RECOVERY FROM NEUROLOGICAL SEQUELAE AFTER CARDIAC ARREST

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Cardiac arrest is a known hazard of surgical operations. Its incidence is difficult to ascertain, since not all the deaths are recognized as due to it. From recent reviews cardiac arrest would seem to occur in between 1:1,000 and 1:5,000 anaesthetics (Milstein, 1956), and in spite of greater recognition its mortality remains high.

The early recognition of cardiac arrest is all-important, for unless the heart can be restarted within three to four minutes irreversible neurological damage may occur (Primrose, 1935; Dark *et al.*, 1952; Leeds, 1953; Louttit, 1954; Milstein, 1956).

The case described below illustrates the remarkable degree of recovery which can take place following such a complication. It also underlines the importance of early recognition of cardiac arrest. The heart was restarted one and a half minutes after the condition was recognized, but the neurological sequelae suggest that the vital time interval may have been longer. In particular, since the arrest occurred just after the injection of neostigmine, the case emphasizes that this procedure should be followed by the utmost vigilance lest cardiac arrest should develop.

Case History

A 39-year-old man was admitted to hospital with a diagnosis of perforated duodenal ulcer of three hours' duration. He gave a long history of previous dyspepsia, was already known to have a duodenal ulcer, and had refused gastrectomy. The perforation was complicated by a moderate haematemesis.

At operation he was found to have a chronic duodenal ulcer on the posterior wall of the first part of the duodenum which had perforated at its postero-superior margin. The only other abnormality noticed was a large smooth fatty liver, enlarged about three fingerbreadths below the costal margin. This was associated with a history of alcoholism. Closure of the perforation proved difficult, and, in view of this, the long ulcer history, and the accompanying haematemesis, it was decided to proceed with partial gastrectomy. This was carried out without difficulty and the abdomen closed, the total operating time being 80 minutes.

Throughout the operation there was no sign of cyanosis, and while closing the wound a bleeding-point requiring ligation produced bright red blood. At the time of suturing the anterior sheath of the rectus muscle atropine, 1/100 gr. (0.65 mg.), was injected intravenously via the blood drip. Following this injection and after the pulse rate had risen to over 100 a minute, neostigmine, 2.5 mg., was injected via the drip. About two minutes later, immediately after final suture of the skin, the anaesthetist reported that the patient's pulse was imperceptible. Oxygenation produced no improvement, and five seconds later the dressing was removed from the abdominal wound and the abdomen rapidly re-entered through the previous incision.

The heart beat could not be detected through the diaphragm, and cardiac massage was immediately begun from the abdomen, the heart being compressed against the sternum. Since the heart beat did not return, 5 ml. of adrenaline 1/1,000 was injected direct into the heart through the fifth left intercostal space some 10 to 15 seconds later, and cardiac massage continued. The heart restarted immediately after the intracardiac injection of adrenaline. Heart beats were initially irregular, but within a second or two powerful regular contractions could be palpated through the diaphragm and the patient recommenced breathing. His general condition appeared excellent, and since approximately only one and a half minutes had elapsed from the time of the initial alarm the prognosis was thought to be good. The abdominal wound was again resutured and the patient returned to the ward.

Six hours later he had not recovered from the anaesthetic; strong pressure over the eyeballs failed to elicit any response, and a state of decerebrate rigidity was present, all four limbs being spastic. Superficial and deep reflexes were absent.

About eight hours after completion of the operation his temperature was 103° F. (39.4° C.), and it was decided to institute cooling. This was accomplished by ice-packs and fans, associated with chlorpromazine hydrochloride medication. The temperature fell to 99° F. (37.2° C.) during the next two to three hours and was maintained at that level by cooling for the next 48 hours.

During the first 24 hours the depth of unconsciousness remained unchanged, all four limbs remaining spastic.

On the second post-operative day the patient was still unconscious, but could be roused by non-painful stimuli, such as shouting. He became very restless and the spasticity in all four limbs had lessened. He was able to swallow water and made incoherent mumbling noises. His temperature had reached the normal range and cooling was discontinued.

By the following day he had become completely conscious and would turn his head towards any sound or voices. He was totally blind, but could speak, though the words were barely recognizable because of extreme dysarthria. Leg-muscle tonus had returned to normal, but both feet gave an extensor response to the Babinski reflex. His arms remained moderately spastic and he had no control of the small muscles of the hand.

During the ensuing week arm spasticity passed off and in the next two months his muscle power returned to normal and no definite abnormality could be found in his motor system. He was able to walk about and to dress and feed himself. At this time peripheral vision had improved, the right eye being 6/36 partly, and left eye 6/24 partly. There was still considerable loss of vision; the pupils were equal and reacted to light, both directly and consensually. Ocular movements appeared to be full in all directions. There was no diplopia or nystagmus. Optokinetic nystagmus was easily elicited, both horizontally and vertically. The right optic disk was pale, particularly in its temporal half; the left optic disk appeared normal, and visual fields showed scotomata. Three months later the optic disks were considered to be normal on ophthalmoscopic examination. Vision in the right eye was 6/36 partly and that of the left 6/24 partly. A distinct reduction in the extent of the central part of the scotomata accompanied a marked subjective improvement in vision.

The patient was able to talk intelligently and no psychosomatic abnormality could be noticed. Apart from his impaired vision he appeared normal in all other respects.

