contributing factor to the poorer results, although we do not deny that other factors may have played a part.

With these reservations the three principal conclusions may be restated in relation to extradural haematoma. (*a*) Delay in surgical evacuation of extradural haematomas leads to increased morbidity and mortality. Specifically it appears that delays exceeding two hours are unacceptable. (*b*) Direct admission of patients to neurosurgical beds from the accident and emergency unit decreased the delay time. The major delay time in the earlier period occurred in hospital and not before admission. (*c*) Direct admission of patients to neurosurgical beds in the later period was associated with a decline in morbidity and major mortality. That this decline was due to shorter delays is suggested by (*b*) but may have been contributed to by other factors.

It therefore seems reasonable to conclude that direct admission of head-injured patients immediately to the primary care of the neurosurgical team is a good policy. It is, of course, possible only in cities where a neurosurgical department is immediately available and staffed to deal with head injuries in this way. Nevertheless, evacuating an extradural haematoma is not difficult, nor is diagnosing its presence and site if a history of the trend of events from injury, especially conscious level, is always obtained. A telephone consultation service round the clock must be available to district hospitals from the nearest neurosurgical department and backed up with a flying-squad service[11] to deal with the more complicated problems of surgical technique. Training district hospital and accident and emergency surgeons for three to six months in a head and spinal injuries service as part of a neurosurgical department at the senior house officer/registrar level has been part of the organisation in Edinburgh for 10 years. Its value is in no doubt.

The most important factors in reducing mortality and morbidity from extradural haematoma are thus early diagnosis and immediate action to reduce intracranial pressure by removing the clot. More often than not the diagnosis may be made clinically, and delays incurred in obtaining radiological confirmation may lead to increased morbidity and mortality if indiscriminately applied to all cases of head injury. Patients with a clear history of deterioration in level of consciousness should therefore be treated surgically with greatest urgency if the aim is to preserve intellect as well as life. From experience in the past few years the CAT scanner is of great value in patients who have remained unconscious from the time of injury or have failed to improve during observation. Such patients should not be confused with the acutely deteriorating group, who require immediate surgical decompression. Clinical vigilance remains the most important factor in all units dealing with head injuries.

We thank the consultants of the department of surgical neurology at the Royal Infirmary of Edinburgh for allowing us to report on their patients. Our thanks also go to Mrs K Mackie for help with the medical records and Miss Picton Phillipps for typing the manuscript.

References

1 McKissock, W, *et al*, *Lancet*, 1960, **2**, 167.
2 Rose, J, Valtonon, S, and Jennett, B, *British Medical Journal*, 1977, **2**, 615.
3 Bartlett, J R, and Neil-Dwyer, G, *British Medical Journal*, 1978, **2**, 813.
4 Documenta Geigy, *Scientific Tables*, 6th ed, p 191. Manchester, Geigy, 1962.
5 Armitage, P, *Statistical Methods in Medical Research*. Oxford, Blackwell Scientific, 1974.
6 Kvarnes, T L, and Trumpy, J H, *Acta Neurochirurgica*, 1978, **41**, 223.
7 Jamieson, K G, and Yelland, J D N, *Journal of Neurosurgery*, 1968, **29**, 13.
8 Hooper, R, *British Journal of Surgery*, 1960, **47**, 71.
9 Lewin, W, *Proceedings of the Royal Society of Medicine*, 1954, **47**, 865.
10 Gallagher, J P, and Browder, E J, *Journal of Neurosurgery*, 1968, **29**, 1.
11 Gillingham, F J, in *Head Injuries*, p 72. Edinburgh, Churchill Livingstone, 1971.

(*Accepted 13 March 1979*)

Hyponatraemia and severity and outcome of myocardial infarction

C T G FLEAR, P HILTON

British Medical Journal, 1979, **1**, 1242-1246

Summary and conclusions

A total of 235 consecutive patients admitted to a coronary care unit were investigated for serum electrolyte and urea concentrations; activities of aspartate aminotransferase, lactate dehydrogenase, and lactate dehydrogenase isoenzymes; electrocardiographic changes; clinical state; and outcome. Hyponatraemia, hypochloraemia, and uraemia were common in patients with confirmed myocardial infarctions, the degree of infarction correlating well with all the above indices of severity. The day-to-day variability of plasma sodium, chloride, and potassium concentrations was often increased above normal. Disturbances were greater in patients given diuretics.

