Nocturnal hypoxaemia after myocardial infarction: association with nocturnal myocardial ischaemia and arrhythmias

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Abstract

Objective—To document the trend in arterial hypoxaemia and electrocardiographic abnormalities on the second to sixth nights after acute myocardial infarction.

Patients—Nineteen consecutive patients with acute myocardial infarction who were monitored continuously during the night (minimum 2300–0700) with a Holter tape recorder and a pulse oximeter. Fifteen patients were monitored for five nights, one patient for four nights, one patient for three nights, and two patients for two nights.

Results—Five patients had >30 episodic oxygen desaturations of $\geq 5\%$ during the nights of monitoring and many patients had episodes with oxygen desaturations to $<80\%$ ranging from 46% to 61% (from 7/15 to 11/18 patients) during the nights of monitoring. Constant hypoxaemia was found in 11–13% (2/15) of the patients. Simultaneous episodic hypoxaemia and episodic tachycardia was seen in 9/17 (52%) patients on the second night, 11/18 (61%) on the third, 7/15 (46%) on the fourth, 8/15 (53%) on the fifth, and 5/15 (33%) on the sixth night. Simultaneous episodic hypoxaemia and ST deviation was seen in 5/17 (29%) patients on the second night, 3/18 (16%) on the third, 4/15 (26%) on the fourth, in no patients on the fifth, and in 3/5 (20%) on the sixth night. Simultaneous occurrence of episodic hypoxaemia and arrhythmias (supraventricular, ventricular ectopy, and atrioventricular blockade) was seen in 5/17 (29%) on the second night, 4/18 (22%) on the third, 4/15 (26%) on the fourth, 2/15 (14%) on the fifth, and in no patients on the sixth night. Overall, simultaneous occurrence of episodic hypoxaemia and electrocardiographic abnormalities (episodic tachycardia, ST deviations, and arrhythmias) was seen in 11/17 patients (64%) on the second night, 13/18 (72%) on the third, 10/15 (66%) on the fourth, 8/15 (53%) on the fifth, and 7/15 (46%) on the sixth night. One patient who died of cardiogenic shock had simultaneously occurring episodic hypoxaemia and non-sustained ventricular fibrillation on the night before she died.

Conclusion—Episodic and constant hypoxaemia are common during the first week after acute myocardial infarction. Episodic hypoxaemia was associated with

electrocardiographic abnormalities in most patients. Thus, episodic nocturnal hypoxaemia may be particularly detrimental to the infarcted myocardium in the early phase after infarction; special attention should therefore be directed towards oxygenation in this group of patients.

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Despite improved treatment, mortality from acute myocardial infarction in hospital is still as high as 8–27%.¹ Apnoeic episodes frequently occur in patients with congestive heart failure,^{2,3} chronic obstructive lung disease,^{4,5} and acute myocardial infarction,^{6,7} and they may cause episodic hypoxaemia and provoke arrhythmias and ischaemia.^{2,8,9} We aimed to document the occurrence of nocturnal hypoxaemia in the subacute phase after myocardial infarction—that is, the second to sixth nights after infarction. Furthermore, we expected myocardium to be susceptible to hypoxaemia and we wanted to document a possible association between electrocardiographic abnormalities and nocturnal hypoxaemia during this time.

Patients and methods

We studied 19 consecutive patients admitted to our coronary care unit with myocardial infarction (table 1). Exclusion criteria were factors predisposing to hypoxaemia: lung disease requiring treatment, excessive obesity, scintigraphically detected pulmonary embolism, and symptomatic or objective signs of congestive heart failure.

All patients were monitored with a Holter

Table 1 Clinical and demographic data on 19 patients with acute myocardial infarction. Results are numbers of patients unless stated otherwise

Characteristic	
Median age (range) (years)	67 (36–91)
No of men/women	16/3
Median No of days in hospital (range)	12 (4–28)
No of smokers	11
Infarction site:	
Anterior	9
Inferior	8
Septal	2
Maximum LDH (U/l):	
<500	5
500–1500	8
1500–2500	6
Thrombolytic treatment	13

LDH = lactate dehydrogenase.

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tape recorder and pulse oximetry. Patients were monitored for a total of five nights from the second to the sixth night after myocardial infarction; thus the acute phase was over. All nights were not of the same duration, but all patients completed eight hours of continuous monitoring on all study nights (2300–0700), and data from these eight hours were analysed.

