ANAESTHESIA IN ADRENO-CORTICAL HYPERFUNCTION¹

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PATIENTS WITH adreno-cortical hyperfunction are not frequently presented to anaesthetists. The proper anaesthetic management for adrenalectomy in these cases requires a basic understanding of the physiopathology of the syndromes caused by hyperfunction of the adrenal cortex.

The adrenal cortex secretes three types of steroid hormones, the glucocorticoids, and the mineralo-corticoids and hormones with androgenic and oestrogenic properties

The majority of glucocorticoids are in the form of hydrocortisone and cortisone, hydrocortisone being the main glucocorticoid secreted. These hormones have been called "S" hormones because of their effect on carbohydrate metabolism, resulting in a decreased tolerance to carbohydrate and an increased tolerance to insulin

Hydrocortisone and cortisone have also been labelled the anti-anabolic hormones since, by diverting amino-acids from gluconeogenesis, they inhibit formation of body protein and eventually there is a marked deficiency of body protein.

The mineralo-corticoids regulate salt and water metabolism Aldosterone which is the most important in man causes retention of sodium and elimination of potassium

The androgens and oestrogens of the adrenal cortex contribute to the development of secondary sex characteristics. Excess of these steroids may lead to suppression of the gonad and distortion of secondary sexual development.

There is no clear-cut separation of the biological activities of these hormones For example, androgens are salt retainers, but not nearly to the same degree as aldosterone Cortisone is a poor mineralo-corticoid compared with aldosterone or even desoxy-cortico-sterone acetate. Due to the overlapping of biological activity, it is therefore not surprising that patients with adreno-cortical hyperfunction are often difficult to categorize clinically.

There are, however, three main syndromes of adreno-cortical hyperfunction which can be attributed to the three groups of hormones produced by the adrenal. These are Cushing's syndrome, virilizing adrenal hyperplasia and primary aldosteronism

Overproduction of glucocorticoids results in Cushing's syndrome and can be produced in man by the administration of cortisone in large doses

In Cushing's syndrome, the influence of glucocorticoids on carbohydrate metabolism is manifested clinically by hyperglycaemia and glycosuria, and these patients may be on fairly large doses of insulin The anti-anabolic effect of the glucocorticoids is manifested clinically by thin skin, easy bruisability due to increased friability of the blood vessels, and delayed wound healing. Deficient

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repair of bone matrix may lead to severe osteoporosis, and vertebral collapse is not uncommon. One of the most striking features of body protein depletion is muscle weakness and wasting. This may be dramatically demonstrated at operation. The loin incision will expose and proceed through much loose adipose tissue and expose a meagre erector spinae mass and later a diaphragm as thin as tissue paper. In addition, patients with Cushing's syndrome may have hypertension and oedema. The female patient may exhibit hirsutism, acne and amenorrhoea to such an extent that differentiation from adrenal virilism is difficult. In many cases such identification is of academic interest only. In Cushing's syndrome there is elevation of the 17-hydroxy-corticoids in the urine and the 17-keto-steroids may also be increased although usually not to the same degree. There may be derangement of the serum electrolyte pattern, with hypokalaemic, hypochloremic, hypernatremic alkalosis, although this is not common.

The commonest syndrome associated with overproduction of the gonadal-like hormones of the adrenal in the adult is virilizing adrenal hyperplasia in the female. This is characterized by hirsutism, amenorrhoea and not infrequently obesity. There may or may not be hypertension. There is elevation of the 17-keto-steroids in the urine, usually with slight or no elevation of the 17-hydroxy-corticoids.

Overproduction of mineralo-corticoids results in primary aldosteronism recently described by Conn (15).

The outstanding clinical finding is that of weakness with episodes of paralysis and tetany attributable to the severe depletion of body potassium that occurs. There is usually hypertension, which is apparently secondary to salt retention, although strangely there is no oedema.

