

properly managed, restored to health is, in my judgment, one of the greatest gains to practical medicine in the last quarter of a century. This is, perhaps, not strictly a gynæcological matter, but it is often so closely connected with it that this passing allusion to it may be pardoned. I hope that even these few words may prove to you that gynæcology has not been at a standstill.

It is my firm conviction that the more scientific basis on which gynæcology is being placed by pathological, histological, and bacteriological research will ere long give it a position which it has hitherto lacked, as is not unnatural in a subject of comparatively recent development. And now, gentlemen, I cannot conclude without a word personal to myself. It is just thirty-five years since I came to London to seek a livelihood, as so many of my countrymen have done since the time of that "high and mighty Prince," James I. of England and VI. of Scotland. It is not an exhilarating thing to put up your brass plate in a city such as this, in which you could count your acquaintances on the fingers of one hand. I was greatly encouraged when in the same year the Council of King's College elected me to be assistant obstetric physician to this hospital. I may say of it, and I say it with the deepest gratitude, I was a stranger and it took me in. In retiring from the active work in connexion with it, which for so many years has been the chief occupation, and interest, and pleasure of my life, it would be a comfort to me if I could carry with me the conviction that the generous confidence thus shown to an unknown young man has not been entirely misplaced. It is a painful thing to say farewell. I cannot do it better than in the words of a prayer often offered up within these walls, "God bless King's College, and King's College Hospital, and all connected with them."

Remarks

ON

6657 ADMINISTRATIONS OF ANÆSTHETICS
CONDUCTED AT THE LONDON HOSPITAL
DURING THE YEAR 1897.

In three Clinical Lectures delivered at the Hospital

By FREDERIC W. HEWITT, M.A.,
M.D. CANTAB.,

ANÆSTHETIST TO THE LONDON HOSPITAL, CHARING-CROSS HOSPITAL,
AND THE DENTAL HOSPITAL OF LONDON.

LECTURE III.¹

Delivered on Feb. 18th, 1898.

GENTLEMEN,—We have to consider to-day the two remaining parts of our subject. These are (1) the cases in which fatal symptoms, partly or wholly referable to the anæsthetic, were recorded; and (2) the after-effects in the various cases in which anæsthetics were administered. I have placed upon the board a simple table which explains itself. Of the 6657 cases in which anæsthetics were given there

Cases in which Fatal Symptoms, partly or wholly referable
to the Anæsthetic, were recorded.

Anæsthetic.	Factors.		
	A, During the administration.		B, After the administration.
	Anæsthetic.	1. State of patient. 2. Operation. 3. Anæsthetic.	1. State of patient. 2. Anæsthetic.
Ether (2910 cases) ...	—	—	1
Chloroform (677 cases)	1	1	—
Totals	1	1	1

¹ Lectures I. and II. were published in THE LANCET of Feb. 19th and March 5th, 1898, respectively.

