

that time. At 2 P.M., and several times afterwards, he vomited; and it was observed that he turned over always to the right side, and almost fell out of bed. He slept a good deal. When he was roused he opened one eye—the nurse thinks the left, but cannot be sure; he seemed to understand when spoken to, and nodded in reply, but never actually conversed. If some beef-tea were offered him, he always looked into the cup and tried to take it, but instead of doing so he pushed it away apparently involuntarily; at one time he almost bit a piece out of the cup, and when it was put to his lips his attempts at swallowing only resulted in the fluid escaping at the sides of the mouth. About 6 P.M. the nurse set him up for the purpose of giving him some beef-tea, and spoke rather sharply to him, thinking he was shamming. He did not succeed in taking the food, and in a few minutes it was observed that his breathing had become stertorous; and when, a short time afterwards, he was seen by the house-surgeon he was found to be quite insensible, in an apparently deepening coma, with a pulse of 54; his left pupil being widely dilated, while the right was firmly contracted. There was apparently no paralysis on either side of face or body.

I saw him at 8.15 P.M., and found him in the condition described. The stertor was loud, and evidently caused by the dropping back of the tongue; in fact, on one occasion the breathing stopped completely, and was only restored on pulling forwards the tongue; a piece of bandage placed under the chin, and fastened to the head of the bed, relieved this difficulty in a great measure. Not only was there no paralysis of the limbs, but a pseudo-voluntary effort was made when the hand was grasped. It was thought at one time that the left side of the face was weaker than the right, but this weakness, if it existed at all, was almost imperceptible. The left temporal region—i.e., that away from the injury—had a doughy feel, as if blood were extravasated amongst the tissues, but there was no fulness of the orbit or of the veins of the conjunctiva or the fundus oculi.

It may be thought that these symptoms were enough to justify the application of the trephine over the middle meningeal artery on the left side—i.e., the side opposite the injury; but it must be borne in mind that the onset of the symptoms, having only been observed by the nurse, was very imperfectly reported, that the only unilateral symptoms consisted in the dilatation of the left pupil and the fulness of the left temporal region, and, lastly, that the patient appeared to retain a sort of voluntary power over his limbs.

Under the circumstances, it was decided to wait a short time, and when I again saw him at 11 P.M. the slight alteration that had occurred in the symptoms seemed rather to be in the way of improvement, the pulse being now 120 and compressible, though somewhat irregular, the left pupil having considerably diminished in size, and the movements of the limbs showing apparently a somewhat more voluntary character. I did not therefore feel justified in performing the operation at the time, and he was left till next morning. During the night the condition scarcely altered; the bowels did not act, the urine was passed freely in the bed, and contained a trace of sugar. There were now coarse mucous râles in the chest; and the temperature was 102°. In the middle of the day the only alteration in the symptoms consisted in the fact that the breathing was gradually becoming more obstructed, and late in the afternoon this difficulty rapidly increased, so that the patient died about 6 o'clock. Throughout the day the vomiting continued at intervals.

The post-mortem examination showed that a fracture of the skull extended from the seat of injury into the middle fossa of the opposite side. The hæmorrhage from this had caused the fulness in the left temporal region. The anterior branch of the middle meningeal artery was ruptured (as was found by injecting water through the external carotid artery) just at the point at which the trephine may be most satisfactorily applied in these cases—viz, two inches above the zygoma, and two behind the external angular process of the frontal bone. From the rupture an extensive hæmorrhage had occurred, a large clot, five ounces in weight, having separated the dura mater from the skull, and greatly indented the brain. The only other lesion discovered consisted of two very superficial spots of bruising on the left temporo-sphenoidal lobe.

Here, then, was a case in which trephining would in all probability have saved the patient. It seems worthy of being placed on record, because the symptoms which were so obscure as to lead us to suspect some more grave injury appear to have depended altogether on the pressure resulting

from the hæmorrhage. It is also of great importance to notice that almost no unilateral symptoms were present, a fact which need hardly surprise us when we remember that the pressure must be equally distributed over the whole of the interior of the cranium. It has been pointed out that a widely-dilated pupil is often found on the same side as the clot in similar cases, and this symptom appears to be one well worthy of notice, though it must be confessed that our knowledge of the meaning of the different conditions of the pupil after injuries to the brain is at present very imperfect.

