with mandelic acid, sulphanilamides should be continued for a week after the urine has become sterile, otherwise relapses may occur.

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ANAESTHESIA IN DENTAL SURGERY*

BY

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The majority of dental operations in this country are performed in the dental surgery under nitrous oxide anaesthesia on ambulant unpremedicated patients. In most cases this agent affords a pleasant and rapid induction, quick recovery, and freedom from after-effects. Ninety per cent. are managed satisfactorily, but the remainder present difficulties, mainly on account of the weak anaesthetic properties of nitrous oxide. Discussion of the conduct of anaesthesia for major dental operations and the special problems of anaesthesia for dental work in children will not be undertaken in this brief review, which deals with the problems arising in the 10 per cent. of patients who prove difficult under nitrous oxide anaesthesia in the dental surgery. Later, cyanosis, anaemia, and respiratory obstruction are discussed, since it is felt that a fuller appreciation of the underlying physiology and pathology of these conditions will make for smoother and safer anaesthesia.

The patient may present difficulties on account of his psychological approach to anaesthesia or because of resistant physical qualities which are difficult to overcome with a weak anaesthetic such as nitrous oxide.

Psychological Considerations

The problem here is to prevent disturbances during induction. The energy for resistance is dissipated early during anaesthesia, so that maintenance is no more difficult than in the average patient. The prospect of being rendered unconscious is not a pleasant one; addiction to anaesthesia is unknown! In the great majority of adult patients this natural apprehension is not sufficient to disturb the even course of anaesthesia. Two classes need special attention—one the undisciplined with poor self-control; the other the basically nervous but superficially controlled patient, who typifies the "stiff upper lip" school and who may not betray any mental disturbance until well after induction is begun. This first type is

uncooperative from the beginning, but the patient whose training has enabled him to mask his fear by rigid self-control presents a different problem. The early part of induction is smooth, but as soon as conscious control is lost his basic fear is manifested by struggling.

For both these types premedication is valuable. It varies from reassurance, the patient's own particular fear being dealt with, to the use of sedative drugs. These should not depress the patient to such an extent that he is unable to leave the surgery within a reasonable time. Simple but effective premedication is 10 grains of aspirin the night previously, and this dose repeated two hours before the operation. If the patient dislikes induction by inhalation it can be avoided by giving a small intravenous dose of barbiturate—for example, 0.2 gramme of evipan or pentothal—and continuing anaesthesia by nasal administration of nitrous oxide. Small doses of pentothal are rapidly eliminated, so that recovery is scarcely if at all longer than after nitrous oxide alone.

Physical Considerations

Certain types of patients are notoriously difficult to anaesthetize, and their resistant qualities are most evident when a weak anaesthetic such as nitrous oxide is used. Although the anaesthetic-resistant patient is usually robust and often hard-living, occasionally a small wiry man displays a resistance which his appearance belies. Resistance to anaesthetics is markedly increased by alcoholism or any other drug addiction.

Nitrous oxide is the weakest of all anaesthetics. If it is to be used successfully for the anaesthetic-resistant patient its weak action has to be reinforced by reducing the oxygen intake to levels where asphyxial signs appear. There is only a narrow margin of anaesthesia between response to a surgical stimulus and asphyxial spasm. An attempt to subdue a difficult patient with nitrous oxide alone results in anoxial muscular spasm which may make operating difficult. If the anaesthetist is unwise enough to persist, spasm of the adductor muscles of the larynx may follow, with resultant respiratory obstruction. Artificial respiration or inflation of the lungs is then impossible because of the tightly closed glottis. The situation is alarming. Since muscular rigidity passes off before death, artificial respiration must be continued so that the lungs will be immediately expanded on relaxation of the spasm. This state of affairs can be avoided by combining the nitrous oxide with a more potent anaesthetic which will allow the introduction of enough oxygen to avoid production of anoxial spasm. The most useful drugs for this purpose are the barbiturates, given intravenously, or ethyl chloride. Anaesthesia should be induced by the former in cases where it is considered improbable that nitrous oxide will be adequate. The extra time involved in the preparation of a sterile intravenous injection is amply justified by the results. If the operation has been started with nitrous oxide and the patient unexpectedly proves to be unmanageable it is technically impossible to give an intravenous injection, and the supplemental use of ethyl chloride is of value because of its potency, ease of administration, and rapidity of action.

