

## THE CAUSES AND TREATMENT OF COLLAPSE ON THE OPERATING TABLE\*

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STEDMAN defines collapse as a condition of extreme prostration similar to shock and due to the same causes, often with the addition of a great loss of fluid. To the anaesthetist, collapse generally signifies one of two things: first, primary cardiac failure, or, secondly, the more gradual deterioration of a patient's condition, which is conveniently described as shock and which, if severe and untreated, may lead to secondary cardiac failure. Thus secondary failure occurs because the patient has died whereas in primary failure the patient is still alive and will remain so for several minutes. Immediate vigorous treatment in this latter type should restore the heart action leaving the patient none the worse for his experience.

Anaesthesia, as it is generally practised by anaesthetists, surgeons, or physicians falls into several well-defined groups. The question of collapse will be considered as it occurs most commonly in each of the following groups.

### A Inhalation Anaesthesia

### B Conduction Anaesthesia

- (1) Spinal and Epidural Anaesthesia
- (2) Regional Block Anaesthesia
- (3) Local and Topical Anaesthesia

### C Intravenous and Rectal Anaesthesia

#### A *Inhalation Anaesthesia*

In this type of anaesthesia, the most dramatic collapse occurs in the form of primary cardiac failure. Usually, in the early stage, the heart is either beating very feebly, in ventricular fibrillation, or quiescent.

Primary cardiac failure can occur in both normal and cardiac patients. This type of failure or arrest may arise from marked vagal stimulation such as stimulation of the trachea or be due to traction on the hilar regions of the lungs, or on splanchnic and mesenteric plexuses or the broad ligaments. It may result from anoxia produced by an over-dosage of the anaesthetic agent or by respiratory obstruction. Hypoxia and hypercapnia are important factors in potentiating the vagal effect on the pacemaker.

In 41 cases of primary cardiac failure, Turk and Glenn (4) list the causes as follows.

Sudden death, either from the condition which brings the patient to the operating room or from a complicating medical condition, may simulate primary cardiac arrest. Patients suffering from severe haemorrhage, trauma, toxæmia, or insulin shock form a part of this group. In any event, if, suddenly, no pulsations

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TABLE I  
PRIMARY CAUSES OF CARDIAC ARREST

Cause	Number of Cases
<i>Vagal Stimulation</i>	
Direct	4
Reflex	1
<i>Anoxia</i>	
Haemorrhage	5
Obstructed airway	
Anaesthesia	2
Head injury	2
Coronary occlusion	1
Sedation	1
<i>Drug idiosyncrasy</i>	2
<i>Direct endocardial stimulation</i>	
Cardiac catheterization	2
<i>Primary arrhythmia</i>	
<i>Acute adrenal insufficiency</i>	
<i>Uncertain</i>	

NOTE A few of these were preoperative and postoperative cases

can be felt and no sounds can be heard over the praecordium, then the diagnosis of primary cardiac failure is made and no time must be lost in instituting artificial respiration and artificial circulation. A thoracotomy is performed and the heart massaged while oxygen from the anaesthetic machine is administered by manual control through an endotracheal catheter, if one is already in place, or by a face-piece. Fluids of any type may be administered intravenously under pressure. Intracardiac infusion of epinephrine (3-5 cc of 1/10,000 solution) may initiate rhythm after massage has been started although ventricular fibrillation may be produced if the myocardium is still anoxic.

Ventricular fibrillation is often a product of light anaesthesia and frequently occurs early in induction or just before the moment of awakening. It may be closely associated with a sudden increase of epinephrine in the blood stream—either endogenous or exogenous. It may also follow another cardiac arrhythmia. The usual reasons for these arrhythmias are:

1. Severe oxygen lack
2. Stimulation of autonomic reflexes with the vagal nerve as the efferent pathway
3. Manipulation of the heart or great vessels
4. Temporary circulatory obstruction.
5. Over-dosage of anaesthetic agent
6. Excessive use of vasopressor drugs
7. Manipulation of epinephrine producing tumours.

