CARDIAC ARREST DURING
SURGERY IN SMALL HOSPITALS
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CARDIAC arrest is a potential hazard of every surgical operation or anaesthetic administration. The actual incidence of this grave emergency is difficult to assess, but recent reviews on the subject would indicate that it occurs once in every 1000 to 1500 anaesthetics.1 The mortality rate is high even in large institutions. Friedberg reports a mortality of 70% in a large series of cases,2 and in small hospitals the mortality rate probably reaches nearly 100%, although it is difficult to obtain accurate figures on this. Nevertheless, it has frequently been pointed out that prompt recognition and immediate institution of a well-planned and co-ordinated program for resuscitation will in many cases result in a favourable outcome. Every hospital large and small whenever an anaesthetic is administered can and should be ready to meet this emergency when it occurs and thus possibly prevent a tragic disaster. All too frequently arrest occurs in young patients in whom some very elective or even minor procedure is being performed. A planned program is simple to set up and requires little in the way of special equipment or knowledge.

There are three main causes of cardiac arrest. These may occur singly or frequently in combination.

MYOCARDIAL ANOXIA

Long periods of inadequate oxygenation or hypoxia with resultant CO₂ build-up are of greatest importance and may cause cardiac arrest during the course of an operation. This occurs usually towards the end of the operation or late during a long procedure. Untreated anaemia with unrecognized blood loss during the course of operation with resultant lowering of blood volume may lead to myocardial anoxia and arrest. Any factor resulting in the decrease of oxygen available to the myocardium will predispose towards cardiac standstill, whether the factor be mechanical and due to faulty administration of O₂ or to a decrease in the total carrying capacity and volume of circulating blood.

MYOCARDIAL DISEASE, TOXICITY
OR SENSITIVITY

This is a second important cause of asystole. A myocardium that is abnormally sensitive or toxic is also less able to tolerate anoxia, and thus a combination of factors may predispose to arrest. Myocardium may be sensitized by an overanxious patient with an increase in epinephrine content of the blood. Hence the importance of adequate pre-operative preparation of the patient. Certain, in fact all, anaesthetic agents may render the myocardium toxic. Recently, trichlorethylene, commonly used by midwives and in obstetrical practice, has been reported as causing cardiac arrest. A case of arrest has been reported following the injection of neostigmine. Accidental administration of a wrong drug or overdosage of a correct one may cause myocardial toxicity and arrest. The importance of correctly labelling all solutions and assuring proper dosage cannot be overemphasized and is illustrated in Case 5 that follows. The possibility of arrest during operation on a patient with a toxic myocardium following an acute disease such as diphtheria must be considered. W. A. Paddon, North West River Hospital, Labrador, in a personal communication reported a fatality due to cardiac arrest in a seven-year-old child undergoing operation for inguinal hernia under open-drop ether anaesthesia. This child had had diphtheria five months previously. The arrest might have been due to residual damage or heart block from the diphtheria, although other causes cannot be excluded.

NEUROGENIC CAUSES

An overdose of anaesthetic agents with a central depressant effect may result in cardiac arrest because of their effect upon the respiratory or cardiac centre. Vaso-vagal reflex caused by postural changes, undue tension on viscera in the abdomen or chest during surgical manipulations, is a frequent cause of hypotension and may even result in standstill. It may occur at any time during the course of an operation.

Whatever the cause, prompt recognition and immediate institution of resuscitative measures is mandatory if a patient is to be saved. Mechanical gadgets and monitors, valuable as they may be, are no substitute for an alert anaesthetist and surgeon. In small hospitals where a nurse may be administering the anaesthetic, the surgeon should be all the more watchful and wary. Constant attention to the colour of the blood, observation of pulsations in major vessels, the rate and depth of respirations, and blanching of the skin, all give important clues to developing anoxia, hypotension, and impending arrest. The anaesthetist should report the state of the patient frequently to the surgeon, and in the event of standstill immediate action should be taken without any delay or debate.

