Haemodynamic effects of frusemide in patients suspected of having acute myocardial infarction

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The haemodynamic effects of intravenous and intramuscular injections of 40 mg frusemide were studied in 17 patients with suspected acute myocardial infarction. Sixteen of the patients had either clinical or radiographic evidence of left ventricular dysfunction. There was an average diuresis of 710 ml during the 60 minutes after the injection. Haemodynamic data were obtained before and at 15, 30, and 60 minutes after the injection.

Sixty minutes after the intravenous injection there were significant falls in the pulmonary artery diastolic pressure (P<0.001), pulmonary arterial wedge mean pressure (P<0.005), cardiac output (P<0.05), and stroke volume (P<0.005). There was also a significant rise in the peripheral vascular resistance at 30 minutes (P<0.005). No significant alterations occurred in the right atrial pressure, arterial systolic pressure, heart rate, or pulmonary arterial and systemic arterial oxygen saturations.

Sixty minutes after the intramuscular injection, a significant fall occurred in the pulmonary artery diastolic (P<0.02) pressure together with a significant rise in the peripheral vascular resistance (P<0.05). There was an insignificant fall in the cardiac output and stroke volume, and for this reason the intramuscular route of administration is preferred.

Ventricular function curves constructed by plotting the cardiac output against the pulmonary arterial wedge pressure suggested that the heart, after an acute myocardial infarction, responds to a diuresis along the ascending limb of the function curve. Discretion should be exercised in the use of potent parenteral diuretics where the severity of left ventricular dysfunction is uncertain. The left ventricular filling pressure, reflected through the pulmonary artery diastolic pressure or the pulmonary arterial wedge pressure, may be used as a guide to therapy.

Until recently, the evaluation of myocardial performance after acute myocardial infarction has been limited to the assessment of the clinical signs, chest radiographs, and other noninvasive procedures (Fowler, 1968; Tattersfield et al., 1969; Karliner and Ross, 1971; Dowling, Sloman, and Urquhart, 1971). With the introduction of haemodynamic studies, it has been shown that left ventricular dysfunction is common after acute myocardial infarction (Fluck et al., 1967; Rackley and Russell, 1972), and that the classical clinical and radiographic signs thought to represent cardiac decompensation are in many cases late in appearing or resolving, and misleading or difficult to interpret, thus hindering any rational timing of therapy (Sjogren, 1970; Lassers et al., 1970; McHugh et al., 1970; Rutherford, McCann, and O'Donovan, 1971; McDonald and McDonald, 1972; Mond, Hunt, and Sloman, 1973b).

The use of diuretic agents after acute myocardial infarction has been said to be safe and effective (Stock, 1970), but little is known of the haemodynamic effects of the potent diuretic agent frusemide (Lasix). It is a rapidly acting diuretic that may lead to excessive plasma water loss, hypotension, and shock (Wolk, Scheidt, and Killip, 1972), and these actions may be particularly hazardous after acute myocardial infarction. Despite this, and the problems of excessive potassium loss, intravenous frusemide has been suggested as a routine after a myocardial infarction irrespective of the presence or absence of clinical evidence of left ventricular dysfunction (Stock, 1970).

In this study a comparison is made of the haemodynamic effects of intravenous and intramuscular injections of 40 mg frusemide, in 17 patients ad-
Haemodynamic effects of furosemide in patients suspected of having acute myocardial infarction

TABLE 1  Clinical features of patients studied and urine output with intravenous and intramuscular furosemide

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age and sex</th>
<th>Infarction</th>
<th>Pulmonary crepitations</th>
<th>Atrial sound</th>
<th>Pulmonary venous congestion on radiograph</th>
<th>Drugs</th>
<th>Urine output (ml)</th>
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<tr>
<td>1</td>
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<td>+</td>
<td>+</td>
<td>Atropine during study</td>
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<tr>
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<td>On procainamide</td>
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<td>-</td>
<td>+</td>
<td>Lignocaine during study</td>
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<td>Mean urine output</td>
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mitted to a coronary care unit with symptoms suggestive of acute myocardial infarction. The aim was to include patients who, after an acute myocardial infarction, might be treated in a coronary care unit with parenteral frusemide on the basis of clinical or radiographic evidence of left ventricular dysfunction and pulmonary venous congestion.