PULSE OXIMETRY

Oxygen saturation was measured with a Nellcor N-200 (software version 2.7) pulse oximeter using an adhesive finger probe,¹⁰ with subsequent data printout on a chart recorder the following morning. The mean oxygen saturation during the night was calculated by estimating average values every 15 minutes throughout the night and taking an average of all the periods.¹¹ Episodic hypoxaemia was defined as a decrease in oxygen saturation of 5% or more occurring within two minutes.¹¹ These desaturations were classified into those with a lowest point equal to or greater than 80% oxygen saturation and those with a lowest point below 80%. Minimum oxygen saturation was defined as the lowest single value for oxygen saturation obtained during the study period. Constant hypoxaemia was defined as a mean oxygen saturation of <90% during the night in question.¹¹

HOLTER MONITORING

Holter monitoring was performed with an amplitude modulated, two channel tape recorder, either model ICR 7200 (Instruments for Cardiac Research, Liverpool, New York) or SpaceLabs model 90205 (SpaceLabs, Redmond, Virginia), with primary signal verification using a monitor (Quadriscope 8034, S and W Medico Teknik A/S, Copenhagen). The frequency response was 0.05 to 100 Hz (–3 dB) and the tape speed 1 mm/s. Two separate bipolar leads were recorded: CM₅ from electrodes placed in the second right intercostal space close to the sternal bone and in the V₅ position, and a modified orthogonal y lead from electrodes just to the left of the spinal column, one on the superior angle of the scapula and one above the iliac crest.¹² The recordings were analysed semiautomatically with a computerised equipment (Pathfinder-3 professional (Reynolds Medical, Hertford)) or an Arrhythmiamaster 90101 (SpaceLabs, Redmond, Virginia). All arrhythmic events detected during the automatic analyses were checked on a full disclosure report from both leads. Results of the ST segment analyses were presented in one-minute graphs from each lead. All episodes of ST segment deviation were verified by at least three strips from each (beginning, maximum, and end of deviation).

CORRELATION BETWEEN ELECTROCARDIOGRAPHIC ABNORMALITIES AND ARTERIAL HYPOXAEMIA

Precise information on simultaneous changes in myocardial arrhythmias or ischaemia and

oxygen desaturation was not possible because the recordings were made on two separate systems (pulse oximeter and Holter tape recorder). Synchronous time markers were placed at the start and end of each recording. Correlations between electrocardiographic abnormalities and oxygen desaturation were defined to exist if the electrocardiographic changes occurred within two minutes on either side of the oxygen desaturation.

ETHICS

The study was in accordance with the Helsinki declaration II and was approved by the local ethics committee. Patients were included if they gave their informed consent.

STATISTICS

Spearman's rank correlation test and Friedman's two way analysis by ranks were used for statistical analyses. Level of significance was chosen at $P < 0.05$.

Results

Tables 2, 3, and 4 summarise the results.

Nineteen patients were included in the study. Eighteen patients were monitored with pulse oximetry on the second night after infarction, 19 on the third night, 17 on the fourth night, 15 on the fifth night, and 16 on the sixth night. Eighteen patients were monitored with a Holter tape recorder on the second and third nights, 16 on the fourth and fifth nights, and 15 on the sixth night. The full monitoring protocol was not completed in one case because the patient died after the third nights, in two cases because the patients did not wish to participate any further, and in the remaining four cases because the patients did not complete a full eight hour monitoring of either pulse oximetry or Holter monitoring and were therefore excluded from the analyses.

PULSE OXIMETRY

Many patients seemed to have the sleep apnoea syndrome (>30 episodic oxygen desaturations of $\geq 5\%$ during the night), the incidence ranging from 6–24% (1/18 patient's to 4/17 patients) and being lowest on the second night (table 2). Many patients had episodes with oxygen desaturations < 80%, ranging from seven out of 15 (46%) to 11 out of 18 (61%) during the night of monitoring, with a median of 0–3 (range 0–41) episodes.

