CLINICAL OBSERVATIONS
UPON
RESPIRATION DURING ANÆSTHESIA;
WITH
SPECIAL REFERENCE TO THE CAUSES OF EMBARRASSED
AND OBSTRUCTED BREATHING.

BY
FREDERIC HEWITT, M.A., M.D.CANTAB.,
INSTRUCTOR IN ANÆSTHETICS AT THE LONDON HOSPITAL AND ADMINISTRATOR
OF ANÆSTHETICS AT CHARING CROSS HOSPITAL.

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Introduction.

So much has lately been written concerning anæsthetics and their physiological effects, that I feel the greatest diffidence in bringing the following observations before this Society—a diffidence which is the greater when I reflect that the Royal Medical and Chirurgical Society has itself done much important work in this particular department of medicine. At the present moment, too, the Report of the Hyderabad Chloroform Commission is before us, and I am afraid it may appear almost presumptuous on my part to submit any facts or theories to the consideration of the Society so soon after the publication

of the important conclusions of that Commission. But the remarks I am about to make are solely the outcome of clinical as opposed to experimental work. It may be said that our store of knowledge concerning the effects of the anæsthetics, with which we are now acquainted, may be increased in two ways: firstly, by direct physiological experiment; and secondly, by clinical observation. With regard to the former mode of progress, I have unfortunately nothing to say. With regard to the latter, however, it seems to me that every one who is specially engaged in administering anæsthetics should endeavour, by making known the symptoms which he is in the habit of observing under particular conditions and circumstances, to contribute to the solution of the problems with which we are at the present time surrounded. The Hyderabad Commission has done a most important service in drawing attention to the necessity for carefully watching respiration during anæsthesia. I do not here propose to discuss the conclusions of the Commission; time itself will be the best judge of their correctness. That respiratory embarrassment or failure frequently precedes cardiac depression under chloroform there can be no doubt; but that such a sequence of events invariably occurs is still, I venture to think, an open question. Apart from this point, however, we must all admit the necessity of maintaining free respiration during anæsthesia; and it is with the hope that some light may be thrown upon the nature and causes of those modifications in respiration, which occur during anæsthesia in the human subject, that I venture to place the following observations on record. In order to avoid respiratory difficulties it is, in the first place, essential that the nature of those difficulties should be understood. I think it will be admitted that there are many conditions in the human subject capable of modifying respiration, which have no true counterpart in the lower animals. Some patients are said to take anæsthetics badly, whilst others are said to take them well. Generally speaking a patient is said to take an anæsthetic badly when the

respiration becomes embarrassed or impaired during the administration. It, therefore, seems to me a matter of the greatest interest and importance to attempt to discover some reasons for the numerous alterations and modifications in respiration which may be met with in actual practice. I am aware that such an attempt is, in the present state of our knowledge, unlikely to be attended by much success. Still, there can be no doubt that if we could discover the reasons of those phenomena, which we from time to time meet with, more especially in difficult and exceptional cases, we should be acquiring information, which might possibly be of future service in preventing those fatalities under anæsthetics, which we are all so anxious to avoid.

The respiration at any particular moment during anæsthesia for a surgical operation will be found to be influenced by one or more of the following considerations :

1. *The anæsthetic employed.*
2. *The method of its administration*, the most important factors in this connection being ; (a) *the rate of administration*, and (b) *the degree of air-limitation permitted.*
3. *The depth of anæsthesia*, for example, the act of deglutition, which cannot occur in profound narcosis, often modifies respiratory rhythm in the earlier and later stages of the administrations.
4. *The nature of the operation*—during the performance of certain operations, such, for example, as ligaturing hæmorrhoids, respiration is sometimes temporarily interfered with by reason of reflex spasm of the laryngeal and other muscles, a condition more especially likely to arise when the anæsthesia is only moderately deep.
5. The presence of any *pre-existing or accidental impediment within the air passages* to the free entry or exit of air ; these will be particularly considered later on (see § 1 and 2).
6. The presence of any other condition capable of hindering free expansion of the lung, such as extreme obesity, abdominal distension, pleural or pulmonary diseases,

senile changes in the framework of the chest, tight lacing, &c.

7. *The activity of the nervous mechanism of respiration.*

The above list very imperfectly represents all the circumstances which may influence respiration during anæsthesia. I have, for example, purposely omitted to refer to the effects upon the respiratory centre of impulses passing along the vagi or other nerves, feeling that any allusions to such points would at once destroy the object of the present paper, which is to consider the practical, rather than the physiological, side of the subject.

