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Original Communications.

THE NECESSITY OF FINDING THE CAUSE OF RECURRENT EARACHE WHICH SUBSIDES WITHOUT APPARENT INJURY TO THE EAR.*

BY HIRAM WOODS, M D.,

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No physician of considerable practice can have failed to have patients consult him on account of recurring earache. In some cases the pain comes in paroxysms of two or three hours' duration, disappears, sometimes spontaneously, sometimes only after the use of hot applications and anodynes, and returns after a long or short interval. Again, there are no distinct paroxysms of pain. The individual is conscious now and then that his ear hurts him. It never confines him to the house, and he may make only an incidental allusion to it when he happens to feel a pang, or thinks of the matter when with his physician. While by no means always so, the first class is usually made up of children, the second of adults. One will find, I think, that the subsequent histories of such cases divide them into three groups: (1) those who "outgrow," as it is called, the earache, or at any rate cease to have the attacks, and retain good hearing; (2) those who continue to have occasional paroxysms, or else now and then feel a twinge of pain, without the development of any special symptoms, save a slight, and possibly transient, defect in hearing; (3) those who after an attack of earache have a serous or purulent otorrhœa. This may subside and leave the ear still useful, but nevertheless impaired, or it may become chronic.

Thus earache may be a trivial matter. Again, it may be a symptom of an inflammation, which will soon show itself by a discharge, or else be the only appreciable indication of conditions which can cause slow changes in the ear and lessen its usefulness. If the diagnosis "earache" be looked upon as good and sufficient, and therapeusis be limited to relieving the patient's suffering, *possibly* no harm will be done; but in the majority of cases important things will be overlooked, and harm will result. The object of this paper is to make a brief study of these cases of earache with special reference to their effects and causes. Barring furuncular and diffuse inflammation of the external auditory canal, painful affections of the ear are due, usually, to catarrhal inflammation of the tympanic cavity, or to reflex neuralgia of the ear from some cause outside of the ear itself. Canal inflammation generally shows itself clearly enough and need not be considered. Of catarrhal inflammation of the drum cavity, many cases pursue the typical course of hyperæmia of the tympanic mucosa, exudation into the drum cavity, perforation of the drumhead, and the establishment of an otorrhœa. Pain is the most prominent symptom of the stages of hyperæmia and exudation, and is relieved when the drumhead ruptures. But all cases do not go so far as perforation. There is hyperæmia of the tympanic mucous membrane, and examination of the ear with the head-mirror, reflected light and ear speculum reveals the vascular changes in the drumhead characteristic of acute aural catarrh; but there is never the bulging of the drumhead indicative of exudation, hearing may not be greatly impaired, the drum can be inflated through the Eustachian tube, and the trouble does not go beyond the stage of hyperæmia. There are a great many such cases of abortive acute aural catarrh observed in an aural clinic. Tympanic hyperæmia may occur once or twice as the result of cold or exposure, and subside without serious results; but when it occurs again and again two things become manifest: (1) the usual results of repeated hyperæmia will probably ensue in the tympanic cavity, and (2) there must be some cause of these attacks more or less closely connected with the ear. That acute catarrh of the tympanum is the most common cause of the repeated earaches frequently observed in children is the opinion of such authors as Woakes, Roosa, and Buck; but the pain soon subsides, the hear-

*Read before the Medical and Chirurgical Faculty of Maryland, at Easton, November 15th, 1892.

ing continues good, and nothing more is thought of the matter till the next attack. If more care were taken to make the tests, there is little doubt but that the hearing of ears which have passed through two or three such attacks would be found impaired. Still, as one can lose nearly one-half of the normal hearing power without being specially inconvenienced, the slow deterioration is not noticed for a long time. This gradual lessening of the hearing power after repeated tympanic hyperæmia is the result of connective tissue formation in the mucous membrane covering the walls of, and ossicles in, the drum cavity. The ossicles become adherent to the walls of the cavity at the points where they touch, the joints between the ossicles become anchylosed, and so the power of conducting sound-waves to the nervous ear is lessened.

