but day by day this diminished. When the tube was withdrawn it continued for a few days, and then entirely ceased. The wound was quite healed in about ten days. Recovery was ultimately perfect.

In boric acid we possess an antiseptic admirably fitted for keeping the external ear and meatus in an aseptic state. It is used dry, but has the property of ready solubility. When in contact with a fluid discharge it first becomes converted into a paste and then undergoes complete solution. It cannot form a retaining plug. It is easily used, and can be managed by an untrained attendant. There is no means of making an effective application or employing any useful treatment to the nasal or pharyngeal cavities. External cleanliness and pure air we can secure. We can see that decayed teeth are removed from the mouth and that proper attention is given to cleansing it. I think, however, that in the treatment of fractures opening into these cavities and into the external meatus trephining may prove to have a useful place. The continual oozing of cerebro-spinal fluid is an important factor in the evolution of septic meningitis; not only does it establish a water way to the interior of the skull, but it produces a sodden state of the tissues very favourable to sepsis. A trephine opening and drainage of the subarachnoid cavity prevents the fluid being under tension and allows it to escape by an arranged safe route. From investigations made in connexion with spinal drainage it apparently does not much matter what part of the cavity is opened; tension is equally relieved by a spinal as by a cephalic drain, but in head injuries there is a practical advantage in placing it at the seat of injury, in that it allows an investigation of the skull and brain at that point which is likely to have suffered from the direct violence. Of lumbar puncture or drainage I have no experience in these injuries. It may be that in the future these will prove useful, but the directness of the trephine opening is to me a recommendation. The opening for drainage is large and not liable to the blocking which may easily close a small tube.

A Lecture

ON

ASTHMA.

DELIVERED IN THE NORTH-EAST LONDON POST-GRADUATE COLLEGE.

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THERE are many varieties of dyspnoea and orthopnoea in so far as they own different causes, or present different pathological features, but apart from these, there is a distinctive idiopathic affection which is termed "asthma." It is sometimes called spasmodic or convulsive asthma, and sometimes bronchial asthma, but if we use the simpler terms "asthma" or "essential asthma" we are committed to no theory as to its pathology or its precise location. Now this disease, as we understand it to-day, has been carefully studied for at least 300 years, since that distinguished Englishman, Thomas Willis, definitely detected its individuality. It is minutely described in many treatises, and were it not that up to the present time its pathology has been the subject of incessant debate, and its treatment correspondingly difficult, there could perhaps be little justification in selecting it as a topic upon which to address you. But I hope to bring forward certain facts which may be of assistance in forming an opinion respecting the nature of this remarkable affection, and, following on that, to offer a few suggestions as to the most propitious method of dealing with it.

HISTORICAL NOTE.

Asthma was confused by the ancients with various affections of the chest. In the seventeenth century Willis recognized its specific features, and propounded the view that it was a convulsive affection of the lungs. Following him came Floyer—himself the subject

of asthma—who in 1698 defined it as due to "contractions of the bronchial muscles and the vesicles of the lungs." He also observed that "the nerves make the same sort of ligature on the arteries, as we observe by the intermitting pulse and the cold hands and feet." The theory of nervous spasm seemed to be established, and was supported by the great authority of Cullen, who flourished from 1770 to 1800. In 1807, however, Dr. Robert Bree, who was also, like Floyer, a martyr to the disease, wrote a treatise in which he combated the prevailing view of a purely nervous spasm. By cogent and ingenious arguments he contended that asthma was a strenuous effort on the part of the lungs to rid themselves of irritating matter. This irritant he believed in most cases to consist in a serous effusion which exuded into the air vesicles. He pointed out the analogies of muscular contractions in other situations to accomplish similar ends. This serous exudation he believed to be chiefly absorbed as the fit proceeded, but a certain portion was expectorated. In some cases, however, those of distinctively "dry" asthma, the irritant was "aerial acrimony" or "subtle matter." There were also certain other subsidiary species of asthma which need not concern us now. Asthma might also be caused by transference to the lungs of certain skin eruptions, by "retrocedent gout," or "viscid mucus secreted by the bronchial glands." Let it be noted that Bree admitted the element of bronchial spasm, which, however, was the outcome of no inscrutable neurosis, but served a definite and salutary purpose.