§ 1. *On the chief Morbid Conditions capable of prejudicially affecting Respiration during Anæsthesia.*

Any pre-existing inadequacy in, or impediment to, respiration should be carefully investigated before administering a general anæsthetic. Moderate or occasional dyspnœa prior to the administration is, for many reasons, liable to pass into considerable difficulty of breathing during anæsthesia, and there are undoubtedly certain cases in which no anæsthetic should be given. Morbid growths within the upper air-passages may increase in size by reason of greater vascularity, principally of venous origin. Such growths, moreover, may, if pedunculated, alter in position, and so give rise to sudden symptoms of an asphyxial character. Should nasal polypi or other formations be present to such an extent as to partially obstruct the nasal air-way, it will often be found that when the patient is deeply under the anæsthetic the nasal obstruction becomes complete from vascular turgescence of the growths, and oral breathing has to be maintained. I have observed a similar result in ordinary nasal catarrh. It is sometimes necessary to give an anæsthetic to a patient in whom considerable and possibly long-standing dyspnœa, depending upon some obstruction within the larynx or trachea, is present. Such cases require special notice. When extraordinary muscles of respiration have been

brought into play in the course of any disease, producing a narrowed air-way, the administration of an anæsthetic may, by throwing such muscles out of action, readily lead to arrested breathing. This event has been observed in cases of aneurysmal pressure upon the trachea, three fatalities having been recorded. In each of these respiration came to a final standstill after but a small quantity of the anæsthetic had been cautiously administered. Respiratory embarrassment from morbid or senile changes within the bronchi, lungs, pleuræ, or thoracic framework is not likely to eventuate in any serious conditions if due care be exercised. In order to maintain respiration satisfactorily under these difficulties three points must be borne in mind. The anæsthesia should never be very profound; a free supply of air should be allowed, and every means should be employed to maintain a good and efficient action of the heart. Those cases would appear to be the most unfavourable in which the respiratory embarrassment is of recent origin, and is, in part, dependent upon the thorax or abdomen (as the case may be) having become useless for respiratory purposes. The effects which impeded or inadequate respiration quickly produce upon the circulation, more especially if the latter be feeble, will be considered in § 4. Before quitting this branch of the subject, however, I would refer to a curious state of the respiratory mechanism which has been noticed in certain cases in which during anæsthesia breathing has come to a standstill, owing to the presence of some pre-existing morbid condition. There has been observed a peculiar rigidity and fixity of the thoracic parietes resisting all artificial attempts at expansion, even though no obstruction between the lungs and external air existed at the moment when such attempts were being made. Two cases of this kind are on record (see 'Brit. Med. Journ.,' March 15th, 1884, p. 508, and May 2nd, 1885, p. 887). At one of these reported by Mr. Holmes I happened to be present, and was able to watch the symptoms which occurred. In each case an enlarged thyroid gland pressed upon the

trachea ; respiration became embarrassed under ether (probably from increase in size of the thyroid), and eventually ceased ; tracheotomy was performed, and although by this operation an air-passage to the lungs was established, no efforts were successful in producing expansion of the chest, which appeared to be rigidly fixed in consequence of spasm of the expiratory muscles. In the case published by Mr. Holmes the heart's action continued for a considerable time whilst attempts were being made to expand the chest.

§ 2. *On the chief Accidental Obstructions and Impediments to Respiration during Anæsthesia.*

During anæsthesia the entry and exit of air to the lungs may become temporarily interfered with, either by reason of some alteration within the upper air-passages leading to narrowing or actual occlusion of the air-way, or by reason of tonic or clonic spasm affecting muscles normally engaged in maintaining efficient lung expansion. The latter condition, although of interest, is not of sufficient importance to need further remark, and I shall, therefore, only consider the former causes, as they are of much greater consequence.

Although it is true that accidental hindrances to respiration may, with a little management on the part of the administrator, be quickly corrected, cases are sometimes met with in which the respiratory embarrassment is of a more obstinate nature. In order that anæsthesia may be safely maintained, it is absolutely essential that a free air-way from the lungs to the external air shall be present throughout the administration, and profound stertor, or any other sound indicative of the commencement or establishment of obstruction, should be carefully watched for. It is often said in describing the symptoms of a particular case that the patient "stopped breathing," an expression which usually means that some impediment to the free passage of air suddenly gave evidence of its

occurrence. Putting out of the question the presence of adventitious bodies we may say that, when respiration becomes accidentally obstructed, such obstruction will be due either to—

1. An altered relation between the parts constituting the boundaries of the upper air-passages ;
2. Vascular engorgement and swelling of those parts ; or
3. To both of these conditions combined.

