cases of self-poisoning coming to hospital and not just with those cases somehow selected for psychiatric investigation.

Those with no psychiatric illness tended to be younger ; those with organic illnesses were generally old. Apart from this the relation between diagnosis and age was not close.

The principal diagnosis was related to the index of endangering life (Table VIII). The acts of patients with a formal psychiatric illness (excluding character disorder) were more life-endangering than those of patients with personality abnormality or those of patients without a psychiatric illness. Yet the majority of acts of even the more seriously ill people were clearly pointed towards survival.

TABLE VIII.—*Diagnosis and Index of Endangering Life*

Predictable Outcome	All Psychiatric Illnesses (292)	Personality Disorder Only (108)	No Psychiatric Diagnosis (113)
Death	25%	12%	12%
Death probable	11%	11%	10%
Death unlikely	20%	26%	30%
Certain to survive	43%	51%	49%

χ^2 * = 18·99.   3 degrees of freedom. $P < 0.001$.
* Calculated by comparing all psychiatric illness against the two other categories combined.

The different diagnostic groups do not vary much in the methods they choose. The only features of note are that those without a psychiatric illness use aspirin rather more often than the others, and that people with an organic illness use coal-gas more. These findings are probably attributable to the ages of those involved.

Quite commonly patients who were suffering from depression had an underlying character disorder. The combination of depression and psychopathy often occurred. This conjunction seems especially prone to manifest in self-poisoning acts. Personality disorder, either as a principal or as an accessory diagnosis, was recognized in 41% of men and 27% of women patients, and about half of these were classed as psychopaths. A smaller proportion appeared to us to be abnormally immature in their outlook. No other sort of personality abnormality occurred frequently.

Associated diagnostic factors included alcoholism, drug addiction, epilepsy, and subnormality. Fifty-two per cent. of men had one or more of these conditions, and the dominant factor was alcoholism. Thirty-nine per cent. of the men and 8% of the women were alcoholics, seasoned drinkers, unquestionably addicted, many of them bearing the physical signs of chronic alcoholism. Alcoholism is a major factor predisposing to self-poisoning. So is alcohol itself : 56% of the men and 23% of the women had been drinking just before the act took place—

a deliberate act in every case. The methods adopted by alcoholics did not differ from those employed by the generality of patients, and the acts which led to their admission, and to the admission of those inebriated at the time, were neither more nor less serious than those of other people. But of the six patients, four men and two women, who killed themselves within a year of discharge, five were alcoholics.

Recommended Disposal

Twenty-six per cent. of the patients were sent for further in-patient psychiatric treatment, 38% were recommended out-patient treatment, which, unless they had previously been under the care of another Edinburgh psychiatrist, was carried out by our own service. For 36% no further psychiatric treatment was arranged (Table IX) : sometimes because the patient refused our suggestion, but in most cases because we did not think that further treatment was called for. This was either because there was no condition present to treat, and the tangled precipitating social web had been unravelled while the patient was still in our care, or because the personality disorder from which the patient suffered was so ingrained that treatment would not avail. Diagnosis is correlated with disposal, but this was not primarily decided on the basis of the diagnosis made. The proportion recommended for in-patient care must inevitably depend upon availability of psychiatric beds. Similarly the extent of out-patient care depends upon the amount of psychiatric time allotted for this.

TABLE IX.—*Diagnosis and Disposal*

		Patients with a Psychiatric Illness (286)	Patients with Personality Disorder Only (103)	Patients with no Psychiatric Diagnosis (112)	All Patients* (501)
Further psychiatric treatment	In-patient	37% } 77%	17% } 51%	8% } 45%	26% } 64%
	Out-patient	40%	34%	37%	38%
No further psychiatric treatment		23%	49%	55%	36%

* Excluding 21 patients who discharged themselves before disposal was arranged.

It does not follow that the patient can benefit from treatment only if he has a psychiatric illness. Nearly half of those without such illness were judged to be helpable by further care, a term which embraces social work as well as psychiatric therapy. Generally this was on an out-patient basis, but 8% of them required to be in-patients so that they might properly be tided over the acute crisis situation which had caused their act.

