out and he remained worried for about seven minutes after the gas was withdrawn. He then made a rapid and complete recovery, and though he said that he had felt no pain he did not wish to have the gas again. Two patients hyperventilated during inhalation and developed generalized muscle rigidity which, however, did not progress into a tetanic state. They made an uneventful recovery after the administration had finished. It was thought that this was a psychological effect of being asked to inhale from a mask.

Only 13 (6%) patients did not use the apparatus properly. This was due to the fact either that they did not hold the mask tightly enough to their face to obtain an airtight fit (six patients) or that they did not breathe deeply enough to trip the tilting demand valve (seven patients), which requires a negative pressure of -1 cm/H_2O. The latter group were all afraid to breathe deeply because of pain, and might have benefited from Entonox delivered from a continuous flow device in the manner described by Keane.[20] Entonox administered in this way must be supervised by a competent doctor, because the inherent safe principles of self-administration with the demand apparatus are then lost.

A few patients complained of the smell of the black antistatic mask which is provided as standard equipment with the apparatus. They found the optional Perspex mask with the latex rubber cuff much more acceptable.

Conclusion

The results of the trial have convinced us that Entonox is safe in the hands of some non-medical personnel such as State-registered nurses and chartered physiotherapists, provided that they are carefully trained and the principle of self-administration is rigidly adhered to. Obviously the gas is not the panacea for all pains, in all people, at all times, but a large proportion of patients gets substantial relief with almost complete freedom from undesirable side effects in circumstances where hitherto often no analgesia was attempted.

In recent years there has been a great deal of attention paid to patient safety—and rightly so. It is, however, also vital that we should look to our resources in providing patient comfort and, within rigid and stringent safety limits, utilize these resources to the full. The patient's feelings regarding his stay in hospital are often highly coloured by painful incidents, many of which can be simply ameliorated.

We would like to thank our medical, nursing, and physiotherapy colleagues at the Royal Infirmary and Frenchay Hospital, Bristol, for their kind and helpful co-operation during this trial. Thanks are also due to Miss Judy Pile for help in producing the manuscript.

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Therapeutic Conferences

Cardiac Arrhythmias—II

FROM THE DEPARTMENT OF THERAPEUTICS AND PHARMACOLOGY, ABERDEEN UNIVERSITY

British Medical Journal, 1971, **2**, 511–513

PROFESSOR MACGREGOR: Our second patient also had ventricular instability but in his case the problem was somewhat different—he didn't look ill on admission, but he was.

Case 2—Myocardial Infarction and Ventricular Arrhythmia

HOUSE PHYSICIAN: This 63-year-old bricklayer, who had enjoyed excellent health since an inferior infarct seven years ago, had some indigestion, vomited, and noticed pain in both shoulders and arms. On admission he was free of pain, and looked well. However, on examination pulse was found to be very irregular and blood pressure was 140/90 mm Hg—somewhat less than his usual reading of 170/105 mm Hg. He had a gallop rhythm but there was no other evidence of cardiac failure. The E.C.G. showed that he was in sinus rhythm and there were new changes suggestive of anteroseptal infarction. He was having frequent ventricular extrasystoles occurring at times almost on top of the T wave of the previous sinus beat (Fig. 1). While I was setting up the E.C.G. monitor he had a run of fast ventricular tachycardia but this stopped spontaneously before I could do anything about it. However, the ectopics persisted at 10 per minute or more.

PROFESSOR MACGREGOR: What did you do?

HOUSE PHYSICIAN: He was already on oxygen, and, as the basic rhythm was normal sinus at a rate of 80-90 per minute

Appointments of Speakers
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R. A. WOOD, B.SC., M.R.C.P.ED., Lecturer in Therapeutics

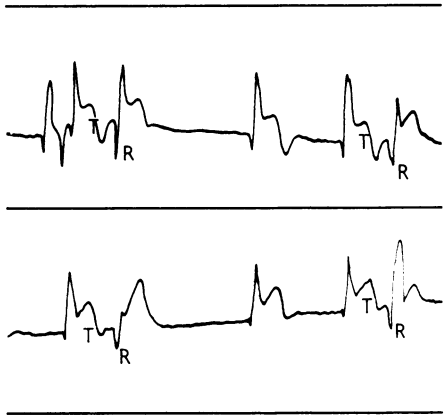

FIG. 1—Anterior infarction. Frequent ventricular extrasystoles.

without heart block, there was no reason to withhold lignocaine. He was given 1 mg/kg of lignocaine intravenously and within two minutes the extrasystoles disappeared. I then put up a drip delivering 1 mg/ml/minute. From time to time the ectopics reappeared during the first two days but on every occasion they were abolished by temporarily increasing the rate of the infusion for a few minutes. On the third day, after 12 hours in which no lignocaine had been given and no extrasystoles had been reported, we stopped intensive monitoring.