Two patients (cases 7 and 10) had constant hypoxaemia during the third to sixth and second to fourth nights, the patient in case 7 having a sinusoidal pattern during the whole night that is often seen in patients with Cheyne-Stokes respiration. Three patients (cases 9, 15, and 16) received supplemental oxygen as 100% oxygen through nasal cannulae. One patient (case 9) received 5 l/min on the second night, 2 l/min on the third night, and 2 l/min intermittently on the fourth night. One patient (case 15) received oxygen at the rate 2 l/min from midnight on the fifth night, when he had an infarction, and at the

Table 2 Electrocardiographic abnormalities and hypoxaemic events in individual patients. Numbers before colons indicate night of monitoring

Case No	No of nights with monitoring	Electrocardiographic abnormalities	Hypoxaemic episodes
1	Holter 5 Oximeter 5	VPC index 2:2, 3:18, 4:20, 5:5, 6:10 ST depression 2*, 3*, 4*, 5, 6 Episodic tachycardia 3*, 4*, 6* Non-sustained VT (few s) 2* SVPCs 3*, 4*	Episodic hypoxaemia with end point ≥80% 2:6, 3:21, 4:10, 5:13, 6:18 <80% 2:15, 3:0, 4:17, 5:0, 6:0
2	Holter 5 Oximeter 5	VPC index 2:6, 3:7, 4:5, 5:6, 6:4 Episodic tachycardia 2* SVPCs 3*	Episodic hypoxaemia with end point ≥80% 2:4, 3:2, 4:1, 5:2, 6:5 <80% 2:1, 3:10, 4:17, 5:0, 6:1
3	Holter 5 Oximeter 5	VPC index 2:4, 3:0, 4:0, 5:0, 6:3 ST depression 2, 3	Episodic hypoxaemia with end point ≥80% 2:0, 3:1, 4:1, 5:6, 6:4 <80% 2:0, 3:0, 4:0, 5:0, 6:1
4	Holter 5 Oximeter 5	VPC index 2:71, 3:68, 4:53, 5:60, 6:40 Episodic tachycardia 2*, 3*, 4*, 5* Non-sustained SVT 2*, 3*	Episodic hypoxaemia with end point ≥80% 2:3, 3:2, 4:31, 5:5, 6:5 <80% 2:4, 3:0, 4:4, 5:3, 6:3
5	Holter 5 Oximeter 5	VPC index 2:1, 3:7, 4:10, 5:200, 6:25 ST depression 3, 4*, 5, 6 Episodic tachycardia* 2, 3, 5 Non-sustained VT 5*	Episodic hypoxaemia with end point ≥80% 2:7, 3:21, 4:34, 5:18, 6:30 <80% 2:5, 3:4, 4:0, 5:2, 6:0
6	Holter 5 Oximeter 5	VPC index 2:100, 3:146, 4:148, 5:116, 6:141 ST depression 2*, 3*, 6 Episodic tachycardia 2*, 3*, 4*, 5*, 6* Triplet VPCs 4*, 5* Isolated episodes	Episodic hypoxaemia with end point ≥80% 2:21, 3:30, 4:5, 5:27, 6:25 <80% 2:0, 3:5, 4:2, 5:0, 6:0
7	Holter 5 Oximeter 5 (nights 3–6)	VPC index 2:4, 3:2, 4:1, 5:0, 6:2 ST depression 6* Episodic tachycardia 3*, 4*, 5*	Episodic hypoxaemia with end point ≥80% 3:320, 4:250, 5:330, 6:290 <80% 3:163, 4:0, 5:4, 6:5 Constant hypoxaemia 3, 4, 5, 6