Subjects and methods

Seventeen patients, 15 men and 2 women, were seen within 48 hours of the onset of the major symptom. Informed consent was obtained from each patient and the nature of the study was carefully explained. They were all rested for at least 30 minutes before the study. The average age was 52 years (range 38 to 66 years). Fourteen patients were subsequently shown to have suffered a definite acute myocardial infarction based on W.H.O. criteria Class 1 (World Health Organization, 1971): 6 of these were anterior, 2 inferior, 4 subendocardial, 1 had no changes on the electrocardiogram, and the other patient had a left bundle-branch block configuration (Table 1). The remaining 3 patients were classified as having suffered a possible acute myocardial infarction (Class 2 by W.H.O. criteria). No patients had cardiogenic shock, severe pulmonary oedema, mitral valve dysfunction, pulmonary disease, or serious arrhythmias. At the time of the study, 9 of these patients had crepitations at the lung bases, 11 had an atrial sound, and 11 had radiographic evidence of pulmonary venous congestion.

No patients had been given diuretics within the 8 hours preceding the study and only 1 patient (Case 17) received intranasal oxygen during the study. Standard posteroanterior chest radiographs, tube distance 6 feet, were taken daily with the patient sitting over the side of the bed, and the presence of pulmonary venous congestion was determined using one or more of the following criteria (Logue, Rogers, and Gay, 1963; Tattersfield et al., 1969): 1) Blurring of the perihilar region; 2) distension of the upper lobe veins; 3) intra-alveolar exudates; 4) Kerley B lines.

The patients were studied at the bedside in the coronary care unit procedure room after full explanation of the procedure and a period of rest. The patient lay with the head on one pillow, and using sterile techniques and local anaesthesia a size 7F Swan-Ganz thermomulation catheter (Edwards Laboratories) was inserted into a right antecubital vein and passed 40 to 50 cm to the superior vena cava where the balloon was inflated. An injection of 2500 units of heparin in 10 ml 5 per cent dextrose was then given. If ventricular ectopics were noted, 50 to 100 mg of intravenous lignocaine (1 %) was also given. While the pressure at the catheter tip was monitored the catheter was passed into the pulmonary artery and wedged. Image intensification was not required. Pressure measurements were made using Sanborn differential transducers type 267A, with the zero level taken at the mid-chest position. The pressure signal was processed using a Hewlett Packard 350-110C carrier amplifier and the pressures recorded on a Sample Electronics SE
3006 ultraviolet recorder using light sensitive paper which provided a high contrast recording within a few minutes under normal light conditions. All pressures were observed on an E.M.I. 4-channel slow scan oscilloscope. From the distal lumen of the Swan-Ganz catheter measurements were made of the pulmonary artery diastolic and pulmonary arterial wedge mean pressures, and a pulmonary artery blood sample was taken for oxygen saturation determination. The blood was collected without any air leak into heparinized glass syringes, placed in ice for transport, and analysed within half an hour using a Beckman Cuvette and Physiological Gas Analyzer (Model 160). From the proximal lumen of the Swan-Ganz catheter the right atrial mean pressure was recorded.

Thermodilution cardiac outputs were performed in duplicate by injecting exactly 10 ml cold 5 per cent dextrose (temperature range 0°C to 5°C) into the right atrium. The temperature change in the pulmonary artery blood detected by the thermistor at the distal end of the

| Table 2 | Haemodynamic data obtained before and after intravenous and intramuscular injection of frusemide |

<table>
<thead>
<tr>
<th>Case</th>
<th>Pressure (Swan-Ganz catheter)</th>
<th>Arterial systolic pressure (mmHg)</th>
<th>O₂ saturation %</th>
<th>Cardiac output (l/min)</th>
<th>Heart rate (min)</th>
<th>Stroke volume (ml)</th>
<th>Peripheral vascular resistance (dynes sec cm⁻²)</th>
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<td>PAD (mmHg)</td>
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<td>Heart rate /min</td>
<td>Stroke volume (ml)</td>
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<tr>
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<td>RA (mmHg)</td>
<td>PAD (mmHg)</td>
<td>PAW (mmHg)</td>
<td>Pulmonary arterial</td>
<td>Systolic arterial</td>
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Mean: 8:1 | 18:6 | 15:1 | 130 | 67 | 94 | 4:9 | 90 | 58:8 | 1447
and: 7:9 | 18:0 | 14:4 | 135 | 64 | 93 | 4:9 | 91 | 57:1 | 1589
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6:6 | 15:9 | 11:2 | 140 | 64 | 94 | 4:3 | 93 | 50:0 | 1866
<0:1 | <0:9 | <0:2 | <0:1 | NS | NS | <0:1 | NS | <0:2 | <0:05