It is concluded that plasma sodium concentrations fall after infarction and that the extent and duration of the fall are indices of the severity of the infarction.

University Department of Clinical Biochemistry and Department of Medicine, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP

C T G FLEAR, MD, FRCPATH, senior lecturer and consultant in clinical biochemistry

P HILTON, MB, BS, medical student (present appointment: registrar in obstetrics and gynaecology, Dryburn Hospital, Durham)

Introduction

Several systemic metabolic changes have been reported to follow acute myocardial infarction in man. These include increased plasma concentrations of catecholamines, free fatty acids, cortisol, glucose, glycerol, and cyclic adenosine monophosphate; decreased triglyceride concentrations; and an initial fall in plasma insulin concentration followed by an early return to normal values.[1 2] Most of these disturbances are also seen after injury and surgery, when hyponatraemia is common.[3] We wondered, therefore, whether plasma sodium concentrations fell after acute myocardial infarction and, if so, whether the extent of their fall was an index of severity, as has been noted in congestive heart failure.[4-6]

We initially carried out a preliminary retrospective study of

data available on 72 consecutive patients (53 men, 19 women) admitted to the coronary care unit. Nine of the men (17%) and two of the women (11%) died. Plasma sodium concentrations were not measured or were measured only once in six of the patients; in 65 of the remaining 66 the concentrations fell. The falls were less than 4 mmol(mEq)/l in 32 patients (48%), 4-8 mmol/l in 17 (26%), and over 8 mmol/l in 16 (24%). Subsequently we carried out a prospective study of 235 consecutive admissions to a coronary care unit. We present here our results.

Patients and methods

All patients were admitted with a provisional diagnosis of acute myocardial infarction. The average age of the 185 men was 56 years and of the 50 women, 61 years. On discharge or death patients were subdivided by the attendant medical staff into four groups. Group 1 comprised those with myocardial infarction who died in hospital; group 2 those with myocardial infarction who were discharged home; group 3 those with evidence of myocardial ischaemia but not of infarction; and group 4 those with no evidence of infarction or ischaemia (table I). A diagnosis of infarction was based on clinical considerations (duration and severity of chest pain, circulatory collapse, etc); rises in serum activity of aspartate aminotransferase (AST), lactate dehydrogenase (LD), and the LD isoenzyme LD1; and characteristic ECG changes. Group 4 comprised patients with minor arrhythmias, simple syncopes, and chest infections, one with pericarditis, and two with carcinomas (of the lung and head of pancreas).

TABLE I—*Details of groups of patients studied*

Group	Men	Women	Total
1 Fatal infarction	22	7	29
2 Non-fatal infarction	131	30	161
3 Ischaemia	23	7	30
4 No evidence of ischaemia or infarction	9	6	15
Total	185	50	235

Twenty-nine of the 190 patients with myocardial infarction died (15·3%). Seven died within two to three hours after admission in severe cardiogenic shock with ventricular fibrillation. One further death occurred on each of days 2, 3, and 4. Nineteen patients died between days 6 and 28 inclusive. Frusemide was given to 66 of the patients with infarcts (35%)—that is, in 54 of the non-fatal cases (34%) and 12 of the fatal cases (41%). Patients were given 40, 80, or 120 mg daily.

Clinical assessment—A proforma was completed daily by medical staff of the coronary care unit. The following factors were graded 0 (not present), 1 (mild), 2 (moderate), or 3 (severe): sweating, raised jugular venous pressure, basal crepitations, pallor, cyanosis, nausea, low pulse volume, oliguria, faintness, and confusion. The daily scores were added together to give a single clinical score for each patient.

Biochemical measurements—Samples of arm-vein blood were collected on admission from all patients except the seven who died within two to three hours, and at about 9 00 am on each of the next seven days. During the remainder of their hospital stay samples were taken at at least weekly intervals. Plasma was analysed for sodium, potassium, chloride, urea, and total carbon dioxide using a Technicon AutoAnalyzer. Serum AST and LD activities were measured by rate reaction; LD isoenzyme patterns were determined by starch gel electrophoresis.[7] Table II shows reference values for plasma sodium, potassium, chloride, and urea concentrations and serum AST and LD activities.