Again, as this thickening advances, the tympanum becomes less able to withstand fresh attacks, and so an otorrhœa is apt to eventually result, with all its attendant inconveniences and perils ; nor do the dangers from tympanic catarrh, not diagnosticable except by objective examination, and presenting only symptoms of severe pain in the ear, stop at the point mentioned. Such attacks are very common among infants. Woakes thinks convulsions are often caused by pressure upon the labyrinth, from an exudation into the tympanum due to an acute aural catarrh, resulting from dentition. He, Politzer and others describe a fold of the meninges, which in infancy passes through the petro-squamosal suture into the drum. Cases of fatal meningitis may thus develop before the tympanic inflammation has caused rupture of the drumhead. While such results may be rare, certain it is that the ultimate production of a foul otorrhœa in infants, after one or more neglected attacks of earache during dentition, is a common occurrence. Possibly the ear is not thought of as a source of pain, until the appearance of the discharge. Deaf-mutism can thus result if the hearing becomes greatly impaired before speech has been learned, even if the child escapes the more fatal dangers of an otorrhœa.

Having thus reviewed some of the consequences of tympanic catarrh of which *pain* is usually the only symptom, I beg to lay stress upon the facts that earache is only a *symptom* ; that diagnosis must extend to the discovery of its cause ; that if this cause, in turn, is due to other abnormal conditions, they must be found ; that the therapeusis of earache, especially recurring earache, must go further than relief of pain. There is not the space in the limits of this paper to enter into the therapeusis of tympanic catarrh, appropriate as such a course might be. To some of its causes, too frequently overlooked, I desire to direct attention.

Chronic abnormalities of the naso-pharynx are a prolific cause of tympanic catarrh. Follicular pharyngitis, post-nasal vegetations and hypertrophied tonsils are, in my experience, the most common throat lesions observed in connection with recurring earache. It is, I think, a more or less common belief that, if chronic follicular pharyngitis does not cause so much throat discomfort as to call attention to itself, or if post-nasal adenoid vegetations do not interfere with nasal respiration, these troubles may be left alone. That they can produce deafness and recurring hyperæmia of the tympanum without special throat or nasal symptoms, I do not think admits of doubt. Situated, as they often are, near the pharyngeal mouths of the Eustachian tubes, these inflamed follicles or vegetations act as irritants, increase the vascularity of the tubes, and cause an Eustachian catarrh. This can reach the tympanum by direct continuity of mucous membrane. Again, as soon as ventilation of the tympanum through the Eustachians is hindered, and the air already in the tympanum has been absorbed—no renewal taking place through the tubes—atmospheric pressure in the external canal drives the drumhead inwards, producing undue pressure upon the ossicles. Impairment of hearing and tinnitus usually follow at once. If unrelieved, hyperæmia and pain follow. Relief comes as soon as the Eustachians again admit air to the drums. Inflation by Politzer's method promptly removes the ear symptoms and the application of a nitrate of silver solution to the mouths of the tubes lessens the secondary catarrh ; but it will surely return, unless the primary trouble is removed. As regards enlarged tonsils, their importance, from an otological standpoint, has been exaggerated. Probably they rarely occur unaccompanied by other morbid conditions of the throat, which more immediately affect the ear. By lessing the air-space, they may, indeed, produce these conditions. This will certainly be the case if they interfere with nasal respiration. The same is true,

however, of any conditions which block the nostrils. Mouth-breaking is a well-known cause of pharyngeal disease, and when nasal respiration is impeded in persons suffering from ear symptoms, it should be re-established. Still, so far as the *direct* influence of hypertrophied tonsils upon the ear is concerned, Roosa states that it is doubtful if they ever enlarge to the extent of pressing upon the mouths of the tubes. He advises their removal upon the grounds I have advanced: that they may "effect the health of the pharynx." I have seen patients cured of middle ear disease by the removal of post-nasal vegetations, although hypertrophied tonsils were also present. Another source of danger to the ears from naso-pharyngeal disease direct microbic invasion through the tubes. This undoubtedly occurs.

