may precipitate familial gout in these circumstances, may be difficult to assess, particularly in a first attack of gout. It can be argued that the combination of secondary polycythaemia, renal impairment, and diuretic therapy is a favourable one to cause gout, whether the patient is predisposed to familial gout or not.

It is reasonable to suppose that gout occurring secondarily to polycythaemia should be seen especially in the severe forms of disorder treated by cardiac and respiratory departments, into which, moreover, patients are not admitted for gout alone. The total incidence of gout in patients attending the Brompton Hospital is not known, for only the in-patients are coded for disease. It is known that the frequency of gout in the general population is low even though the disease is often unrecognized. In a general-practitioner survey based on consultations the figures were 1.2 per 1,000 for men and 0.4 per 1,000 for women (Logan and Cushion, 1958). Higher incidences may be expected in patients attending a hospital. In a general-hospital series of 200 patients, not admitted for the treatment of gout, there were two with gout (Beckett and Lewis, 1960), and in 200 consecutive in-patients with a haemoglobin level below 120% attending the Brompton Hospital there were also two with gout. Even so, the incidence of four diagnosed cases of gout in the 88 patients with cardiac or respiratory disease (Table I) and a raised haemoglobin concentration appears to be higher than expected by chance.

For a diagnosis of gout secondary to polycythaemia certain criteria can be postulated. The cardiac or respiratory disease which causes the polycythaemia should be long-standing and should antedate the gout. In such cases a family history of gout is not usually obtained. There should be clinical evidence of plethora, with an elevated haematocrit and haemoglobin level, but no abnormality of the white cell or platelet count or splenomegaly to suggest polycythaemia vera. In these patients the arthritis clinically resembles primary gout, but tophi should not always be expected, as they are often an index of long-standing gout. The response of the joint pain to colchicine is not a reliable diagnostic test, since in an appreciable minority the expected benefit is not seen (Dixon, 1960, personal communication). If these criteria are satisfied the diagnosis is likely, especially in congenital cyanotic heart disease where the polycythaemia is most extreme (Whitby and Britton, 1950) but cannot be proved without special studies.

Recognition of the significance of the joint pain in those with heart disease is of importance in distinguishing it from other causes of arthritis such as rheumatic fever, as well as from vascular thromboses and the embolic manifestations of bacterial endocarditis.

Summary

The incidence of gout was investigated in 90 patients who had a haemoglobin level of 120% or more, all but two of whom had cardiac or respiratory disease. There were four patients in whom a diagnosis of gout was made. It was considered that a haemoglobin level of 120% indicated the presence of secondary polycythaemia.

In a group of 200 patients with a cardiac or respiratory disease and a haemoglobin level below 120% there were two patients with gout.

The serum uric acid levels were estimated in 21 of the 90 patients with a haemoglobin level above 120%. The highest values of serum uric acid were seen in patients with congenital cyanotic heart disease.

It is considered that the importance of recognizing the association of gout with cyanotic heart disease is to distinguish it from other forms of arthritis, vascular thromboses, and bacterial endocarditis.

The association of gout in patients with a high haemoglobin level was seen more often with cardiac than respiratory disease.

I thank the consultant staff of the Brompton Hospital for allowing me to study their patients, and Dr. J. F. Goodwin, of the Hammersmith Hospital, for permission to include one of his patients. I wish to acknowledge the technical assistance of Miss J. Gardner in estimating the serum uric acid.

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SUCCESSFUL CARDIAC MASSAGE AFTER MYOCARDIAL INFARCTION IN A CASUALTY DEPARTMENT

BY

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Cardiac massage after cardiac arrest under anaesthesia or during minor operative procedures has been commonly practised for many years. The first successful attempt was by Igelsrud in 1901 (Keen, 1904), and in 1953 Stephenson *et al.* were able to collect 1,200 cases. However, cardiac massage has been much less often attempted for cardiac arrest after myocardial infarction. The first successful attempt was made in 1953 by Reagan *et al.* (1956), and since then fewer than a dozen successes have been reported (Reagan *et al.*, 1956; Beck *et al.*, 1956; Celio, 1956; Mozen *et al.*, 1956; Hannon *et al.*, 1957; Bloomfield and Mannick, 1958; Nickel and Gale, 1959; Walton, 1960). The only one recorded in this country (Walton, 1960) occurred in a ward patient with a known myocardial infarct. In

the case reported here cardiac arrest occurred in a man waiting for attention in the hospital casualty department.