During the past eighteen months we have had eight patients suffering from adreno-cortical hyperfunction presented for adrenalectomy. There were five patients with Cushing's syndrome and three with virilizing adrenal hyperplasia. This small number of cases is inadequate for compilation of a statistical analysis. Because cases of this type are being encountered sporadically by anaesthetists across the country, we feel justified in attempting a discussion of the anaesthetic problems involved. In addition, anaesthesia for adrenalectomy is always an exercise in the anticipation and treatment of emergency situations.

Two cases are presented as examples.

Case 1

The first case was that of an acute fulminating Cushing's syndrome in a 57-year-old man. A probable chromophobe adenoma of the pituitary had been diagnosed five years ago and he had not improved on deep X-ray therapy.

He was sent to Montreal early in January after a period of several weeks in hospital in New Brunswick. The last admissions were because of visual disturbances, swelling of the feet, polydipsia, polyuria and easy fatigability. His hypertension had increased to 220/140 and he had consistently elevated blood sugars. He was obese, plethoric, with a moon-shaped face and excess fat on the face, shoulders and trunk, sparing the extremities. The skin was thin and inelastic and he had numerous ecchymoses and some purpuric spots on the abdomen. There was marked pitting oedema of the legs and the hands were slightly oedematous. The fundi showed a Grade III hypertensive retinopathy with increased light reflexes, A-V nicking, haemorrhages and cotton-wool exudates.

He was orthopnoeic with a persistent productive cough. Rhonchi were heard over the entire chest and there was definite evidence of congestion at the lung bases. The heart was slightly enlarged to percussion. He had episodes of auricular fibrillation.

The laboratory reports on this patient were voluminous and are summarized here. (1) urine showed persistent albuminuria; (2) variable Glycosuria occurred during diet and insulin therapy for his diabetes; (3) blood proteins were decreased—total protein 5.3 gm. per cent, albumin 3.4 gm. per cent, globulin 1.8 gm. per cent; (4) blood cholesterol was elevated to 492 mg. per cent, (5) endocrine assays showed elevated 17-hydroxy-corticoids

Electrocardiogram taken January 21, 1957, revealed left ventricular hypertrophy or alternatively, incomplete left bundle-branch block.

Early in the morning of January 26, the patient had an episode characterized by nausea, vomiting and hypotension, clinically simulating myocardial infarction. Serum electrolytes showed a hypochloremic alkalosis with a serum K of 2.9 mEq. He responded to KCL and electrolyte therapy.

We were asked to see the patient in consultation, and on January 29, Dr. Bromage wrote that he was reluctant to administer an anaesthetic because of the "difficulty in maintaining an adequate respiratory exchange during a long operative procedure in the prone position and during the immediate post-operative period. This would be aggravated by anticipated bronchospasm and secretions during surgery. If operation was imperative, the choice of anaesthesia would be combined continuous extradural block and light endotracheal anaesthesia, with a tracheotomy to be performed before leaving the Operating Room, in order to facilitate frequent tracheo-bronchial toilet. Continuance of the extracural block would provide analgesia for chest pain and result in more effective coughing."

We were advised that because of the fulminating state of his disease, bilateral adrenalectomy offered the only hope for the patient's survival, and the family were well aware of this and of the grave risk involved. On January 31 the patient underwent bilateral adrenalectomy, after preparation with cortisone. Hydrocortisone at the rate of 5 mg. per hour was given in an intravenous infusion during surgery. A reproduction of the anaesthetic chart is shown in Figure 1. The operation was uneventful.

At the point where the arrow marked "EEG" appears, the patient's electro-encephalogram showed diversion from a fast pattern associated with light anaesthesia to delta waves indicative of cerebral anoxia. This occurred in the absence of any clinical manifestations of hypoxia. The patient's colour, blood pressure, respirations and pulse rate showed no changes.

There was no evidence of respiratory obstruction and no audible evidence of secretions in the tube. For this reason aspiration through the endotracheal tube was delayed. The delta waves persisted. After three or four minutes the tube was aspirated and a considerable amount of tenacious mucus removed. Within a few minutes the normal rhythm of light anaesthesia was re-established. It was a striking example of the value of electro-encephalography.

At the completion of surgery a tracheotomy was performed.

On moving the patient from the table, excoriation of the skin of the thighs was noted. We had not considered his tender skin and had allowed direct contact between the skin and the special supporting mattress. This is a lesson we have not forgotten.

