

The president's concluding remarks on anæsthetics, with special reference to chloroform and ether- their physiological action, their relative value, their dangers, and their mode of administration : discussion on anæsthetics in the Medico-Chirurgical Society of Glasgow, 31 st. October, 1890 / William Macewen.

Contributors

Macewen, William, Sir, 1848-1924.

Publication/Creation

[Glasgow] : [The Society], [1890?] (Glasgow : Alex. Macdougall.)

Persistent URL

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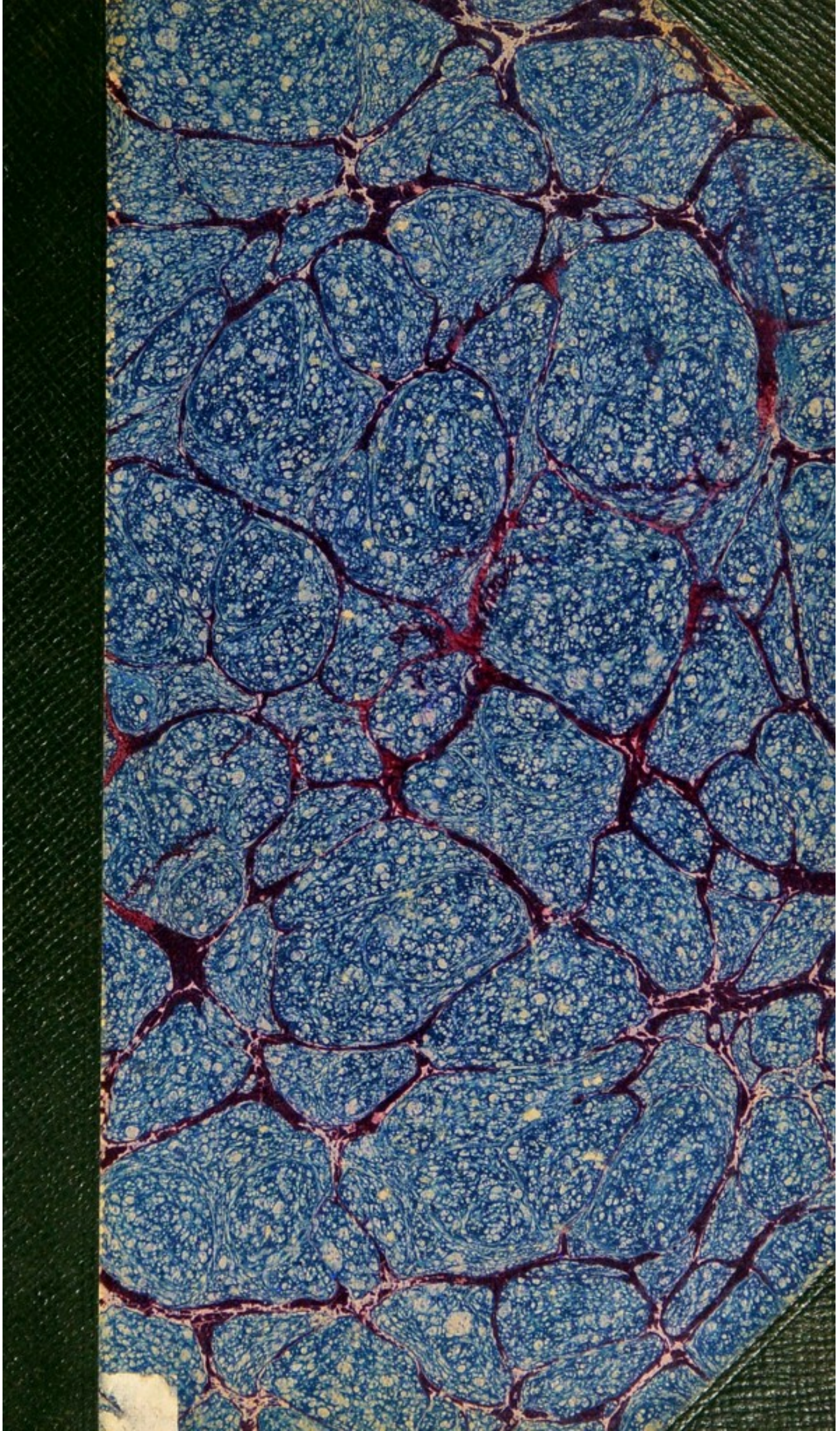
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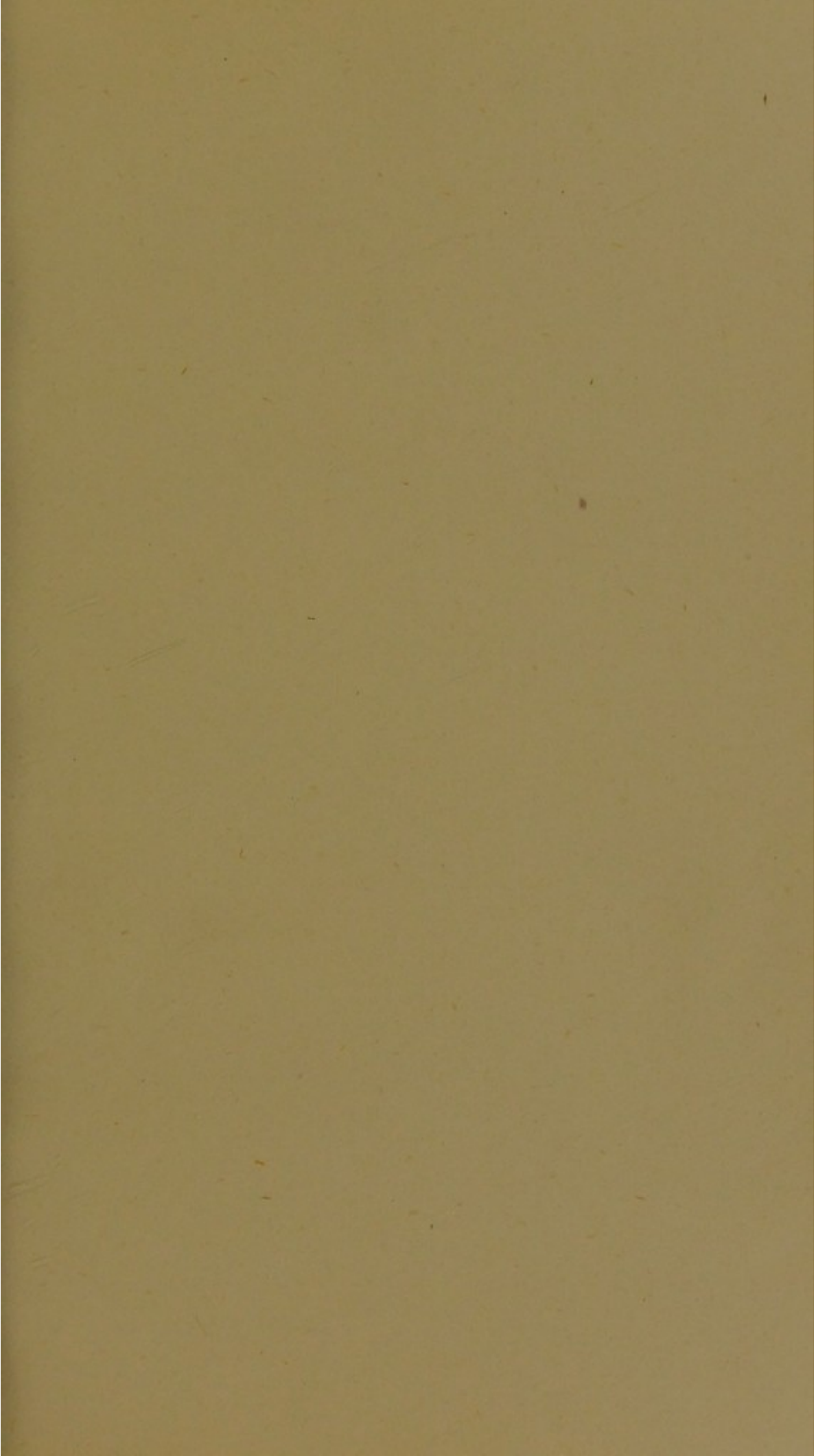


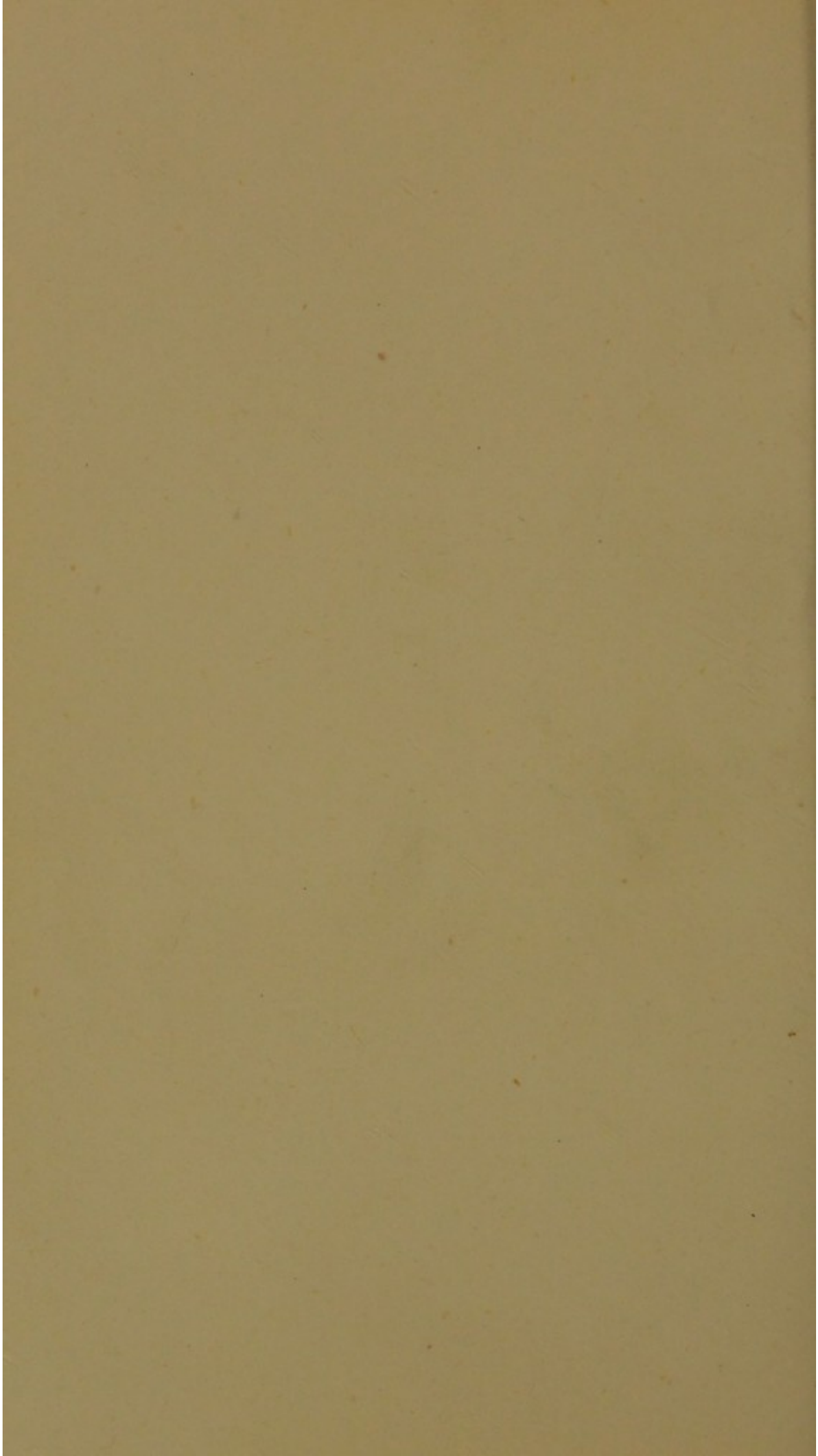
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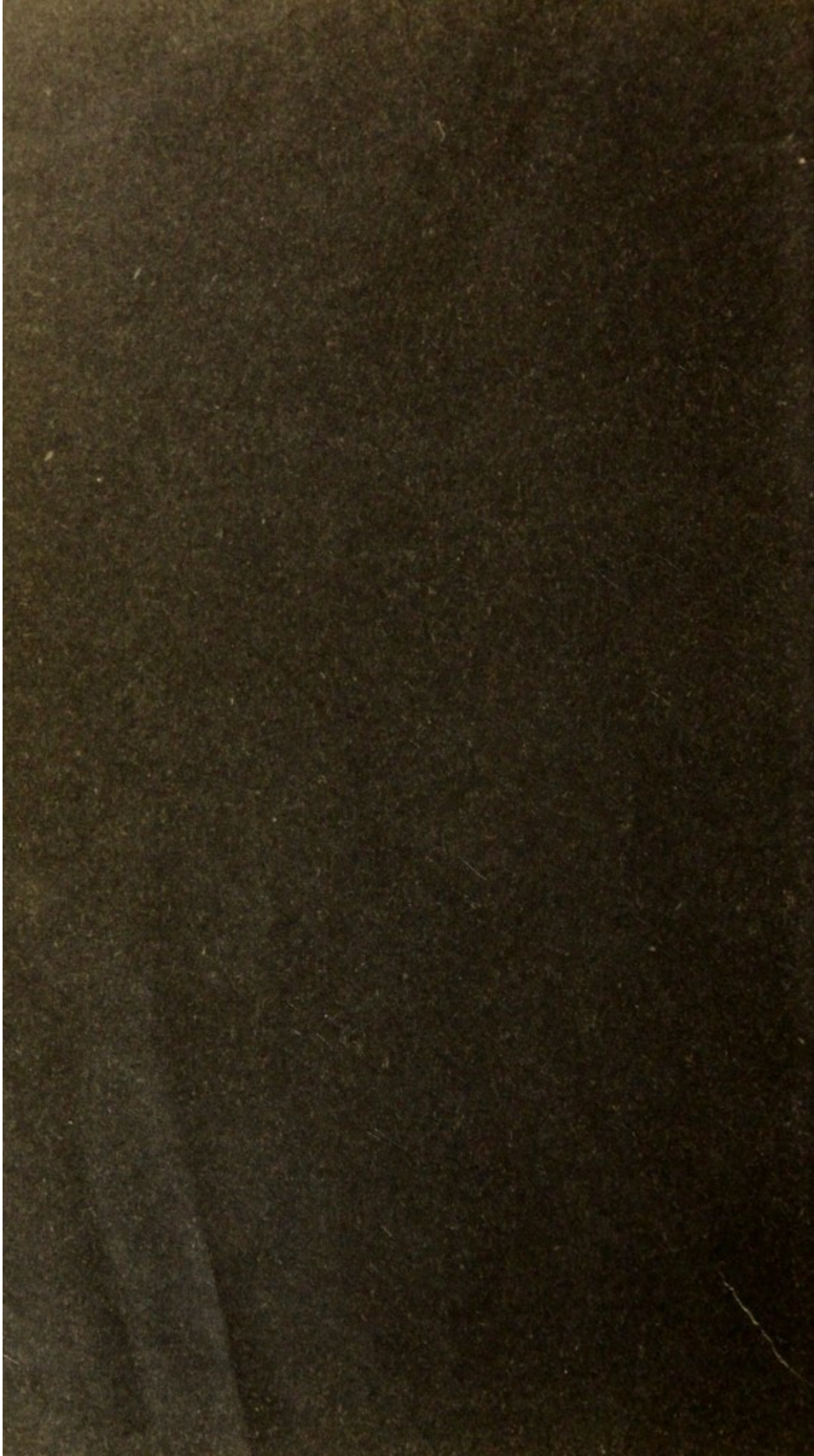
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DISCUSSION ON ANÆSTHETICS
IN THE
MEDICO-CHIRURGICAL SOCIETY OF GLASGOW.
31ST OCTOBER, 1890.

The President, DR. WILLIAM MACEWEN, in the Chair.

THE PRESIDENT'S CONCLUDING REMARKS ON
ANÆSTHETICS, WITH SPECIAL REFERENCE TO
CHLOROFORM AND ETHER—THEIR PHYSIOLOGI-
CAL ACTION, THEIR RELATIVE VALUE, THEIR
DANGERS, AND THEIR MODE OF ADMINISTRA-
TION.

GENTLEMEN,—It has fallen to my lot, as President of this Society, to perform the somewhat onerous task of making the concluding statement on this discussion on anæsthetics. Whatever may be the outcome of this discussion, those who have regarded for three nights in succession the large audience assembled in this hall, must have been convinced that the profession have an intense interest in the subject of anæsthesia, and have shown a manifest desire, while extending that inestimable blessing, the alleviation of pain, to do their utmost to secure the safety of the patient.

The subject has been discussed from two distinct stand-points, that of the physiological experimenter, and that of the clinical observer. The majority of speakers have confined their remarks to either one or other of these sides. It ought not to be forgotten that both must draw their conclusions from physiological phenomena, the one supplied by the lower animals, the other by man. The experimenter on lower

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animals has the advantage of making direct and exact observations graphically recorded, which, however, are sometimes vitiated by the necessary interference with the functional subtleties of the complex mechanism embodied in animal life. The surgeon is often content with the consummation of complete anaesthesia, without analysing each step in the process by which it has been obtained. The evanescent nature of the phenomena render the clinical observer doubtful on many points which he is debarred from settling by an appeal to direct experiment. Such can only be ascertained clinically by retaining in the midst of abundant experience a sufficiently receptive and retentive mind, which, while noting the constantly recurring phenomena, can detect the deviations from the normal, and the circumstances under which they occur.

Within a comparatively recent period, four reports have been issued on the physiological action of chloroform—that of the Glasgow Committee on anaesthetics; that of the Hyderabad Commission; that of Dr. M'Williams, of Aberdeen; and that by Dr. Kirk, on primary shock under chloroform. From a perusal of these reports, it is clear that the observers have not come to a unanimous finding; they differ from one another in many important points.

The most prominent of all the reports is that of the Hyderabad Commission. One cannot help expressing admiration at the princely generosity of the Nizam of Hyderabad in issuing the chloroform Commission. His act evinces deep human sympathy, guided by the intelligent appreciation of a cultured mind, and is well worthy of imitation.

Let us look briefly at a few points discussed in these reports, and in doing so, attention is directed to, first, the diminution of blood pressure; second, the causation of primary syncope.

BLOOD PRESSURE DIMINISHED.

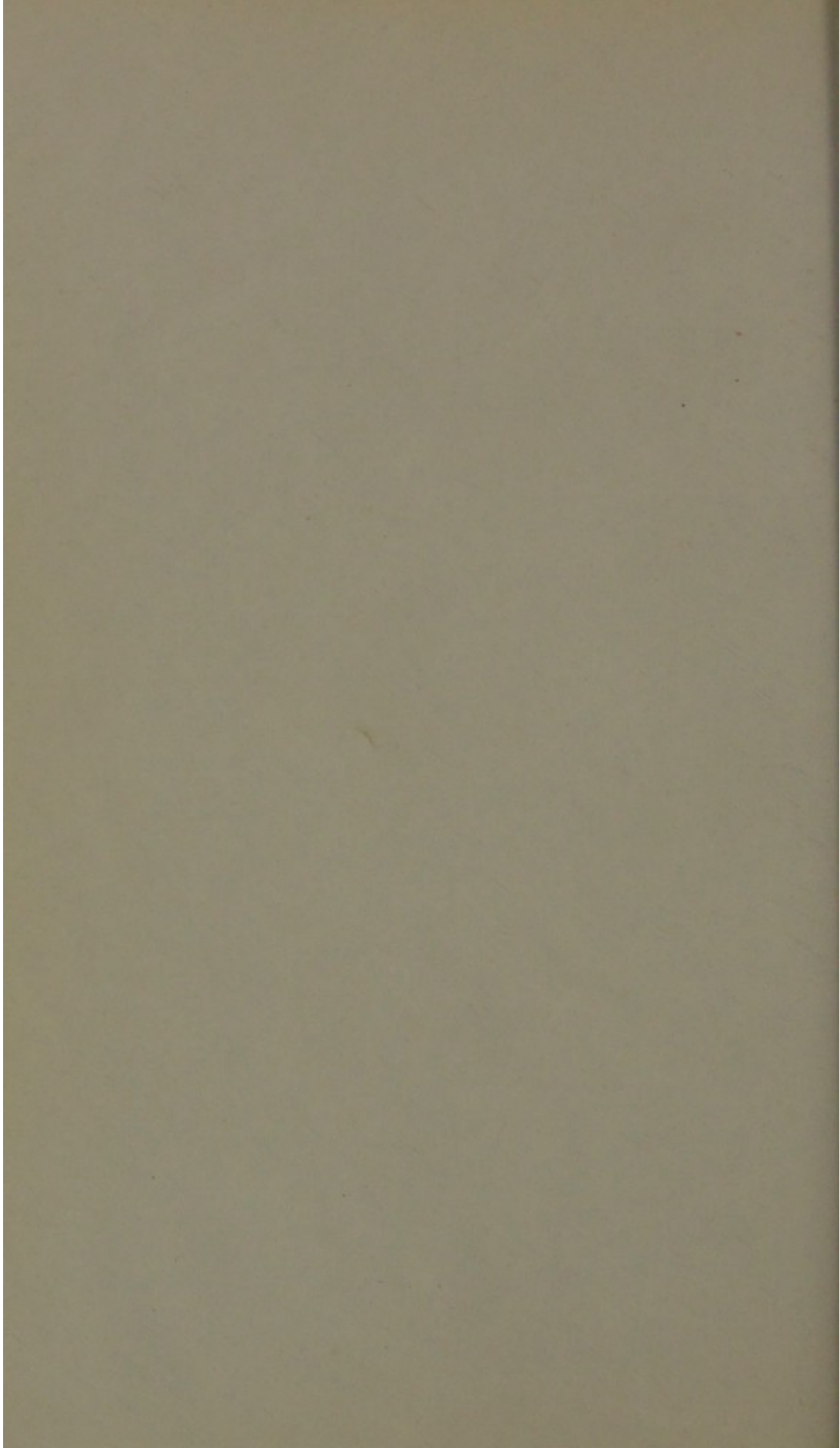
All are agreed that under chloroform blood pressure is diminished, though they are not equally so as to the cause of this. Dr. Coats believes it due either to cardiac weakness, or to diminution of peripheral resistance, brought about by dilatation of the arterioles. Dr. M'Williams rather favours the latter view, regarding it as primarily due to a depressing influence on the vaso-motor system, leading to arterial relaxation, and, when very marked, is accompanied by pronounced cardiac dilatation. He reminds us that blood pressure records cannot be relied on as giving accurate information as to the state of the cardiac action; and shows that carotid

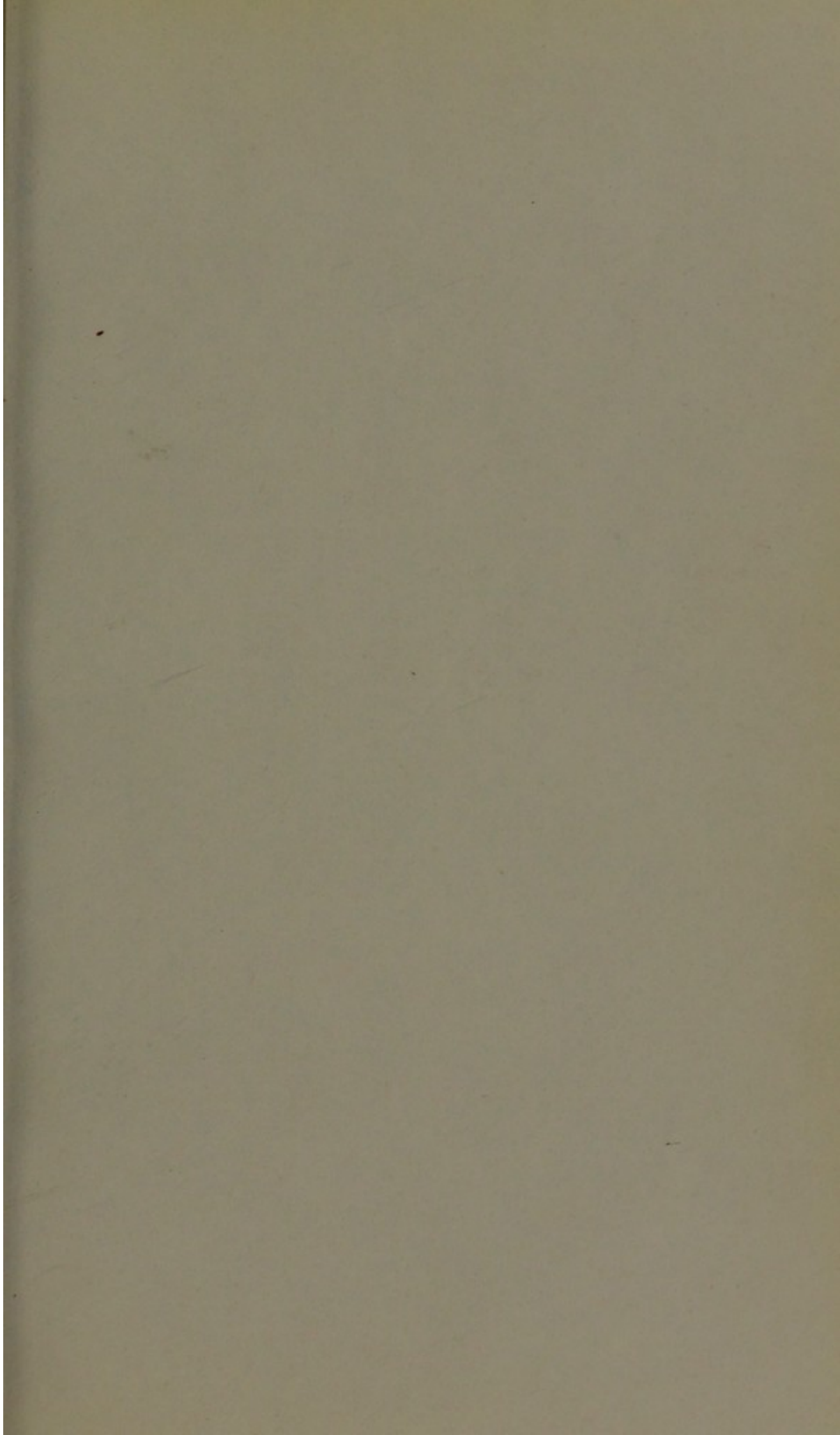
pressure can be easily raised by firm pressure on the abdominal aorta. The Hyderabad Commission report that chloroform causes a fall in blood pressure at a rate which bears a close relation to the degree of concentration of the anæsthetic administered. When given gradually, the fall is gradual; and if given concentratedly, the fall is more rapid; but, however concentratedly it may be administered, it never produces sudden death from stoppage of the heart.

