been found to be greater than was previously thought (Stone, Luhby, Feldman, Gordon, and Cooperman, 1967), sometimes no doubt owing to the slow return to normal of folate status following a previous pregnancy (Temperley *et al.*, 1968). This sequence of events could occur if those patients on iron alone and with low postnatal whole blood folate levels soon became pregnant again.

Conclusions

Folate depletion in late pregnancy is again found to be common when assessed by the whole blood folate level, but only when this is tested some weeks after delivery, because of the delay in its reflection of folate status.

This depletion is prevented by the prophylactic administration of 330 μg. of folic acid a day, and the whole blood folate level six weeks after delivery is then close to the initial pre-treatment levels in early pregnancy in this group of patients.

Though the whole blood folate is slow to reflect changes in folate status (compared with the serum level) it is a valid index of this in that a low value in early pregnancy indicates an increased liability to development of overt megaloblastic changes in late pregnancy.

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Circulatory and Metabolic Effects of Oxygen in Myocardial Infarction

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Summary: The circulatory and metabolic effects of inhalation of oxygen in high concentration were investigated in 50 patients with acute myocardial infarction. The heart rate, arterial blood pressure, cardiac output, blood gas tensions, pH, and lactate and pyruvate levels were measured. In general, oxygen inhalation produced a fall in cardiac output and stroke volume and a rise in blood pressure and systemic vascular resistance. In a small number of patients with very low cardiac outputs there was a rise in output. A substantial rise in arterial oxygen tension was obtained even in patients with low initial values. The raised arterial blood lactate levels which were frequently present were reduced after oxygen. The therapeutic implications of these effects are discussed.

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Introduction

The circulatory disturbances associated with myocardial infarction have recently been the subject of several detailed studies (Broch *et al.*, 1959; Malmcrona and Varnauskas, 1964; Thomas *et al.*, 1965a). In addition to haemodynamic changes, one of the important findings has been a reduction in arterial oxygen tension, especially in the presence of left ventricular failure and cardiogenic shock (MacKenzie *et al.*, 1964; McNicol *et al.*, 1965; Valentine *et al.*, 1966). Hypoxia may be largely responsible for some of the serious metabolic abnormalities which take place and may also contribute to the production of hypotension. Though administration of oxygen has long been recommended in the management of myocardial infarction (Dunlop and Alstead, 1966; Friedberg, 1966) there is little information available concerning its effects on the underlying circulatory derangements (MacKenzie *et al.*, 1964; Thomas *et al.*, 1965b; Cameron *et al.*, 1966). The object of the present investigation, therefore, was to determine the haemodynamic and metabolic effects of the administration of oxygen in high concentration to patients with myocardial infarction and to assess its therapeutic value.

Patients and Methods

Fifty men with acute myocardial infarction were studied. Their ages ranged from 34 to 77, with a mean of 57.6 years. The diagnosis was based on a history of cardiac pain lasting more than 30 minutes, of acute left ventricular failure, or of unconsciousness, and was confirmed by electrocardiograms which showed pathological Q waves and sequential ST–T-wave changes. Investigations were carried out in all cases within 24 hours of infarction, and Table I summarizes the time intervals elapsing between the onset of symptoms and the beginning of the investigations. Serial serum aspartate aminotransferase estimations were carried out and the maximum values obtained are shown in Table II.

TABLE I.—*Duration of Myocardial Infarction Before Commencement of the Investigation in 50 Patients*

Time (hours)	0–6	6–12	12–18	18–24
No. of cases	38	6	4	2

TABLE II.—*Maximum Serum Aspartate Aminotransferase Levels*

Frankel units per ml. ..	50–200	201–300	301–400	Above 400
No. of cases	20	10	8	12

