found to be normally situated. The swelling was returned to the pelvic cavity with great difficulty, by continuous traction from inside and pushing from outside. After reduction of the swelling and opening of its anterior surface, the ureteric orifices were visible and urine was seen collecting in the opened lumen. It was thus confirmed that the prolapsed mass was a complete bladder.

The urethra could easily admit two fingers, clearly an unusual condition in a child of two and a half years. A strip of the urethra was excised longitudinally and the urethra was secured over a 14 French gauge rubber catheter. The bladder was closed completely in two layers and fixed to the anterior abdominal wall by two stitches. A drain was put in the cave of Retzius and the abdominal wall was closed. The urethral catheter was left indwelling for 14 days, after which the patient was continent with perfect control except for very slight stress incontinence.

Five groups of urinary bladder inversion are recognized: (1) inversion of a diverticulum; (2) inversion of only the bladder mucosa; (3) partial inversion of all layers of bladder wall; (4) total inversion of the whole viscus; and (5) inversion through a vesicovaginal fistula. Inversion occurs through the urethra in the first four groups. Sutherland and Merki[2] point out the difficulty of replacing the bladder through the urethral sphincter. In their case of acute inversion during labour, as in others, the sphincter was ruptured during replacement. In the present case, the swelling was reduced without apparent damage to the sphincter. Probably the lapse of 18 hours between the onset of the condition and its treatment allowed time for some relaxation of the sphincter and thus the bladder could be returned to the pelvic cavity without rupture of the sphincter.—We are, etc.,

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1 Campbell, M. F., (ed.) *Urology*, Vol. 3, p. 2048. Philadelphia, Saunders, 1963.
2 Sutherland, J. C., and Merki, R. T., *British Medical Journal*, 1964, 2, 991.

Air Embolism during Haemodialysis

SIR,—The paper by Dr. M. K. Ward and others (10 July, p. 74) suggests that all cases of air embolism in patients on haemodialysis occurred with infusion sites placed proximal to the blood pump. In the event of any leakage, air is bound to be sucked into the circuit. Perhaps the authors do not realize that this problem can be overcome with great ease. In this unit we place an R.85 insert (Avon Medicals) for heparin infusion immediately after the blood pump and before the dialyser. In this position there is always a positive pressure within the line and any potential leakage would be from inside to out. Any other infusions are given direct into the bubble trap, another high pressure area in the circuit. This necessitates the use of a high drip stand, but presents no other problems and is somewhat cheaper than the £8,000 for plastic bags quoted.—We are, etc.,

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Sterilization of Young Wives

SIR,—Permit me to join Dr. R. J. Houseman (17 July, p. 184) when he asks that the nature of tubal ligation should be explained to both the husband and wife, but may I further suggest that this golden rule should also apply to the male sterilization operation—and that this explanation should be offered by both the surgeon concerned and the family doctor.

It is my belief that the family doctor has the key role in any sterilization decision. His knowledge of the physical and mental health of a husband and wife (and their children), and his awareness of their socio-economic circumstances and how these might adversely affect the health of one or more members of the family unit renders him the ideal, perhaps the *only*, person capable of advising on which partner should be sterilized. For example, it would be a nonsense for a man to be sterilized without it being known that the wife was already suffering from gynaecological symptoms necessitating relief by a hysterectomy.—I am, etc.,

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Heparin in Acid Solutions

SIR,—The *Drug and Therapeutics Bulletin*[1] declared that heparin is "unstable in acid solutions (for example, dextrose)." Previously, dextrose solution had been considered the vehicle of choice for heparin infusion and although this statement has since been withdrawn[2] it is evident that some uncertainty still exists.

We have conducted experiments designed[3] to clarify the situation. Sterile heparin concentrates, 25,000 units/ml, were aseptically diluted with autoclaved 5% Dextrose Injection B.P. to arrive at a series of concentrations over the range in which heparin is likely to be infused clinically. At various times afterwards, potencies were determined in duplicate. Up to at least 24 hours at room temperature (23±2°C) there was never any indication of a loss. We have observed deterioration only after 60 hours at a minimum concentration of 1,000 units/ml. In normal hospital practice it is unlikely that heparin infusions would be kept for more than 24 hours. Typical results were as shown below.

pH of dextrose solution	pH of dextrose after the addition of heparin pH 7·2	Initial mean potency (u/ml)	mean potency at: (hours after preparation) 12		24	
			u/ml	% of initial	u/ml	% of initial
4·4	7·0	964 (950-977)*	970 (961-980)	101	956 (947-964)	99
4·4	6·8	200 (199-201)	197 (194-200)	99	199 (197-201)	100
4·4	6·6	40 (39-40)	40 (39-40)	100	40 (40-41)	100

*fiducial limits of error.

—We are, etc.,

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1 *Drug and Therapeutics Bulletin*, 1970, 8, 55.
2 *Drug and Therapeutics Bulletin*, 1971, 9, 24.
3 *British Pharmacopoeia*, p. 1345. London, General Medical Council, 1968.

Infectiousness of Glandular Fever

SIR,—In view of the supposed rarity of Paul-Bunnell positive glandular fever occurring within several members of a family, as recently pointed out by Dr. R. M. Whittington (26 June, p. 772), I would like to describe such an occurrence recently seen in general practice.

The initial case was in a girl, aged 10, presenting with earache, cervical adenitis, and a few yellow spots seen on a non-inflamed left tonsil. She recovered completely within two weeks and returned to school. Her brother, aged 7, began to be ill two weeks later, complaining of a sore throat. On examination he had similar yellow spots on his tonsils, and also cervical lymphadenopathy. He remained quite ill for six weeks with generalized malaise and abnormal liver function tests. The third sibling, aged 10, presented eight weeks later with swellings on both sides of the neck, and she had been mildly off colour for the preceding two weeks. Blood tests were not carried out because this particular child is very sensitive, having severe eczema. The fourth sibling, a boy aged 8, became ill a month later with a sore throat, enlarged cervical glands, necrotic looking tonsils, and a positive Hoagland's sign. Only in the last case was the spleen palpable.

The interval between the first and second case was 74 days, between the second and third was 60 days, and between the third and fourth was 33 days.

In the most extensive field survey of glandular fever in general practice carried out by Hobson and Lawson in 1958[1] all the familial outbreak patients were always Paul-Bunnell negative, as was also noted by Shubert and Collee in 1954[2] in epidemics in close communities. Perhaps other practitioners have seen similar familial episodes and it may not really be so rare as indicated in the literature.—I am, etc.,

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1 Hobson, F. G., Lawson, B., and Wigfield, M., *British Medical Journal*, 1958, 1, 845.
2 Shubert, S., Collee, J. G., and Smith, B. J., *British Medical Journal*, 1954, 1, 671.

SIR,—I was interested in Dr. R. M. Whittington's (26 June, p. 772) experience with glandular fever. I have on two occasions observed an interval of six weeks