Clinically, experience shows that in many, possibly the majority of cases, there is a fall in blood pressure, and this is marked where chloroform narcosis is prolonged. It is questionable whether this fall, if kept within bounds, is objectionable, and, in certain patients, it may be of advantage.

THE CAUSATION OF PRIMARY SYNCOPE.

This is a point over which physiologists are at variance. The Hyderabad Commission deny its existence as arising from the action of chloroform, though they admit it may arise from reflex action, or from shock, when the patient is not fully under the protecting influence of the anæsthetic. Dr. Coats, while believing that chloroform may directly cause primary syncope, attributes it to various causes, but mainly to reflex inhibition of the intrinsic cardiac ganglia. Therefore, though not ostensibly, he yet so far agrees with the conclusion of the Hyderabad Commission in attributing the primary syncope to reflex action. He believes, however, that chloroform has a direct influence in bringing this about, by removing the control of the higher centres, and so permitting the reflex centres to act with greater potency than they otherwise would do. Dr. M'Williams looks upon primary syncope as the direct outcome of the chloroform upon the heart, causing primary dilatation of all its cavities—auricles and ventricles on both sides. Dr. Kirk states that primary syncope never occurs from the action of the chloroform, but it may do so from the sudden withdrawal of the anæsthetic, once the patient is under its influence. Dr. Kirk thinks that chloroform has the effect of causing a retardation or stagnation of the blood in the vessels, and, when this effect has been produced, and the chloroform is suddenly withdrawn, the circulation resumes its normal rapidity and causes thereby a sudden emptying of the heart. He contends, it is not the strength of the chloroform, but its sudden withdrawal, which occasions the mishap. Dr. Kirk's theory stands quite apart from the others; it has been argued with much force and skill, and from a polemical point





of view much might be said on it. The reasoning in support of the theory is purely deductive, and whether there be facts behind the scenes which would support the hypothesis, they have at least not been disclosed. Until the data are more fully supplied an opinion cannot be expressed on the subject. There can be no doubt that chloroform administration is suddenly stopped on many occasions without syncope following, and I am not aware of any case in which syncope arose from this cause. Nor is it the case that, during chloroform narcosis, there is any retardation of the blood stream, at least perceptible to the naked eye. During operations there is abundant hæmorrhage, oozing from minute vessels, welling from veins, and brisk spouting from arteries, while the patient is anæsthetised. It is one of the advantages of chloroform that it does not check bleeding while the patient is under its influence, otherwise secondary hæmorrhage would follow when consciousness was regained. Secondary hæmorrhage is seldom ever seen now, whereas, before chloroform was introduced, it was common, as the patient often suffered from shock, causing the vessels to contract and to be hidden from the surgeon at the end of the operation; they were, therefore, not secured, and secondary hæmorrhage occurred as reaction set in.

Though Dr. M'Williams shows that the heart dilates from the action of chloroform, it is important to note his conclusion that notwithstanding this direct cardiac action, yet in the great majority of cases of chloroform collapse, the respiration stops before the heart action becomes entirely ineffective. In healthy animals the heart, though dilated, is quite able to play its part in maintaining blood pressure, which though low, is yet compatible with the continuance of life. Though the heart be dilated it is able sufficiently to perform its function and continues to do so until respiration is arrested. The cardiac change is not usually sufficient by itself to induce a fatal result; when death occurs, the depression of the heart is usually associated with vaso-motor and respiratory failure. He gives three cases however, in which the state of the heart nearly caused death, though artificial respiration, along with rhythmical contractions of the ventricles with the fingers, restored the animals. Dr. Coats agrees with the view, that the risk of early and sudden syncope is minimised by continuous and sufficient administration, and when it does occur it is less dangerous than the other form, inasmuch as it is reflex and leaves the heart intrinsically unaffected. "Artificial respiration when used early should always produce recovery in this case." The Hyderabad Commission state that collapse may be recovered

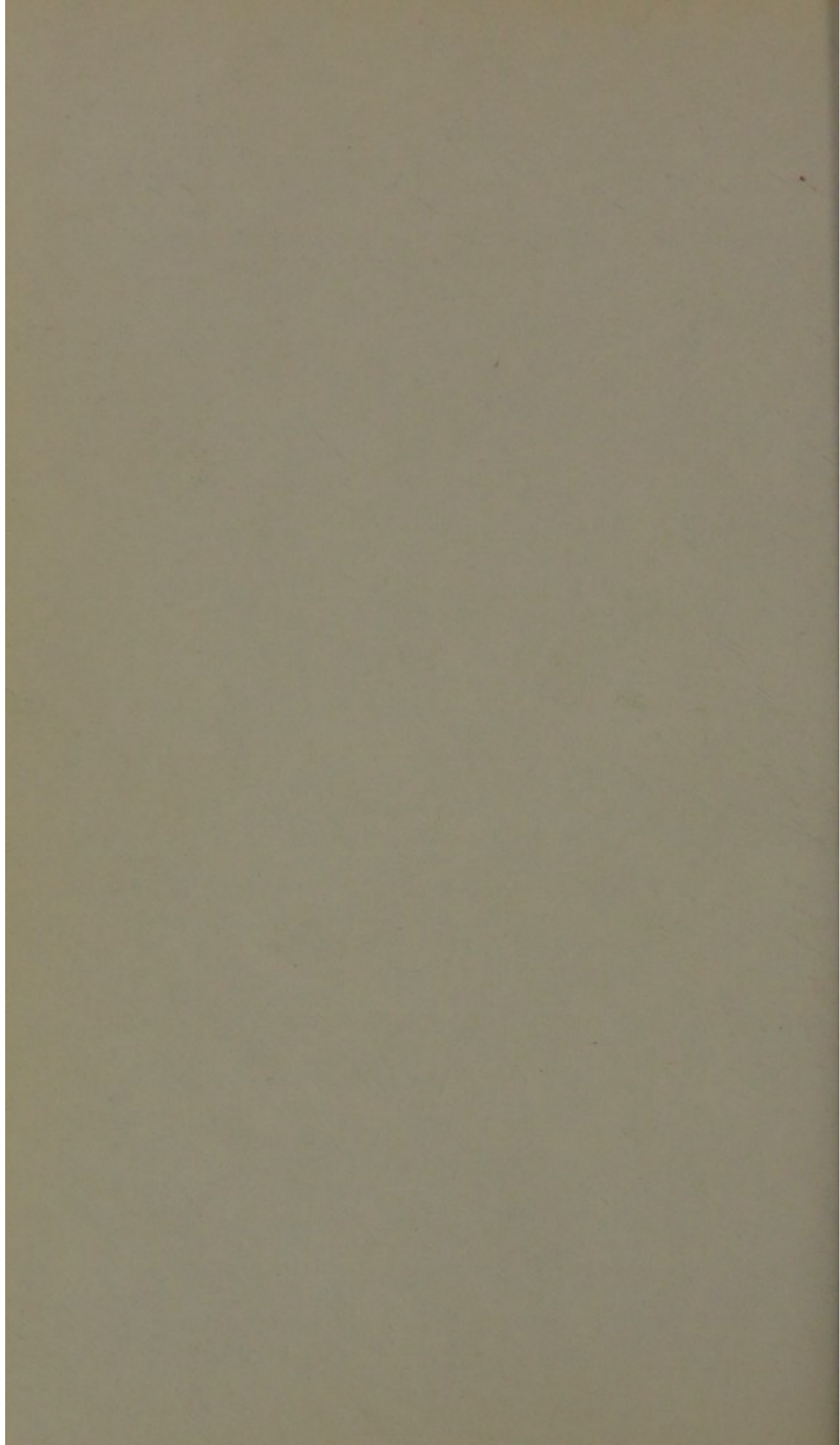
from, provided artificial respiration be commenced *within thirty seconds* from the time of the cessation of respiration.

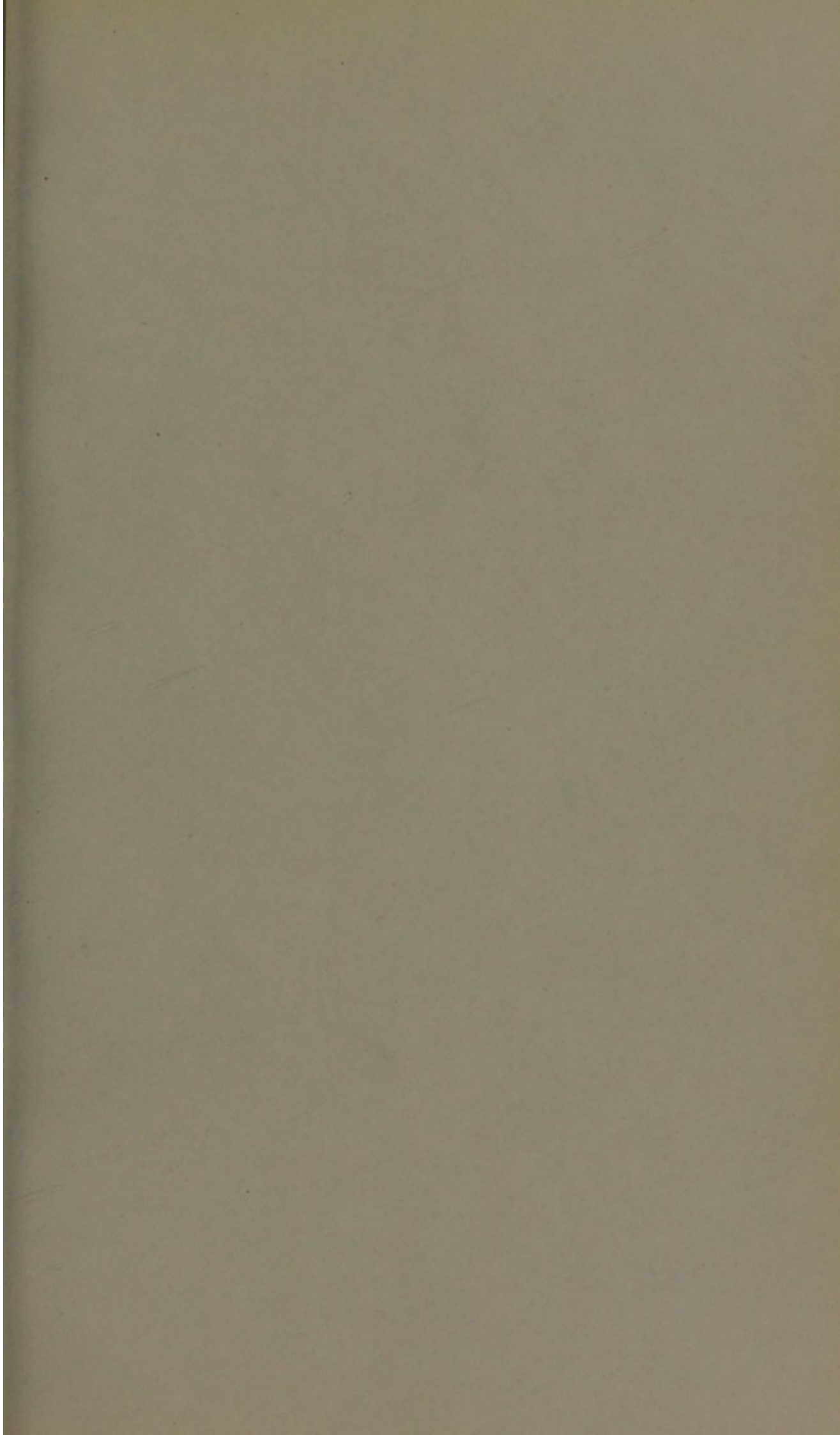
So that, whatever diversity of opinion be entertained by physiologists regarding the causation of primary syncope, they are much nearer unanimity as to its prevention. They agree that careful continuous administration of the anæsthetic, with vigilant attention to the respiration, will, in the great majority of instances, prevent primary syncope. Let us look at some clinical experience as bearing upon primary syncope.

Primary syncope is exceedingly rare during the early stage of chloroform narcosis, but many accidents occur during the early stage of chloroform inhalation which are erroneously attributed to syncope. Accidents in the primary stage of chloroform inhalation generally occur about the period of "struggle." It is important to bear in mind that all accidents occurring while a patient is under the influence of an anæsthetic are not necessarily *from* the anæsthetic.

THE PERIOD OF STRUGGLE IN CHLOROFORM ANÆSTHESIA :
IS IT DUE TO ACCUMULATION OF CARBONIC ACID IN THE
BLOOD ? AND DOES IT THROW LIGHT ON SOME FORMS OF
PRIMARY SYNCOPE ?

From the manner in which chloroform is often administered, there is a period of struggle before the patient passes fully under the influence of the drug. The intensity of the struggle varies greatly, and seems to bear some relation to the mode of administration. Concentrated doses increase the struggle, while abundant dilution of the chloroform vapour permits the majority of patients either to escape it or to have it only in a very modified form. If an analysis be made of this stage of struggle, when it has assumed an intense form, it is seen that besides the violent contractions all over the body, it is accompanied by violent expiratory efforts, with little or absolutely no inspiratory ones. The aspect is one of suffocation, and so intense are the muscular contractions, it is often with great difficulty that the lower jaw can be prized open. If one has observed the pulse, he will perceive that just preceding this stage there is a rise in the arterial pressure, and it may be presumed (as one cannot examine the pulse, as a rule, during this stage of struggle) that this arterial pressure is maintained or greatly increased during this period of violent expiratory effort. Immediately after this period of struggle has passed, the patient is found to be fully anæsthetised. Occasionally, however, he is plunged into a deep state of coma, which passes





quickly into a highly dangerous condition. The muscles are absolutely limp, the reflexes abolished, the pupils widely dilated, the breathing shallow or suspended, the blood pressure reduced to zero—the pulse being imperceptible.

Some would attribute this state to syncope, but the cardiac failure is here distinctly secondary.

The condition described during struggle very closely resembles the second stage of carbonic acid poisoning (see *Landois and Stirling*, vol. i, p. 283). Is it possible that carbonic acid poisoning may be the cause of this state? It is not necessary that it be administered to the patient in order to induce carbonic acid poisoning. All that is needed is to diminish the elimination of the carbonic acid from the body of the animal by depriving it of a sufficient supply of oxygen. This is seen in a marked degree when nitrous oxide is administered. Nitrous oxide has no intrinsic anæsthetic property, but it produces anæsthesia indirectly by depriving the blood of oxygen, and so preventing the elimination of the carbonic acid, which then acts on the nerve centres, producing anæsthesia. This may also obtain when chloroform vapour is administered in too concentrated a form, preventing a sufficient supply of oxygen, and hindering the elimination of carbonic acid.* It is also possible to induce it by mechanical obstruction to respiration, such as by paralytic closure of the glottis. So it is possible some sudden syncopes seen under chloroform may be due to reflex action, induced by the action of carbonic acid gas upon the periphery of the vagus in the lung. Therefore, such syncope is secondary to the asphyxia, and a direct result of it.

* Wood has shown that "carbonic acid, when breathed into the lungs alone, sometimes causes a slight and temporary rise in the arterial pressure, but usually at once, and always after a very short time produces a very decided fall of arterial pressure. On the other hand, when carbonic acid is taken *properly diluted*, it distinctly increases the arterial pressure, apparently having most power when mixed with oxygen in the proportion of two to one." Dr. Wood's experiments also point to the fact that carbonic acid has a double action, that "by stimulation of the vaso-motor centre it acts to increase the pressure, while by inhibiting the heart, it acts to lessen the pressure. It is also clear that the carbonic acid acts on the branches of the vagus in the lungs, as when the vagi were cut the inhalation of carbonic acid no longer produced the distinct slowing of the pulse." (*A Research to Determine the Action of Nitrous Oxide, Nitrogen, Oxygen, and Carbonic Acid upon the Circulation, with Special Reference to Nitrous Oxide Anæsthesia.* By H. C. Wood, Philadelphia, P.H. George S. Davis, publisher, Detroit, Mich., 1890.)

It is well to remember that Professor Rutherford arrived at the conclusion that the cardiac arrest was not due to direct reflex action, but to stimulation of the cardio-inhibitory fibres of the vagus, by the state of the blood resulting from arrest of respiration.

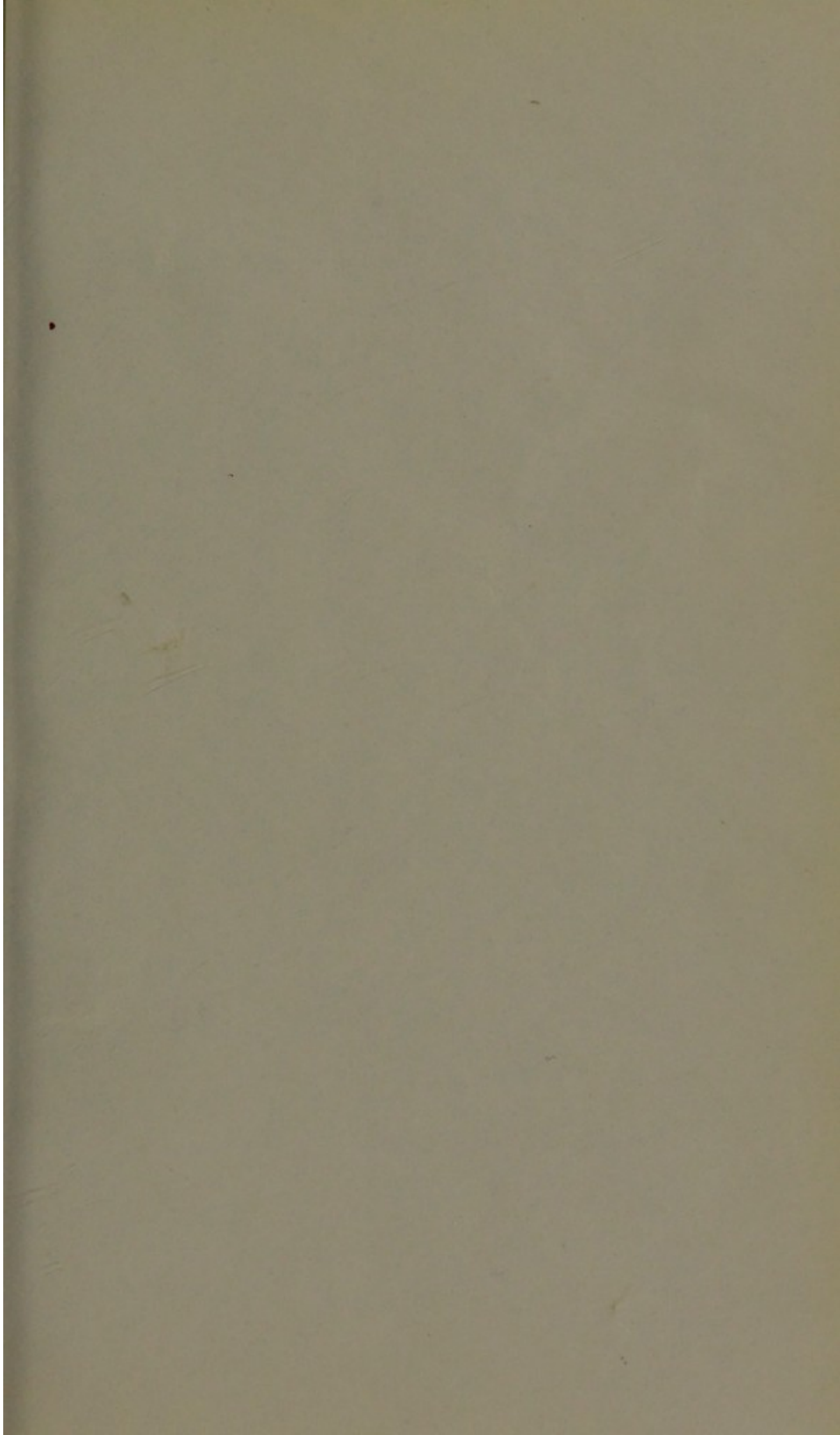
ANÆSTHETIC EFFECT OF CARBONIC ACID GAS IN LARYNGEAL OBSTRUCTION CAUSED BY DIPHThERITIC DEPOSITS.

