Melbourne (*Daily Telegraph*, 30 June). A plastic tube 4 inches (10 cm.) long is reported to have been used, but I doubt if it will be successful as it has no cilia and no secretion.

There is yet time to develop in Britain a live transplant which, even if eventually rejected, would serve to secure at least one conception.—I am, etc.,

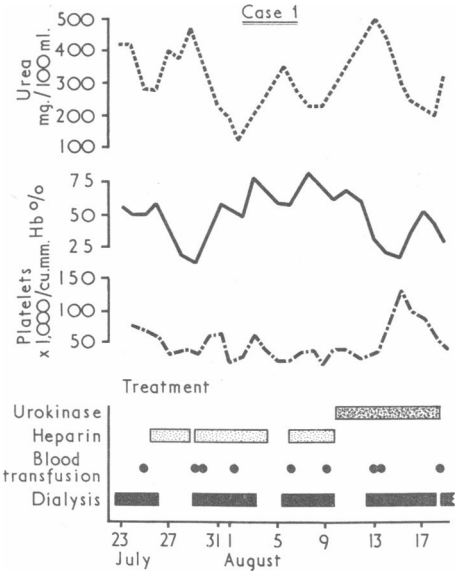
N. L. MAXWELL READER.

London S.W.19.

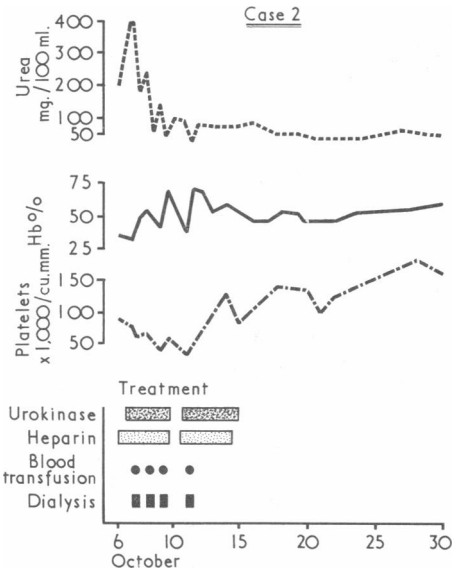
Microangiopathic Haemolytic Anaemia

SIR,—Microangiopathic haemolytic anaemia (M.H.A.) has been described in association with a wide range of clinical situations. Since Good and Thomas in 1953[1] showed that heparin could prevent the experimental Schwartzman reaction, this drug has been widely used in the conditions associated with M.H.A. Although heparin may effectively prevent further coagulation, it has little or no fibrinolytic effect, and in many patients severe renal damage has already occurred at the time of presentation. The use of specific fibrinolytic agents is suggested in this group of patients, and we would like to report two children in whom the plasminogen activator urokinase (Leo Laboratories Ltd.) was used.

A 10-year-old girl was admitted to hospital in July 1968 with a week's history of vomiting and weight loss, jaundice, a petechial rash, and acute renal failure. The blood film showed the typical features of M.H.A. She was treated with transfusions of fresh blood, peritoneal dialysis, and intravenous heparin 5,000 units six hourly. She failed to improve (Fig. 1) and repeated transfusions were required. Intravenous urokinase, 100,000 Ploug units daily, increasing to 200,000 units daily, was added on 11 August. The platelet count rose for the first time, reaching 150,000/cu. mm. on 15 August, but the improvement was transient and she died on 21 August. Renal histology showed widespread necrosis of the small arteries.



In September 1969, a 9-year-old boy had a short febrile illness and was admitted to hospital two weeks later with acute renal failure. Again, the blood film was typical of M.H.A. He was treated with intravenous heparin, 20,000 units per 24 hours, blood transfusion, and haemodialysis. Intravenous urokinase, 100,000 units per 24 hours, was added on the second day of treatment. Although a diuresis began on the second day, the haemoglobin and platelet levels continued to fall (Fig. 2). On 10 October a catheter was introduced into the lower of two left renal arteries, and the infusion of urokinase and heparin continued at this site for a further five days. From 12 October a steady rise in the platelet count and haemoglobin level occurred. A renal biopsy performed six weeks after admission showed acute proliferative glomerulonephritis.



In our first case, a rise in platelet count on the third and fourth days of treatment with urokinase was not maintained. In the second case the platelet response occurred one week after starting treatment with heparin and urokinase. An intravenous pyelogram during the convalescent phase showed uniform excretion throughout both kidneys. Fibrin degradation products were detected both before and after treatment with urokinase.

The effect of heparin on fibrinolysis is not clear. Von Kaulla *et al.*[2] found in vitro that heparin stimulated urokinase-induced fibrinolysis, but Holemans *et al.*[3] showed inhibition of Urokinase activity by concentrations of heparin greater than 30 μg./ml. The effects in vivo, however, are uncertain. The extremely high cost of urokinase makes it difficult to justify the routine use of this drug without more evidence of its efficacy. However, we feel that urokinase may be indicated when other measures fail in the treatment of acute microangiopathic disease.—We are, etc.,

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REFERENCES

1 Good, R. A., and Thomas, L., 1953. *Journal of Experimental Medicine*, **97**, 871.
2 Von Kaulla, K. N., and McDonald, T. S., 1958, *Blood*, **13**, 811.
3 Holemans, R., Dionysios, A., and Horace, J. F., *Thrombosis et Diathesis Haemorrhagica*, 1963, **9**, 446.

Cardiac Arrest and Bone Cement

SIR,—In the first six months of this year we have seen four deaths following the use of the Thompson prosthesis in patients with fractured femurs. One of these collapsed and died on the operating table. All showed evidence of severe fat embolism after histological examination of the lungs, brain and kidneys. We shall report them in more detail later.

Dr. J. N. Powell and colleagues (8 August, p. 326) did not exclude fat embolism as a cause of death in their cases. None of our cases had cutaneous petechiae at necropsy, so that this sign is not always a reliable indication of fat embolism. Dr. J. N. Powell and others did not comment on petechiae in their patients.—We are, etc.,

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Workload of Anaesthetists

SIR,—We write to answer Dr. J. C. Ainley-Walker's question (18 July, p. 161).

We took as our definition of the duration of an operation list the time from the induction of anaesthesia in the first case to the time the last case left the operating theatre. This is not intended to imply that we think that this is or should be the limit of the anaesthetist's involvement with his patient. We used this because it is the incontrovertible minimum requirement of anaesthetic time. The difficulty with the other definitions is that the beginning and end of the occupancy of an anaesthetist with his patients are definable in so many ways that there is almost no limit to the amount of work that one could allege was involved.

The purpose of our paper (4 July, p. 39) was to make clear the type of staffing structure implied by the Godber Report[1] in view of the present sessional obligations of the two departments in which we work. The points at issue are: first, how many sessions are to be covered, and, second, are these sessions fully used? Because of such factors as Dr. Ainley-Walker mentions we suggest that if the average session has a minimum requirement of the order of three hours it is very unlikely that any reorganization of the work could lead to significant reduction in the number of sessions to be covered. This is the sole value of quantifying them in this context.

It must be obvious to practising anaesthetists that lists vary greatly from one week to another. We have expressed our own experience of this in the large standard deviation of list length compared with the mean length. In the day-to-day organization some of this variation can be allowed for, since it is in part predictable once the list is published. For the broader considerations we wished to discuss the relevant factor is the number of sessions that must be staffed. This sessional obligation, we suggest, must be taken as the present number of sessions covered, unless there is evidence of under-use. There are many ways of examining this, but we think the definition we have used suffices for the purpose, and does not depend on factors