Hypoxaemia and supplemental oxygen therapy in the first 24 hours after myocardial infarction: the role of pulse oximetry

ABSTRACT – *Objective:* To assess the incidence and degree of hypoxaemia in patients with acute myocardial infarction and evaluate the nation-wide perception and usage of oxygen therapy.

Design: Postal survey of all coronary care units (CCU) in England of their use of prescribed oxygen and pulse oximetry. Prospective randomised study of 50 patients presenting within 24 hours of onset of myocardial infarction, half of whom received oxygen therapy. Oxygen saturation (SpO_2) as continuously measured by pulse oximetry, and arrhythmias and ST segment changes were recorded on simultaneous 24-hour ambulatory Holter monitors.

Results: In 53% of UK coronary care units oxygen is not routinely prescribed but in only 3% is a pulse oximeter used to aid management. In patients presenting with acute myocardial infarction the incidence of hypoxaemia (SpO_2 < 90%) was 70% and severe hypoxaemia 35% in those not given oxygen, compared with only 27% and 4% in patients given oxygen therapy. The only patient to receive oxygen on clinical grounds had an oxygen saturation of 71%. Severe hypoxaemia (SpO_2 < 80%) occurred significantly less often (1 and 7 patients, $p < 0.05$) in patients given oxygen. There were no differences in arrhythmias or ST segment changes between groups.

Conclusion: Hypoxaemia occurs frequently in patients in the first 24 hours after acute myocardial infarction. It is effectively and easily treated with supplemental oxygen which can be guided by pulse oximetry. This is rarely done. Measurements of oxygen saturation are therefore justified in all patients to guide oxygen therapy unless there is a decision to give all patients supplemental oxygen: this we believe to be unnecessary.

Arterial desaturation and hypoxaemia following acute myocardial infarction has been documented for years[1] and is worse in patients with heart failure. It can be corrected by increasing the inspired concentration of oxygen – but whether doing so improves prognosis or reduces arrhythmic complications has not been proven. An unsigned editorial in the *British Medical Journal* in 1965[1] suggested a controlled trial of oxygen therapy to answer this question but so far none of any size has been done. The biggest randomised clinical trial which has been performed[2] involved 157 patients with acute myocardial infarction. That trial showed no differences in mortality, arrhythmias or analgesic usage. More recent reviews[3,4] have suggested that there is little evidence of benefit in the immediate period following an infarction.

The major development in the management of acute myocardial infarction in the last 20 years has been the use of thrombolytic drugs. These agents help to reperfuse ischaemic myocardium and reduce infarct size and mortality. This is usually achieved by increasing blood flow, but in other ischaemic settings the same benefit is achieved by increasing oxygenation. Both methods decrease cellular acidosis and ultimately cell death. In the context of acute myocardial infarction, the emphasis in recent years has been on re-establishing coronary blood flow. By contrast, oxygen therapy and the treatment of hypoxaemia is rarely mentioned in the cardiological literature.

We are now in the post-thrombolytic era and cardiologists recognise the importance of reperfusion. It is timely therefore to re-examine the value of full oxygenation of the circulation. A recent editorial on reperfusion injury following an acute myocardial infarction in the *British Medical Journal* by Grech and colleagues[5] stated that it is the restoration of *oxygenated* blood to ischaemic myocardium, which is reperfusion, that halts the process leading to infarction. In support of this, Kelly *et al*[6] have demonstrated that breathing high oxygen concentrations decreased infarct size and increased left ventricular ejection fraction in conjunction with reperfusion therapy in dogs. Samaja and colleagues[7] have shown that the major determinant of the reperfusion injury, that occurs after thrombolysis, is low oxygen supply rather than low coronary flow.

At the time of acute myocardial infarction, apart from thrombolysis, patients are nearly always given narcotic analgesics. Hypoxaemia occurs after opiate analgesia[8] and oxygen therapy reduces this[9,10]. Perioperative hypoxaemia has a temporal relationship with myocardial ischaemia[11,12].

Over twenty years ago, two studies documented a reduction in infarct size in dogs[13], and a decrease in ST segment elevation in infarct patients[14] by oxygen inhalation. Since then, little progress has been made in the evaluation of oxygen therapy in these patients. In 1991 Saito and colleagues[15] demonstrated the occurrence of sleep apnoea after infarction and postulated its deleterious effects on the ischaemic myocardium. This study has been repeated with continuous measurement of arterial oxygen saturation, arrhythmia and ST segment analysis by Galatius-Jenson and colleagues[16] in 1994, but not in the first 24 hours following infarct; this study did

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demonstrate a temporal relationship between hypoxaemia and ST segment depression, indicating ischaemia and arrhythmia.