It is interesting in this respect to recall the anæsthetic effect of carbonic acid on the human being labouring under diphtheritic laryngeal occlusion from false membrane. In the latter stages of this affection, there is, along with the lividity of the face and skin, a blunting of all the senses. A time arrives when the forced respiration is maintained by the power of the will which is only exerted fitfully. In the intervals the patient doses; the moment this occurs the respiration slows down gradually, and at last ceases, when the patient awakes with a great gasp, followed by a series of violent and forced inspiratory efforts. The pulse in such cases is barely perceptible, the arterial pressure is very low. An anæsthetic is not required in such a case for the performance of tracheotomy. The patient may wince a little when the skin is severed, but remains absolutely passive during the remainder of the operation. A slight whiff of chloroform (sufficient to destroy the intelligence) in such a state causes instant collapse, cessation of respirations, imperceptible pulse, and intensification of the lividity. The carbonic acid is apparently increased by the chloroform vapour getting into the lungs, while the channel for normal respiration is so much restricted that the usual amount of oxygen is excluded. In such a state instant opening of the trachea, and the employment of artificial respiration, with remittent pressure upon the heart—(the ribs yield in children)—quickly brings him round.

ASPHYXIA MISTAKEN FOR PRIMARY SYNCOPE, AND HOW THE MISTAKE MAY OCCUR.

Some cases of death under chloroform attributed to primary syncope are in reality due to asphyxia, the occurrence of which has been overlooked by the administrator on account of his having taken the heavings of the chest and abdomen as a guide to the respiration, believing the patient to be breathing as long as these continue. This is an entirely fallacious sign, and if depended on must occasionally lead to plunging the patient into a dangerous or fatal condition. If toward the end of this state the condition of the patient becomes alarming, and someone there examines the pulse, they find it imperceptible at the wrist, and they conclude that death has occurred by primary syncope. Conclusion No. 28 of the Hyderabad Commission report states that many cases of reported dangerous





failure of the heart under chloroform are really due to asphyxia, which the administrator has allowed to occur from *want of attention*. I agree with them in believing that many cases of apparent failure of the heart under chloroform are due to asphyxia, but I show that this may not be due to want of attention on the part of the administrator, but to a radical error inculcated by many authorities in the mode of watching the breathing, and which the Hyderabad Commission seek to perpetuate by "Practical Conclusion X," in which they say—"If possible, the patient's chest and abdomen should be exposed during chloroform inhalation, so that the respiratory movements can be *seen* by the administrator." On many occasions in the past, while a patient was under chloroform, I have observed the cessation of breathing and called the anæsthetist's attention to it, when he has assured me the breathing was right, pointing in support of his observations to the still heaving chest and abdomen, while not a particle of air was entering the lungs. Many fatalities in the lower animals occurring under the influence of the anæsthetic is due to this defective mode of *watching* the heavings of the chest and abdomen. If one observe an animal in this state, the abortive respiratory efforts which it makes cause rhythmical expansion and contraction of the lungs, which continue for a variable period, but occasionally last for thirty seconds or longer after the cessation of respiration. The same phenomena occur in man. The extreme seriousness of this mistake becomes manifest, when it is remembered that it is just during the first half minute after the cessation of respiration that one is able easily to restore the patient by the present use of artificial respiration. Artificial respiration ought to be resorted to even after the lapse of several minutes from the cessation of respiration, but the restoration becomes more and more difficult the longer it is delayed. (The proper mode of observing the respiration is given further on.)

PRIMARY SYNCOPE FROM SHOCK OR POWERFUL PERIPHERAL IRRITATION.

This may be caused in a variety of ways. First, by shock produced by peripheral irritation inhibiting the intrinsic cardiac ganglia. Such shock occurs quite independently of the chloroform, and may be seen in any recipient of severe injury. It was not unusual in patients who were subjected to painful operations before the days of anæsthetics. Whether a small quantity of chloroform increases the danger of syncope, by

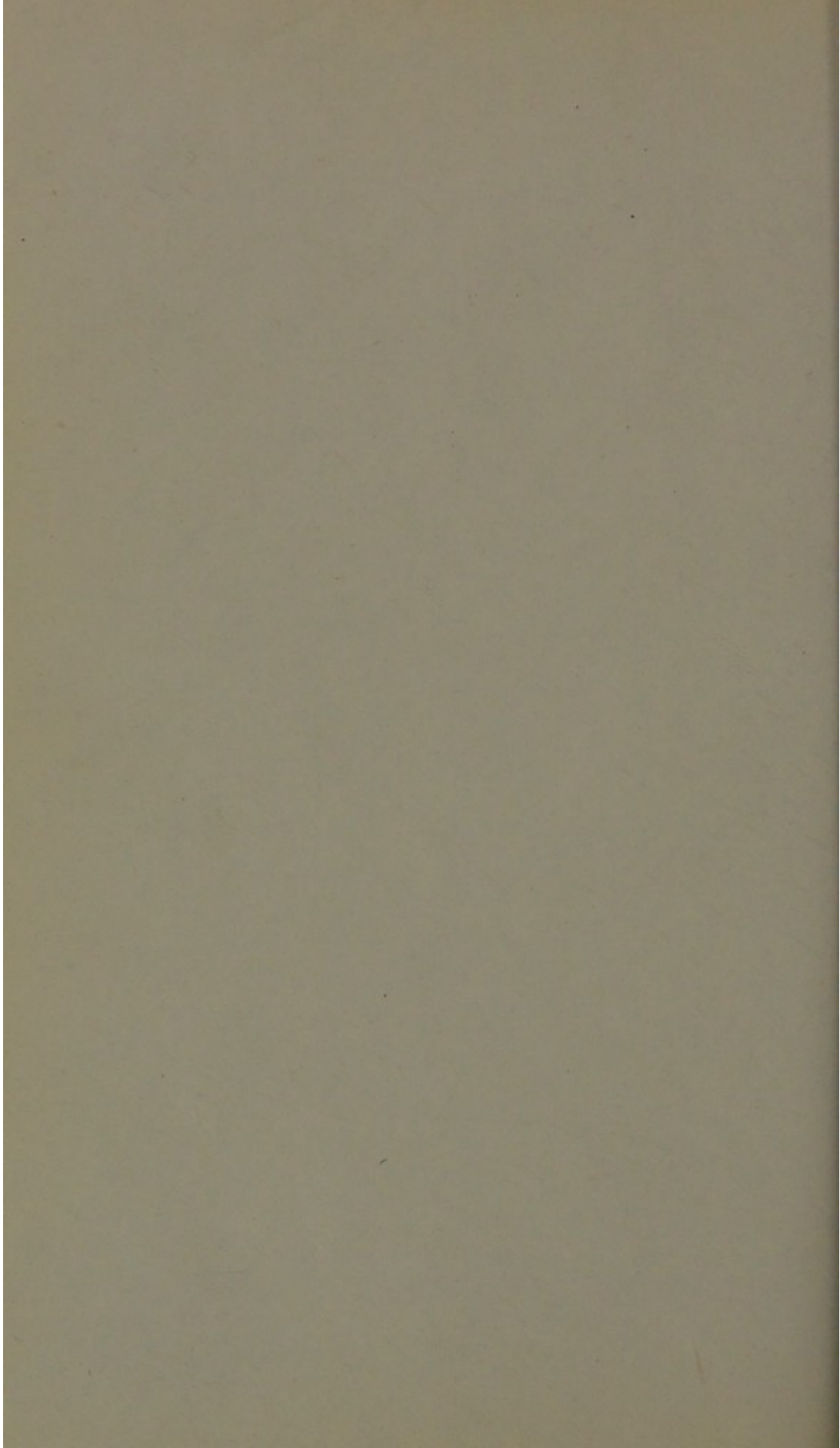
diminishing the power of the higher centres, and thus allowing the reflexes to become more dominant, is questionable, though it is quite conceivable and has an analogical foundation. In people who have a "highly nervous temperament" very slight peripheral irritation may be sufficient to induce syncope.*

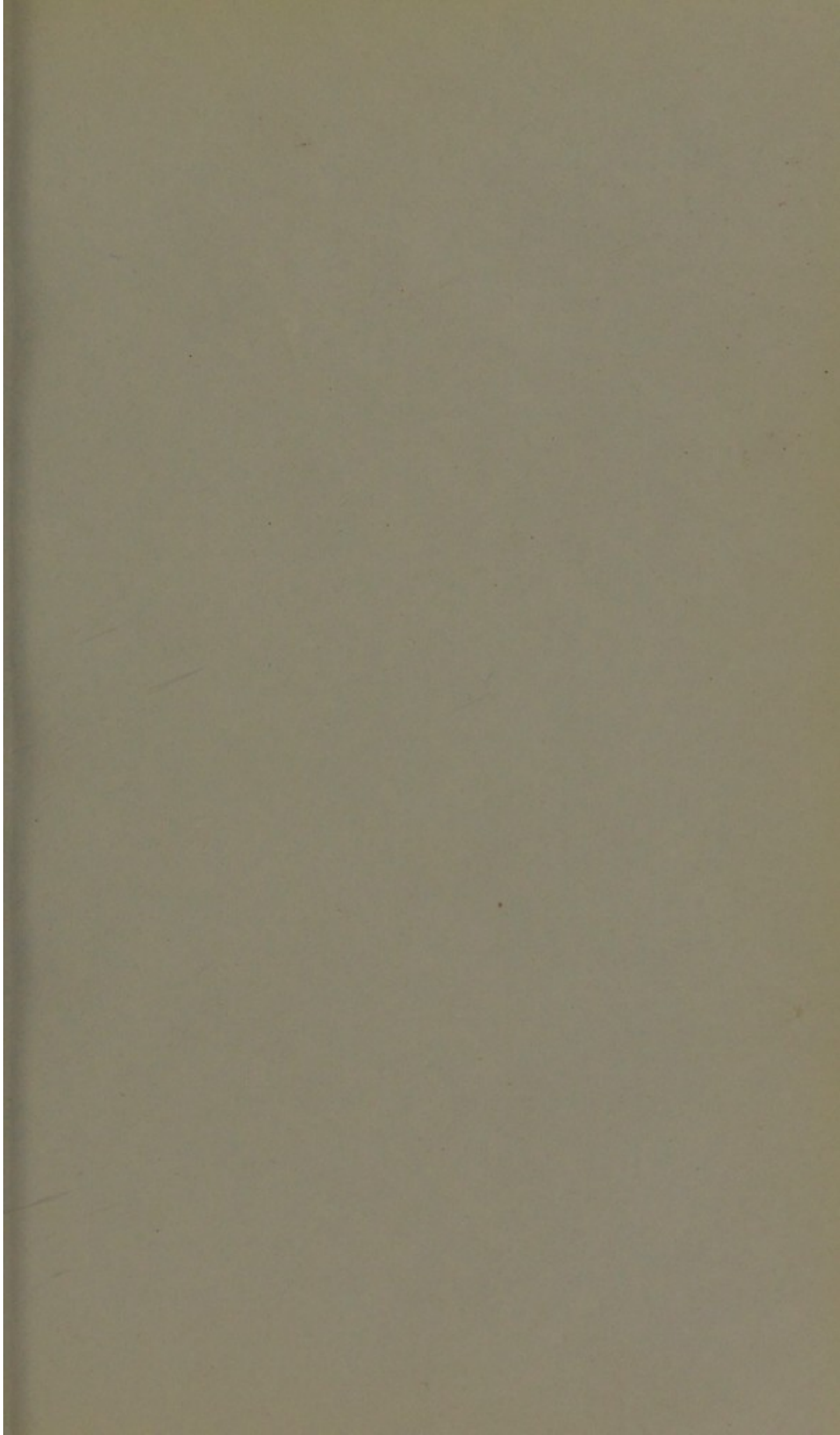
When primary syncope does occur, one of its chief causes is admittedly reflex inhibition of the cardiac ganglia. This form of primary syncope is clearly preventible. Not only is it not caused by the chloroform, but, were a sufficient amount of chloroform administered, it would safeguard the heart from such peripheral impressions. In order to secure this end, the chloroform ought to be administered until the reflex function of the central spinal axis is abolished, and the muscles lie perfectly relaxed. Before this stage has been reached, no act calculated to produce pain ought to be done to the patient, and, of course, no surgical operation ought to be permitted. It is important to note that in some, at least, of these cases the conjunctival reflexes are said to have been abolished before the operation was commenced. I believe, when primary syncope does occur, it must often do so in this way. This form of primary syncope is preventible, inasmuch as the patient ought to be placed fully under the influence of the anæsthetic before any act which could produce pain, or an operation, is commenced. It ought always to be remembered that chloroform protects from this very kind of mishap, but it only does so when it has produced abolition of the reflexes.

ABOLITION OF CONJUNCTIVAL REFLEXES NOT ALWAYS A TRUE TEST OF THE GENERAL REFLEX ACTION BEING PLACED IN ABEYANCE, HENCE THERE IS DANGER IN TRUSTING IT ALONE.

Many surgeons and physiological experimenters take the conjunctival reflex as an absolute guide to the abolition of all the other reflexes. Though this obtains as a broad general rule, there are many exceptions. Often the conjunctival reflex is abolished when the other reflexes are yet active. This is apt to occur when chloroform vapour is permitted to come into direct contact with the conjunctiva, such as when the

* A case of primary syncope occurring in one of the London Hospitals was due to reflex inhibition. The patient, who had cardiac disease, was operated on before chloroform narcosis was induced—only having had a few whiffs. Amputation of penis was performed with one sweep of the knife, when patient suddenly died from syncope. Dr. Cameron has detailed to us another case of primary syncope occurring in his own practice.





eyes are kept open while chloroform is administered on a towel. The abolition of the reflex is then due to the local effect of the anæsthetic vapour on the conjunctiva. This has been observed by me in over fifty cases. It is clear that, were the surgeon judging the depths of the anæsthesia by the conjunctival reflex alone, and then commencing a severe and painful operation, primary syncope might ensue from reflex action. There has also been observed, in a number of cases, persistence of conjunctival reflex, while the other reflexes were in abeyance. Therefore, conjunctival reflex when abolished does not always indicate a corresponding abolition of reflex action generally.

THE EFFECT OF POWERFUL MENTAL IMPRESSIONS AND EMOTIONAL STORMS, OCCASIONED WHILE UNDER THE ANÆSTHETIC, IN THE PRODUCTION OF PRIMARY SYNCOPE.

Primary syncope may also be induced during first stage of chloroform administration by powerful mental impressions, or violent emotional storms, arising while the patient is going under the influence of the anæsthetic. Everyone knows of instances of powerful mental impressions producing syncope in ordinary life. On one occasion a man of 28 years of age, who was supposed to have had no cardiac affection, was crossing a street when his road was blocked by a vehicle which he had not observed. He started back, turned deadly pale, and fell in a heap, dead. He had had no physical injury; the vehicle was not near enough to do so. Again, a few words written on a paper have been known to fell a man as effectually as a blow from a sledge-hammer. Under chloroform it is quite clear that mental processes are carried on, as may be learned from the conversation of the half anæsthetised patient, ranging from amatory declarations to violent altercations, accompanied by tremendous struggles. A state of mental terror would produce a marked depressing effect upon the heart, already enfeebled by the action of the anæsthetic. In a patient who was on three occasions placed under the influence of chloroform, there occurred, during the first stage of narcosis, on each occasion, indications of deep mental depression, with wringing of the hands and a burst of grief, during which the pulse lowered in volume till it became imperceptible at the wrist, and this was accompanied by excessive blanching of the face and feeble respiration. They were, on each occasion, relieved by lowering the head for a few minutes, after which she passed easily under the anæsthetic, without further disturbance. This patient had no recollection of her grief when she

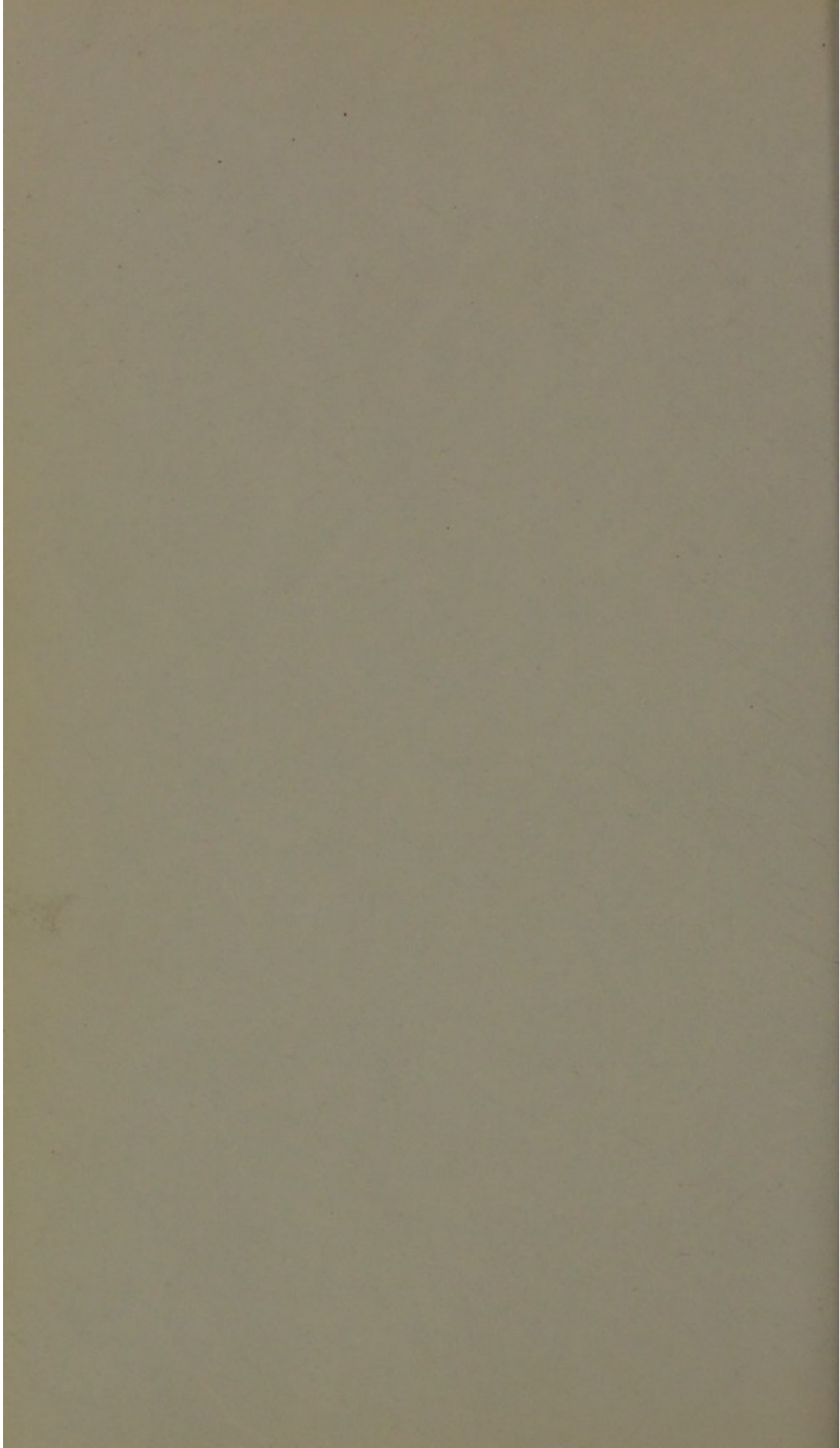
recovered from the influence of the anæsthetic. She, however, said that anything which caused her mental annoyance sent a "stoon" to her heart. Another patient did remember the state of mental anguish she passed through in the first stage of chloroform narcosis, which had a somewhat similar effect to the above. The bearing of the recollection of these vivid dreams upon the groundless accusations sometimes formulated against medical men who have placed patients under an anæsthetic is obvious. This condition depends on the state of the patient's mind more than on the anæsthetic. One cannot blame sleep for producing nightmare.

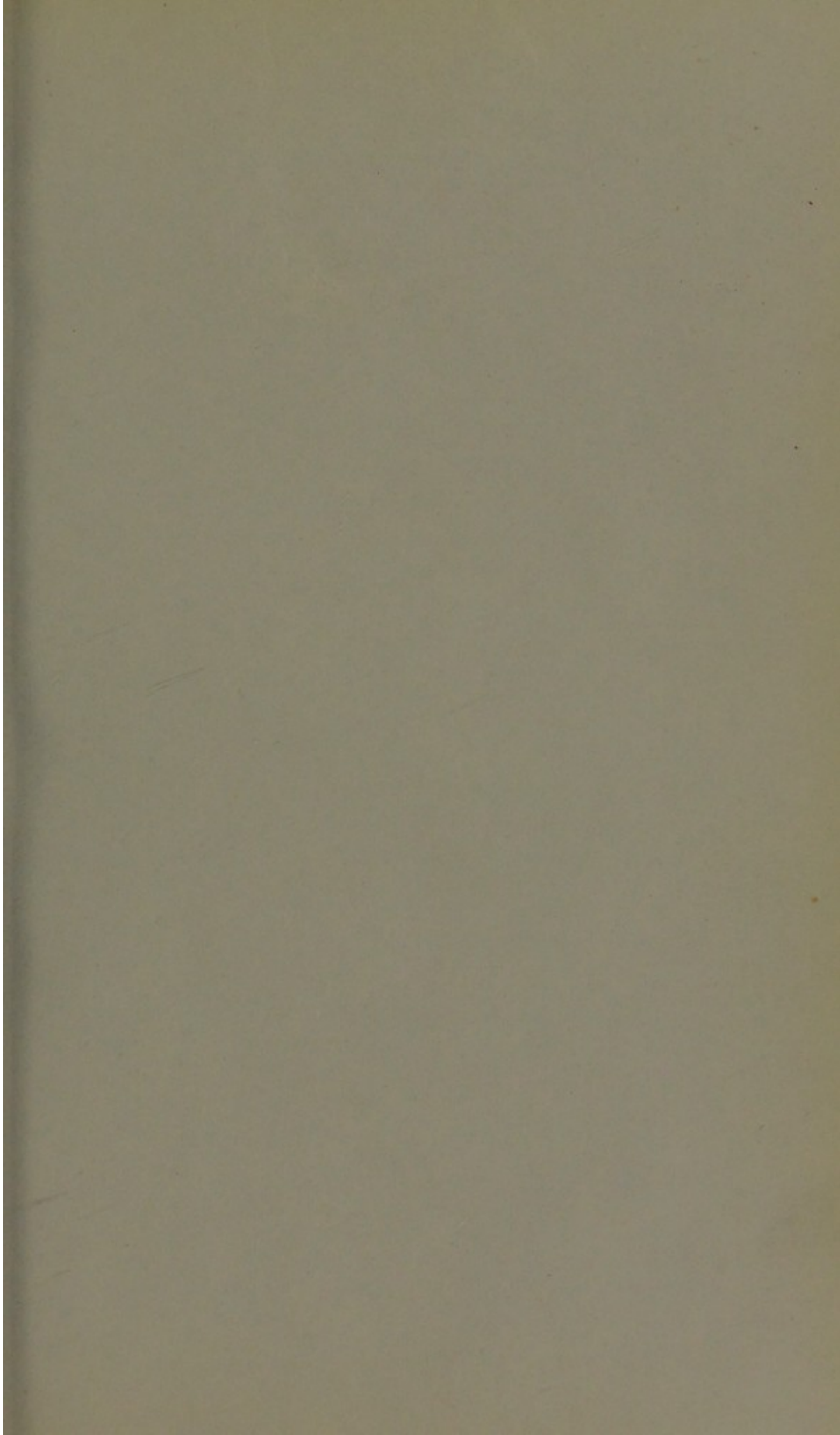
THE EFFECT OF FRIGHT OR TERROR PRODUCED PRIOR TO ANÆSTHESIA ON THE PRODUCTION OF PRIMARY SYNCOPE.

