THE EFFECTS OF EXPIRATORY RESISTANCE, OXYGEN AND HYPOXIA IN RELATION TO DENTAL ANAESTHESIA

PART I: INTRODUCTION

By

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SUMMARY

The establishment of the safety of anaesthetic techniques which might be expected to reduce cerebral and cardiac oxygenation requires the support of experimental and clinical evidence. This article outlines the factors which should be considered and reviews some of the literature with particular reference to hypoxia and cardiovascular depression. It serves as an introduction to an experimental study designed to explore the effects of expiratory resistance and variations in inspired oxygen concentration.

During anaesthesia in the dental chair, adequate cerebral and cardiac oxygenation are essential for safety. Anaesthetic techniques which might reduce the oxygenation of these vital tissues can be justified only if their safety is established by clinical and experimental evidence. Some evidence which suggests that traditional dental anaesthetic techniques may not be without danger is reviewed in this article. Part II describes a study designed to explore the effects of expiratory resistance and variations in inspired oxygen concentration upon the systolic blood pressure, pulse rate and respiration in conscious sitting subjects. This was undertaken as a preliminary to a subsequent investigation of the same factors during dental anaesthesia.

Cerebral and cardiac oxygenation are dependent upon the tissue blood flow, the blood oxygen content, respiration, alveolar-capillary gas exchange and the partial pressure of oxygen in the inspired gases. The study of these variables during routine dental anaesthesia is difficult, and some cannot be measured directly. Cerebral blood flow, for example, cannot be measured under these circumstances but, in the sitting position, it can be expected to follow variations in blood pressure which can be measured.

Arterial blood pressure during dental anaesthesia.

Arterial blood pressure is a factor which the dental anaesthetist may find difficult to assess and over which he has little direct control (except by tilting the chair to the horizontal position in the event of a fall in blood pressure). Goldman, Cornwell and Lethbridge (1958) and Walsh (1958) maintained that the usual tendency is for the blood pressure to rise during dental anaesthesia, although it was admitted that it might fall slightly at the end of anaesthesia, upon the introduction of oxygen, and occasionally during induction. Bourne (1957, 1960), on the other hand, emphasized the dangers of fainting in the sitting position, particularly when this coincides with hypoxia.

The incidence and causes of hypotension during dental anaesthesia are not clear. Many of the possible causes have been reviewed by Bourne (1960). Emotional factors may be concerned when hypotension occurs before or during the early stages of anaesthesia (Sharpey-Schafer, 1958) but this explanation is less convincing when hypotension occurs towards the end of, or following, anaesthesia.

Many factors may contribute towards changes in blood pressure during dental anaesthesia and the sudden reversal of an environmental change may be of as much importance as its introduction. Some of the more obvious considerations are: the pharmacological properties of anaesthetic agents; movement and struggling; surgical stimuli during light anaesthesia; hypoxia, pre-oxygenation and re-oxygenation; the application and the removal of raised airway pressures due to the use of high flow rates and of apparatus with high resistance; the nature of the surgical procedures and the
duration of anaesthesia. Factors less easy to define are those concerning the individual patient. Apart from race, sex, age, physique, disease, alcohol or drug tolerance, and individual patterns of emotional, autonomic, humoral and vasomotor control, the intensity and duration of pre-operative pain, the recent quota of sleep, the time of the last meal, previous exercise and activity, and the heat and humidity of the day may all play a part.

Apart from the difficulty in investigating such a variety of factors which may act alone or in combination and have different relative effects in different people, it should be appreciated that in assessing the safety of any anaesthetic procedure the reaction of the atypical individual is important as well as that of the typical. To give an example from dental anaesthetic practice, Bourne (1957, 1960) concluded that there were about 500 cases per year in the United Kingdom of delayed recovery of consciousness of half an hour or more following dental anaesthesia. If the total number of anaesthetics administered per annum be taken as 2,000,000 then one such delayed recovery might be expected every 4,000 dental anaesthetics. This gives some idea of the size of the problem.