Case Report

A 39-year-old Pole, who worked as a cleaner in a biscuit factory, came to the casualty department of King's College Hospital on February 6, 1960, at 8.15 p.m. At 8.30 p.m. he was found slumped in a chair in the middle of a long queue of patients. He was semiconscious, with obstructed respiration but with a good colour and a regular pulse of poor volume. He was quickly carried into the nearest examination-room. Within a few minutes his respirations became gasping. Nikethamide ("coramine") 5 ml. intravenously was given with no apparent effect. Shortly afterwards his radial pulse disappeared, and within a minute respiration also ceased. He rapidly became deeply cyanosed. At this time his past history was not known, and he had not been questioned or examined prior to his collapse. As it was apparent that respiratory failure had occurred subsequent to cardiovascular failure, a diagnosis was made of cardiac arrest, probably secondary to myocardial infarction. It was then 8.42 p.m.

His clothes were cut away, and, after brief auscultation over the praecordium, the chest was opened by a 14-cm. incision in the fifth left intercostal space. Simultaneously a cuffed endotracheal tube was passed. No bleeding occurred from the chest wall, and the heart was found motionless. Cardiac massage was begun, without opening the pericardium, within three minutes of the disappearance of the radial pulse. At the same time the lungs were inflated with oxygen from Boyle's machine by intermittent positive pressure. Initially the ribs were held apart with a Mason gag, but this proved inadequate, and after a few minutes the fourth rib had to be broken to relieve pressure on the operator's wrist.

The patient's colour improved slowly, although a peripheral pulse could not be felt. At 8.47 p.m. nikethamide 5 ml. was injected into the cavity of the left ventricle. Within a minute slow spontaneous respirations returned. At the same time weak ventricular movement began. During the next 10 minutes the patient's condition continued to improve, but at 8.50 p.m. ventricular fibrillation developed. Cardiac massage, at about 40 compressions a minute, and manual intermittent positive-pressure oxygenation were maintained throughout. The patient remained deeply unconscious, with slightly cyanosed face and limbs, widely dilated pupils, and cold perspiration on his forehead.

An intravenous transfusion of group O rhesus-negative blood was set up, and at 9.10 p.m. an anaesthetist arrived with a cardiac defibrillator. Cloth-covered electrodes, moistened with saline, were applied across the ventricles, and at 9.20 p.m. defibrillation was attempted. A single shock of 150 volts, a double shock of 150 volts, and a single shock of 200 volts were ineffective. At 9.22 p.m. a double shock of 200 volts restored strong regular contractions, 40 minutes after cardiac arrest had originally occurred. Cardiac massage was therefore stopped and at 9.25 p.m. the systolic blood-pressure, previously unrecordable, was 100 mm. Hg; the patient's colour was good, and his radial pulse easily palpable.

Over the next 20 minutes his level of consciousness rose steadily. He became very restless, and 50% nitrous oxide was added to the oxygen. His restlessness continued, however, and was controlled by repeated 50-mg. doses of suxamethonium ("scoline"). After 400 ml. of blood had been given an infusion of 5% glucose in 0.2 N saline was started.

At 9.55 p.m. the systolic blood-pressure was 140 mm. Hg. One ampoule of "crystamycin" (penicillin G 500,000 units and streptomycin 0.5 g.) was emptied into the pleural cavity, atropine 0.6 mg. intravenously was given, and closure of the chest was begun by a surgical registrar. Nitrous oxide, oxygen, and intermittent suxamethonium were used to maintain anaesthesia. After 200 ml. of the glucose-saline solution had been given the infusion was changed to triple-strength plasma to minimize the danger of cerebral oedema.