The patients were admitted to a special unit with facilities for coronary care and investigation. After initial clinical assessment, fine polyethylene catheters were introduced percutaneously under local anaesthesia into a brachial artery and antecubital vein and advanced centrally. The intravascular pressures and the electrocardiogram were monitored continuously by means of Devices strain gauge transducers and direct-writing recorder. Cardiac output was measured by a dye-dilution technique, the procedure described by Taylor and Shillingford (1959) being followed. Coomassie Blue (sodium anoxynaphthonate) was injected from a calibrated syringe, the average dose being 40 mg. The concentration of dye in the plasma was estimated by dye extraction or by a modification of the method of Deane *et al.* (1966). The cardiac output was measured in duplicate at each stage and the mean of the results was taken. The systemic vascular resistance was calculated from the formula:

$$\frac{\text{Mean arterial pressure (mm. Hg)}}{\text{Cardiac output (litres/min.)}} \times 80 \text{ dynes sec. cm.}^{-5}$$

Left ventricular work was determined from the formula:

$$\frac{13.6 \times \text{mean arterial pressure (mm. Hg)} \times \text{cardiac output (litres/min.)}}{1{,}000} \text{ kg.-m./min.}$$

Blood gases were measured with an Instrumentation Laboratory Inc. system (Model I.L. 113). The arterial oxygen tension was measured with a Clark electrode. The carbon dioxide tension was measured with a Severinghaus electrode and the *p*H with a glass electrode. The system was calibrated with known gas samples and carefully tonometered blood. Arterial blood lactate and pyruvate levels were estimated by Boehringer enzymatic methods.

Oxygen was administered by means of a close-fitting facemask connected to a humidifier and low-resistance demand valve (McDowall *et al.*, 1965). When oxygen is breathed this

TABLE III.—*Haemodynamic Findings in 50 Patients with Acute Myocardial Infarction Breathing Air and Oxygen*