Five minutes is probably a safe maximum that the brain can withstand circulatory cessation and recover. Hence, the treatment must be prompt and definite and the diagnosis must be made within a minute of its occurrence. The most important sign is absence of pulse pressure; as soon as this is noted, measures should be taken to re-start the circulation.
TREATMENT

1. Adequate Ventilation

The patient must be oxygenated, and to do this properly intubation should be carried out at once, if it has not already been done. Hence, a laryngoscope functioning properly with suitable endotracheal tubes should be readily available together with apparatus for administering oxygen under positive pressure. Initially, if time is a factor, a tight-fitting face mask will do until the anaesthetist can carry out intubation, but where possible this should be done immediately.

2. Cardiac Massage

As soon as the diagnosis is made the surgeon should re-start the circulation by massaging the heart; by mechanically pumping the heart with adequate pressure the pulse will become perceptible again. He has three to four minutes to carry this out if the patient is to recover without sequelae. A rate over 60 is ideal, but a rate of 40-60 per minute is sufficient. Without wasting time for sterile drapes or preparation, the chest is opened with a long sweeping left anterolateral incision through the fourth or fifth interspace, and massage immediately started. Massage may be carried out through the diaphragm from below if operating in the abdomen, and it is not necessary at first to open the pericardium, but this is done if the circulation does not promptly re-start. The heart is compressed rhythmically between the thumb and forefingers, expelling a quantity of blood into the great vessels. Massage is carried out if it is contracting weakly or is in ventricular fibrillation, and is continued until the contractions spontaneously become regular and forceful.

3. Drugs

Certain drugs are useful and should be used in proper dosage and sequence. If the heart is in standstill, 5-10 ml. of 10% calcium chloride should be injected into the ventricle. This helps balance the potassium liberated by ischemic tissues. Adrenaline 0.5-1 ml. of 1:1000 is useful in restoring forceful contraction, but should not be used if cyclopropane has been used as an anesthetic. If the cause of the standstill is thought to be a vagovagal reflex, then atropine 1/100 grain should be given directly into the ventricle. Sodium bicarbonate 3-7 g. may be given intravenously if acidosis develops. Procaine amide or hydrochloride (2-10 ml. of 1% solution) will decrease the irritability of the ventricles and prevent ventricular fibrillation. Procaine may be used topically or directly into the ventricle.

4. Treatment of Ventricular Fibrillation

Ventricular fibrillation may develop after the heart action is restored and must be abolished if recovery is to ensue. The first step in its control is the continuation of rhythmical contraction manually to maintain circulation. The fibrillating mass consumes oxygen; therefore, adequate oxygenation is as important as in standstill. If the beat does not readily return to normal rhythm after these measures and the instillation or application of procaine, then an electric defibrillator should be used, giving a shock of 110-220 volts for 0.1-0.2 second. The electrodes may be placed directly on the heart, one over the apex, the other over the right atrium. Usually after a single or a second shock the heart will return to normal rhythm. We use a Mark I explosion-proof defibrillator, and it is set up and ready in the operating room at all times. After defibrillation, acidosis may be treated with intravenous bicarbonate.

5. Treatment of Shock

Shock should be treated and the blood volume restored with whole blood as soon as possible. Initially clamping the aorta for a few minutes at a time will direct more blood to the cerebral circulation. A noradrenaline drip is most useful and may be necessary for some time after the heart returns to normal rhythm.

The chest should be closed only after adequate time for observation has elapsed (at least one-half hour). Hæmostasis should be attended to and bleeding points will now be seen. The chest should be closed in the routine manner, leaving a drainage tube attached to a water-sealed bottle or mild negative pressure.

It is essential that all those in the operating room know exactly what they are to do when the emergency occurs. We have posted a detailed chart outlining the duties of each member of the staff, the routine set-up and essential drugs which must always be kept ready. This chart is posted in a conspicuous place in the scrub room and the nurses’ wash-up area, and by the anesthetist’s table and by the nurses’ table in the operating room. In this way all are reminded constantly of their duties, so that when the need arises they speedily fall into the proper routine. It is important to follow a detailed procedure such as this step by step and not haphazardly, hence the value of having it readily available. This chart is modified from one described by the Christian Medical College, Vellore, India, in the British Medical Journal last year. The chart is shown in Table I and is recommended for general use.

