Correspondence

Fatal *Yersinia enterocolitica* transfusion reaction

Although septicaemia is a rare complication of blood transfusion, episodes of transfusion associated sepsis may be fatal. A recent review demonstrated that about half of all reported cases of sepsis resulting from transfused erythrocytes involved *Yersinia enterocolitica* alone.¹ These transfusion reactions are presumed to result from a chain of coincidences in which a mild infection in the donor gives rise to a transient bacteraemia during donation. As *Y. enterocolitica* is one of the few human pathogens that can grow at 4°C, after storage for one to three weeks at 1–6°C a unit of blood could contain numerous bacteria and associated endotoxin.

Human *Y. enterocolitica* infections are particularly frequent in Belgium.² Mild diarrhoea is a common manifestation of *Y. enterocolitica* infection and it often goes unrecognised. After the enteritis the organism may persist for some time in mucosal, submucosal or lymphoid tissues, and give rise to episodes of symptomatic or cryptic bacteraemia.³

We report a case of fatal *Y. enterocolitica* septicaemia in an 82 year old man caused by a contaminated unit of red cells that was collected from an apparently healthy asymptomatic blood donor. The patient had a history of severe cardiovascular disease and chronic renal insufficiency. Three weeks before admission to hospital he developed atrial flutter for which coumarin treatment was started.

On 19 August 1995, the patient was admitted to hospital because of a two day history of anal blood loss, abdominal discomfort, and vomiting. He was haemodynamically stable but blood examination revealed an international normalised ratio (INR) of 10.8 and a haemoglobin of 88 g/l. Colonoscopy revealed a tumour of the ascending colon as cause of the bleeding. The patient was treated with vitamin K. Even after correction of coagulation (INR 1.53), blood loss per anum persisted and haemoglobin further decreased to 73 g/l. One unit of packed cells was given. During the transfusion the patient developed a temperature up to 38.7°C. After transfusion of about 200 g red cell concentrate, the transfusion was stopped and three blood culture sets were taken. Also a sample from the unit of packed red blood cells was inoculated on a separate culture set. Meanwhile the transfusion bag was stored in the refrigerator. The fever was transient, but a few hours later the patient developed shock with hypotension and dullness. Plasma expanders, sympathicomimetics, and antibiotics (amoxicillin and gentamicin) were started, but shock and multiorgan failure were progressive. The following day, after incubation at 36.5°C, there was growth of motile Gram negative rods in the culture set inoculated with the packed red blood cells. These were identified as *Y. enterocolitica* serotype O:9, biotype 2. Therefore, antibiotics were switched to fluoroquinolones. The patient died four days after the transfusion because of septicaemic shock.

Identification was confirmed by the Belgian Reference Laboratory for *Yersinia* (G Wauters, Université Catholique de Louvain,

Brussels). In addition, blood from the blood-bag side tube of the transfusion bag was inoculated on an aerobic blood culture bottle and remained sterile. Absence of *Y. enterocolitica* in the low volume tubing segment is consistent with a low level of bacterial contamination at the time of collection.

The donor of the blood sample was traced. He was a 58 year old healthy man who had spent his holiday in Switzerland the week before the blood donation. He did not recall any gastrointestinal disturbance, nor did any member of his family. However, he had been very tired after his last blood donation on 31 July 1995, possibly indicative of a subclinical infection. A coproculture was performed 50 days after the donation but it was negative for enteropathogens even after prolonged enrichment on modified Rappaport medium. Serum taken at the same time demonstrated an antibody titre against *Y. enterocolitica* serotype O:9 of 1:200 suggesting a recent infection as serum taken at the time of donation showed no agglutination.

About 40 cases of transfusion reaction caused by *Y. enterocolitica* have been described in the English language literature with a mortality rate higher than 60%. *Y. enterocolitica* is the organism most commonly implicated in red cell related transfusion sepsis.⁴

The association between *Y. enterocolitica* and transfusion related sepsis can be explained by the fact that this microorganism can grow at refrigerator temperature, and by the stimulation of its growth by exogenous iron (in most reported cases red cell units were more than 25 days old). After several weeks of conservation these units may contain sufficient free haemin to stimulate the multiplication of *Y. enterocolitica*.⁵ The unit with packed red blood cells transfused in our patient was three weeks old.