After Bree came the preponderating influence of Laënnec, who espoused the doctrine of an essential neurosis, and the distinctive views of Bree were gradually lost sight of. Since the time of Laënnec we are confronted with the spectacle of two opposing camps—the disputants concerning themselves not so much with the cause of asthma, as that is unknown, but with the anatomical conditions in the bronchi which occasion the dyspnoea, and these conditions have been narrowed down to spasm on the one hand *versus* hyperaemia or turgescence on the other.

RECENT ANATOMY AND PHYSIOLOGY.

With regard to the bronchial muscles, Aufrecht, by a special method of staining, has discovered the existence of a fine longitudinal layer, closely interwoven with the circular fibres. Herein we are furnished with a new conception of the expulsive mechanism of the bronchi, and it is evident that in the case of asthma, so soon as the imprisoning forces relax, the vermicular contractions of these longitudinal muscles would serve to expel both air and secretion. Then, with respect to their innervation, careful experiments by Dixon and Brodie have demonstrated that the sole supply is from the vagus, which contains both broncho-constrictor and broncho-dilator fibres. The bronchioles are readily constricted by various drugs (for example, muscarine, pilocarpine, barium chloride) acting either on the nerve-endings or on the muscle. This constriction is not accompanied by any congestion or secretion. In like manner, other agents (for example, morphine, atropine, hyoscyamine) cause bronchiolar dilatation. There appears to be no central tonus. The same investigators made another series of experiments with reference to the pulmonary vessels, in which it was proved that they are devoid of vaso-motor nerves. They are entirely passive, accommodating themselves readily to the needs and fluctuations of the circulation generally. Thus, when adrenalin is given via the veins, there is constriction of all the arterioles throughout the body, but the pulmonary vessels become flushed with blood, owing to the backward pressure. Indeed, it was found that when the lung itself is perfused with adrenalin solution, its vessels actually dilate. In poisoning by adrenalin, pulmonary oedema is a marked feature. Such a safety-valve action on the part of these vessels is a remarkable provision of Nature. It is to be remembered, however, that in so far as the bronchi are concerned we are dealing, though not solely, with the small bronchial system of vessels, and these are subject to the ordinary constricting effects of adrenalin. Anatomists state that the bronchial mucosa is supplied by both the pulmonary and the bronchial arteries, and in the case of the bronchioles, the principal supply is not from the bronchial but from the pulmonary artery. This becomes

of importance in connexion with the pathological anatomy of asthma.

SPASM OR HYPERAEMIA?

The typical fit of asthma begins suddenly, usually in the early hours of the morning while the patient is asleep. So sudden is its onset, and so intense may the dyspnoea shortly become, that even *prima facie* the idea of a bronchial spasm seems alone adequate to account for it. If you look through a microscope at a section of lung, and observe a very small bronchus in close proximity to a small artery, you may, unless you be an experienced observer, encounter some difficulty in deciding which is which. They look, generally speaking, very much alike, more especially as regards their muscular tunics. It is also to be noted that during life each undergoes alternate expansion and contraction. And if on various occasions of provocation the vascular tubes are so liable to contractions more or less excessive, is there likely to be any reason for denying a similar possibility or necessity ever to occur in the case of the bronchial tubes?

But in order to show that bronchial constriction is operating in the intense dyspnoea which characterizes the asthmatic paroxysm it may be well to offer some criticisms, and describe certain observations relative to the rival theory of hyperaemia. In the first place, will any one affirm that such an alleged hyperaemia could suddenly cause a much greater obstruction of the air tubes than is produced by acute inflammation? Yet even generalized acute inflammation of the bronchioles is not attended with a dyspnoea anything so severe as we find in asthma. Again, a comparison is sometimes drawn betwixt the turgescence of the nasal mucosa—such, for instance, as we find in hay fever—and a hypothetical swelling of the bronchial mucosa. But, as I endeavoured at one time to demonstrate in a series of measurements taken by means of the camera lucida, such comparison is unwarrantable. The turbinal bones are covered by cavernous erectile tissue, which measures over a sixth of an inch in thickness, while the thickness of the mucous membrane in the smaller bronchi is only $\frac{1}{8}$ inch.