ST-segment elevation and dysrhythmia—ECGs were continuously monitored during the acute phase of the patients' illness in the coronary care unit; continuous recording was not made. Persistent arrhythmias were recorded in the case notes. After discharge daily records were examined, and ST-segment elevation was measured, to the nearest 0·5 mm, on the day of maximum changes in the lead showing maximum deviation.

Results

Plasma sodium concentrations—Mean daily sodium concentrations showed a progressive fall until day 4 and a rise thereafter (fig 1). In addition, the concentrations commonly varied more from day to day than is usual in healthy adults (table II). The incidence of excessive variability ranged from 69% for patients in group 4 to 94% for those in group 1. We defined hyponatraemia as a plasma concentration of less than 135 mmol/l at any stage during admission. This occurred in 85 (45%) of all the patients with infarcts (19 (66%)) of the fatal cases and 66 (41%) of the non-fatal cases); in 2 (13·3%) patients in group 4; and in 1 (3·3%) of the patients with ischaemia. Hyponatraemia was present on admission in 20 (10·5%) of the patients with infarcts and in 3 (7%) others. We calculated the mean and standard deviation (SD) of the plasma sodium concentrations in each

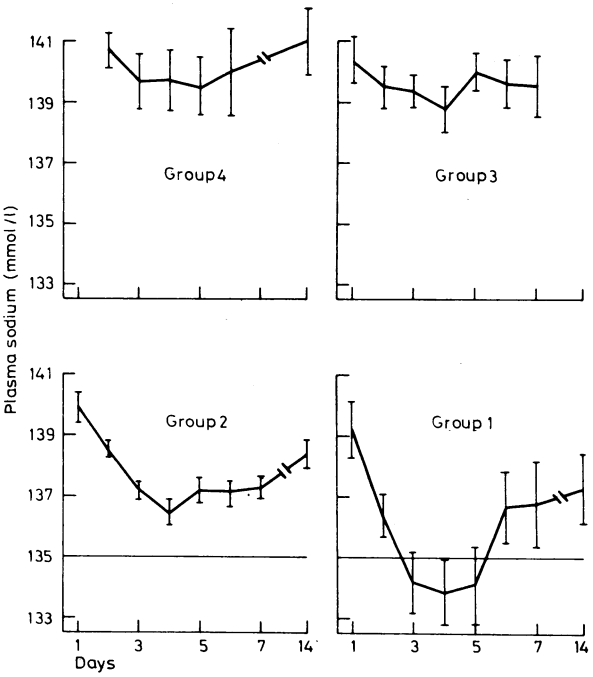

FIG 1—Mean (±1 SE) daily plasma sodium concentrations in each group. Horizontal lines indicate lower 95% limit of reference values.
Conversion: SI to traditional units—Plasma sodium: 1 mmol/l = 1 mEq/l.

TABLE II—*Reference values for plasma concentrations and serum enzyme activities*

	Plasma values (mmol/l)				Serum enzyme activity (U/l)	
	Sodium*	Potassium*	Chloride*	Urea*	LD†	AST†
95% confidence limits	135-146	3·4-4·8	96-105	3·3-6·6	50-220	4-20
Daily variation in SD‡	1·85	0·20				

* From values obtained in 127 healthy adults (C M Singh and C T G Flear, unpublished observations).
†From values obtained in 2000 hospital patients (controls) over six months (A W Skillen, unpublished observations).
‡Average of SD of values (9 00-10 00 am) determined on each of days 8-11 over 14 days in nine healthy adults (C M Singh and C T G Flear, unpublished observations).
LD = Lactate dehydrogenase.
AST = Aspartate aminotransferase.
Conversion: SI to traditional units—Plasma sodium, potassium, and chloride: 1 mmol/l = 1 mEq/l. Plasma urea: 1 mmol/l ≈ 6 mg/100 ml.

patient, and the grand means of both sets of data for each group. The grand means for daily concentrations could be ranked group 1 < 2 < 3 < 4 and for the SDs group 4 < 3 < 2 < 1. The differences between the grand means of the daily concentrations in groups 1 and 2, 2 and 3, and 3 and 4 were all significant ($P < 0.001$). Similarly, the differences between the grand means of the SDs for groups 1 and 2, 2 and 3, and 3 and 4 were all significant ($P < 0.01$).

