less sensitive to early rejection than the biopsy procedure. Acute rejection episodes confirmed histologically are treated by increased immunosuppression using methyl-prednisolone, actinomycin D, and ATG. Heparin is also given to prevent acute vascular occlusions during this period. The technique of cardiac transplantation remains that described from Stanford in 1969.[7]

After discharge from hospital patients are followed closely and maintained on a low-cholesterol, low-sodium diet and warfarin. Antiplatelet agents are used to prevent platelet aggregation and their deposition on the endothelium, which leads to atheroma-like occlusion of the arteries in the grafted organ. (The development through this process of cardiac ischaemia in the cardiac graft does not produce clinical angina because the heart is denervated.) Retransplantation has been used to treat both advanced atherosclerosis in the graft and intractable rejection. Seven patients so far have undergone a second transplantation, three of them successfully.

So the one-year survival rate at Stanford has improved from 42% in 1968 to 68% in 1977. Infection caused a third of all deaths (the lungs being the most common site), and the Stanford workers point out that infection is more common after cardiac than after renal transplantation because more intense immunosuppression is needed to maintain cardiac function during rejection. Rejection itself accounted for only a tenth of deaths.

Enlightened lay recognition of brain death in California led to legislative action which permitted the removal of the beating heart from brain-dead donors back in September 1974. Recent techniques of cardiac preservation after removal have enabled the hearts to be transported (up to 450 miles so far) to the operating room at Stanford. (Only last month a kidney was brought from the United States to Britain by Concorde.) In Britain the clinical recognition of "brain death" was clarified by a conference of Royal Colleges and Faculties, whose statement[8] we published in 1976. The diagnostic criteria for brain death have now been accepted as being sufficient to distinguish between those patients with a chance of even partial recovery "from those in whom no such possibility exists."

In a letter to all hospital doctors, administrative medical officers, and hospital administrators the Chief Medical Officer wrote that "once the diagnosis of death has been made the actual moment at which a respirator is switched off may be influenced by the need to maintain the kidneys or other organs in the best possible condition before they are removed for an eventual transplant; this will remain a matter for the judgment of the doctor in clinical charge of the potential donor. In all cases the requirements of the Human Tissue Act (1961) must be met." It is distressing that despite this circular there is a continuing desperate shortage of kidneys, most organs for transplantation coming from the same minority of hospitals—the ones who have received the message and acted on it.

The success rate for cardiac transplantation at Stanford is now comparable to or superior to that reported for renal transplantation from unrelated donors, and thanks to the unrelenting efforts of Shumway and his team over nearly two decades the operation can now provide real palliation for patients with terminal heart disease. The feasibility of transporting donor hearts has transformed the possibilities for obtaining viable donor organs—provided that public and professional ignorance about brain death can be overcome.

This time around Stanford's success will not be the signal for a new series of sporadic transplants because it is now understood that their success has been achieved only as a result of massive research and team work. The emergence of any centre in this country should be on a similar basis: that is, an established successful major cardiac centre, preferably placed close to units already transplanting other organs; a centre wherein there is already a continuing research programme with full support services in immunology, pathology, and microbiology; and, of course, without prejudice to its regular service programme in cardiovascular surgery.

[1] Barnard, C N, *South African Medical Journal*, 1967, **41**, 1271.
[2] Griepp, R B, *et al*, *Surgery*, 1971, **70**, 88.
[3] *British Medical Journal*, 1967, **4**, 757.
[4] *British Medical Journal*, 1968, **2**, 315.
[5] Goodwin, J F, and Oakley, C M, *American Heart Journal*, 1969, **77**, 437.
[6] Hunt, S A, *et al*, *Circulation*, 1976, **54, suppl III**, 56.
[7] Stinson, E B, *et al*, *American Journal of Surgery*, 1969, **118**, 182.
[8] *British Medical Journal*, 1976, **2**, 1187.

Drinking and drowning

A problem is not a problem until it reaches sufficient prominence in the public eye to become one—and then it becomes fashionable. For example, how big a part does alcohol play in the deaths of the 150 000 victims of drowning around the world each year? Evidence is accumulating that drinking is a factor in many of these deaths, but its importance varies with nationality and geography.