Note: The mean and 'P' values are given at the end of each study.
RA, right atrial mean; PAD, pulmonary artery diastolic; PAW, pulmonary arterial wedge mean.

catheter resulted in an off-balance output from a Wheatstone bridge which was amplified and recorded at 4 mm/sec on a Kipp and Zonen BD 3 micrograph potentiometric recorder calibrated before each study. The thermodilution catheter had previously been calibrated over a temperature range 33° to 39°C, using a Marconi Universal Bridge T.F. 2700. The area under the temperature time curve obtained on the Kipp and Zonen recorder was determined by planimetry and the cardiac output calculated from the following equation.

\[
CO = \frac{0:535 \times (T_b - T_i) \times T_a}{5A_p}
\]

where, CO is the cardiac output in litres/minute; T_b is the temperature of the blood (°C); T_i is the temperature of the injectate (°C); T_a is the area of calibration; and A_p is the area under the temperature time curve.

This working formula was derived from the equation recommended by the manufacturers of the catheter (Ganz and Swan, 1972).

Arterial pressure recordings were made in 14 patients using a fine 4F portex cannula passed by the Seldinger technique into the femoral or brachial artery. In 14 patients arterial blood samples were taken and the oxygen saturation determined. In 3 patients arterial pressures were measured by a cuff sphygmomanometer.

After the recording of the baseline measurements, 40 mg frusemide was given by intravenous injection over 5 minutes in 10 patients and by the intramuscular route in 7 patients. The haemodynamic measurements were repeated at 15, 30, and 60 minutes after the injection. Before the study the patient was requested to empty
his bladder and this was repeated at the end of one hour or earlier if necessary. The volume of urine passed over one hour was measured.

Stroke volume (ml) was obtained from the following formula:

\[
\text{Cardiac output ml/min} = \frac{\text{Peripheral vascular resistance (dynes sec cm}^{-5})}{\text{Heart rate/min}}
\]

\[
1332 \times 60 \times \frac{\text{mean arterial pressure (mmHg) - mean right atrial pressure (mmHg)}}{\text{Cardiac output (ml/min)}}
\]

There were no complications associated with the study apart from sinus bradycardia in one patient which required treatment with atropine sulphate.

Results

The clinical features of the patients are listed in Table 1, and the haemodynamic data in Table 2.

Urine output

There was an excellent urinary response to both the intravenous and intramuscular injections of frusemide, the mean volumes being 770 ml over the 60 minutes for the intravenous study (range 400 to 1300 ml) and 910 ml over the 60 minutes for the intramuscular study (range 300 to 1680 ml). The mean volume for all cases was 830 ml.

There was no relation between the initial haemodynamic data, the haemodynamic response to frusemide, and the volume of urine passed.

1) Intravenous frusemide (10 patients)

Right atrial mean pressure There was a gradual fall in the mean right atrial pressure over the 60 minutes after the intravenous injection of frusemide (7.1 mmHg falling to 5.7 mmHg). However, this fall was not statistically significant (P > 0.1).

Pulmonary artery diastolic pressure (Fig. 1) A statistically significant fall in the mean pulmonary artery diastolic pressure occurred after 60 minutes following the injection (17.3 mmHg falling to 14.4 mmHg) (P < 0.001). However, in the 2 patients with the highest initial pulmonary artery diastolic pressure, this pressure rose at 30 minutes, and coincided with the complaint of a painful distended bladder. After micturition the pulmonary artery diastolic pressures fell.

Pulmonary arterial wedge mean pressure (Fig. 2) There was a statistically significant fall in the mean pulmonary arterial wedge pressure at 60 minutes after the injection (14.1 mmHg falling to 10.1 mmHg) (P < 0.005). Though the fall was significant at 15 minutes, the mean pulmonary arterial wedge pressure rose again at 30 minutes. This was caused by a sharp rise in the pulmonary arterial wedge pressures of the same 2 patients who showed a rise in the pulmonary artery diastolic pressures. These patients had the highest pulmonary arterial wedge pressures and after micturition the pressures fell.

Arterial systolic pressure, blood gases, and heart rate There was little alteration in any of these measurements over the 60 minutes after the injection.