Treatment of ventricular fibrillation is, again, artificial respiration and circulation. Massage is instituted immediately to ensure good colour and tone of the heart muscle and then electrodes are placed on the heart to carry out the defibrillation technique. Two or three shocks, each lasting about one second,

are generally sufficient to bring the fibrillation to a stop and then massage and epinephrine treatment are instituted as for cardiac arrest. The usual defibrillating current required is 1–2 amperes of a 60 cycle, 110 volt current. It has been shown in the dog heart that currents of less than one ampere regularly produce fibrillation whereas the above-mentioned amperage stops fibrillation. Procaine hydrochloride injected into the right ventricle in amounts of 3–4 cc. of 1 per cent solution bathed over the epicardial surfaces may also prove helpful in initiating normal rhythm where fibrillation has been prolonged. Brock and Milstein state that in a series of thirty cases of fibrillation they felt that procaine in doses of 50–200 mg. intravenously was effective in abolishing nine episodes of fibrillation. All these steps in the treatment of ventricular fibrillation may have to be repeated several times before normal rhythm is regained and maintained. However, it may also happen that recovery to normal rhythm may occur while resuscitative measures are being prepared.

Operative shock or progressive hypotension is often a prelude to secondary cardiac failure unless the continuing deterioration is reversed. A major fall in blood pressure may simulate primary cardiac failure because of absent pulses, but cyanosis and pallor occur more slowly in the secondary form of failure. Often obvious haemorrhage or traction on viscera may be the clue to the cause of hypotension

The usual causes of secondary failure can be enumerated as:

1. Loss of fluids through sweating, respiration, haemorrhage, extravasation.
2. Prolonged exposure to trauma.
3. Stimulation of traction reflexes.
4. Loss of heat from exposed viscera and cold inhaled vapours
5. Over-dosage of anaesthetic, either relative or absolute.
6. Severe anoxia.
7. Sudden changes or extremes in positioning of patient.
8. Perforation of a viscus

Blood replacement is the treatment for haemorrhage; but plasma and plasma substitutes can be utilized until blood is available. Vasopressors, moderate head down position, adequate oxygenation, and intravenous fluids are the accepted quartet for treatment of increasing hypotension, provided the noxious stimulus or other exciting factor has been removed, and blood loss has not been excessive. Levophed may be necessary during prolonged surgery or excessive trauma to maintain approximate normal blood pressure but should not be utilized as a substitute for blood replacement. Thus the avoidance of deeper anaesthesia than necessary, the prevention of loss of heat and moisture, the avoidance of rough handling of tissues, and the maintenance of a high alveolar oxygen level throughout the operation are the main points to be kept in mind, in order to prevent the onset of increasing hypotension. In a normal patient, surgical or anaesthetic shock need never be fatal, but in patients with marked myocardial degeneration, coronary stenosis, chronic debilitating disease with or without advanced age, death may occur rapidly. Thus the issue resolves itself into one of balance between cardiac effort and cardiac support in the matter of tissue oxygenation and replacement of blood or fluid loss. Jacoby, Flory *et al* (8) report a successful out-

come in a case in which 54 pints of blood were given in 2½ hours to offset the blood loss during surgery. They also gave one gram of calcium gluconate for each 2-3 pints of transfused blood to offset the deleterious effects of hypocalcemia.

The circulatory factors relevant to collapse in inhalation anaesthesia not only include accidents due to decreased blood pressure, but those due to increased blood pressure as well. Severe hypertension may be produced as follows

1 By rapid asphyxia which occurs occasionally during an anaesthetic and is augmented at the start by initial struggling and breath-holding.

2 A slow increase in blood pressure caused by poor technique which prevents adequate removal of carbon dioxide

3 Increased epinephrine in the circulation due to fear, excitement, and struggling.

4 Overhandling of a toxic thyroid gland or adrenal tumours.

These four factors may produce acute cardiac dilatation or rupture of a blood vessel. Cardiac dilatation may restrict coronary flow, causing the heart to stop, while rupture of a vessel may cause immediate death. Obviously the treatment of these two conditions lies only in their prevention with emphasis on smooth, quiet induction and the maintenance of a free airway with attention to adequate removal of accumulating carbon dioxide

Respiratory failure under general anaesthesia can be partial or complete but in either case, if allowed to persist long enough, will cause vasomotor collapse through prolonged anoxia. Respiratory failure can be considered as either central or peripheral respiratory failure

Central respiratory failure is generally caused by

1. Overdose of pre-anaesthetic sedative
2. Overdose of the anaesthetic agent
3. Increase of intracranial pressure

One of the above factors may lead to serious trouble but a combination of two or more of these factors may well be disastrous

When pre-anaesthetic medication has lowered the respiratory rate, the increased level of carbon dioxide in the blood will raise the normal respiratory threshold. Hyperpnoea of induction as in vinethene or ether induction will lower the carbon dioxide level and respirations may cease. Anaesthesia in a closed circuit will maintain blood carbon dioxide at more normal levels so that apnoea can be avoided. However, under prolonged closed anaesthesia elevated carbon dioxide levels may drop seriously at the conclusion of the anaesthetic with resultant hypotension and/or respiratory failure as is occasionally seen in so-called "cyclopropane shock". This somewhat rapid drop in blood pressure can be avoided by gradual elimination of the opiate, anaesthetic, and carbon dioxide towards the end of the operation by conversion to a high flow of nitrous oxide and oxygen in a semi-closed system.