PROFESSOR MACGREGOR: Ventricular arrhythmia following infarction occurs in many patients and may pass unnoticed if the patient is not on an E.C.G. monitor. Often little needs to be done but the incidence of ventricular tachycardia and ventricular fibrillation can be greatly reduced if certain types of extrasystoles can be suppressed.

ACTION OF LIGNOCAINE

HOUSE PHYSICIAN: I know that lignocaine is very effective. But how does it act? Is it because it has a local anaesthetic action?

DR. WOOD: This is not certain, but it is probably due to an effect on the area which is being re-excited. Ventricular ectopic beats are an electrical consequence of the normal heart beat that precedes them. Electrical impulses do not travel through infarcted muscle and move only slowly on its periphery. Proximal to the area of blocked conduction the muscle cells have a brief action potential and rapidly regain their excitability—that's to say, in time to be reactivated by the original wave of depolarization reaching the area on a longer course around the infarct. This process can be self-perpetuating, giving rapid ventricular tachycardia. If the discharge from the reactivated area spreads only sluggishly the entire ventricular myocardium can be desynchronized—in other words, ventricular fibrillation. This may happen when the extrasystole falls within the period when the rest of the heart is only partly repolarized —the "R on T" phenomenon on the E.C.G.

Lignocaine renders the myocardium inexcitable until it is fully repolarized and often prevents re-excitation. If re-excitation does occur it sets up a more vigorous wave of depolarization that is much less likely to desynchronize the heart. Even if the extrasystoles remain the risk of a serious outcome is less if lignocaine has been given. Lignocaine usually converts ventricular tachycardia to normal sinus rhythm.

STUDENT: I had always thought that the ventricular extrasystoles were the result of spontaneous pacemaker activity.

DR. PETRIE: The increased pacemaker activity in the ventricle is the reason why the ventricular rate in heart block tends to be higher following an infarction. On the other hand, the faster ventricular tachycardias are usually due to the mechanism Dr. Wood described.

DR. WOOD: The risk of ventricular arrhythmia is greatly increased when the area responsible is easily re-activated—because of potassium ions leaking from the damaged muscle or because of hypoxia or circulating adrenaline.

DR. PETRIE: The important indications for treatment with lignocaine are the "R on T" coupled ectopic, and when the ectopics are self-perpetuating—bursts of ventricular tachycardia. But most physicians usually begin lignocaine when the ectopics are frequent (say, 5-6 per minute), irrespective of situation, or when they originate from more than one area. The drug should also be used to prevent recurrence of the arrhythmia after successful D.C. shock therapy in ventricular tachycardia or ventricular fibrillation. Rarely, when defibrillation fails despite increasing the current applied a further shock may be successful after lignocaine has been infused.

SIDE EFFECTS OF LIGNOCAINE

STUDENT: What are the side effects of lignocaine?

DR. PETRIE: If large doses are infused rapidly convulsions can occur. A few patients notice light-headedness when the ordinary loading dose is given. At up to 2-3 mg/minute no serious haemodynamic effects are seen. When adverse effects do occur they are transient because the drug is rapidly metabolized in the liver.

STUDENT: Hypoxia, adrenaline, and potassium leakage were listed as factors favouring ventricular arrhythmia. Do you treat them?

PROFESSOR MACGREGOR: Certainly we must not forget oxygen. Hypoxaemia can be severe after myocardial infarction and although it is only partially reversible in patients with pulmonary oedema, in whom there is a shunt through the lungs, we use oxygen routinely in the first 48 hours.

HOUSE PHYSICIAN: If adrenaline is important why don't we use β-blocking drugs, which antagonize the effects of adrenaline on the heart?

PROFESSOR MACGREGOR: Theoretically this is the obvious thing but these drugs have disadvantages. They reduce cardiac contractility and many patients can ill afford a drop in cardiac output, or a lengthening of the A-V conduction time. These drugs are antiarrhythmic, but in the case of propranolol this effect partly comes about through local anaesthetic action.

DR. PETRIE: We can cut down the levels of circulating adrenaline to the minimum by keeping the patient comfortable and free of pain. I think we have all noticed how the extrasystoles are more frequent during a ward round: fear is an undesirable emotion and some critics of coronary care units think that the environment produces the arrhythmias. With good nursing this need not be the case. This man did develop radiological pulmonary congestion on the second day and has had frusemide and potassium chloride supplements since then.

DR. WOOD: This brings us to the third of the student's points. It is not possible to stop potassium leaking from damaged cells. However, diuretic-induced hypokalaemia is also arrhythmic and is preventable if the potassium chloride supplements are given.