The Grenfell Hospital, St. Anthony, Newfoundland, is a small hospital of 140 beds, and is the headquarters of the International Grenfell Association which serves Northern Newfoundland and Labrador. All major surgery for the area is brought into this institution. In the past 12 years some 6000 operations requiring anaesthesia were carried out, and five cardiac arrests resulted, an incidence of 1:1200 procedures, which is in line with that reported elsewhere.¹ Last year the program just out-
TABLE I.—MANAGEMENT OF CARDIAC ARREST

Don't—Wait for electrocardiograph or cardioscope to diagnose and begin treatment. An absent B.P. and absent pulse in a major vessel (e.g., carotid) means cardiac arrest.
Don't—Give drug of any type until time and cause of arrest is determined.
Don't—Fiddle and hope the heart restarts. You have only 3 minutes to establish circulation before irreversible damage occurs. So cut and massage.

Duties of personnel:

Anesthetist
1. When carotid pulse cannot be felt or heart cannot be heard inform surgeon and ask assistant to record time.
2. Stop all anesthesia.
3. Respirate patient with 100% oxygen.
4. Tilt patient head down.
5. Pass endotracheal tube as soon as possible.
6. When cardiac massage is established, check radial or carotid pulse.
7. Check B.P.

Surgeon
1. When possible, confirm presence or absence of pulsation in aorta.
2a. Open left chest in 4th interspace with transverse incision from sternum to mid-axillary line.
2b. Massage the heart. *
3. When adequate massage has been continued for 3 minutes, pause to see if contractions have returned; if not, follow column 2 below.

Aide
1. As soon as absent pulse is reported bring emergency thoracotomy set.
2. Bring emergency tray of syringes and drugs.
3. Massage the heart.
4. Observe the heart.
5. If shock is ineffective (or defibrillator is not available) give adrenaline 1:1000 0.5-1 ml. into cavity of right ventricle.
6. If spontaneous best returns follow column 1.
7. If heart is in asystole follow column 2B.

Assistant surgeon
1. Insert rib spreader and cut 1 or 2 adjacent cartilages for adequate exposure.
2. Stand by to relieve surgeon massaging the heart.
3. Start i.v. if no one else is available.

Others
1. One person call out time at one minute intervals until adequate massage is established.
2. One person start i.v. and assist anesthetist.

POSSIBLE SITUATION EXISTING AFTER ADEQUATE MASSAGE AND OXYGENATION

1. The heart remains a normal beat
   (a) Continue massage in rhythm with beat until contractions are vigorous and B.P. is maintained at 80 systole.
   (b) If contractions do not improve or if they become weaker give adrenaline 1:1000 0.5-1 ml. into the cavity of the right ventricle.
   (c) If the heart is contracting well but the B.P. remains low a noradrenaline (4 mg. in 500 ml. 5% glucose) drip should be started.
   (d) Observe the heart for at least 30 minutes of adequate action before closing the chest.
   (e) Close the chest with adequate hemostasis.
   (f) If acidosis develops, give 3-7 g. sodium bicarbonate i.v.

*Cardiac massage
Adequate cardiac massage should produce a palpable peripheral pulse and should be carried out thus:
Grasp the heart with the whole hand, apply pressure with the palm surfaces of the fingers and the thenar eminence. Do not use the finger-tips. If the heart is large use both hands. 80-90 contractions per minute is the most effective rate, but this may be difficult to maintain. The rate must be slow enough for the heart to fill during diastole, and the maximum rate possible may be only 50-60 per minute. The cerebral and coronary circulations can be improved by occluding the aorta below the origin of the left subclavian artery. The clamp should be released every 20 minutes to allow circulation to the kidney.

