

OBSTRUCTIVE SLEEP APNOEA

Treat sleep apnoea before people fall asleep at the wheel

We are the father and uncle of Toby Tweddell, who was killed in August 2006, his car crushed from behind by a large goods vehicle; the driver had fallen asleep at the wheel, and was subsequently found to have sleep apnoea.¹

After the inquest into Toby's death, the coroner reported in August 2008 that the driver had visited his general practitioner during April 2006 complaining of tiredness. Blood and urine samples were taken to test for diabetes, and when the results were returned negative he was advised that he was most probably suffering from stress. Critically, sleep apnoea was not diagnosed.

So, yes, obstructive sleep apnoea is easily missed by general practitioners,¹ with dreadful consequences.

After Toby's inquest, the coroner took the unusual step of issuing a Rule 43 Report to the Lord Chancellor calling for a toughening of the licensing regimen for commercial drivers as it relates to obstructive sleep apnoea. But as yet the Department for Transport and Driver and Vehicle Licensing Agency continue to resist a sharper focus on identifying and treating people with obstructive sleep apnoea before they cause an accident.

We call for a coherent and evidence based framework that:

- Makes clear the connection between obesity and obstructive sleep apnoea
- Indicates the likely prevalence of the condition among vocational drivers, given the prevalence of obesity among them
- Encourages general practitioners to identify and treat potential cases of sleep apnoea¹



- Requires vocational drivers to be regularly screened, rather than only once they reach the age of 45.

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1 West SD, McBeath HA, Stradling JR. Obstructive sleep apnoea in adults. *BMJ* 2009;338:b1165. (7 April.)

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COMBATING MALARIA

Try public health measures in Africa too

The director of the Global Fund for AIDS, Tuberculosis and Malaria says: "There is no reason any child should die from malaria anymore" because we have "impregnated bed nets" and "effective drugs to treat those who fall ill."¹

Not a word about environmental hygiene or public health, which curtailed malaria considerably not only in the colonial days of the Gold Coast/Ghana but also in present day Singapore, Cuba, and Trinidad with the same hot and humid climate.² In equally hot and humid Orlando, Florida, goldfish are farmed in vast areas of standing water to devour mosquito larvae—hence no malaria.

Has frenetic malarial therapeutics with apparent exclusion of public health as practised in politically disciplined Singapore, Florida, Cuba, and Trinidad more to do with the interests of the pharmaceutical companies than with the health of the natives?³

As I wrote on millennium development goals: "The West will only give you tablets and vaccines, and will even prevent your goods being sold on the European market" but "we natives seize on the word 'development' and think about agriculture, covering of open drains, and pipe borne water."⁴ How many of the Global Fund experts advising us in the Tropics have had the guts to tell our leaders: "If you do not cover your open drains within 12 months, do not come to us begging for funds to fight malaria"? Or "Why don't you send health teams to Singapore to find out how they do things?"

What saddens me as I get older is that experts sent to us in Africa ostensibly to help "roll back malaria" prefer to talk therapeutics to pulling us up sharply on environmental sanitation. Not many of them think and talk like Zuckerman:

"Get rid of vectors, and malaria tends to disappear."⁵

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- 1 Moszynski P. New funding mechanism is launched to combat malaria. *BMJ* 2009;338:b1627. (20 April.)
- 2 Konotey-Ahulu FID. Averting a malaria disaster. *Lancet* 1999;354:258.
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Public health measures work

Having lived my first 21 years in Trinidad without having malaria or knowing anyone who did, I am committed to the public health measures I saw being used there which made this possible. In Ghana several of my friends and relatives had serious side effects after a course of the new artemisinin combination therapy,¹ which also did not guarantee long or short term immunity.

Good old fashioned public health programmes, which could be implemented with effective supervision at every point in the healthcare chain, should form the basis of the healthcare programme of any government serious about eradicating or minimising malaria. One of my memories is that the sanitary inspector, who was easily recognisable by his uniform, had the authority to impose fines on residents who did not comply with simple and inexpensive preventive measures. The large workforce in this disease prevention programme also probably helped to keep unemployment to a minimum.

Shouldn't African governments now take full responsibility for the health of their people? Why are these decisions still under the purview of the World Health Organization and other groups? Of course, drug companies stand to gain from the perpetual presence of malaria in Africa and elsewhere. This is how the system works and has worked for decades, so why change it? Because it is not at all in the interest of African and other people in poor countries.

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- 1 Moszynski P. New funding mechanism is launched to combat malaria. *BMJ* 2009;338:b1627. (20 April.)