Case No.	Heart Rate (per min.)		Arterial B.P. (mm. Hg)		Mean Arterial B.P. (mm. Hg)		Cardiac Output (l./min.)		Systemic Vascular Resistance (dynes sec. cm.$^{-5}$)		Stroke Volume (ml.)	
	Air	Oxygen	Air	Oxygen	Air	Oxygen	Air	Oxygen	Air	Oxygen	Air	Oxygen
1	125	117	135/90	137/92	105	107	4·83	4·56	1,739	1,877	39	39
2	68	66	127/65	136/70	86	92	8·70	6·51	791	1,131	128	99
3	58	55	105/45	125/60	68	82	2·00	1·73	2,720	3,791	35	31
4	54	58	124/60	154/64	80	98	5·76	4·66	1,111	1,682	107	80
5	72	71	80/52	77/55	61	62	3·50	3·48	1,394	1,425	49	49
6	68	70	112/90	120/77	97	90	6·24	4·97	1,243	1,448	92	71
7	56	58	75/40	77/40	52	60	3·70	3·85	1,124	1,247	66	66
8	100	96	137/65	155/80	85	107	7·01	5·14	970	1,665	70	53
9	66	66	150/90	158/120	110	133	6·21	5·45	1,417	1,953	94	83
10	72	76	157/98	180/112	115	142	7·50	5·70	1,227	1,993	104	75
11	80	76	140/92	140/82	100	100	6·15	5·96	1,301	1,343	77	78
12	66	70	137/83	138/90	94	105	3·80	3·62	1,980	2,320	58	52
13	124	112	152/85	150/90	95	100	4·45	4·16	1,708	1,923	36	37
14	100	108	75/47	107/65	57	77	5·20	4·66	877	1,322	52	43
15	70	68	114/55	145/70	80	88	4·44	4·14	1,441	1,700	63	61
16	120	100	125/75	110/67	80	82	7·83	6·66	817	985	65	66
17	120	110	112/55	117/60	70	77	9·55	7·78	586	792	80	71
18	80	64	65/30	87/42	50	54	4·93	3·44	811	1,256	62	54
19	84	84	117/70	125/70	87	90	5·68	4·92	1,225	1,463	68	59
20	72	74	110/45	120/60	62	75	7·19	6·65	690	902	100	90
21	60	56	102/55	102/52	70	70	5·59	5·42	1,002	1,033	93	97
22	68	58	87/45	97/55	60	70	3·84	4·15	1,250	1,349	57	72
23	64	66	140/82	125/75	102	95	3·66	3·56	2,229	2,135	57	54
24	74	82	87/57	113/75	67	88	4·28	4·40	1,252	1,600	58	54
25	64	76	180/80	180/90	115	125	5,58	5·88	1,649	1,701	87	77
26	100	100	130/100	140/110	110	124	6·11	4·53	1,440	2,190	61	45
27	57	55	100/52	132/66	68	88	3·47	3·77	1,568	1,867	61	69
28	56	54	108/74	122/72	88	94	4·53	4·10	1,554	1,834	81	76
29	83	83	96/58	94/60	68	72	4·41	3·41	1,234	1,689	53	41
30	83	83	152/74	152/68	100	96	5·40	5·16	1,481	1,488	65	62
31	61	42	136/54	76/28	78	44	3·41	2·03	1,830	1,734	56	48
32	75	65	240/137	230/125	180	160	3·61	5·39	3,989	2,375	48	83
33	83	85	100/64	132/72	80	90	5·80	5·01	1,104	1,437	70	59
34	115	116	106/74	102/66	88	80	2·82	3·11	2,496	2,058	25	27
35	102	114	60/40	52/40	44	40	1·32	2·56	2,667	1,250	13	23
36	107	125	150/96	154/100	114	118	5·17	4·96	1,764	1,903	48	40
37	82	70	118/84	134/74	96	94	3·46	3·67	2,220	2,049	42	52
38	88	88	160/104	162/112	116	126	6·16	5·57	1,506	1,810	70	63
39	96	105	128/53	146/71	68	97	4·66	3·79	1,167	2,047	48	36
40	48	51	116/56	116/58	77	82	3·30	3·63	1,867	1,807	69	71
41	90	99	100/48	102/50	62	72	5·79	5·25	857	1,097	64	53
42	71	74	136/68	132/64	90	88	5·60	5·86	1,286	1,201	79	79
43	98	89	142/76	144/74	88	100	5·71	4·73	1,233	1,691	58	53
44	81	96	116/70	126/70	81	96	5·08	4·64	1,276	1,655	63	48
45	81	92	140/91	145/95	110	110	5·63	5·72	1,563	1,538	69	62
46	73	73	94/56	106/62	70	72	4·58	4·28	1,223	1,346	63	59
47	100	99	138/44	150/70	66	100	7·53	6·41	701	1,248	75	65
48	83	93	90/56	112/62	66	78	6·11	5·63	864	1,108	74	61
49	94	108	90/60	92/64	70	73	5·86	6·27	956	931	62	58
50	71	69	100/64	136/70	76	92	7·43	6·41	818	1,148	105	93
Mean values	81	81	120/68	127/72	84	91	5·21	4·75	1,424	1,610	66	61

TABLE IV.—*Arterial Blood Oxygen and Carbon Dioxide Tensions, pH, and Lactate and Pyruvate Values in 50 Patients with Myocardial Infarction Breathing Air and Oxygen*