The state of fright into which some patients are plunged by the mere anticipation of an operation tends to produce syncope during the earlier stages of chloroform narcosis. Fear may be induced by ostentatious examination of the heart just before the patient is to be anæsthetised, or by making injudicious observations in the hearing of the patient calculated to excite alarm.

The following case has been related to me. A weak woman was placed on the operating table, and was about to be anæsthetised. The anæsthetist took up a chloroform bottle and was about to pour some of its contents upon the towel which covered her face, when the surgeon abruptly intercepted him, declaring he was never to give chloroform to a patient of his, as it was a most dangerous drug and occasioned many deaths. The surgeon then directed the anæsthetist to use ether. It is positively stated that the patient did not inhale any chloroform, and yet, when the folded towel was removed from the patient's face to substitute it for the ether apparatus, she was seen to be deadly pale, the heart had ceased to act, and, notwithstanding all their efforts, its function was not restored. This woman died from fright, and in the opinion of some of those who were present, the fright was occasioned by the injudicious remarks made at a critical moment in her hearing. Supposing a small quantity of chloroform had been administered in this case, the death might then have been attributed to primary chloroform syncope, instead of to its rightful cause—fright.

On many occasions I have seen patients who were in dread while being placed under the influence of the anæsthetic, become very much depressed during the first stage of both





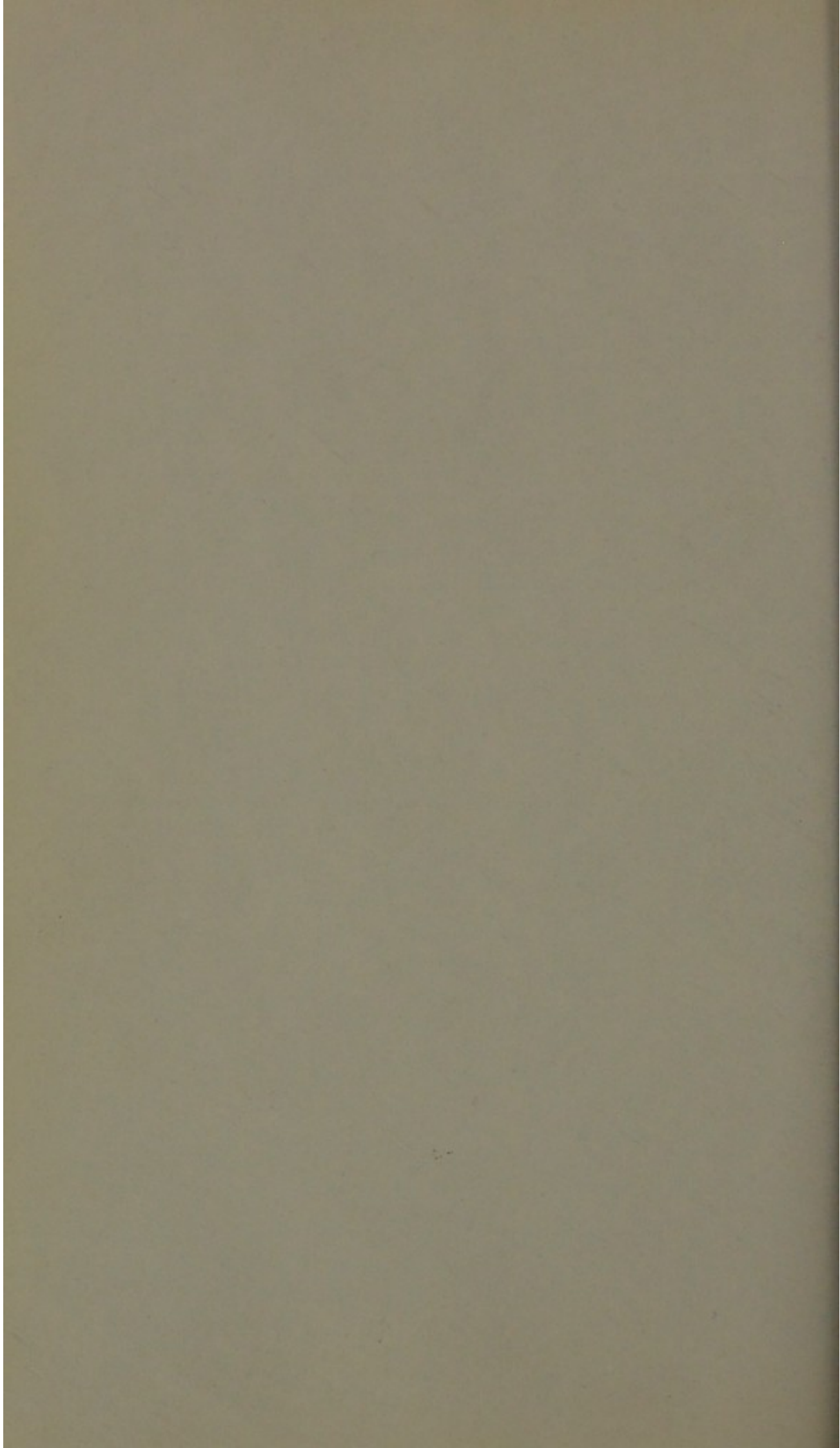
chloroform and ether narcosis, though much more often while under the former. Syncope occurred in some, and in others the depression went very near to it.

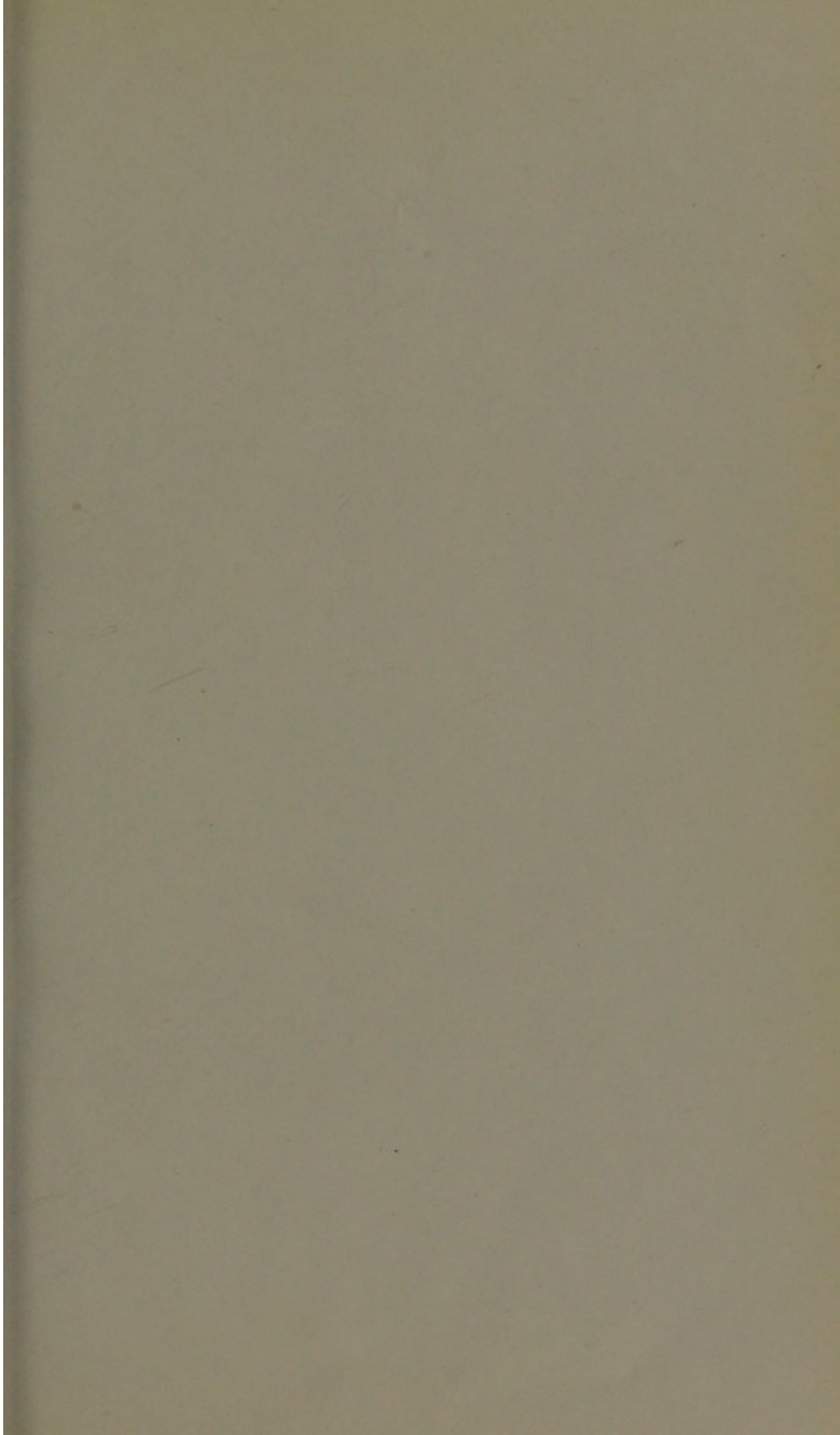
Under such circumstances, when the operation can be postponed, it is better to do so rather than subject the patient to the risk of syncope. But the administration of brandy before the anæsthetic is given is a powerful guard against this effect. In an adult an ounce, or even two if the fright be great, given five minutes before the commencement of the anæsthetic, steadies the pulse, dispels the fright, shortens the first stage of chloroform narcosis, and enables the patient to slip quietly under the influence of the anæsthetic. It has been alleged that alcohol causes vomiting. This is difficult to prove, owing to the frequency with which vomiting occurs under chloroform; but even if it be granted, the good derived greatly outweighs the trouble occasioned by the vomiting. I have never seen reason to regret the giving of this form of Dutch courage; on the other hand, a mixture of one part of chloroform to two of ether serves excellently well in such cases, the stimulating effect of the ether being of great service.

PRIMARY SYNCOPE OCCURRING DIRECTLY FROM THE EFFECT OF THE ANÆSTHETIC UPON THE DISEASED BRAIN.

Many surgeons deny that primary syncope can occur in any other way than by reflex action, and the Hyderabad Commission are particularly strong in supporting this conclusion. Some surgeons also declare that no death can occur under chloroform narcosis except from negligence. As I cannot agree with these statements, you will allow me to relate the only case of death under an anæsthetic which I have seen in my own practice. The patient was a shoemaker about 59 years of age, the subject of pronounced carcinoma of the tongue and floor of the mouth. He was a well developed man, of rather stout build, though he had been losing flesh during a few months prior to admission. He was active and intelligent, of a cheerful disposition, and fond of a joke. He had been in the habit of drinking whisky to excess, but had stopped it for some years, because it did not "do with him." The chloroform was administered by a competent and highly intelligent house surgeon who now occupies one of the foremost positions as a lecturer. The pulse was observed by another man, quite independently of the anæsthetist. All preparations for the operation were ready, and I was standing by the patient's side

waiting till he was fully under. There was no talking going on. The anæsthetist was listening to the breathing, which was free and regular. I was looking at the patient, believing he was going easily under, when he was suddenly seized with a tremor-like shudder which ran from head to foot all over his body, at the termination of which the pulse-observer called out, "The pulse has suddenly stopped." I placed my ear instantly over his chest and could hear no cardiac impulse. There was a decided pallor over his face. He was breathing naturally and regularly at that time, as was verified by the anæsthetist, the pulse-observer, and myself. He continued to take four or five full, easy inspirations, after which the inspiration ceased. All the usual restoratives were tried, along with artificial breathing and attempted cardiac stimulation, but they were of no avail. The patient was dead. The *post-mortem* showed that though his other organs were in a fair condition for a man of his age and habits, his brain was the seat of chronic œdema, accompanied by considerable serous distension of the lateral ventricles. This condition could not have supervened during the administration of the anæsthetic, though the latter may have augmented the serous effusion to a slight, though a sufficient, extent to produce a fatal result. Subsequently his master and his companions were interviewed in the hope that some light might be thrown on the history of his disease. They declared that he had been in the same employment for over thirty years, and they regarded him as being in good health until his tongue began to be affected. He however, had been a heavy whisky consumer for a great many years, and could take six or seven glasses of whisky of an evening without "being the worse of it." Within the last two years he had to reduce the quantity very considerably, because even a small amount made him insensible, and, once he became so, he remained in this state for several days. At the New Year holiday, eight months before his admission, he had been induced to take two glasses of whisky, after which he became insensible, and remained so for three days. From this statement it was probable that the œdema of the brain had been present for the last two years of his life, and was gradually increasing during that period. A very little engorgement of the cerebral vessels, such as was occasioned by the small quantity of alcohol—two glasses—was sufficient to throw him into a comatose state. It is probable that the œdema had increased during the eight months which had elapsed before admission, and had he tried the experiment after his admission to the infirmary, a much smaller quantity of whisky





would have produced a like or even a more serious result. The small extra engorgement caused by the primary stage of chloroform anæsthesia was sufficient to increase the œdema, and to paralyse the already enfeebled cardiac centres. Thirteen years ago, when this occurred, I had not so many opportunities of becoming acquainted with cerebral diagnosis, but in making a retrospect through the experience since acquired, I hesitate to believe a positive diagnosis of this cerebral œdema could have been arrived at, with the data at our disposal before the operation. Granted the administration of an anæsthetic in such a case, the result was inevitable. Ether would have acted more surely in bringing about the same result, and would have killed in a shorter time, as it induces greater cerebral congestion.

Although presenting this fatality under chloroform as clearly due to the action upon the brain producing primary syncope, it is necessary to add, such an occurrence is rare, the majority of cases of death occurring under chloroform being no doubt due to asphyxia.

I have since found, in cases of cerebral disease accompanied by œdema, or in those in which œdema is readily produced, any anæsthetic is apt to increase the œdema. Ether does so rapidly, and is dangerous in these states; chloroform does so, but much more slowly than ether. Prolonged administration of chloroform has in such cases a marked effect. It is also interesting to see how chloroform sometimes reveals brain defects which cannot be seen when patient is not under the influence of the anæsthetic. Squints, for instance, arising from functional defects in special groups of muscles have been frequently seen while under chloroform, and these disappeared as the patient recovered from the anæsthetic. This is a subject requiring special attention elsewhere, but information has occasionally been obtained under these circumstances which helped the diagnosis of the disease.

Chloroform, as well as ether, in concentrated form, may act directly upon the brain centres, and, if pushed far enough, will interfere with the functions of the cardiac and respiratory centres. They will do so all the more easily in diseased brain conditions.

LATE SYNCOPE DUE TO RETURN OF REFLEX ACTION WHEN ANÆSTHESIA IS PASSING OFF.

When anæsthesia is passing off, one of the first indications of the returning reflex action is that of vomiting, with its

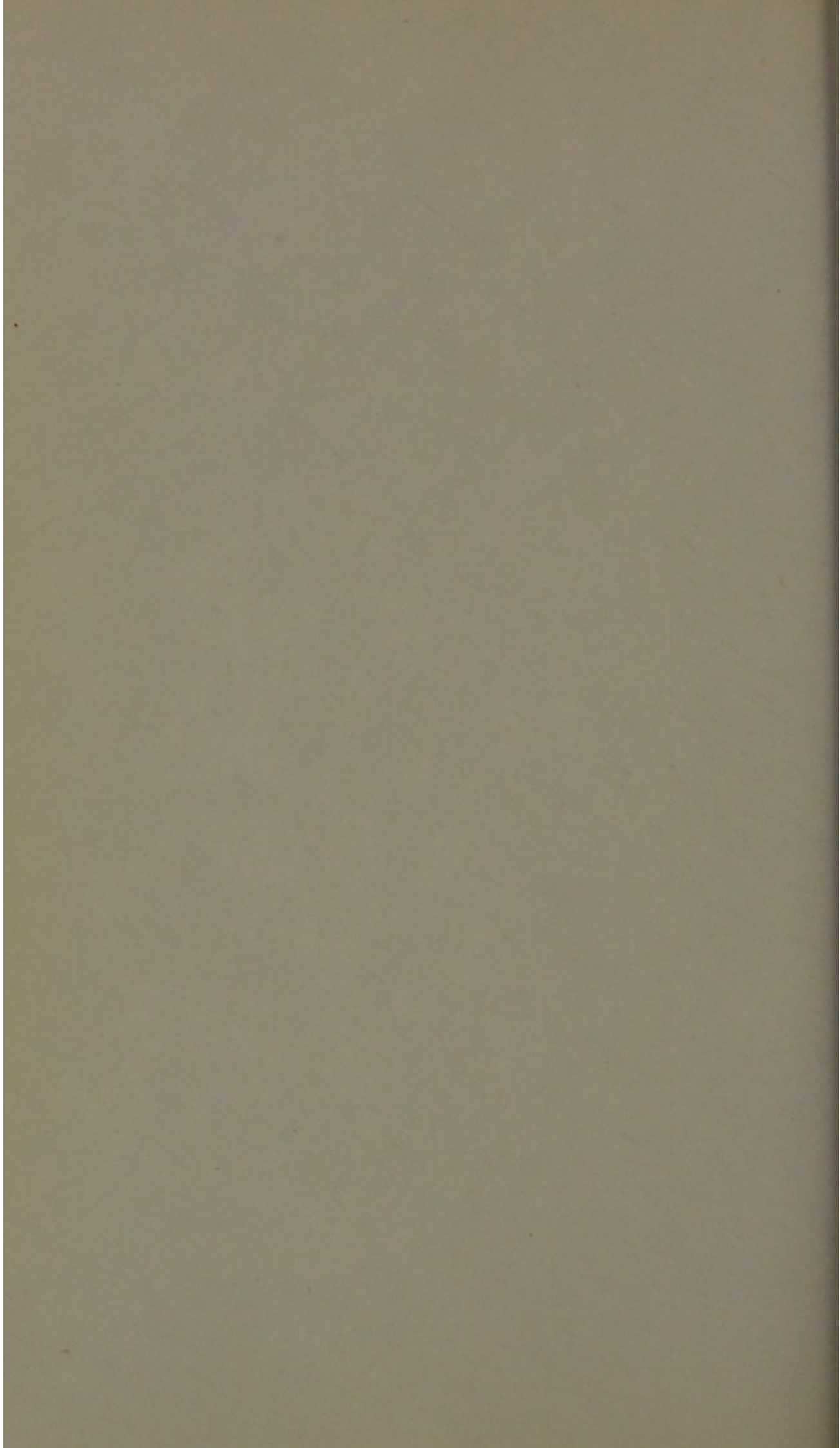
accompanying sickness. In severe sickness, even without chloroform, there is a marked depression of the pulse, pallor of the skin, dilatation of the pupils, blanching of the face, coldness of the extremities, and tendency to syncope. Under chloroform the same phenomena appear, but the tendency to syncope is the most alarming feature, and the cause of it is often overlooked, while the syncope is erroneously attributed to the direct action of the chloroform upon the heart. The depression due to the sickness may last for some minutes, occasionally for a much longer period, before being relieved by the vomiting. After the vomiting the depression disappears, and the patient's circulation is restored. Vomiting during an operation is, as a rule, an indication of a return of reflex action, and shows that the administration of the anæsthetic has not been sufficiently evenly pursued. When the administrator is sufficiently active, vomiting does not occur during the operation, though it may do so when the anæsthetic is removed. A good anæsthetist seldom permits vomiting during an operation. When vomiting is about to show itself, a little more of the anæsthetic prevents its occurrence.

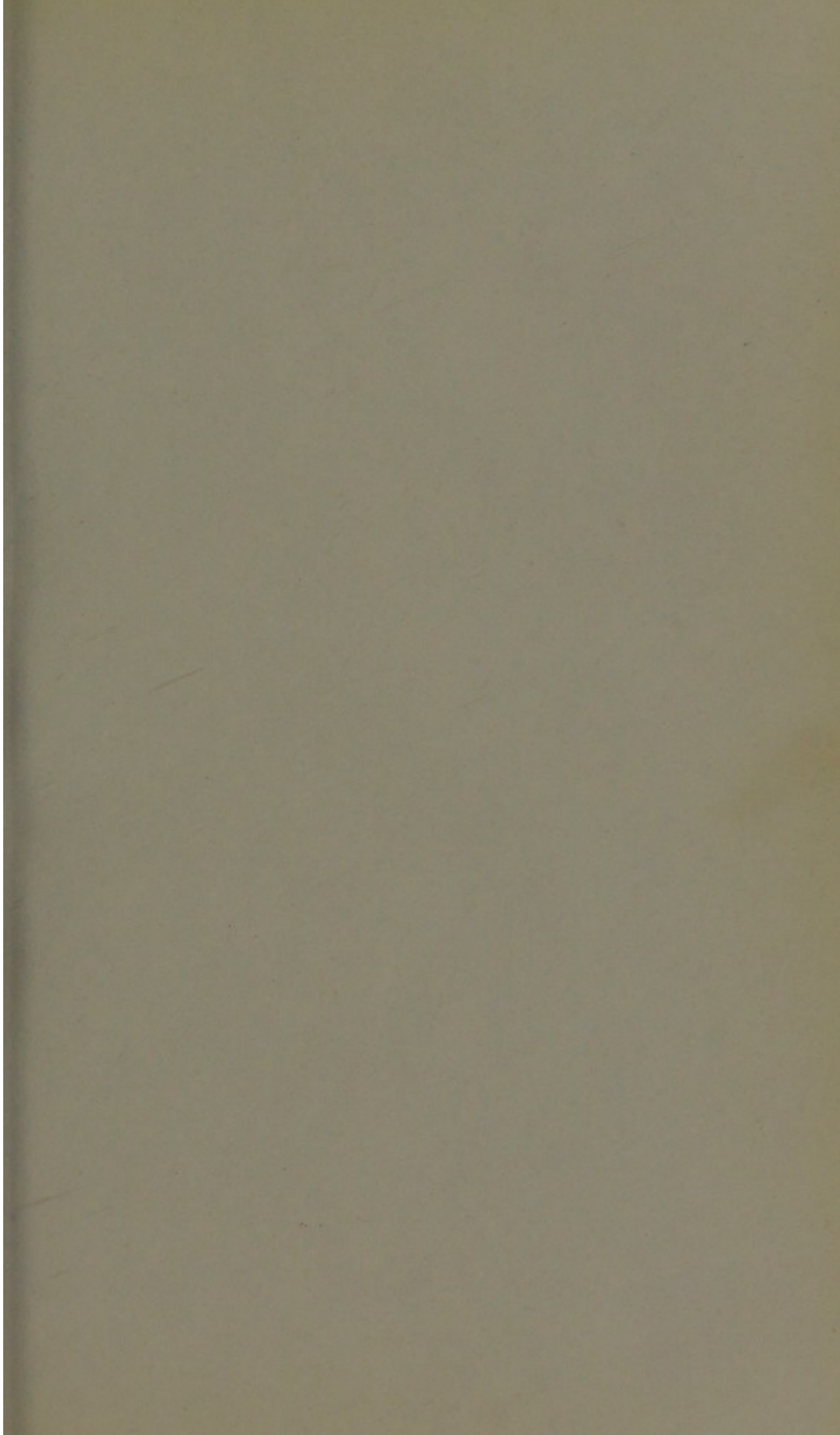
WARNING AS TO VOMITING BEING ONLY A REFLEX ACT AS FAR AS STOMACH AND ŒSOPHAGUS ARE CONCERNED.