were only three in which the anæsthetic could in any way be held responsible for the fatal issue, and you will see that of these there is only one in which the anæsthetic was the sole factor. In the remaining two cases other factors more important than the anæsthetic must be regarded as having been present. I shall first of all deal with the case in the third column. It is one in which the fatal symptoms arose after the administration of ether, the state of the patient being the primary factor and the anæsthetic the secondary. The case is one which, so far as my experience and reading are concerned, is quite unique, and it is certainly of very great importance. It was this case coupled with one or two others in the course of this year's work which made me feel that our experience in this hospital should be put on record. The patient was a male fourteen years of age. About three months before admission he contracted typhoid fever and the attack was a severe one. He subsequently developed symptoms of appendicitis and was admitted to the hospital on Jan. 5th, 1897, to be operated upon for that affection. On Jan. 15th, the day of operation, the boy appeared to be in very fair health; he had good heart sounds, there was no cough or difficulty in breathing, and the abdomen was not distended. Ether was given to him by means of a Clover's inhaler and the administration lasted forty minutes. The induction of anæsthesia was perfectly smooth and there was no difficulty of any kind, but when the patient was fully under ether I noticed a great peculiarity about his breathing. In order to explain this to you I must first say that if you take the trouble to observe the respiration of patients under anæsthetics you will find that in most cases the abdomen rises with inspiration and falls with expiration, and that the sternum either remains practically stationary during both inspiration and expiration, or actually slightly recedes with the former and rises with the latter. The meaning is simple. It is that the diaphragm does most of the inspiratory work and by vigorously contracting produces such sudden negative pressure in the chest that the sternum actually falls in during the inspiratory contraction of the diaphragm. This is, however, not the case with all patients. One sometimes sees, especially in perfectly healthy young men and women, both chest and abdomen rising with inspiration, provided that no obstruction such as that expressed by stertor be present. If even mild stertor be audible the increased inspiratory effort needed to overcome the obstruction upon which the stertor depends will be made by the diaphragm and the sternum will at once begin to recede during respiration. I need hardly remind you that patients with rigid chest-walls will not display these phenomena to the extent observed in patients with yielding parietes. So far, then, as the abdomen is concerned we may say that it almost invariably rises during the inspiratory phase and falls during the expiratory. I had observed this in so many cases that you may judge of my surprise when I saw that in this boy under ether an exact reverse of the usual state of things was present, and that the abdomen instead of rising with inspiration was receding, and the sternum instead of receding was markedly rising. In other words, the breathing was entirely thoracic, the diaphragm being wholly inactive. I was so struck by the peculiarity that I called the operator's attention to it. There was also another unusual feature in the case. Although a plentiful supply of air was given—such a supply as under ordinary circumstances would have ensured a florid colour without any duskiness—this patient remained slightly cyanosed throughout. But beyond these two points, which might easily have escaped my attention had I not been interested in the different forms of breathing during anæsthesia, there was nothing to note. There was no difficulty of any kind; the respiration was regular and not hurried, the pulse was excellent, the pupils were as a rule moderately contracted, and there was no abnormal secretion of mucus. The operation was successfully performed and the patient was taken back to bed. Later in the day, however, it was noticed that more mucus than usual seemed to be present in the air-passages, that the respiration was 32 per minute, and that the pulse was 112. Breathing did not improve during the night, but it did not get much worse. The patient slept fairly well but complained of slight pain at the base of the left lung anteriorly. At 8 A.M. the next morning the pulse was 160, the respiration was 40, and there was a good deal of rattling in the chest. At 12 noon the breath sounds were harsh, with râles and rhonchi all over the chest except at the base of the left lung anteriorly, which was dull to percussion. The heart sounds

were almost indistinguishable. There was no increase of vocal resonance. The apex beat of the heart was not displaced. There were no signs of fluid or definite pneumonia. The respiration was 52. Stimulants were given, the inhalation of oxygen was employed, and the patient was propped up in bed. At 4 P.M. the respiration was 81, the pulse was 150, and the temperature was 102° F. At 10 P.M. the patient grew very dusky and he was bled, but only one and a half ounces of blood issued. No tubular breathing was audible, but both bases posteriorly were dull and the breath sounds were deficient. Strychnine and digitalis were injected every two hours, but there was no improvement and the patient died thirty-four hours after the operation. Unfortunately, no post-mortem examination was allowed, but everything seems to point to acute pulmonary oedema as the cause of death.