ACTION OF AMYL NITRITE AND ADRENALIN.

If the chest of a normal individual be observed through the screen, and an inhalation of nitrite of amyl—a drug which is often capable of quickly dispersing the asthmatic paroxysm—given, it will be found in the course of two or three minutes that the chest expands, and the lungs will be seen to flare up considerably. This added brightness lasts for a few minutes and then gradually subsides. Incidentally, it may be mentioned that if measurements be taken of the heart's shadow before and during the action of the drug, it will be found that its transverse diameter diminishes concomitantly with the expansion of the lungs to the extent of 1 in. or even $1\frac{1}{2}$ in. Now it is evident that this expansion of the lungs after amyl nitrite can only be explained by a dilating action of the drug on the bronchial muscles, and it is also evident that if the bronchial tubes were swollen by an inordinate vascular turgescence, this dilatation would be mechanically frustrated. Apart from its action on the bronchial muscles, the amyl nitrite greatly diminishes the amount of blood in the pulmonary system, while it flushes the systemic and therefore the bronchial vessels. It might be suggested that this draining away of the pulmonary blood would render the lungs more distensible, but in the case of asthma it is to be remembered that the lungs are already distended, and moreover contain comparatively little blood.

In 1900, Solis-Cohen found that adrenalin was capable of temporarily arresting the fit—an observation easily confirmed by giving a subcutaneous injection of 10 minims of a 1 in 1,000 solution. In this reaction fresh and convincing evidence was thought to be furnished in favour of the theory of hyperaemia. The adrenalin must act by constricting the turgid bronchial vessels. It was therefore rather disconcerting afterwards to discover that adrenalin given subcutaneously—at least, in these small quantities—had no appreciable influence on the blood pressure, as can readily be proved by means of the Riva Rocci apparatus. I have noticed, however, that when the injection is made during the fit, a rise of 10 or 15 mm. Hg may be registered after a few minutes, concurrently with the cessation of the spasm. But this rise cannot be ascribed

to the adrenalin, for it is permanent, and merely represents the return of the normal pressure. We probably, therefore, must refer the prompt action of adrenalin in asthma, even when given subcutaneously, to inhibition of the respiratory centre. In experimental work a shallowing or even stoppage of the respiratory movements is obtained. But even granting, for the sake of argument, that this substance were administered in asthma so as to constrict the bronchial vessels, it would, at the same time, dilate the pulmonary vessels which furnish the main supply of blood to the bronchioles, and these effects are precisely the reverse of those produced by nitrite of amyl. It seems perfectly evident, therefore, that the mechanical condition of the bronchial or pulmonary blood vessels cannot be of much moment in relation to the asthmatic paroxysm.

THE PATHOLOGICAL BASIS OF ASTHMA.

If spasmodic contraction of the bronchial muscles be the main factor relative to the dyspnoea, we know that this forms only a part, and probably only a minor part, of the pathological processes which occur in the bronchial tubes alone. As the attack progresses, and especially towards its termination, we obtain evidence from the characters of the expectoration that the secretory and other structures of the tubes have been engaged in a severe conflict. We find the expectoration to contain numerous epithelial cells, sometimes casts, either epithelial or fibrinous; many eosinophile leucocytes, polynuclear leucocytes, Leyden's crystals, and the remarkable spiral formations of Curschmann, all embedded in masses of mucus. No theory which has been hitherto advanced seems adequate to explain the formation of the spirals, but this much is certain, that they indicate pathological forces acting with great intensity. As regards the eosinophile cells, their presence in the blood, and their local chemiotactic afflux to the bronchial tubes, indicate the existence of a local lesion or irritant. Eosinophilia is found in many skin diseases, and, according to Neusser, in conditions associated with much irritation in the sympathetic nervous system. Some have found reason to believe that the granules of these cells are of the nature of alexins, and are attracted to substances which are associated with the destruction of epithelium. However, these various bodies, which go to make up the expectoration, can only be regarded as the outcome of the conflict which has been raging inside, and they may not inaptly be likened to fallen soldiers on the battle-field.