Plasma chloride concentrations—Mean daily chloride concentrations showed a similar pattern to that of plasma sodium concentrations. Calculated values for the grand means of the concentrations could be ranked group 1 < 2 < 3 < 4. The grand means of the SDs ranked similarly to those of sodium. For both sets of grand means the differences between the groups were all significant ($P < 0.001$).

Plasma potassium concentrations—Mean daily potassium concentrations fell during the first three days and rose thereafter. This trend was less apparent when the data for the groups were considered separately (fig 2). The grand means of the daily concentrations and

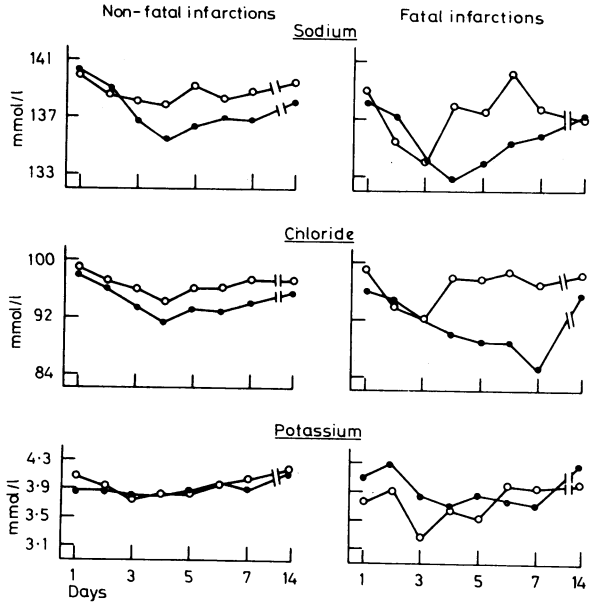

FIG 2—Mean daily plasma concentrations of sodium, chloride, and potassium in patients with infarctions.
—○—=Patients not given diuretics. —●—=Patients given diuretics.
Conversion: SI to traditional units—Plasma sodium, chloride, and potassium: 1 mmol/l = 1 mEq/l.

the SDs did not differ significantly between the groups. In all groups the day-to-day variability was commonly higher than normal. The incidence of excessive variability ranged from 79% for group 4, to 96% for patients with infarctions. Wilhelm reported raised potassium concentrations after myocardial infarction,[8] and Dyckner *et al* commented on the importance of hypokalaemia.[9] Hypokalaemia was present on admission in 29 (15%) of the patients admitted with acute infarction. We found hypokalaemia (plasma concentration less than 3·4 mmol (mEq)/l) at some time after admission in 42 (17·9%) of our patients. We also found hyperkalaemia (plasma concentration of over 4·8 mmol/l) in 25 (10·5%).

Plasma urea concentrations—Mean daily concentrations rose significantly only in groups 1 and 2, the rise being greater in group 1. The highest mean daily urea concentration occurred on day 5 (figs 3 and 4).

Serum AST and LD activities—Serum LD activity increased more in group 1 than group 2; AST activity showed little or no difference between these two groups (figs 3 and 4).

Plasma sodium concentrations as index of severity—Significant negative relations were noted between the lowest plasma sodium concentrations and the maximum serum LD activity, maximum ST-segment elevation, and total clinical score. Mortality and minimal plasma sodium concentrations were related: 10 out of the 147 patients (7%) with normal minimal plasma concentrations died, as did one of the six (17%) with minimal concentrations between 135 and 131 mmol/l and 18 of the 82 (22%) with concentrations of 130 mmol/l or less. Minimum plasma sodium concentrations averaged 132 mmol/l