Children, because of inadequate pre-anaesthetic sedation, often show hyperpnoea throughout induction with resultant blow-off of carbon dioxide and precipitate anaesthesia which further depresses respiration. Apnoea and cyanosis occur, and these are followed by frantic resuscitative measures on the part of

the anaesthetist. This situation can be avoided through adequate preoperative sedation and therefore quiet induction.

Any intracerebral or medullary pathology may elevate the respiratory threshold so that further elevation by opiates before operation or administration of the anaesthetic may completely paralyse respiration

Peripheral respiratory interference occurs in such conditions as:

1. Breath-holding—due to direct irritating vapours.
2. Relaxation of jaw allowing tongue to occlude respiratory tract.
3. Laryngeal spasm—due to direct irritation of vocal cords or from abdominal reflex origin.

These conditions may lead to apoplexy or acute cardiac dilatation or to the effects resulting from prolonged hypoxia

4. Aspiration of debris during induction or anaesthesia, which can lead to partial or total collapse of lung with subsequent hypoxia, collapse, and death.

The usual causes for this accident are:

1. Vomitus—in accident cases, obstetrical patients, those with intestinal obstruction, or poorly prepared patients.
2. Excess mucus
3. Pus from pharyngeal or peritonsillar abscesses
4. Bronchiectasis or lung abscess
5. Haemorrhage from surgery or anaesthetic trauma to nose, throat, and mouth
6. Broken teeth or suction tips
7. Sponges or pharyngeal packs

The use of lavage where a full stomach is suspected is a safety precaution that cannot be overemphasized. Often, however, particles of undigested food will still remain in the stomach so that smooth induction, the presence of adequate suction, an operating table that can provide immediate Trendelenburg, and an oro-tracheal cuffed catheter are all indispensable implements in the handling of the suspect "full stomach"

However, the above-mentioned accidents still occur and the restoration of pulmonary function is the prime concern in the institution of resuscitation. If no foreign obstruction is present in the pharynx or in the glottic region then intubation, oxygenation, and suction are the prime points in immediate resuscitation. Bronchoscopy should be carried out to complete the removal of vomitus, blood clots, or foreign material from the trachea or bronchi. Anoxia during the bronchoscopy should be studiously avoided, since the patient's hypoxic state can easily be further aggravated during the insertion and manipulation of the bronchoscope.

Other pulmonary conditions which, if left unattended, can lead to hypoxia and collapse are.

1. Internal or external paradoxical respiration
2. Bronchopleural or tracheal fistula.
3. Bilateral pneumothorax
4. Acute emphysema.
5. Pleural effusion
6. Pulmonary oedema

External paradoxical respiration can be treated by providing an unobstructed

orotracheal airway. Internal paradoxical respiration occurs in the open chest and is handled by controlled respiration. Bronchopleural or tracheal fistula are generally accidentally produced and allow escape of vital oxygen and anaesthetic agent. High flow oxygen, intravenous anaesthesia, if necessary, and repair of the damage are the necessary points in the treatment. Bilateral pneumothorax may occur in surgery of the oesophagus, trachea, or the great vessels and controlled respiration via an endotracheal catheter is the bulwark against collapse. Acute emphysema following ball-valve obstruction of a bronchus may require bronchoscopy or bronchial exploration to relieve the obstruction. Rupture of a lung cyst following positive pressure by the anaesthetist may cause pulmonary collapse so that immediate reparative surgery may be required before this complication progresses

Undiagnosed pleural effusion may be present in cardiac patients or patients presenting with sub-phrenic or pancreatic abscesses. Respiratory embarrassment may become quite marked during or at the end of the operative procedure.

