since errors in dosage can occur even in the best units. The other practical problem in the case of outpatients is that the patient has to be brought up to hospital six hours before the dialysis.—Ed, *BMJ*.

¹ Maher JF. In: Drukker W, Parsons FM, Maher JF, eds. *Replacement of renal function in dialysis*. The Hague: Nijhoff, 1978:384.

² Papadimitriou M, Memmos D, Metaxas P. *Nephron* 1979;24:53-7.

³ Bowerson DW, Winterbauer RH, Stewart GL. *N Engl J Med* 1973;289:84.

⁴ Anderson RJ, Gambertoglio JG, Schrier RW, Springfield, Illinois: Charles C Thomas 1976:92.

Haemodynamic monitoring in the intensive care unit

SIR,—In acute myocardial infarction a low-cardiac-output condition can be caused by inadequate intravascular volume, and an inappropriately low left ventricular filling pressure is an important diagnostic feature. Your leading article (12 April, p 1035) suggests that the incidence of the condition is small, but as long ago as 1974 Sobel¹ reported that nearly 25% of patients sent to his unit from community hospitals for the treatment of cardiogenic shock and power failure after myocardial infarction had this hypovolaemic disorder. It can kill within an hour or two of its onset if it is not treated by volume expansion, and so many patients must have died before they could reach the community hospitals or arrive at Sobel's unit.

More patients might be recovered if the condition was suspected more often, and if the diagnosis was not thought to depend on direct or indirect measurement of the left atrial pressure. The diagnosis can usually be made from the patient's response to intravenous test infusions of 200-400 ml 5% dextrose, and special equipment is not required to detect the recovery of palpable arterial pulses and recordable blood pressure, the improvement in the peripheral cutaneous circulation, the wakening from unconsciousness, and the resumption of urine flow, which are achieved when the circulation gets the fluid it needs. The ensuing diuresis commonly brings the left ventricular filling pressure below the effective threshold and so the infusion may need to be repeated over and over again during a period of one to seven days. Occasionally an inotropic agent requires to be used, and adrenaline is excellent.²⁻⁶

The infusions cause variable central venous pressure responses. When venous constriction is severe an infusion of 200 ml 5% dextrose may cause the pressure to rise by 20 cm of water, but the level quickly falls as the veins relax, and the infusion of 200 ml 5% dextrose when the hands are pink and warm may cause a pressure rise of less than 2 cm water.³⁻⁶

The majority of patients resuscitated by volume expansion at Charing Cross Hospital had inferior myocardial infarction and presented two remarkable features. Firstly, the heart block which was commonly present disappeared with the first test doses of infusion, but pacemaking without infusion did not relieve the shock. Secondly, the magnitude of the infarction seemed small for such a catastrophic illness when it was judged subsequently from the patient's physical ability and freedom from heart failure.

The diagnosis of hypovolaemic shock in acute myocardial infarction can be excluded when the test doses of infusion fail to produce a rapid and obviously favourable clinical response. The myocardial injury is usually a

severe anterior infarction, and adrenaline infusion may occasionally improve the left ventricular function to a level which enables the patient to recover.

The principles which encouraged the rational investigation and treatment of the low output states in acute myocardial infarction were clearly stated in your leading article—namely: (1) "Changes in [right] atrial pressure may be due to alterations in venous tone. . . ." (2) "In a small [sic] but important group of patients with low-output states complicating myocardial infarction the left atrial pressure is normal or even low." (3) "The use of diuretics to promote the flow of urine may be dangerous. . . ." These principles were waiting to be observed by anyone who turned his attention from the problems of open-heart surgery to acute myocardial infarction, and probably were discovered in many parts of the world at the same time; but I do think they were first published in England.²⁻⁶

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¹ Sobel E. In: Braunwald E, ed. *The myocardium: failure and infarction*. New York: HP Publishing Co, 1974:285-93.

² Nixon PGF, Ikram H, Morton S. *Lancet* 1966;ii: 1077-9.

³ Nixon PGF, Ikram H, Morton S. *Am Heart J* 1967; 73:843-5.

⁴ Nixon PGF, Taylor DJE, Morton SD. *Lancet* 1968; i:1230-2.

⁵ Nixon PGF. In: Julian DG, Oliver MF, eds. *Acute myocardial infarction*. London: E & S Livingstone, 1968:191-200.

⁶ Nixon PGF. In: Baron DN, Compston N, Dawson AM, eds. *Recent advances in medicine*. London: J & A Churchill, 1968:383-415.