The patient was kept under immediate supervision in the recovery room for 48 hours. Members of the resident staff attended him continuing on 4-hour shifts during this period in order to control secretions and supervise the administration of hydrocortisone, insulin, fluids and electrolytes.

Postoperatively the patient exhibited an exaggeration of his pre-existing psychosis. According to Starr, psychosis occurs in 65 per cent of patients with Cushing's syndrome.

The tracheotomy tube was removed on the seventh day. He was discharged three weeks after operation.

His insulin requirements dropped considerably after operation.

ANAESTHETIC RECORD
ROYAL VICTORIA HOSPITAL

← BLUE OPERATION REPORT ALONG HERE →

RESPIRATORY	SURGEON	CODE 11 12	77	57-1175	M/ 57
C.V.S.	DATE OF OPERATION 13 19	TIME	8.10-2.15		
NEUROLOGICAL	PRE OP DIAGNOSIS		Cushing's Syndrome	OPERATION	Bilateral Adrenalectomy
G.U.	ANAESTHETIST	CODE 17 19	2	WEIGHT	165
G.L.				BLOOD PRESSURE	220/140
MISC.					
GOOD	PRE OP MEDICATION		Demerol 100 mgm, Atropine gr 1/150 at 7.0 a.m.		
FAIR	PRE OP REMARKS		Emphysematous chest. Bronchospasm.		
POOR			Secretions --. Orthopneic. V.C. =		
VERY POOR			1500 ml. Hypertensive retinopathy.		
EMERGENCY GOOD			Isopropylnoradrenaline preoperatively.		
EMERGENCY POOR					
SATISFACTORY					
UNSATISFACTORY					
WIL					
BARBITURATE					
ANALGESIC					
CURARE					
RELAXANT					
ANTAGONIST					
ETHER					
WINTHANE					
CHL					
NIO					
ICE					
NUPERCAINE					
PONTOCAINE					
EYLOCAINE					
OPEN DROP OR INS					
ABSORPTION					
PARTIAL REBETH					
NON REBETH					
OROTRACHEAL					
NASOTRACHEAL					
ENDOSMO OR BLS					
SPIRAL I. M. H.					
EPIDURAL					
CAUDAL					
N. BLOCK					
INFILTRATION					
TOPICAL					
DIAG THERAP					
RECTAL					
HYPOTENSIVE					
HYPOTHERMIA					
OTHER					

CHART COPY

FIGURE 1 A reproduction of the anaesthetic record in Case 1

Case 2

The second interesting case encountered was a 39-year-old man who was rejected for army service some fifteen to seventeen years before because of hypertension.

In October, 1950, he was admitted to the Royal Victoria Hospital for investigation, with a diagnosis of Cushing's syndrome. He had a recent gain in weight and complained of general weakness, particularly of the legs, blurring of vision, pain in the back and praecordium. Objective examination revealed definite obesity of the trunk, fat pads in the supra-clavicular areas and over the upper dorsal vertebrae. Blood pressure was 195/155.

The patient was hypochondriacal and showed impairment of recent memory and of concentration. Glucose tolerance curve was 105, 213, 288, 238, 166 and 83 mg. per cent. The 17-keto-steroids and chemical corticoids were elevated, the former after ACTH. Skull X-rays were negative.

The patient refused adrenal exploration and was discharged. He was readmitted seven years later.

There was a recent weight gain of 15-20 pounds. He developed nocturia and day-time frequency associated with abnormal thirst. Blood pressure was 240/160 with a pulse rate of 100. Fundi showed vascular tortuosity, A V. nicking, and one small haemorrhage and some cotton-wool exudate were found in the right eye. Endocrine assay revealed elevated 17-keto-steroids and corticoids

Anti-hypertensive drugs had very little effect and he was scheduled for bilateral adrenalectomy February 19, 1957. He was prepared with cortisone and moderate pre-medication. No conduction anaesthesia was used. He received intravenous Pentothal, Demerol, intracheal nitrous-oxide, oxygen and a relaxant. A polyethylene catheter was inserted in the right femoral vein. His anaesthetic chart shows that all went well until adrenal manipulation commenced.