A few studies were done regarding the growth and endotoxin production of *Y. enterocolitica* in packed erythrocytes. When such units were inoculated with low levels of *Y. enterocolitica* (0.1–1 colony forming units (cfu)/ml) the organism proliferated to high titres (> 103 cfu/ml) after a lag period of 10–20 days. Endotoxin was detected only after three weeks of conservation.⁶

Several solutions have been proposed to prevent this life threatening infection. Questioning the donors about gastrointestinal symptoms does not appear a sensitive predictor of *Y. enterocolitica* occult bacteraemia. Only two thirds of donors implicated in *Y. enterocolitica* transmission recalled gastrointestinal illness before donation.⁷ Serology gives numerous cross reactions and false negative results in acute infection.⁸ A decrease in storage time of packed red blood cells to less than three weeks would have a devastating effect on blood supplies. Also, the addition of antibiotics may cause greater problems than it resolves because of anaphylaxis or other adverse drug reactions. Culturing of all units would be logistically very difficult. Furthermore if culture is done soon after donation, when fewer than one organism per sample volume is present, it will not be detected.

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Alcohol estimation at necropsy: epidemiology, economics and the elderly

Having read this paper¹ with interest, we feel that it raises several important issues pertinent to necropsies performed on behalf of HM coroner or the procurator fiscal, of which the authors appeared to be unaware.

It is a common misconception that, given the apparently rich source of material from coroners' necropsies, forensic pathologists seem reluctant to undertake any research using this material. In Britain there have been no previous publications concerning blood alcohol levels in a medicolegal necropsy population because there is no provision under present law for such a study.

Postmortem examinations in England and Wales are carried out either at the request of the coroner, using powers as set out in the Coroners Act 1988² or, with the permission of the person lawfully in possession of the body, under the conditions set out in the Human Tissue Act 1961.^{3,4} In Scotland, the procurator fiscal's power to request a post-mortem examination is grounded in common law rather than statute. The Human Tissue Act 1961 applies to Scotland as to England and Wales.

Where a coroner has decided to open an inquest, he or she may direct that samples be removed from the body during the course of the postmortem examination for "special examinations" including toxicology.⁵ The person carrying out the postmortem examination is required to preserve material that bears upon the cause of death for as long as the coroner thinks fit.⁶ In England and Wales, the sole purpose of the coroner's post-mortem examination is to assist the coroner in inquiries that are essentially limited to who the deceased was and how, where, and when they came to their death. Tissue cannot be removed or preserved for any other purpose under the coroner's authority. In Scotland, the procurator fiscal's enquiries are directed towards ascertaining "the truth or otherwise of the information given to him as to the death: to investigate the circumstances impartially and in the public interest without fear or favour and to get to the truth: to ensure that any dangerous or faulty practices are exposed so as to prevent their recurrence: to preserve from corruption the sources of evidence: to ensure that homicide does not go undetected and to make, when required, a true report to the officers of the Crown".⁷ Neither the coroner nor the procurator fiscal

have any authority to authorise the collection or retention of specimens for other purposes, including research. If samples are required for teaching or research, then the requirements of the Human Tissue Act have to be met, whether the necropsy be a normal hospital one or a medicolegal postmortem examination. When the examination is a medicolegal one, then the removal of material for research requires not only the consent of the person in lawful possession of the body, who, in the absence of a request by the deceased that his parts of his body be used for purposes of inter alia research, has to establish that the surviving relatives of the deceased have no objection to the removal of material for that purpose,³ but also that of the coroner or the procurator fiscal.⁴

While there is no sanction for failure to comply with the Human Tissue Act set out in the Act itself, the common law crime of disobedience of a statute may be relevant. A man who impersonated a medical practitioner was successfully prosecuted for disobedience of a statute after he removed the eyes from a body contrary to section 1(4) of the Human Tissue Act 1961.⁵

In the absence of a history of alcohol abuse or a death in which alcohol is considered not to have contributed or caused death, the retrieval, retention and analysis of samples for alcohol content falls outside present legislation. Of 1730 cases quoted in this paper, 1046 were deaths from natural disease.¹ Only 91 of these had a history of alcohol use; therefore, it appears that the remaining 955 analyses were undertaken with no legal authority.

Given the quoted price of each test (£15), it seems unfortunate that the authors felt that they needed to analyse 1046 cases to prove what most coroners and full time forensic scientists are well aware of; namely that routine testing for alcohol is not cost effective. Some pathologists believe that the retention and analysis of material from medicolegal necropsies is not justified where the coroner refuses to pay for such work. Coroners can only pay for further investigations when they have decided to hold an inquest and they are satisfied that they are pertinent to the cause of death. Pathologists should be aware that retaining material not relevant to the cause of death places them out with current legislation.