If it is decided to reinforce nitrous oxide by the use of a barbiturate, a small dose (for example, 0.2 to 0.4 gramme) of evipan or pentothal injected rapidly will produce unconsciousness. A large proportion of the drug is carried to the brain by the vast carotid circulation, causing immediate unconsciousness. Anaesthesia is continued at once by nitrous oxide administered nasally. Provided a clear airway is maintained the barbiturate can

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be disregarded, since its effect, given in these doses to this type of patient, is transient. In the course of a very short time its distribution from the brain to tissues with a less abundant blood supply results in a lightening of anaesthesia. If the barbiturates are slowly injected a larger dose is required to produce the same depth of anaesthesia, and recovery is correspondingly delayed. Rapid injection is recommended only when small doses of the drug are used. The small amount of barbiturate given will afford a pleasant induction and will increase the margin of anaesthesia. The recovery period is scarcely longer than after nitrous oxide used alone, being in marked contrast to the slow recovery after large doses.

When ethyl chloride is necessary to deepen the effect of nitrous oxide it should be sprayed on to the bib turned up over the mouth. If the patient is mouth-breathing, anaesthesia deepens rapidly and, even if nose-breathing, enough of the vapour reaches the pharynx to augment the action of the nitrous oxide. Application of ethyl chloride must stop at the onset of automatic respiration, and anaesthesia is continued with nitrous oxide and enough oxygen to avoid cyanosis, the import of which is considered later. If during the operation the patient shows signs of returning consciousness, anaesthesia should be deepened again by giving ethyl chloride as before. Preparatory to this care is taken to see that the mouth is free from debris and a fresh sponge left in position. Small supplemental doses of ethyl chloride seldom cause post-operative nausea in this type of patient, but vomiting and prostration may occur if deep or prolonged anaesthesia has been attained mainly by the use of ethyl chloride.

Cyanosis

Before dealing with the anaemic patient the significance of cyanosis must be considered. At the outset it can be stated that when it occurs during nitrous oxide anaesthesia its presence is not necessarily a sign of danger or its absence an assurance of safety. With other anaesthetics cyanosis is a sign of respiratory obstruction or arrest. With nitrous oxide, as a result of essential reduction in the oxygen intake, its occurrence in the average case is inevitable.

The discussion of cyanosis is facilitated by classifying patients according to the haemoglobin content of the blood, which in an average case is taken as 15 grammes per 100 c.cm. This amount will be greater for the plethoric patient, but in severe anaemia it may be as low as 5 grammes per 100 c.cm. Cyanosis can occur only if the absolute amount of reduced haemoglobin is 5 grammes or more per 100 c.cm. of blood. Inhalation of a mixture containing less oxygen than in the air results in diminished alveolar oxygen tension and hence incomplete oxygenation of the circulating blood. A definite proportion of the haemoglobin is oxygenated irrespective of the total amount present. This is shown in the well-known association-curve of haemoglobin.

To illustrate the application of these facts, the effect of inhalation of nitrous oxide mixed with enough oxygen (about 10 per cent.) to oxygenate two-thirds of the circulating haemoglobin may be considered. In the plethoric patient with, say, 18 grammes of haemoglobin per 100 c.cm. of blood, 12 grammes will be oxygenated, 6 grammes remaining in the reduced form. In a normal patient having 15 grammes of haemoglobin per 100 c.cm. of blood, 10 grammes will be oxygenated, 5 grammes remaining reduced; while in an anaemic patient with only 6 grammes of haemoglobin per 100 c.cm. of blood, 4 grammes will be oxygenated, leaving 2 grammes reduced. There is thus definite cyanosis in the plethoric individual, mild cyanosis

in the normal patient, and no change in the colour of the anaemic patient.