It must not be forgotten that vomiting is only a reflex act as far as the stomach and œsophagus are concerned, and that the contents of the stomach remain in the mouth and pharynx, unless voluntarily expelled, or are favoured in their expulsion by the action of gravity. Under an anæsthetic, when vomiting does occur, it is the anæsthetist's duty to see that the vomited matter be removed from the pharynx. Deaths, both under ether and chloroform, have occurred from a neglect of this precaution. Letting the head hang over the table assists in preventing asphyxia by allowing the contents of the stomach to pass away from the larynx, and swabbing the pharynx out with a sponge on a handle is the surest way to get rid of foreign matter lodged in the pharynx. It is well to have a sponge on a handle ready for such cases.

HEART DISEASE IN RELATION TO ANÆSTHETICS, AND THE RELATIVE VALUE OF ETHER AND CHLOROFORM.

Ought a person, suffering from any of the forms of heart disease, be subjected to anæsthesia for surgical purposes?



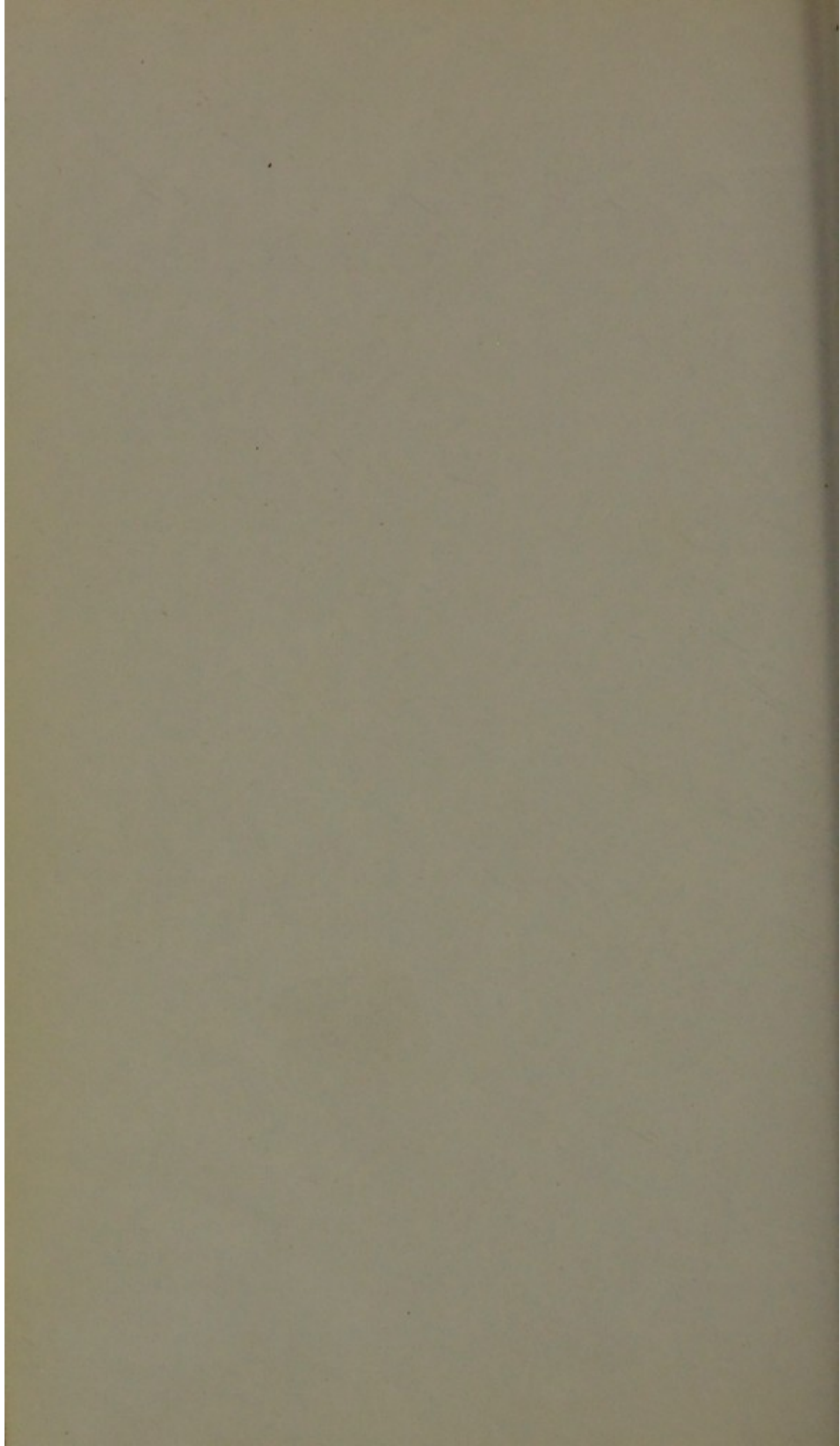


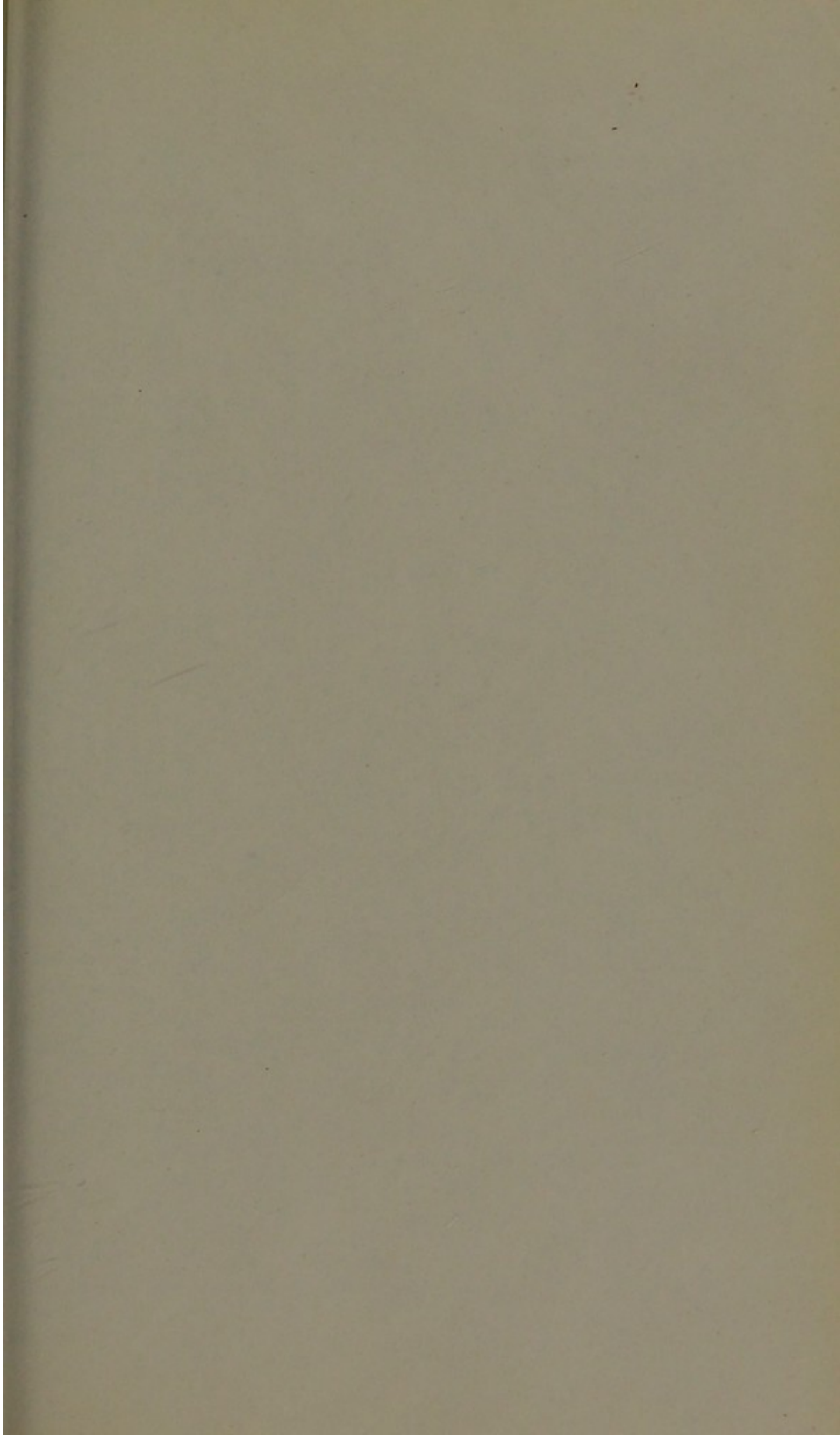
No doubt every surgeon would prefer to deal with a patient who had a healthy heart, and in the presence of advanced heart disease he would be inclined to avoid performing an operation of mere convenience. Granting, however, that an operation be required, would it be better to do this operation without an anæsthetic in the presence of heart disease? To operate without an anæsthetic would be to expose the patient to the danger of sudden death, arising from shock acting upon a weak heart. To place them under the influence of an anæsthetic would be to guard the heart against this peripheral shock. To do this efficiently, it is, however, necessary to place the patient so thoroughly under the influence of the anæsthetic, as to place the reflex actions completely in abeyance, and to judge of this latter, not by conjunctival reflex alone, but by the contraction of the pupils as well as the general condition of the patient. In this stage no peripheral shock can reach the heart, and the operation can be performed with safety. To commence the operation before thorough anæsthesia has been induced, is to expose the patient to imminent danger.

Granted that an anæsthetic has to be administered, whether will ether or chloroform be the better? Many unhesitatingly declare for ether, under these circumstances—because it stimulates the heart's action, while chloroform depresses it. No one doubts that ether is a powerful cardiac stimulant, while chloroform diminishes the blood pressure and depresses the heart. Before coming to a conclusion as to the relative value of each, we require to ask—Is it necessary that the heart be stimulated while patient is under an anæsthetic, and, if so, is the stimulation derived from ether inhalation sufficient to maintain a normal cardiac action, or does it exceed this? is it, in fact, a stimulation which can be controlled or regulated? Again, is the depressent effect of the chloroform more than sufficient to exercise a calmative effect upon the heart?

I am bound to answer, from my own experience, that the heart does not require stimulation in the great majority of cases, and, even where the cardiac action is feeble from disease, if one avoids nervous excitement, the chloroform acts well, and when the patient is fully under its influence the blood pressure, as far as it can be measured by the pulse, is good, and, in many instances, even better than during the patient's wakeful moments. When the patient can be examined before the operation, the whole of his internal organs are looked to, so that an idea of the weak points are ascertained. This is not done at the moment the anæsthetic is about to be administered, which would be a mistake, but

days previously if possible. In many cases of known cardiac weakness, aortic regurgitation, flabby and suspiciously fatty heart, valvular disease, &c., chloroform has been administered without any hitch. During the last 18 months there were, in my practice, 29 cases of cardiac affections anæsthetised with chloroform without any difficulty. In several of these the pulse was weak and soft prior to the operation; after full anæsthesia was induced the pulse became markedly improved, so much so, that those detailed to watch the pulse remarked the improvement and commented on it. So that, in many cardiac cases, the chloroform, instead of exercising a prejudicial effect upon the pulse, improves it. The *rationale* of its action in these cases may not be clear, but the fact remains. There are cases, however, where some form of stimulation would be an advantage. Ether, under these circumstances, might be employed, if its stimulating power was controllable. Ether stimulates the heart in many instances to excess; it also is apt to cause forced breathing, both of which, continued for a long period, are exhausting to the patient. Ether also tends to produce pulmonary and other internal congestions, which, of course, increase the amount of work thrown upon the heart. One would not advise for a fatty heart, or one where there was even aortic regurgitation, an increased amount of work, such as running rapidly up a hill, and yet that is much the same as what is done when the patient is put under ether. Even the advocates of ether admit that those who have a tendency to venous congestion are bad subjects for ether anæsthesia, and serious consequences are apt to arise from its use. Those who fear chloroform, fear it because in their minds they believe it is apt to induce syncope, yet this is admittedly the very way in which ether kills. Dr. Hartley pointedly made reference to this, and Mr. Vincent Jackson's case at Wolverhampton, mentioned the other day in the *Lancet*, was another instance of death under ether occurring from syncope. Further, were we regarding the danger from peripheral shock as an important point, chloroform would then be the safer, as it abolishes reflex action much more absolutely than ether, unless the patient is plunged profoundly under its influence, when ether may become dangerous. On the whole, in the majority of cases of cardiac disease chloroform is the safer anæsthetic; it calms the heart, it protects from the effects of peripheral shock, and the patient may be stimulated, if need be, by alcohol, before or during the operation. Ether not only may cause exhaustion during the operation—if it be prolonged—but the tendencies to venous





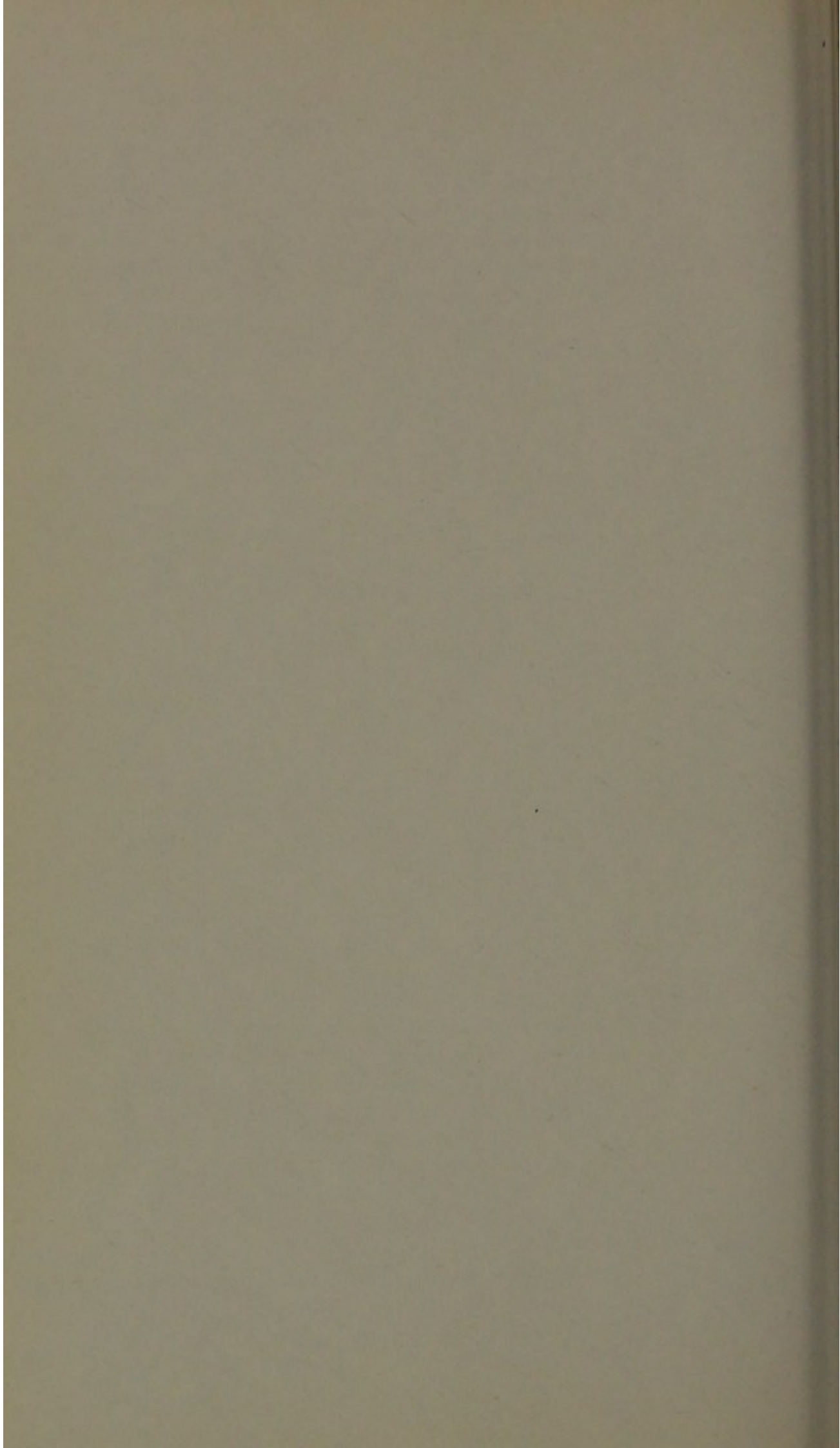
congestion in the lung exercise a further exhausting influence on the heart for many hours, and it may be for days, after the administration.

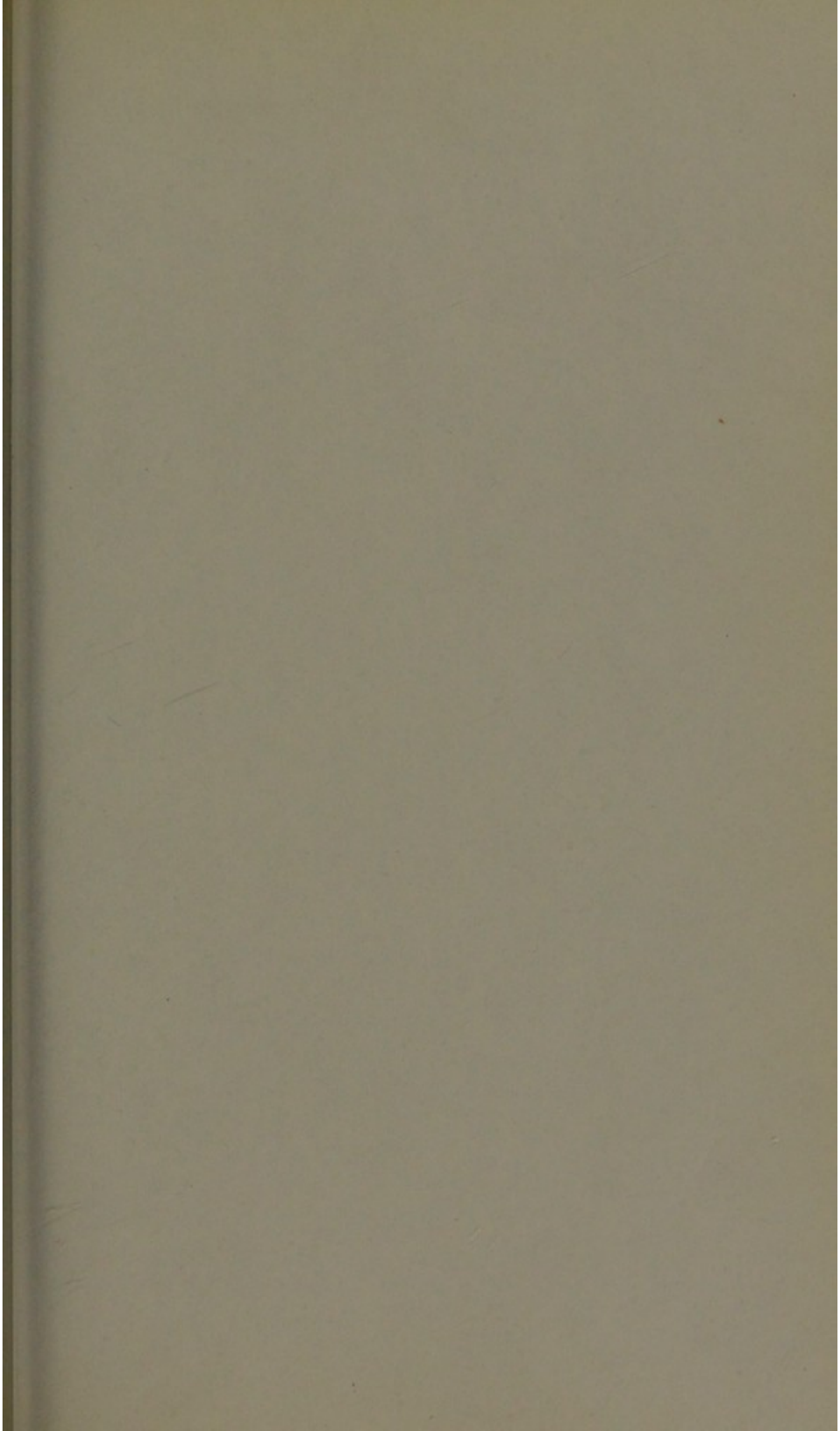
IS ETHER SAFER THAN CHLOROFORM?—STATISTICAL EVIDENCE.

It has been said, however, by some of the speakers that ether is for all purposes safer than chloroform, and the only proof of this assertion seems to be a statistical one—quoted from certain text-books, that with ether the fatalities are 1 in 5,000, and with chloroform 1 in 2,000 or thereby. I cannot understand on what data this conclusion is arrived at. It must be a matter of great difficulty to ascertain the number of administrations in a single city like this, and a statistical conclusion, to have any value, must be based, not on a limited number of ascertained cases, but on a very wide induction, extending over a great area, and over a considerable period of time. The figures given by the various speakers at this discussion will not bear out this assertion. Thus, Sir George Macleod computed his cases as 15,000, with one death *under* chloroform (though Sir George denied that it was from chloroform); Dr. Buchanan, with some 9,000 cases (the number he said was under the mark), had one death, and many others occupied a somewhat similar position. From these two alone there is combined 24,000 cases, with two deaths while *under* the anæsthetic, one of which was not due to the anæsthetic. I have recently met in the Midlands, two surgeons of high standing, one who has been practising for nearly 30 years, another for over 20, neither of whom have had a death from chloroform, though it was constantly used in their practice to the exclusion of ether. The statement which Dr. Laurie of the Hyderabad Commission makes is a striking one: that he and the late Professor Syme conjointly have administered chloroform on 45,000 occasions without a death. Even suppose these figures are doubted, as they do look large—regard his statement from another aspect. The late Professor Syme and Dr. Laurie have given chloroform from its introduction in 1847, the former till his death, the latter to the present date, without a death occurring in the hands of either of them. That is a sufficiently striking experience. Taking the statement in any way one likes, making all allowances and deductions as to the figures, it remains a very remarkable fact that chloroform has been given for over 40 years almost daily, by first one, then another person, and in two very

different climates, cold and hot, without one death. And this statement finds analogy in the experience of two English surgeons who have been equally fortunate. It is also a point of importance that the Hyderabad Commission have so strongly concluded in favour of chloroform. It must be remembered that the conclusions arrived at by that Commission cannot be considered as due to the man who dominated the first Hyderabad Commission, as the *Lancet* doubted its conclusions, and sent out as a representative of their own views a man of the highest scientific status, Dr. Lauder Brunton, who, after the most searching experimental investigation which has ever been made on the subject, returned a convert to the opinions promulgated by the first Commission, which were in accordance with those held and taught by Syme and Simpson.