I cannot help thinking that if, as was stated above, the introduction of antiseptic surgery has rendered trephining a far less formidable operation than it used to be, we ought to become less shy of resorting to it. If a distinct interval of consciousness have occurred after the accident, and symptoms so severe as those occurring in the present case supervene, and if there be anything at all to guide us to the side on which the hæmorrhage have taken place, I do not think we are justified in denying the patient the chance which the performance of the operation affords.

Henrietta street, Cavendish-square, W.

AN ADDRESS PRELIMINARY TO THE DISCUSSION ON RICKETS,

*Delivered before the Pathological Society of London,
Nov. 16th, 1880.*

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It is unpleasant on such an occasion to have to begin with anything like an apology; but unless I explain how I come to be here to-night, you, when you have heard me, will assuredly think that I have done very little justice to my task. On being asked by your Council to open a discussion on Rickets I wrote, as I thought, to decline. I said that I had no new observations to offer you; for although I have always felt a general interest in the subject, I have kept no notes of the cases which I have seen. But your Secretary told me that what was needed was not so much a series of new facts, as a general statement of the questions in regard to the disease which seemed most to require elucidation and discussion, and this I thought I could furnish to you. Other members of the Society, he assured me, had valuable observations on particular points to bring forward; and in offering to you the following remarks, I would disclaim for them any higher position than that of being simply introductory.

There are, I think, good reasons why the subject of rickets should now be taken up by this Society. One is, that in spite of the labours of Sir W. Jenner, and of many others, there is still too great a tendency to attribute to local causes the deformities caused by it, and to overlook the fact that these deformities belong to a general disease. Only a few weeks ago I heard a very eminent physician, in examining the body of a child who had died of broncho-pneumonia, speak of the pigeon-breast as if it were merely a result of the pulmonary affection. In that case there were at the wrists and ankles no obvious signs of a rachitic change. But a section through the lower end of the tibia at once showed an abnormal state of the epiphysal line. Is it certain that a pigeon-breast is ever seen in a child whose bones are absolutely healthy? I should like to put this question to the Society, and to follow it with another—Does a rachitic thorax ever acquire the characteristic grooves on each side near the ends of the rib-cartilages without the intervention of some degree of bronchial catarrh? A very slight cough, lasting only a few days, may undoubtedly flatten the sides of the chest in a rachitic infant. But can this result be effected by the mere elasticity of healthy lungs?

The settlement of these questions, and of many others, is greatly retarded by the circumstance that during life it is impossible to determine absolutely the fact that rickets is not present in a slight degree. Even in a child not very fat I have more than once failed to detect any "beading" of the rib carti-

lages through the skin, notwithstanding that at the autopsy they were found to be diseased. And I may notice that the enlargement is often much more marked on the pleural side than towards the surface. On the other hand, there is naturally a slight elevation where the cartilages and the bones meet; and in thin children I have seen this mistaken for "beading," when in reality not the slightest rachitic change was present.

This difficulty as to the clinical recognition of rickets in certain cases is, I think, of great importance in regard to what will probably be the chief subject discussed here this evening—the relation, namely, between the disease and the recently discovered osseous lesions of inherited syphilis. M. Parrot, indeed, ignores rickets altogether. For him a rachitic change in a bone is merely a modification of the syphilitic affection, occurring only in children over six months of age, and attended with an enlargement of the extremities of the bone, and with the formation of a pearly-white osteophyte in its interior and on its surface. He would seem to be almost unacquainted with those histological characters which enable rachitic lesions of bones to be traced from their very commencement. My friends, Dr. Barlow and Dr. Lees, of course, take up a very different position from this. But it has seemed to me that even they, in their observations, have been too ready to assume the absence of rickets when they found no obvious beading of ribs and no marked swelling of wrists or ankles. They do not appear to have been fully alive to the importance of slicing the bones at an autopsy, and of examining their epiphysal lines, both with the naked eye and microscopically. Until this procedure shall have been systematically carried out in a sufficient number of cases, I think that it will still remain doubtful whether cranio-tabes is really related to syphilis rather than to rickets.