Then, as regards the systemic blood changes, P. Salecker, who has devoted considerable attention to this point, affirms that both during and after the attack the fluctuations in the blood picture, more especially as regards the polynuclear leucocytosis, admit of a degree which is only found as a consequence of conditions of toxæmia. And so we come to recognize that the asthmatic paroxysm from start to finish—the spasm of the bronchial and inspiratory muscles, the inflation of the lungs, the carbonæmia, the leucocytosis, the output of mucous bodies, spirals, eosinophiles, and the rest—is truly and strictly a process of defence.

The disease, therefore, or specific source of irritation, is for the time being in the lungs; and, whatever its nature, it seems early to excite contraction of the bronchial muscles, probably much in the same way as impure blood is believed to excite contraction of the arterioles. But this symptom of spasm, owing to its painful and obtrusive domination, has so monopolized attention as to have impaired the view of many respecting the limitations of essential asthma. In the absence of a specific organism, it is the secretion—the expectoration—and the blood changes, on which we are to rely in the diagnosis of asthma, and not the spasm. After cessation of the spasm the disease is still there.

Reflex bronchial spasm can be set up in many other ways, and especially by irritation of the nasal mucosa. We have also gastric, bronchitic, gouty, and other forms of asthma, but all these are spurious varieties. They do not show the characteristic expectoration of genuine asthma, and so when you hear that an asthma has been cured by the application of a cautery to the turbinal body or nasal septum, or by a few occasional sniffs from a spray bottle, you know that the disease was not true asthma, and that it was either unattended by secretion, or else that the

secretion never contained the various bodies coexisting, of which mention has been made. It is nevertheless true that the spasmodic element in asthma may often be greatly relieved by intranasal or other treatment directed to any source of reflex irritation. When you are called to a case of asthma, and see, sooner or later, a pot by the patient containing gummy sago-like masses of expectoration, you know you have got the genuine article to deal with. This expectoration may be less or more, but it is never entirely absent, and its composition differs from that produced in any other disease of the lungs. Again, some call asthma a neurosis, but let us beware lest that term merely serves as a cloak to our ignorance. Is it easy to believe that the assemblage and expression of the pathological phenomena we have just been considering may be due merely to a certain vicious and irresponsible action on the part of the bronchial nervous system? It would be more correct to say that asthma is a disease to which a pronounced neurotic element is attached. Iodide of potassium, which more than any other drug influences the status asthmaticus, is not indicated in the purely neurotic affections. Certain of the so-called neuroses are far from being functional disorders, such, for instance, as epilepsy, and so the term is certainly an elastic one. But in chronic asthma, any wreckage found *post mortem* is solely confined to the chest, while the nervous system is healthy, and so we may safely affirm that asthma is a disease of the lungs.