Drug tray
1. Adrenaline—1:1000 solution
2. Calcium chloride—10% solution
3. Atropine—graft 1/100
4. Procaine HCl—1% solution
5. Normal saline—100 ml.
6. Noradrenaline—one 4 ml. ampoule
7. Sodium bicarbonate—i.v. ampoules

2. The heart remains in arrest
   (a) Open the pericardium.
   (b) Observe the action of the ventricles for diagnosis.
   (c) Observe the colour of the heart: if it is well oxygenated it will be a good pink colour.

A. The heart is in fibrillation
   1. Procaine 1% 2-10 ml. into cavity of right ventricle.
   2. Massage for 3 minutes awaiting action of procaine.
   3. If no result use electric defibrillator.
   4. If shock is ineffective (or defibrillator is not available) give adrenaline 1:1000 0.5-1 ml. into cavity of right ventricle.
   5. If spontaneous best returns follow column 1.
   6. If heart is in asystole follow column 2B.

B. The heart is in asystole
   1. Continue adequate massage.
   2. Give CaCl2 10% 5-10 ml. into cavity of right ventricle.
   3. If asystole persists and the heart is a good colour, give adrenaline 1:1000 0.5-1 ml.
   4. Atropine 1/100 grain in 10 ml. normal saline into cavity of right ventricle if of reflex or vagal origin.
   5. If heart beat returns follow column 1.
   6. If asystole persists continue massage up to two hours or until the heart fails to fill in diastole.

Sterile instrument package
1. Rib spreader (1)
2. Hemostats (3)
3. Medicine glasses—1 oz. (4)
4. Scalpel with blade
5. Scissors
6. Forceps (2)
7. Syringes—10 c.c. (4), 2 c.c. (2)
8. Sponges
9. Hypodermic needles—18 x 1½ (2), 22 x 1½ (4)

Defibrillator apparatus to be always ready.

lined was set up, and since then there has been one arrest. This case was saved, the only survival in the group.

Case 1:
A 22-year-old woman was admitted for Caesarean section because of pelvic disproportion and an old tuberculous hip. The fetal head was floating. On June 28, 1949, a spinal anaesthetic, 10 mg. of pontocaine, was administered, and three minutes after this the patient's heart stopped. An incision was quickly made into the uterus; the baby was delivered and it breathed spontaneously. The patient's heart was massaged through the diaphragm. Coramine (nikethamide) was given. The heart beat returned for a time, but then ceased. The length of time between the onset of arrest and the start of massage was not recorded. The cause of arrest was undoubtedly the sudden hypotension following the spinal anaesthetic. We no longer use spinal anaesthesia for Caesarean sections. If it is used, an intravenous drip should be started first and drugs should be readily available to treat hypotension.
Case 2:
A 58-year-old man was admitted with carcinoma of the hepatic flexure of the colon. On September 17, 1951, an extensive procedure was carried out under spinal pontocaine anesthesia, 15 mg., and near the end of it the heart went into arrest. The heart was massaged and the heart action returned, but he developed fibrillation, which continued and finally the heart stopped again. A defibrillator was not available. The cause of arrest here was thought to be possibly the extensive procedure, and blood loss with shock. The length of time between the onset of arrest and the start of massage was not recorded.

This case illustrates the need for prompt replacement of blood loss, and treatment of hypotension as soon as it occurs. Probably there was an element of vaso-vagal reflex due to traction and pain, as the spinal anaesthetic might have been wearing off towards the end of the procedure. Had a proper routine and electrical defibrillator been available, this case might have been saved. We would no longer use spinal anaesthesia for a case of this kind, and, in fact, do not use it at all.

Case 3:
A 62-year-old Eskimo was admitted with a small ventral hernia. He had a history of coronary thrombosis one year before. An operation was done on February 6, 1952, for repair of hernia under local procaine anesthesia. The procedure was short and uncomplicated, but just after reduction of the hernia the patient went into asystole. Immediate massage was carried out through the chest but the heart was flabby and dilated, and did not respond at all. The cause of arrest was felt to be another coronary thrombosis, but this was not proved at autopsy. The length of time between the onset of arrest and the start of massage was not recorded.