[*Part II will appear in our next issue.*]

Pulmonary Function in Acute Myocardial Infarction

M. W. McNICOL, M.B., M.R.C.P. ;   BRIAN J. KIRBY,* M.B. ;   K. D. BHOOLA,* M.B., B.CH., B.A.O., PH.D. ;
M. E. EVEREST,* M.B., M.R.C.P. ;   H. V. PRICE,* M.B., D.C.H. ;   S. F. FREEDMAN,* M.B., M.R.C.P.

In studying the disturbance of arterial blood gases in patients with acute myocardial infarction it soon became apparent that many had evidence of pulmonary congestion (McNicol *et al.*, 1964). This paper reports the changes in arterial blood gases found in a large number of patients with acute myocardial infarction and the results of several procedures carried out with

a view to assessing the effect of pulmonary congestion on the function of the lungs.

Material

Observations were made in the years 1962–4 on patients admitted to Central Middlesex Hospital with acute myocardial infarction and pulmonary congestion from other causes ; those

* Cardiothoracic Department, Central Middlesex Hospital, London.

with chronic bronchitis or pulmonary embolism were excluded. The patients' ages ranged from 38 to 83 years, and they were classified into the following groups:

Group 1: Uncomplicated myocardial infarction (14 patients).

Group 2: Myocardial infarction with " shock " (2 patients).

Group 3: Myocardial infarction with " shock " and pulmonary congestion (13 patients).

Group 4: Myocardial infarction with pulmonary congestion (44 patients).

Group 5: Pulmonary congestion from other causes (19 patients).

Diagnostic Criteria

Myocardial infarction was diagnosed when there was an appropriate history accompanied by electrocardiographic evidence of recent transmural infarction (W.H.O., 1959) or by lesser electrocardiographic changes suggestive of myocardial infarction if these were accompanied by a rise in serum glutamic-oxaloacetic transaminase or serum hydroxybutyric dehydrogenase (Wilkinson, 1962), or by the finding of acute coronary occlusion or myocardial infarction at necropsy. Pulmonary congestion was diagnosed when examination of the chest showed basal or more widespread crepitations not cleared by cough, or rhonchi in the absence of a history of bronchitis, or a combination of these signs. Twenty-three of the 57 patients in groups 1 and 4 with these signs were acutely dyspnoeic and orthopnoeic ; the remainder were not breathless.

" Shock " was diagnosed if there was persistent hypotension —systolic blood-pressure of less than 90 mm. Hg for more than 30 minutes. Most of the patients in this group had hypotension for much longer and had other manifestations of circulatory failure—pallor, coldness of the skin, restlessness, etc.

Methods

Blood was taken by percutaneous puncture of the brachial or femoral arteries, a No. 1 hypodermic needle (21 S.W.G., 0.8 mm. external diameter) being used. All patients were in bed, and, unless dyspnoeic, were lying supine. When more prolonged studies were made a Teflon catheter was inserted into the brachial artery.

All samples were analysed immediately. Oxygen saturation was measured with a Brinkman Haemoreflector (Kipp and Zonen, Delft). In some patients oxygen tension was measured directly with a polarographic electrode (Beckman macro-electrode, Beckman Instruments Inc.). In the remainder oxygen tension was calculated from the oxygen saturation by the use of Dill's dissociation curve, taking into account the effect of pH. The overall accuracy of this procedure is ± 5 mm. Hg at oxygen saturations below 90% and ± 10 mm. above 90%.

Carbon dioxide tension was measured by a Severinghaus CO_2 electrode (Severinghaus and Bradley, 1958) and pH with an Astrup micro-electrode (Radiometer, Copenhagen). Plasma bicarbonate was calculated from the observed values by use of the Henderson-Hasselbach equation.