It will now be seen that this view of asthma is essentially that propounded by Bree; but whereas Bree regarded the expectoration as the cause of the attack, we now know that it is merely a result. As to the causative agent, this can merely be a matter of speculation. The long duration of the affection is not necessarily against its being microbial. It may, however, be a toxin or leucomaine, which is either of distinctly pathological origin or else a product of normal metabolism, which gradually accumulates in the blood by reason of some defect, congenital or acquired, in the excretory function of the lungs. There can be little doubt that the lungs are avenues for the excretion of unknown poisons. Asthma commences usually about 2 or 3 a.m., when the opsonin or preopsonin content of the blood reaches its minimum; and we have, perhaps, a parallel instance in cramp, which is generally believed to be due to a toxin acting on the muscles. We also find in asthma, both before and during the attack, profound disturbances in the sympathetic system, and sometimes the affection alternates with eruptive lesions. So when an attack followed the retrocession of certain cutaneous affections, such as urticaria or eczema, it has been thought that the skin lesion actually became transferred to the bronchial tubes. We can only, however, regard such skin lesions as incidents of the toxæmia, and as probably also affording a temporary and vicarious immunity. Evidence of toxæmia is also forthcoming in the various visceral disturbances so frequently encountered. Again, there is nothing prejudicial to this conception of the disease in the circumstance that the fit may be induced by influences acting through the senses or emotions. A fit of malaria is often induced in the infected subject by nervous shock. A fright may easily precipitate an attack of whooping-cough in a child suffering from the disease. And in the case of asthma in this connexion, the influence known as "force of habit" also comes into play.

But if the asthmatic paroxysm be really a defensive process, one is justified in asking if the excessive bronchial constriction with its accompanying distress can possibly serve any useful purpose. This question is most pertinent, and there need be no hesitation in replying that we have here an instance of the well-known fact that (as it appears to us) Nature frequently acts in excess of the requirements. Many examples might be quoted, such as the phenomenon of ileus or of ruptured uterus. It is only right, therefore, that this spasm should be repressed by art, just as we assuage the excessive pain or temperature which are factors in the defensive operations of inflammation. At the same time it is to be remembered that the dyspnoea in asthma is not exclusively due to bronchial spasm. As the fit proceeds, it is partly due to the congestion and secretion in the tubes, and it is likewise maintained and promoted by the excess of carbon dioxide in the blood.

MANAGEMENT OF ASTHMA.

The management of the asthmatic patient generally resolves itself into two distinct periods. One concerns itself with the actual attack, and the other with the intervals of freedom, or comparative freedom.

It is unnecessary to recount the multimiform agents employed to subdue the attack. It is a curious circumstance that asthmatics have peculiarities or idiosyncrasies, so that what benefits one may be useless to another. Just as we are ignorant of the morbid agent which excites the disease, so we are often perplexed as to the action of our remedies. The most we can do during the actual fit is to relieve the dyspnoea, and in view of the considerations which have already been submitted, it must be evident that any attempt to check the fit suddenly by powerful agents is not an ideal method of practice. The relief so produced is but ephemeral, and though it may invest both the patient and physician with a short-lived halo of satisfaction, it nevertheless often betrays temerity rather than wisdom, and may court ultimate failure rather than reasonable success. Certainly the spasm must be mitigated, but it is best to accomplish this if possible by agents which, like the nitrites (as in the ordinary fuming inhalations) and iodide of potassium, do not at the same time imperil the natural order of cure. Such drugs as morphine, atropine, cocaine, chlorbutol, and paraldehyde, may assuredly be required, but ought to be used sparingly and with caution. In the spurious varieties of asthma they may even be occasionally curative, but in essential asthma, while they produce a temporary suspension of the spasm, the secretion, on the other hand, is liable to be suppressed, and this can only act prejudicially by diverting the disease into fresh channels. The arrest of the fit is also too commonly followed by a more speedy recurrence and a longer period of trouble. On the other hand, we promote the best interests of the patient by so repressing the spasm as to permit Nature to effect her purpose with a reasonable degree of comfort to the sufferer. Drugs which numb the nerve centres, or render the bronchial mucosa insensitive, not only tend to arrest or pervert those metabolic processes designed to rid the system of the asthma poison, but are likely also, unless carefully employed, to occasion the necessity for a too frequent resort to their use, so that the patient, instead of obtaining and utilizing the opportunity which is available for the only real means of cure, is made the slave of a habit which is so harmful because so insidious.