1. *Altered Relations between the Parts constituting the Boundaries of the Upper Air-way.*

Sometimes, more especially in the completely relaxed subject, certain parts may fall together, as when the lips of an edentulous patient meet, or the flaccid tongue and lower jaw fall backwards. On other occasions, more especially during the period immediately preceding deep anæsthesia, the obstruction is due to reflex spasm of muscles whose contraction leads to a diminution in the size of the air-way, such spasm being due, either to too strong an anæsthetic vapour or to operative procedures upon extremely sensitive parts. Respiration, which usually takes place wholly or partially through the nose during anæsthetic sleep, is often rendered difficult or impossible by allowing the head to be bent too much forwards. In this position the soft palate will meet the pharynx, and if the mouth be closed and the tongue applied to the teeth respiration will at once cease. Moreover, tilting forwards the head may have the same effect as depressing the lower jaw, that is, it may bring the base of the tongue and epiglottis into contact with the pharynx and so prevent either nasal or oral respiration. The stoppage of breathing, which sometimes occurs when the mouth is opened by a Mason's gag, is due to this cause. In the next place respiration is frequently interrupted and sometimes suspended for a brief period by the closure of the larynx during an act of deglutition. Deglutition is often performed in separate stages, and, thus it happens, that the

larynx may remain applied for a time to the epiglottis, rendering respiration impossible till descent of the larynx has taken place. The completion of the act of swallowing is most likely to be delayed for a considerable period, in other words, respiration is most likely to be suspended from this cause for a considerable period, just as the patient is entering upon, or emerging from, the state of deep anæsthesia. The apnœa thus induced is usually capable of correction either by pushing the lower jaw forwards from behind, by raising the chin, or (less conveniently) by the so-called complete extension of the head and neck. By the kindness of Dr. MacDonald and Mr. Harvey (at the Hospital for Diseases of the Throat) I was enabled to watch, in a patient anæsthetised and relaxed by ether, the readiness with which, by simply pushing the lower jaw forwards from behind, the epiglottis could be made to leave the larynx. Whilst examining the throat of an etherised patient I was also able to satisfy myself of the uselessness of forcible tongue traction in moving the epiglottis—a point which Sir Joseph Lister has, from observations upon his own throat insisted upon, and one which other observers, by experiments upon the cadaver, have corroborated. Pushing the lower jaw forwards from behind usually suffices to remove any obstruction due to the epiglottis. Sometimes, however, there co-exists with the half-performed act of deglutition much tonic spasm of the muscles of the neck, jaws, floor of the mouth, and other parts, rendering remedial measures difficult to apply, and under these circumstances neither pushing the lower jaw forwards nor extending the head and neck may be practicable. When breathing is thus arrested the mouth must be at once opened with some form of gag in order to admit air. The preliminary insertion of a small mouth gag, in those cases in which this difficulty is likely to arise, is strongly to be recommended. The so-called “complete extension of the head and neck” (which undoubtedly has the power of removing the epiglottis from the larynx in the relaxed cadaver) cannot be depended

upon for the relief of apnœa thus arising. There is usually considerable dissimilarity between the upper air-passages of an anæsthetised patient and those of the cadaver, as will be admitted when we take into account the absence of spasm and vascular engorgement in the latter. The part played by venous turgescence in causing stertor and in occluding the upper air-passages will be more particularly discussed presently.

2. *Vascular Engorgement and Swelling of Parts constituting the Boundaries of the Upper Air-passages.*

Stertor and respiratory embarrassment from this cause are more liable to arise in plethoric, florid, or stout subjects than in anæmic or fragile individuals. There is reason, indeed, for the belief that in the former patients temporary hindrance to respiration is often chiefly, if not exclusively, the outcome of vascular turgescence of the tongue, palate, pharynx, nasal cavities, and adjacent parts. The more plethoric the patient the more are stertor and temporary alteration in the rhythm of respiration liable to arise. Anæmic and cachectic persons, as well as those advanced in years, are not liable to exhibit respiratory difficulties from the cause referred to—a fact that is sometimes expressed by stating that such subjects “take chloroform well.” The worst subjects for anæsthetics are bloated, flabby, and alcoholic patients, and the difficulties which arise during the administration to these patients are often due to the cause here indicated. This turgescence is doubtless of vascular origin, and is apparently chiefly venous in its nature. The tongue of a plethoric patient often becomes much swollen during anæsthesia, and by its increase in size obstructs the air-way. It has seemed to me, moreover, that the more air-limitation is practised in administering an anæsthetic to such subjects the greater the swelling and the greater the difficulties in keeping the air-way patent. The tongue of a short-necked, florid man under nitrous oxide, administered in the usual manner,