The chart shows the hypertensive responses to this part of the procedure and the

FIGURE 2 Anaesthetic record showing the hypertensive responses due to adrenal manipulation in Case 2.

incomplete reversal produced by 5 mg. doses of Regitine. Needless to say these episodes resulted in quite a distraction. As a result, inadequate attention was given to blood replacement during this period. The later hypotensive episode resulted when the inferior vena cava was accidentally injured and active haemorrhage occurred.

During the immediate postoperative period he had a prolonged apnoea following a total dose of 950 mg. of succinylcholine over a 6-hour period. Adequate tidal exchange occurred following the intravenous administration of atropine, 0.4 mg. followed 5 minutes later by 1.5 mg. of prostigmine.

In the recovery room he appeared to be in good condition. He was rousing and maintaining his airway with no apparent difficulty. We left him. Within 5 minutes he suddenly became cyanosed and apnoeic. Fortunately, a staff member was nearby and he was re-intubated at once. He was extubated 10 minutes later and was conscious almost immediately. We have been unable to explain this episode satisfactorily. Eight days later he was re-anaesthetized uneventfully for removal of a pack in the right loin incision.

During convalescence his blood pressure was extremely labile and his response to antihypertensive drugs such as ansolysen rather unsatisfactory. He complained greatly of weakness.

According to our present knowledge, bilateral adrenalectomy offers the most effective method of arresting the metabolic dysfunctions in Cushing's syndrome. Surgeons are generally in favour of a one-stage procedure. In their opinion, this removes some of the hazards from poor wound healing and the low resistance to infection occurring in these patients. Bilateral exploration may reveal adrenal hyperplasia or functioning tumours. The latter may be benign or malignant. Adrenal carcinoma metastasizes slowly and if surgical removal is early, the chances of a cure are good. In some cases of frank Cushing's syndrome, bilateral exploration has not revealed any perceptible abnormality in the adrenals.

The two cases described reflect the anaesthetic problems involved. Some cases are not so well defined and may present manifestations of hypersecretion of one portion of the cortex. For instance, the results of hyper-aldosteronism may present the important manifestations. Preoperative preparation is carefully handled by the Department of Medicine, and we rely on their presence and advice during surgery. Our problem is one of alert management. The technical procedure of anaesthesia need not be stereotyped.

These patients are always obese with poor muscle tone. There may be fluid retention. Care must be taken to avoid pressure on the eyes and skin. The thin skin and friable blood vessels may be responsible for considerable difficulty with intravenous infusions. A polyethylene catheter should be inserted after cutting down on a large vein in the leg.

Most patients have been on a diabetic regimen. Diabetic acidosis is rare. If they are receiving insulin, they are better to come to surgery with some elevation of their blood sugar. Hypoglycaemic reactions must be avoided.

Osteoporosis is common. Many of these patients have spontaneous fractures of the vertebrae. Great care must be taken to avoid trauma to the spine during posturing and during surgery.

The prone position requires controlled or assisted respirations in order to assure an adequate tidal exchange and avoid hypercarbia. Careful tracheal toilet is essential. Controlled respirations with a cuffed tube are also necessary in case the pleura becomes accidentally torn during removal of the twelfth rib.

Mushin (2) correctly advocates readiness for dealing with hypertensive crises during adrenalectomy for any reason. The second patient described here reacted dramatically during the period of adrenal manipulation. It is well to point out that phaeochromocytomas are not the only suprarenal lesions responsible for these episodes.

Phentolamine (Rogitine) is the best adrenergic blocking agent for the treatment of these reactions. The effects are reliable and the agent should be readily available.

Adrenal insufficiency is a hazard. It is difficult to understand why a patient showing many of the signs of Cushing's syndrome can be potentially adrenal deficient, but Thorn (4) has pointed out that this can be the case and such people do not respond normally to stress. Marquardt (16) has also pointed out that, in this disease, adrenal hyperplasia may be associated with atrophy of the contralateral gland. This may be the result of ACTH inhibition by the overactive gland.