Regarding personal statistics, it is difficult to form an accurate estimate, as chloroform is so often administered without note being taken, such as for preliminary examinations, for diagnostic purposes, painful dressings, secondary suture, &c. If Sundays, and 30 days of holiday be deducted, a year is left of 283 days, and approximately an anæsthetic is administered thrice on the remaining days (occasionally it is five or six times in one day). It is therefore calculated that in my wards of the Royal Infirmary, the Children's Hospital, and in private practice, anæsthetics have been administered and personally supervised by me between eleven and twelve thousand times (11,886) during the last 14 years. But suppose 1,000 be deducted, so as to place it within instead of over the mark, that would leave over ten thousand cases. About five hundred of these have been ether, ether alone, or ether with nitrous oxide; in several hundred instances ether has been added to the administration of the chloroform in prolonged operations, for possibly ten minutes or so, once or twice during the continuance of the operations. On many occasions ether and chloroform mixed have been given. The remainder have been chloroform administrations, occasionally assisted by alcohol, given before the commencement of the chloroform narcosis, and sometimes during the anæsthesia by rectal injections. Out of these, the only case of death is the one already related arising from syncope, due to increase of œdema in a chronically œdematous brain. As far as can be ascertained, respiratory difficulties occurred in 40 cases, and in 30 of them artificial respiration was resorted to for a variable period. Lowering of the force of the pulse has been frequently seen, especially when associated with sickness, which was relieved as soon as

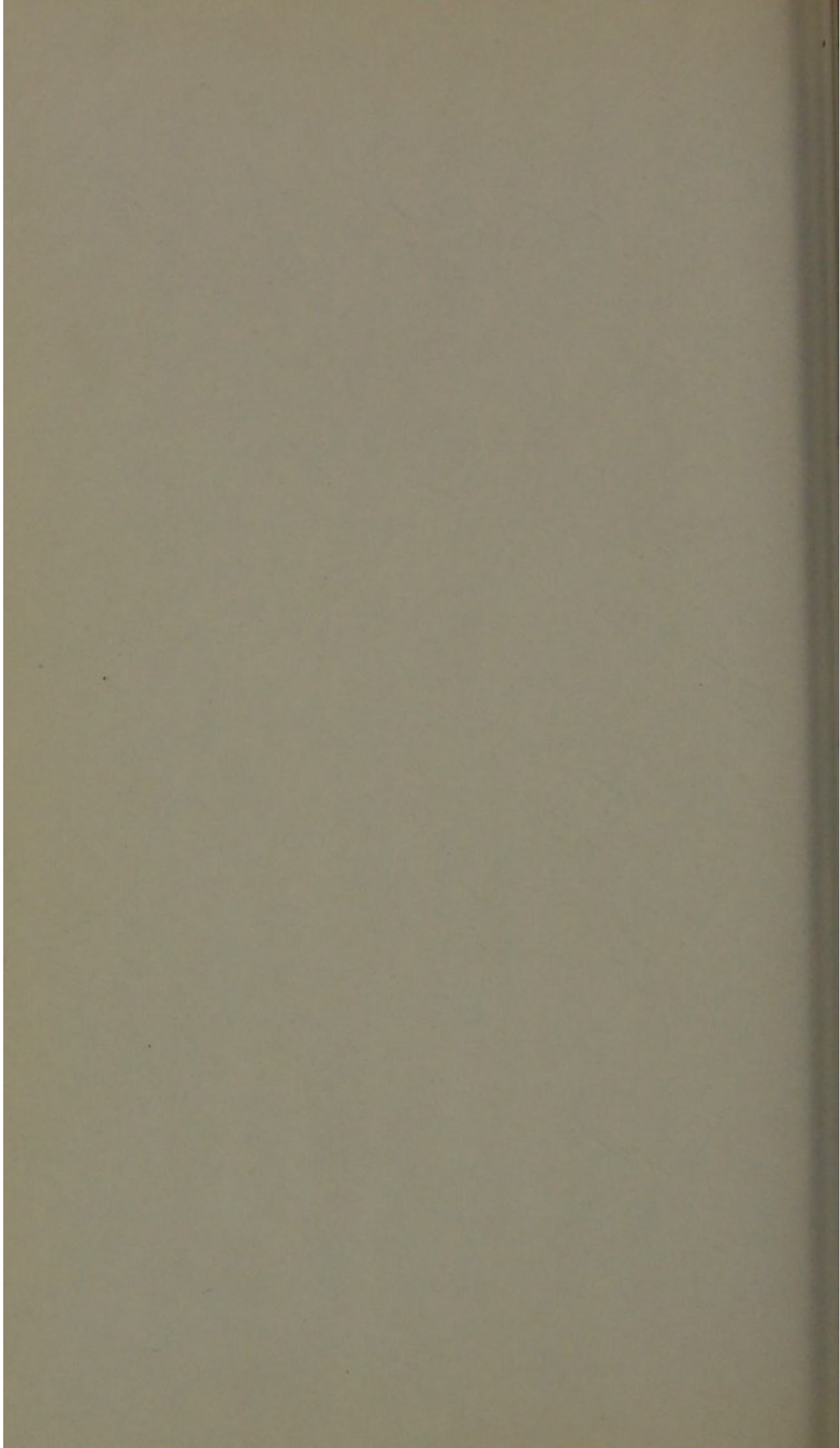


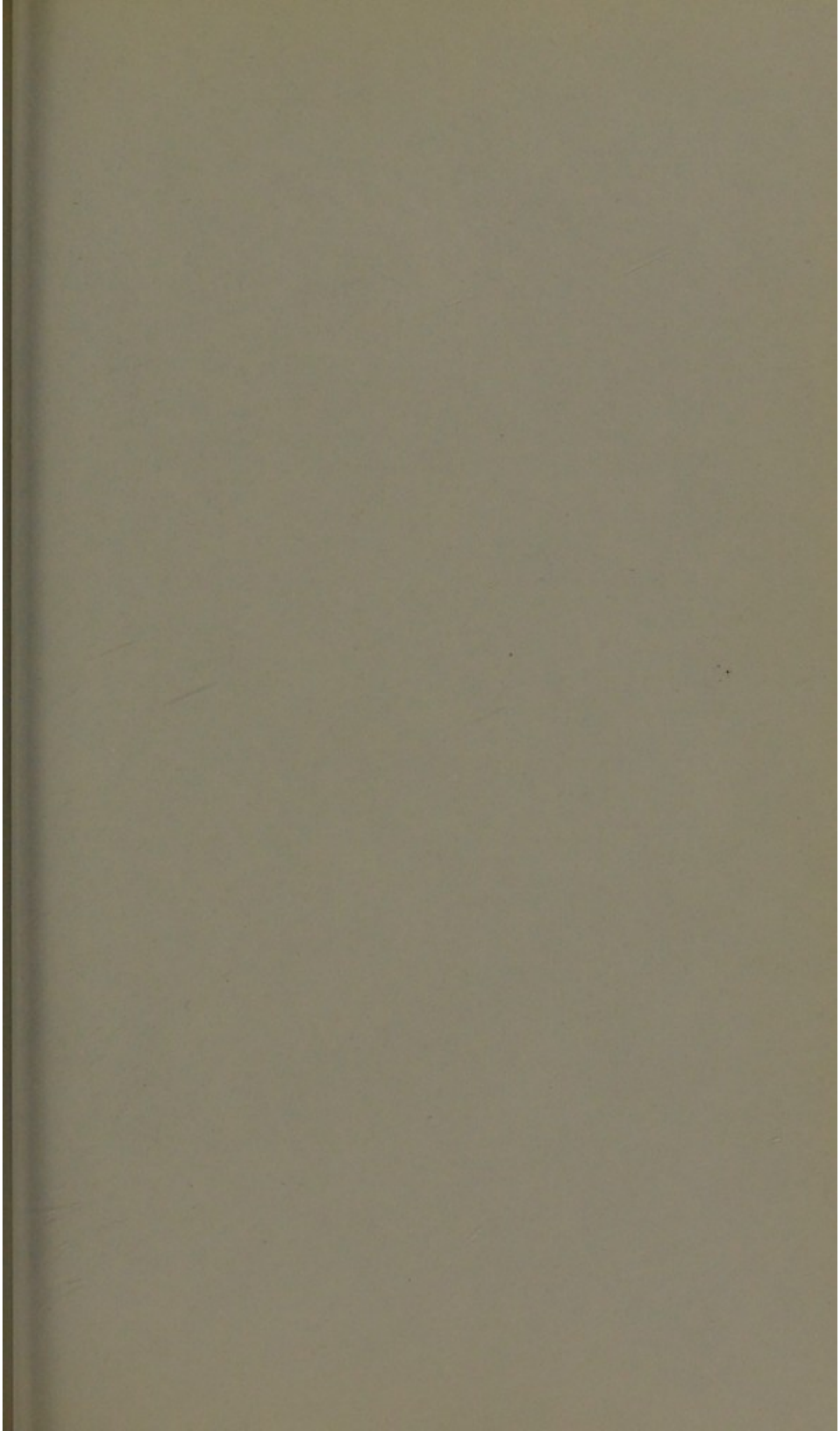


vomiting occurred. Depression at the termination of prolonged operations (lasting several hours) has been noted, and the administration of ether in such has produced nothing further than a short lived filip. When ether is given, however, at intervals, during a prolonged administration of chloroform, it acts well as a stimulant. In many instances the administration of ether and chloroform either in equal parts by weight, or in the proportion of 2 of ether to 1 of chloroform by measure, makes a very agreeable anæsthetic, stimulates the heart and circulation generally, while the chloroform prevents the irritating effects of the ether. To have such in a perfect condition, the mixture ought to be effected at the time the inhalation is about to take place, and not over night. It is best administered in a Clover's inhaler.

Some who have spoken on the side of ether have so strongly advocated its use, as to lead one to suppose that death could not happen under it. This is unfortunately far from being the case. Of those who have advised its use in this discussion, one only has had a sufficiently extended experience to make his observations of weight statistically, and he, Mr. Hartley, has seen two deaths from ether. In the *British Medical Journal* for 1885, there is a paper by Dr. Ernest H. Jacobs of Leeds, on "Deaths from Anæsthetics in 1884," occurring in Great Britain, and which were recorded in the Journals or as had come to his knowledge. From it, we see that there were nine deaths from chloroform; two from mixtures of chloroform and ether; three from methylene—two of these from syncope, the cause of death of the third is not stated; six were from ether. That is a very instructive table. When it is remembered how frequently in Britain chloroform is administered, in comparison with ether (possibly 10 to 1), this list of fatal cases from chloroform is much below the average of deaths from ether: chloroform 9, ether 6. Even suppose chloroform were only administered twice as often, its mortality would require to have mounted to twelve, in order to have been as great as that of ether. The mortality from methylene is enormously higher than from either chloroform or ether in that year, when one considers how seldom it is given. A few months ago, Mr. Vincent Jackson of Wolverhampton, reported a case of death under ether, due to syncope, after a very brief administration of ether. In this case, the patient had had chloroform about two weeks previously, and took the anæsthetic well, and again, a week before death he had nitrous oxide without ill effect, yet he succumbed to ether, and his death was due to syncope. Here was a patient who was

anæsthetised with chloroform without ill effect, and who shortly after died from ether. It is strange that some of the Journals who are so much in favour of ether have not commented upon that fact. In Germany, and over the Continent generally, surgeons administer chloroform, almost to the complete exclusion of ether. In Schede's klinik in Hamburg, two English speaking surgeons who were preaching a crusade against chloroform, asked him to permit them to show him the administration of ether, as they alleged it was so much safer than chloroform. Dr. Schede allowed them to do so, though he expressed himself as satisfied with chloroform. They administered ether, but the patient died on the table from its effect before any operation was performed. In my own experience, I have had no deaths from ether, but on several occasions patients have become so alarmingly ill from its effects that its use had to be discontinued and in at least two of these chloroform has been substituted, and complete anæsthesia established by it without any difficulty. In one of these the anæsthetist was an experienced ether administrator. It was the case of a gentleman who had come from the south of England to have an operation performed on him, but he stipulated that ether should be given him by his own medical attendant, who had a strong ether bias. The patient was also imbued with distrust of chloroform. He stated, that though he liked Scotch surgery, he detested chloroform and would not have it as long as ether would serve the same purpose. When examined by me, I believed he had a fatty heart and a tendency to venous congestion. He was told that he would be much safer with chloroform than with ether. He insisted upon having ether. It was given him, and before operation was commenced his pulse became very weak and then ceased at the wrist. The ear placed over the chest found his heart in a quiver, without any distinct rhythmical contractions. It closely resembled the condition known as delirium cordis. It was fully ten minutes before patient was out of danger, during which many restoratives were applied. After he had fairly recovered, he took chloroform without any difficulty, during which the operation was performed. It has also occurred several times, when ether has been continued for several hours, that the pulse has gone down to a very low ebb, and was only somewhat revived after the cessation of the anæsthetic and the administration of restoratives. It seemed in these cases to exhaust the energy, and when this does occur, there is more difficulty in restoring the exhaustion than in a similar state from chloroform.





DANGEROUS AFTER-RESULTS FROM ETHER.

Again, patients who have been kept under the influence of ether during prolonged operations, occasionally exhibit, hours or days thereafter, bronchial affections which may lead to death. Yet those deaths are often not recorded as due to the ether. I have not had any deaths from such, though I have seen cases with pneumonia or bronchitis occurring after prolonged ether inhalation. It is said these only occur where the ether has been badly given, or with improper instruments. But it has been administered in my wards by etherists of great experience, who were visiting the wards, and each of them have warned me to place the patient in a warm room with a fire for a few days after the operation, lest bronchitis should arise, and they acknowledged that sometimes death did arise from such affections days after the administration of the anæsthetic. In Mr. Jacobs' list of cases already referred to, there is a case of a patient operated on for ovariectomy who took ether well, but who died seventeen hours after operation from congestion of the lungs, which manifested itself twelve hours after the anæsthesia. The ether was administered by a competent person. Chloroform is not followed by such consequences. Some are coerced into using ether, who openly confess they prefer chloroform and believe chloroform the better anæsthetic, but use ether because in the district in which they live the opinion prevails that ether is safe and chloroform is dangerous. As an American surgeon told me, he did not use chloroform, because, if a death occurred under it, there would be an outcry; whereas, if it occurred under ether every one would be satisfied, the people believing that a death from ether was brought about by the hand of God, and a death from chloroform by the hand of man!

THE ACTION OF THE PUPIL UNDER CHLOROFORM.

As the condition of the pupil under chloroform is erroneously described in many physiological works, while in others no reference is made to it, and as very few surgeons have a clear appreciation of its value in relation to reflex action, and as a guide to dangerous development, I will here give in summary what has been written by me in detail in a paper on the "Pupil in its Semiological Aspects," published in the *International Journal of Medical Science*.

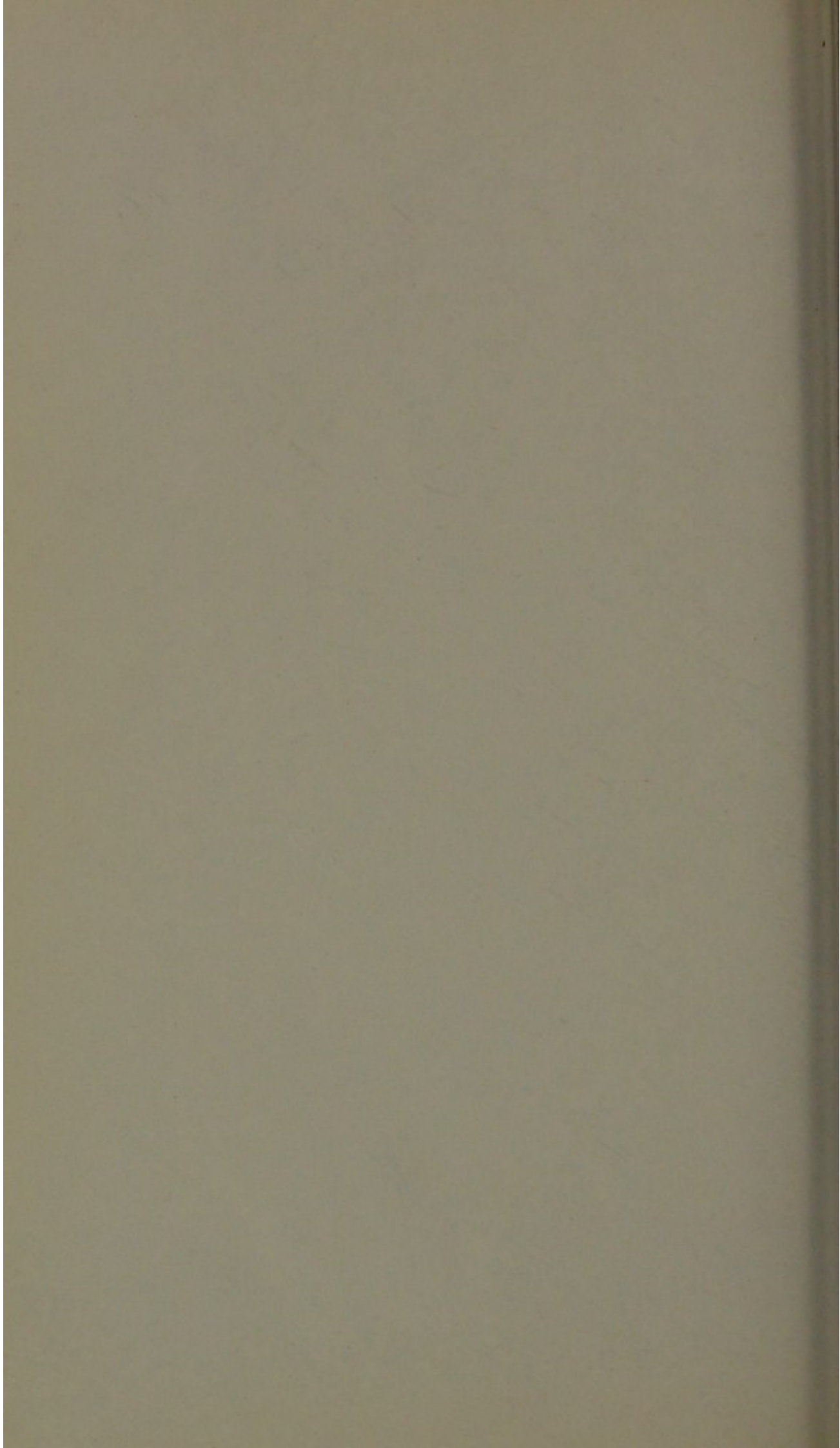
In the first stage of anæsthesia from chloroform, the pupils are mobile, and follow no rule regarding their size. In the

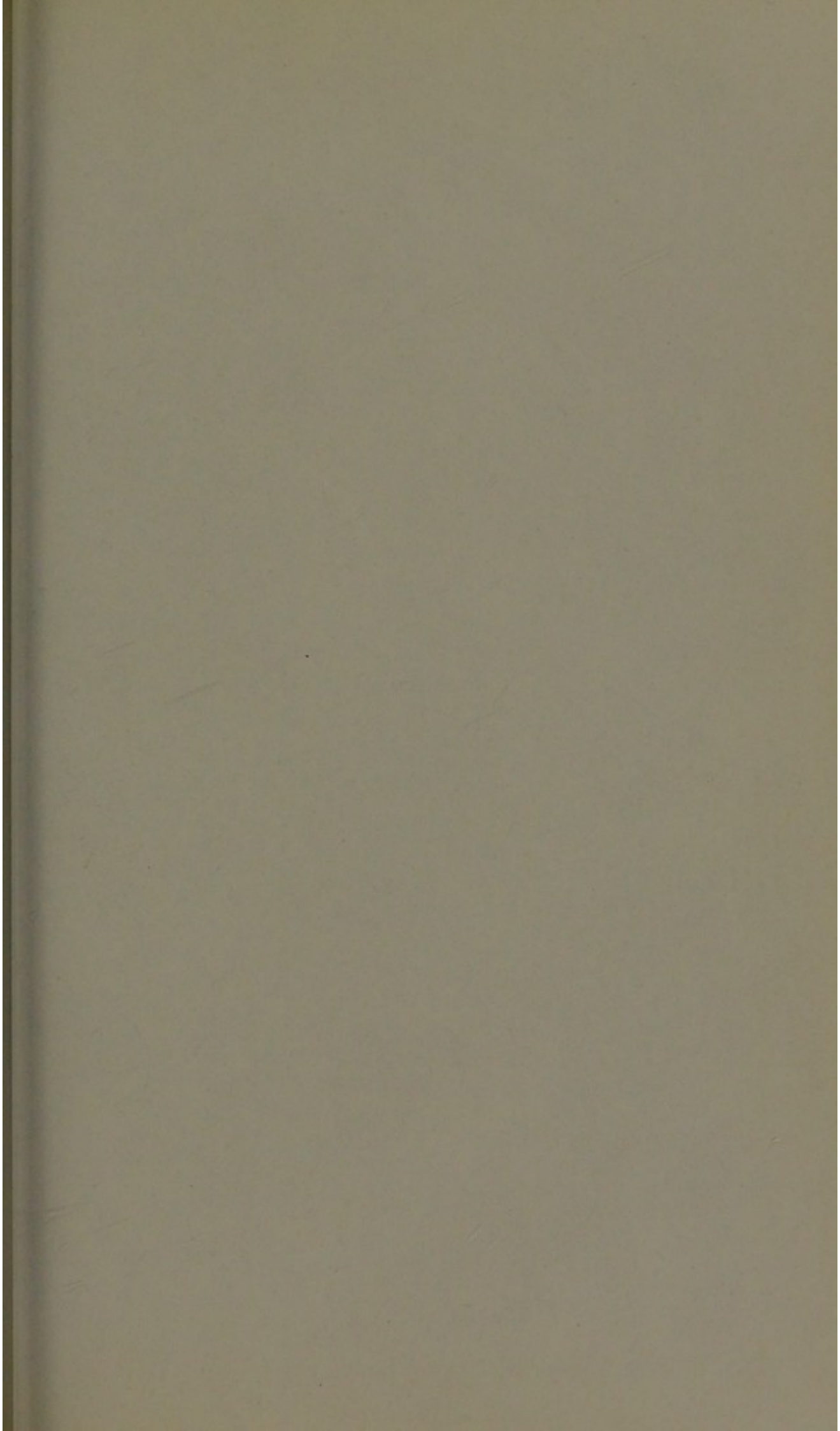
second stage, which is identical with the abolition of reflex action, they become contracted and fixed. The stabile myosis continues as long as the patient remains in that state. But if the chloroform be pushed to a dangerous extent, and the function of the cardiac and respiratory centres becomes involved, then the pupils become widely dilated and fixed. So that this third, or dangerous state, is always indicated by the pupillary condition. The contracted and fixed pupil indicates the safety zone of chloroform narcosis. When this stage has once been reached, and a sudden dilatation ensues, it betokens extreme danger, which, however, may yet be overcome by prompt action on the part of the administrator. It is true that sudden dilatation does ensue in the presence of intense sickness, and though this may alarm the anæsthetist it is an error on the safe side, and is not lost, as the patient requires extra attention at that time. The anæsthetist ought to keep the patient in the contracted pupil stage, as in it the reflexes are abolished.

EDUCATION OF STUDENTS IN ADMINISTRATION OF ANÆSTHETICS.

At present, at all the examining boards in the kingdom, a man may receive his licence to practise medicine and surgery without giving evidence of having been taught the theory of anæsthetics, or of having administered chloroform on even a single occasion! It has been maintained that a student ought never to be allowed to administer anæsthetics. If this be the creed of those who are responsible for the teaching of students, why are they not sufficiently logical to pursue this belief to the end, and refuse to grant those students degrees to practise medicine and surgery? The mere legal "qualification" to practise won't teach that man how to administer an anæsthetic. The granting of licences and degrees to those who have never been allowed to administer anæsthetics is reprehensible.