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CLOPIDOGREL

Increasing concordance

Chua and Ignaszewski review the evidence base for clopidogrel in acute coronary syndromes.¹ Knowing the number needed to treat and number needed to harm for each trial would enable more informed decision making with patients.

For example, in the CURE trial the number needed to treat for the primary composite outcome was 48 and the number needed to harm for major bleeding (disabling, requiring transfusion) was 100. In other words, for every 100 patients treated for an average of 9 months with clopidogrel plus aspirin instead of aspirin alone, two non-fatal myocardial infarctions would be prevented at the expense of one major bleed caused. Overall, one positive outcome occurs for every 100 patients so treated. Is this explained to patients so a joint decision can be made (concordance) on whether to take clopidogrel as well as aspirin?

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- 1 Chua D, Ignaszewski A. Clopidogrel in acute coronary syndromes. *BMJ* 2009;338:b1180. (14 April.)

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Bleeding and resistance

The review of clopidogrel in acute coronary syndromes does not mention the management of bleeding in patients taking clopidogrel.¹ Unlike aspirin, whose antiplatelet effects can be predictably overcome by platelet transfusion, clopidogrel has no reliable antidote. Another omission is clopidogrel resistance, the subject of a recent review.²

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Competing interests: None declared.

- 1 Chua D, Ignaszewski A. Clopidogrel in acute coronary syndromes. *BMJ* 2009;338:b1180. (14 April.)
- 2 Dominick J, Angiolillo MD. Variability in responsiveness to oral antiplatelet therapy. *Am J Cardiol* 2009;103(suppl 1):27-34A.

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More on proton pump inhibitors

Although proton pump inhibitors reduce the rates of gastrointestinal bleeding in patients with acute coronary syndromes receiving dual antiplatelet treatment,¹ recent evidence points to sinister and potentially far more injurious side effects.^{2,3}

Co-prescription of proton pump inhibitors known to inhibit hepatic cytochrome P450 2C19 (omeprazole, lansoprazole, and rabeprazole) with clopidogrel resulted

in a 40% relative increase in the risk of recurrent myocardial infarction.² The risks of rehospitalisation, revascularisation procedures, and death were higher in patients with acute coronary syndromes receiving a proton pump inhibitor and clopidogrel.³ However, pantoprazole and esomeprazole have shown no negative effects on the cardioprotective action of clopidogrel,^{2,4} probably because of their differing metabolic pathways.⁵

Not all proton pump inhibitors are born equal, and further studies are urgently required to establish the safest combination.

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- 1 Chua D, Ignaszewski A. Clopidogrel in acute coronary syndromes. *BMJ* 2009;338:b1180. (14 April.)
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to reduce ulcer complications.³ Concomitant treatment with proton pump inhibitor reduced the excess risk of bleeding associated with all antiplatelet treatments, but H2 antagonists were effective without any dose-response relation.²

Thus prophylaxis with proton pump inhibitors for the highest risk patients taking aspirin and clopidogrel is indicated: on limited data the choice is between esomeprazole (which does reduce rebleeding⁶) and pantoprazole (which has the greater body of data showing no interaction with clopidogrel).

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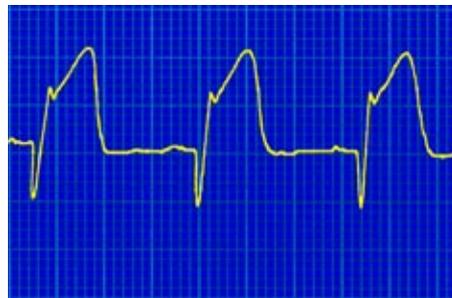
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Authors' reply

In response to our correspondents,¹ administration of a proton pump inhibitor with clopidogrel increases the risk of recurrent myocardial infarction and adverse cardiovascular outcomes, probably owing to inhibition of the cytochrome P450 2C19 pathway. Pantoprazole may be devoid of this interaction with clopidogrel on the basis of its lack of cytochrome P450 2C19 inhibition and subgroup analyses of two recent retrospective cohort studies.^{2,3} However, translating the results of subgroup analysis of these retrospective cohort studies into clinical recommendations is premature.

Emerging data on the interaction between clopidogrel and proton pump inhibitors contradict the suggestion that pantoprazole shows no negative effects on the cardioprotective action of clopidogrel. In their post hoc analysis of the CREDO trial Dunn et al found that concurrent use of proton pump inhibitors was associated with increased cardiovascular events in patients taking clopidogrel or placebo and was independently associated with increased adverse cardiac outcomes.⁴ Similar results were recently found in the Clopidogrel in Medco Outcomes Study—the largest trial to date investigating the interaction between clopidogrel and proton pump inhibitors in 16 690 patients.⁵ In this retrospective study, the use of any proton pump inhibitor concurrently with clopidogrel was associated with an increased risk of major adverse



cardiovascular events, with pantoprazole having the highest risk of cardiac events.