Case 4:
A 19-year-old Eskimo was operated on for far advanced bilateral pulmonary tuberculosis on January 14, 1953. A left six-rib thoracoplasty had been done in three stages, and was being followed by a right extrapleural pneumonolysis with paraffin plombage. Anaesthesia was by thiopentone, gas, O₂, and ether. There was considerable difficulty with the anaesthetic and the patient was anoxic. Just at the end of the procedure the heart stopped. The wound was quickly closed and the patient was turned on his back; an incision was made on the left side and massage started. Normal heart action re-started, and after about one-half hour of artificial respiration, breathing re-started. The patient, however, never regained consciousness, developed rigors and convulsions, and died two days later. At autopsy there were marked petechial hemorrhages scattered throughout both cerebellar hemispheres, and one haemorrhage in the pons measuring 0.5 cm. in diameter. It is felt that there was too long a time between the onset of arrest and start of massage, and the importance of not taking any time to close a wound but immediately starting massage is emphasized by this case. The cause of arrest was undoubtedly the anaesthetic and prolonged anoxia. Had massage been started earlier, he likely would have recovered. Had there been adequate ventilation during the procedure, arrest would probably not have happened.

Case 5:
A 19-year-old girl was admitted in labour on December 5, 1958, with a diagnosis of pre-eclampsia and occipito-posterior position. Labour progressed, and at 11:30 a.m. she was prepared for forceps delivery. A pudendal block of 2% procaine was given. Immediately after this she developed clonic movements, seizures, and cardiac arrest. Later it was discovered that a mistake had been made in the solution and about 10 c.c. of 1% pontocaine had been injected rather than procaine. Both the nurse and doctor had recently arrived on this side of the Atlantic, and apparently were unfamiliar with our terms or labels, hence the error. Since then pontocaine in the operating room has been placed in a red bottle with a prominent label "not for injection". However, the possibility of causing cardiac arrest by giving in error a toxic drug or an overdose of a drug is illustrated by this case.

The operating team was immediately called and was available, having just finished another operation in the next room, and the planned measures were immediately instituted. The emergency thoracotomy set was taken out, and the chest wall immediately opened through the fifth interspace, the pericardium opened, and cardiac massage started. The heart was in asystole, flabby, and did not contract at all. Meanwhile, an intratracheal tube was passed, and positive pressure artificial respiration with 100% oxygen was started. After massaging the heart for about one minute, fine contractions were noted. Ten c.c. of calcium chloride was injected into the right ventricle, and after this, good contractions resumed, assisted by hand massage. After several minutes, the contractions became very rapid and weaker, and 0.5 c.c. of 1:1000 adrenaline was injected into the right ventricle. Following this, contractions resumed with good, forceful beats and better rhythm. Blood pressure came back to 120/80 mm. Hg, but then began to drop again; therefore, noradrenaline drip was started, maintaining the blood pressure at about 110/70 mm. Hg. Considerable manipulation with the drip was needed to maintain the pressure, which was very labile. Approximately four to five minutes elapsed from the time cardiac arrest developed until massage was adequately started, and approximately ten minutes elapsed before the heart returned to beating satisfactorily on its own. At 12 noon massage was stopped, and the heart allowed to beat on its own. It was observed for a half hour before the chest was closed.

As the circulation re-started on its own, the patient developed fine twitches and then seizures which were controlled by intravenous thiamylal (Sutural) and later paraldehyde rectally. The cause of this was possibly the continued absorption of residual pontocaine. She regained consciousness at 9 p.m. the same evening. The following day she appeared normal except for amnesia back to the day before massage. She has fully recovered with no sequelæ and no further seizures. Had the routine and set-up not been readily available, this patient would not have survived.

Each of these cases illustrates a different cause of arrest. With the exception of Case 3, it is felt that with prompt institution of a planned procedure and adequate measures each patient might have been saved.
SUMMARY

Cardiac arrest occurs once in every 1000 to 1500 anaesthetics.