There is no doubt, I think, that in this case complete diaphragmatic paralysis was present before the anæsthetic was given. At the conclusion of the operation, indeed, the dresser of the case informed me that he had himself observed the peculiarity in the patient's breathing on the preceding day. With the cause of the paralysis of the diaphragm we are not, of course, concerned, but it is most probable that it arose as a result of the attack of typhoid fever, peripheral neuritis being one of the recognised sequelæ of this disease. As is well known, inaction of the diaphragm may be present without causing the patient any distress, provided that no great exertion be taken and that the thorax be sufficiently mobile to carry on respiration. In this case the elastic thorax was perfectly competent to take over the work of the diaphragm whilst the patient was at rest in bed, but when under the influence of ether both respiration and circulation became increased the thoracic movements were doubtless incompetent to fully oxygenate the blood passing through the pulmonary capillaries, and hence a certain degree of cyanosis was, as we have seen, produced. Moreover, when we bear in mind the fact that the pulmonary circulation is very largely dependent for its efficient maintenance upon free lung expansion the question arises whether the thoracic movements were sufficient during the administration to properly carry on this circulation, especially at the bases of the lungs. It is probable that they were sufficient, for had they not been more symptoms of distress would most likely have been noted. On the other hand, it is not, I think, unreasonable to suppose that the bases of the lungs were not expanded so efficiently by thoracic movement as they would have been had the diaphragm been in working order, and if this view be correct we can well imagine incipient pulmonary oedema from a comparatively sluggish circulation through the vessels in the bases of the lungs to have begun during the administration. Again, it must not be forgotten that the tranquil breathing which always accompanies recovery from ether anæsthesia must have been eminently favourable to the development of pulmonary stasis and oedema in a patient whose diaphragm was paralysed. One frequently sees instances, under all anæsthetics, of the respiratory centres resting, so to speak, after unwonted activity, and there can be little doubt that the phase of tranquil breathing through which this patient must have passed—a phase which commenced with the withdrawal of the ether inhaler and terminated with re-establishment of consciousness—was specially favourable in such a subject to the continuance of the hyperæmic pulmonary condition brought about by the administration of ether. It is well known that patients with diaphragmatic inaction are particularly prone to congestive and inflammatory lung complications, so that it is not very surprising that after forty minutes' etherisation such symptoms should have arisen in this patient. By this hypothesis it is, I think, easy to explain the occurrence of the oedema which unfortunately proved fatal. Had we not observed the diaphragmatic paralysis the case would undoubtedly have been lost sight of with others whose etiology is perfectly obscure. It is true that even with the data now at our disposal we cannot say for certain what was the immediate pathology of the pulmonary oedema, but I think you will agree with me that by the careful study of such cases as these we shall at all events tend to advance our knowledge as to the circumstances under which the pulmonary sequelæ of ether may arise.

Before dismissing the above case we should carefully consider whether it is capable of teaching us any lesson which may be of use for future guidance. It is, of course, somewhat difficult to say whether the chances of pulmonary

complications would have been less had chloroform or the A.C.E. mixture been employed instead of ether, but the probability is certainly in this direction. Again, with the experience of the case to help us, it is now easy to see that the unusual cyanosis met with during the administration might with advantage have been taken to indicate that a change from ether to chloroform should have been effected. I think we shall not be far wrong in the future if, on finding that the patient presented to us for anæsthetising has diaphragmatic paralysis, we adopt all means in our power to throw as little stress as possible upon both respiration and circulation. Possibly the use of chloroform in conjunction with oxygen would be the most suitable anæsthetic for such cases. There is just one other point which occurs to me in connexion with the case. It is that after the administration of an anæsthetic to a patient with diaphragmatic paralysis it may be advisable to assist respiration by intermittently compressing the lower ribs till consciousness has returned, in order to prevent as far as possible any circulatory stasis at the bases of the lung.