But, while advocating that every student ought to have the opportunity of administering chloroform or ether, I as strongly hold that they ought not to be called upon indiscriminately and without preparation to do so. This practice of indiscriminate choice of students from the benches of a large class, without the operator knowing anything as to their individual knowledge of the subject, has conduced to mischief, and has alienated teachers from permitting students to administer anæsthetics. The teacher ought first to teach theoretically the physiological action of anæsthetics, pointing out their dangers and how to





obviate them. He ought, secondly, to ascertain, by written and oral examination, the knowledge of the student upon the subject. To those who have shown proficiency in this theoretical examination tentative certificates ought to be given, which would enable the students to present themselves for the practical part of their instruction. They ought then to be permitted to administer the anæsthetic on at least twelve occasions, under the superintendence of a thoroughly competent person, who could be responsible for the safety of the patient. In our hospitals this duty would fall naturally to the surgeon and house surgeons. Thus the safety of the patient is secured, while the student is taught practically. A certificate is then granted to him of having received theoretical instruction in the effect of anæsthetics, and of having administered anæsthetics on at least twelve occasions. This practice obtains in my wards, and I have had no reason to regret it.

WHAT IS A SAFE DOSE OF CHLOROFORM?

In medico-legal enquiries into deaths from chloroform the question is often asked—How much chloroform was used? The answer is supposed to be—Not more than 2 drachms! or if it be more, an overdose is supposed by the authorities to have been given. This question, with its stereotyped answer, is one which must have been formulated at a very primitive stage of chloroform exhibition, and presumes that chloroform is given mechanically or by measure, as one would give a dose of laudanum. The dose of chloroform ought to be judged by its physiological effects, and not by a definite quantity poured out of a bottle. Two drachms, if given in the wrong way, might kill; whereas several ounces might not even put the patient under. Besides, the quantity poured out of a bottle is no criterion of the amount inhaled by the patient, especially when the towel is used, as a great part of the chloroform is wasted, and is not taken into the patient's system. The quantity of chloroform used will depend upon the susceptibility of the patient, the form of apparatus used in its administration, and the duration of the operation. The dose must be judged alone by its physiological effects.

VARIOUS INHALERS.

Many administrators of chloroform deprecate the use of an apparatus, though they will use the corner of a towel caught with a safety pin, and a drop bottle! Don't let us be

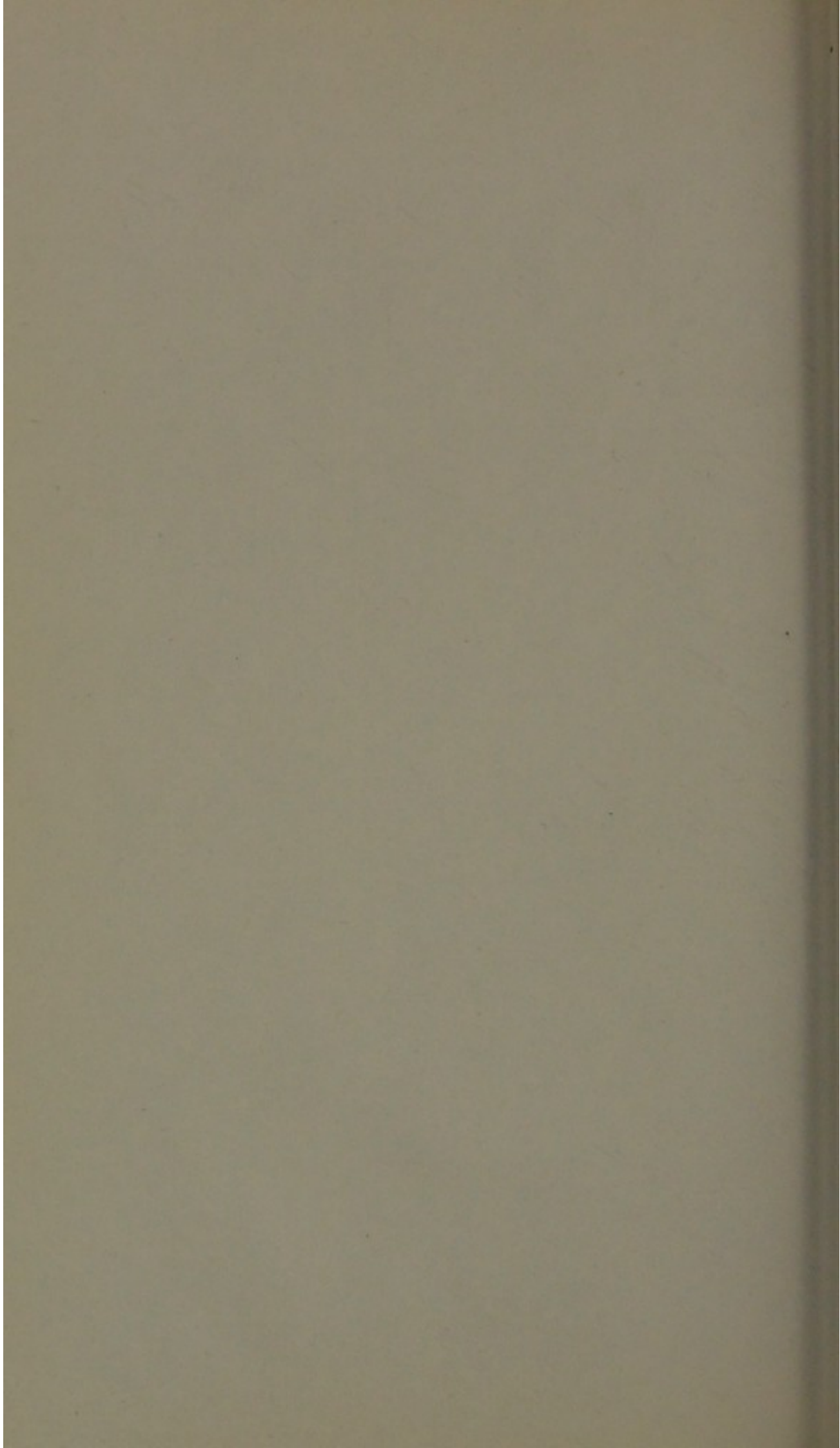
frightened by the name apparatus, but let us enquire into the best way in which chloroform can be conveyed into the patient's system. The desideratum is to have the anæsthetic thoroughly under the control of the administrator, so that he can regulate with precision the quantity given at each inspiration; and while the anæsthetist can do so, it ought to be quite beyond the power of the patient, voluntarily or otherwise, to inhale more than the anæsthetist gives.

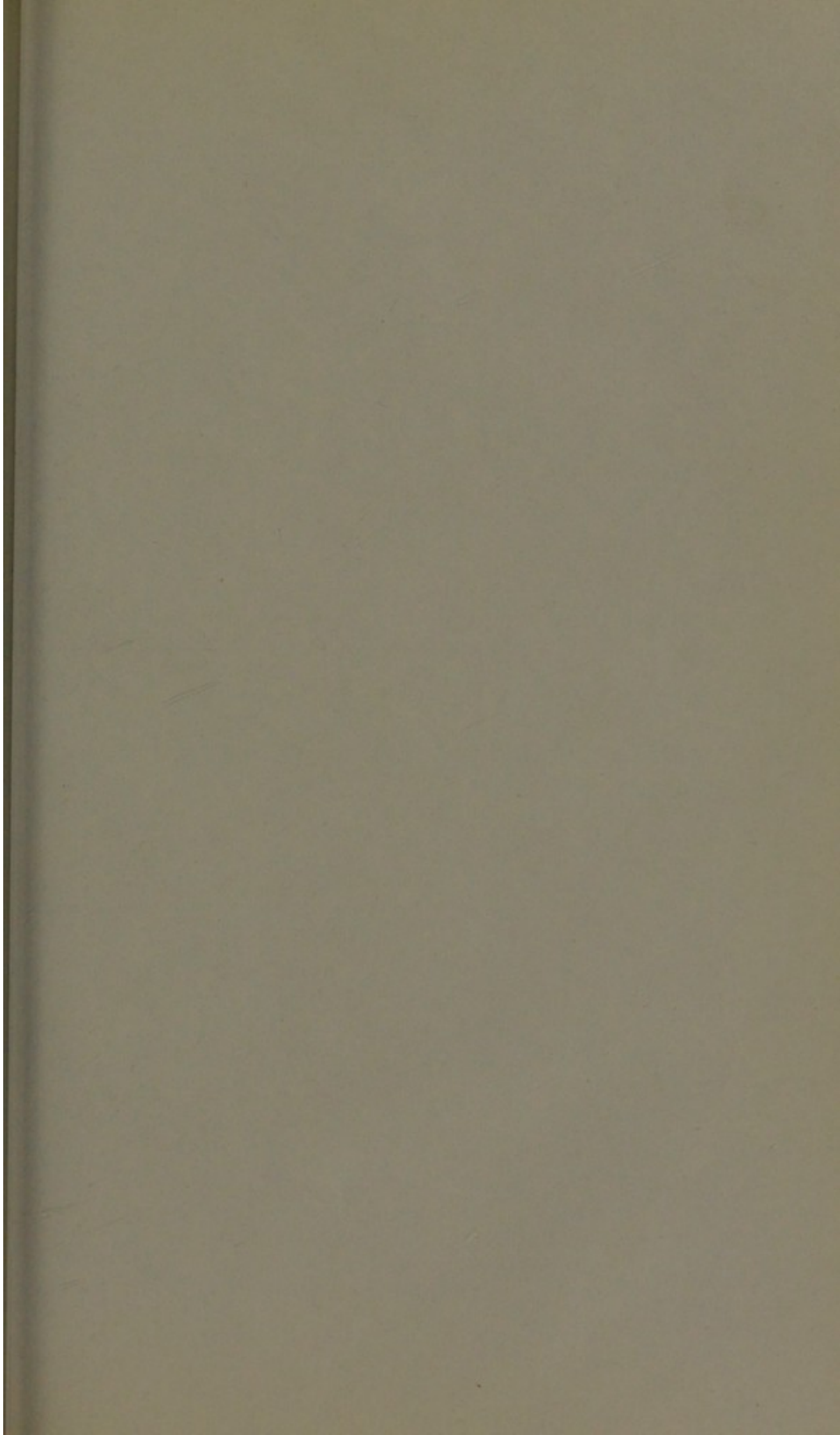
The folded towel has been, and is still, very much used. Many surgeons who never have had a fatal case use it, and are satisfied with it. The dose, however, is irregularly given: first when the chloroform is poured on, it is rather concentrated, and it gradually becomes weaker until the chloroform is again added. It places a reservoir of chloroform at the command of the patient, who inhales a greater or less quantity according to the depth of his inspirations. No doubt, one practised in its use can overcome these difficulties. The danger still exists for novices. The towel is handy, most extravagant on the chloroform, irregular in the distribution of the anæsthetic, and contains an element of danger in unskilled hands, inasmuch as it presents an uncontrolled reservoir of chloroform to the patient.

Allis's inhaler probably produces anæsthesia more rapidly than any other, and it effects a saving in chloroform, but it is a dangerous instrument in the hands of those who do not know its power, and especially to one who is accustomed to administer chloroform on a towel. It also presents an uncontrolled reservoir of chloroform over the patient's mouth and nose.

The flannel or other media stretched on a frame, whether in the form of Esmarch's, or the simple corner of a towel, with a drop bottle, is a much better instrument, as the uncontrolled reservoir over patient's mouth is more limited, and it is possible to work the apparatus in such a way as to render it almost controlled.

To blow the chloroform vapour in regulated quantity over patient's face, leaving the face exposed, would be one of the best methods, were it not that the vapour of chloroform diffuses itself so quickly in the atmosphere that it takes a long time to anæsthetise the patient, though this has several times been done by me in this way. If the vapour so blown be received into a cup-shaped vessel, which is placed over mouth and nose, then the vapour may be inhaled sufficiently concentratedly to permit of anæsthesia being produced. This is found in Jünker's inhaler. It is simply a pump forcing a





definite quantity of atmospheric air into a bottle, which there volatilises a measurable quantity of chloroform. This is then blown over patient's face in a known state of dilution. It possesses the following advantages :—There is no uncontrolled reservoir left near patient's mouth ; each stroke of the bellows suffices only for one inhalation, and the amount of chloroform expelled is exhausted before another inhalation takes place. So that, whether the patient breathes irregularly, very shallow, or very deep, he cannot take more than is given him by the anæsthetist at each pressure of the ball, and the anæsthetist has that under his complete control. Second—by placing five drachms of chloroform in the bottle, for every complete compression of the bellows one minim of chloroform fluid is evaporated, which becomes diluted, as inhaled, with six thousand times its bulk of atmospheric air. While placing the patient under, the bellows is blown more rapidly, and when the patient is fully anæsthetised the blowing is lessened to about one half the rate. Again, as the chloroform liquid gets less, there is a smaller quantity of liquid taken up with each blast of the bellows, until, when one drachm remains, there is only one quarter of a minim evaporated with each impulse, and this is diluted to one in every twenty-four thousand times its bulk as inhaled. These exact quantities have not been tested by me. They are given in a table which is issued along with the instrument, but, from my experience of the instrument, I believe they are approximately correct. Third—there is no waste of chloroform, and those accustomed to a folded towel would be astonished at the small quantity of chloroform necessary to induce anæsthesia. Fourth—the stage of struggle is often avoided absolutely, and when present is so to a much slighter extent than is usual with other forms of inhalers. This is very important, seeing that syncope is apt to follow this period of struggle. It is the safest inhaler for the administration of chloroform. As a rule it takes longer to place the patient "under" with this inhaler than with Allis's. I do not think that a drawback.

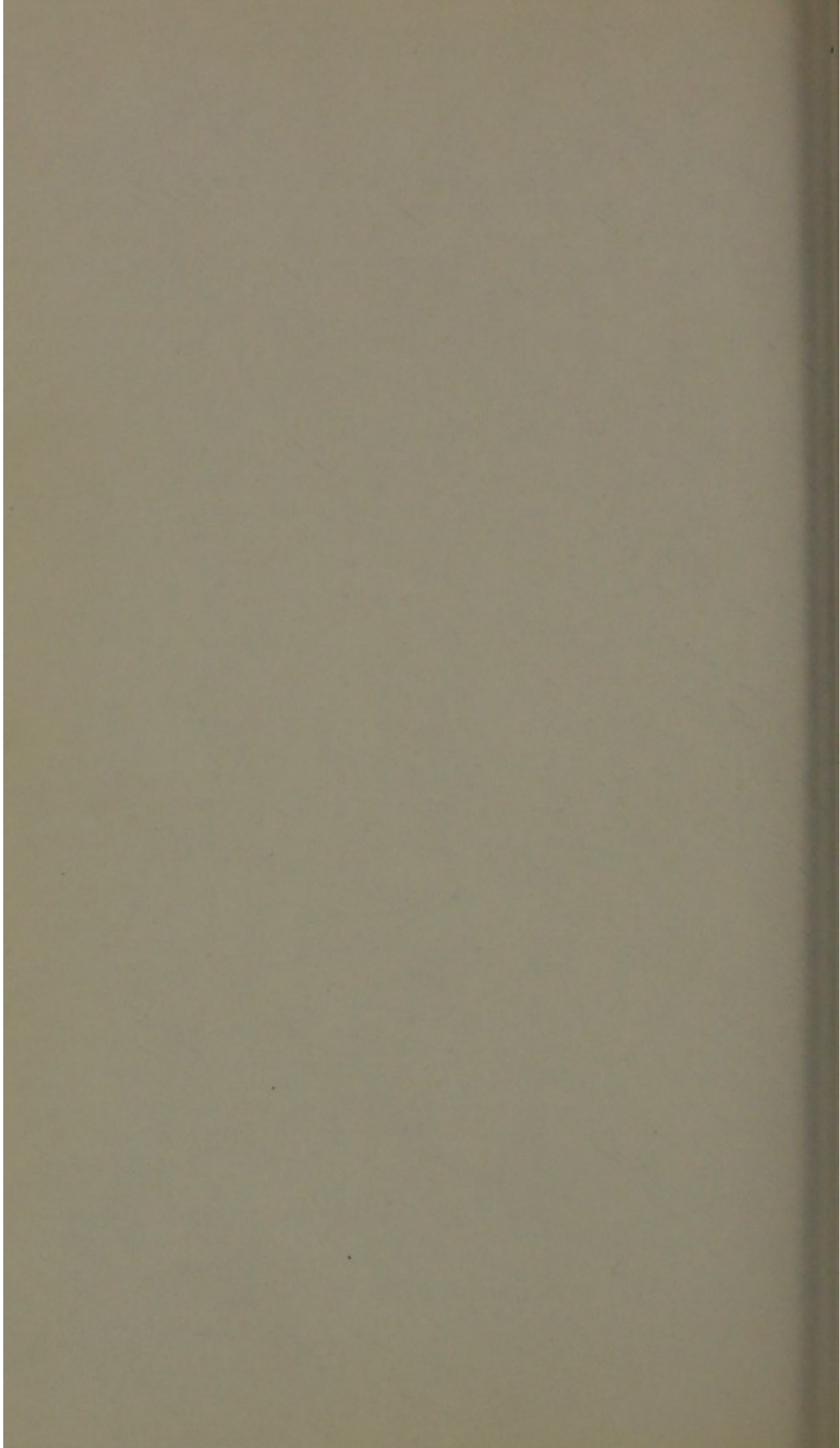
It is, however, to be borne in mind that no inhaler is absolutely safe—that a fatal issue can arise even with the safest inhaler ; after all, it is the physiological action of the drug on the individual which must be taken as a guide, though an inhaler such as Jünker's permits definite doses to be given, and, in this way, secures at least precision of dose and definite dilution. A competent chloroform anæsthetist can administer chloroform without any form of apparatus, the towel is quite sufficient for him ; he judges by the physiological action, and trusts to nothing else.

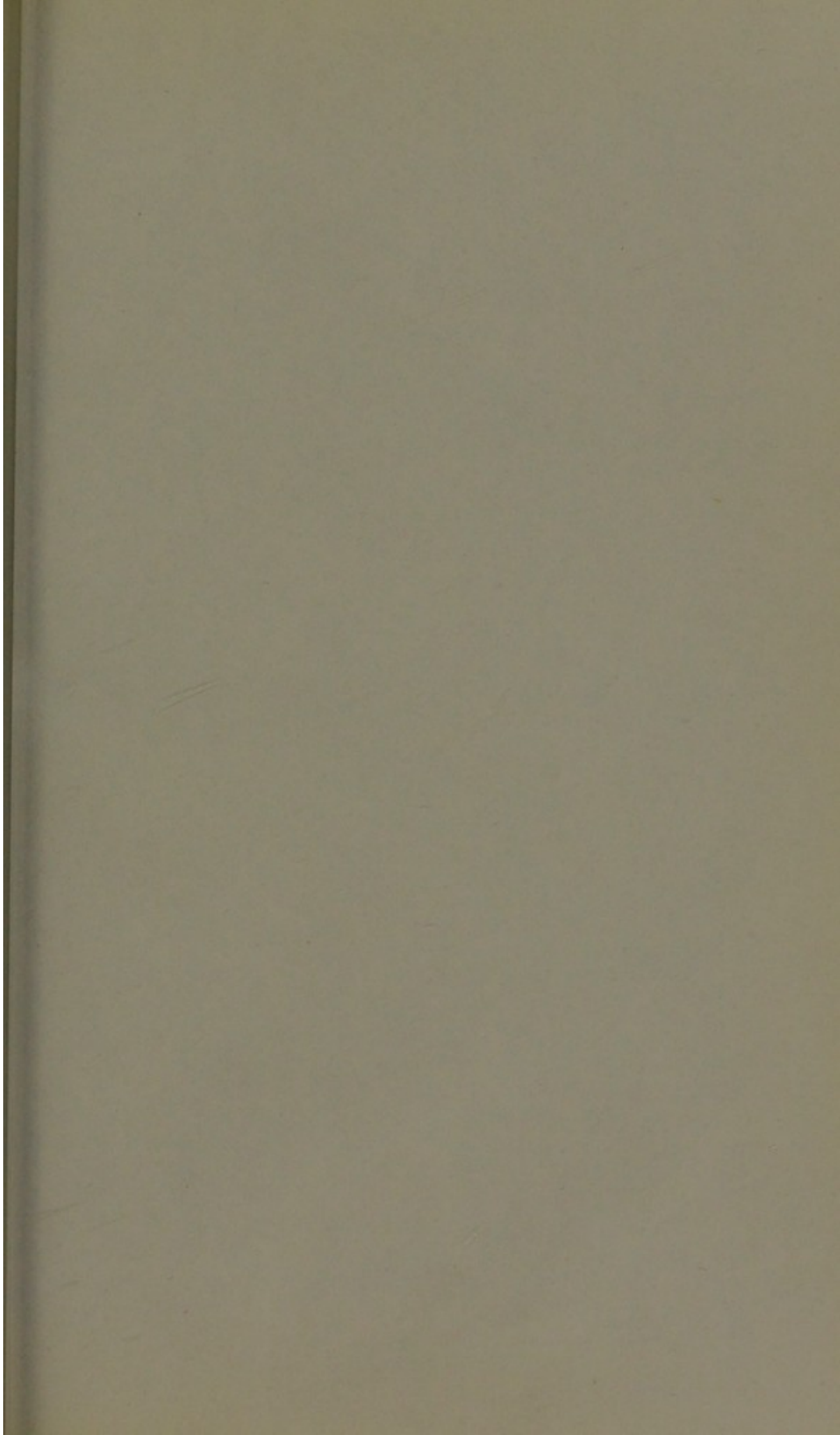
MORPHIA AND OTHER DRUGS AS ADJUNCTS TO ANÆSTHESIA.

The use of morphia, administered hypodermically, has been advised as an aid to anæsthesia, and as a preventive of accidents. It certainly is an aid to anæsthesia, but it may narcotise the patient so deeply as to lead to death. It must not be forgotten that morphia becomes intensified in its action in the presence of chloroform. I have seen one-twelfth of a grain injected subcutaneously in a fairly healthy woman ten minutes before administration of chloroform produce profound narcosis, lasting for several hours after the completion of the operation, the patient exhibiting the opium pupil. A case has been reported to me of a fatal issue, following one-sixth of a grain of morphia hypodermically injected twenty minutes before chloroform was administered. The death took place eight hours subsequently. It is quite likely to diminish primary syncope, and there may be good in the combination provided it were accurately wrought out. Meantime, it is occasionally a source of danger. Opium given immediately after an operation, before the patient is fully out from the influence of the chloroform, is likewise somewhat dangerous, especially if a large dose be given. Many other combinations I have tried, as recommended and lauded on their introduction, but they have been all found wanting, with the single exception of alcohol, of which I have already spoken. In cases of fright or of weak heart, an ounce of brandy or whisky, five minutes before the patient is anæsthetised, stimulates the heart, avoids shock, and facilitates anæsthesia. Before the administration of ether one would avoid giving alcohol. The best method of administering ether is that of Mr. Teale, of Leeds.

METHOD OF ADMINISTRATION OF CHLOROFORM.