Perkoff *et al.* (7) found that hypotension was more common after surgery for Cushing's syndrome than after adrenalectomy for other reasons. For this reason Latham (8) recommends large doses of cortisone for patients with Cushing's syndrome.

This form of preoperative preparation and support during surgery should be ordered by the members of the medical staff and their presence during the surgical procedure is of great value in designating the amount of hydrocortisone required.

These patients were prepared with doses of up to 600 mg of cortisone intramuscularly over a period of 60 hours before operation, followed by a final dose of 100 mg, by mouth, one hour before operation.

During operation, a continuous infusion of hydrocortisone .2 per cent was administered and continued during the recovery period. A total of 200 mg. was administered during the first twenty-four hours. This was reduced to 100 mg during the second twenty-four hours.

Hypotension is most common immediately after removal of the adrenals and, of course, may occur in the presence of adequate cortisone.

ANAESTHETIC MANAGEMENT

(a) Premedication. These patients may have a potential adrenal insufficiency. Some years ago Thorn (4) warned against the low tolerance to opiates exhibited by patients with adrenal insufficiency. It is better to use Demerol in preference to morphine premedication.

(b) Dundee (5) has stressed the necessity for care in the use of Pentothal in patients with a decreased metabolic rate. We have not noted an abnormal depressant effect due to Pentothal in these patients.

(c) Ether anaesthesia should be avoided because of the diabetic tendency of these patients.

Of interest is a fact mentioned to us by Dr Leroy Vandam recently. He has noted an increase in blood gluco-corticoid levels in volunteers during ether anaesthesia.

(d) Cyclopropane is contra-indicated because of the possibility of liberation of large amounts of endogenous adrenaline and noradrenaline.

(e) Conduction techniques are probably of value in blocking the afferent stimuli responsible for the reflex liberation of adrenergic substances and corticoids. This has been confirmed by Vandam (14). On the other hand both spinal and epidural anaesthesia may result in vasomotor instability.

Four of our patients received continuous epidural anaesthesia combined with intratracheal N_2O and O_2 . The remainder received Pentothal, Demerol, intratracheal nitrous oxide and oxygen relaxants. One patient had a prolonged apnoea following succinylcholine.

Induction of all these patients has been time consuming. After induction with anaesthesia a cut-down is performed on a large vein in the leg before posturing the patient.

Patients are postured on a special mattress to avoid interference with respiratory function. Cortisone is administered by infusion into a wrist vein.

RECOVERY ROOM CARE

Continuous postoperative care is required for these patients during this period. There are important considerations not encountered in the ordinary postoperative case. A continuous blood pressure chart must be kept and fluid balance carefully recorded.

Postoperative stability is a manifestation of adequate cortisone therapy. Under these circumstances adrenal crisis is rare (10), but should be suspected if vomiting, hypotension, hyperthermia and extreme weakness occur. Serum chemistry may not be confirmatory (7).

If this diagnosis is confirmed by medical consultation, hydrocortisone succinate, 100 mg., is administered over a four- to five-minute period. In cases of adrenal crises, prompt improvement will occur.

Postoperative haemorrhage must be watched for. Patients with adreno-cortical hyperfunction are obese, exposure is difficult and the area is vascular. The occurrence of hypotension with tachycardia and other manifestations of shock suggests possible haemorrhage. This condition was found in the second patient who was returned with a gauze pack in one incision.

Vasomotor instability may be revealed by hypotension. With adequate cortisone this is rare (10). It was not of serious consequence in any of our patients. Support with norepinephrine over a period of several hours is usually adequate.

There is a tendency towards over-zealous treatment with norepinephrine in order to maintain arbitrary blood-pressure levels. It is better to assess the patient's condition—general appearance, blood pressure, capillary refilling time and urinary output. Determining the latter may be difficult during the postoperative period of oliguria. The use of noradrenaline may present problems. After noradrenaline infusion of several hours duration, discontinuance may result in a sharp drop in blood pressure. This has been explained by Burn (11) and others as due to the ganglion-blocking effect of noradrenaline, which they have demonstrated experimentally. Cessation of the infusion decreases the vasoconstrictive effect of the noradrenaline while the ganglion-blocking effect persists.

resulting in hypotension. Burn advocates the use of vasopressin when the weaning from noradrenaline is difficult.