By 10.17 p.m. the chest had been closed, with an underwater drain through a stab incision in the sixth intercostal space. The systolic blood-pressure was then 150 mm. Hg. The patient was still unconscious, with dilated pupils, generalized spasticity, ankle clonus, and extensor plantar responses. He was also extremely restless, with thrashing uncoordinated movements of his limbs. Pethidine 25 mg. intravenously was given four times in the next 25 minutes, and the patient became adequately sedated. Digoxin 0.75 mg. intravenously was also given.

Up to this point the whole procedure was carried out in a small examination-room in the casualty department. At 11.05 p.m. the patient was transferred to the ward, and half an hour later an electrocardiogram recorded on a cathode-ray oscilloscope showed sinus rhythm with large Q waves over the praecordium and an elevated S-T segment. During the next three hours the patient was retching and extremely restless, and eventually 150 mg. of pethidine, 50 mg. of chlorpromazine, and 10 mg. of perphenazine were given. An elective tracheostomy was performed under local anaesthesia in anticipation of a chest infection, and treatment with crystamycin 1 phial b.d. and penicillin 1 mega unit six-hourly was started.

It was subsequently discovered that the patient had been in King's College Hospital from August 19 to October 6, 1959, with a transmural myocardial infarct in the antero-septal region. Moreover, three days before cardiac arrest occurred he had attended an out-patient clinic complaining of retrosternal pain on exertion for the past week.

The post-operative period was uneventful. Digitalization was maintained, diuretics were given and mephentermine ("mephine") 30 mg. intramuscularly was given hourly to maintain a systolic blood-pressure above 100 mm. Hg. Next day the patient was alert and orientated, and described experiencing a severe crushing pain in his chest the previous afternoon, exactly similar to the pain he had had at the time of his previous infarction, six months before. He remembered coming to the hospital and collecting his casualty card, though not the events that followed. A chest x-ray film taken that day showed congestion of both lungs, greater on the left, and an E.C.G. showed evidence of further transmural infarction (Fig. b).

On February 8 the administration of mephentermine was stopped, the tracheostomy tube was removed, intravenous fluids were stopped, and oral feeding was started. At this time, however, the patient became confused and disorientated, probably as a result of cerebral oedema, and did not recover fully for three days. He was then left with a memory loss, from midday on February 6 to the evening of February 10, which he has never regained. On February 9 the thoracic drain was removed and on February 10 digitalis and diuretics were stopped and anticoagulation with phenindione was started. This has been maintained ever since. On February 15 a chest x-ray film showed that the left lung field had cleared completely, although the diaphragm was still tethered to the chest wall, and on February 19 all antibiotics were stopped. The chest wound healed rapidly with no sign of infection. The blood-pressure remained at about 95/60 mm. Hg for several weeks, but at no time did cardiac failure or arrhythmia develop. The patient was allowed out of bed for the first time on March 16 and discharged to a convalescent home on April 5. A chest x-ray film on March 29 showed that the heart was actually smaller than it had been six months previously and that all adhesions at the base of the left lung had cleared. An E.C.G. taken on the same day (Fig. c) showed an unchanged general pattern with the normal changes of a healing infarct.

A few days before he left hospital the patient's I.Q. was estimated to be 76 on the Wechsler scale, placing him in the "dull and backward" range. Because he was a foreigner with no lifelong acquaintances and an imperfect command of English it was difficult to assess his previous I.Q., but it is unlikely that it was significantly higher. He had never had or sought any skilled job, and his landlord, his friends, and the houseman responsible for his care the previous year

could not detect any change in his intellect or his personality, except that he appeared less worried by his health than in the past.

