; those such as the National Freight Corporation that chose individual employee ownership and flotation had shorter lives. The government's initial provision of £10m (€11.7m; $1.6m) for advice for groups seeking to make the transition is a good start.

The approach of the commissioning bodies will be key. A simple minded, lowest cost approach allows outsourcing companies to nullify their initial low quotes through expensive "change of scope" provisions. Services provided by owner-employees should prove more honestly constructive.

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Competing interests: The Baxi Partnership supports organisations exploring alternative business models.

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OPEN LETTER TO BMA

BMA's reply

Since the government published its NHS White Paper in July, the BMA has played a leading part in responding critically to the proposals.[1]

The BMA's position is rooted in policies agreed through our democratic processes, is shaped by ongoing feedback and consultation with members, and follows several debates on the issue, endorsed by most of the elected council. The BMA represents all doctors in the UK, and so the range of views will always be broad: our job is to represent and support them all as fairly as possible.

The BMA has rightly acknowledged the aspects of the proposals that reflect improvements the profession has been calling for, such as a greater role for clinicians in the design and planning of services, and a focus on quality and outcomes rather than crude targets.

However, the BMA has been most vocal on the issues that most concern the majority of doctors. We have been very clear about the consequences of rushing ahead with the planned reforms, particularly at a time of financial pressure, and the dangers of increased market based competition in the NHS. We have emphasised that an NHS based on a competitive rather than a cooperative model is likely to lead to a fragmentation of services and create significant inefficiencies, ultimately resulting in poorer quality of care for patients.

The proposed NHS reforms have profound and far reaching implications—some potentially good, some potentially bad, and many more as yet unknown. Some of the proposals are already becoming a reality across the country, with doctors often at the forefront of the changes. If we turn our backs on the reform process now the changes may continue but without crucial professional leadership to ensure the highest standards of care for patients. We should question the evidence for change, reflect the impact of policy on the frontline of patient care, and, above all, ensure our professional values remain at the core of what doctors do. The consequences of not being involved would be far more damaging to the NHS we are seeking to protect and preserve.

We are working closely with patient organisations, professional bodies and trades unions, and other stakeholders, as well as parliamentarians. Our views are respected—and shared—by many. We must stand together and, through continued argument, evidence, and practice, work to get the best possible service for our patients and the wider public.

The BMA has no intention of ceasing its pressure on government to reconsider its position on the areas of most concern to the profession and patients, and we will be stepping up our lobbying and member engagement activities once the bill has been published and during its passage through parliament.

Continue sending us your views on the government's plans and sharing with us your experiences of any developments locally. Give your views on the member feedback form on our website (www.bma.org.uk) or email us at info.whitepaper@bma.org.uk.

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1 Peedell C and cosignatories. Open letter to the BMA about the health white paper. *BMJ* 2011;342:d7. (4 January.)

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See OPEN LETTER TO THE BMA, p 146.

For further responses to the Open Letter from Peedell *et al* see bmj.com