Cardiac output (Fig. 3) There was a fall in the mean cardiac output after the injection, this fall being most significant at 30 minutes (6.4 l/min, falling to 5.2 l/min) (P < 0.005). At 60 minutes the fall was still significant but the mean cardiac output had risen again to 5.7 l/min (P < 0.05).

Stroke volume (Fig. 4) There was a significant fall in the mean stroke volume. This was maximum at 30 minutes (83.5 ml falling to 69.3 ml) (P < 0.001), but the fall was also significant at 15 minutes (P < 0.01) and 60 minutes (P < 0.005).

Peripheral vascular resistance There was a statistically significant rise in the peripheral vascular resistance 30 minutes after the injection (1234 dynes sec cm\(^{-5}\) rising to 1471 dynes sec cm\(^{-5}\)) (P < 0.005).

Ventricular function (Fig. 5) The cardiac output, before injection and 60 minutes later, was graphed against the pulmonary arterial wedge pressures at those times. In 9 cases there was a fall in both the cardiac output and the pulmonary arterial wedge pressure. In the other case a rise in the cardiac output occurred from the preinjection value of 5.3 l/min to 5.6 l/min at 60 minutes.

2) Intramuscular frusemide (7 patients)

Right atrial mean pressure There was a gradual fall in the mean right atrial pressure over the 60 minutes following the intramuscular injection of frusemide (8.1 mmHg falling to 6.6 mmHg). However, this fall was not statistically significant (P > 0.1).

Pulmonary artery diastolic pressure (Fig. 6) A statistically significant fall in the mean pulmonary artery diastolic pressure occurred after 60 minutes following the injection (18.6 mmHg falling to 15.9 mmHg) (P < 0.02).
Haemodynamic effects of frusemide in patients suspected of having acute myocardial infarction

**FIG. 1** Effect of an intravenous injection of frusemide on the pulmonary artery diastolic pressure. There was a significant fall at 60 minutes ($P < 0.001$).

**FIG. 2** Effect of an intravenous injection of frusemide on the pulmonary arterial wedge mean pressure. There was a significant fall at 15 minutes ($P < 0.02$) and again at 60 minutes ($P < 0.005$).

**FIG. 3** Effect of an intravenous injection of frusemide on the cardiac output. There was a significant fall at 30 minutes ($P > 0.005$) and again at 60 minutes ($P < 0.05$).

**FIG. 4** Effect of an intravenous injection of frusemide on the stroke volume. There was a significant fall at 15 minutes ($P < 0.01$), 30 minutes ($P < 0.001$), and 60 minutes ($P < 0.005$).
Pulmonary arterial wedge mean pressure  A fall in the mean pulmonary arterial wedge pressure occurred in the 60 minutes after the injection (15±1 mmHg falling to 11±2 mmHg). However this fall was not statistically significant (P < 0.2).

Arterial systolic pressure, blood gases, and heart rate  There was little alteration in any of these measurements over the 60 minutes after the injection.

Cardiac output (Fig. 7)  There was a fall in the cardiac output after the injection (4·9 l/min falling to 4·3 l/min at 60 minutes), but this fall was not statistically significant (P < 0·1).

Stroke volume  There was a fall in the stroke volume after the injection (58·8 ml falling to 50·0 ml at 60 minutes), but this fall was not statistically significant (P < 0·2).

Peripheral vascular resistance  There was a statistically significant rise in the peripheral vascular resistance at 30 minutes (1447 dynes sec cm⁻⁸ rising to 1761 dynes sec cm⁻⁸ (P < 0·001), and 60 minutes (1866 dynes sec cm⁻⁸ (P < 0·05) after the injection.

Ventricular function (Fig. 8)  The cardiac output, both before the injection and 60 minutes later, was graphed against the pulmonary arterial wedge pressure at those times. In 5 cases there was a fall in both the cardiac output and pulmonary arterial wedge pressure. In one case the cardiac output rose (this patient had the highest pulmonary arterial wedge pressure present) and in another case the pulmonary arterial wedge pressure rose 1 mmHg 60 minutes after the injection.