In the Australian city of Geelong Plueckhahn reported[1] that three-quarters of the men aged 26 or over who had drowned had taken alcohol shortly beforehand; at necropsy over half of these had a blood alcohol concentration of 100 mg/100 ml or more. Giertsen[2] stated that one-third of adults who drowned in Finland had been under the influence of alcohol, and claimed that half the Norwegian seamen who drowned were in a similar state. In England and Wales the Home Office report[3] states that alcohol was associated with drowning in 14% of cases and in 29% in Scotland. Careful analysis by the Home Office working party showed that in 1974-5 alcohol was a direct contributory factor in 77 out of 526 drowning accidents. Of these, 45 people had fallen into water while walking alone and 24 while in the company of friends, while 16 were seamen returning to ship who had fallen into the harbour. Only eight had entered the water deliberately. In other words, in those accidents in which alcohol was known to be an important factor 90% of those who drowned were so drunk that they fell into the water.

Moreover, the figures suggest that the association between drinking and drowning is underestimated, since blood alcohol concentrations are not always estimated and a reliable history may not be available. More accurate data are required. The Australian Surf Life Saving Association, whose members save thousands of bathers from the water each year, uses a resuscitation report form filled in on the spot by the lifeguard. In one recent series of these reports at least a third of 67 survivors of immersion who required cardiopulmonary resuscitation on the beach had recently drunk alcohol.[4]

Quite apart from its effect of increasing bravado while diminishing the ability to cope physically, alcohol exerts a more subtle influence on the survival chances of the victim. It depresses hepatic gluconeogenesis, so that when, after physical exertion, the glucose stores are consumed the blood sugar concentration will fall. Haight and Keatinge[5] have shown that not only may this fall in the blood sugar concentration be profound, but that the resulting hypoglycaemia impairs the

temperature control mechanisms, leading to rapid cooling. The process can be corrected by restoring the blood glucose concentration to normal. Hypothermia is a more common cause of death in the open sea than drowning.

The important points that emerge from these observations are, firstly, that people who drink alcohol, even in moderate amounts, before entering the water are at risk; secondly, that this risk applies to swimmers and sailors alike, summer or winter; and, thirdly, that the public is apparently unaware of these dangers. Perhaps water safety organisations should emphasise the dangers of drinking rather than giving the time-honoured advice about eating before swimming—which has not yet been shown to place the victim at risk.

[1] Plueckhahn, V D, *Medical Journal of Australia*, 1972, **2**, 1183.
[2] Giertsen, J C, *Medicine, Science, and the Law*, 1970, **10**, 216.
[3] Home Office, *Report of the Working Party on Water Safety*. London, HMSO, 1977.
[4] Surf Life Saving Association of Australia, *Resuscitation Report Form Statistics 1974-7*. Sydney, Surf House,1977.
[5] Haight, J S J, and Keatinge, W R, *Journal of Physiology*, 1973, **229**, 87.

Drugs in threatened preterm labour

Spontaneous preterm labour (21 days or more before term) occurs in about 7% of pregnancies in Britain—but 36% of perinatal deaths are in this group.[1] The risks are highest and the problems greater in those women whose labours begin before the 32nd week of pregnancy and whose babies weigh under 1·5 kg, for these very small infants are most vulnerable to the disorders associated with extreme prematurity and they require specialist facilities if they are to survive undamaged.

Many drugs have been claimed to suppress preterm labour only to be discarded in the light of continued experience. In the last few years, however, interest has centred on prostaglandin inhibitors and beta-sympathomimetic agents, both of which have a rational basis of action and are widely used. Indeed, in West Germany alone one million ampoules and six million tablets of one beta-sympathomimetic drug (Feneterol) are said to be used each year[2]—yet this has not been associated with any fall in the numbers of infants of low birthweight.