Prompt resuscitative measures must be instituted if recovery is to ensue, because the brain can withstand complete anoxia for not more than three to five minutes.

A program is outlined for the management of arrest.

Five cases are reported with one recovery.

The author wishes to express appreciation and thanks to Dr. John M. Gray for helpful criticism in preparing this paper.

REFERENCES


GONADAL DYSGENESIS WITH FEMALE HABITUS*
REPORT OF TWO CASES

Y. Goulet, M.D., B. Lebecuf, M.D., J. Grignon, M.D., F.R.C.P.[C], and C. E. Grignon, M.D., Montreal

The past decade has witnessed important advances in our knowledge of abnormal sex differentiation. Better understanding of such aberrations stems mainly from two sources: experiments on sex embryogenesis and studies dealing with cellular morphology in regard to genetic sex. These have allowed a rational classification of these disorders, based on chromosomal sex as determined at fertilization time as well as on morphological features developed through sexual differentiation. Two cases of hitherto so-called "ovarian agenesis" will be reported and comments will be made, stressing the current lines of thought on the pathogenesis of this syndrome.

CASE REPORTS

Case 1.-M.N.G. was first seen in February 1954, at the age of 15, because of stunted growth. She reported that, until the age of 7 or 8, she had been of normal weight and height for her age, but that she then stopped growing. Secondary sex characteristics had never appeared. For the past two years, she had been given injections of chorionic gonadotrophins, receiving 1000 units twice a week. She was free from any other complaint, and had not suffered from any disease other than whooping cough, measles and scarlet fever. There was no familial incidence of retarded growth or sexual development.

*From the Department of Medicine, Notre-Dame Hospital, Montreal.

She was a well-nourished short girl (Figs. 1a, 1b) with broad chest. Physical characteristics are given in Table I. No webbed neck was noted. Sparse axillary hair was present, with absent breast development. The eyes were normal. Blood pressure was 110/70 mm. Hg. Examination of the genitalia showed underdeveloped clitoris, labia minora and labia majora. No uterus or adnexa were felt by rectal digital examination.

TABLE I.—ANTHROPOMETRIC MEASUREMENTS

<table>
<thead>
<tr>
<th>Measurement</th>
<th>M.N.G.-15</th>
<th>G.P.-17</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actual</td>
<td>76 (34.5)</td>
<td>80 (36.3)</td>
<td>lb. (kg.)</td>
</tr>
<tr>
<td>Weight age</td>
<td>11 ½ yrs.</td>
<td>12 yrs.</td>
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<tr>
<td>Average for age</td>
<td>108 (49)</td>
<td>114 (51.7)</td>
<td>lb. (kg.)</td>
</tr>
<tr>
<td>Height</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actual</td>
<td>50.5 (128.3)</td>
<td>52 (132.1)</td>
<td>inches (em.)</td>
</tr>
<tr>
<td>Height age</td>
<td>9 yrs.</td>
<td>9.5 yrs.</td>
<td></td>
</tr>
<tr>
<td>Average for age</td>
<td>62.5 (158.7)</td>
<td>63.7 (161.8)</td>
<td>inches (em.)</td>
</tr>
</tbody>
</table>

Radiological studies showed a skull of essentially normal appearance. Osseous age was estimated to be between 13 and 14 years. Results of laboratory examinations were as follows: normal blood urea, glucose and cholesterol; red blood cell count 3,825,000 and white blood cells 6250 per c.mm. Urine was normal. Basal metabolic rate determination gave a result of +6%. Urinary hormonal levels were as follows:

Follicle stimulating hormone (F.S.H.) > 211.2 and < 316.8 mouse units (m.u.)/24 hrs.

*Estrogens: < 40 m.u./24 hrs.

17-Ketosteroids: 3.6 mg./24 hrs.

The patient was treated by cyclical administration of 1 mg. diethylstilbestrol. One year later, she weighed 103 lb. (46.7 kg.) and had grown 2½ in. (6.35 cm.). Development of secondary sex characteristics is shown in Figs. 1c and 1d. She experienced vaginal bleeding on...