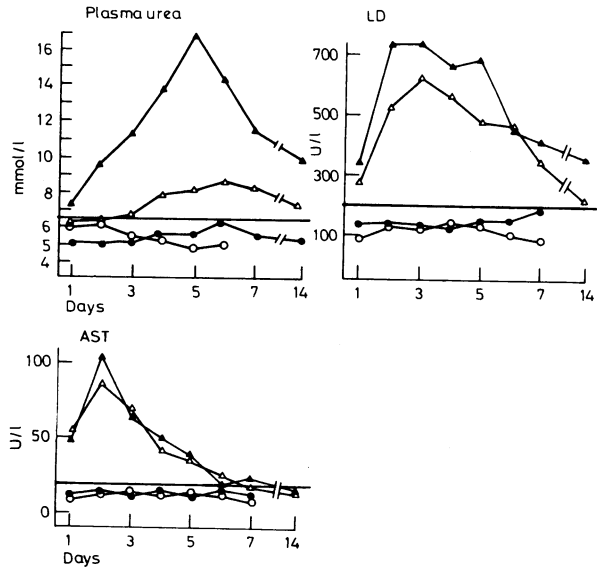

FIG 3—Mean daily plasma urea concentrations and activities of serum lactate dehydrogenase (LD) and aspartate aminotransferase (AST). Horizontal lines indicate upper 95% limits of reference values.
—▲—=Group 1. —△—=Group 2. —●—=Group 3. —○—=Group 4.
Conversion: SI to traditional units—Plasma urea: 1 mmol/l ≈ 6 mg/100 ml.

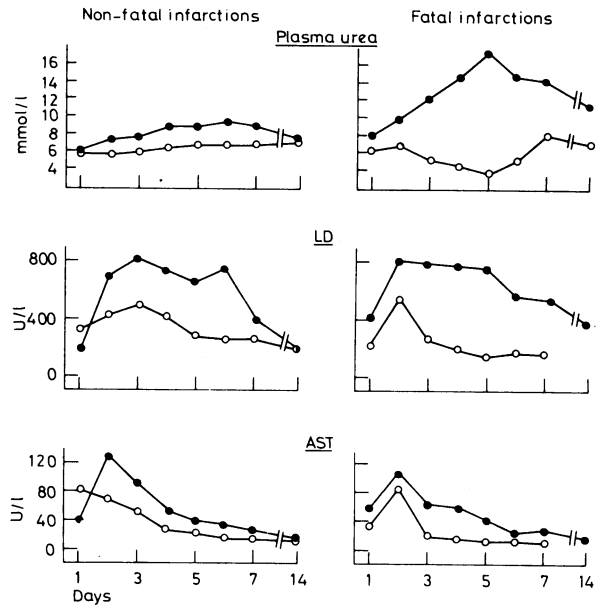

FIG 4—Mean daily plasma urea concentrations and activities of serum lactate dehydrogenase (LD) and aspartate aminotransferase (AST) in patients with infarctions.
—○—=Patients not given diuretics. —●—=Patients given diuretics.
Conversion: SI to traditional units—Plasma urea: 1 mmol/l ≈ 6 mg/100 ml.

in patients with episodes of ventricular fibrillation; 133 mmol/l in those with conduction defects (bundle block; first-, second-, or third-degree heart block); and 136 mmol/l in patients with no dysrhythmias or with only atrial or ventricular ectopic beats.

Diuretics: plasma concentrations and mortality—Patients in groups 1 and 2 given diuretics showed greater and more prolonged falls in plasma sodium and chloride concentrations and higher urea concentrations and LD and AST activities than did those not given diuretics (figs 2 and 4). These differences were all more pronounced in those who died. Changes in plasma potassium concentrations were not obviously different between the two categories. Frusemide was given to 66 (35%) of patients with infarcts: three out of 44 (7%) given 40 mg/day died, as did six out of 17 (35%) given 80 mg/day and three out of five given 120 mg/day.

Plasma concentrations in individual patients—Plasma concentrations

showed the same trends in individual patients as in the grouped data. Fig 5 and 6 show data from two patients in group 2. Notes from the nursing cardex are included. In both patients the plasma sodium concentration reflected the clinical state.

