SOME ASPECTS OF ANAESTHESIA FOR HEAD AND NECK SURGERY

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IN HEAD AND NECK SURGERY, as well as in neurosurgery, prolonged tranquil anaesthesia is essential. Subdued respiratory function results from our attempts to maintain a stable anaesthetic free from reflex disturbances. Even with an airway established with the largest possible endotracheal tube, it may appear that the patient is underventilated for a considerable time. Does the resultant degree of carbon dioxide retention present a hazard, or is it tolerable, partially compensated and reversible with the anaesthetic state?

With regard to induced hypotension, can we assist the prevention of unnecessary blood loss by discreet lowering of the arterial pressure to a point that will maintain adequate perfusion of vital organs without the hazard of cellular anoxia and intravascular clotting?

In an attempt to integrate the anaesthetic techniques with the clinical results, we have reviewed the records of 125 patients presented for maxillo-facial and radical neck surgery over a three-year period. Included is a small number of laryngectomies and laryngo-fissures.

Figure 1 shows that a large proportion of these patients fell into the older age groups. You will note the incidence of either hypertension or arteriosclerotic cardiovascular disease.

TECHNIQUES OF ANAESTHESIA

These operations have all been performed under general anaesthesia and the non-rebreathing system has been used in the majority. Spontaneous respirations are maintained with minimal resistance by using one of the non-rebreathing valves (Ruben, Fink, Stephens-Slater) or a Bullough circuit. Sudden increases in pressure within the Magill circuit may obstruct the exhalation port on the Fink valve and this also occurred occasionally with the Ruben valve. Because of this hazard, a blow-off valve is inserted at the machine end of the Magill circuit. The advantage of these two types of valve, however, is that they permit the employment of controlled respirations when necessary. With this system the anaesthetic machine can be well isolated from the surgical field. With the Stephens-Slater valve, respirations cannot be controlled unless the exhalation valve is compressed by the anaesthetist's thumb.

The arrangement used in laryngectomies after preparation of the laryngeal stoma is as follows. A breathing tube fitted with Stephens-Slater valve is placed in position before the patient is draped. The Boyan tube is cold-sterilized,

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FIGURE 1

inserted into the stoma, and the cuff inflated. It is then connected to the valve under the drapes.

In a small number of cases regional nerve block was used in combination with supplementary general anaesthesia.

INDUCTION

Extra time taken to assure a tranquil induction pays handsome dividends in the form of diminished surgical haemorrhage. The acquisition of definite surgical anaesthesia is essential. It is fortified by the administration of a good topical anaesthetic nebulized through the vestibule of the larynx or injected through the cricothyroid membrane. A quiet nasal or oral intubation may then be performed. The absence of reflex disturbances avoids sudden increases in venous pressure. This in turn prevents the increased incisional bleeding that results from venous and capillary stasis. Omitting the use of a relaxant prior to intubation avoids interruption of spontaneous breathing.

ANAESTHETIC AGENTS

These patients were all maintained on a mixture of nitrous oxide and oxygen, and a non-rebreathing circuit was used in most instances.

During the preliminary stages of induction, a large number of these patients received a mixture designated as DLP which consists of meperidine (Demerol®) 100 mg., chlorpromazine (Largactil®) 50 mg., and promethazine (Phenergan®) 50 mg. in a total volume of 20 cc. Some received varying amounts of this mixture plus thiopentone and meperidine. The incidence of respiratory depression and reflex disturbances in this group was high. This situation was improved by combination with volatile anaesthetic agents. With the addition of various volatile agents the anticipated effects were noted.

With trichlorethylene, there was a high incidence of tachypnoea, not reliably controlled by intravenous meperidine.

Ether was found to be quite satisfactory in cases where the use of electric cautery or coagulation was avoided. However, the incidence of post-anaesthetic nausea and retching was higher in this group.

Halothane began to be used late in 1956 and it has since been used in the majority of cases associated with a DLP induction. We note a smooth, easy induction and a decreased incidence of laryngospasm. Capillary oozing appears to be decreased. We have paid more attention to the patient's responses than to an accurate assessment of the concentration delivered. By varying the concentration within certain limits a reversible hypotensive effect is available and with care, cardiac disturbances rarely occur. The non-inflammability is an asset and a smooth undisturbed recovery common even after prolonged administrations. Our main concern has been the extremely high cost of this agent, particularly when used in a non-rebreathing circuit.