Discussion

Parenteral frusemide will result in a prompt diuresis and significant haemodynamic changes apparent between 30 and 60 minutes in patients with suspected acute myocardial infarction. With the intravenous injection, there were significant falls in the cardiac output, stroke volume, pulmonary artery diastolic, and pulmonary arterial wedge pressures, together with a significant rise in the peripheral vascular resistance. Similar haemodynamic trends occurred with the intramuscular injection of frusemide; however, the only significant alterations were the fall in the pulmonary artery diastolic pressure at 60 minutes and the rise in peripheral vascular resistance at 30 and 60 minutes.

After an intravenous injection of frusemide in patients with acute myocardial infarction, other workers have reported a fall in the pulmonary artery pressure (Sjogren, 1970; Tattersfield and McNicol, 1970; Davidson et al., 1971), left ventricular end-diastolic pressure (Amsterdam et al., 1972), and pulmonary arterial wedge pressure (Kiely et al., 1972). This fall in the left ventricular filling pressure measured either directly or indirectly via the pulmonary artery diastolic or pulmonary arterial wedge pressures (Sapru, Taylor, and Donald, 1968; Forsberg, 1971; Balcon, Bennett, and Sowton, 1972), together with a fall in the stroke volume, suggests intravascular fluid depletion, resultant upon the diuresis.

Although only statistically significant after the intravenous injection, the cardiac output was shown to fall after parenteral frusemide. The blood pressure however was maintained, because of a concomitant rise in peripheral vascular resistance, presumably caused by the activity of baroreceptor reflexes (Loeb et al., 1969). Sjogren (1970) showed a similar decrease in the cardiac output and stroke volume together with a rise in the peripheral vascular resistance in some of his patients after an intravenous injection of frusemide, but the mean values overall did not show significant change. Other authors do, however, show significant falls in the cardiac output and stroke volume (Tattersfield and McNicol, 1970; Davidson et al., 1971).

After the intravenous administration of frusemide, the failure of the right atrial pressure to show a significant fall, despite the fall in pulmonary arterial wedge pressure, further emphasizes the failure of this pressure to reflect changes in the left ventricular filling pressure. The right atrial pressure, being dependent on the peripheral venous tone and compliance of the right ventricle, correlates poorly as a static pressure with the pulmonary artery diastolic, pulmonary arterial wedge, and left ventricular end-diastolic pressures (Rapaport and Scheinman, 1969; Ramo et al., 1970; Gunnar and Loeb, 1972; Russell et al., 1972; Mond, Hunt, and Sloman, 1973a). Despite this finding, one group has shown a significant reduction in the right atrial pressure after an intravenous injection of 40 mg frusemide in patients with pulmonary congestion after acute myocardial infarction (Coltart and Hamer, 1971).

The pulmonary artery and systemic arterial blood oxygen saturations did not alter significantly in the 60 minutes after the intravenous injection of frusemide. It has been emphasized by other workers that the hypoxaemia accompanying left ventricular failure fails to improve rapidly after frusemide, and the much slower clearance of interstitial oedema, as contrasted with the rapid reduction in pulmonary blood...
Haemodynamic effects of frusemide in patients suspected of having acute myocardial infarction

**FIG. 5** The cardiac output is plotted against the pulmonary arterial wedge mean pressure, both before and 60 minutes after an intravenous injection of frusemide. In all cases there was a fall in the pulmonary arterial wedge pressure, and in 9 cases there was a concomitant fall in the cardiac output suggesting that the heart functioned along the ascending limb of the ventricular function curve. In one patient with a high initial pulmonary arterial wedge pressure the cardiac output rose after frusemide.

**FIG. 7** Effect of an intramuscular injection of frusemide on the cardiac output. There was a gradual fall in the output over 60 minutes but this was not statistically significant \((P < 0.1)\).

**FIG. 6** Effect of an intramuscular injection of frusemide on the pulmonary artery diastolic pressure. There was a significant fall at 60 minutes \((P < 0.02)\).

**FIG. 8** The cardiac output is plotted against the pulmonary arterial wedge mean pressure both before and 60 minutes after an intramuscular injection of frusemide. In 5 cases there was a fall in both the pulmonary arterial wedge pressure and cardiac output. In 1 case the cardiac output rose and in another case the pulmonary wedge pressure rose 1 mmHg, 60 minutes after the frusemide.
volume, probably explains the slow improvement in gas exchange (Iff and Flenley, 1971).

By graphing the cardiac output against the filling pressure of the left ventricle (the pulmonary arterial wedge pressure), ventricular function curves were constructed for both the intravenous and the intramuscular frusemide studies. They showed that cardiac function after a diuresis followed the ascending limb of the function curve. These results are in agreement with those of Stampfer et al. (1968) and Lal et al. (1969).

