The first kidney donor had suffered a head injury in a road traffic accident. After donor nephrectomy the kidney was immediately perfused with Perfudex for 10-20 minutes and placed with the perfusate in a sterile bag and stored in ice until transplantation six hours later. The blood group and tissue type of the patient was A1, RhD −; HLA-A1, 2 B5, 8, and of the donor A1, RhD + (Dr Barbara Dodd, department of forensic medicine, London Hospital Medical College); HLA-A1, 2 B5A, 8 (B5A is a local "split" of HLA-B5). Eighteen days after the transplant and one day before transplant nephrectomy no RhD antibodies were detected in the patient's serum. Two months later, before a successful second graft, RhD antibodies were first detected and have remained present to the same titre (1/4 by serum albumin and indirect antiglobulin technique) in all subsequent samples tested to date. No cytotoxic antibodies were detected after routine screening of the same samples against a reference panel representing all HLA-A, B, and C antigens.

The first grafted kidney never functioned and two weeks after transplantation a biopsy showed total infarction and the kidney was removed shortly afterwards. Histology of the kidney was therefore non-informative.

Discussion

The development of RhD antibodies is most probably a response to previous sensitisation by the first transplant, as this was the only transfused or transplanted tissue from an Rh + donor. The appearance of Rh antibodies within three months of the graft is also consistent with this explanation[1]; and the absence of cytotoxic anti-HLA antibodies does not preclude the formation of red cell antibodies.[2] Conventional immunotherapy with azathioprine and steriods, used after both transplants, did not apparently suppress the antibody response. Probably passenger red cells, not completely washed out by perfusion, were responsible for the sensitisation rather than kidney cells per se as the Rhesus antigen has not been detected on other tissue. Gunson *et al* have pointed out that less than 0·5 ml of D-positive red cells is sufficient for sensitisation.[3]

As the particular kidney graft was considered a primary non-functional graft it was not possible to establish whether the Rh antibodies contributed to its failure. It would be of interest to know if there are any other similar cases of Rh antibody production and the fate of such transplanted organs.

[1] Gunson, H H, Stratton, F, and Phillips, P K, *British Journal of Haematology*, 1976, **32**, 331.
[2] Ross, J M, and James, D C O, *Journal of Pathology*, 1973, **26**, 367.
[3] Gunson, H H, Stratton, F, and Phillips, P K, *British Journal of Haematology*, 1976, **32**, 317.

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Electronic monitoring of urinary incontinence in the elderly

Training and retraining for sanitary competence is common practice in childhood and in the elderly. In both cases the procedure is based on the Pavlovian conditioned reflex principle. In childhood the endeavour is to imprint the habit de novo; in the elderly all that can be achieved is a revival of the life-long habit that has fallen into disarray.

Toilet training in the elderly is a common nursing technique. It is often started before diagnosis is made, but usually it is based on a charted record of bowel and bladder evacuations.

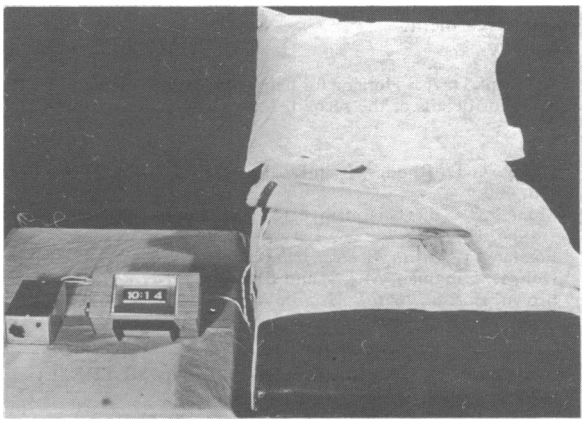
Clearly, to achieve success most easily the principles that have been investigated in detail by Pavlov[1] and others must be followed. This means providing a conditioning stimulus (sitting on a bedpan, commode, etc) when the bladder or rectum is full enough to provide sensory stimulation—before, and not after, evacuation.

Often the last thing to be corrected in the elderly is the episode of true nocturnal incontinence unassociated with delay in providing facility, high beds, etc. The observation of wetness by the nursing staff must be accurate to be of any use and be recorded. Nowadays, nurses are scarce at night and accurate charting is difficult to achieve. Under these circumstances some sort of monitoring technique is required.

Method

The basic requirements are: accuracy of method and no disturbance of the patient by noise or wetting. The time recorded when the patient is asleep will represent the longest period before bladder evacuation, since emotional and sensory stimuli are removed.

A technique that provides these conditions using an Acrylan pad in combination with an under pad and an electrode was described by Willington.[2] To achieve accurate timing independent of nursing availability an electronic device was produced by Ball and McFadden.[3] The important points of this are that the current flows to only about 10 m/s and is of very small amplitude (about 5μV) (see figure). When a series of events has been timed the nursing staff can determine with some accuracy the time they must wake the patient to use the commode. Events can then take place in the correct order: (*a*) full bladder; (*b*) vesical sensation; (*c*) sit on commode; (*d*) evaucation.



General lay out of apparatus. Corner of Acrylan pad has been turned back to show polyethylene and electrode.
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Discussion

The normal conditioning stimulus used for this purpose is the sensation of fullness of bladder or rectum. Simultaneous presentation of the accustomed means and conditions produces a successful result —that is, bladder evacuation (reinforcement). Skill is required by child or adult to void without a sensation of fullness. This is the social requirement demanded from young and old alike; it is the result of prolonged practice by the child and accepted without insight by most adults.

In the treatment of childhood enuresis buzzer alarms are popular and successful. There has been much discussion, but Woodmansey[4] believes it to be a traumatic experience that the child must overcome. The procedure described here would enable training to occur without psychological trauma or skin hazard and shorten the training time because events may take place in the correct order. It can also be used to record the effects of drug treatment on nocturnal incontinence.

[1] Pavlov, I P, *Lectures on Conditioned Reflexes*, vol I. London, Lawrence and Wishart, 1963.
[2] Willington, F L, *Gerontologia Clinica*, 1969, **2**, 330.
[3] Ball, J A C, and McFadden, M H, *British Medical Journal*, 1975, **3**, 465.
[4] Woodmansey, A C, *British Medical Journal*, 1972, **3**, 161.

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