A recent review[3] looked at 18 clinical trials of hormones, ethanol, or beta-sympathomimetic agents. In only five of these trials were the drugs used therapeutically rather than prophylactically and compared with placebo in a design that was adequate. In only two trials was the drug found more effective than placebo in postponing delivery, and a favourable effect in terms of fetal outcome was found in only one. One of the more satisfactory trials was that of Wesselius de Casparis *et al.*[4] In this double-blind controlled trial preterm labour was postponed in 80% of patients receiving ritodrine (a beta-sympathomimetic drug) compared with 48% in the placebo group.

Perhaps we expect too much of these drugs if the effect sought is delay of labour for several days if not weeks. Inhibiting uterine activity for even a short period may be of great value if the time gained is used to give the child a better chance of survival. The respite may, for instance, be used to give corticosteroids to reduce the risk of respiratory distress syndrome,[5] or to transfer the patient, if necessary, to a centre with special paediatric facilities. Furthermore, the tacit assumption that inhibiting preterm labour is necessarily beneficial should not go unchallenged. Indeed, preterm labour may often be nature's best option, in that the precipitating cause may be acute or chronic impairment of placental function. The condition of the fetus should be carefully evaluated, using cardiotocography, before the decision is made to attempt to inhibit uterine activity.

The drugs used may themselves have detrimental side effects. For example, treatment with prostaglandin antagonists may lead to premature closure of the fetal ductus arteriosus with resultant pulmonary hypertension.[6] Adverse effects on the mother from beta-sympathomimetic agents may include tachycardia, palpitations, and hypotension. Drugs such as ritodrine, which are relatively selective for beta receptors, have fewer cardiovascular effects in proportion to their action on the beta receptors of the myometrium. A combination of a beta-mimetic agent and corticosteroids may cause maternal pulmonary oedema and right-sided heart failure in susceptible patients.[2 7] In such cases there may be an underlying cardiomyopathy; a careful examination for cardiac disease is needed before treatment of this kind is given.

On balance and in terms of fetal outcome the use of drugs to inhibit labour is usually unnecessary, frequently ineffective, and occasionally harmful. Indeed, when all cases of threatened and progressive preterm labour are analysed retrospectively, specific treatment to try to stop labour is found to be of potential value in only relatively few patients, either because of complicating factors indicating a need for delivery or because the patient is in advanced labour at the time of admission. Improvement in perinatal mortality and morbidity is more likely to come from concentrating efforts on the identification of high-risk pregnancies, on early admission, and on measures to ensure that infants at risk are delivered in optimum condition in centres of perinatal skill.

[1] Chamberlain, G, *et al*, *British Births 1970*, vol 2. London, Heinemann, 1978.
[2] Kubli, F, in *Preterm Labour*, ed A Anderson *et al*, p 218. London, Royal College of Obstetricians and Gynaecologists, 1977.
[3] Hemminki, E, and Starfield, B, *British Journal of Obstetrics and Gynaecology*, 1978, **85**, 411.
[4] Wesselius de Casparis, A, *et al*, *British Medical Journal*, 1971, **3**, 144.
[5] Caspi, E, *et al*, *British Journal of Obstetrics and Gynaecology*, 1976, **83**, 187.
[6] Rudolph, A M, in *Preterm Labour*, ed A Anderson *et al*, p 231. London, Royal College of Obstetricians and Gynaecologists, 1977.
[7] Elliott, H R, Abdulla, U, and Hayes, P J, *British Medical Journal*, 1978, **2**, 799.

Common waiting lists

One of the more futile political interventions in the NHS has been this Government's exclusion of private practice from the hospital service.[1] It angered consultants already demoralised by deteriorating standards of hospital care and, paradoxically, has since boosted private practice outside the NHS. Yet, as the profession is tired of pointing out, private beds formed barely 2% of total NHS beds.[2]

The Health Services Board—surely one of the country's least constructive Quangos (Quasi Autonomous Non-Governmental Organisations)—is grinding steadily ahead reducing beds and facilities for private patients (p 146), and just before Christmas[3] the Government announced the first step towards fulfilment of another of its pet objectives: a scheme for a common waiting list for urgent and seriously ill