Discussion

The immediate cause of death in myocardial infarction is usually either cardiac failure or cardiac arrest. The distinction between these two is important (Reid *et al.*, 1958). In cardiac failure death occurs because the myocardium is so extensively damaged that it is no longer capable of providing a sufficiently powerful contraction to maintain an adequate circulation. In cardiac arrest death occurs because the specific conducting tissue of the heart fails, although the myocardium itself may not be severely damaged. This may be due to a gross and histologically demonstrable lesion of the conducting tissue. More commonly,

however, it is due simply to a temporary electrical instability, a disturbance of the normal self-repeating pattern of electrical impulses by local injury currents from damaged cells in the neighbourhood. In such cases, as Beck *et al.* (1956) illustrated so vividly with their analogy of the clock in which the pendulum is jarred, the mechanism is motionless but intact, and only needs to be triggered into activity, to be given a second chance, to function just as before. This is why resuscitation may be possible after cardiac arrest but not after cardiac failure.

The available literature suggests that if cardiac massage is attempted soon after arrest has occurred the chance of successful resuscitation is surprisingly high. The 11 cases of successful resuscitation so far published are accompanied by only three failures, and in each of these there were good reasons why resuscitation was unsuccessful; either the patient's general condition was very poor, oxygenation was inefficient, or other lesions were also present. In any case, in so grave a situation there is everything to gain and nothing to lose in attempting resuscitation.

It has often been demonstrated in animal experiments, and since confirmed clinically by experience of cardiac arrest under anaesthesia, that irreparable brain damage occurs after between three and four minutes of circulatory arrest. If the heart has been slowly failing before arrest occurs this interval will be reduced. Thus resuscitation, to be effective, must be immediate. Though there are reported cases of successful resuscitation by pounding on the chest wall, by pricking the heart with needles, or by intracardiac injections, such cases are rare. We believe that attempted resuscitation by these means is not only unlikely to be successful but is dangerous, for it serves only to delay cardiac massage. Immediate massage is the most effective means of restoring a spontaneous heart-beat because it re-establishes the coronary circulation and oxygenation of the myocardium, and consequently restores the ability of the myocardium to respond to stimulation. It is also the only technique that maintains an adequate cerebral circulation during the period before a spontaneous heart-beat returns.

Reluctance to perform an immediate and unsterile thoracotomy when even the diagnosis of cardiac arrest might be in doubt is understandable. However, the diagnosis is confirmed by failure of the initial skin incision to bleed, and under these circumstances, even if the heart is found still beating, the cerebral and coronary circulations are almost certainly inadequate, and so thoracotomy is completely justified. The dangers of performing a thoracotomy without sterile precautions seem surprisingly slight. In the 11 cases previously mentioned infection was a serious problem in only two, and in only one was it eventually fatal. To delay cardiac massage in the hope that it may be avoided greatly increases the risk of death or severe cerebral damage, and is thus more hazardous than immediate thoracotomy.

Cardiac arrest is a fairly common cause of death in a very common disease. The opportunity for resuscitation must therefore occur frequently in hospital practice, both in the wards and in out-patient and casualty departments. Because of the vital time factor it will generally be a house officer who is presented with this opportunity. We therefore believe it should be the duty of all resident staff to acquaint themselves with the basic principles of cardiac massage. The only apparatus needed is a scalpel, for it has been amply demonstrated that direct mouth-to-mouth respiration can maintain

(a) E.C.G. recorded on September 4, 1959, two weeks after the original myocardial infarct, showing a transmural antero-septal lesion.

(b) E.C.G. recorded on February 7, 1960, the day after cardiac arrest occurred, showing a recent transmural anterior and apical infarct in addition to the previous lesion. Leads 7.V5 and 7.V6 were taken instead of V4-6 because of the thoracotomy dressings.

(c) E.C.G. recorded on March 29, 1960, seven weeks after cardiac arrest occurred, showing a healing transmural anterior and apical infarct. The positive potential R in lead V1 suggests that right ventricular enlargement has taken place.

adequate oxygenation for as long as 15 minutes if Boyle's machine and an endotracheal tube are not immediately available (Mozen *et al.*, 1956; Nickel and Gale, 1959). An emergency thoracotomy is much easier to perform than, say, an emergency tracheostomy, and once cardiac massage has been started and an airway established expert anaesthetic and surgical assistance can be awaited in comparative leisure.