Pulmonary oedema is a serious complication which, fortunately, develops rarely. It is characterized by gentle wheezing and the appearance of pinkish fluid in the tracheobronchial tree. The usual causes centre around damage to pulmonary capillaries by

1. Prolonged shock.
2. High pulmonary intravascular pressure from cardiac dilatation or cardiac tamponade.
3. Excessive negative intratracheal pressure, i.e. respiratory obstruction.
4. Direct injury as in an anaesthetic explosion
5. Prolonged extreme head down position
6. Overhydration
7. Anaphylactic reaction
8. Congestive heart failure

Treatment consists of tracheobronchial toilet and the application of slight positive pressure during expiration. Intravenous infusion should be stopped or slowed and the table tilted to a 30° Fowler's position. Aminophylline (7½ gr i.v.) should be given slowly and may be repeated if necessary. If the oedema is considered to be cardiac in origin, then digitalization should be carried out with 0.5–1 mg of digoxin which may be repeated if necessary. Cortisone therapy may be required if oedema was due to explosion or anaphylactic reaction. Phlebotomy and administration of hypertonic glucose may prove useful. If blood was administered during the surgical procedure, then a recheck of the blood grouping should be carried out.

There are many other accidents that can precipitate collapse under general anaesthesia, a few of which deserve separate mention

(1) *Tracheal Collapse* This may occur following removal of a large thyroid gland with resultant respiratory obstruction. Intubation either should be done at the start of the thyroid operation or at least be anticipated throughout the procedure

(2) *Convulsion* Deep ether convulsions occur chiefly in children but may occur in adults as well. They may also occur in light anaesthesia. Controversy

exists as to whether deficiency or excess of carbon dioxide is the chief cause although quite often a febrile condition co-exists. Regardless of the cause, treatment consists of removal of the anaesthetic, administration of oxygen, and the intravenous administration of Pentothal.

(3) *Venous or Arterial Air Embolism* These conditions prevent adequate cardiac emptying and filling with marked reduction in cardiac output and may lead to shock or immediate death. Massive amounts of air may be sucked or forced into the circulatory system after intravenous fluids are exhausted. Air embolism can also occur in neurosurgery and pelvic surgery and may lead to sudden cardiac failure. The patient should be placed in the supine position, turned on the left side, and given oxygen. If a normal heart beat is not restored, a needle puncture of the right ventricle should be performed in an attempt to aspirate the air, and if this fails, the air should be aspirated through a thoracotomy incision and massage instituted if necessary.

(4) *Ether Accidents* (a) Liquid ether in lungs may occur when the ether vaporizer is wrongly connected. Violent pulmonary congestion, oedema, and death may ensue. This apparatus should always be tested before use. The use of a Flagg can will allow ether to be spilled into the lungs if it is tipped. These cans should always be strapped to the table after the patient is positioned. (b) Boiling ether is another factor. Some vaporizers have a heating arrangement to vaporize ether more efficiently, especially when adults are being anaesthetized with pharyngeal insufflation. When anaesthesia deepens the hook should be removed from the mouth—it is not sufficient to turn off the air current.

(5) *Miscellaneous* Explosions or fire, metabolic emergencies, mechanical or human errors in anaesthetic techniques are mentioned for the sake of completeness and should not be minimized.

## B *Conduction Anaesthesia*

Toxic reactions to the drugs employed in the use of spinal and epidural anaesthesia, regional and local anaesthesia are due either to: sensitivity to the drug or to a high blood level of the drug. Sensitivity to the drug provides less than 2 per cent of the reactions seen and the wheals, itching, hypotension, and asthmatic breathing are treated symptomatically.

High blood levels of the agent used account for more than 98 per cent of the reactions seen and these reactions are more severe in nature. High levels arise mainly from inadvertent intravascular injection or injection into a highly vascular area. The use of hyaluronidase spreads the absorptive area and may give excessively high blood levels. Errors in dosage and faulty detoxification mechanisms in the patient also raise the blood levels.

The signs and symptoms are central in origin and are listed as follows:

1. Mental—drowsiness, dullness and coma.
2. Cardiovascular—Tachycardia progressing to bradycardia and hypotension, possible cardiac arrest.
3. Respiratory—Depressed shallow respirations, dyspnoea or bouts of apnoea.
4. Neurological—Minimal twitching or generalized convulsions.

These signs may be immediate or delayed up to thirty minutes. Thus the anaesthetist must be prepared to administer oxygen, vasopressors, atropine, barbiturates, and even perform cardiac massage if necessary for at least one half hour after the block has been given. It cannot be overstressed that immediate access to operating room facilities is of prime importance whenever block procedures are contemplated because of the possibility of toxic reactions to the drugs employed.