There is, however, another and a somewhat different question—namely, whether syphilis may not be one of the causes of rickets. At first, when one is taught that inherited syphilis produces certain definite osseous lesions, one is naturally disposed to think that what has to be done is to draw a hard and fast line of distinction between these lesions and those due to rickets. But what we know of syphilis in adults surely points to a different conclusion. We know that besides producing an immense variety of affections peculiar to itself, it also acts as one of the two causes of the lardaceous change in the viscera, and many suppose it to be also a frequent cause of atheroma of arteries, and perhaps of phthisis.

The characteristic changes of rickets are far more obvious in fresh bones than in those which have been kept in spirit; but I have brought with me a few specimens which have been collected during the last few months, and which show the irregularity of the epiphysal line, the enormous increase in the breadth of the semi-transparent bluish zone of proliferating cartilage, &c.

I have already remarked that rickets is a general disease. The points which show that it is not confined to the osseous system are indeed such as singly might appear trivial, but taken together they possess considerable significance. I am not now referring to the visceral lesions described by Sir Wm. Jenner, and afterwards by Dr. Dickinson, as affecting spleen and liver and lymphatic glands. I have failed to find these lesions in the great majority of rachitic children whose bodies I have examined, whereas I have found a large fleshy spleen in many children who have been free from rickets. And I am much disposed to agree with Dr. Gee, who, after stating that the appearance of the spleen in rickets generally differs in no respect from that which is seen after ague, or in inherited syphilis, adds that in his opinion the affection is really a result "not of the rickets, but of the general state of health which caused the rickets." This, however, is a matter on which valuable experience will no doubt be forthcoming in the course of the present discussion.

Nor can I attach much importance to the so-called "prodromata" of rickets—sickness, diarrhoea, tumefaction of the abdomen, languor, drowsiness, &c. It seems to me that all of them are really either independent effects of the improper dieting and other conditions which give rise to rickets, or else themselves accessory causes of the disease. And it is certain that when one is called to a case of croup or of broncho-pneumonia one often has to deal with an advanced stage of rickets, although the child's mother may have thought it perfectly well up to the time when the acute illness began.

But there are three well-known symptoms of rickets which appear to have no direct connexion with the osseous changes. One is a restlessness at night, which impels the child to throw off its bedclothes, even in cold weather. Another is the tendency for profuse perspiration to break out over the head, neck, and chest, especially during sleep. The third is the sensitiveness of all parts of the body to even gentle pressure. Dr. Gee has shown that this tenderness is by no means confined to the bones, and that the muscles of the loins or of the abdomen are sometimes no less painful when pressed upon, however cautiously.

There is one point with regard to the urine, on which I should be glad to elicit further information. Some months ago I was asked by Dr. Paddon of Putney, to see with him a rachitic boy, whose most obvious symptom was that he passed large quantities of uric-acid crystals. Such a circumstance is surely not common in a child. Since then I have met with another case in which I was told that the urine was extremely irritating and caused scalding pain during micturition. Can this have any bearing upon the supposed presence of lactic acid in the urine? which is certainly one of the points that needs further confirmation.

As to the state of the brain in rickets there are some discrepancies of opinion among different writers. The skull is obviously large, out of all proportion to the narrow face and stunted body. But Ritter von Rittersbain showed by accurate comparative measurements that as a rule it is not bigger in rachitic children than in healthy children of the same ages. If so, a very improbable notion suggested by Trousseau falls to the ground—namely, that the softness of the cranium allows of the more easy development of the nervous centres, and that rachitic children consequently possess intellectual faculties in advance of their years. Surely it would be more reasonable to attribute the precocity of such children rather to their being thrown so much into the company of adults, and to the contemplative habits induced by an unfitness for rough play and games. And Dr. Gee speaks of the brain as being dwarfed like all other structures, and describes an effusion of fluid within the cranial cavity to fill up a vacant space there. I should like to ask the Society whether it is certain that hydrocephalus is really a frequent complication of rickets, as all writers seem to say. It is at any rate certain that the skull is sometimes greatly increased in size, without there being any fluid in its interior. I well remember, although I have unfortunately no notes of, a case which I saw in the Evelina Hospital, and which was taken without question for one of very advanced hydrocephalus, until at the autopsy the brain was found to fill the cranial cavity completely. When a grown-up person has a rounded projecting forehead, as was the case with the novelist Thackeray, is the popular opinion correct that an hydrocephalus existed in infancy and has been recovered from? Might there not have been simply a rachitic state of the bones of the skull?