Because nitrous oxide is carried in solution in the blood plasma the concentration of this agent is the same in the three types discussed. Of these the anaemic patient, despite the fact that he is not cyanosed, will be the most depressed, since the oxygenation of his blood is lowest. In contrast to this the plethoric man, although cyanosed, will hardly be unconscious, since he has still a large amount of oxyhaemoglobin circulating. From this the unreliability and danger of using cyanosis as a sign of depth of anaesthesia with nitrous oxide is obvious. The occurrence of cyanosis in advanced anaemia is impossible, and the anaesthetist will find that during deepening of nitrous oxide anaesthesia in such a patient there is no colour change. Respirations become more and more shallow, and the patient will in fact "fade away" and die without any suggestion of cyanosis. On the other hand, with a plethoric patient, if the anaesthetist regards cyanosis as a sign of anaesthesia he may allow the operator to begin before the patient has even lost consciousness.

The Anaemic Patient

The anaemic patient is particularly susceptible to the action of any anaesthetic, or oxygen-lack. It is often erroneously stated that nitrous oxide is unsuitable for such a patient. On the contrary, a weak anaesthetic is obviously indicated for the type of patient easily depressed by narcotics. It is essential to appreciate that cyanosis, muscular rigidity, or jactitation, which are frequently seen at some stages of nitrous oxide anaesthesia in an average or robust patient, are completely absent if the degree of anaemia is severe. The anaemic patient is easy to anaesthetize, and danger lies only in interpreting the absence of cyanosis and muscular signs as proof that anaesthesia is not deep. Properly handled, nitrous oxide can be the safest anaesthetic for this type, but if handled carelessly it can be as rapidly lethal as any other, causing death from asphyxia, without the physical signs usually associated with this condition.

Anaesthesia in Laryngeal Obstruction

Oedema of the larynx may follow extension of any neighbouring inflammatory process. In dentistry this may be encountered in a patient with an inflamed impacted wisdom tooth. Clinically it is revealed by signs of partial respiratory obstruction. When conscious the patient increases his normal inspiratory effort by use of the accessory muscles of respiration. He is thus able to expand his lungs despite the obstruction produced by the narrowed laryngeal opening. These accessory muscles are under conscious control, and their action ceases with the loss of consciousness. Without the extra power afforded by this voluntary act the automatic respiratory muscles alone are not sufficient to draw the air through the narrowed glottis, respiratory efforts become ineffective, and, unless immediate tracheotomy is performed, the patient will die of asphyxia.

If the patient is dyspnoeic and is using his accessory muscles of respiration, death will follow the production of deep unconsciousness by any means. Such fatalities are only too frequent and, since the underlying pathology is not appreciated, they are generally attributed in error to the particular anaesthetic used. If the accessory respiratory muscles are in action, unconsciousness should not be produced unless tracheotomy is first performed under local anaesthesia. This preliminary tracheotomy removes the respiratory obstruction and with it any contraindication to the use of general anaesthesia.

Obstruction of the Airway during the Operation

In an unconscious patient the possibility of obstruction of the airway by the tongue must be borne in mind. With an unconscious patient in the dental chair, the tongue is not as likely to fall back and occlude the laryngeal opening as when he is recumbent, but it may be pushed back inadvertently by the dentist. It is the anaesthetist's duty to keep the airway clear, but he may, in practice, wittingly allow obstruction of this type to persist unrelieved in a fit patient for five to ten seconds if it assists the operator in a difficult extraction. The anaesthetist should, however, appreciate that the anaesthetic is being complicated by respiratory obstruction and consequent anoxaemia, and if this time is exceeded he must insist on co-operation from the dentist in re-establishing a clear airway. The condition is most likely to occur during extractions in the lower jaw. With his non-extracting hand the dentist is inclined to obtain purchase by depressing and pushing back the lower jaw, and with it the tongue. To prevent this type of obstruction the anaesthetist should exert counter-pressure by pushing the lower jaw firmly forwards from the angle. The dentist should also assist by pulling the lower jaw forwards and upwards with his non-extracting hand instead of pushing it backwards and downwards.