Expired-air collections were made, a low-resistance valve (Siebe Gorman) and a lightweight Douglas bag being used. Collections were taken over two minutes after an initial period of acclimatization to the mouthpiece. Gas volume was measured with a dry-gas meter (Parkinson-Cowan). The expired air was analysed for oxygen and carbon dioxide by means of Lloyd's modification of the Haldane apparatus (Lloyd, 1958). From these figures and the values of simultaneously taken arterial blood the following were calculated: respiratory exchange ratio ; dead-space/tidal-volume ratio and physiological dead space ; alveolar oxygen tension ; alveolar-arterial oxygen tension

gradient. When no expired air collection was made alveolar-arterial oxygen tension gradient was calculated, an R.Q. of 0.8 being assumed.

Oxygen (100%) was given by means of a valve-box and reservoir bag. Blood samples were taken after 20 minutes.

In 11 patients blood was taken before and four hours after the intravenous administration of a diuretic (chlorothiazide 0.5 g. or ethacrynic acid 100 mg.). In these patients the F.E.V.$_1$ and F.V.C. were also measured with a portable bellows spirometer (Vitalograph, Garthur).

Results

Arterial Blood Gas Analysis

Oxygen (Table I).—Oxygen saturation was normal only in group 1 (uncomplicated myocardial infarction), but even in this group the mean oxygen tension was below normal and the alveolar-arterial oxygen tension gradient increased. " Shock " (group 2) and pulmonary congestion (group 4) were associated with greater changes and arterial desaturation. The most severe changes were found in patients with both " shock " and pulmonary congestion (group 3). Pulmonary congestion not due to acute myocardial infarction (group 5) produced changes similar to those seen in groups 2 and 4.

TABLE I.—*Arterial Blood : Oxygen*

Group	Diagnosis	No. of Cases	Mean O_2 Satn. (%)	Mean Pao$_2$ (mm.Hg)	A-a Gradient (mm.Hg)
1	Myocardial infarction	14	93 ± 3*	73 ± 14*	31 ± 16*
2	Myocardial infarction with shock	2	89	61	57
3	Myocardial infarction with shock and pulmonary congestion	13	82 ± 8	53 ± 14	58 ± 14
4	Myocardial infarction and pulmonary congestion	44	87 ± 8	59 ± 12	45 ± 13
5	Pulmonary congestion from other causes	19	86 ± 8	55 ± 13	54 ± 15
Normal values (Comroe *et al.*, 1962)			Above 93	85–95	10

* The figures following the mean values represent one standard deviation.

Carbon Dioxide Tensions, pH, and Plasma Bicarbonate (Table II).—Group mean values for carbon dioxide tension were at the lower limit or slightly below normal. Group mean values for pH and bicarbonate were normal. Individual patients with " shock " showed a fall in pH and bicarbonate.

TABLE II.—*Arterial Blood : Carbon Dioxide, pH, and Bicarbonate*

Group	Mean pH	Mean Pco$_2$ (mm.Hg)	Mean Plasma Bicarbonate (mEq/l.)
1	7·41 (7·35–7·48)*	37 ± 5†	23 ± 3†
2	7·41	26	16
3	7·32 (7·17–7·55)	32 ± 8	21 ± 7
4	7·39 (7·32–7·48)	37 ± 5	22 ± 4
5	7·43 (7·37–7·49)	33 ± 6	21 ± 3
Normal values (Comroe *et al.*, 1962) ..	7·36–7·45	36–45	20–24

* One standard deviation range.
† One standard deviation.

Expired-air Collections (Table III)

The dead-space/tidal-volume ratio was raised in all the patients in whom it was measured and the physiological dead space was raised in the majority, sometimes strikingly so (Cases 5 and 88). In only two patients was the value of the respiratory exchange ratio above 1, indicating that most of the gas collections were satisfactory.