The aims of our study were to document the occurrence of hypoxaemia in the first 24 hours following an acute myocardial infarction and to assess the nationwide utilisation of supplemental oxygen and pulse oximetry.

Patients and methods

Postal survey

A postal survey was sent to all the coronary care units in England. It was addressed to the nurse in charge and enquired into the use of oxygen supplementation in the first 24 hours after myocardial infarction and the availability and usage of a pulse oximeter on the unit.

Interventional study

The study was approved by the hospital ethics committee and all patients gave informed consent.

Fifty consecutive patients with acute myocardial infarction, admitted to the coronary care unit at the Royal Hallamshire Hospital, participated in the study within six hours of the onset of thrombolytic therapy. Since we only use thrombolytic drugs in patients who present within the first 24 hours of a myocardial infarction, all patients were studied within this time frame. Myocardial infarction was confirmed by a typical history of chest pain and the presence of new electrocardiographic changes of ST elevation, pathological Q waves or new left bundle branch block. In all cases the 'cardiac' enzymes (creatinine phosphokinase, aspartate and alanine transaminases and lactate dehydrogenase) showed a greater than twofold rise.

Patients with central cyanosis, pulmonary disease requiring oxygen independent of the cardiac status or those in whom blood gas estimation showed a $pCO_2 >$ 5.5 kPa, and patients with left ventricular failure requiring inotrope support, were excluded from the trial.

Patients were randomly allocated to one of two groups by means of sealed envelopes. In group 1 patients were given continuous oxygen supplements at 4 litres per minute by face mask and in group 2 they had no supplemental oxygen.

Oxygen could be given by the nursing or medical staff for clinical cyanosis or respiratory distress and this was documented in all cases.

Oxygen saturation

All patients were monitored with a Minolta pulsox-7 oximeter, which has a 12-hour memory and printout and therefore was downloaded and reapplied after this time. Recordings were made at 4-minute intervals. Patients were studied for the first 24 hours following admission to the coronary care unit.

Saturation readings were only considered accurate if the level of saturation was maintained for more than 4 minutes and the pulse rate did not differ from the simultaneous Holter monitoring recording. This reduced isolated artefactual recordings that may occur with finger probe measurement of oxygen saturation, particularly in cases of poor peripheral perfusion or during finger movement. The medical and nursing staff looking after the patient were unaware of the pulse oximeter recordings.

Hypoxaemia was defined as an oxygen saturation below 90% and severe hypoxaemia as an oxygen saturation at or below 80%.

Electrocardiographic recordings

Simultaneous 24-hour electrocardiographic recordings were made with a Reynolds Tracker, four-lead, two-channel recorder. The recorder clock and pulse oximeter clock were synchronised. The 24-hour electrocardiographic recordings were analysed by a Reynolds Professional semi-automatic analyser. Hourly mean maximum and minimum heart rates, ectopic frequency, tachyarrhythmias and bradyarrhythmias were measured. ST segment change of ± 2mm was judged to be significant.

Statistical analysis

Differences between the groups were analysed by the *t* test, analysis of variance (ANOVA) or the χ^2 test where applicable.

Results

Postal survey

Two hundred and seven of 252 questionnaires were returned (82%). Nine units did not answer the question on supplemental oxygen policy or on oxygen prescription. In 105 units (51%) routine oxygen therapy was not used, yet 81 (77%) of these had a pulse oximeter. Only 3% said they measured oxygen saturation in all patients although 14% said they measured it if blood gases were poor. In 93 units (45%) oxygen therapy was routinely given and pulse oximetry was available in 76 (80%) of these. However, oxygen saturation was routinely measured in only 6% and measured in 8% when indicated by poor arterial blood gases (Table 1).

Intervention study

Forty-two patients completed the 24-hour trial period, 22 in group 1 and 20 in group 2. Of the remainder, 1 patient died, 1 patient had a cerebrovascular accident,

Table 1. Use of pulse oximetry and supplemental oxygen in coronary care units in England.*

	No routine oxygen therapy	Routine oxygen therapy
Number	105	93
Pulse oximeter available	81 (77%)	76 (80%)
Measured Spo_2 in all patients	3 (3%)	5 (6%)
Measured Spo_2 if poor ABG	15 (14%)	7 (8%)
Other indications	6 (6%)	5 (6%)

*Nine units out of 207 did not answer this question.
ABG: arterial blood gas analysis.