PROFESSOR MACGREGOR: This patient has done well and has had no recurrence of arrhythmia. He has been in for ten days and I think that we might let him sit out of bed today. We had no trouble controlling the arrhythmia in this man but lignocaine is not always successful and we should discuss other drugs.

OTHER DRUGS

DR. WOOD: Sometimes it is just a matter of increasing the dosage of lignocaine. Many doctors assume that if lignocaine fails procaineamide will fail. This is not necessarily so although both drugs are local anaesthetics. Procaineamide has different effects on the heart and in therapeutic dosage—100 mg/min. intravenously for up to five minutes—is thought to act by reducing conduction of the impulse that initiates the ventricular extrasystole. Procaineamide may be more effective than lignocaine when the interval between the normal beat and the extrasystole is a long one. We saw this in one patient recently (Fig. 2).

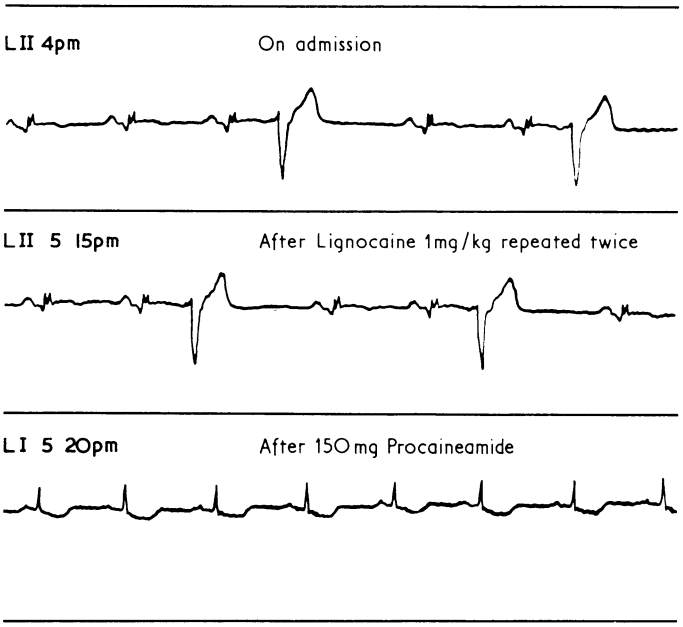

FIG. 2—Acute inferior myocardial infarction.

DR. PETRIE: Many doctors give their patients procaineamide, 250-500 mg six hourly orally for 4-6 weeks, after coming off a lignocaine infusion. This is to prevent patients getting a "late" cardiac arrest.

PROFESSOR MACGREGOR: Procaineamide is much more cardiodepressant than lignocaine and is more likely to provoke heart block or heart failure. I prefer not to use it long term. Most patients recover from their ventricular instability in two or three days but a small minority continue to get extrasystoles, especially when they are tired or excited, for as long as they live. Such patients are liable to attacks of ventricular tachycardia.

DR. WOOD: I believe that they should be given a supply of procaineamide to take—say, 250-500 mg by mouth in an attack.

PROFESSOR MACGREGOR: Lignocaine is a safer drug and in most cases it should be possible for the doctor to make an urgent call to administer it and have any patient with ventricular tachycardia admitted for intensive monitoring.

DR. PETRIE: If lignocaine and procaineamide do not work it is worth looking for some other treatable factor—such as a metabolic acidosis—before further complicating the issue with other drugs. I have known several patients severely ill who did not respond to lignocaine until the base deficit was corrected, using 50-100 ml of 4·2% sodium bicarbonate solution.

DR. WOOD: It is quite wrong to give one drug after another in rapid succession. Various other drugs have been used to treat ventricular extrasystoles—including the anticonvulsant phenytoin, propranolol, the antihistamine antazoline, and, more recently, the obsolete hypotensive drug bretylium. There is no evidence that any of these is safer or more effective than lignocaine.

LIGNOCAINE IN GENERAL PRACTICE

STUDENT: Where do you stand in the controversy over general practitioners giving lignocaine before the patient comes to hospital?

PROFESSOR MACGREGOR: We know how dangerous the first hour or two after an infarct are. The ambulance journey is another critical time. If the pulse was over 70 per minute and extrasystoles were present I would give lignocaine, 50-75 mg intravenously. And, even if there were no extrasystoles, I would give the same dose just before the patient went into the ambulance.

We have been dealing with an area in therapeutics where the patient's welfare is in the hands of the general practitioner and the most junior hospital doctors. The general practitioner must take therapeutic decisions and accept that at times the precise disturbance of rhythm may not be identified. Residents must reach a basic level of proficiency in the E.C.G. interpretation of cardiac rhythm and be able to use the standard drugs intelligently. Too many patients still die unnecessarily from arrhythmias following myocardial infarction.

The participants wish to thank the medical illustration department, University of Aberdeen, for help in the preparation of diagrams.