TABLE III.—*Expired-air Collections*

Patient No.	Group No.	Resp. Exchange Ratio	VD/VT	VDphys. (ml.)
1	3	0·93	0·75	—
2	5	0·85	0·53	182
3	2	1·36	0·43	171
4	3	0·82	0·51	143
5	3	0·83	0·54	550
6	3	0·96	0·42	155
13	4	0·86	0·36	139
19	4	1·01	0·45	—
23	4	0·77	0·55	220
69	4	—	0·44	294
79	5	0·75	0·41	211
84	4	0·79	0·49	182
85a	4	—	0·48	130
85b	4	0·84	0·71	217
88	4	0·72	0·41	310
89	4	0·76	0·57	288
Mean values		0·87	0·50	228
Normal values		0·80	0·30	150

Effects of Oxygen (Table IV and Fig. 1)

Administration of 100% oxygen by means of a mouthpiece and valve-box resulted in a rise in the oxygen tension in all the patients studied. In seven patients a normal rise in oxygen tension was not obtained. This was especially striking in two patients. The reasons for this are discussed below. The diagnostic categories of these patients are shown in the Table.

TABLE IV.—*Effect of 100% Oxygen*

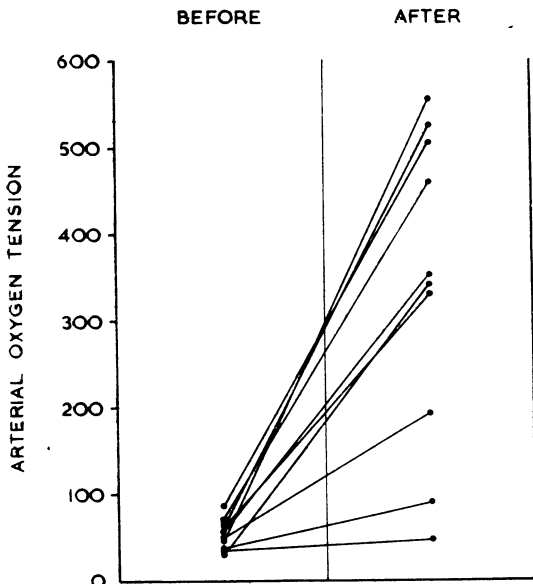| Patient No.	Group No.	Pao₂ (mm.Hg) Before	Pao₂ (mm.Hg) After
59	3	68	525
63	4	59	194
79	5	47	555
80	3	71	460
81	3	38	92
82	4	37	341
85	4	38	48
88	4	63	350
89	4	56	330
90	4	88	505

FIG. 1.—Effects on arterial oxygen tension of the administration of 100% oxygen.

Effects of Diuresis (Tables V and VI, Fig. 2)

When the initial oxygen saturation was less than 90% a significant rise in the arterial oxygen tension was produced by diuretics (P<0.01), together with a fall in alveolar-arterial gradient. The F.E.V.₁ and F.V.C. also rose in these patients.

TABLE V.—*Effect of Diuretics. Arterial Oxygen*

Patient No.	Before O₂ Satn. (%)	Before Pao₂ (mm. Hg)	Before A–a Gradient (mm. Hg)	After O₂ Satn. (%)	After Pao₂ (mm. Hg)	After A–a Gradient (mm. Hg)
	Saturation less than 90%					
57	86	53	53	95	77	38
60	89	54	57	90	58	54
69	89	55	56	93	63	45
73	89	59	54	94	73	39
82	85	61	35	94	75	34
84	86	51	55	91	58	52
85	71	37	77	66	36	66
89	77	41	67	81	45	51
	Saturation over 90%					
61	96	87	22	97	93	15
63	92	63	38	91	61	45
87	90	61	42	92	65	43