Case No.	Pa_{O_2} (mm.Hg)		Pa_{CO_2} (mm.Hg)		*p*H		Arterial Blood Lactate (mM/l.)		Arterial Blood Pyruvate (mM/l.)	
	Air	Oxygen	Air	Oxygen	Air	Oxygen	Air	Oxygen	Air	Oxygen
1	69	575	37	26	7·42	7·55	—	—	—	—
2	85	430	36	34	7·45	7·47	—	—	—	—
3	86	491	39	36	7·46	7·41	—	—	—	—
4	98	486	17	30	7·58	7·58	2·555	1·538	—	—
5	97	295	38	32	7·33	7·42	—	—	—	—
6	97	538	—	—	—	—	—	—	—	—
7	88	522	40	44	7·30	7·30	—	—	—	—
8	53	261	38	42	7·43	7·40	—	—	—	—
9	90	435	44	40	7·40	7·53	—	—	—	—
10	67	281	39	39	7·46	7·37	—	—	—	—
11	50	260	34	35	7·44	7·44	1·356	1·057	—	—
12	100	342	39	39	7·40	7·38	—	—	—	—
13	43	390	29	34	7·47	7·47	1·658	1·968	0·048	0·042
14	24	141	37	36	7·25	7·37	5·700	4·539	0·172	0·157
15	61	367	37	33	7·45	7·44	1·969	1·658	0·047	0·076
16	63	470	41	38	7·45	7·46	2·528	1·177	0·036	0·042
17	84	423	32	41	7·44	7·47	3·357	2·582	0·029	0·035
18	70	406	37	38	7·40	7·44	2·020	2·069	0·043	0·027
19	59	406	35	39	7·52	7·51	0·910	1·334	0·083	0·045
20	61	496	34	36	7·48	7·45	—	—	—	—
21	52	481	34	31	7·50	7·55	2·502	1·565	0·049	0·039
22	60	565	37	37	7·50	7·49	0·874	1·177	0·047	0·051
23	81	542	31	31	7·53	7·49	2·582	1·946	0·059	0·057
24	82	603	33	36	7·46	7·45	1·322	1·334	0·042	0·031
25	54	366	39	42	7·45	7·43	1·283	1·334	0·037	0·047
26	51	486	44	41	7·43	7·38	1·632	1·346	0·044	0·040
27	65	625	46	46	7·40	7·48	1·929	0·681	0·028	0·025
28	70	610	42	50	7·47	7·47	1·512	0·775	0·021	0·020
29	51	459	37	39	7·44	7·44	1·217	0·990	0·059	0·054
30	56	496	45	48	7·38	7·32	4·722	3·397	0·025	0·032
31	68	378	—	—	—	—	1·256	1·230	0·015	0·024
32	58	475	—	—	—	—	4·491	3·357	0·009	0·009
33	56	423	37	43	7·45	7·42	2·742	1·432	0·025	0·027
34	73	649	35	34	7·32	7·33	6·654	2·327	0·033	0·037
35	54	181	32	35	7·35	7·32	7·056	6·434	—	—
36	73	452	36	37	7·45	7·45	—	—	—	—
37	59	440	39	44	7·48	7·42	1·485	1·070	0·062	0·019
38	61	474	41	50	7·43	7·37	1·163	0·626	0·043	0·020
39	34	237	40	45	7·39	7·35	2·808	2·134	0·086	0·052
40	59	542	47	39	7·41	7·50	0·496	0·495	0·024	0·039
41	79	571	41	43	7·42	7·43	1·310	1·458	0·030	0·042
42	73	311	34	30	7·43	7·48	—	—	—	—
43	63	396	35	38	7·42	7·44	2·477	1·203	0·043	0·025
44	51	510	35	36	7·55	7·57	2·011	1·043	0·023	0·024
45	72	555	39	27	7·48	7·52	4·058	0·963	0·016	0·010
46	56	435	40	41	7·45	7·45	0·735	0·547	0·042	0·033
47	73	237	39	42	7·37	7·38	3·130	2·287	0·025	0·031
48	51	441	32	37	7·50	7·43	3·370	2·463	0·077	0·056
49	28	395	35	36	7·44	7·44	3·785	3·385	—	—
50	62	305	35	37	7·51	7·48	2·603	2·328	—	—
Mean values	65	433	37	38	7·44	7·44	2·520	1·817	0·043	0·040

delivers a concentration of inspired gas in the region of 90% as measured by direct sampling at the mouth.

Initial observations were made with the patient breathing air. The patient was then connected to the oxygen supply and after one hour the measurements were repeated. During the investigation no drugs were required by any of the patients. After the period of study they remained in the coronary care unit for 48 hours and during this time oxygen administration was continued.

The results were treated statistically, Student's paired *t* test and χ^2 test being used where applicable (Fisher, 1954).

Results

The detailed haemodynamic findings and mean values in the 50 patients while breathing air and oxygen are shown in Table III and the blood gas and metabolic results in Table IV.

Heart Rate and Rhythm.—After the administration of oxygen there was a rise in heart rate in 22 patients, a fall in 21, and no change in 7. In the group as a whole, however, there was no significant change. Seven patients had a sinus bradycardia (60/min. or less) on admission, and in these oxygen caused a further fall in four and a rise in the remainder. Eleven patients had a sinus tachycardia (100/min. or more); after oxygen there was a rise in four, a fall in six, and no change in one. Apart from extrasystoles no arrhythmia developed during the period of study.