The next case occurred under chloroform, and there is very little doubt that the anæsthetic was the chief if not the sole factor. It is true that the patient was not in a perfect state of health, as he was the subject of slight chronic bronchitis, but there is no evidence that this contributed in any way to the fatal issue. He was a labourer, twenty-six years of age, well built and muscular. Owing to his bronchial affection it was thought advisable to administer chloroform rather than ether. He had, however, previously had ether for a short operation and no difficulties had arisen during the administration. I have before pointed out to you how very cautious you should be in giving chloroform, *ab initio*, to muscular men, and this case will illustrate far better than any remarks I have made how vigorous patients may die comparatively early in the administration of chloroform. The contemplated operation was for tuberculous epididymitis and the anæsthetic was given from a Skinner's mask. The patient took the chloroform fairly well till excitement commenced; there was then very considerable struggling which was attended by rigidity of the chest, arms, and legs. The rigidity lasted from thirty to forty seconds and necessarily led to embarrassment of breathing. At the end of this time, however, it passed off; two deep breaths were taken and the anæsthetic, which had been discontinued, was resumed. The report goes on to say that in about half a minute the conjunctival reflex was lost and the pupils were moderately contracted. But whilst the patient was being wheeled into the theatre it was noticed that breathing had ceased and that the face was cyanosed. The pulse, however, was then palpable at the wrist. Remedial measures were promptly applied (artificial respiration, strychnine, and brandy), but the pulse disappeared and, with the exception that about twenty respirations were subsequently taken at intervals of about a quarter of a minute, no further signs of animation were observed. Artificial respiration was nevertheless kept up for some time.

So many deaths of this kind have been recorded in our medical journals that it will, I think, be well worth while to carefully consider the facts just narrated. The first element in the case was undoubtedly the struggling. This led to a fixed condition of the thoracic and abdominal parietes, during which the absorption of the chloroform previously given and now locked up, as it were, in the air passages steadily continued. Under such circumstances as these the right side of the heart would find itself less able than usual to perform its work. In the first place, the struggling would tend to force an abnormal quantity of blood into its cavities; in the second place, the impeded pulmonary circulation would prevent those cavities emptying themselves as easily as in unrestricted breathing; and, in the third place, the heart would be to a certain extent dilated from the effects of the chloroform upon its muscular tissue. The absorption of the anæsthetic vapour during totally arrested breathing is an important point to bear in mind in studying fatal cases of this category. The muscular rigidity of the so-called struggling stage is very often associated with mechanically obstructed breathing from partially performed deglutition movements or other causes, so that neither can air enter the lungs nor can the imprisoned vapour leave them. So important is the absorption of chloroform during temporarily suspended breathing that I think it should be a rule to withhold the anæsthetic whilst the rigidity is subsiding and for some little while afterwards, and only to recommence the administration if there is a conjunctival reflex present. In the particular case before us it is stated

that the anæsthetic was not resumed till after two deep breaths had been taken at the conclusion of the rigid stage. But if we picture to ourselves the state of the circulation at that moment we shall, I think, agree that the blood must then have contained a good deal of chloroform and that the right side of the heart must have been somewhat overtaxed and distended as the immediate result of the struggling and self-asphyxiation through which the patient had just passed. It is clear that by recommencing the administration before a proper state of equilibrium has been established between the pulmonary circulation and the right cavities of the heart the fresh chloroform distributed to the heart muscle by the coronary arteries can only have the effect of still further dilating those cavities and rendering them unable to meet any further strain that may be imposed upon them. What the precise nature of this additional strain may have been in this case it is rather difficult to say. It is, however, fairly certain that some additional respiratory embarrassment arose and acted as the last straw. Either the upper air-passages again became mechanically obstructed as the patient was being moved into the theatre or the respiration came to a standstill as the immediate result of the toxic effects of the previously absorbed chloroform upon the nervous centres, or both conditions were present. But whatever may have been the cause of the arrest of breathing the final result upon the embarrassed and poisoned heart muscle was the same—viz., to arrest its action.