TABLE VI.—*Effect of Diuretics. F.E.V. and F.V.C.*

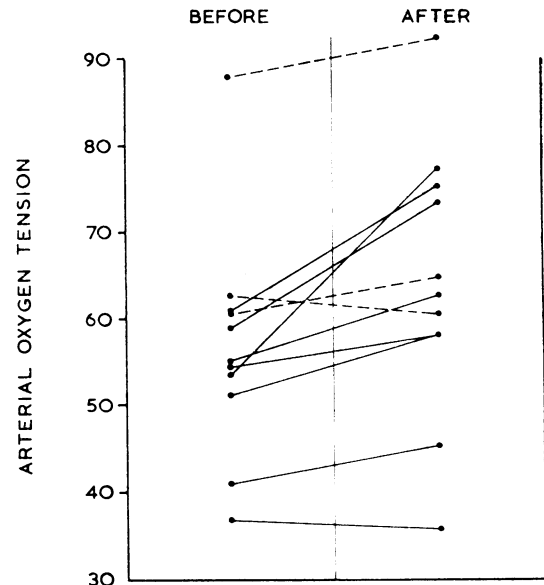| Patient No.	Before F.E.V.₁ (L.)	Before F.V.C. (L.)	After F.E.V.₁ (L.)	After F.V.C. (L.)
	Saturation less than 90%			
57	0·7	0·9	0·7	1·0
60	0·5	1·0	1·3	1·8
69	1·6	1·8	1·45	2·2
73	1·1	1·35	1·25	1·6
82	0*	0*	0*	0·2
84	1·3	2·05	1·6	2·45
85	1·1	1·4	1·5	1·9
89	0·45	0·8	1·15	1·6
	Saturation over 90%			
61	2·05	2·55	2·35	2·85
63	1·1	1·3	1·35	1·55
87	0·8	1·45	0·95	1·6

*Unrecordable.

FIG. 2.—Effect on arterial oxygen tension of the administration of an intravenous diuretic. The continuous lines denote patients in whom the oxygen saturation was below 90% at the start; the broken lines denote those with oxygen saturations above 90% before diuresis.

Discussion

Of the 73 patients with acute myocardial infarction 57 (78%) had evidence of pulmonary congestion as defined by strict clinical criteria. Other complications, notably " shock," were much less common—15 cases (21%). That pulmonary congestion is a very common complication of myocardial infarction is not surprising. Lee (1957) has recorded an almost invariable rise in central venous pressure in acute myocardial infarction ; a rise in pulmonary venous pressure must be even more common, for the left ventricle is predominantly involved in

myocardial infarction. It might be suggested that our diagnostic criteria for pulmonary congestion were so wide as to be of little significance, but the results of blood-gas analysis on our patients show clearly that even these minor clinical signs were associated with a well-marked deterioration of pulmonary function. Like previous observers (Vitale *et al.*, 1954), we have found no correlation between the clinical assessment of severity of pulmonary congestion and the functional changes it produces. A few basal crepitations may be associated with as great a functional disturbance as many crepitations in both lung fields. Our findings show that not only is pulmonary congestion a common complication of myocardial infarction, but also that it produces significant impairment of oxygenation by the lung.

The characteristic change in the arterial blood gases in myocardial infarction is a fall in arterial oxygen tension with an increase in the alveolar-arterial gradient. The fall in oxygen tension is not due to underventilation of the lungs ; in most of the patients the low carbon dioxide tension indicates overventilation. These changes were not pronounced in those patients with uncomplicated myocardial infarction, but were most often found in those with pulmonary congestion. Similar changes were found in those with " shock," but " shock " was almost invariably accompanied by pulmonary congestion.

Our investigations are not sufficient to give precise information on the mechanism of this disorder of pulmonary function. Either pulmonary congestion with a rise in pulmonary venous pressure or a fall in cardiac output consequent on myocardial infarction (Malmcrona and Varnauskas, 1964) could cause disturbance of ventilation–blood-flow relationships in the lungs. Pulmonary congestion with interstitial oedema would impair gas transfer from the alveolus to pulmonary capillary. Either of these mechanisms would be associated with a rise in alveolar-arterial oxygen gradient. The increase in alveolar-arterial oxygen gradient in the two patients with myocardial infarction and persistent hypotension suggests that ventilation–blood-flow disturbance secondary to change in cardiac output can be important ; this would be expected in an organ like the lung, whose perfusion is largely determined by gravity (West, 1963), and the pattern of perfusion of which will be very sensitive to changes in cardiac output. The gas collections showed a large physiological dead space, indicating ventilation of unperfused alveoli in keeping with such a hypothesis. Breathing of pure oxygen should abolish the hypoxaemia produced in either of these ways, and 7 of the 10 patients to whom pure oxygen was given showed such a response. In the remaining three there was a much smaller rise in oxygen tension. It seems unlikely that an anatomic shunt is present in these cases. It is suggested that the failure to achieve a satisfactory rise in oxygen tension is due to a very severe ventilation–blood-flow imbalance with perfusion of completely unventilated alveoli, the circulatory counterpart of the increased dead space.