Discussion

Hyponatraemia is common after infarction, and clinical improvement is accompanied by a rise in the plasma sodium concentration. These findings are similar to the systemic biochemical changes that occur after injury. Hyponatraemia occurs more often (86%) and is more pronounced in patients who die in hospital than in others (41%). It is an epiphenomenon and should not be treated directly with, for example, saline. A low plasma sodium concentration is more appropriate to circumstances attending infarction than a so-called normal concentration would be. Hypoxia and ischaemia increase the permeability of sarcolemma to sodium—for example, Flear *et al* found a considerable increase in sodium permeability during elective ischaemic anoxic arrest during heart surgery.[10]

A reduction in the plasma sodium concentration reduces the influx of sodium into cells and so limits the resultant increase in energy expenditure on sodium pump activity.[11] It also lessens the likelihood of imbalance in transmembrane sodium exchanges and net sodium accumulation. Accumulation of sodium by myocardial cells reduces diastolic membrane potentials, impairs contraction and relaxation, and impairs or abolishes electrical coupling between cells,[12] and in-vitro evidence attests that the heart withstands anoxia better when the sodium concentration in the medium is reduced[13 14] and that an increase in fibre sodium depresses contraction when active sodium efflux is depressed.[15]

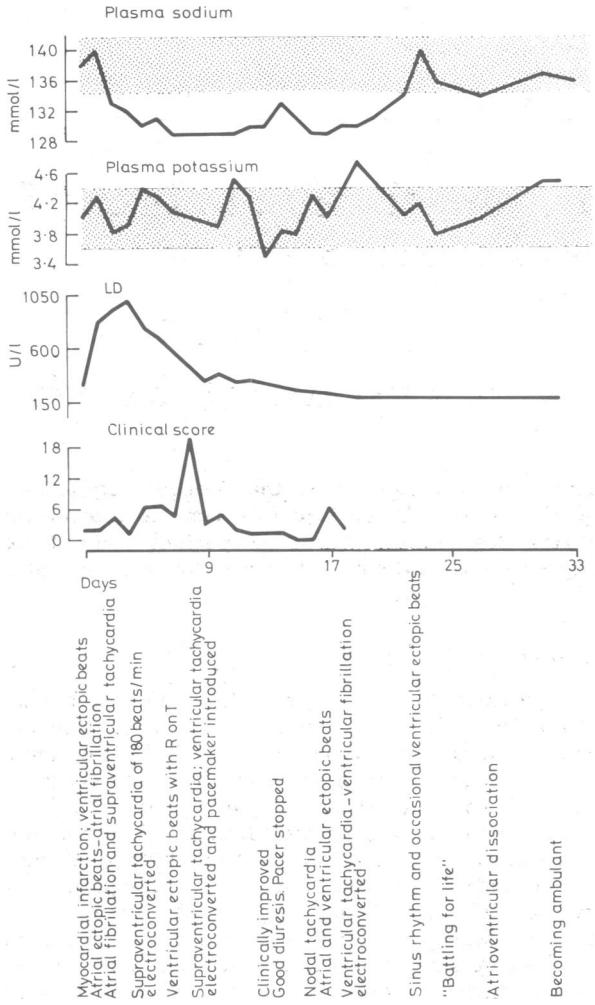

FIG 5—Profile of patient, aged 61, in group 2, who was not given diuretics. Plasma concentrations of sodium and potassium and activity of serum lactate dehydrogenase (LD) given, together with clinical score and notes from nurses' reports throughout admission. Stippled areas indicate extent of normal day-to-day variation in plasma concentrations (±2 SD (from table II) on either side of initial values).

Conversion: SI to traditional units—Plasma sodium and potassium: 1 mmol/l = 1 mEq/l.