A curious complication of rickets, of which I have seen an example, is a chronic cerebritis. A child, eighteen months old, who had had frequent fits since the age of four months, was admitted into Guy's Hospital in a state of insensibility. It lay on its left side, and whenever it was moved into any other position its whole body became rigid, the mouth being affected with clonic spasms. It died in a few days, and the cerebral membranes, which themselves were thin and transparent, were found to be everywhere adherent to the surface of the brain, so that a thin superficial layer of the cortex peeled off with them. The white substance of the hemispheres was markedly indurated, and had a yellowish colour, and the limit between it and the cineritious matter was ill-defined. This was especially the case at the antero-lateral part of the brain on the right side.

But in many cases spasmodic affections occur without any change in the brain being discoverable. Of laryngismus stridulus, I believe that many observers say that it never occurs except in rachitic children. A year or two ago I had to examine the body of an infant, concerning whom all that I could learn was that it had died suddenly. The ribs showed the appearance characteristic of rickets, and this suggested to me that the cause of death was, perhaps, laryngismus, an idea which was afterwards confirmed by the mother's statement that it had suffered from that kind of disorder. I have certainly seen one case of laryngismus in a little girl, whose general bodily configuration, although she had been brought up by hand, seemed at first to negative the idea that she could possibly be rachitic. But afterwards, when this child got an attack of bronchial catarrh,

her ribs became for a time slightly flattened, and I began to think that her case might not really be an exception to the rule. Tetany is another affection that occurs chiefly in those who are the subjects of rickets.

Both the chemistry and the histology of rickets have been carefully studied, but with regard to each of them I think that there are still points open to discussion. The most recent analyses that I have met with are those which Friedleben made, and the results of which are recorded in the *Jahrbuch für Kinderkrankheiten* for 1860. He found the percentage of earthy salts to be from 33 to 52, which is considerably higher than that obtained by earlier investigators, though it is of course much below the normal percentage of 63 to 65, yielded by the bones of healthy children. But it seems to me that we cannot too carefully guard ourselves against the supposition that any figures of this kind represent the actual composition of the osseous tissue itself, or even that they prove it to differ from the normal. No one can examine the microscopical specimens which I have placed on the table in front of you without seeing that in the bones from which they were taken, if dried and submitted to analysis, there would have been a large proportion of various other structures besides the osseous. Even within the bony trabeculae themselves there are often to be seen very numerous and large islets of residual cartilage-matrix. Why the point is of importance is that, if there were in rickets a real alteration in the chemical constitution of the bones, affecting every particle of their substance, we could hardly doubt that the disease must exist at a very early period, if not from the first setting-in of the process of ossification within the foetus. It does begin earlier than used to be supposed, being not unfrequent in children less than six months old. But whether it is ever present at birth seems to be very uncertain. Cases of foetal rickets have been described by several observers; but all that is as yet known with regard to their histology leads to the conclusion that the lesion is altogether different from that with which we are now concerned.

The curvatures which arise in the shafts of the long bones in rickets show, of course, that these bones are throughout soft and yielding. But it does not necessarily follow that the whole of their substance must be altered in composition. Their diminished resistance may fairly be ascribed to the preponderance of a light and spongy texture, not only in the parts formed since the commencement of the disease, but also in older parts, where it may well result from an over-activity of the absorptive processes which are always at work in their interior.