Unlike the changes associated with " shock " in myocardial infarction which are resistant to treatment (MacKenzie *et al.*, 1964), we found it was possible to influence the changes associated with pulmonary congestion not only by oxygen therapy but also by the use of diuretics. A significant improvement was produced within four hours, not only in arterial blood gases but also in ventilatory function, by the use of a rapidly acting intravenous diuretic in those patients in whom there was a marked physiological disturbance (O_2 saturation less than 90%). Patients with less abnormality of blood gases did not show any significant response. Thus the most common hypoxaemia in acute myocardial infarction can be relieved by treatment—" symptomatically " by oxygen therapy and more fundamentally by diuretics—and the more severe the hypoxaemia the more likely it is to be relieved.

There was no difference in the severity of the blood-gas changes in those with pulmonary congestion from acute myocardial infarction and those with pulmonary congestion from other causes. Within the scope of our investigation there were no distinguishing features in the physiological changes produced by either cause.

The abnormalities we have described and their response to treatment may be of considerable significance. The patients who died had significantly greater arterial hypoxaemia than those who survived (mean oxygen tension of those who died, 52 mm. and O_2 saturation 83%, compared with mean O_2 tension of 63 mm. and O_2 saturation of 89% in survivors—P<0.01). Of the 23 patients with oxygen saturation below 85% 13 (56%) died, and of the 64 with oxygen saturation above 85% 12 (19%) died. The more severe hypoxaemia may only reflect greater severity of the disease process, but it may also be a contributory factor in the fatal outcome, as was found by Freeman and Nunn (1963) in experimentally induced shock, when anoxia caused respiratory depression, not respiratory stimulation. In acute myocardial infarction oxygen therapy will rapidly relieve the hypoxaemia most commonly found—that due to pulmonary congestion—and diuretic therapy will bring about more fundamental improvement.

Follow-up studies on these patients are not yet complete ; they will be published at a later date. The abnormalities of arterial oxygenation present in the acute stage have greatly diminished or disappeared in 37 of the patients so far studied. Disappearance of the chest signs has occurred in 30 of these cases.

Summary

Of 73 patients with acute myocardial infarction 57 (78%) showed evidence of pulmonary congestion. This was associated with arterial hypoxaemia, which could be relieved by oxygen therapy or, if severe, by diuretics. It is suggested that pulmonary congestion is the commonest cause of hypoxaemia in myocardial infarction and that its treatment may improve the prognosis in acute myocardial infarction.

REFERENCES

Comroe, J. H., Forster, R. E., Dubois, A., Briscoe, W. A., and Carlsen, E. (1962). *The Lung*, p. 323 *et seq.* Year Book Medical Publishers, Chicago.
Freeman, J., and Nunn, J. F. (1963). *Clin. Sci.*, 24, 135.
Lee, G. de J. (1957). *Brit. Heart J.*, 19, 117.
Lloyd, B. B. (1958). *J. Physiol. (Lond.)*, 143, 5 P.
MacKenzie, G. J., Taylor, S. H., Flenley, D. C., McDonald, A. H., Staunton, H. P., and Donald, K. W. (1964). *Lancet*, 2, 825.
McNicol, M. W., Kirby, B. J., Everest, M. S., Freedman, S., and Bhoola, K. D. (1964). Ibid., 2, 1180.
Malmcrona, R., and Varnauskas, E. (1964). *Acta med. scand.*, 175, 1.
Severinghaus, J. W., and Bradley, A. F. (1958). *J. appl. Physiol.*, 13, 515.
Vitale, A., Dumke, P. R., and Comroe, J. H. (1954). *Circulation*, 10, 81.
West, J. B. (1963). *Lancet*, 2, 1055.
Wilkinson, J. H. (1962). *An Introduction to Diagnostic Enzymology*, p. 250. Williams and Wilkins, Baltimore.
World Health Organization (1959). *Techn. Rep. Ser.*, No. 168.