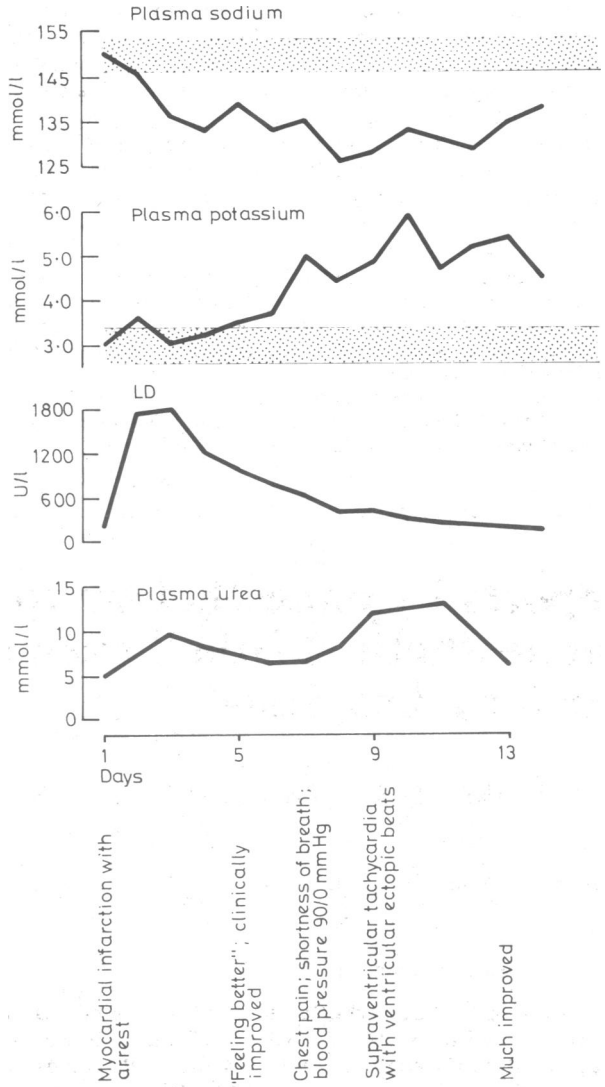

FIG 6—Profile of patient, aged 59 years, in group 2, showing plasma concentrations of sodium, potassium, and urea and serum activity of lactate dehydrogenase (LD). Stippled areas indicate extent of normal day-to-day variation in concentrations (±2 SD (from table II) on either side of initial values).

Conversion: SI to traditional units—Plasma sodium and potassium: 1 mmol/l = 1 mEq/l. Plasma urea: 1 mmol/l ≈ 6 mg/100 ml.

Depletion of both salt and potassium can cause hyponatraemia,[16–18] but neither mechanism is likely to be responsible for hyponatraemia after infarction. Time is too short for widespread depletion to arise. Impaired circulation after infarction could cause hyponatraemia as a result of either redistribution of body water and solutes caused by a widespread increase in cell-membrane permeability[18 19] or a net gain of water caused by an appropriately provoked increase in secretion of antidiuretic hormone. The increased circulating concentrations of catecholamines after acute infarction[2] also increase membrane permeability. Hudson *et al* (submitted for publication) found

that infusions of adrenaline or noradrenaline in dogs had this effect. Water retention might be expected to lower plasma concentrations of potassium and urea as well as sodium. In fact, uraemia was common and hyperkalaemia sometimes seen (figs 5 and 6). Retention of some 1·7 l of water would be needed to reduce a plasma sodium concentration from 139 to 134 mmol/l (fig 2) in a 70 kg adult; and of some 8·4 l to reduce the concentration from 150 to 125 mmol/l (fig 6).

We found that hyponatraemia and uraemia and rises in serum LD and AST activities were all more severe and prolonged in patients given frusemide (figs 2 and 4). Mortality also increased with increased doses. These findings may have occurred because diuretics were not given to the patients with the least severe infarcts, and the highest dose was given to those with the most severe infarcts. Nevertheless, diuretics may conceivably aggravate events after infarction, and this possibility deserves further investigation. It is perhaps pertinent that observations in congestive heart failure[20] have similarly prompted the suggestion that diuretics might enhance catabolism of amino-acids and thereby increase the concentration of urea in body fluids.

Biochemical analyses were carried out by the department of clinical biochemistry. We thank Dr R A L Brewis, consultant physician; resident medical and nursing staff of the coronary care unit; Dr D Appleton, of the department of medical statistics; and Professor D G Julian for their help.

Requests for reprints should be sent to: Dr C T G Flear, University Department of Clinical Biochemistry, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP.