We have found a single intramuscular dose of 20 mg. of methoxamine of value in these cases. Methoxamine seems to maintain the vasoconstriction while the ganglion blockade resulting from norepinephrine is wearing off. Mushin (12) commented recently on this problem.

The total fluid intake during the day of surgery must be watched carefully. Over-hydration may result in hyponatremia or pulmonary oedema.

Pulmonary complications must be anticipated after prolonged surgery in the prone position with the added problems of obesity and poor muscle tone. One patient developed a right upper lobe broncho-pneumonia probably superimposed on an immediate atelectasis. The condition was suspected before he left the recovery room and responded to conservative treatment.

RÉSUMÉ

L'on n'a pas souvent l'occasion d'anesthésier des patients souffrant d'hypercortico-surrénalisme. La bonne conduite de ces cas de surrénalectomie requiert une parfaite connaissance de la physiopathologie des syndromes d'hypercortico-surrénalisme.

La cortico-surrénale produit trois genres de stéroïdes: les gluco-corticoïdes, les minéralo-corticoïdes et des hormones androgènes et estrogènes. Les principaux gluco-corticoïdes sont la cortisone et l'hydrocortisone, appelées hormones "S" à cause de leur action sur le MB des hydrates de carbone. On les appelle aussi hormones anaboliques, car elles bloquent la gluconéogenèse à partir des acides-aminés et inhibent la formation de protéines dans l'organisme, ce qui a pour résultat une baisse marquée des protéines. Les minéralo-corticoïdes régularisent le MB de l'eau et du sel. L'aldostérone est la substance la plus importante de ce groupe: elle règle la rétention de Na et l'excrétion du K. Quant aux hormones androgènes et estrogènes, elles contribuent au développement des caractères sexuels secondaires.

Il faut dire que ces actions biologiques ne sont pas aussi nettement dissociées, les androgènes agissent aussi sur la rétention du sel. D'où la difficulté de différencier les syndromes d'hypercortico-surrénalisme.

On reconnaît trois grands syndromes, correspondant à chacun des trois groupes de produits de la cortico-surrénale: le syndrome de Cushing, le syndrome adrénogénital et l'aldostéronisme primaire.

Le syndrome de Cushing vient d'une surproduction de gluco-corticoïdes. (L'injection de doses excessives de cortisone amène les mêmes résultats.) On y trouve de l'hyperglycémie, de la glucosurie, une fonte musculaire frappante, parfois de l'hypertension et de l'œdème ou, chez la femme, de l'hirsutisme, de l'acnée et de l'aménorrhée. On y trouve encore une élévation des 17-hydroxy-corticoïdes dans l'urine, parfois une augmentation des 17-kétostéroïdes, enfin une diminution du K, du Cl et du Na sanguins.

Le syndrome adrénogénital, ou virilisme surrénalien, provient d'une surproduction surrénalienne de substances gonadotropes. Il se rencontre le plus souvent chez la femme où il se caractérise par de l'hirsutisme, de l'aménorrhée, souvent

de l'obésité, de l'hypertension, une augmentation des 17-kétostéroïdes urinaires et des 17-HOGS.

L'aldostéronisme primaire, récemment décrit par Conn, provient d'un excès de minéralo-corticoïdes; il se manifeste ainsi: faiblesse, épisodes de paralysie et de tétanie due à une déficience marquée de K; hypertension apparemment secondaire à une rétention de Na, mais sans œdème.

Depuis dix-huit mois, nous eûmes à anesthésier pour surrénalectomie huit cas d'hypercortico-surrénalisme, dont cinq syndromes de Cushing et trois syndromes adrénogénitaux. La rareté de ces cas et surtout leur intérêt majeur pour les anesthésistes justifient la présentation de cet article.

Voici deux de ces cas.