becomes greatly enlarged, but when the factor of oxygen-starvation is removed by administering 10 per cent. of oxygen with the nitrous oxide, the enlargement of the tongue will be found to be much less marked or even absent altogether. We can readily understand, with the facts which Dr. George Johnson has lately brought before our notice (see § 3), why air-limitation or deprivation during anæsthesia should lead to venous engorgement, and it seems but rational to infer that in full-blooded persons the accumulation of blood in the venous system would be more liable to cause noticeable turgescence of parts than in anæmic and thin individuals. I have on three occasions given anæsthetics to stout, full-blooded patients with glandular or other tumours at the root of the neck—tumours which undoubtedly interfered, by reason of pressure made upon them during operation for their removal, with the return of venous blood from the structures within the mouth. In each of these cases the tongue was greatly enlarged, and in one of them had to be kept half out of the mouth during the administration. The engorgement in question becomes of considerable importance when the air-way is previously encroached upon by morbid growths (see § 1).

In this connection a few remarks must be made upon stertor, which, often affords timely warning of impending obstruction to the passage of air. Dr. Robert Bowles has brought many interesting facts concerning stertor before this Society. The soft palate may vibrate against the tongue during oral respiration, or against the pharynx during nasal respiration, and stertor may thus arise. Or the base of the tongue, more especially if the organ be congested and enlarged, may vibrate against the back of the pharynx and thus produce another variety. Then, as Sir Joseph Lister has shown, the aryæno-epiglottidean folds may vibrate and so cause another kind of stertor. The peculiar throat-sound of nitrous oxide narcosis will be more fully considered below. It seems probable that the venous turgescence, to which the tongue and other parts become liable under anæsthetics, is

an important factor in most forms of stertor. As a practical fact we know that the more plethoric the individual the deeper will be the stertor. When stertor is very profound the anæsthetic should be given cautiously, and the need for admitting air considered, whilst the lower jaw should be pressed well forwards from behind.

Before concluding this section I would refer to a curious condition which, so far as I am aware, has not been described, and which may be met with in connection with apnœa arising from accidentally obstructed breathing. It is well known that thoracic and abdominal movements will continue for a while after complete obstruction to the entry and exit of air has taken place. Now, if from any of the causes which have been mentioned, temporary obstruction at, or about, the superior aperture of the larynx should arise, the continuance of thoracic and abdominal movements will have the effect of increasing the difficulty by sucking the tongue or epiglottis more firmly into that position which originally led to this apnœa. It is hence very desirable that obstructed respiration should be immediately corrected, for the longer it persists the greater will be the difficulty in overcoming it.

§ 3. *On Certain Effects which may be produced by Diminished Oxygenation of the Blood (from Obstructed Respiration or other Causes) during Anæsthesia ; with Remarks concerning the Respiratory Phenomena of Nitrous Oxide Narcosis.*

Though there are several objections to the view that general anæsthetics produce their effects solely by preventing or retarding the normal processes of oxidation within the central nervous system, there can be little doubt that the diminished oxygenation of the blood, which takes place in one way or another during the administration of these agents, often contributes very largely to the establishment of the anæsthesia with which we are familiar in practice. The anæsthesia produced by ether is, for example, more

quietly, more quickly, and in the vast majority of cases more satisfactorily established when air-limitation is practised to a moderate degree than when the anæsthetic is administered with a free supply of air. This, indeed, explains the now almost universal plan of administering ether by what has been termed the close method. Numerous instances, moreover, might be given of the marked influence which a diminished supply of oxygen has in intensifying or prolonging the usual effects of anæsthetic agents. Although moderate air-limitation during anæsthesia is, generally speaking, attended by no untoward symptoms there are undoubtedly certain cases in which this factor should be carefully avoided. To these I shall presently refer. It will be well, before proceeding further, to mention the chief ways in which diminished oxygenation of the blood may arise during the use of general anæsthetics. It may result from :—

1. Intentional air-limitation, as when a bag inhaler is employed.

2. The anæsthetic vapour excluding air to a greater or less extent, as when ether is freely administered from a cone-inhaler.

3. Some special action of the anæsthetic upon the blood, as when by the administration of nitrous oxide the oxyhæmoglobin of the blood becomes reduced.