Discussion

This case illustrates the remarkable recovery which may follow neurological damage occurring during cardiac arrest. It indicates that even a state of deep unconsciousness should not be regarded as completely hopeless, and probably the

use of hypothermia is justified in these cases, since it may reduce the metabolic needs of damaged nervous tissue.

As has been recorded, cardiac arrest developed in this patient within two minutes of the injection of neostigmine. The neostigmine had been given following intravenous atropine. In recent years some controversy has developed regarding which of these two drugs is the more responsible for cardiac arrest. Bain and Broadbent (1949) thought atropine partly responsible. They noted that subcutaneous injections of atropine first slowed the heart rate, tachycardia occurring only 15 to 20 minutes later. Pooler (1957) also blamed atropine. It has been shown by Johnstone (1951) that hyperventilation with CO₂ produced electrocardiographic irregularities, and Pooler noticed that intravenous atropine during CO₂ retention produced a wildly irregular pulse.

In our patient intravenous atropine produced no detectable irregularity of the pulse: tachycardia was produced, not bradycardia, and intravenous atropine produced no detectable irregularities of the pulse when given during anaesthesia after muscle relaxants in over 40 other patients.

These observations suggest to us that neostigmine was the most likely cause of cardiac arrest in the present case. This finding is in keeping with the views of many other workers as recorded by Pooler (1957). It emphasizes the need for the utmost vigilance immediately after the administration of neostigmine. The neurological sequelae in this case suggest that some valuable time may have been lost before the alarm was given.

Unfortunately, neostigmine is administered at the end of the operation, when an anaesthetist is often forced to busy himself with the many tasks involved in terminating one anaesthetic and preparing for the next. Some visual or audible method of monitoring the patient's pulse might be of great service both in the operating theatre and in the recovery-room, saving valuable seconds between the cessation of the pulse and the application of adequate cardiac massage.

Only by such methods can delay in recognition be prevented, and this factor may well contribute to the continuing high mortality of cardiac arrest in spite of more adequate surgical management.

Summary

A case of recovery from neurological damage following cardiac arrest is described.

Neostigmine was given two minutes before arrest of the heart, and was thought in this case to be the most responsible agent.

The need for some mechanical device to monitor the pulse during operation is emphasized, so that treatment could be initiated in the shortest possible time following stoppage of the heart.

We thank Professor C. F. W. Illingworth, in whose wards this patient was treated, for guidance in the preparation of this paper, and Dr. M. W. Paterson, of the eye department, Western Infirmary, for superintending the ophthalmological investigations carried out on this patient.

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The *Transactions and Report of the Liverpool Medical Institution* for the year 1957 contains the inaugural address by Dr. MARGARET E. THOMAS, the first lady president of the Institution, who took for her subject "The Artist's Contribution to Medicine." Also included are abstracts of papers read at ordinary meetings.

HAEMORRHAGE DURING PHENINDIONE THERAPY

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Anticoagulants have an accepted place in the immediate treatment of thrombotic diseases. Their value in the prophylaxis of thrombosis has yet to be established. The risk of haemorrhage, and possibly death as a consequence, is a hazard to which increasing numbers of patients are exposed as the use of these drugs and the duration of treatment extend.

Reports of the incidence of haemorrhage attributed to anticoagulant therapy relate mainly to the more extensively used coumarin group of drugs. The frequency of this complication has varied, but it would appear that experience has not succeeded in reducing the risk of haemorrhage during long-term anticoagulant therapy. The incidence of haemorrhage in patients treated with anticoagulants over a period of five years was 36% in Nichol and Borg's (1950) series and 42% in the series of Keyes *et al.* (1956); Peyman (1957), over a period of one year, found a haemorrhagic incidence of 23%. Similarly with short-term anticoagulant therapy, the frequency of haemorrhage has not decreased: Olwin (1949) reported an incidence of 15%, Gilchrist and Tulloch (1954) 11%, and Peyman (1957) 16%.

It is possible that significant haemorrhage during anticoagulant therapy may be forecast by using a simple chemical tests ("occultest") to detect microscopic haematuria (Peyman, 1956). Should significant bleeding occur with anticoagulant therapy, it is essential that adequate means should be available to overcome this complication. Toohey (1953) has discussed the indications for oral and intravenous vitamin K₁ in combating haemorrhage induced by anticoagulants. This paper is concerned with the treatment of haemorrhage and factors which may have influenced the onset of bleeding in a series of patients receiving phenindione ("dindevan").

Method

Patients were treated with twice-daily doses of phenindione, and prothrombin estimations were done each day by Quick's one-stage method, using brain extract as a source of thromboplastin. Phenindione dosage was assessed from the previous response of the patient to the drug, so that the prothrombin time was kept within the desired therapeutic range between two and three times the control prothrombin time. The duration of phenindione treatment varied between individual patients from 3 to 12 weeks, the majority receiving this therapy for a period of three to four weeks. A total of 92 patients were treated with phenindione: myocardial infarction 69, venous thrombosis 3, pulmonary embolism 15, others 5. Six deaths occurred during the period of observation; in one case death was attributed to phenindione. Four died in left ventricular failure and one from mesenteric embolus one week after stopping phenindione.

Major haemorrhage occurred in seven patients (7.6%) (see Table), one of whom died as a direct result of this complication. Minor haemorrhage—for example, microscopic haematuria (Peyman, 1956)—was not sought.