ACUTE NON-FATAL CHD EPISODES: MULTIPLE LOGISTIC REGRESSION-DERIVED RATE RATIOS ASSOCIATED WITH SLEEPING PATTERNS AND OTHER ESTABLISHED OR SUSPECTED RISK FACTORS

Variable (and groups or units)	Rate ratio (and 90% confidence interval)*
Schooling years (yr)	1·06 (0·97, 1·16)
Occupation (manual;† non-manual)	2·91 (1·26, 6·71)
Residence (rural;† urban)	1·15 (0·48, 2·75)
Weight (5 kg)	0·95 (0·80, 1·15)
Systolic BP (10 mm Hg)	1·11 (0·94, 1·35)
Cholesterol (20 mg/dl)	1·19 (1·00, 1·42)
Glucose (10 mg/dl)	1·12 (1·00, 1·25)
Smoking (non- and past;† current smokers)	0·45 (0·19, 1·08)
Alcohol (wine) (non-drinkers;†	
< 4 glasses/day;	0·50 (0.18, 1·37)
⩾ 4 glasses/day)	0·34 (0·12, 0·98)
Coffee (cups/day)	1·55 (1·21, 1·99)
Night sleep (30 min)	0·97 (0·85, 1·11)
Afternoon sleep (30 min)	0·71 (0·54, 0·93)

*All rate ratios are mutually and age adjusted.
†Baseline.

duration dependent and significant (two-tail $p < 0.04$) association between afternoon sleep with the occurrence of (at least) non-fatal CHD episodes, indicating that a half-hour siesta may be related to an almost 30% reduction in CHD incidence. Among patients reporting a siesta the association of its duration with CHD rate was even stronger: the point estimate of the rate ratio for a 30 min increase in the duration of siesta is 0·53, indicating that psychosocially[4] conditioned self-selection in the afternoon rest group is not a likely confounder in the observed association.

These findings should be interpreted with caution. The results were based on small numbers and although all identifiable confounders were allowed for in a multifactorial model, some residual confounding may have persisted. Nevertheless, the differential distributions and relative risks associated with type A behaviour[4] and drinking of alcoholic beverages[5] are not as large as to explain our findings, and physical activity[6] cannot generate residual confounding in the suspected direction.

There have been no other epidemiological studies of the long-term relation of siesta to mortality in general or to acute CHD episodes in particular, but there are several linking the duration of night's sleep with the risk of death and the incidence of CHD.[1,2] Furthermore, shiftwork has been linked to high CHD risk in a Swedish study,[7] a finding our inquiry confirms. Finally, there are many studies linking various aspects of sleep with several normal and abnormal functions of the circulatory system;[8,9] they could provide an insight into the mechanism of the long-term association between afternoon siesta and CHD episodes, if such an association were confirmed.

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TESTING OF BLOOD DONATIONS FOR NON-A, NON-B HEPATITIS

SIR,—In their contribution to the current debate in *The Lancet* our Scottish colleagues (July 4, p 36) argue the case for surrogate testing of blood donations for non-A, non-B hepatitis (NANBH). We take issue with several points.

Has the time for a prospective study already passed? This seems to imply that the longer an unproven test is used, the greater becomes the pressure to use it. This is not an argument that should commend itself to those practising transfusion medicine. Why should we have to wait 3–4 years for an answer? If the problem is serious this will be revealed, in *acute* NANBH, within a year of initiating the study. The need for controlled studies of the incidence of NANB post-transfusion hepatitis will not disappear with the introduction of routine screening of blood donations with tests of unproven value. Indeed, trials are necessary in different countries, where the incidence of NANB post-transfusion hepatitis is likely to vary as widely as does the incidence of positivity for putative markers such as anti-HBc. We sympathise with transfusion centres in countries where HBV infection is common, who would, with the policy Dr McClelland and colleagues support, have to replace the 20–30% of blood donors who are positive for anti-HBC.

How far can the argument stretch that "all known methods" should be used to avoid the risk of NANBH after transfusion? The bulk of NANBH may still be transmitted even after surrogate screening. Are we certain that patients would succeed in a legal action if they contract NANB hepatitis after the transfusion of blood untested for anti-HBc? Why should NANB post-transfusion hepatitis be such a special case that we have to make tremendous efforts to prevent occasional infections? What about the transmission by transfusion of agents such as cytomegalovirus or HTLV-I for which there are *specific* tests but which are not screened for routinely?