Chloroform, as is well known, tends to cause suspension of the functions of the brain in a definite order. It is said to give rise to a period of excitement, but I am inclined to regard this excitement as due to the attendant circumstances, rather than to the effect of the vapour. In the first stage of chloroform narcosis, the brain loses its power of sensation and voluntary motion. But in this stage reflex action is still present, and if the anæsthesia did not go further, not only would these reflex actions be a source of annoyance, but the heart would be exposed to the danger of shock from the operation. In this stage the pupils are somewhat dilated, but





may be either wide or medium, and they are mobile, still reacting to light. In the second stage the reflex functions of the cerebro-spinal axis is abolished, and the voluntary muscles lie perfectly relaxed. The parts concerned in the respiratory movement and the sympathetic cardiac ganglia are exceptions. In this stage the pupils are contracted and fixed. This is the safety zone of chloroform narcosis. The narcosis ought never to go beyond this stage. But if the chloroform be pushed, then the functions of the respiratory and cardiac centres become interfered with, the pupils become widely dilated and death is imminent, and will ensue unless prompt preventive measures are adopted. Any impediment to breathing producing complete occlusion may produce this dangerous stage if not at once remedied.

Sickness often causes a marked cardiac depression, when severe, akin to syncope, during which the pupils become widely dilated. When the stomach is relieved this condition passes off. Sickness ought seldom to occur during chloroform narcosis as it is generally indicative of a return of reflex action, and may be avoided by an even administration of the anæsthetic. Vomiting, on return of reflex action, is often attributed erroneously to the anæsthetic used, whereas, it is generally due to the profound insensibility produced.

Where it is possible to have the patient examined medically, that ought to be done, days before the administration of the chloroform. When the patient's case is being enquired into surgically, the state of the lungs, heart, kidneys, and liver can be ascertained at the same time. The information so obtained will place the surgeon in a better position to guard the weak points, and to act instantaneously if danger arises. It is true that there are diseases of the viscera which cannot be determined by physical signs, and which may prove a source of danger, but that is no reason why defects ascertainable physically ought not to be determined beforehand. In my practice, this is regularly done, and is a source of comfort. This examination can scarcely apply to accidents, where, manifestly, an operation is imperative without delay. An examination of the heart ought never to be ostentatiously made, just as the patient is about to be anæsthetised, as the nervous excitement to which it may give rise, might be prejudicial to the patient. Patients with grave pulmonary lesions ought to have very great care bestowed on them while being anæsthetised.

PREPARATION OF THE PATIENT.

(a) Chloroform is best administered on an empty stomach. If possible, just a little before, or about the time patient would be expecting a meal. If he is kept too long without food, bile is apt to flow into stomach and induce vomiting during chloroform.

(b) A purge twenty-four hours previous to the anæsthetic, and if the patient is to be operated on in the morning, it is advisable to let him have a good night's rest without being disturbed by purgation. The patient ought to be kept as cheerful as possible.

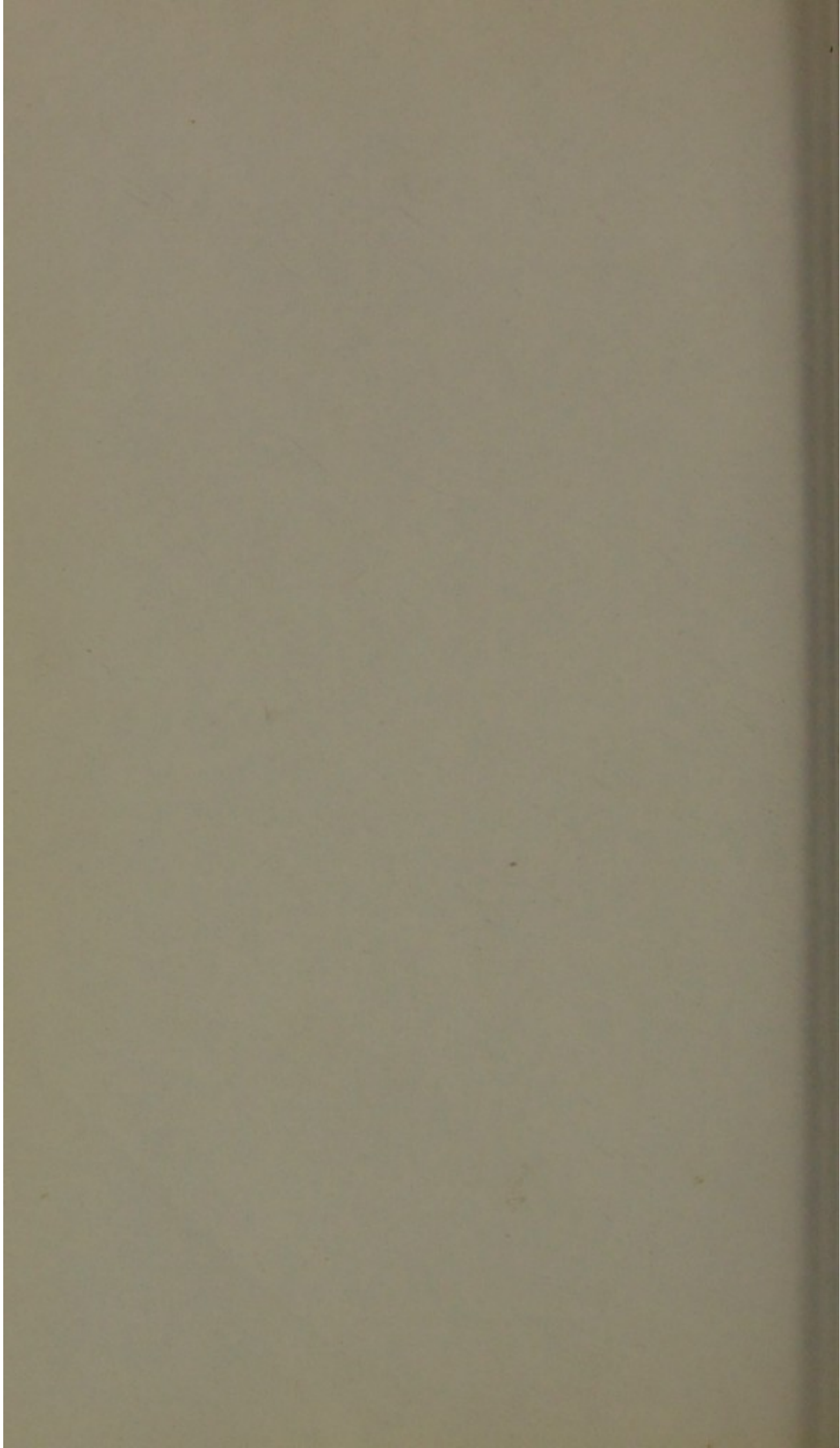
(c) If he be weak, nervous, or in any way afraid, or has a weak heart, an ounce of brandy with slight dilution ought to be given ten minutes before the anæsthetic is administered.

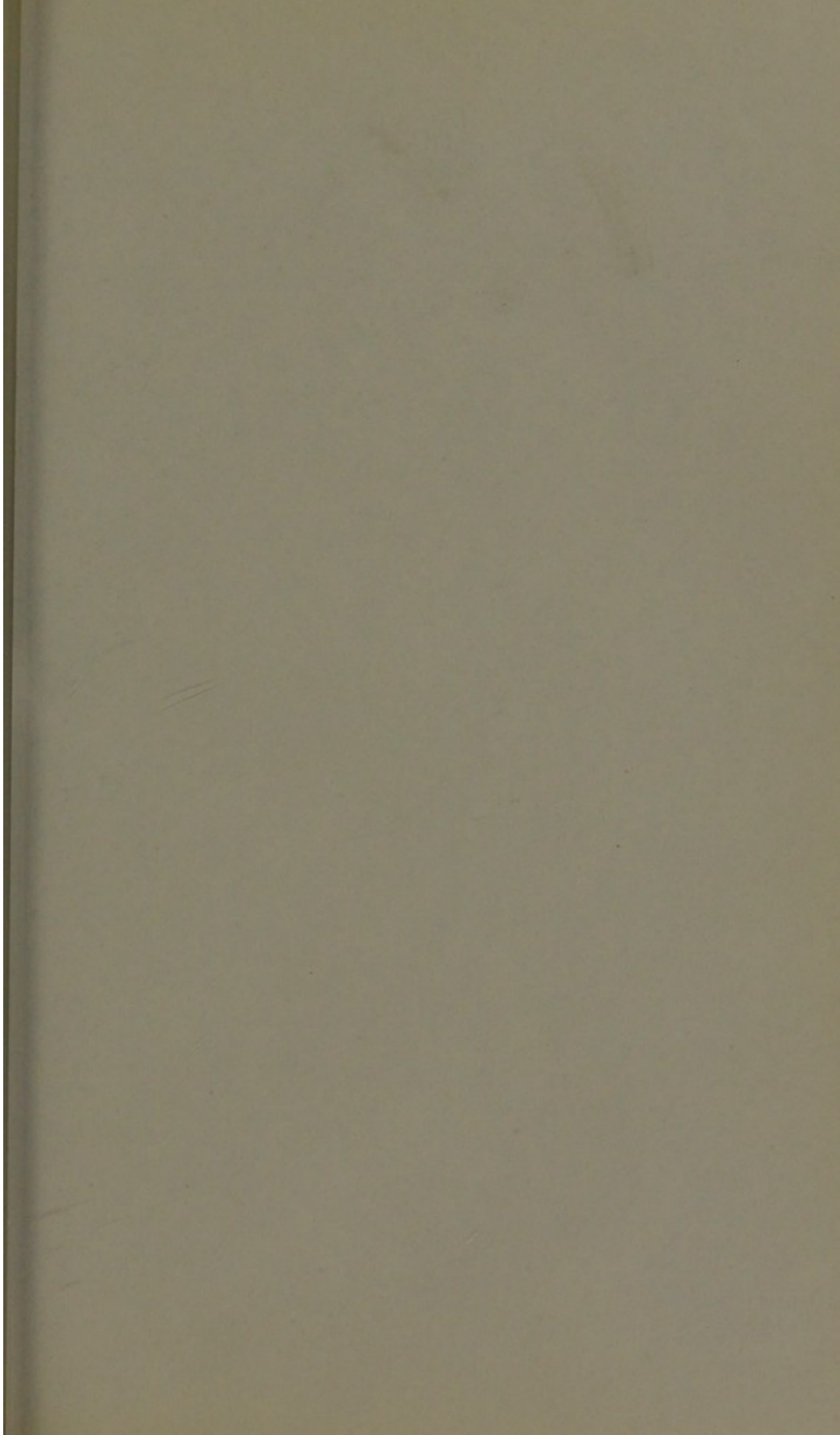
(d) The patient ought to be kept warm throughout the operation. It ought to be remembered that the patient is more or less exposed during the operation. An atmosphere which is comfortable for surgeons and attendants with their ordinary clothing, would be quite too cold for a patient more or less divested of theirs, and who is at the same time depressed by the circumstances.

HOW TO ADMINISTER THE CHLOROFORM.

The anæsthetist ought to endeavour to take the patient into his confidence, and explain to him what he wishes him to do during the inhalation of the anæsthetic. This often facilitates anæsthesia. He is to ask patient to shut his eyes, as the chloroform nips them. Then to breathe easily and quietly, and if he does not like the chloroform just to blow it out, which ensures a subsequent deep inspiration.

He ought to request the patient to count aloud and slowly, from one to one hundred, and between the announcement of each number, to take an easy, deep breath. The object of that is, first, to divert the mind of the patient; second, he feels that as long as he is able to count, he is able to communicate with the anæsthetist or the surgeon, a knowledge which somewhat soothes him; third, it shows the surgeon that the breathing is progressing favourably—a very great point. Eighteen years ago, I commenced this practice, and since have found it of great service. When it is possible, some one, independent of the anæsthetist, ought to watch the pulse, during the administration. That person's duty is to tell of any weakness which may be perceptible. It is a guide to the surgeon, as well as to the





anæsthetist. The latter ought to be armed with an instrument for opening the mouth, and a forcep for pulling out the tongue if need be.

The duty of the anæsthetist is to concentrate his attention on the breathing, the colour of the face being a help regarding the state of the circulation; and the state of the pupils, as well as the conjunctival reflexes, being a guide to him as to the depth of the anæsthesia.

HE OUGHT NOT to watch the heavings of the chest and abdomen as a guide to the respirations; if he does so, death may ensue from asphyxia before these heavings cease. If he only becomes alarmed at the cessation of the heavings of the chest and abdomen, he may be quite too late to save his patient. Many patients' lives have been lost owing to this fallacy.

Each breath which the patient draws ought to be recorded on the *tympanum* of the anæsthetist; there can be no fallacy as to the patient's breathing under these circumstances. The alternate hot and cold sensations communicated by the expired and inspired air to the finger of the hand which holds the towel or inhaler, placed near the lips, is an additional help. The anæsthetist's attention ought not to be diverted from his duty from the beginning to the end of the anæsthesia. A good anæsthetist will keep a patient in the second stage of chloroform narcosis throughout the whole operation, neither plunging him too deeply nor allowing the return of reflex action. He will find a Jünker's inhaler of use in thus regulating the stage of anæsthesia. He ought also to recognise the undoubted fact, that the *majority* of deaths under chloroform have been due to asphyxia.

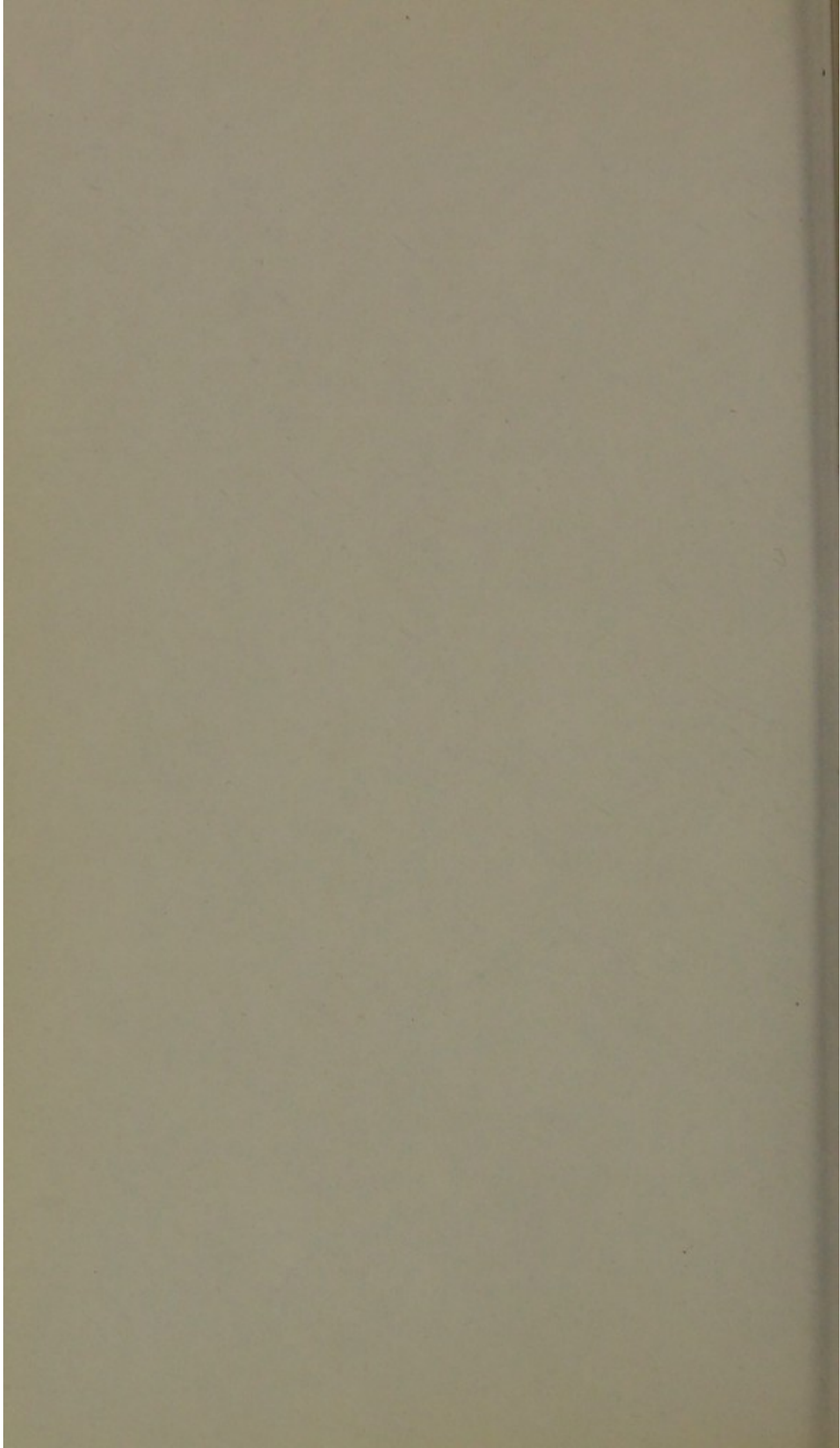
Asphyxia may arise under chloroform from various causes—first, from mechanical obstruction due to paralytic action of the ary-epiglottidean folds, or to accumulation of mucus in the pharynx, or from the contents of the stomach getting into the trachea; secondly, to too concentrated an atmosphere of chloroform preventing an elimination of carbonic acid; and, thirdly, from the effects of too concentrated an atmosphere on the respiratory centre, paralysing it. The second and third may be obviated by giving the chloroform well diluted with atmospheric air, and by keeping the patient in the safety zone. The mechanical difficulties require prompt action when they do arise. The falling in of the epiglottidean folds, along with the backward displacement of the tongue, producing stertorous breathing, or an absolute stoppage of respiration, can be remedied easily in the majority of cases by throwing the angle of the lower

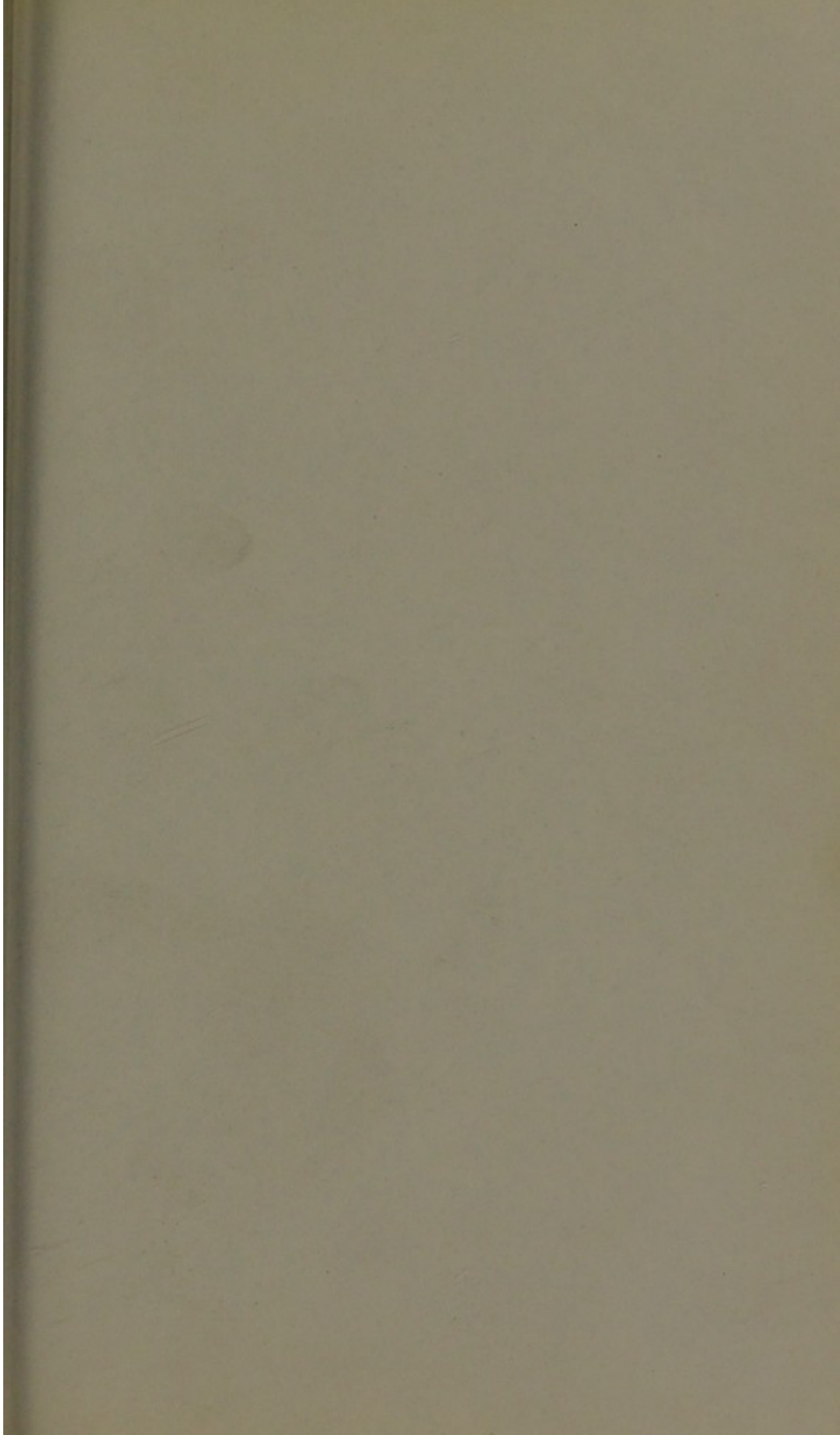
jaw forward, while the weight of the head acts as a counter extending force. Simple as this is, the majority of men don't know how to do it. It ought to be seen and taught to the students during their practical course. Some try to open the mouth while throwing forward the jaw—the one effort defeats the other. The head ought to hang, and the angle of the lower jaw ought to be thrown forward by the thumbs of the anæsthetist. Often a little movement in this direction is sufficient. If it fails, then open the mouth and pull out the tongue, first upwards, then over the teeth toward the chin. At the same time a sponge mounted on a stick should swab out the pharynx. The larynx being clear, if the breathing is still deficient or absent, artificial respiration ought to be resorted to, and if the pulse be weak, elevate the lower part of the body and depress the head. In carrying out artificial respiration, the arms of the patient ought to be pressed forcibly on the lower part of the chest so as to empty it, next they ought to be pulled out from the sides fully, then stretched outward from the body over the top of the head and extended. This movement must fill the chest with air which ought to be heard to rush in. Don't be satisfied otherwise. Then leave this air in the chest for five seconds, when the process may be repeated.

Bile vomited may get into the trachea and cause occlusion. This is apt to occur if the vomiting is so quietly performed as to escape the anæsthetist's attention. Whenever vomiting occurs, the anæsthetist ought to lower the head, open the mouth, and swab the pharynx out with a sponge. Let him remember that vomiting is only reflex as far as the pharynx, and there it must lie until removed or inspired. Let the anæsthetist continue by the patient until he is able to speak, and afterwards leave a nurse with instruction how to act should the patient become sick or vomit.

CONCLUSION.

In conclusion, it may be that the future will disclose a safer anæsthetic agent than any which we yet possess. However right and commendable it is for experimenters to endeavour to procure new and better anæsthetics, surgeons ought to hesitate before they try upon their patients every new and untried agent, however much it may be lauded from animal experiment. Let the student study the physiological action of chloroform, learn that it is an agent which places the functions of the cerebro-spinal axis in abeyance, but does so





seriatim, in a definite order, and which may, if pushed far enough, suspend them all. Let him be fully alive to its dangers, and how to counteract them, and let him recognise the safety zone of chloroform narcosis, and he will find it an anæsthetic powerful, it is true, but controllable, and though not infallible, yet a most valuable and useful servant. Meanwhile, chloroform is one of the safest and best anæsthetics, though it requires for its proper administration knowledge, skill, and assiduous attention.

