50 mg and heparin 10 000 U are given 30 minutes before the expected time of removal. Mannitol acts synergistically with both these drugs and may be included in the regimen in future.[18] Phenoxybenzamine is supplied by the transplant surgeon since the drug is now available only under special licence. A single dose of 100 mg is given intravenously five minutes before removal. A long-acting muscle relaxant such as pancuronium 4 mg may be given at the same time to prevent contractions of the abdominal muscles at laparotomy.

Postmortem examination should be carried out on every donor to exclude occult neoplasm, which might affect the health of the recipient.

Conclusion

The techniques and laws relating to organ removal from a heart-beating donor are well known; public attitude is sympathetic, and relatives seldom refuse to give consent for donation; and statistics show that there is a surplus of potential donors. Nevertheless, the supply of viable cadaveric kidneys falls far short of the need. The fault lies with the medical profession and must be due to lack of knowledge and skills or to inappropriate attitudes of hospital medical staff. Table III lists the requirements for participation in a transplantation programme.

TABLE III—*Skills, knowledge, and attitudes required for participation in transplantation programme*

Skills:
IPPV
Monitoring circulation
Appropriate fluid treatment
Knowledge:
Organisational protocol for donation
Legal aspects of donation
Awareness of shortage of donor kidneys
Attitudes:
Obligations versus rights
Attitudes towards organ donation/brain death/transplantation programme

The relevant knowledge and techniques have been described. Appropriate attitudes can be learned only from the example of others.

We have shown that a district general hospital can make appreciable contributions to a transplantation programme. The procedure has been facilitated by the use of flow charts. These prevent errors and simplify and hasten the procedure. In our hands the time taken from the diagnosis of brain death to the removal of kidneys ranges from three to six hours.

One estimate suggests that a small district general hospital should be able to supply six donors a year.[1] We calculate that if other hospitals of comparable size were to follow our example there would be a surplus of donor kidneys. This would allow surgeons to choose only those kidneys removed under optimal conditions for transplantation. The initial high failure rate would be expected to improve. A far greater number of patients undergoing long-term dialysis could be offered transplantation and hence the chance of an improved quality of life. Moreover, with a faster turnover more patients with chronic renal failure could be accepted for dialysis.

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SHORT REPORTS

Persistent psychiatric symptoms after eating psilocybin mushrooms

Abuse of the hallucinogenic mushroom *Psilocybe semilanceata* was described by Hyde *et al.*[1] I report a case in which persistent panic attacks developed after eating psilocybin mushrooms.

Case report

A 24-year-old man presented to the outpatient department with a three-month history of attacks of tension, anxiety, fear that something was about to befall him, depersonalisation, palpitations, bounding pulses, dryness of the mouth, and "butterflies in the stomach." Sometimes the attacks were accompanied by disturbed vision—"everything becomes misty, as though I am looking into a fog." Two weeks before onset he had eaten 25 psilocybin mushrooms in the company of friends in a pub, where he had drunk two pints of beer. Three hours later he had become emotionally labile. One of his friends had asked, "Where's Howard?" He repeated the question twice. "Then I reeled back. It was all black behind him. The whole room blacked out then cleared in seconds." The patient felt well next day.

The first attack occurred while the patient was talking to workmates on the first day of a new job and lasted for a quarter of an hour. He had a further attack that evening. Episodes occurred daily since then. He controlled the symptoms with lorazepam 2·5 mg, without which they could have lasted all day and into the night. Chlorpromazine 50 mg twice daily had no effect.

The patient had no other psychiatric history. He described a happy childhood and life and was socially well adjusted. In the months before his illness he had been subjected to stress in interpersonal relations. He had eaten psilocybin mushrooms on two other occasions and smoked cannabis infrequently—all without ill effect. Since the onset of his illness he had felt suicidal several times.

Comment

Psilocybin is a class A drug under the Misuse of Drugs Act 1971. Charges have been brought for possessing the mushroom. Judge Blomefield ruled at Reading Crown Court in 1976 that because the mushroom itself is not mentioned in the Act possession is not an offence. In September 1977 Goodchild successfully appealed to the law lords against a conviction for possessing a marijuana plant, albeit without the flowers and fruiting tops then mentioned by the Act.

The law was subsequently changed to make possession of the plant illegal. Thus unless the Act is amended to name the psilocybin mushroom possession is legal.

Apparently increasing numbers of teenagers and others are harvesting and eating the mushrooms.[2] Doctors should therefore be aware of possible long-term side effects of such agents. One of these is the flashback phenomenon described by about 5% of LSD users.[3-5] The development of severe and uncharacteristic anxiety symptoms, as in the present case, may be another.