Arterial Blood Pressure.—In 38 patients oxygen caused a rise in mean arterial blood pressure, in three there was no change, and in nine a fall occurred. The average value for the group was 84 mm. Hg before oxygen, rising to 91 mm. Hg after treatment ($P<0.001$). The average rise in systolic pressure was 7 mm. Hg and in diastolic pressure 4 mm. Hg. Ten patients had a systolic pressure of 81–100 mm. Hg initially, and in all but one of these the pressure increased after oxygen. A further five patients had a systolic pressure of 80 mm. Hg or less; in three of these a rise occurred while in two there was a fall.

Cardiac Output.—The mean value for cardiac output while breathing air was 5.21 litres/min., and after inhalation of oxygen it fell by 0.46 l./min. to 4.75 l./min. ($P<0.001$). In 37 patients the initial cardiac output was greater than 4 l./min., and in 32 of these a fall occurred after oxygen; in 13 patients the cardiac output was less than 4 l./min., and in this group only five fell. The difference in response of these two groups was statistically significant ($P<0.001$).

Stroke Volume.—With oxygen the stroke volume fell in 34 patients, rose in 11, and was unchanged in 4. The mean fall of 5 ml. per beat was highly significant ($P<0.001$). In 32 patients the initial stroke volume was greater than 60 ml.; in 13 it was between 40 and 60 ml., and in 5 it was less than 40 ml. Of this last group three patients showed a rise, one a fall, and one no change in stroke volume after oxygen.

Systemic Vascular Resistance.—After oxygen the systemic vascular resistance rose in 40 patients and fell in 10. For the whole group there was a mean rise of 186 dynes sec. $cm.^{-5}$, which was highly significant ($P<0.001$). A raised systemic vascular resistance (1,600 or more dynes sec. $cm.^{-5}$) was present in 13 patients breathing air and in 26 after breathing oxygen. On the other hand, 12 patients had a low systemic vascular resistance initially (less than 1,000 dynes sec. $cm.^{-5}$) but only

four after oxygen. In the group of 13 patients with a cardiac output of less than 4 l./min. a high systemic vascular resistance was present in nine, but only two of these showed a further rise with oxygen. In the other four patients of this group, who had a normal systemic vascular resistance initially, a rise occurred with oxygen.

Left Ventricular Work.—While there were some changes in the left ventricular work after oxygen in individual patients, the mean figures showed a slight fall which was not statistically significant.

Arterial Blood Gases and pH.—The arterial blood oxygen tension was reduced in 38 patients, the mean value being 65.4 mm. Hg. After oxygen the mean value increased to 433 mm. Hg, a substantial rise being obtained even in those patients in whom the initial tension was very low. The arterial carbon dioxide tension was within normal limits in most patients and showed no significant change after oxygen. The mean values for *p*H were the same before and after oxygen.

Arterial Blood Lactate.—The arterial blood lactate before and after oxygen was measured in 37 patients. The level was raised in 22 patients (normal range 0.999–1.776 mM/l.) and after oxygen it fell in 21 of these, though in only eight did it return to normal. The fall in the mean value was significant ($P<0.001$). While the highest levels were obtained in those patients with a very low cardiac output and arterial oxygen tension, raised values were also found in many patients who did not have marked circulatory disturbance.

Arterial Blood Pyruvate.—The arterial blood pyruvate levels were measured in 32 patients before and after oxygen. The value was raised (normal range 0.045–0.068 mM/l.) in only four patients while breathing air, and there was no significant change after oxygen.

Mortality.—Eight patients died during the period of admission to hospital, but only two deaths (Cases 14 and 35) occurred in the first 48 hours while oxygen was being given continuously. The numbers involved are, of course, too small to allow any conclusion to be reached concerning the effect of oxygen on mortality.