But it is after the passing of the acute attack, during the periods of remission, that the real possibility of subduing the disease is presented, and in order to accomplish this the intelligent co-operation of the patient is essential. The supposition, should it exist, that asthma is an incurable disease, must be dismissed. No doubt when the pathology of the affection is better understood a speedy and effective remedy will be forthcoming, but even with the means now at our disposal, cures may be effected, or, short of actual cure, a life of comparative comfort, with possibilities of regular work, may be substituted for a régime of invalidism, and I am able to show you several patients illustrative of that contention. Now, the first thing to be done in tackling an asthmatic case is to make a careful examination of any region, more especially within the jurisdiction of the pneumogastric nerve, where an irritative lesion might be likely to exist capable of exciting or assisting bronchial spasm. The nose, naso-pharynx, and the stomach are our chief concern. Of the latter, mention will shortly be made. The nasal cavities present areas the irritation of which, even in the normal state, by chemical or electrical stimuli, may occasion reflex bronchial spasm. A good many years ago Albert Abrams found that this spasm could be induced by packing the healthy nostrils with cotton-wool, and that it failed to appear if cocaine were first applied to the interior of the nares. Hence we find that cocaine applied in this fashion may mitigate the asthmatic attack. Any nasal thickenings or hyperæsthetic areas must therefore be rectified, and the same applies to enlarged tonsils, adenoids, granular pharyngitis, etc.

After this, the general or constitutional treatment is to be considered. We note, in the first place, that asthmatics exhibit idiosyncrasies in respect to environment, just as they do to drugs. Some do well in cities, others in the

open country; some at high atmospheric pressures, others at low. Discrepancies are also observed in regard to foods and beverages and sensory impressions. In the case of the well-to-do, a suitable climate and locality can generally be obtained, and the patient should begin, when possible, a systematic course of respiratory exercises. The lungs must be properly ventilated. A regulated amount of hill-climbing is an excellent form of pulmonary exercise. Marcet has a high opinion of the value of cycling as a training for the respiration, as it increases chiefly the depth of breathing, carrying the tidal air through the lungs. I have often advised swimming as an ideal form of exercise for asthmatics, since it not only serves to increase the depth of the breathing, but it also brings into play the accessory muscles of respiration, and so improves their functions, which become disorganized by the asthmatic attacks. It is likewise a tonic to the nervous system. These various exercises also, by improving the pulmonary circulation, tend to correct the peculiar anaemia which is a feature in most sufferers from this disease. There is undoubtedly an asthmatic "cachexia," which must be reckoned with. There is a form of anaemia visibly present, which we may designate "pulmonary anaemia," and there is a defect of metabolism, the burden of which comes to be thrown on the lungs. A careful regulation of the various excretory functions is therefore of paramount importance. This is chiefly to be obtained through careful dieting, by spa treatment, and especially by promoting elimination through the skin, which is not infrequently affected in asthma, as though the toxin sought an outlet there. The Turkish or electric light baths, regularly employed, may effect a surprising amount of good. In regard to diet, the "acarbonizing" system of Hare—that is, an adequate restriction of the carbonaceous intake—is to be carefully considered, and the same applies to the purins. Generally speaking, the directions as to diet given by Cullen cannot very well be improved upon—namely, "none of them bear a large or full meal, or any food that is slow and difficult of solution in the stomach, but many of them bear animal foods of the lighter kinds and in moderate quantity. The use of vegetables which readily prove flatulent is always very hurtful. Water or cool, watery liquors are the only safe and fit forms of drink, and all liquors ready to ferment and become flatulent are hurtful. Few asthmatics can bear any sort of strong drink." In addition to these measures, the avoidance of worry and the preservation of a tranquil and cheerful disposition are most helpful.

The medicinal treatment of the asthmatic patient is of high importance. Most cases receive great benefit from iodide of potassium, which is often prescribed in association with arsenic. I have had excellent results from the iodide with Peruvian balsam, and in emaciated patients exhibiting a marked bronchitic element the balsam with cod-liver oil is an excellent remedy.

Mention has been made of the peculiar anaemia which characterizes the asthmatic diathesis, and for this a prolonged course of iron, either alone or with arsenic, is attended with the greatest benefit. The value of iron was recognized by Bree and also by Laënnec, but it is not used as it deserves to be. Certain of these remedies may be alternated with quinine and strychnine, both of which are highly beneficial. The action of adrenalin in the attacks might seem to indicate its employment as a specific tonic and alterative, but it has been found to be useless.