It was interesting to consider whether or not the cost could be decreased by reverting to the use of chloroform. With this in mind, chloroform was used in a small number of cases, intending to switch to ether to avoid prolonged administration. These patients were all attached to a cardiac monitor. Despite the fact that some of us had previous experience in the use of chloroform, we were surprised that the inductions were not as placid as with halothane. The incidence of cardiac arrhythmias was high, but may have been related to an unnecessarily high concentration. In one case chloroform was discontinued because of the occurrence of shifting pacemaker, extrasystoles, and hypotension. Halothane 0.5 per cent was substituted and no arrhythmias recurred over a period of several hours. Although a more detailed study is in order, our impression is that we cannot obtain comparable safe anaesthesia with chloroform.

This general method of anaesthesia for head and neck surgery produced satisfactory practical and clinical results. The inclusion of the ataractic cocktail naturally resulted in a prolonged recovery time, but this has been quite acceptable in patients undergoing such drastic and often disfiguring surgery. A common observation was that the patient's respiratory function appeared to be subnormal. There are two schools of thought concerning breathing during operation. One group believes any form of general anaesthesia results in respiratory inadequacy and the patient is better off if paralyzed by relaxants and ventilated artificially. This may result in a purposely induced alkalosis. The other more old-fashioned school maintains that the patient may be as well if not better off breathing on his own, provided respiration is not unduly depressed.

For the purpose of this series we allowed our patients to breathe spontaneously. Ventilation studies were carried out on a number of these cases after a steady state of anaesthesia had been established and had been in progress for three or four hours. Tidal and minute volumes were measured and arterial samples taken to determine pH, $p\text{CO}_2$, and blood bicarbonate levels.

Table I shows the results obtained by Dr. Bromage in estimating the minute volume and arterial CO_2 tensions, in fourteen of these patients using a non-rebreathing system. For comparison we have included results in a comparable

TABLE I
SPONTANEOUS RESPIRATION: VENTILATION AND ARTERIAL $p\text{CO}_2$

Technique		No. of Cases	Minute Volume (L.)		Arterial pCO ₂	
			Mean	Range	Mean	Range
DLP N ₂ O	(Head and neck)	14	5.17	(3.3-8.0)	49.1	(43.0-55.0)
Epidural Thio. N ₂ O	(Abdominal)	16	5.5	(4.2-8.0)	45.0	(37.0-56.0)
End tidal pCO ₂						
Nunn's Series (1958) Thio. N ₂ O peth.		26	4.6	(1.95-9.10)	46.3	(36.5-65.5)
Robin <i>et. al.</i> (1958) Normal sleep		11	5.86	(3.87-8.41)	44.6	(41.0-49.0)

series of abdominal cases anesthetized with continuous epidural anaesthesia using a thiopentone and nitrous oxide/oxygen supplement. Recently published figures by Nunn (16) showing end-expiratory CO_2 tensions under the conditions designated, and during sleep as established by Robin (17) are also noted.

Figure 2 illustrates in a bicarbonate, pH diagram that the arterial CO_2 tensions in these patients ranged between 43 and 55 mm. Hg.

This available evidence indicates no serious degree of respiratory acidosis. Possible effects of our administrations on metabolic rates can only be speculated on. In addition, certain workers have demonstrated reductions in CO_2 production and a coincidental decrease in oxygen uptake under Fluothane anaesthesia. However, the reasons for this are by no means clear.

CONTROL OF BLOOD LOSS

A conservative attitude has seemed essential in considering the application of induced hypotension to these patients. Experiences have pointed to the potential hazards of prolonged reduction in arterial pressure, particularly as applied to patients in the older age group. Among the older patients in this series, the incidence of hypertensive cardiovascular disease was high. We had no knowledge concerning the degree of atheromatous or medial changes in their arterial systems, and preoperative blood volume determinations were seldom done. Extreme and prolonged degrees of arterial hypotension were therefore avoided. In younger patients with no evidence of vascular or cardiac disease, more active hypotensive methods were used. It was felt that a contribution could be achieved by the

RADICAL SURGERY OF HEAD & NECK : SPONTANEOUS RESPIRATION pCO₂ IN 14 PATIENTS

FIGURE 2

application of discreet arterial hypotension for a minimum period of time. The word discreet refers to the maintenance of average levels of arterial pressure in a range of between 80 and 100 mm. Hg in normotensive individuals. In patients suffering from essential hypertension only, the highest levels at which an effective decrease in arterial haemorrhage could be obtained was used.

Elevations in venous pressure are avoided by preventing sudden increases in airway pressure. These may be of mechanical or reflex origin. Posture also assists greatly in the provision of adequate venous drainage of the head and neck as well as being an aid in acquiring the optimal degree of arterial pressure.

Figure 3 shows in a diagrammatic fashion the method used.

(1) The volume of the DLP mixture given varied between 8 and 20 cc. depending on the individual response. It was usually discontinued if the blood pressure reached 90 to 100 mm. Hg or the pulse rate exceeded 96 per minute.