Thus, clinical studies have consistently shown that proton pump inhibitor use concurrently with clopidogrel is associated with higher rates of cardiovascular events. However, whether one particular proton pump inhibitor is safer remains unclear and the evidence to date is conflicting.

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- 1 Sadler GJ, Poullis AP. More on proton pump inhibitors. *BMJ* 2009;338:b1931.
- 2 Juurlink DN, Gomes T, Ko DT, et al. A population-based study of the drug interaction between proton pump inhibitors and clopidogrel. *CMAJ* 2009;180:713-8.
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NICOTINE REPLACEMENT THERAPY

Authors respond to criticism that treatment is ineffective

Siegel is surprised that we concluded that nicotine replacement therapy was effective and cannot think of other interventions that would be regarded as effective with such a high failure rate.¹ He chooses a secondary outcome from our review to make his point, but as it excludes 76% of all the people who stopped smoking for a long time, this is not an accurate measure of the impact of the programme. Overall, offering up to one year of treatment with nicotine replacement therapy helped 6.75%–3.28%=3.47%, or 1 in 29 users sustain long term abstinence. About half will become lifetime abstainers, about half of whom will be prevented from premature death,² which gives a number needed to treat (NNT) for prevention of premature death of about 120.

This NNT compares favourably with other preventive interventions. Five years of treatment with statins in those at risk of cardiovascular disease gives an NNT of 931 for preventing one death, while one year of treatment for hypertension in middle aged people gives an NNT of 850 for stroke prevention.³ Such treatments are widely accepted as effective, but the absolute benefits are small, or the treatment

is a failure for nearly everyone who takes it.

Smoking is largely sustained by tobacco addiction and reinstatement after abstinence is characteristic of addiction, resulting in high relapse rates. Treatment for unwilling quitters could be compared with treatment for willing quitters or other addictions. Smokers trying to stop using nicotine replacement therapy with and without modest behavioural support results in about 8% quitting.⁴ More intensive support and medication results in about 15% quitting in the long term.⁵ These treatment effect sizes are not very different from the 7% or so observed in unwilling quitters and treatment effects are not small judged in this context.

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- 1 Siegel M. Nicotine replacement, effective? *BMJ* 2009;338:b1730. (29 April.)
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THE DEATH OF DNR

Training is needed to dispel confusion around DNAR

If "allow natural death" (AND) is substituted for "do not attempt resuscitation" (DNAR), staff will become even more confused about the difference between end of life care and a DNAR decision.¹ DNAR is appropriate for all patients for whom end of life care is the agreed path, but not all patients with a DNAR decision are necessarily at the end of their expected life. Consequently, AND and DNAR are not synonymous. DNAR is intended to provide an immediate and unambiguous instruction to healthcare professionals on just one point—the patient to whom it relates should not undergo cardiopulmonary resuscitation (CPR) in the event of cardiorespiratory arrest. It does not relate to any of the many other important aspects of care planning that should take place and be recorded clearly and separately.

The principles underpinning CPR decisions are set out in the 2007 joint statement from the BMA, Resuscitation Council (UK), and Royal College of Nursing (www.resus.org.uk). The Resuscitation Council (UK) has subsequently



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published standards for recording DNAR decisions and a model DNAR form (www.resus.org.uk), and we welcome feedback. The forms were developed to help to ensure that a DNAR decision was readily accessible, given that the default action is to start CPR immediately.

Healthcare professionals require training to understand what DNAR means. Healthcare organisations must ensure that CPR decisions are made by healthcare professionals with training and experience in making and recording them. Professionals should discuss the issues clearly and sensitively with patients and families. Guidance on DNAR decisions has existed for many years, but high quality training supported by a properly structured implementation plan have been lacking.

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- 1 Sokol DK. The death of DNR. *BMJ* 2009;338:b1723. (30 April.)

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Personal resuscitation plans

We have developed personal resuscitation plans instead of do not resuscitate orders (DNRs) for children with severe neurodisability and life limiting or life threatening conditions.¹ Children's nurses asked us for clear information on appropriate interventions when a particular child's condition deteriorates, not just an instruction not to call the arrest team.