With regard to pooled plasma products, can anyone really feel confident that the 30% decrease in virus load predicted from US studies as a result of surrogate testing would have any obvious impact on transmission of NANBH when 70% of virus remains in a plasma pool?

How can the impact of transfusion-transmitted AIDS be compared with that of transfusion-transmitted NANBH, whose consequences seem minor? We doubt if the public (since consumer reassurance is being invoked) sees NANBH in the same light as AIDS. The significance of HBV and HIV infection are well known; the clinical importance of NANBH has to be sought.

Transfusion services must not bow to irrational pressure for measures whose efficacy is unproven. In the UK, transfusion centre directors resisted commercial pressure for premature introduction of unsatisfactory screening tests for anti-HIV; they should show the same resolution with NANBH.

At our transfusion centre 400 000 blood components are available for transfusion per annum. We have received an average of only 4 reports of NANB post-transfusion hepatitis annually for the past 10 years, and we repeatedly remind clinicians of the need to report infective complications of blood transfusion. A realistic estimate of the annual cost of surrogate screening for NANBH in the UK would be £9 million. Our colleagues' estimate of £2 per test (which test?) seems too low, and to it must be added the cost of counselling 3–4% of blood donors.

It is vital to extend the few available studies of transfusion recipients with more complete follow-up of untransfused patients to find out what role sporadic NANBH has in the hepatitis attributed to transfusion. And before we accept that 50% of cases of NANB post-transfusion hepatitis progress to chronicity and that 10% of chronic cases progress to liver cirrhosis, larger studies must be done. New studies are also essential because published data on NANBH transmission by transfusion relate to transfusions given before the "clean-up" of donor panels in the wake of the AIDS epidemic.

Even those arguing most forcefully for the significance of NANB post-transfusion hepatitis recognise that studies on the natural history of chronic NANBH have been limited both in size and in duration of follow-up and that the ultimate prognosis of this disease has not been established.[1]

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SIR,—The mandatory introduction of surrogate testing of blood donations in the USA with alanine aminotransferase (ALT) and hepatitis B core antibody (anti-HBc) as markers has raised the issue of whether such tests should be used by all transfusion services. Dr Anderson, Dr Gillon, and Dr Dow and their colleagues (April 18, p 912; June 13, p 1366) indicate that the cost-benefit of such testing is doubtful, given the reported low incidence of post-transfusion hepatitis in the UK.

A similar low incidence of reported post-transfusion hepatitis occurs in Ireland. However, we have studied the effect on blood supplies, the cost of introducing surrogate testing, and the cut-off level that is appropriate to our population. The use of a cut-off level determined elsewhere is arbitrary and may be misleading. We followed the recommendations of the American Association of Blood Banks (AABB) for the determination of this level.

4136 random blood-donors had their donated blood tested for ALT level at 25°C with a 'Viatron' sampler with Bio-Mérieux reagents. 140 samples were unsuitable for testing because of lipid interference, which left 3996 valid tests with a mean value of 12·9 IU/l. The AABB recommendation for determination of the cut-off level is the log of the mean normal plus two standard deviations. Our cut-off was therefore 30·7 IU/l. There were 109 donors with an ALT level higher than the cut-off, which resulted in a rejection (or deferral) rate of 2·7%.

We also tested a further 1000 donors for ALT levels at 37°C with an Abbott 'Biochromatic Analyzer' with Boehringer reagents. There was no lipid interference with this system. The mean value was 17·5 IU/l, and the cut-off was 39·6 IU/l. There were 25 donors with an ALT level over this cut-off, which resulted in a rejection rate of 2·5%.

The group of 4136 donors were also tested for anti-HBc by an enzyme-linked immunoassay (Wellcome Diagnostics). 42 were positive, which represents a rejection rate of 1·0%. 2 (0·05%) donors had both a raised ALT level and anti-HBc.

If ALT testing were introduced for routine screening of blood donors in our service, the deferral rate would be about 2·5%. If ALT and anti-Hbc testing were introduced, the deferral rate would be 1·0% plus 2·5% minus the overlap of 0·05%, equals 3·45%.

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SHOULD TRANSPLANT CENTRES EXCHANGE KIDNEYS?