8	Holter 5 Oximeter 5	VPC index 2:8, 3:3, 4:4, 5:0, 6:3 ST depression 2, 3, 4, 6 Episodic tachycardia 3*	Episodic hypoxaemia with end point ≥80% 2:1, 3:2, 4:6, 5:25, 6:8 <80% 2:0, 3:1, 4:0, 5:0, 6:0
9	Holter 5 Oximeter 5	VPC index 2:0, 3:2, 4:0, 5:1, 6:0 Episodic tachycardia 3*, 5* Triplet VPCs 2* AV blockade 3*	Episodic hypoxaemia with end point ≥80% 2:0, 3:18, 4:18, 5:2, 6:19 <80% 2:0, 3:0, 4:0, 5:0, 6:0 Supplemental oxygen 2, 3, 4
10	Holter 5 Oximeter 5	VPC index 2:2, 3:0, 4:0, 5:0, 6:0 ST depression 2*, 3*, 4*, 5, 6 Episodic tachycardia 6*	Episodic hypoxaemia with end point ≥80% 2:7, 3:4, 4:4, 5:6, 6:9 <80% 2:0, 3:0, 4:0, 5:0, 6:0 Constant hypoxaemia 2, 3, 4
11	Holter 3 (nights 2–4) Oximeter 5	VPC index 2:7, 3:4, 4:10 Episodic tachycardia 2*, 3*	Episodic hypoxaemia with end point ≥80% 2:10, 3:9, 4:10, 5:1, 6:4 <80% 2:10, 3:0, 4:0, 5:0, 6:15
12	Holter 4 (nights 2, 3, 5, 6) Oximeter 5	VPC index 2:2, 3:0, 5:0, 6:1 ST depression 2 Episodic tachycardia 3*	Episodic hypoxaemia with end point ≥80% 2:10, 3:9, 4:10, 5:1, 6:4 <80% 2:10, 3:0, 4:0, 5:0, 6:1
13	Holter 5 Oximeter 5	VPC index 2:0, 3:1, 4:0, 5:0, 6:0 ST depression 2–5 Episodic tachycardia 4*, 5* SVPC 4*	Episodic hypoxaemia with end point ≥80% 2:1, 3:4, 4:19, 5:22, 6:25 <80% 2:10, 3:0, 4:0, 5:1, 6:18
14	Holter 5 Oximeter 5	VPC index 2:0, 3:0, 4:0, 5:0, 6:0 ST depression 2*, 3, 4*, 5, 6* Episodic tachycardia 2*, 3*, 5*, 6*	Episodic hypoxaemia with end point ≥80% 2:62, 3:57, 4:52, 5:52, 6:62 <80% 2:17, 3:41, 4:5, 5:10, 6:17
15	Holter 5 Oximeter 5	VPC index 2:420, 3:360, 4:200, 5:48, 6:4 ST depression 5, 6* Episodic tachycardia 2*, 4*, 5*, 6* Bigemini 2* SVPC 4* Torsade de pointes, reinfarction 5	Episodic hypoxaemia with end point ≥80% 2:4, 3:10, 4:18, 5:13, 6:10 <80% 2:3, 3:30, 4:20, 5:8, 6:0 Supplemental oxygen 5, 6
16	Holter 5 Oximeter 5	VPC index 2:4, 3:60, 4:30, 5:64, 6:0 ST depression 2–6 Episodic tachycardia 2*, 4*	Episodic hypoxaemia with end point ≥80% 2:10, 3:18, 4:29, 5:15, 6:20 <80% 2:0, 3:1, 4:1, 5:7, 6:6 Supplemental oxygen 2
17	Holter 2 (nights 2–3) Oximeter 3 (nights 2–4)	VPC index 2:0, 3:2 ST depression 2*, 3 Episodic tachycardia 2, 3	Episodic hypoxaemia with end point ≥80% 2:25, 3:26, 4:2 <80% 2:2, 3:10, 4:0
18	Holter 1 (night 3) Oximeter 2 (nights 2–3)	VPC index 3:755 Episodic tachycardia 3* Non-sustained VT 30 s 3*	Episodic hypoxaemia with end point ≥80% 2:5, 3:30 <80% 2:11, 3:9 Died during daytime after night 3
19	Holter 1 (night 2) Oximeter 2 (nights 2–3)	VPC index 2:2	Episodic hypoxaemia with end point ≥80% 2:18, 3:20 <80% 2:2, 3:0