4. Some pre-existing condition mechanically preventing free access of air to the pulmonary capillaries, as pressure upon the trachea, pulmonary œdema, &c.

5. Some accidental embarrassment to respiration during the administration (see § 2) ; or—

6. Inadequate respiratory movements, as when the nervous mechanism of respiration becomes paralysed, or the bony framework of the chest from senile changes or morbid states of the lungs or pleuræ expands imperfectly.

In administering anæsthetics, one is frequently reminded that the action of the heart, more especially if that organ be functionally or organically deranged, is closely dependent upon the manner in which respiration is being per-

formed. Any marked interference with the respiratory functions—whether such interference be due to the presence of an obstruction, whether it arise from an absence of sufficient oxygen in the gas or vapour, which is being inhaled, or whether it be produced by the use of an inhaler which is not suitable to the case—will tend to depress an already enfeebled heart. Many instances bearing upon this point might be given. In illustration of the effects of temporarily obstructed breathing, I may mention that it is not an uncommon thing, when feeble old persons are under chloroform, for the slight interference with respiration incidental to the insertion of a mouth-gag, to be immediately followed by weakness or irregularity of the pulse, and for such symptoms to quickly vanish when the impeded breathing is relieved by pushing the lower jaw forwards. As an instance of the effects which may be produced upon a feebly acting heart by the inhalation of an anæsthetic free from, or nearly free from, oxygen, I may say that I have known symptoms of cardiac failure to manifest themselves during the administration of nitrous oxide to a young man, who was the subject of aortic regurgitation, symptoms, which were immediately dispelled by two or three compressions of the chest, the heart's action becoming restored immediately air was made to pass into the lungs. As an example of the deleterious effects of an unsuitable inhaler, I may direct attention to cases which have been recorded in which alarming or even fatal symptoms arose during the administration of ether to extremely asthenic subjects by means of Clover's inhaler. The air-limitation thus brought into play, coupled with the stress which would be thrown upon the respiratory mechanism by making the patient breathe through the narrow channels of this inhaler, might easily combine to seriously depress an already enfeebled circulation. Dr. George Johnson has devoted much attention to the effects which air deprivation produce upon the circulation, and his views receive considerable support from what we observe in administering anæsthetics. The

deprivation of air to any great extent leads, as he has shown, first to a constriction of the systemic arterioles, and consequent over-distension of the left side of the heart, and then to constriction of the pulmonary arterioles and over-distension of the right cavities. When the quantity of oxygen which the blood receives during the inhalation of an anæsthetic becomes much reduced, or when the respiratory movements are from any cause so feeble that they do not materially assist in the maintenance of the pulmonary circulation, we have evidence of the correctness of Dr. George Johnson's views in the general venous distension which is present, and which is more especially noticeable in the cervical region. When nitrous oxide unmixed with oxygen is, for example, pushed to its fullest extent, we find that the tongue becomes swollen, the veins considerably distended, and that venous blood freely issues from a cut surface; whereas if this gas be administered with 10 per cent. of oxygen, these phenomena, as well as others of similar significance (to which reference will be made below), are generally speaking absent. It may be said with regard to all anæsthetics at present employed, that, when from any cause the heart's action is weak or much embarrassed prior to the administration, the pulse will quickly show signs of failure should deficient oxygenation of the blood be allowed to occur to any considerable degree, an observation which applies with greater force to chloroform than to ether or nitrous oxide. The more vigorous the heart, the better, of course, will it be able to withstand the depressing influence of the deprivation of air. There is reason to believe that in many of the cases in which syncope has arisen during chloroform anæsthesia, the cardiac failure has in reality been the immediate result of embarrassed or impaired respiration. We are well aware that many fatalities with this anæsthetic have occurred during the period of excitement or during reflex respiratory embarrassment, *i. e.* whilst some degree of apnoea was present, and a considerable strain was being thrown upon the heart. Ether compares

very favorably with chloroform in this respect, for cardiac depression during excitement, and temporary apnoea under this anæsthetic are practically unknown.