(2) The patient was then postured in a variable degree of a head-up tilt in an attempt to establish a stable arterial tension between 80 and 100 mg. Hg.

(3) If the blood pressure rose during the early part of the operation and arterial bleeding became more active, more hypotension was induced with the use of hexamethonium, thiophanium, or trophanium for minimal effective periods.

(4) If considered preferable, alterations in the concentration of halothane could be depended upon to induce a lesser hypotensive effect. This method appears to us to be very effective, but unfortunately we have no control series with which to compare.

FIGURE 3

Blood loss is routinely measured throughout surgery in a simple and helpful manner. We assumed accuracy within a range of 200 to 400 cc.

- (1) Sponges are weighed individually as discarded.
- (2) Suction bottles are marked off in units of 100 cc.
- (3) If any wet sponges are used, they are moistened in a known volume of saline. This volume is checked before and after the operation.
- (4) We estimate the volume of blood on the drapes.

Despite a lack of absolute accuracy, the method is helpful and better than simple guesswork. Its simplicity allows daily application even with frequent changes in operating room personnel.

COMPLICATIONS DURING ANAESTHESIA

In laryngeal cases, if a proliferative neoplasm exists in the vestibule or on one of the cords, the anaesthetist should make a point of inspecting the lesion before commencing anaesthesia. It is important to decide whether or not the lesion can be safely passed by an adequate tube. In performing a nasal intubation, traumatic epistaxis should be considered as an avoidable complication.

If there are obvious hazards to either the induction of general anaesthesia or endotracheal intubation, preoperative tracheotomy under local anaesthesia is indicated. If early tracheotomy is necessary in laryngectomies, invasion of the surgical field by obstructing tracheotomy attachments can be avoided by preparing the laryngeal stoma under local block, and inserting the special right-angled tube before the induction of general anaesthesia.

Once anaesthesia is underway, we are all aware of the problems caused by kinked tubes, a pharynx inadequately sealed off from the trachea by proper packs, or an inadequate inflatable cuff, and the frustrations caused by connections that come loose under the drapes.

Once a tranquil anaesthetic has been established, there are many routine duties to attend to. In neck cases there are two important complications to anticipate. These are: (i) disturbances due to the carotid sinus reflex, and (ii) accidental tension pneumothorax.

REFLEX DISTURBANCES

Several types of parasympathetic reflex disturbances may occur. They have not been encountered frequently in these cases. The most important reflex resulted from manipulation of the carotid sinus. In one instance a similar vagal effect resulted from pressure on the pharyngeal plexus deep to the posterior pharyngeal wall.

On three occasions the carotid sinus reflex resulted in a marked bradycardia and hypotension. In all of these instances, the routine preliminary injection of the area with local anaesthetic had been overlooked. All responded to the cessation of the stimulus and the intravenous administration of 1/100 to 1/50 of a grain of atropine.

In one instance marked bradycardia, hypotension, and respiratory depression occurred with stimulation of the active carotid sinus and the condition persisted despite the cessation of stimuli and the use of intravenous atropine. Prompt improvement resulted from the intravenous administration of methedrine 5 mg. and a subsequent carotid sinus infiltration with 1 per cent lignocaine.

A cardiac monitor fitted with a small amplifier is of value in the early identification of these reflexes.

TENSION PNEUMOTHORAX

We are indebted to Dr. Olga Schweizer and Dr. William Howland of the Memorial Center in New York for the excellent contributions concerning this complication (6). They reported ten cases occurring in their hospital in one year but did not mention the relative incidence. Akin and Smith (19) in 1952 reported eight cases with a 50 per cent mortality. Thomas and Hucks (10) reported four such incidents in thirty-seven radical neck dissections:

We have had three such occurrences in three years. The surgical literature tends to relate the occurrence of this complication to respiratory obstruction, but Schweizer and Howland's explanations are of more value to the anaesthetist. They describe the direct communication of the space between the middle and deep layers of the deep cervical fascia with the superior mediastinum. Mere exposure of the trachea or dissection of the internal jugular vein will open this space. However, the pneumothorax is caused not by the opening of the space, but by air being sucked into the mediastinum by laboured respirations. Trapping occurs with narrowing of the superior mediastinal orifice on expiration. This is shown diagrammatically in Figure 4, and this increasing pressure in the mediastinal pleura may result in a pneumothorax. Mediastinal air under tension may, of

FIGURE 4

proud, but it well illustrates the probable train of events if the anaesthetist is not alert to the possibility of this complication.