VPC = ventricular premature contraction, VT = ventricular tachycardia, SVT = supraventricular tachycardia, SVPC = supraventricular premature contraction.

*Episodic hypoxaemia and electrocardiographic events occurring within two minutes of each other. ST deviations ≥0.1 mV were defined as significant. Episodic tachycardia was defined as an episodic increase in heart rate of 10% or more from baseline. Episodic oxygen desaturation was defined as a decrease in oxygen saturation of 5% or more occurring within two minutes. Constant hypoxaemia was defined as a mean saturation ≤90% on given night.

rate of 2 l/min on the sixth night. Another patient (case 16) received oxygen at a rate of 2 l/min on the second night. Severe hypoxaemia (oxygen saturation = ≤85%) was seen for shorter lengths of time in three patients on the second night (in case 4 saturation was

80% for 15 minutes, in case 9 saturation was 84% for 40 minutes, and in case 15 saturation was 85% for 60 minutes). On later nights no periods of oxygen saturation of ≤85% were detected that lasted for more than two minutes.

Table 3 Frequency of electrocardiographic abnormalities and hypoxaemic events

	Night of monitoring: No or proportion of patients
Holter recording data	
No of patients monitored	2:18, 3:18, 4:16, 5:16, 6:15
No of patients with:	
ST deviation	2:10/18, 3:10/18, 4:7/16, 5:7/16, 6:9/15
ST deviation*	2:5/18, 3:3/18, 4:4/16, 5:0/16, 6:4/15
Tachycardia*	2:18/18, 3:11/18, 4:7/16, 5:8/16, 6:6/15
Arrhythmias*	1:7/18, 2:4/18, 3:11/16, 4:3/16, 5:0/15
ECG abnormalities (total)*	2:11/18, 3:12/18, 4:9/16, 5:9/16, 6:7/15
Pulse oximetry data	
No of patients monitored	2:18, 3:19, 4:17, 5:15, 6:16
No of patients with constant hypoxaemia	2:2/18, 3:2/19, 4:2/17, 5:2/15, 6:2/16
No of patients with hypoxaemic episodes with end point ≥80%	2:16/18, 3:19/19, 4:17/17, 5:15/15, 6:16/16
No of patients with hypoxaemic episodes with end point <80%	2:11/18, 3:11/19, 4:9/17, 5:7/15, 6:9/16
No of patients with >30 hypoxaemic episodes (regardless of end point)	2:1/18, 3:4/19, 4:4/17, 5:2/16, 6:3/16

ECG = electrocardiographic.
*Episodic hypoxaemia and electrocardiographic events occurring within two minutes of each other.

Table 4 Medians (ranges) of individual mean values from pulse oximetry and electrocardiography on the second to sixth nights after acute myocardial infarction

	Night after infarction				
	2	3	4	5	6
Oxygen saturation (%):					
Mean	94 (90–98)	93 (87–98)	93 (89–97)	94 (89–98)	94 (88–97)
Minimum	79 (45–93)	79 (45–92)	82 (45–90)	81 (47–91)	80 (55–91)
No of hypoxaemic episodes:					
≥80%*	6 (0–62)	18 (1–320)	10 (0–250)	13 (1–330)	19 (4–290)
<80%	3 (0–17)	1 (0–41)	0 (0–20)	0 (0–10)	1 (0–17)
VPC index	4 (0–755)	3 (0–366)	5 (0–200)	1 (0–200)	3 (0–141)
Heart rate:					
Maximum	99 (74–173)	94 (56–120)	91 (69–123)	94 (69–163)	94 (71–151)
Minimum	59 (45–75)	60 (39–80)	59 (46–78)	59 (45–74)	58 (44–75)
Mean	73 (61–107)	72 (59–94)	67 (53–91)	75 (52–88)	73 (54–100)

VPC = No of ventricular premature beats per hour of monitoring.
*P < 0.05 by Friedman test.

HOLTER MONITORING
The results are summarised in tables 2, 3 and 4. The index of the number of ventricular premature contractions per hour was >100 on several nights in three patients (cases 6, 15, and 18). In case 6 the premature contractions commonly coincided with electrocardiographic abnormalities and episodic hypoxaemia on all nights. The patient in case 15 had an index >200 before he had a reinfarction on the fifth night. The index fell to 48 on the fifth night and 4 on the sixth night probably because he received antiarrhythmic treatment for torsades de pointes ventricular tachycardia that developed after his reinfarction.

One and a half hours before thrombolysis, he showed a 0.3 mV ST elevation in an electrocardiogram, but at no time did oxygen saturation fall below 90%. One patient (case 18) had an index of 755 on the night before she died of cardiogenic shock. Electrocardiographic abnormalities did not coincide with episodic hypoxaemia in two cases: one patient (case 3) had an ST depression on the second and third nights and another (case 19) had no electrocardiographic abnormalities on the second night, when he wore the Holter monitor.