We use a template with a list of increasingly invasive interventions to discuss with parents, and when possible the young



ALPHOTO/SPL

person, to agree the most appropriate treatment. We emphasise what will be beneficial and what care will be given in an emergency rather than what won't be done. Also the plan can change over time as the child's condition deteriorates and certain interventions are no longer appropriate and the family is ready to accept that. Approaching a family to draw up what they can see is a useful emergency care plan is much easier than approaching them with a DNR form.

Our audit shows that 14 of the 19 children with personal resuscitation plans would have no more invasive treatment than suction, oxygen, and a trial of bag and mask ventilation in the event of an acute collapse. Half of the families found it difficult and upsetting to complete a plan but all would recommend it to other families. Comments included: "It's important to plan ahead and for everyone to know what to do," "It gives me peace of mind, there will be no misunderstandings about his treatment," "It will give us more control of the situation." The nurses say that the plans are empowering and reassuring when they are managing a dying child whether at home or in hospital.

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1 Sokol DK. The death of DNR. *BMJ* 2009;338:b1723. (30 April.)

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The undesirability of acronyms

The statement "allow natural death" (AND) is much less clear than "do not resuscitate" (DNR).¹ A term that needs defining before it may be used will always be open to interpretation and misinterpretation by patients, relatives, and staff, and have different meanings in different hospitals—a disaster in the high pressure context of a cardiac arrest.

A better statement would be: "In the event of cessation of heartbeat and/or breathing, please do not attempt to perform cardiopulmonary resuscitation. No implications regarding any other care for this patient are intended by this statement." Although long winded, this statement cannot be misinterpreted.

Perhaps what is revealed by Sokol's analysis is that trying to abbreviate an extremely important clinical directive to three letters will always be fraught with problems.

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1 Sokol DK. The death of DNR. *BMJ* 2009;338:b1723. (30 April.)

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BARRIERS TO ORGAN DONATION

Shouldn't be a family affair

I don't understand why I am able to give all my goods to whomever I specify after my death and there is no involvement of my family or anyone else whereas when I try to make a gift of organs my family have an entitlement to deny my wishes. This makes no sense at all.

Efforts to increase the availability of organs should concentrate on changing the law to make it clear that the wishes of the donor, expressed in advance, are sufficient to proceed.¹ We can discuss how to ensure that everyone is guided into expressing his or her wishes.

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Competing interests: None declared.

1 Simpkin AL, Robertson LC, Barber VS, Young JD. Modifiable factors influencing relatives' decision to offer organ donation: systematic review. *BMJ* 2009;338:b991. (21 April.)

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Improve staff infrastructure

In the United States, where most of the studies in Simpkin and colleagues' systematic review were performed,¹ a protocol for identifying potential organ donors and a procedure for trained staff to request organ donation when a patient meets the criteria (death or brain death) are legal requirements.

Consent rates were higher when the request was made by staff from the organ procurement organisation or transplant centre with the hospital staff.¹ In the United Kingdom inhouse transplant coordinators, who have the expertise in gaining consent, have thus become embedded in intensive care units. However, most requests for organ donation are still performed by hospital staff who are pressed for time and have little

formal training in this area. Until training and support for hospital staff is improved nationally, organ donation rates are unlikely to change.

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Teach medical students

Someone skilled and confident in a task is likely to achieve a better outcome. So why not include tackling organ donation in communication skills at medical school along with breaking bad news? Discussing organ donation could also be introduced as a common station to cover in postgraduate examinations. It should be covered as early as possible so doctors feel confident to raise the issue with relatives.¹ By the time students graduate or complete postgraduate examinations they will have covered it in theory many times so will feel more confident in practice.

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1 Simpkin AL, Robertson LC, Barber VS, Young JD. Modifiable factors influencing relatives' decision to offer organ donation: systematic review. *BMJ* 2009;338:b991. (21 April.)

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INSULIN PUMPS EXPOSED TO HEAT

Put an indicator on the bottle

Some years ago I tried to persuade several insulin manufacturers that they should put an indicator on the container that would change colour permanently if the insulin had been left outside the desirable temperature range and so become unreliable to use. Then the user would know what had happened. Currently, as was the case with the girl whose insulin pump had been exposed to heat and sunlight and who developed diabetic ketoacidosis on using it afterwards,¹ there is no way of knowing whether this has happened.

None of the manufacturers was the slightest bit interested in this suggestion, but perhaps this lesson of the week in the *BMJ* might stimulate some action.

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1 Pryce R. Diabetic ketoacidosis caused by exposure of insulin pump to heat and sunlight. *BMJ* 2009;338:a2218. (5 January.)

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