The effects of hypoxia.

Schneider (1918) subjected seventy fit male volunteers to progressively decreasing inspired oxygen concentrations during rebreathing experiments lasting up to half an hour, and found that the occasional subject suddenly breathed less and then fainted. Some subjects responded to the increasing hypoxia with an initial acceleration of the pulse rate but then, after maintaining a constant rate for a while, the heart suddenly slowed and this was a sure indication that the limit of endurance had been reached. The change in systolic blood pressure in response to hypoxia was variable. In what Schneider described as the "optimal" type of response, the systolic blood pressure remained unchanged until the inspired oxygen concentration had fallen to between 14 and 9 per cent; the blood pressure then rose gradually. Some subjects inhaled mixtures containing as little as 6.6 per cent oxygen before the systolic blood pressure rose. In other cases the systolic blood pressure rose by 40 to 60 mm Hg. A few subjects showed a fall in systolic blood pressure at about the time that the pulse rate began to rise and these were less tolerant of low inspired oxygen concentrations. Many subjects showed a sharp and sudden fall in systolic pressure at low inspired oxygen concentrations and this, if allowed to continue, led to fainting. Normal blood pressures were usually recovered on return to air, although after fainting full return to normal might take up to 1 or 2 hours.

It is worth noting that if a man were even slightly below the best physical condition, his tolerance of hypoxia was reduced. A cold, indigestion, late hours or worry might reduce his resistance to hypoxia temporarily by the altitude equivalent of many thousands of feet. More serious indisposition affected him to a corresponding extent. Caution should be exercised, however, in interpreting these results in relation to the hypoxia which may occur during dental anaesthesia. The two sets of conditions are not the same. In Schneider's experiments hypoxia was introduced gradually and it was progressive and prolonged. During dental anaesthesia the introduction of hypoxia is usually sudden and often profound, but it should not be prolonged and it is usually progressively reduced. Keys, Stapp and Violante (1943), however, reported fainting in ten subjects between 2.8 and 15 minutes after starting to breathe about 10 per cent oxygen. The possibility that hypoxia may be one of the causes of hypotension in the dental chair deserves serious consideration. It was expected by Sharpey-Schafer (1958) but not proved.

Hypoxia and tourniquets.

Anderson and his colleagues (1946) investigated the circulatory changes occurring during fainting induced by oxygen lack. Inspired oxygen concentrations between 6 and 10 per cent were used and faints were induced in three out of thirteen subjects by this means. The charts of their results indicate that fainting did not occur until after about 5 minutes from the start of hypoxia but these subjects lay on a couch with their backs supported at about 45 degrees. Their legs were not dependent, although one volunteer subsequently fainted much sooner when tourniquets were used to dam back blood in his legs. His pulse rate fell to 58 and blood pressure to 80/65 mm Hg. He was breathing 8.4 per cent oxygen. Inhalation of this hypoxic mixture was continued,
yet within 2 to 3 minutes his blood pressure began to rise again. Five other subjects also recovered from fainting under similar circumstances. This suggests the possibility of a patient both fainting and recovering from the faint while still anaesthetized in the dental chair.

The effects of re-oxygenation following hypoxia.

Schneider (1918), commenting upon the significance of hypoxia in relation to aircraft accidents, observed: “In another class of accidents, or near accidents, the pilot’s failure occurs near the end of a rapid descent. Often he has no recollection of how the crash occurred; he had lost consciousness, was asleep. This is highly significant; for this is a state which is induced by ample supply of oxygen, or fresh air at full pressure, after a period of deprivation. This interval of unconsciousness, or collapse, is often met by students of low oxygen problems. It occurs in a wide variety of conditions, in miners escaping from ‘after damp’, city firemen coming out of smoke, and low oxygen experiments in the laboratory.”

