Fears have basis in reason

Entorr—A deep seated fear may yet be rational. The fear of being declared dead while still alive, in the case of “brain dead” patients, is a fear with a basis in reason.1 If such patients are not dead, they certainly will be after unpaired vital organs are removed for transplantation. Rather than being “settled,” the acceptability of criteria for brain death is the subject of intense international debate.

As early as 1974, the philosopher Hans Jonas wrote in opposition to brain death criteria; a lengthy article by Byrne et al followed nine years later (reprinted in an anthology by Potts et al). More recently, the neurologist Alan Shewmon reversed his previous support for brain death criteria.2 In 1990, the anthology Beyond Brain Death was published, with contributors from the United States, the United Kingdom, Japan, and Liechtenstein.3

There are many reasons for this growing opposition. Shewmon’s accounts of long term survivors of whole brain death empirically falsify the claim that whole brain death marks the end of a unified human organism.4 Even if Shewmon’s claims are unfounded, a prognosis that brain death will lead to immanent somatic death (in the sense of the loss of a unified functioning organism) is not the same thing as a diagnosis that somatic death has occurred (Byrne et al). Evans, among others, discusses the mounting evidence that brain activity persists in a number of patients declared brain dead.5 Coimbra identifies the dangers in a test used to diagnose brain death, the apnoea test.6 Philosophers, such as Hans Jonas and Josef Seifert, have attacked the dualism of brain and body (Jonas’ term) that is inherent in criteria for brain death.7

This debate should raise serious doubts concerning whether brain dead people are dead and lead to a rethinking of the entire enterprise of removing vital organs from such patients. A fundamental goal of medicine is to do no harm (non-maleficence). Any action that directly causes the death of a patient, even if it is for the good of others, opposes the goal of medicine not to harm the individual patient. Any attempt to downplay the importance of the brain death debate in the interests of organ transplantation is therefore fundamentally wrong. It is precisely whether transplantation kills the donor that is the key issue that cuts to the heart of the goals of medicine.

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Staphylococci may indeed cause acute dental infections

Entorr—In their letter Ribeiro and Cousin question the likelihood of a causal relation between the development of an epidural abscess after root canal treatment and the isolation of Staphylococcus aureus in the report by O’Rourke et al.8

We disagree and support the original proposal of O’Rourke et al because recent studies using both molecular technology and conventional culture techniques indicate conclusively that Staphylococcus spp are not uncommon in dental root canal infections.9 Furthermore, in a stringent and comprehensive investigation that we are currently conducting on the microflora of endodontically involved teeth, staphylococci were isolated from root canals in eight out of 86 patients (unpublished data). Indeed in two cases, staphylococci were the sole and major isolate from the asepically opened, infected root canals.

We emphasise, however, that most endodontic infections are polymicrobial in nature. Using the polymerase chain reaction, Munson et al recently found a mean of 17 taxa in endodontic samples and a total of 30 new phylotypes.10 Therefore, acute dental infections are still best treated by antimicrobial agents such as penicillin, amoxicillin, cindamycin, and metronidazole, but the possibility of staphylococci causing acute exacerbations or bacteremia should not be overlooked.

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1. Ribeiro N, Cousin G. Staphylococci are unlikely to cause acute dental infections. BMJ 2002;324:1477. (15 June.)

Children are still seen but not heard

Entorr—Kroenke and Gask and Underwood describe in some detail the multiplicity of psychological presentations in patients.1 No mention is made of the relief of the many children seen in general practice and by paediatricians who have primarily mental health problems, psychological factors associated with their physical illness (such as diabetes management), or medically unexplained symptoms (such as recurrent abdominal pain). The best estimate of the prevalence of psychiatric disorders in young people in the United Kingdom is the Office for National Statistics figure of 10% of all children.2 This figure increases appreciably in inner cities and when chronic ill health, and particularly neurological disorders, are present. But only one in five of these attend mental health services for children and adolescents. Some evidence exists that family doctors can provide effective treatment for this group.3

Children are in the unique position of being presented to their family doctors by parents, rather than seeking help themselves. They are therefore vulnerable in “the consultation” to the effects of mental illness and personality disturbance in their parents, which can range from the genuinely (over-) anxious to the homicidal.

Quite apart from the genetic transmission, the psychosocial impact of parental mental illness, separation, and divorce is considerable and may well present as psychosomatic symptoms in vulnerable children. At the other end of the scale, a few parents and carers actively harm children and then seek help for their illness.

Patterns of help seeking behaviour are established in childhood. Practising psychological medicine in childhood may well reduce undesirable adult patterns of health care use. If it is to truly encompass the whole person, child and adult, psychological medicine must consider all members of the family, not only adult patients.

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References from quality of life scales are not simple

Entorr—As someone who is guilty of adding to the large number of disease specific quality of life (QOL) scales, let me add a note of caution to the pleas made by Garratt et al for guidance and recommendations for the users of these scales—however understandable that plea is.1 QOL scales are not like thermometers or spirometers, where the reading is independent of the type of patient.

A QOL scale is just a shopping bag of experiences (or questions) that are put together to form a scale, rather like the retail price index. The retail price index is a shopping bag of goods for an “average” shopper, even though most people are not that average shopper. The scale value obtained from a QOL scale depends on the overlap between the items in a scale and the patient’s own experience of disease. So, for example, if there is a generic QOL scale and there are many pain items but no items on sleep disturbance, then arthritis will come out worse than asthma. The same logic applies to disease specific scales. If there are


Children who present with disease-specific QOL scales are often asked to compare their illness with that of others, but this comparison may only be meaningful when the illnesses being compared are similar. For example, children may be asked to compare their asthma to the asthma of other children as a guide to whether their asthma is a problem. If the QOL scales are not accurately reflecting the children’s own experience of their illness, then they are not differentiating between children who have similar experiences of asthma and children who have different experiences of asthma. This leads to the question of whether the QOL scale is valid for use in these children. If it is not valid, then the QOL scale is not providing the information that is needed to guide the child’s treatment. Therefore, it is important to ensure that the QOL scale is valid for use in these children before it is used to guide the child’s treatment.