Hypokalaemia and diuretics

SIR,—We would like to congratulate Drs D B Morgan and C Davidson for their painstaking analysis of publications on hypokalaemia and diuretics (29 March, p 905). We would, however, like to make a few comments particularly relevant to the older population, 37.4% of whom are on regular diuretic therapy.¹

We surveyed 400 consecutive admissions to our unit; 85 patients were taking diuretics for more than three months and 17 of these were hypokalaemic (see accompanying table). It is interesting to note that 33% of the patients on frusemide alone were hypokalaemic as compared to 4% of those receiving frusemide in combination with potassium supplements or potassium-sparing diuretics. We therefore disagree with the authors' suggestion that the risks of hypokalaemia are small when loop diuretics are administered. Elderly patients are likely to develop hypokalaemia for several reasons: their dietary intake of potassium tends to be reduced,^{2,3} the gastrointestinal absorption may be diminished,⁴ and the reduced muscle bulk may further diminish the total body potassium reserves.^{5,6} If in addition they are challenged by a diuretic that increases the urinary potassium loss, hypokalaemia may develop.

It is interesting to note that in our survey, 27% of the patients on Navidrex K (cyclopentiazide and potassium chloride) became hypokalaemic. In elderly patients therefore the amount of potassium

supplements incorporated in these tablets may either be insufficient to replace the amount lost or not be completely absorbed from the gastrointestinal tract. From our data it may also be argued that even the potassium-sparing diuretics may lead to hypokalaemia: 18% of our patients on 50 mg hydrochlorothiazide and 5 mg amiloride (combined in one tablet) became hypokalaemic. Our findings regarding this combination are, however, in agreement with those of another study⁷ and it has been suggested that the formulation amiloride 5 mg and hydrochlorothiazide 50 mg may in some way interfere with the biological availability of amiloride, as when these two compounds are taken separately no significant drop in the serum potassium could be detected.⁷ On the other hand, the combination of 25 mg hydrochlorothiazide and 50 mg triamterene seems more effective in preventing potassium loss: several hundred elderly patients on this formulation are closely followed by the European Working Party on Hypertension in the Elderly, and no significant drop in their serum potassium has been reported. In another series of 484 patients treated with a combination of hydrochlorothiazide (25 mg) and triamterene (50 mg), only 6% developed hypokalaemia.⁸

We consider unacceptable the statement that "Hypokalaemia would almost disappear as an important complication of diuretic treatment if it was defined as a value less than 3.0 mmol[mEq]/l rather than as a value less than 3.5 mmol/l." Hypokalaemia in the elderly may be associated with postural hypotension and giddiness on standing up. In our series the hypokalaemic patients exhibited an average drop in systolic blood pressure on standing up of 20 mm Hg (from 148 to 128 mm Hg) despite the fact that none had a serum level below 3.0 mmol/l. Apart from the other known manifestations, hypokalaemia may also present in a non-specific manner with lethargy, apathy, and easy fatigability; whereas these manifestations may not be "harmful," the quality of life of older patients may be improved by preventing hypokalaemia from developing.

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¹ Williamson J. *Practitioner* 1978;220:749-55.

² Macleod CC, Judge TG, Caird FI. *Age Ageing*, 1975; 4:49-51.

³ Judge TG, Caird FI, Leask RG, Macleod CC. *Age Ageing* 1974;3:167-73.

⁴ Warren PM, Pepperman MA, Montgomery RD. *Lancet* 1978;ii:849-50.

⁵ Lye MDW, Tucker JS. *Proceedings of the Annual Meeting of the British Geriatric Society* 1978: abstract 21.

⁶ Cox R. *Geriatr Med* 1980;10:(4):22.

⁷ Antcliff AC, Beavers DG, Hamilton M, Harpur JE. *Br J Clin Pract* 1972;26:413-6.

⁸ Hansen KB, Bender AD. *Clin Pharmacol Therap* 1967; 8:392-9.

Beta-blockers in immediate treatment of myocardial infarction

SIR,—On behalf of my colleagues, may I thank Professor G A Rose for his comments? He rightly points out that it is not possible to conclude from our results (29 March, p 885)

Diuretic treatment and hypokalaemia in 85 elderly patients

Diuretic agent	No of patients	Average age (years)	No with hypokalaemia
Frusemide	12	83	4
Frusemide and potassium supplement or potassium-sparing diuretics	23	81	1
Navidrex K (cyclopentiazide and potassium chloride)	26	79	7
Moduretic (amiloride hydrochloride and hydrochlorothiazide)	17	80	3
Others	7	81	2