HYPOXAEMIA LINK WITH ELECTROCARDIOGRAPHIC ABNORMALITIES
Tables 2, 3 and 4 summarise the results. Simultaneous occurrence of episodic hypox-

Figure 1 Simultaneously occurring hypoxaemia and episodic tachycardia in case 6 on third night after infarction.

aemia and episodic tachycardia (increase in heart rate of more than 10% from baseline) (figure 1) ranged from 33% (5/15) (sixth night) to 61% (11/18) (third night) of the patients. Simultaneous occurrence of episodic hypoxaemia and ST deviation (figure 2) ranged from 0 (fifth night) to 29% (5/17) (second night) of the patients. Simultaneous occurrence of episodic hypoxaemia and arrhythmias (supraventricular, ventricular, or atrioventricular blockade) (figures 3 and 4) was seen in ranging from 0 (sixth night) to 29% (5/17) (second night) of the patients. On the second night we found a significant correlation between maximum heart rate and minimum oxygen saturation ($r_s = -0.54$, $P = 0.03$) and between maximum heart rate and number of oxygen desaturations with a minimum value below 80% ($r_s = 0.14$, $P = 0.004$); we found no significant correlation between maximum heart rate and mean oxygen saturation during the same night ($P = 0.09$). In summary, the simultaneous occurrence of episodic hypoxaemia and electrocardiographic abnormalities (episodic tachycardia, ST deviation, and arrhythmias) ranged from 46% (7/15) (sixth night) to 72% (13/18) (third night) of the patients.

One patient (case 18) died of cardiogenic shock during the day after the third night. She had one episode of non-sustained ventricular tachycardia (22 s) and simultaneously occurring episodic hypoxaemia (oxygen saturation 70% for one minute) on the night before she died (figure 4).

Discussion
We found a high incidence of episodic hypoxaemia and a possible association between episodic hypoxaemia and electrocardiographic abnormalities in most patients with acute myocardial infarction. Previous studies have shown a high incidence of sleep apnoea in patients with myocardial infarction.^{6,7} Several studies have shown coincidence between arrhythmias, ischaemia, and hypoxaemia in patients with left heart failure,^{2,3} with the sleep apnoea syndrome,^{8,9} and after major surgery,¹¹ and these findings have now been confirmed in the sub-

Figure 2 Simultaneously occurring ST depression and hypoxaemia in case 6 on second night and in case 14 on fourth night after infarction.

Figure 3 Simultaneously occurring non-sustained supraventricular tachycardia and hypoxaemia on second and third nights after infarction in case 4.

Figure 4 Simultaneously occurring non-sustained ventricular tachycardia and hypoxaemia in case 18 on third night after infarction. Patient died on following day of cardiogenic shock.

acute stage after acute myocardial infarction. To our knowledge, ours is the first study to show the incidence of constant and episodic nocturnal hypoxaemia in the subacute stage after myocardial infarction and the association between hypoxaemia and electrocardiographic abnormalities by continuous monitoring during the first nights after an infarction.

Some of the episodes of hypoxaemia observed during monitoring by pulse oximetry might be artefacts, the result of the probe moving or being displaced.¹³ A study in patients with sleep apnoea or lung disease showed, however, that more than 99% of the observed episodes of desaturation were reflected in transcutaneous oxygen tensions,¹⁴ suggesting that overnight pulse oximetry gives a reliable estimate of episodic hypoxaemia. Furthermore, in our study the probe was secured with adhesive tape, making displacement and therefore artefacts highly unlikely. Finally, the findings of simultaneously occurring episodic hypoxaemia and electrocardiographic abnormalities when obtained by two different methods of measurement (pulse oximetry and Holter monitoring) verify that episodic hypoxaemia measured by pulse oximetry may represent pathophysiological events rather than artefacts. Nevertheless, the issue of reliability of pulse oximetry after myocardial infarction with suspected high incidence of heart failure needs further study.