From the nature of the work the danger of respiratory obstruction is ever present in dental surgery. An operation in the mouth should not be performed without a sponge attached to a tape in position to safeguard the larynx from obstruction by blood or debris. A sponge should never be left in the mouth unless a tape is attached to it. If it becomes soaked in blood or mucus it can obstruct respiration by slipping into the larynx, and without an attached tape may be very difficult to remove. A soiled sponge must be substituted by a fresh one as often as necessary.

The first essential in anaesthesia for any type of surgery is the maintenance of a clear airway. Neglect of this rule is the most fruitful cause of lack of smoothness in anaesthesia, and of danger. Complete protection of the air passages during dental work can only be afforded by endotracheal anaesthesia, which assures a clear airway and enables packing-off of the pharynx to be perfect. For practical purposes, however, this is advisable only for major dental operations, which should not be undertaken in the dental surgery, but in a hospital or nursing home.

Summary

The problems of nitrous oxide anaesthesia for the ambulant unpremedicated dental patient are discussed, and the psychological and physical factors which affect the course of anaesthesia are dealt with.

Methods of extending the scope of nitrous oxide in resistant cases by the supplementary use of intravenous barbiturate or of ethyl chloride are described.

The effects of nitrous oxide in anaemic and plethoric patients are reviewed, and the significance of cyanosis is discussed.

The special problem of anaesthesia for a patient with respiratory obstruction due to oedema of the larynx receives attention, and the danger of general anaesthesia for such a case without preliminary tracheotomy is stressed.

Prevention of the common causes of respiratory obstruction during operation is reviewed.

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THE CEREBROSPINAL FLUID IN ANTERIOR POLIOMYELITIS

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During the outbreak of anterior poliomyelitis in Swansea and the neighbourhood in 1938 we had occasion to examine fifty-five cerebrospinal fluids taken from fifty-three suspected cases. These were all subjected to enumeration of cells, differential count, and full chemical analysis, and the following points emerge from a general survey of the examinations correlated with the clinical aspects. The cases examined come under the following groups:

A. Poliomyelitis with definite paresis before lumbar puncture	16
B. Limb weakness and loss of reflexes at the time of lumbar puncture, subsequently diagnosed as poliomyelitis	15
C. Cases later found to have poliomyelitis	2
D. Fatal cases proved histologically to have poliomyelitis (post-mortem samples of fluid)	2
E. Illness of doubtful origin	3
F. Cases ultimately diagnosed as having other diseases	11
G. Recovered after a short period as suspects, no illness materialized	4

In summary, there were thirty-five cases definitely shown to have poliomyelitis, three probably mild cases, four false alarms or abortive cases, all suffering from other diseases.

Analysis of Cerebrospinal Fluid

Analysis of the fluids in Group A showed the following characteristics: an increase of cells with an average of 51 lymphocytes and 6 polymorphs per c.mm., and a range of 4 to 250 and 0 to 31 per c.mm. respectively; an increase in the protein value of range 10 to 100 mg. per 100 c.cm., with an average of 41 mg. per 100 c.cm.; the presence of globulin; and a definite though small Lange curve, with a maximum at Tubes 4 and 5 and seldom rising above the level of 2. There was a fine fibrin clot in five of the cases. The glucose and chlorides in all the fluids were within the normal limits.

In Group B the findings were similar but the total cell count was somewhat higher and the number of polymorphs decreased, the ranges being 3 to 225 and 0 to 9 per c.mm. of lymphocytes and polymorphs, with averages respectively of 78 and 2 per c.mm. The average protein was 48 mg. per 100 c.cm., with a range of 30 to 95 mg. per 100 c.cm., and globulin was present in all the fluids; the Lange curves resembled those of Group A but shifted slightly to the right, the maximal tubes being 5 and 6. Clot occurred in six of the fluids. The glucose and chlorides were within normal limits. It is of interest to note that those fluids with the lower protein value all had a definite increase in cell count.

The cerebrospinal fluids from the two cases in Group C had the same characteristics as those of the first two groups. The two fluids taken post mortem, Group D, had similar changes, but there was a higher degree of