In the first half hour of spinal anaesthesia the most dramatic form of collapse is that of a combined respiratory and circulatory depression. The tone of the entire vascular bed is maintained both by the action of the sympathetic innervation and by the tone of the voluntary muscles. Thus the combined effect of sympathetic paralysis, hypoxia due to respiratory depression, and loss of muscular tone is to produce circulatory and respiratory depression which varies directly as the height of anaesthesia progresses. This condition may go on to severe collapse and cardiac slowing by paralysis of the cardiac accelerator nerves.

Treatment consists of

1. Intravenous infusions of saline or glucose with vasopressor administration through the rubber tubing
2. Oxygen administration by mask with positive pressure
3. Horizontal positioning of patient since head up or head down positions may further advance the hypobaric or hyperbaric spread of the anaesthetic solution or conversely with result in imperfect anaesthesia so that added agents will be required after normal tension has been restored.

With profound vasomotor and respiratory collapse cardiac standstill may occur. Treatment consists of

1. Instant recognition
2. Scultetus position to provide blood for the vital centres
3. Cardiac massage
4. Oxygen mask under positive pressure—later by endotracheal tube

Respiratory and circulatory collapse under spinal anaesthesia can occur individually or combined and it must be stressed that either can set up a chain of events that will bring about the other.

Hypertension may occur with resultant cardiac dilatation, rupture of a cerebral vessel, or cardiac arrest. The cause may be asphyxia, adrenalin excess, too much sympathetic stimulation prophylactically or from the toxic effect of the vasoconstrictor agent used. Treatment of hypertension consists of assisted respirations in a closed circle filter, aminophylline 3½ gr. i.v., 50 mg doses of Pentothal, or if available a nitroglycerin tablet 1/100 gr placed under the tongue.

Epidural anaesthesia can produce hypotension to about the same degree as spinal anaesthesia although respiratory paralysis is not as marked. Inadvertent puncture of the outer meninges and flooding of the canal with the agent used may produce spinal anaesthesia past the foramen magnum with profound vasomotor and respiratory collapse. This same complication may also occur in stellate ganglion block, thoracic or lumbar paravertebral block, caudal block, and even in a brachial block. The usual precautions of aspiration, followed by the injection of a small amount of the drug and several minutes' scrutiny for any untoward



reactions, should always be taken. In the event of a misadventure, treatment is the same as that already outlined for spinal circulorespiratory collapse.

The use of topical anaesthetics such as cocaine, butyn, pontocaine, nupercaine as employed in rhinology, laryngology, ophthalmology, and genito-urinary procedures, is fraught with the same dangers as those encountered in regional or local block procedures. With regard to the use of barbiturates before operation to offset these toxic phenomena, Moore (2) is of the opinion that barbiturates given before operation are useful only to allay apprehension. Moore states that to prevent a toxic reaction, barbiturates would have to be given in anaesthetic levels. Stimulation of the central nervous system, progressing to eventual depression and paralysis of respiratory and vasomotor centres, is the usual sequence of signs of drug toxicity. The treatment for such reactions consists of oxygen, suction, vasopressors, and small doses of intravenous barbiturates to stop convulsions.

### C *Rectal and Intravenous Anaesthesia*

The relative or absolute over-dosage of anaesthetic agents in rectal or intravenous anaesthesia constitutes the main cause of mishaps in these forms of anaesthesia. Barbiturates by rectum or intravenously can cause profound respiratory and circulatory collapse and the treatment is supportive. Other forms of rectal anaesthesia produce their ill effects through respiratory depression and resultant anoxia. Intravenous procaine carries with it the dangers of toxic manifestations and in higher doses may lead to cardio-respiratory collapse through its action on central and autonomic nervous systems.

### SUMMARY

This article has been designed to review some of the more important forms of collapse on the operating table and in most instances the cause and treatment of each form of collapse have been outlined briefly. Collapse under inhalation and conduction anaesthesia has received the bulk of attention in this review since rectal anaesthesia is not a common procedure and intravenous anaesthesia in all its newer forms was considered to be beyond the original scope of this article. Individual surgical techniques which in themselves might prelude collapse were purposely omitted and instead an effort was made to keep the subject within the anaesthetist's domain.

### RÉSUMÉ

Nous avons passé en revue les différentes formes de collapses pouvant survenir sur la table d'opération ainsi que leurs causes et leurs traitements, nous avons envisagé le problème selon qu'ils surviennent au cours de (a) l'anesthésie par inhalation; (b) l'anesthésie loco-régionale, (c) l'anesthésie rectale et intraveineuse.