Patients with myocardial infarction may have a disturbed sleep pattern with a primary depression in REM sleep followed by an apparent rebound increase in REM sleep until the ninth night after myocardial infarction,¹⁵ with a potential increase in sleep disordered breathing and episodes of apnoea. Sleep apnoea may be central, due to a neurological disturbance with a cessation in respiratory activity, obstructive, due to occlusion of the upper airways, or have mixed causes.¹⁶ The number of apnoea episodes required for the sleep apnoea syndrome to be diagnosed varies from 5 apnoeas per hour^{17,18} to 10 apnoeas per hour.^{19,20} Gould *et al* have suggested that episodes of hypopnoea with arterial oxygen desaturation are equally clinically important, they summed episodes of apnoea and hypopnoea and chose 30 per night as the threshold for a diagnosis of the sleep apnoea syndrome.²¹

As we did not monitor respiratory movements but knew that apnoea in the sleep apnoea syndrome is usually associated with hypoxaemia,²¹ we chose 30 episodes of oxygen desaturation per night as a threshold for suspected apnoea syndrome. We are aware, however, that our patients may well have developed sleep apnoea or episodic hypoxaemia as a result of the infarction with resulting heart failure, instead of having had apnoeas present before having been admitted. Because of the design of the study, however, we have not been able to pursue this hypothesis. According to the definition of 30 desaturations per night as a threshold, we found an incidence of suspected sleep apnoea syndrome varying from 6% on the second night

after infarction to around 20% on the following nights. This suggests a rise in the number of episodic desaturations in the later stage after acute myocardial infarction and a higher incidence of suspected sleep apnoea among patients with myocardial infarction than among controls, in whom the incidence has been reported to be between 4%⁶ and 10–15% in unselected middle aged people.¹⁶ Although our study was small and adjusted for factors leading to sleep apnoea, our finding is consistent with that of Hung *et al* who found sleep apnoea in 36 out of 101 men after myocardial infarction when they used an apnoea index of 5 per hour.⁶ On the first night of monitoring we found a lower incidence of suspected sleep apnoea, which might be explained by the depression in REM sleep after trauma among several of our patients. We found prolonged episodes of oxygen saturation below 85% in three patients (cases 4, 9, and 15) only during the first night of monitoring.

Coincidence between arrhythmias, ischaemia, and hypoxaemia has been found in patients with chronic left heart failure²³ and after operation¹¹ and between arrhythmias and hypoxaemia in patients with myocardial infarction.⁷ None of the patients had sustained life threatening arrhythmias.^{23,7} Hypoxaemia was temporally associated with ischaemia (fig 2) and arrhythmias (figs 3 and 4). The occurrence of arrhythmias after acute myocardial infarction predicts a poorer prognosis²² and experimental studies have suggested that repeated episodes of ischaemia may have a cumulative effect and eventually cause myocardial infarction.²³

We chose nocturnal monitoring because we expected more episodic hypoxaemia during the night. After myocardial infarction sleep is disturbed, with a decrease in REM sleep and a subsequent rebound.¹⁵ Such a sleep disturbance with rebound of REM sleep bears a risk of an increase in apnoeas and episodic hypoxaemia as seen after abdominal surgery.²⁴ Whether episodic hypoxaemia is causally related to arrhythmias and ischaemia, or both (reflecting, for example, brainstem hypoperfusion and autonomic instability) is not known, but the association between electrocardiographic abnormalities and hypoxaemia after acute myocardial infarction is interesting and requires further study.

Treatment of patients with acute myocardial infarction after the acute stage have mostly focused on reducing oxygen demand, but the question of increasing oxygen supply has been more or less neglected. Previous studies of oxygen treatment after acute myocardial infarction have suggested an apparent benefit in reducing the infarct size,²⁵ and an ischaemic injury estimated from the sum of ST elevations was reduced in patients when supplemental oxygen was given.²⁶ In patients with congestive heart failure both supplemental oxygen²⁷ and application of continuous positive airway pressure²⁸ reduced the number of episodic oxygen desaturations, just as in patients with a sleep apnoea syndrome.²⁹

Only a few of our patients received supplemental oxygen, but because the study was not controlled for oxygen treatment we were not able to evaluate the influence of oxygen on arterial oxygenation after infarction. Controlled studies of oxygen treatment and oximetric monitoring of patients after acute myocardial infarction are warranted to rationalise oxygen treatment in such patients.

In conclusion, we found that patients have both episodic and constant hypoxaemia and that this is often associated with electrocardiographic abnormalities on the second to the sixth nights after acute myocardial infarction. Episodic and constant nocturnal hypoxaemia may be detrimental to the infarcted myocardium, and special attention should therefore be directed towards nocturnal hypoxaemia in the early phase after infarction. Further studies are needed to show to what extent routine monitoring and oxygen treatment would be beneficial and to evaluate the possible causal relation between hypoxaemia and electrocardiographic abnormalities.

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