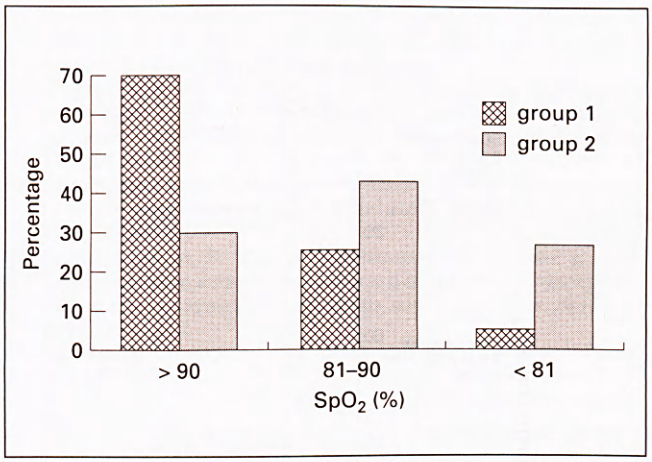

Fig 1. Illustration of oxygen saturation (SpO_2) in three divisions, normoxia, hypoxaemia and severe hypoxaemia, and the percentage of patients from groups 1 and 2 in each division.

4 withdrew consent before the end of the study and 2 had incomplete data collection. There were more men (25) than women (17) in the study. The groups were comparable for the number of smokers (5 and 7, respectively), diabetics (2 and 2) and mean ages (64 and 65 years).

Oxygen saturation

Twenty of the 42 (48%) patients had periods of at least moderate hypoxaemia ($Spo_2 < 90\%$) and 8 (19%) patients had severe hypoxaemia ($Spo_2 \leq 80\%$) (Fig 1). Of the severely hypoxaemic patients, 7 of the 8 (88%) were in group 2 ($\chi^2 = 6.3$, $p < 0.05$), which received no supplemental oxygen, and were clinically undetected in all but one case. The mean lowest Spo_2 level was significantly lower in group 2 (81%) than in Group 1 (85%) (t test, $p < 0.05$) (Table 2). The only patient to receive oxygen on the grounds of clinical hypoxaemia (the presence of central cyanosis) had a saturation of 71%.

There were no significant differences in the prescription of drugs between groups. There was a high incidence of opiate prescriptions in both groups (73% and 90%) with no significant difference in the incidence of hypoxaemia between those who received opiates and those who did not (Table 3). However, in those who received opiates but no oxygen supplementation, hypoxaemia tended to follow shortly after opiate administration, as illustrated in Fig 2.

Table 3. Association of opiate prescription and hypoxaemia.

Opiate prescription	Moderate hypoxaemia	Severe hypoxaemia
Yes	5	7
No	7	1

Electrocardiographic recordings

There were no significant differences between groups in the incidence of arrhythmias (11 in each group) or ST segment changes (3 and 4, respectively) (Table 4), or the type of arrhythmia in each group (Table 5).

Discussion

The majority of coronary care units in England do not measure oxygen saturation despite possessing a pulse oximeter. In about half the units oxygen is routinely

Table 2. Hypoxaemia and opiate use in patients with supplemental oxygen therapy (group 1) and those without (group 2).

Group (number)	Opiates	Hypoxaemia present (%)	Lowest oxygen saturation mean (range)	Severe hypoxaemia present (%)
Both (42)	34	30 (48)	83 (65–89)	8 (18)
1 (22)	16	6 (27)	85 (77–89)	1 (4)
2 (20)	18	14 (70)	81 (65–88)*	7 (35)**

Hypoxaemia = $Spo_2 < 90\%$ and severe hypoxaemia = $Spo_2 < 80\%$.
* t test, $p<0.05$ ** $\chi^2 = 6.3$, $p < 0.05$.