Sous anesthésie générale, nous avons l'impression que la défaillance cardiaque primitive survient fréquemment à la suite d'un effet vagal sur le centre d'automatisme résultant d'une stimulation vagale soit directe soit réflexe. L'anoxie et l'hypercapnée sont des facteurs importants quand survient la défaillance cardia-

que primitive. Une complication médicale ou chirurgicale peut également produire le même tableau clinique d'arrêt cardiaque. Les premiers gestes de la resuscitation sont les suivants: le diagnostic précoce, la thoracotomie, le massage cardiaque, l'oxygène sous pression et l'adrénaline intracardiaque si nécessaire. La fibrillation ventriculaire survient sous anesthésie légère, en présence de quantité excessive d'adrénaline, en déficience d'oxygène ou, encore, à partir d'une autre arythmie; on convertit cette fibrillation en arrêt cardiaque par la défibrillation électrique puis on institue le traitement de l'arrêt cardiaque. Certains auteurs ont employé avec succès de la procaine à 1 pour cent soit en injections intracardiaques (3-4 cc.) soit en injections intraveineuses (50-200 mgm.) dans le but de restituer un rythme normal lorsque la fibrillation s'est prolongée.

La défaillance cardiaque secondaire est une autre sorte de collapse, elle peut survenir au cours de l'anesthésie générale et elle est le résultat d'une aggravation progressive de l'état du malade. Cette sorte de collapse peut résulter soit d'une perte de liquides, d'un traumatisme prolongé, de réflexes de traction, soit d'un surdosage anesthésique surtout si ces facteurs ne sont pas contrôlés souvent. Dans ces cas, il ne faut jamais employer de vasopresseurs à la place de sang ou de plasma. Au cours de cette sorte d'anesthésie, il peut survenir, à l'occasion, une hypertension marquée. Une élimination inadéquate du gaz carbonique, de l'appréhension et de l'agitation, des manipulations exagérées d'une thyroïde toxique ou des surrénales peuvent conduire à cette situation et causer une dilatation du cœur ou la rupture d'un vaisseau.

Sous anesthésie générale, la défaillance partielle ou complète de la respiration, pour peu qu'elle se prolonge, peut également entraîner un collapse circulatoire par anoxie prolongée. La défaillance respiratoire centrale peut survenir à la suite de surdosage de la prémédication ou de l'agent anesthésique ou encore d'une augmentation de la pression intracrânienne. La défaillance respiratoire périphérique est plus fréquente, elle comprend plusieurs formes d'obstruction des voies respiratoires, mécaniques, physiologiques ou pathologiques, obstructions qui peuvent réduire ou empêcher l'oxygénation.

Avec l'anesthésie loco-régionale, on observe occasionnellement des réactions aux drogues employées. Ces réactions apparaissent dans les cas de sensibilité particulière aux médicaments ou lorsqu'il y a un taux élevé du médicament dans le sang. Moins de 2 pour cent des réactions anormales sont dues à une sensibilité spéciale et elles peuvent être traitées symptomatiquement, tandis que 98 pour cent de ces réactions sont provoquées par un surdosage et elles peuvent être sérieuses. On peut observer des manifestations psychiques cardiovasculaires, respiratoires et neurologiques dont le traitement requiert de l'oxygène, des vasopresseurs, de l'atropine, des barbituriques et, dans un nombre de cas plutôt restreint, le massage cardiaque peut devenir nécessaire. En conséquence, quand on projette de faire un bloc, il faut toujours avoir à sa portée les facilités dont on dispose dans les salles d'opérations.

Au cours de la rachianesthésie, la complication la plus dramatique est le collapse simultané de la circulation et de la respiration. La bradycardie peut aussi devenir un facteur important. Le traitement doit tendre à lutter contre l'hypotension et la manque d'oxygène. L'hypertension et ses complications peu-

vent avoir pour causes: le manque d'oxygène, l'excès d'adrénaline, endogène ou exogène, ou encore, une stimulation sympathique prophylactique trop intense. Les blocs épiduraux également peuvent produire de l'hypotension; toutefois les collapsés plus sérieux qui surviennent sont dus à l'inondation imprévue de l'espace sous-arachnoïdien par l'agent anesthésique. Cette éventualité peut également se produire à l'occasion d'un blocage du ganglion étoilé, d'un blocage paravertébral thoracique ou lombaire, d'un blocage caudal ou brachial. Pour prévenir cette inondation subarachnoïdienne, les détails importants sont: l'aspiration, l'injection de petites quantités et des périodes d'attente

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