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Oil immersion magnification without the oil

Most of us have a 100× oil immersion lens as part of our standard microscope kit. However, many, like me, must be reluctant to use it very often because of the inevitable mess, and the inability to switch between high and low power without cleaning the slide every time. These days, many demonstration microscopes are fitted with a video camera and monitor, and I have found that this equipment allows the use of the 100× lens without oil. Simply closing the iris diaphragm of the condenser and observing the image on the video screen rather than through the eye pieces results in an acceptable high power view. The use of oil does, of course, improve the quality of the image, but the video screen method without oil provides a high power image of a quality that is adequate for many diagnostic or demonstration purposes. The cost of video cameras and monitors has fallen steadily over recent years, and it could be argued that the purchase of a video system is better value for money than an expensive high power oil free lens, assuming that there is already a 100× oil immersion lens fitted, allowing high power magnification with and without oil, and the added benefit of video.

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Book reviews

Pathology in Dentistry. E Sheffield. (Pp 281; £56.95.) Oxford Medical Publications. 1996. ISBN 0 1926 2421 0.

This is a brief but comprehensive text. The first four chapters cover general pathology and the remaining 12 deal with systemic disease. The text is confined to the medial two thirds of each page giving an impression of rather uneconomical use of paper although the broad margins clearly will be very useful for annotation. Where appropriate the margins contain photomicrographs, gross photographs, tables, and diagrams. The photographs are of extremely high quality but some suffer from a rather small size and less than adequate labelling.

A particularly valuable feature is the introductory paragraph at the start of most of the chapters on systemic disease, which gives an overview of the pathological principles peculiar to that system together with a summary of normal structure and function. Each chapter is followed by a helpful and succinct summary box.

There are indexing errors and some text references to a topic treated elsewhere are followed by (P000) instead of the page number. The text is clear and readable. The multitude of demands on undergraduates' time makes one question whether a dental student should be required to have such a wide knowledge of pathology, but as long as those who design the curriculum deem that this should be so there will be a definite place for this textbook. At £56.95 it is good value

and should be seriously considered by both medical and dental undergraduates as an introduction to pathology.

L W L HORTON

Clinical Reproductive Medicine. Cowan BD, Seifer DB, eds. (Pp 400; £60.75.) Lippincott-Raven. 1997. ISBN 0 3975 1186 8.

This is a very elegantly produced volume that should prove popular in its target market—those seeking to become board certified in reproductive endocrinology in the USA. Thus it contains all of the appropriate background material for specialist examinations, including basic reproductive physiology and biochemistry. The student is greatly helped by the numerous original diagrams, though it is not immediately obvious why some figures are duplicated at the beginning of each chapter, other than as pure decoration. From a factual point of view the material is relevant and up-to-date, though obviously it is possible to criticise a number of omissions. For example, there is no material on the receptor deficiency syndromes, a very active clinical research topic at the present time. There is also no mention of the use of progesterone containing intrauterine devices for the treatment of dysfunctional uterine bleeding (perhaps reflecting the US origins of the book), and very limited information on the immunological treatment of recurrent abortion (which is surprising because this is very widely practised in the US). Overall, however, the book can be recommended to a young clinician seeking to become better informed in this particular topic.

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Physician's Guide to the Laboratory Diagnosis of Metabolic Diseases. Blau N, Duran M, Blaskovics ME, eds. (Pp 508; £65.00.) Chapman and Hall Medical. 1996. ISBN 0 4125 7604.

In the preface to this book the editors state that most books dealing with inborn errors of metabolism are detailed, complex, time consuming, and difficult to comprehend. Their target audience are general physicians whose knowledge of metabolic disease is not large. As more and more metabolic disease variants present in the older age groups adult physicians will occasionally come into contact with these disorders. However, genetic metabolic disease and its diagnosis will remain mainly in the hands of specialist paediatricians and laboratories. I cannot see changes in health delivery systems affecting this, and lawyers would have a field day if it did. On the other hand successful treatment of many of these disorders will result in an increasing adult population requiring treatment.

A prestigious list of international authors (regrettably few from the UK) has been assembled by the editors. Clearly a lot of thought and a great deal of work has been put into the construction of the chapters and the result is a uniform structure involving nomenclature, background biochemistry, clinical signs and symptoms, diagnosis, and treatment. This makes it easy to find one's way around initially, however, when one attempts to get into the detail of the book it is very obvious that it is highly condensed and far from simple. For example, many general physicians faced with a metabolic pathway with