Premier cas

Syndrome de Cushing suraigu. homme de 57 ans présentant polydypsie, polyurie, hypertension artérielle, faciès lunaire, obésité localisée à la face, aux épaules et au tronc, mais non aux extrémités, congestion des bases pulmonaires, peau mince, dépourvue d'élasticité, œdème à godet des jambes, orthopnée, toux productive persistante, psychose aiguë, hyperglycémie, pléthore sanguine, hypoprotéïnémie, hypercholestérolémie, augmentation des 17-hydroxy-corticostéroïdes.

A cause de l'état cardio-pulmonaire du patient et à cause de l'opération (surrénalectomie bilatérale), il eut comme anesthésie une épidurale continue associée à une anesthésie endotrachéale légère. A la fin de l'opération, une trachéotomie fut faite pour faciliter l'aspiration bronchique.

Les résidents se relayèrent pour surveiller étroitement le traitement et les suites post-opératoires, qui ne furent marquées d'aucun incident.

Deuxième cas

Patient de 39 ans admis une première fois en 1950 pour syndrome de Cushing; réadmis en 1957, obésité tronculaire, hypertension grave, augmentation des 17-kétostéroïdes, courbe positive d'hyperglycémie provoquée.

Il subit une surrénalectomie bilatérale sous anesthésie générale: Pentothal, protoxyde d'azote, curare. Une crise d'hypertension provoquée par les manipulations chirurgicales fut en partie contrôlée par des doses de 5 mg. de Régitine. A la fin de l'opération, il eut une apnée prolongée: il avait reçu 950 mg. de succinylcholine pendant une période de 6 heures. Cette apnée fut corrigée par de l'atropine 0.4 mg i-v, suivie de prostigmine 1.5 mg. après 5 minutes.

Pour résumer, les problèmes qui se posent à l'anesthésiste sont les suivants: obésité, asthénie, hypertension, congestion pulmonaire, surcharge liquidienne, fragilité des tissus, des vaisseaux et des os, crises hypertensives per-opératoires, hypotension post-opératoire, position ventrale, durée prolongée de l'intervention, nécessité d'une surveillance post-opératoire étroite et prolongée.

Dans les crises hypertensives, c'est le phentolamine (Régitine) qui agit le mieux comme sympatholytique.

L'insuffisance surrénalienne post-opératoire est toujours à craindre, car ces patients répondent mal au stress, surtout chez les syndromes de Cushing.

Ces patients reçoivent de fortes doses de cortisone avant l'opération et une solution d'hydrocortisone 0.2% pendant et après l'intervention.

Conduite anesthésique

- (1) Prémédication: Démérol de préférence à la morphine.
- (2) Induction: Pentothal, à dose prudente.
- (3) Maintien: éviter l'éther à cause de l'hyperglycémie; éviter le cyclopropane qui pourrait libérer de fortes doses d'adrénaline et de nor-adrénaline.

Les techniques d'anesthésie par conduction ont probablement de la valeur par leur action sympatholytique, mais la rachi et l'épidurale peuvent amener une instabilité vasomotrice.

Quatre de nos patients eurent une épidurale continue associée au N₂O. Les autres reçurent du Pentothal, du Démérol, du N₂O et du curare.

- (4) Soins à la salle de réveil: surveillance continue et enregistrement précis de la T.A., des liquides.

La stabilité de la T.A. sera le signe d'un traitement adéquat à la cortisone. Il faut soupçonner une déficience de ce côté, en cas de vômissement, d'hypotension, d'hyperthermie et d'extrême faiblesse. Alors, le succinate d'hydrocortisone 100 mg. en 4 à 5 minutes agit bien.

Il faut aussi avoir l'œil ouvert sur la possibilité d'hémorragie dans la plaie, à cause de sa riche vascularisation.

L'hypotension peut venir aussi de l'instabilité vasomotrice. La nor-adrénaline en goutte à goutte pendant plusieurs heures après l'opération soutiendra la T.A. Il faut cependant en restreindre la quantité car la nor-adrénaline semble posséder aussi une action ganglioplégique qui peut se manifester par de l'hypotension aigue quand on la cesse. La méthoxamine (20 mg. i-m) agit bien contre cette complication.

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