This effect has since been called the “oxygen paradox”. Latham (1951) has reviewed the relevant literature and investigated it further. He demonstrated the paradox reaction in thirteen out of fifty-two subjects. It began between the 9th and 12th seconds after starting to breathe oxygen following a period of hypoxia. The initial effect was a disturbance of the central nervous system as shown by an increase in writing test errors. A secondary rise in the number of test errors written coincided with cardiovascular changes which were most pronounced after about 30 seconds of oxygen breathing. There was sometimes a sharp fall in systolic pressure within the first 2 to 4 seconds which Latham explained by an increase in the capacity of the pulmonary capillary bed causing a momentary reduction in cardiac filling. A greater fall in blood pressure was recorded after about 30 seconds and this coincided with an increase in forearm blood flow. The forearm blood flow usually began to increase at about the 9th or 10th second, reached a maximum at about 35 seconds, and had returned to normal within 60 seconds. There was a significant change in forearm blood flow during the preliminary period of hypoxia. Latham suggested that a constitutional phenomenon is terminated by a cardiovascular collapse.

It may be relevant to repeat here that Goldman, Cornwell and Lethbridge (1958) sometimes found a slight fall in blood pressure at the end of anaesthesia and upon the introduction of oxygen. They did not record any dramatic changes, but their series was small. It may also be highly significant in this context, that the majority of the cases of death in the dental chair of which details were given by Bourne (1957, 1960) also occurred at the end of anaesthesia, and two of these patients went pale when oxygen was administered.

Anaesthesia as a protection against fainting.

It may be objected that fainting does not occur during anaesthesia. de Wardener and associates (1953) found that controlled haemorrhage sufficient to trigger the fainting reflex in a high proportion of conscious subjects did not produce fainting in any of fourteen patients under light cyclopropane or light ether anaesthesia during operations on varicose veins. They suggested that the fainting reaction could be prevented by anaesthesia. If this is true, then it might possibly provide an explanation for some of those cases in which fainting, whatever its cause, appears to have been postponed until the end of dental anaesthesia.

On the other hand, it should be remembered that it takes an appreciable time to obtain the full effect of nitrous oxide, and that early loss of consciousness during a nitrous oxide induction may be produced as much by hypoxia as by the anaesthetic properties of nitrous oxide. It is possible that there may be no protection against fainting under these circumstances. Furthermore, hypotension with bradycardia, whatever its mechanism, can occur as a result of surgical stimulation during nitrous oxide, oxygen and relaxant anaesthesia.

The carotid sinus.

Burstein (1949) suggested that application of digital pressure in the carotid sinus area when supporting the jaw might result in bradycardia and hypotension. External pressure on the carotid sinus could easily occur during dental extraction.

The effects of external resistance to respiration.

Some of the literature relevant to the effects of external resistance to respiration has been
reviewed elsewhere (Smith, 1961). While considering the safety during dental anaesthesia an observation of Barach and his colleagues (1947) is worth recalling. They reported a syncopal reaction in a young male subject while breathing against a pressure of 20 cm H₂O (14.7 mm Hg), without hypoxia and while wearing a compensating vest, although the duration of the pressure breathing before syncope occurred was not mentioned. Pressures of this magnitude may be approached for short periods during dental anaesthesia when the pressure in the mask is deliberately increased, with the intention either of deepening anaesthesia or of preventing dilution with air (Rolleston and Moncrieff, 1938; Minnitt and Gillies, 1945; Clement, 1951). Consideration should be given to the possibility that even lower mask pressures imposed for short periods may have similar effects when combined with hypoxia, or with any of the other possible contributory causes of fainting in the dental chair.

REFERENCES

SOMMAIRE
L'évaluation de la sécurité d'une technique anesthésique dont on peut attendre une réduction de l'oxygenation cérébrale et cardiaque doit être basée sur des preuves expérimentales et cliniques. Cet article souligne les facteurs qu'on doit prendre en considération et analyse une partie de la littérature spécialement en ce qui concerne l'hypoxie et la dépression cardiovasculaire. Cet article sert d'introduction à une étude expérimentale qui se propose d'explorer les effets de la résistance expiratoire et les variations de la concentration de l'oxygène inspiré.

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