For there is in rickets one essential and characteristic histological change, which certainly is not diffused through the entire mass of the bones, but affects only their growing surfaces, when they join cartilages, or lie beneath periosteum. Indeed, it would be strictly accurate to say that rickets is not a disease of the osseous tissue at all, but of the soft tissues. Even the so-called "beading of the ribs" is really an enlargement of the costal ends of their cartilages rather than of the ribs themselves. I shall not attempt to describe to you the details of the remarkable morbid process which is observed, and of which I have a series of beautiful specimens, prepared for me by the kindness of my colleagues Mr. Symonds and Dr. Carrington. It is well known that the cartilage cells of the intermediate zone, instead of forming short vertical columns, which should be arranged with much regularity side by side, multiply, so that they become converted into rounded masses of enormous size, some of which bulge towards the cartilage, while others dip and extend far into the bone. The cells themselves have an abnormal appearance, which is aptly indicated by the epithet "dropsical" applied to them in a recent paper by Klebs. Rindfleisch defines rickets as depending upon an acceleration of the changes which usher in and prepare the way for the formation of bone, without the actual ossification keeping pace with them; but, in regard to this definition, I doubt whether the amount of proliferation which must normally take place has been sufficiently appreciated. It seems clear that for a long bone to increase in length, so much as it does from childhood onwards, there must be a far more abundant multiplication of the cells at each epiphysal line than would be indicated by the short columns which are visible at any one time. It may be, after all, that what takes place in rickets is not so much an excessive as an irregular and perverted process of growth. I must also remark that the formation of the so-called "cartilage bone," the occurrence of "provisional calcification" of cartilage-

cells, has appeared to me a much less prominent feature of rickets during its active stage than I should have expected from the accounts given by writers on morbid histology.

With regard to the origin of rickets, it is perhaps worth while to allude briefly to a recent revival of the old "lactic-acid" theory. Wegner of Berlin showed in 1871 that if, while administering minute doses of phosphorus to young animals, he withheld lime-salts from their food, there arose an affection of the bones exactly like rickets. He supposed that the phosphorus acted as a stimulant to the ossifying tissue. Heitzmann has since stated that lactic acid is capable of acting in a similar way. The hypothesis, therefore, as it has been promulgated by Senator, is that the disease is the result of an irritant action upon the growing bones of that acid (which is thought to be formed in the alimentary canal in excess from milk or other articles of food), there being at the same time a deficiency of phosphate of lime, consequent either on its being ingested in too small quantity, or on its being carried away through the bowels by diarrhoea. The weak point in this speculation is of course the circumstance that no excessive formation of lactic acid has been proved to occur.

As to the causes of rickets, again, there are a great variety of opinions; but I think not very many accurately determined facts. In this country the prevailing doctrine is that improper feeding of infants is the chief, if not the sole cause. But among Germans it is interesting to notice that Vogel, in the last edition of his work, says not a word about the possible influence of diet, and insists on defective ventilation—the want of fresh air—as the most important factor in the etiology of the disease. And of ourselves there are some who think that it is especially apt to be caused by farinaceous food, such as potatoes, when given too early; others attribute it mainly to a premature deprival of the natural food of a baby—its mother's milk.

Most English observers doubt whether rickets is capable of direct hereditary transmission. If it occurs in the children of a rachitic parent, they think that in each generation the hygienic influences have been adverse. But Vogel says he knows many families in which, the parents showing distinct signs of having been formerly affected, the children have all become rachitic in turn, in spite of every precaution. Sir Wm. Jenner has expressed a doubt whether impairment of a father's health has any tendency to induce rickets in his offspring; whereas Ritter von Rittershain thought that he traced it to the presence of tuberculous disease in the father more often than in the mother. But, among the poor, how are we to say what links there may have been between the supposed cause and the effect? The illness of a labouring man may deprive his wife of nourishment, throw heavy work upon her, and in many different ways render her likely to bear weakly infants; it may prevent the children from receiving proper food, may interfere with their being taken out into the open air, may confine them in small and cramped rooms, and may limit their supply of warm clothes.