We next come to the consideration of the respiratory phenomena of nitrous oxide narcosis. Respiration is deepened and quickened under this gas, the alteration in amplitude and rate of the respiratory movements being chiefly, if not entirely, due to the absence of free oxygen. After a variable though short period, the hurried but rhythmical movements undergo a peculiar and almost characteristic modification ; they become markedly arrhythmical, and often a trifle slower. The alteration in rhythm is usually chiefly dependent upon repeated elevations of the larynx towards the epiglottis, by which the air-way becomes at irregular intervals momentarily occluded. In some cases, however, the rhythm of respiration becomes altered chiefly or wholly by reason of clonic contractions of thoracic and abdominal muscles, engaged in maintaining breathing. The peculiar throat-sound of nitrous oxide administration is due to the irregular elevations and depressions of the larynx towards the epiglottis. Other kinds of stertor (see last section) may, however, occur either independently of, or in association with, this characteristic throat-sound. The clonic contractions of the respiratory and extrinsic laryngeal muscles occur at or about the same time that clonic muscular phenomena in the extremities manifest themselves, and there can be little doubt that they are, like the latter, due to an absence of free oxygen. I have lately administered at the Dental Hospital of London for Dr. George Johnson pure nitrogen, as well as nitrogen with small percentages of oxygen, and the respiratory phenomena which have manifested themselves, as well as the general muscular movements, have so closely resembled those of nitrous oxide as to be indistinguishable from them. I have also conducted about 500 administrations of nitrous oxide with varying percentages of oxygen, and find that it is possible with 10 to 12 per cent. of oxygen to secure perfect anæsthesia without the occur-

rence of any asphyxial phenomena. With this percentage of oxygen, respiration never becomes jerky or laboured, the ordinary stertor of nitrous oxide is absent, there is no lividity of the features, and there are no clonic movements in the extremities. In many of the cases breathing becomes so calm as to be barely perceptible. I would strongly urge that when it is necessary to secure a brief period of anæsthesia in anæmic and feeble individuals, or in those suffering from any serious cardiac or pulmonary affection, nitrous oxide mixed with 10 or 12 per cent. of oxygen should be employed in preference to nitrous oxide alone.

§ 4. *On Failure of Respiration occurring independently of Obstruction or Other Impediment.*

It is not my intention to refer at any length to this form of respiratory failure. When respiration comes to a standstill, without any indication of impediment or obstruction, it is usually a matter of difficulty to decide what factors have principally contributed to the condition. The question is one which is, perhaps, more suited for physiological investigation than for clinical study. In its graver forms respiratory failure of this kind is fortunately rarely met with in practice, though by careful observation early indications of its approach may occasionally be detected. Speaking practically this form of cessation of breathing is in the vast majority of cases associated with a feeble or imperceptible pulse, and there are numerous reasons for regarding it, in many instances at all events, as the outcome of the latter condition, *i. e.* as due to cerebral anæmia. Thus profuse hæmorrhage during an operation may lead to arrested breathing, the respiration becoming re-established as cardiac action increases. It may be said, in fact, that syncope sometimes asserts itself by feeble or suspended respiration, a circumstance which has led many observers to overlook cardiac depression as a factor in respiratory failure in

the human subject. The undeniable effect of inversion in almost immediately restoring respiration which has become paralysed during chloroform inhalation is doubtless due to correction of cerebral anæmia. It is a significant fact that in a moderately healthy individual ether may be administered in very large doses, that is to say, till the pupils are widely dilated, all reflex action is abolished, and respiration is becoming feeble, shallow and irregular, without any such cardiac depression as that which occurs in the case of chloroform administered in similar toxic quantities. When respiration fails independently of obstruction from an overdose of ether it would seem that the failure is the direct effect of the anæsthetic upon the respiratory mechanism, the circulation remaining sufficiently good to negative the possibility of cerebral anæmia contributing to the suspension of breathing. The reason why resuscitation of the patient from an overdose of ether is far easier than from an overdose of chloroform lies in the fact that in consequence of the intensely stimulating effects of the former anæsthetic, the heart's action is still maintained even though the respiratory mechanism has been paralysed by the anæsthetic.

In concluding this section let me add a few words concerning certain effects which may arise from the use of morphine in combination with general anæsthetics. Time will not permit me to enter at length into this matter. Respiration may readily become slow, feeble, or even arrested, from the combined effects of a very small quantity of morphine and general anæsthetics. Whenever the patient is even slightly under the influence of an opiate general anæsthesia must be induced with the greatest caution, and as a general rule it is best to administer the anæsthetic in very small quantities, and not to abolish reflex action.

Practical conclusions.

As the outcome of the foregoing observations the following practical conclusions may be deduced.

1. Any marked deprivation of oxygen (as by the incautious use of bag-inhalers) should be avoided (*a*) in patients with a very feeble circulation; (*b*) in persons who are the subjects of extreme obesity; and (*c*) in those whose respiratory apparatus has become seriously crippled by disease, in other words, when anæsthetising such patients no method of administration should be adopted by which the supply of free oxygen is greatly diminished.