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Steam sterilisation of sandpits infected with toxocara eggs

Public parks, playgrounds, and children's sandpits contaminated with toxocara-infected faeces of pets poses a public health problem.[1 2] Embryonated toxocara eggs may infect man (children forming a bad-risk group) and cause the so-called larva migrans syndrome.[3] Under optimal conditions eggs mature to the embryonated and infectious forms in three weeks, and in the temperate European climate this process occurs only in summer time. For this reason public sandpits are replenished with new sand each year. This laborious and expensive process has only a transient useful effect because contamination is continuous. Hence ideally a cheap method of killing the embryonated eggs and possibly other micro-organisms while leaving the sand in the pit should be employed. For practical and public health reasons chemicals cannot be used, but a good alternative is to heat the sand in the pit, since thick-shelled nematode eggs are destroyed by pasteurisation.[4] We have tried to sterilise sandpits by steam and report our results.

Methods and results

Steam at a temperature of 160°C and a pressure of about 10 atmospheres was introduced under thick plastic covers. Initially steam treatment was carried out for five hours to ensure that the high temperature had reached the depth of the pit. Samples (weighing 150-300 g) from the surface and from a depth of 40 cm were collected before and after treatment for examination for toxocara eggs. The latter were isolated by sieving followed by zinc-sulphate flotation and filtration through a Millipore (5 μm) filter. After counting and assessing the stage of development the eggs were incubated in moist chambers for three weeks at 28°C, their maturation being examined under the microscope every week.

All samples contained toxocara eggs, most being found in the superficial layer. None of the eggs had undergone any natural development. This was to be expected since we performed the sampling in January. Maturation to the embryonated stage during laboratory incubation at 28°C occurred in 30% of the eggs from the superficial sample taken before treatment. No development took place in eggs after treatment.

Pasteurisation (20 minutes at 73°C)[4] or temperatures of 60°C for one hour[5] will kill nematode eggs, and hence possibly treatment time might be reduced. For this reason we monitored the temperature continuously at several points and different depths during steaming. In the superficial layer of the pit (up to 15 cm) the temperature reached an equilibrium of 100°C within half an hour (table). At lower levels an equilibrium of a lower temperature was reached later. From these data an appropriate steaming time may be calculated. If one hour at 60°C is enough to kill the eggs, the treatment time (S, in hours) should be $S = d/15 + 1$ (where d is the depth in centimetres).

Temperature (°C) in a sandpit at different depths during steaming at 160°C

Depth in cm	Steaming time in hours				
	1	2	3	4	4½
15	100	100	100	100	100
30	42	58	68	74	76
45	26	44	60	70	75
60	16	34	48	60	66

Comment

As well as giving satisfactory results, steaming is very economical, and given all the costs (wages, fuel, water, electricity, etc) cheaper than removing 15 cubic metres of sand. Moreover, treatment can be repeated several times in the warm season and hence safety can be maintained throughout.

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Extreme hyperphosphataemia causing acute anuric nephrocalcinosis in lymphosarcoma

Acute hyperphosphataemia occurring during chemotherapy for leukaemia or lymphoma may be accompanied by mild or severe acute renal dysfunction[1] of uncertain mechanism. We describe a patient affected with lymphosarcoma in whom extreme hyperphosphataemia was followed by anuria with histologically proved nephrocalcinosis, suggesting a direct relationship between the metabolic disorder and the onset of acute renal failure (ARF).

Case report

A 43-year-old man was admitted to the renal intensive care unit on 15 June 1976 because of ARF. Six years earlier poorly differentiated malignant lymphoma of the lymphocytic type had been diagnosed by histological examination of one of several enlarged cervical lymph nodes; abdominal lymph nodes were enlarged on lymphangiography. Radiation therapy, cyclophosphamide, and vincristine were administered. Thereafter the patient became asymptomatic and remained so until 1976 on maintenance treatment with cyclophosphamide and chlorambucil.

In June 1976 a relapse occurred with diffuse lymphadenopathy, ascites, palpable abdominal masses, and severe pancytopenia. On 10 June 1976 blood urea was 10 mmol/l (60 mg/100 ml), serum creatinine 109 μmol/l (1·2 mg/100 ml), potassium 5·5 mmol(mEq)/l, urate 785 μmol/l (13·2 mg/100 ml), calcium 2·5 mmol/l (10 mg 100 ml), and phosphate 1·4 mmol/l (4·3 mg/100 ml). Bone marrow was infiltrated by vacuolated deeply basophilic lymphoblasts (80% of the marrow elements). Allopurinol and urate oxidase were given for two days. On 12 June, while the blood urate concentration was 357 μmol/l (6 mg/100 ml), the patient received intravenously cyclophosphamide (1·2 g), doxorubicin (100 mg), vincristine (1·5 mg), and methylprednisolone (120 mg). No shock or fever was observed over the next few days. On 15 June ascites and oedema of the legs were still present, while abdominal masses and peripheral lymphadenopathy had disappeared. Blood urea was 32·5 mmol/l (196 mg/100 ml), serum creatinine 272 μmol/l (3·1 mg/100 ml), and serum potassium 6·7 mmol/l. Urine output was 1·5 l/day. The same day the patient was admitted to the renal unit. On admission blood urate was 327 μmol/l (5·5 mg/100 ml), calcium 1·43 mmol/l