Occasionally one will observe a patient who has earache, and possibly defective hearing, and find one or more of the throat lesions mentioned, but the examination of the ear will be negative. The drumhead does not present any increased vascularity. Evidently there is no tympanic inflammation. The pain is not so severe or lasting as in tympanic catarrh. It is felt as a shooting neuralgic pain in the ear. I have had under my care two sisters who have shown this condition. One consulted me for occasional attacks of deafness and earache some time ago. I reported her case in the *Maryland Medical Journal* of Dec. 26th, 1891, in an article upon post-nasal vegetations as a cause of deafness. I frequently examined her ear when painful, but there was no inflammation. When I removed the vegetations with Mackenzie's forceps, she experienced severe pain in both ears. Her sister has follicular pharyngitis and tonsillitis. Earache with her is not a marked symptom, but her hearing has been poor. I have, however, often produced an otalgia, or ear neuralgia, with her by simply pressing the tonsils with a probe, or applying an applicator to the naso-pharynx.

I experienced myself last spring a definite and painful proof of the power of throat disease to cause reflex earache without inflammatory changes. I was suffering from an attack of acute tonsillitis on the left side. The afternoon of the second day, my left ear gave me some pain. This steadily increased until by night it was agonizing. I obtained some relief from anodynes, but very little. I could still hear fairly, and could inflate the drum through the Eustachian. Early in the morning I sent for Prof. Chisolm. I feared that he would find an acute aural catarrh. Greatly to my relief he did not. His words were: "It is reflex. The drumhead is not even congested." The correctness of his diagnosis was proven by the sequel. I obtained some relief from the large doses of salicylate of sodium he ordered, but the pain did not cease till my tonsil was well. No ear trouble followed. These cases prove, I think, the power of throat lesions to produce a purely neuralgic earache. Whether or not this reflex can eventually cause organic lesions in the ear, I am not prepared to say; still, they bring us straight back to my theme—the necessity of finding the cause of earache. In the cases of the two sisters mentioned, the causes were of themselves capable of damaging the ears through the Eustachians. The channel of transmission in these cases was almost certainly the glosso-pharyngeal nerve, which supplies the tonsils, pharynx and tympanum with sensory fibres.

The teeth, and more particularly dentition, constitute a source of ear disease which is not sufficiently appreciated. The occurrence of otorrhœa in babies during dentition is frequently observed. Earache in infants, I am sure, is not always recognized as promptly as it should be. I see babies with otorrhœa whose clinical history is very clearly read backwards from the otorrhœa to dentition, but the pain the little one then had in the ear was not attributed to that organ. I have now a little patient nineteen months old, who first had otorrhœa when one year old, the sequel of measles. Both ears are affected. Twice have I succeeded in stopping the discharge, and twice has the boy had a relapse, each time at the cutting of a new tooth.

Sexton, of New York, who has given the subject of oral irritation careful study, considers irritation from the mouth a most prolific cause of ear disease. He goes so far as to condemn amalgum fillings, vulcanite plates, and retention of teeth which have lost their nerve pulp as dangerous to the integrity of the ears. I have tried to make some clinical observations upon this subject. While I have seen nothing to lead me to accept all Sexton says, I have over and over again seen

earache, sometimes accompanied by hyperæmia of the drumhead, and sometimes not, cured only after a carious tooth had been removed, or cleaned out and filled.

The channel of transmission from the teeth to the ears may be directly through the fibres of the fifth from the dental to the auriculo-temporal branch. This may be the case in those patients whose trouble is only *pain* of a reflex character unaccompanied by inflammatory changes; but it will not explain the acute aural catarrh and suppurative otitis of dentition. Two explanations of these lesions are given: (1) extension of the inflammation from the gums to the middle ear by direct continuity of tissue. Roosa says he has seen this. Woakes, on the other hand, holds that the intermediate tissues are healthy, and offers, as an explanation of the tympanic catarrh, vasomotor disturbance. He traces the irritation from the teeth along the afferent sympathetic fibres accompanying the dental branches of the fifth to the otic ganglion. Here the nervi vasorum of the carotid plexus are met and receive reflex irritation. This causes dilatation of the tympanic branch of the internal carotid going to the drumhead. Thus a hyperæmia of the drumhead is produced. Its vessels anastomose freely with those of the drum cavity, and tympanic hyperæmia results—the first step in the production of tympanic catarrh.