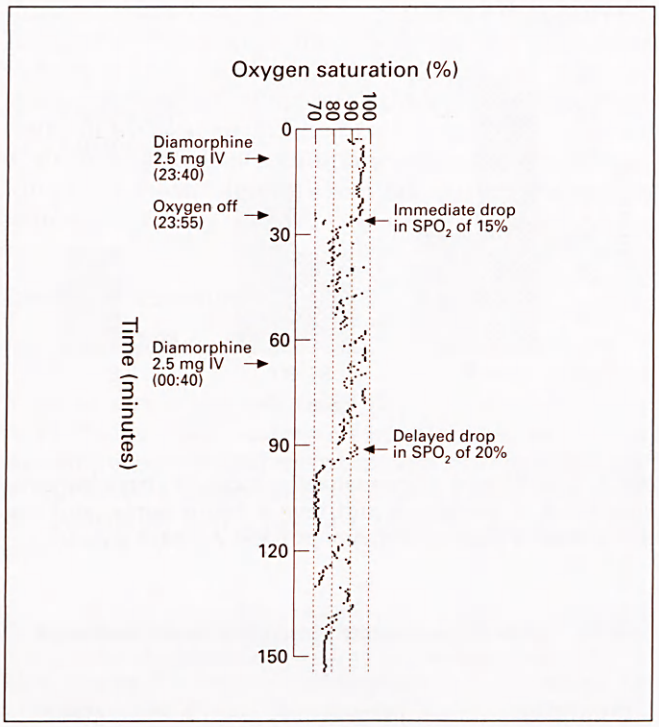

Fig 2. Example of a decrease in oxygen saturation following opiate analgesia with no supplemental oxygen.

prescribed. Hypoxaemia, as measured by pulse oximetry, is common in patients recovering from an acute myocardial infarction (70% of those who did not receive prescribed oxygen). Supplemental oxygen reduced the incidence of severe, and usually unnoticed, hypoxaemia (Spo_2 < 80%) from 35% of patients to just 4%.

It seems there is a reluctance to measure oxygen saturation in patients on coronary care units. We have demonstrated that this is not because clinicians are good at detecting hypoxaemia, nor is it because of lack of availability of a pulse oximeter or that oxygen is used routinely in all patients. Oxygen is often given empirically to ill patients after myocardial infarction, especially if they are breathless, but such patients were excluded from our study. There is general acceptance that thrombolytic revascularisation after infarction is of benefit but few clinicians seem to be concerned that it may be with poorly oxygenated blood. There is a reluctance amongst clinicians and nurses to give oxygen in otherwise stable or comfortable patients because patients do not like oxygen face masks[17]. Not all patients are hypoxic after myocardial infarction so a simple continuous measurement of oxygen saturation would demonstrate non-invasively which of them are hypoxic and therefore require oxygen therapy. Compliance with oxygen therapy could be far higher on coronary care units if the nursing staff were made aware of the patient's hypoxaemia by continuous monitoring.

In conclusion, we have demonstrated that hypoxaemia in the first 24 hours after an acute myocardial infarction is a frequent and predictable occurrence and that this remains undetected by the medical and nursing staff unless a pulse oximeter is used. We found no statistical correlation between hypoxaemic events and adverse cardiac events, but others[16] have previously demonstrated such an association; our study is too small to assess outcome.

We have been waiting since 1965[1] for a sufficiently powered controlled trial of oxygen therapy to demonstrate convincingly whether it is useful in the recovery period after an infarct. Thus far, only underpowered trials have been performed and have not demonstrated clinically important benefits. Until such a trial has been undertaken we should assume that hypoxia is indeed bad for patients after acute myocardial infarction. With pulse oximetry it is now easy to measure hypoxia non-invasively and to correct it with oxygen, which has a proven safety record. We recommend that pulse oximetry be used routinely to guide the use of inhaled supplementary oxygen in patients after acute myocardial infarction.

Table 4. Arrhythmias and ST segment changes in patients with oxygen therapy (group 1) and those without (group 2).

Group	Arrhythmias (%)	ST segment changes (%)
Both (42)	22 (52)	7 (18)
1 (22)	11 (50)	4 (18)
2 (20)	11 (55)	3 (15)

Table 5. Description of arrhythmias in patients with oxygen therapy (group 1) and those without (group 2).

Group	VT + salvo	VT	Salvo	Brady (< 35)	Tachy (< 130)	SVT
1	5	0	2	2	0	2
2	4	1	3	1	2	0

VT: ventricular tachycardia; salvo: run of ventricular beats; brady: bradycardia; tachy: tachycardia; SVT: supraventricular tachycardia.

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