Discussion

The present investigation has confirmed that in patients with myocardial infarction inhalation of oxygen in high concentration causes a small but significant drop in cardiac output and rise in arterial pressure. Since there is no alteration in heart rate the reduction in cardiac output is due to a lowered stroke volume. While the factors responsible for these changes are not fully understood, perfusion experiments on isolated segments of dog arterioles have shown that oxygen has a vasoconstrictor effect (Carrier and Guyton, 1963) and narrowing of the retinal vessels during oxygen breathing in man has been observed by Dollery *et al.* (1964). It therefore seems likely that the haemodynamic effects of oxygen are secondary to arterial vasoconstriction, and this concept is supported by the rise in systemic vascular resistance noted in our patients. Administration of high concentration of oxygen to healthy subjects is also associated with a reduction of cardiac output and a rise in systemic vascular resistance. In contrast to the findings in myocardial infarction, however, there is a significant fall in the heart rate which is probably mediated through the vagus with little or no change in stroke volume or arterial blood pressure (Daly and Bondurant, 1962 ; Whalen *et al.*, 1965 ; Murdoch *et al.*, 1968). Thus the change in cardiac output in healthy subjects is largely rate-dependent. The reason for this different response is not clear.

The therapeutic effects of these changes in patients with myocardial infarction are difficult to assess. For example, the reduction of cardiac output which is the usual response to oxygen might be undesirable and a decrease in coronary flow would be particularly disadvantageous. It is known, however, that in the presence of myocardial ischaemia the coronary arteries are maximally dilated and blood flow is largely pressure-dependent (Gorlin *et al.*, 1959). Despite the fall in cardiac output, therefore, the accompanying rise in arterial pressure will result in improved coronary perfusion, and this is achieved without any significant increase in left ventricular work. On the other hand, in many of those patients with initially very low cardiac outputs oxygen caused a rise which could well be beneficial.

In addition to the local anoxia at the site of the infarction generalized arterial hypoxia is often present, even in patients in whom cardiac output and blood pressure are well maintained. With regard to this latter group the present findings confirm previous reports that correction of the hypoxaemia is usually accomplished readily (MacKenzie *et al.*, 1964). Moreover, with the methods of oxygen administration used in this study it has been possible to raise the arterial oxygen tension considerably in excess of normal values even in those patients with very low initial levels. However, the availability and amount of oxygen delivered to the tissue obviously depends not only on the arterial oxygen tension but also on the blood flow. Direct measurement of blood flow to individual parts of the body is difficult in severely ill patients, but indirect information regarding the adequacy of perfusion of the tissues can be obtained by study of the products of anaerobic metabolism. In any such investigation it is essential that simultaneous measurements of lactate and pyruvate levels should be made (Huckabee, 1958).

In this series a raised arterial blood lactate was a surprisingly common finding in patients with acute myocardial infarction, though the pyruvate level remained normal. While very high lactate levels were found in association with the syndrome of hypotension, hypoxia, and low cardiac output, raised values were often present in patients with less pronounced circulatory disturbances. It seems likely, therefore, that tissue hypoxia is present in many patients though it may not be clinically apparent. Inhalation of high concentrations of oxygen caused a reduction of arterial lactate levels in most cases, indicating that improved tissue oxygenation had occurred despite the reduction of cardiac output.

There are no clear indications whether oxygen should be used routinely in the treatment of myocardial infarction. At the present time there is little available evidence from clinical trials of its effects on mortality (Cameron *et al.*, 1965). It has been found in the present work that the blood lactate is often raised and the arterial oxygen tension reduced in acute myocardial infarction. Since these changes are indicative of tissue hypoxia, and can be at least partly corrected by the administration of high concentrations of oxygen, there may well be grounds for advocating this treatment in all patients, irrespective of the clinical severity of the illness.

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Late Ventricular Dysrhythmias after Myocardial Infarction

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Summary: Serious ventricular dysrhythmias occurred in hospital after discharge from a coronary intensive care unit in 11 out of 142 patients with myocardial infarction. Previous rhythm changes, hypotension, and left ventricular failure were common findings; only one of these patients had an uneventful previous course. Four patients were resuscitated and left hospital; six were resuscitated but died at varying periods up to eight days after the event; one patient could not be resuscitated. Recent coronary occlusion or further myocardial infarction was demonstrated in 7 of these 11 patients and presumably accounted for the dysrhythmia.