Kiely et al. (1972) have shown that in patients after acute myocardial infarction, a good diuretic response to frusemide indicated a good prognosis, whereas a failure indicated a group with poor renal perfusion and a high mortality. None of the patients in our series had severe pulmonary oedema and all had an excellent urinary response to frusemide whether it was given by the intravenous or intramuscular route.

There was very little difference between the response to the intravenous and intramuscular injections of frusemide in this study. Though statistically the intravenous route had a more significant haemodynamic response, this was not striking when the mean values were compared. Both groups had similar haemodynamic data at the beginning of the study. The fall in cardiac output after parenteral frusemide was more obvious in the intravenous study and had reached its lowest level at 30 minutes. After the intramuscular injection of frusemide the drop was more gradual, suggesting that this route of administration might be safer in patients with left ventricular failure after acute myocardial infarction.

Serious complications of potent diuretics, namely excessive plasma loss, hypotension, and shock, as suggested by Wolk et al. (1972), were not seen in this study, nor in other reported series where frusemide in intravenous doses of 40 to 80 mg was given to patients after acute myocardial infarction (Sjogren, 1970; Stock, 1970; Tattersfield and McNicol, 1970; Coltart and Hamer, 1971; Davidson et al., 1971; Amsterdam et al., 1972; Kiely et al., 1972). However, in a previously reported case, overenthusiastic diuretic therapy combined with inadvertent fluid restriction resulted in a very low left ventricular filling pressure (Mond et al., 1973a).

Because of the rapid and potent action of parenteral frusemide (Sjogren, 1970; Davidson et al., 1971; Tattersfield, 1972), it has been recommended in the treatment of left ventricular failure after acute myocardial infarction (Stock, 1970). The value of potent diuretic treatment in patients with mild left ventricular failure after an acute myocardial infarction is, however, debatable. In this study the fall in the left ventricular filling pressure reduced the cardiac output, but the blood pressure remained unaltered because of an increased peripheral vascular resistance. This situation suggests a reduction in the cardiac work load (Stampfer et al., 1968; Lal et al., 1969; Sjogren, 1970), and by this means the reduction of the left ventricular filling pressure may be beneficial. However, we know of no reports to suggest that early and vigorous treatment of left ventricular dysfunction with diuretics aids the survival of such patients.

After acute myocardial infarction there is a reduction in the compliance of the left ventricle, even in the early phase (Diamond and Forrester, 1972), and consequently a raised left ventricular filling pressure may be beneficial in order to distend the stiffened ventricle. Thus, a raised filling pressure may be a compensating mechanism advantageous to the failing left ventricle. We, therefore, emphasize the need for caution in the use of vigorous intravenous diuretic therapy in patients with lesser degrees of left ventricular failure (McDonald and McDonald, 1972).

Russell et al. (1972) have recommended that the left ventricular end-diastolic pressure after acute myocardial infarction should be regulated between 18 and 22 mmHg to obtain the maximum cardiac output. They further suggest that in patients whose initial filling pressures are above 25 mmHg reduction of this pressure is certainly indicated by the use of diuretics. If one accepts these criteria only 4 of our patients should have been given frusemide. This problem is further complicated by the unreliability of the clinical signs of left ventricular dysfunction after acute myocardial infarction (Sjogren, 1970; Lassers et al., 1970; McHugh et al., 1970; Rutherford et al., 1971; Mond et al., 1973b).

In summary, parenteral frusemide should not be given routinely to patients after acute myocardial infarction, but should be reserved for those cases with definite clinical, radiographic, or haemodynamic evidence of left ventricular failure. Because of the gradual fall in cardiac output, the intramuscular route of administration is preferred. In subjects with left ventricular failure, immediate administration is indicated but in any case where there is clinical or radiographic doubt, the left ventricular filling pressure, as reflected through the pulmonary artery diastolic and pulmonary arterial wedge pressure, should be measured before therapy is decided.

References
Haemodynamic effects of frusemide in patients suspected of having acute myocardial infarction


Requests for reprints to Dr. G. Sloman, Cardiac Department, Royal Melbourne Hospital, Victoria 3050, Australia.
Haemodynamic effects of frusemide in patients suspected of having acute myocardial infarction.
H Mond, D Hunt and G Sloman

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