It will be well in this connexion to say something on the much-discussed question whether under chloroform the respiration fails before the circulation or the circulation before the respiration. In considering this question it is necessary in the first place that two considerations should be borne in mind. The first of these is that failure of the wrist pulse is by no means synonymous with failure of the heart. The second is that there are three distinct ways in which breathing may come to a standstill under chloroform—the state of the circulation at the moment when breathing ceases being very materially dependent upon the form of respiratory failure. (1) With regard to the first of these considerations I may tell you that I have seen many cases in which the wrist pulse has become almost or completely imperceptible although the heart has been acting, though feebly, at the time. The clinical question whether the pulse fails before respiration or *vice versa* must therefore not be confounded with the physiological question whether the heart ceases before the respiration or *vice versa*. Many have imagined that because they have met with cases in which the pulse has vanished at the wrist whilst respiration has been proceeding the physiological dictum that respiration stops before the heart is opposed to clinical evidence, but this is not really so. During the fall of blood-pressure in deep chloroform anæsthesia the pulse may disappear whilst the heart is still acting; then the breathing ceases; and, finally, the heart. You will remember that I have in a previous lecture emphasised the necessity for watching the pulse in deep chloroform anæsthesia; and it is by doing this that you will often be able to avoid disaster. (2) The next consideration is important, and it is one, moreover, to which physiologists have not perhaps directed sufficient attention. During anæsthesia breathing may become arrested (*a*) from mechanical obstruction, (*b*) from spasm of the respiratory muscles, or (*c*) from paralysis of the respiratory nervous mechanism. Now the first two forms of respiratory failure are most likely to be met with during comparatively light anæsthesia—i.e., when the general systemic circulation is fairly good; and under these circumstances the pulse may remain palpable at the wrist for a considerable time after breathing has ceased. In the third form of respiratory failure, however, the arterial tension at the moment of the arrest of breathing is so low that either there is no pulse to be found at the wrist or it is barely palpable, or if it is fairly palpable it quickly disappears. In the human being under chloroform there is a very great tendency to mechanically obstructed breathing from a variety of causes; and it is this tendency which, to my mind, places the human being upon a somewhat different footing, so far as the physiological action of chloroform is concerned, to lower animals. Considerable experience is needed to detect some of the more insidious forms of mechanically impeded breathing. Indeed, in many cases this element of obstruction plays a far more important part in the administration than the chloroform itself. It is impossible on this occasion to enter upon a consideration of

the various ways in which breathing may become obstructed. All I wish to say now is that the lower animals do not display these tendencies and it is therefore, in my opinion, erroneous to argue from the one to the other without taking these differences into consideration. It is in the highest degree probable that in the case last quoted some obstructive condition acted as the last straw in upsetting the circulatory balance. It is quite certain that respiration was primarily interfered with, for the pulse was palpable when breathing had become arrested. The final gasps, at long intervals, only came on after the muscular system had completely relaxed and cardiac action could not be restored, and they cannot, I think, be regarded as indicating the continuance of true respiration. They were analogous to the final gasps met with in the course of asphyxia, but by the time they appeared it was impossible to restore cardiac action. From what I have placed before you you will, I think, see that in cases of anxiety under chloroform one of three clinical conditions may be present. You may either have (1) the respiration ceasing before the pulse, (2) the pulse ceasing before the respiration, or (3) both ceasing simultaneously; and you must bear in mind that neither of these conditions is incompatible with the physiological fact that in an overdose of chloroform respiration ceases before the heart.