Introduction

Serious ventricular dysrhythmias may still occur after the acute phase of cardiac infarction has passed, and their cause remains undecided. These dysrhythmias could be due to persistent electrical instability of the heart muscle, or be the result of a further ischaemic episode. As this problem has obvious therapeutic consequences, an analysis was made of ventricular dysrhythmias both in and after discharge from the Coronary Care Unit of St. Mary's Hospital.

Electrocardiographic monitoring of patients has shown an incidence of 70–90% of cardiac dysrhythmias in the first 48 hours after acute myocardial infarction (Julian *et al.*, 1964; Killip and Kimball, 1967; MacMillan *et al.*, 1967). The increasing use of coronary care units has facilitated the recognition and correction of otherwise fatal dysrhythmias and also their prevention (Spracklen *et al.*, 1968). In the absence of recurring or continuing complications it is unusual for patients to be kept in one of these units for more than six days (Day, 1965; Yu *et al.*, 1965; MacMillan *et al.*, 1967; Restieaux *et al.*, 1967; Pentecost and Mayne, 1968; Thomas *et al.*, 1968).

Patients and Results

A total of 216 patients with suspected myocardial infarction were admitted to the coronary intensive care unit between October 1966 and April 1968. Of these, 163 were subsequently shown by electrocardiographic and enzyme studies to have suffered a myocardial infarction. In the absence of significant ventricular dysrhythmias, hypotension, or cardiac failure, patients were transferred to a general ward after five days. Ventricular tachycardia occurred in 11 patients and cardiac arrests due to ventricular fibrillation or asystole in 27 (16.6%) while in the coronary unit. Of these 27 patients, 8 could not be resuscitated, 13 were resuscitated but died later in the unit, and 6 were resuscitated successfully and discharged from the unit and the hospital. Resuscitation in this early phase was most successful in patients with a ventricular dysrhythmia occurring within 24 hours of the onset of infarction: four out of nine such cases survived to leave hospital. A total of 142 patients were discharged from the unit in a stable rhythm. Subsequent "late" cardiac arrests occurred in nine of these patients (6.3%) while they were still in hospital. This was caused by ventricular fibrillation in eight and by asystole in one. Ventricular tachycardia occurred in a further two patients. These 11 patients were readmitted to the unit and form the basis of the present study.

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Readmissions to Coronary Unit

Relevant details concerning these 11 patients are presented in the accompanying Table. The time of the so-called "late" dysrhythmias varied from 7 to 64 days after initial admission to the coronary unit. It is notable that in only one instance was the patient's previous stay in the unit entirely uneventful. Of these 11 patients the dysrhythmia was precipitated by a pulmonary embolus in one, and in seven there was subsequent evidence of a further ischaemic episode. Of the latter patients necropsy demonstrated the presence of a recent coronary occlusion by a fresh thrombus in three. In four surviving patients there was electrocardiographic and enzyme evidence of further myocardial infarction. In three patients the cause of the terminal dysrhythmia remains undecided. Enzymes were increased in all three, but this by itself may have been due to repeated attempts at resuscitation. Thus in three patients persistent electrical instability is a possible but unproved cause of the ventricular dysrhythmia, whereas in the majority it would appear that a further ischaemic episode was responsible. In only one patient (Case 8) was the dysrhythmia preceded by cardiac pain. Only 2 of these 11 patients were on anticoagulant drugs at the time of the dysrhythmia. Of the 131 patients who left the unit and did not have further complications during the remainder of their hospital stay 34 were on anticoagulant therapy. There were no other late hospital deaths following myocardial infarction in this series.

Non-esterified fatty acid estimations had been determined on 18 occasions in six of these "late dysrhythmic" patients during the first five days of their initial admission to the coronary unit. The first blood sample, however, was usually obtained more than 12 hours after onset of the patient's symptoms. Thus