2. Temporarily suspended respiration is far less likely to be attended by symptoms of cardiac depression under ether than under chloroform. The latter anæsthetic should, therefore, be more particularly avoided in those patients in whom excitement or reflex respiratory embarrassment is likely to be well marked; *i. e.* in those who are likely to require considerable quantities of the anæsthetic. There are, of course, many cases in which the operation could hardly be performed under ether (such for example as those of cleft palate and many operations on the mouth and nose), but even in these cases it is generally speaking better to *induce* anæsthesia with nitrous oxide and ether or the A.C.E. mixture, in other words, to tide over the initial stages with ether, and to *keep up* the anæsthesia with chloroform, than to administer the last-named anæsthetic from the beginning.

3. The initial difficulties in respiration which sometimes attend the administration of ether more especially in stout, muscular, or plethoric subjects, may be prevented by first gradually administering a small quantity of the A.C.E. mixture with a copious supply of air, and replacing this anæsthetic by ether from an Ormsby's inhaler, the change being effected when the respirations of the patient are becoming deep and uncontrolled, *i. e.* just before the stage in which muscular movements and other indications of

excitement sometimes arise. This method of producing anæsthesia has much to recommend it for general use, as it is simple to manage and the administration is not disagreeable to the patient.

4. In administering an anæsthetic the slightest impediment to respiration should be corrected as soon as possible in order to prevent an apnœa of a more abiding character from becoming established.

APPENDIX.

1. *References bearing upon § 1.*

'Brit. Med. Journ.,' March 29th, 1884, p. 645 (Bailey, on case of carcinoma of tonsil and on case of goître); 'Practitioner,' 1887, pp. 99—103 (Remarks on the use of anæsthetics in obstructive dyspnœa).

Of the fatalities which have occurred during anæsthesia in persons suffering from some pre-existing impediment to respiration the following may be referred to:—'Brit. Med. Journ.,' March 18th, 1882, p. 380 (Tumour across trachea); 'Brit. Med. Journ.,' Feb. 24th, 1883, p. 351 (Aneurysm of aortic arch); 'Lancet,' August 8th, 1885, p. 245 (Innominate aneurysm); 'Brit. Med. Journ.,' Sept. 29th, 1888, p. 719 (Aortic aneurysm); 'Lancet,' Nov. 15th, 1884, p. 895, and 'Brit. Med. Journ.,' May 2nd, 1885, p. 887 (Bronchocele); 'Brit. Med. Journ.,' March 15th, 1884, p. 508 (Bronchocele); 'Lancet,' Sept. 1st, 1888, p. 442, and 'Brit. Med. Journ.,' Sept. 15th, 1888, p. 625 (Pharyngeal tumour causing dyspnœa); 'Brit. Med. Journ.,' May 19th, 1883, p. 978 (Fatal asphyxia probably due to growth of tongue); 'Lancet,' June 18th, 1881, p. 1014 (Diphtheria); 'Brit. Med. Journ.,' Feb. 23rd, 1884, p. 378 (Cancerous growth in gums and throat); 'Practitioner,' 1887, p. 101 (Innominate aneurysm); 'Brit. Med. Journ.,' July 13th, 1889, p. 88 (Stoppage of respiration after removal of tongue, case complicated by extensively adherent pleuræ); 'Lancet,'

June 25th, 1887, p. 1297, and 'Brit. Med. Journ.,' same date, p. 1397 (Pleural effusion); 'Brit. Med. Journ.,' Feb. 20th, 1886, p. 356 (Empyema); 'Brit. Med. Journ.,' Oct. 10th, 1868, p. 407 (Nitrous oxide in phthisis).