References

1 Oliver, M F, *Circulation*, 1972, **45**, 491.
2 Vetter, N, *et al*, *Lancet*, 1974, **1**, 284.
3 Flear, C T G, *Journal of Clinical Pathology*, 1970, **4**, 16.
4 Flear, C T G, *Postgraduate Medical Journal*, 1960, **36**, 104.
5 Flear, C T G, in *Electrolytes and Cardiovascular Diseases*, ed E Bajusz, p 357. Basle, S Karger, 1966.
6 Flear, C T G, in *Compartments, Pools, and Spaces in Medical Physiology*, ed P-E E Bergner and C C Lushbaugh, p 53. Washington, US Atomic Energy Commission, Division of Technical Information, 1967.
7 Latner, A L, and Skillen, A W, *Lancet*, 1961, **2**, 1286.
8 Wilhelm, S K, *American Journal of Clinical Pathology*, 1951, **21**, 146.
9 Dyckner, T, *et al*, *Acta Medica Scandinavica*, 1975, **197**, 207.
10 Flear, C T G, Singh, C M, and Nandra, G S, communication to the Cardiac Muscle Research Group, Edinburgh, 1976.
11 Flear, C T G, Bhattacharya, S S, and Nandra, G S, in *Nutritional Aspects of Care in the Critically Ill*, ed J R Richards and J M Kinney, p 195. Edinburgh, Churchill Livingstone, 1977.
12 De Mello, W C, *Journal of Physiology*, 1976, **263**, 171.
13 Hercus, V M, McDowall, R J S, and Mendel, D, *Journal of Physiology*, 1955, **129**, 177.
14 McDowall, R J S, Munro, A F, and Zayat, A F, *Journal of Physiology*, 1955, **130**, 615.
15 Glitsch, H G, Reuter, H, and Scholz, H, *Journal of Physiology*, 1970, **209**, 25.
16 McCance, R A, *Lancet*, 1936, **1**, 823.
17 Maffly, R H, and Edelman, I S, *Progress in Cardiovascular Disease*, 1961, **4**, 80.
18 Flear, C T G, and Singh, C M, *British Journal of Anaesthesia*, 1973, **45**, 976.
19 Flear, C T G, *Lancet*, 1974, **2**, 164.
20 Domenet, J G, and Evans, D W, *Quarterly Journal of Medicine*, 1969, **149**, 117.

(*Accepted 16 March 1979*)

Reports by anaesthetists to procurators-fiscal: analysis of "anaesthetic deaths" over 10 years in four Scottish teaching hospitals

A L GILLIES, D S ARTHUR, A L FORREST, J I M LAWSON, A H B MASSON, A A SPENCE

British Medical Journal, 1979, **1**, 1246-1247

Summary and conclusions

Review of 489 "anaesthetic deaths" reported to procurators-fiscal over 10 years disclosed only 30 that were thought to justify such reporting. Most of the remainder occurred in patients so desperately ill at the time of operation that death was expected. Postmortem examinations ordered by the Crown authorities in nearly all cases were probably largely unrewarding and mostly unnecessary.

The results suggest that the present regulations on reporting should be revised to focus more attention on the few deaths that occur in patients who have no apparent contraindication to anaesthesia or operation.

University Department of Anaesthesia, Western Infirmary, Glasgow G11 6NT
A L GILLIES, MB, FFARCS, registrar anaesthetist
A A SPENCE, MD, FFARCS, head of department
Department of Anaesthesia, Royal Infirmary, Glasgow
D S ARTHUR, MB, FFARCS, consultant anaesthetist
Department of Anaesthesia, Ninewells Hospital, Dundee
A L FORREST, MB, FFARCS, consultant anaesthetist
J I M LAWSON, MB, FFARCS, consultant anaesthetist
Department of Anaesthesia, Royal Infirmary, Edinburgh
A H B MASSON, MB, FFARCS, consultant anaesthetist

Introduction

The requirement to notify deaths associated with anaesthesia or operation or both to a procurator-fiscal in Scotland or to a coroner in England and Wales was introduced in 1904 (although there have been minor modifications to the regulations from time to time). Deaths in three categories must be reported: (1) death occurring during anaesthesia or operation; (2) death clinically attributable to the anaesthetic; and (3) death occurring in a period not exceeding 12 hours after operation. Inquiries resulting from notification are intended to serve the public interest by establishing whether criminal negligence may have occurred. In almost all cases in Scotland the fiscal's medical adviser conducts a postmortem examination; the report is retained by the fiscal and transmitted to the office of the Lord Advocate. The deceased's doctors are not informed of the findings.

Some anaesthetists believe that the regulations are inappropriate to modern practice,[1 2] since techniques of inducing anaesthesia and the distribution of causes of death during and after operation have changed since 1904. That this belief is shared by others is evident from comments in the *Report of the*