Lastly, some medical men in America have recently recommended the diphtheria antitoxin, both as a remedy in the attack and also as a cure. It is given in full doses, up to 4,000 units. At present it is being tried here, and the results certainly justify an extended investigation. These, however, are probably due to the horse serum; that is a question which remains to be decided.

When the disabling effects of prolonged asthma are regarded, there is no disease of which it may with greater truth be affirmed that it requires to be consistently and watchfully treated. Fortunately or unfortunately, there is no one plan of treatment applicable all round. Each case must be individually studied, and the various functional or organic defects considered in their correlative aspects. It is thus only that you can hope to grapple successfully with an affection having so inscrutable an origin, so erratic a course, and so firm an attachment to its host.

PYREXIA DURING THE PUERPERIUM.

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PYREXIA is a symptom common to a number of conditions, some of which it would be misleading to describe as morbid, whereas others are amongst the most serious with which we have to deal.

It is the most frequent accompaniment of puerperal infection, to which references are found in many of the earliest medical works, and which still occupies a considerable space in the professional press of to-day.

Under the title "puerperal fevers," the textbooks even of our fathers described a number of distinct diseases, which had nothing in common beyond their frequent occurrence in puerperal women. "Milk fever," "ephemeral fever or weed," "intestinal fever," "miliary fever," and others, each had its chapter, with description of symptoms, diagnosis, and treatment. Since, however, the close analogy between puerperal and surgical fever was pointed out, and more rational methods for the prevention of wound infection were introduced, the etiology and treatment of this group of diseases has been greatly simplified.

My object in this paper—which is based upon the examination of 1,196 cases of childbirth attended during twelve consecutive months by the extern midwifery department of St. Bartholomew's Hospital—will be to show, as far as possible, the frequency, origin, and course of all abnormal rises of temperature noted during that period.

Not every rise of temperature is abnormal; considerable variation within certain limits may occur even in a perfectly healthy individual, quite apart from the puerperal state. Mental and physical activity tends to increase the body temperature, which therefore, except in night-workers, attains its maximum during the day, and its minimum at night.

During gestation the temperature is usually the same as in the healthy non-pregnant state. During labour the unusual muscular activity increases the temperature, which, usually normal at the beginning, gradually rises as labour progresses. As might be expected, the temperature is somewhat higher in primiparae than in multiparae, higher in difficult and protracted than in short easy labours, especially when the second or expulsive stage is unduly prolonged.

After the end of labour there is normally a slight steady rise, followed by a gradual fall. Dr. Chalmers Cameron has stated that this maximum is normally reached in primiparae in twelve hours, and in multiparae in six or seven hours, the gradual fall occupying about ten to twelve hours in both cases.

From the limited number of normal cases in which I have had the temperature taken almost every hour during the first day, I have been unable to find any such definite period for the maximum and minimum in multiparae and primiparae respectively. It appears, however, that this rise and fall after labour is modified by the usual diurnal rise and fall, so that the actual temperature in any given case is really the resultant of these two. It is evident, therefore, that the normal temperature of the first day hinges upon the time when labour terminated. If this slight rise after labour coincides in point of time with the normal daily rise, the resultant temperature may run up to 100°, or possibly 101° F.; but, if counterbalanced by the daily physiological ebb, there may be little or no observable elevation of temperature. In a general way it may be said that normal births during the night are generally followed by little or no rise in the first twenty-four hours; those during the day by a marked rise, though within the limits regarded as normal.

As to the limits of a normal puerperium, great difference of opinion still exists, and there is no uniform standard of morbidity in general use in the various maternity hospitals of the kingdom.

At the annual meeting of the British Medical Association at Leicester in 1905 a special committee was appointed "to inquire into the matter." A schedule of questions as to their standard of morbidity was sent to maternity