2. *References bearing upon § 2.*

'System of Surgery,' 2nd ed., 1870, vol. v, p. 492 (Lister on the effect of tongue traction, &c.); 'Trans. Roy. Med. and Chir. Soc.,' vol. xliii, 1860, p. 49 (Bowles on stertor); "Stertorous breathing in Apoplexy, and the management of the apoplectic state," by Dr. Bowles, published by the 'Brit. Med. Association,' 1881; 'Brit. Med. Journ.,' Jan. 12th, 1889, p. 65 (Bowles on raising the epiglottis); 'Trans. Roy. Med. and Chir. Soc.,' vol. xlvii, p. 403, cases 9, 14, and 17 (Recoveries from obstructed breathing under chloroform); 'Brit. Med. Journ.,' Feb. 14th, 1874, p. 201 (Clover on beneficial effect of raising the chin to remove epiglottis from larynx); 'Lancet,' Oct. 11th, 1879, p. 539 (Jacob on respiratory difficulties during etherisation); 'Lancet,' Oct. 27th, 1888 (Howard on raising the epiglottis); 'Brit. Med. Journ.,' Jan. 26th, 1889, p. 185 (On raising the epiglottis); 'Trans. Odont. Soc.' vol. iii, new series, p. 36 (Case of spasm of glottis under nitrous oxide); 'Trans. Odont. Soc.,' vol. iii, new series, p. 117 (Case quoted of stout plethoric man under nitrous oxide: tongue (probably from enlargement), causing obstruction to respiration); 'Lancet,' Feb. 16th, 1889 (Silk on "Anæsthetic Apnoea"); 'Brit. Journ. Dent. Science,' vol. xix, 1876, p. 229 (tonic spasm of respiratory and other muscles, probably of reflex origin and due to tooth extraction during imperfect anæsthesia, under nitrous oxide).

The following fatalities may be referred to as bearing upon the points discussed in § 2.—'Brit. Med. Journ.,' Jan. 8th, 1881, p. 62 (Struggling: laboured followed by obstructed breathing: cardiac failure); 'Lancet,' March

12th, 1881, p. 42, and 'Brit. Med. Journ.,' March 19th, 1881, p. 440 (similar case); 'Brit. Med. Journ.,' Feb. 24th, 1883, p. 350 (similar case); 'Lancet,' March 5th, 1881, p. 401 (similar case); 'Lancet,' July 29th, 1882, p. 153 (Tongue or epiglottis probably causing asphyxia); 'Brit. Med. Journ.,' Sept. 29th, 1888, p. 719 (Embarrassed respiration leading to fatal syncope); 'Lancet,' Sept. 3rd, 1881, p. 430 (similar case); 'Brit. Med. Journ.,' Nov. 22nd, 1884, p. 1027 (similar case); 'Trans. Roy. Med. and Chir. Soc.,' vol. xlvii pp. 377—396; see cases 17, 50, 79, 80, 83, 87, and 101; 'Trans. Odont. Soc.,' vol. ix, 1877, p. 182 (A death from nitrous oxide, probably due to the non-relief of obstructed respiration occurring during the administration).

3. *References bearing upon § 3.*

"An Essay on the Physiology of Asphyxia (Apnoea)," by George Johnson, M.D., F.R.S., 'Brit. Med. Journ.,' July 15th, 1882 (Lawson Tait on etherisation by close inhalers); 'Lancet,' April 27th, 1889, p. 832 ("On the Anæsthesia produced by the administration of mixtures of Nitrous Oxide and Oxygen").

The following fatalities may be referred to as bearing upon the points mentioned in § 3:—'Lancet,' Sept. 4th, 1880, p. 376 (Extreme asthenia: syncope during the arrest of breathing incidental to vomiting); 'Brit. Med. Journ.,' July 15th, 1882, p. 103 (Cardiac failure apparently due to asphyxia caused by the close method of etherisation in an asthenic subject); 'Brit. Med. Journ.,' Jan. 15th, 1881, p. 103 (Asthenia: cardiac failure during etherisation: two cases); 'Brit. Med. Journ.,' March 13th, 1886, p. 489 (quoted by Dr. Jacob: emaciated girl of ten years: syncope); 'Lancet,' Dec. 3rd, 1887, p. 1132, and the 'Lancet,' article in the same number (Syncope apparently due to asphyxia produced by the administration of ether by close method to a cachectic patient with morbus

cordis); 'Journ. Brit. Dental Association,' Oct. 1880, p. 504 (Syncope under nitrous oxide); 'Journ. Brit. Dent. Association,' 1889, p. 689 (Syncope under nitrous oxide).

4. *References bearing upon § 4.*

"Anæsthetics, Ancient and Modern," by George Foy, F.R.C.S., 1889, pp. 105—109 (Cases bearing upon the effect of inversion in restoring respiration); 'Practitioner,' 1887, p. 103 (On morphine and general anæsthetics); 'Brit. Med. Journ.,' January 8th, 1881, p. 69 (Morphine and general anæsthetics); 'Brit. Med. Journ.,' June 14th, 1890, p. 1346 ("Remarks on the Report of the Second Hyderabad Chloroform Commission," by the Members of the British Medical Association Committee on Anæsthetics: see their remarks relative to cessation of respiration dependent upon feebleness of cardiac action).

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. iii, p. 33.)