In his summary of events, the surgeon reported that as he was about to close the incision following radical neck dissection, respirations became laboured and the patient began to buck. He complained to the anaesthetist who remarked that the patient was a bit light. The surgeon proceeded, but the laboured respirations continued and he complained again. This time the anaesthetist remarked

that the tube must be too close to the carina and she adjusted it. There was still no improvement and a few minutes later respirations ceased owing to cardiac arrest. A thoracotomy was performed and within two minutes the heart responded to direct cardiac massage. At this time marked shifting of all mediastinal structures to the left was noted. The right tension pneumothorax was treated by the insertion of a tube and underwater drainage, and radiological examination revealed almost complete expansion of the right lung a few hours later. The patient was maniacal for a few days, but there was no evidence of permanent serious cortical damage afterwards. She is still alive and well.

One of our patients was chronically bronchitic and the 4-hour anaesthetic was disturbed on six occasions by the necessity to aspirate secretions through the endotracheal tube. Towards the end of operation the patient's colour deteriorated, respirations became laboured and inadequate aeration to the right chest was noted. With the establishment of underwater drainage, a considerable amount of air escaped from the chest. The patient's condition improved immediately.

The diagnosis of this complication is easy, particularly if one is aware of the possibility. Laboured respirations, bronchospasm, and a disparity between laboured respiratory excursions and minimal movement of the rebreathing bag occur. As the tension increases, the last sign becomes more obvious. The tachycardia, cyanosis, cardiac arrhythmias, and hypotension are, of course, reflections of the existing hypoxia. The clinical identification of diminished breath sounds, increased resonance, and evidence of mediastinal shift may be inconclusive. One should not wait for radiological confirmation.

Prompt establishment of underwater catheter drainage of the affected pleural cavity will usually result in the expansion of the collapsed lung within a few hours. With prompt diagnosis and proper treatment this accident should not present a serious hazard.

POSTOPERATIVE CARE

The patient should be kept under continual close supervision and nursed in the head-up position. Venous drainage of the head and neck is thereby improved and this position is also of value in the reduction of intracranial pressure.

Postoperative facial cyanosis is common but does not necessarily indicate serious venous obstruction. Schenken *et al.* have postulated that this may be a manifestation of adequate flow of blood from intracranial to extracranial areas with resultant reduction of venous congestion within the cranial vault.

Postoperative tracheotomy should be performed whenever reactive laryngeal oedema might be anticipated and when venous drainage of the neck has been affected bilaterally.

Postoperative increase in intracranial pressure is a potential hazard following removal of the second internal jugular vein or following a simultaneous bilateral neck dissection. The complication occurs occasionally despite the presence of an extensive collateral venous system. Headache, cyanosis, respiratory depression, hypertension, and papilloedema may occur and the diagnosis confirmed by the

finding of a marked increase in spinal fluid pressure. This complication did not occur in any of our cases.

SUMMARY

A non-rebreathing technique is useful in maxillo-facial and radical neck surgery because it enables adequate control with the anaesthetist and his equipment well isolated from the surgical field. With the method described there appears to be some decrease in spontaneous ventilation, but severe respiratory acidosis does not occur. Efforts to produce a moderate reduction in arterial pressure, and the avoidance of increases in venous pressure are of definite value in minimizing blood loss. Certain complications must be continuously anticipated.

In many cases postoperative tracheotomy is required to prevent respiratory obstruction in the postoperative period. The routine use of a head-up position is an important aspect of postoperative care.

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RÉSUMÉ

Au cours de la chirurgie qui se pratique sur la tête et le cou, il faut habituellement une anesthésie de longue durée et bien calme.

Pour arriver à cette fin, nous employons une technique sans réinspiration; ce système, tout en permettant à l'anesthésiste de conserver un bon contrôle sur le malade, isole l'anesthésiste et son matériel du champ opératoire. A part cela, nous suivons habituellement la ligne de conduite suivante: induction avec Phénergan, Largactil et Demerol, intubation après injection de curare si nécessaire, mais après anesthésie locale systématique; le maintien de l'anesthésie se fait avec du protoxyde d'azote ajouté ou associé à un autre agent anesthésique. Ainsi anesthésié, le malade conserve son rythme respiratoire. S'il survient de l'hypoventilation et de l'acidose respiratoire dans ces circonstances, ce n'est rien de très marqué.

Pour diminuer les pertes sanguines, une légère hypotension artérielle est toujours efficace de même qu'une tension veineuse dans les limites de la normale.

Il peut survenir certaines complications, telles: l'obstruction des voies respiratoires par une tumeur, un réflexe du sinus carotidien ou un pneumothorax. L'anesthésiste doit essayer de prévenir ces ennuis et les diagnostiquer précocement s'ils surviennent.

Au cours des suites opératoires, il s'impose de laisser la tête élevée. Dans bien des cas, comme mesure préventive de l'obstruction respiratoire, nous suggérons fortement la trachéotomie.

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