A point on which Sir Wm. Jenner has laid great stress is that the first child of a family, or even the first two or three children, are often found free from rickets, when later ones are affected with the disease; and, again, that when a woman has once had a rachitic infant, those that follow are almost sure to suffer. But this may be due either to the progressive enfeeblement of the mother's health by repeated childbearing, or (among the poor) to the overcrowding and deficient food and clothing which are implied by a large family; while children of even the better classes are often kept far too much in-doors, when there is only one nursemaid for several of them.

The only conclusion seems to me to be, that the conditions of life in large towns are too complex, and too little capable of being isolated from one another, to allow of a separate study of their effects. Hitherto it has not been possible to trace rickets to any one special exciting cause; but, on the other hand, we are surely not justified in saying that no such cause exists.

It is clear that if rickets can be set up by feeding a child improperly, or by any other unfavourable conditions of life, it is not, in a strict sense, a diathesis. Thus one can no longer, with Sir William Jenner, set forth a contrast between it and other diathetic conditions. But its relation towards tuberculosis is a question of great interest and well deserves further elucidation. Dr. Eustace Smith, while doubting the diathetic character of rickets, remarks that it "never occurs in children in whom the tubercular disposition is well marked." At first this appears a striking state-

ment, but it loses all its significance when we remember what are supposed to be the signs of a tubercular disposition; since for a rachitic child to present them would be almost a contradiction in terms. And it would seem that it must be impossible to uphold Sir Wm. Jenner's distinctions between scrofulosis and tuberculosis, now that so many pathologists find grounds for maintaining that cheesy glands and chronic joint- and bone-diseases commonly form the starting-point of an infective tuberculosis. For my own part, I must confess that I doubt whether any special configuration of body carries with it a *positive* tuberculous tendency. It seems to me that the only character presented in common by the majority of phthisical and scrofulous individuals is a *negative* one—namely, the absence of a perfectly robust and vigorous frame. In whatever direction the body deviates from the highest standard of health, except as the result of the actual presence of some other disease, I believe that the liability to tuberculosis is increased. If so, however, one would certainly expect that rachitic children should often be also tuberculous. Sir Wm. Jenner, indeed, himself states that rickets does not by any means exclude tubercle, and I have lately met with a case in which both diseases were present in the same infant. As regards family tendencies to tubercle in those who are the subjects of rickets, Ritter von Rittershain proved that a large proportion of the fathers of rachitic children were tuberculous, whereas Sir Wm. Jenner alludes to a table made for him by Dr. Edwards, which appeared to show that the offspring of phthisical parents were actually less likely than those of non-phthisical parents to become affected with rickets.

In conclusion, I would urge as slight additional incentives to the study of rickets by English physicians, its great frequency among us, and the fact that one of the traditional names for it is *morbus Anglicus*. The suggestion was even once made that it originally spread from this country to the Continent; but the name in question probably had its origin in the title of a treatise published by Whistler at Leyden in 1684, "*De Morbo Puerili Anglorum*." The term rachitis was first proposed by Dr. Glisson, of Cambridge, two years later. One can hardly doubt that he really adopted it on account of its similarity in sound to the word rickets, which was a popular name for the disease in the west of England, where it seems to have been first recognised. But Glisson, in his work on the subject, offers to his readers the choice of a Greek root, *ῥαχίς*, on the ground that the spine is one of the first parts to be attacked! It has probably seldom happened that the fabricator of a scientific appellation has thus himself made apparent the weakness and inaccuracy of its etymology. But I have little doubt that there are many other terms which, having been made, instead of growing spontaneously, have no better origin; and, if so, what can be more futile than to spend time and labour in searching out for such bastards a legitimate pedigree, to which they really have no claim?

THE ADMINISTRATION OF ANÆSTHETICS.

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It may be assumed that the anæsthetic agents usually employed in practice are ether and chloroform. Moreover, it may be affirmed that they are so safe and satisfactory that while we all shall welcome any better, it is not wise to abandon these well-tried means for every new compound possessing anæsthetic properties, with nothing else to recommend it but its novelty.