The next case is one in which death occurred during the administration of chloroform, but the anæsthetic had very little indeed to do with the fatality. In a great many hospitals it is customary to attribute such deaths entirely to surgical shock, but one cannot, I think, wholly exonerate the anæsthetic. The patient was a male, suffering from empyema, and he was in a very unsatisfactory general condition. It was proposed to remove two pieces of rib. Chloroform was given, but only in small quantities, and while the pus was escaping, the patient stopped breathing and died. The case must, I think, be considered as one in which the state of the patient was the primary, the operation the secondary, and the anæsthetic the tertiary factor. With regard to the use of anæsthetics in cases of empyema I may say that the most favourable ones are the extremely chronic and the worst the very acute. If a patient has had an empyema for some weeks or months and his respiration has become adapted to the altered circumstances there is very little special risk. But if you should be called upon to give an anæsthetic to a patient with pleuropneumonia running on to empyema you should be very cautious. The worst cases of all are those in which the patient is muscular or obese, the temperature high, the pulse quick, and the face flushed and dusky. In such patients the respiratory apparatus has not yet become accustomed to its altered circumstances. In cases of empyema one of the most important points is the posture of the patient. If the patient be a heavily built man with but one lung available for respiration, and if he be turned over to his sound side it will be obvious that the weight of the trunk may seriously impair the working of the sound lung. Whenever it is practicable, therefore, you should endeavour to place your patient in such a posture that his breathing is as little impaired as possible. In chronic cases ether may be given perfectly well, and I have even used it with advantage in moderately acute ones in which a short and partial anæsthesia seemed indicated; but if ether be used it is imperatively necessary that it should be given by the open method with plenty of air. All inhalers with bags must be avoided. In the acute and comparatively recent cases struggling and rigidity are more likely to arise than in others and, as I have already pointed out to you, this stage of struggling is the most dangerous stage under chloroform. If, therefore, you suspect from the type of your subject that struggling and rigidity will arise I would strongly advise you to endeavour to pass over that stage under ether and, if necessary, to change to chloroform subsequently. If a muscular patient with recent empyema become rigid the heart may quickly fail. It is, indeed, cardiac rather than respiratory failure that is to be feared in cases of this kind.

It now remains for me to direct your attention to the records of after-effects. The gastric disturbances which are unfortunately liable to follow the administration of chloroform, ether, and allied anæsthetics deserve special attention, for from the point of view of both patient and surgeon they are often exceedingly objectionable. We are, perhaps, too prone to look upon some degree of nausea and vomiting as necessarily consequent upon ether and chloroform anæsthesia and to imagine that if we have arranged the diet of the patient before the operation we have done everything that

is possible in the way of preventing these unpleasant sequelæ. Only those who have undergone operations under ether or chloroform and have suffered severely from after-vomiting can thoroughly appreciate the importance of studying this branch of the subject. Let me first of all deal with the facts at our disposal. It is a very difficult matter to collect statistics of after-effects, for, in order that any case may be complete it is necessary that we should have details as to the kind of nourishment last taken, the interval between the taking of this nourishment and the operation, the anæsthetic used, the length of inhalation, the nature of the operation, and the extent of the nausea, retching, or vomiting afterwards. I shall refer only to gastric after-effects following the use of other anæsthetics than nitrous oxide. Out of our 6657 cases I am sorry to say that I have only been able to obtain full details as to after-effects in 275, and I find that these can be arranged in four classes, as follows:—

Gastric After-effects of Anæsthetics other than Nitrous Oxide (275 cases).

Class I. (no after-effects)	109
Class II. (slight after-effects)	109
Class III. (moderately bad after-effects)	35
Class IV. (severe after-effects)	22
Total	275

The subjoined table shows the anæsthetics given in these 275 cases.

	Class I.	Class II.	Class III.	Class IV.	Total.
Ether	43	44	18	6	111
A.C.E.	7	7	—	2	16
Chloroform	6	1	—	1	8
Gas and ether	19	25	8	5	57
A.C.E. and ether	9	10	3	3	25
A.C.E. and chloroform	5	5	—	—	10
Ether and chloroform	8	9	4	2	23
A.C.E., ether, and chloroform	9	5	—	1	15
Gas, ether, and chloroform	3	3	2	1	9
Gas, ether, A.C.E., and chloroform	—	—	—	1	1
Total	109	109	35	22	275

Unfortunately the number of cases in which chloroform after-effects were recorded is so small that it is impossible to draw any conclusions as to the relative severity of gastric disturbances after this as compared with other anæsthetics. The following calculations are, however, of interest. We can compare the cases in which ether was either used throughout or was preceded by nitrous oxide or the A.C.E. mixture as preliminary anæsthetics with those in which ether was given for the first part of the administration and was subsequently changed for chloroform. Thus:—