Looking back on this case, we are aware that it was mismanaged in several ways, partly through ignorance and partly through lack of thought at the time. The heart was massaged incorrectly, against the ball of the thumb instead of against the chest wall or thenar eminence; the foot of the couch was not raised to aid cerebral blood flow; procainamide or quinidine should have been administered rather than digitalis; neither blood nor saline should have been given in view of the risk of cerebral oedema; and too large doses of pethidine and phenothiazine derivatives were given in the immediate post-operative period. Yet, in spite of these errors, in spite of a previous transmural infarct, and in spite of 40 minutes of cardiac massage with no detectable pulse, the patient survived, and survived, moreover, without apparent cerebral damage. This would suggest that cardiac massage does not demand ideal circumstances and expert knowledge to be successful, and reinforces our belief that it should become the accepted treatment in hospital practice of cardiac arrest after myocardial infarction in reasonably young and fit patients.

Summary

The case is described of a man who developed cardiac arrest after his second myocardial infarction, and was successfully resuscitated by cardiac massage in a hospital casualty department.

A distinction is drawn between cardiac arrest and cardiac failure, and the reason why resuscitation may be possible after arrest is discussed.

It is suggested that immediate cardiac massage should be the recognized treatment in hospital practice of cardiac arrest after myocardial infarction.

It is stressed that the opportunity for resuscitation after cardiac arrest will normally be presented to junior resident staff and that all house officers should therefore be acquainted with the principles of cardiac massage.

[ADDENDUM.—Ten months later the patient is in good health, with no dyspnoea or angina, and has returned to a light job with his previous employers.]

We are very grateful to Dr. John Anderson for permission to publish this case, to Dr. Clifford Hoyle and Dr. A. Deacock for their encouragement and advice, and to Dr. Leon Resnekov for interpreting the cardiograms.

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THE ORAL GLUCOSE-TOLERANCE TEST IN TROPICAL MALABSORPTION SYNDROME

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Thaysen (1926) showed that when glucose was administered by mouth to patients suffering from idiopathic steatorrhoea the rise in blood-sugar levels was lower than would be expected in normal people. Subsequently the glucose-tolerance test has been widely used as an index of intestinal function in various disorders of the small intestine, and not least in the investigation of tropical sprue and tropical malabsorption syndrome (Serra, 1929; Thaysen and Norgaard, 1929; Fairley and Bromfield, 1932; Fairley, 1936; Frazer, 1950; Gardner, 1956; Baker, 1957).

A maximum rise of 40 mg./100 ml. in the blood sugar in the glucose-tolerance test (G.T.T.) is often taken as the limit of normal, any rise less than this being regarded as abnormally low or "flat." In studying subjects without any evidence of malabsorption we were struck with the frequent occurrence of a "flat" G.T.T. result as defined above, and therefore undertook a comparative study of G.T.T.s in patients with tropical malabsorption syndrome and in normal controls. This report presents the results of this study.

Materials and Methods

Subjects Studied.—Fifty consecutive cases of tropical malabsorption syndrome with steatorrhoea (a fat excretion of more than 5 g. a day on a 50-g. intake) were used for this study. These patients were all Indians, of differing social status, and with symptoms and signs covering the whole range of clinical pictures from the mildly affected almost symptomless cases to those with fully developed classical "sprue" (Baker, 1957). Twenty-four subjects living on a high-carbohydrate diet associated with rice-eating, in good health, and without any evidence of intestinal disease, were used as suitable controls.

Oral Glucose-tolerance Tests.—Subjects fasted for 10 hours before the test. An initial sample of venous blood was collected, and then the subjects drank 50 g. of glucose dissolved in 200 ml. of water. Thereafter blood samples were drawn at 30-minute intervals for a period of two hours. Blood-sugar levels were estimated by the method of Folin and Wu (1920) modified to give true glucose values. The "rise" in the G.T.T. was defined as the highest blood-sugar level recorded in the test (usually at half or one hour) minus the fasting level.

Results

Height of "Rise" in Glucose-tolerance Test.—As a group the patients with malabsorption syndrome have a lower rise of blood sugar above the fasting level than do