But these agents are neither satisfactory nor safe unless properly administered with due discrimination of the cases suitable to each. The reports of deaths from both chloroform and ether which appear nearly every week point to the urgent need for those who have larger practical experience than ordinary to formulate their opinions. Even at the risk of being considered dogmatic, I shall try to state accurately what are the methods I use, the precautions I have found necessary, and the errors I have learnt to avoid in the administration of anæsthetics; and I hope by clearly enun-

ciating my own views to raise certain questions in a definite manner, which shall be capable of being affirmed or denied, but at any rate must be answered.

The agent to be preferred.—As a general rule, I prefer ether, because I believe it to be safer, the public believes it to be safer, it is a perfectly satisfactory anæsthetic, and its after-effects are less depressing than those of chloroform. The kind of ether I use is Macfarlane's methylated ether, as made for Dr. Keith, because it is cheaper, and in every way as good as the more expensive kinds. The apparatus I employ is a towel folded lengthwise, with three or four thicknesses of paper between the folds, made into a cone by twisting it on one hand, and fixing it with a few safety pins.

Preliminary arrangements.—No solid food should have been taken for at least three hours before the time fixed for the administration. I can recommend the plan proposed and practised by my friend Mr. Priestley Smith of administering a dose of chloral hydrate an hour before. Do not give brandy or any other stimulant just before administering ether; it is unnecessary, will probably be vomited, and introduces another factor into the conditions which we should try to keep as simple as possible. Examine the chest and make inquiries as to cough in all cases. Inflammation of the lungs or air-passages forbids the use of ether. The vapour of ether irritates healthy lungs, often to an excessive degree, and sometimes causes a slight bronchitis for a day or two, while occasionally it gives rise to fatal oedema of the lungs, even where no previous disease existed in these organs. It is therefore plain that all inflammatory conditions of the lungs are likely to be made worse by ether. Chloroform is to be preferred in all such cases. Cardiac disease *per se* does not contra-indicate ether, as the drug aids a weak heart. In aortic incompetence with badly filled arteries the circulation becomes better during the administration of ether. In mitral disease the case is somewhat different. It must be remembered that ether frequently causes spasmodic dyspnoea, which ordinarily need cause no alarm, and calls for nothing but temporary suspension of the administration, but during which there is great venous turgescence, and the right side of the heart is necessarily overloaded with blood. So that wherever I have reason to believe that the right side of the heart is weak and dilated I should prefer chloroform to ether. The same would hold good of dilatation of the right ventricle apart from mitral disease.

Fractures, herniæ, and other conditions in which complete muscular relaxation is required are cases in which, *cæteris paribus*, I should use chloroform.

Operations about the face can sometimes be performed only with difficulty, or not at all, while ether is being administered; in these chloroform must be employed.

Young children take chloroform with such ease and safety that it is to be preferred for them.

Method of administration.—The orifice of the cone should be large enough to cover the lower two-thirds of the patient's face, and take in the chin and lower jaw. It is always preferable to have the patient lying down with his shoulders a little raised, and his head not much higher than his shoulders; the pillow should be firm and flat; unfasten anything that is round the patient's neck; ask him to turn his head with the *right* cheek on the pillow, to shut his eyes and mouth, to breathe through his nose; tell him to try to go to sleep, and assure him that the ether will be given him cautiously. Pour about an ounce of ether into the cone, and approach it slowly towards the patient's face; with a little encouragement he will soon submit to having it brought quite close, for partial anæsthesia is rapidly induced. When once it is close to his face it should not be removed for some minutes, in spite of any struggles or protests. Fortunately, patients rarely recollect what occurs at that time if the cone has been approached gradually. The ether should be given liberally, as atmospheric air is being excluded, and the patient is respiring nothing but ether vapour. Stertorous breathing is a sign that the patient is "over," and that the operation may begin. If there is much lividity, stop giving ether for a short time, and the natural colour will soon return. The ether must be given almost continuously throughout the operation. Stertorous breathing is not a warning of danger. On the contrary, I like to hear this noisy breathing, as I feel sure my patient is going on all right.

Cautions.—It is absolutely necessary that one person should do nothing else but administer the anæsthetic. He should never leave his post to assist or perform other duties. His business is to give the anæsthetic and to watch the