Ether.	Ether followed by Chloroform.
Ether 111	Ether and chloroform 23
Gas and ether 57	A.C.E., ether, and chloroform 15
A.C.E. and ether 25	Gas, ether, and chloroform ... 9
Total 193	Total 47

When worked out in percentages we find the following results:—

Ether.	Per cent.	Ether followed by Chloroform.	Per cent.
Class I.	36·78	Class I.	42·55
Class II.	40·95	Class II.	36·19
Class III.	15·02	Class III.	12·75
Class IV.	7·25	Class IV.	8·51
Total	100·00	Total	100·00

It is only right to bear in mind in studying these figures that they are after all based upon very few cases; still, they

seem to point to certain conclusions which we have arrived at in other ways, so I have placed them before you. You will see that whilst 36 per cent. of the ether cases had no after-effects 42 per cent. of the others were similarly fortunate; whereas whilst the frequency of severe after-effects in the ether cases is represented by 7·2 per cent., this frequency comes out at 8·5 per cent. in the others. In other words, patients are more likely to suffer from slight after-effects when ether has been used, but the chances of protracted vomiting are greater when chloroform has been administered. Further data are, of course, needed to make this point finally certain. The influence of the length of abstinence from food is very fairly shown by the following figures:—

Average Abstinence from Food.

Classes I. and II. combined (no after-effects or very slight)	4 h. 4 min.
Classes III. and IV. combined (moderate or severe after-effects)	3 h. 50 min.

There can, I think, be no doubt that an abstinence of at least four hours should be enforced, and if it be somewhat longer so much the better. Clear soup or beef-tea without any solids is on the whole the best form of nourishment before an operation. Milk and eggs should be avoided. In private practice an endeavour should be made to interfere as little as possible with the patient's usual hours for meals. Thus, for early morning operations (up to 10 A.M.) he should have a light dinner the night before at his usual time and nothing whatever after. In the case of an operation about 2 P.M. he should have a light breakfast at his usual time and nothing after. The practice of giving a patient beef-tea at 6 A.M. before a 9 o'clock operation is open to objection, and the same remark applies to beef-tea or soup at 11 A.M. before a 2 o'clock operation. The stomach should not only be empty when an anæsthetic is given but digestion should have finished for some little time.

The next statistics are interesting, for they show the very decided influence which the length of administration has in determining gastric after-effects:—

Average Duration of Administration.

Class I.	30 min. 3 sec.
Class II.	34 min. 52 sec.
Class III.	41 min. 39 sec.
Class IV.	41 min. 51 sec.

The lesson to be learnt from these figures is that patients should not be kept longer under anæsthetics than is necessary, for it is clear that, other things being equal, the longer a patient inhales an anæsthetic the greater will be the chances of after-effects.

I may here say something as to the prevention and other treatment of the above sequelæ of anæsthesia. I have already alluded to the regulation of the diet. In addition to this it is essential that the bowels should be thoroughly evacuated. This is best done by administering a purgative the night but one before the operation and an enema on the morning itself. As regards the administration the patient should be rendered deeply insensible as quickly as is compatible with safety; a deep anæsthesia, free from swallowing movements, should be maintained; the head should be kept upon the side for the escape of mucus and saliva and the mouth should be frequently wiped out. If it be necessary to move the patient from the operating table to a bed this should, if possible, be done during deep anæsthesia. When in bed he should be turned well upon his side, the bed should not be moved, and the room should be kept quiet. Putting exceptional cases on one side, no nourishment whatever should be given until the patient himself asks for it. If nausea, retching, or vomiting be present the first thing to do is to give at frequent intervals two or three ounces of very hot water to drink. The taste of ether is best overcome by moistening the lips with lemon juice. If there appears to be a neurotic element present, enemata of twenty grains of bromide of potassium in two ounces of water will often answer well. I have found the inhalation of vinegar from a towel very useful in arresting vomiting and I may say the same of the application of a mustard leaf to the epigastrium. By one or other of these plans you will find that you will be able to successfully treat the unpleasant after-effects of anæsthesia.