SIR,—Professor Salaman and Mr Ross (June 27, p1480) question the need for transplant centres to exchange kidneys to improve donor/recipient matching. Their arguments, based on experience at Cardiff in 1985/86, are: (1) more kidneys were exported from Cardiff than were imported; (2) matching is of no real benefit; and (3) local kidneys perform better than imported ones, the latter having a high frequency of technical failures.

In any short period some centres will certainly be net losers, but just as many will be net winners. Perhaps in 1987/88 Cardiff will import more kidneys than they export. The second and third points are more serious and have led us to look at Newcastle-upon-Tyne experience in relation to matching and performance of imported kidneys.

We considered 310 consecutive first cadaver grafts done between 1979 and 1986 with at least 6 months of follow-up. Kidneys

GRAFT SURVIVAL RATES (ALL LOSSES)

	No	6 mo	1 yr	2 yr	4 yr
HLA-DR mismatches					
None	105	86%	84%	83%	81%
One or more	205	79%	74%	71%	67%
Source of kidney					
Local	150	82%	78%	74%	70%
Imported	160	81%	78%	77%	73%

beneficially matched on the HLA-DR locus (no mismatches) performed significantly ($p < 0·03$) better than the less well matched (table). Beneficial matching improved graft survival for both imported and local kidneys and was also advantageous for both conventionally and cyclosporin treated patients. In the cyclosporin group, for instance, one-year graft survival rates were 93% for no HLA-DR mismatches and 73% otherwise ($p < 0·001$). There were no differences between the total survival rates of local and imported kidneys (table), nor any significant increase in technical failures among imported (8·75% for imported, 6% for local). These findings were confirmed by a formal multifactor statistical analysis allowing for age, sex, dialysis time and method, pre-transplant transfusions, and immunosuppression, as well as matching and source of kidney.

We conclude that matching is well worthwhile and that imported kidneys perform as well as local ones do. With over half of the well-matched kidneys in Newcastle being imported we see a continuing need for exchange between centres. The frequency of technical failures associated with organ exchange does not exceed the benefits to be had and it is not time to call a halt to exchange.

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CRYPTOSPORIDIOSIS: A CASE OF AIRBORNE TRANSMISSION

SIR,—*Cryptosporidium* causes mild to severe diarrhoea, especially in children and in immunocompromised individuals.[1] Little is known about the epidemiology and pathogenesis of cryptosporidiosis. Zoonotic transmission is well recognised, and mammals, particularly cattle, are believed to constitute a major reservoir in certain areas.[2] Faecal-oral transmission is thought to be the major route in the transmission of infective oocysts from cattle to man, and from man to man.[1] We describe here a case of airborne transmission of *Cryptosporidium* oocyst causing gastroenteritis.

A veterinary scientist was caring for a calf experimentally infected with *Cryptosporidium* oocysts. The calf was severely dehydrated because of profuse watery diarrhoea, and in an attempt to save the animal, the scientist gave fluid therapy via a stomach tube. During the procedure she was wearing gloves and protective clothing, and great care was taken to avoid any possible contamination, especially since the scientist was still breastfeeding her baby. The only unprotected contact she had was when smelling for gastric odour, to check on the position of the tube. Neither before nor after this event has she had any other contact with calves experimentally infected with *Cryptosporidium* spp. 7 days later she had influenza-like symptoms with general weakness, nausea, loss of appetite, moderately increased body temperature, and generalised muscle pain. Abdominal pain and diarrhoea developed after a further 3 days. The diarrhoea was watery and non-bloody with 5–8 motions daily and lasted for 4 days with a weight loss of 4 kg. Abdominal cramps and bloating persisted for another 10 days, followed by constipation. A stool sample collected 16 days after exposure contained many *Cryptosporidium* oocysts while a sample 11 days later was negative. On day 60 post-exposure she had *Cryptosporidium* serum antibody titres of IgG 250 and IgM 64, in an indirect immunofluorescence antibody test (IFAT). Her breast-fed child became febrile with general malaise on day 27 but a stool sample was negative for *Cryptosporidium*. Breast milk on day 26 had *Cryptosporidium* antibody titres of IgG 50 and IgM 4 by IFAT.

There seems little doubt that this woman was infected as a result of inhaling droplets containing oocysts. *Cryptosporidium* spp can