Justifiable conclusions from the foregoing are that the diagnosis in cases of recurrent earache must include the condition of the drumhead, pharynx, nose and teeth; that therapeusis must include the treatment of disease found in these structures.

DIAGNOSIS OF DIPHTHERIA.*

BY A. S. FRASER M D., SARNIA, ONT.

The diagnosis of diphtheria is comparatively easy in the majority of cases. The thick white patch with its red border of inflamed mucous membrane is not likely to be mistaken for anything else. Unfortunately, however, the characteristic appearance is not formed in a large proportion of the cases met with in practice, and as the divergence from the typical form increases, the difficulty of recognizing the disease becomes greater, until at last a point is reached where it will be impossible for even the most experienced to decide whether an existing morbid condition is diphtheria or not.

The constitutional symptoms of pharyngitis are exactly like those of the milder forms of diphtheria, and although the exudation in pharyngitis is usually soft and easily washed or brushed from the mucous membrane, it becomes at times firmer and adheres to the underlying tissue with considerable tenacity, and is then easily mistaken for the false membrane of diphtheria. In mild cases of follicular tonsillitis the exudations from several lacunæ often coalesce and form a somewhat thick white mass, which may adhere to the surface of the tonsil for several days having much the same appearance as diphtheria. The symptoms of a severe attack of follicular tonsillitis are similar to those of the graver forms of diphtheria. In the former, however, the temperature is higher, albuminuria, when present, occurs later and does not last long, the exudation is not so extensive and there is less nervous depression. The inflamed lympathic glands usually found in diphtheria are frequently met with in tonsillitis, while paralysis of the palate and pharynx may follow either.

In endeavoring to arrive at a conclusion as to whether the diseased condition of the pharynx in a given case is diphtheria or not, some other sources of error besides those already referred to have to be guarded against. In making an examination of the pharynx small diphtheritic deposits are liable to escape notice unless the patient makes an effort, to vomit when the tonsils will revolve outward and expose their posterior surfaces; occasionally a laryngoscopic mirror may be useful in making this examination. It will sometimes happen that after a case of pharyngitis has pursued the ordinary course for several days, a false membrane will form and other symptoms of a serious attack of diphtheria become developed, but it is generally admitted that there is a form of diphtheria in which no false membrane is found, and which has no constant or reliable symptoms to distinguish it from ordinary pharyngitis. Sir Morell McKenzie believed that in this form of diphtheria the disease was arrested in its first stage of development, and that its nature could not be known until the occurrence of paralysis, or until there was unmistakable evidence that diphtheria had been contracted from the patient.

*Read before the Ontario Medical Association, June, 1892.

It has been held by some observers that the exudation of follicular tonsillitis while adhering to the tonsil may become invaded by the germs of diphtheria, but owing to the absence of lymphatics in this locality, and to the fact that some persons are not at all susceptible to diphtheria, no result follows so far as the individual in question is concerned, although he is no doubt as much a source of danger to others as if he had the disease in the usual manner. It is impossible to make a diagnosis of laryngeal diphtheria in the early stages of the disease, unless a diphtheritic membrane can be seen in the pharynx. Without this there is no way of distinguishing it from catarrh of the larynx until the mucous secretion becomes free, when, if there is some rise of temperature, we may infer that it is the latter complaint we are dealing with. Cases will sometimes occur, however, in which it will be perfectly certain that there is catarrh of the larynx, and yet the stenosis will go on increasing instead of becoming less, as we might expect, until there is complete obstruction of the larynx; usually, however, some pieces of false membrane will be expelled before death occurs. In this connection a short account of some cases that have come under my own observation may not be without interest.

CASE 1.—A little girl, seven and a half years of age, was taken ill about the first of January, 1890. At the time of my visit, twenty-four hours from the beginning of her illness, she was slightly feverish and had a harsh dry cough. She was suffering considerably from nervous depression, there was no indication of diphtheria, but she was the subject of a chronic naso-pharyngeal catarrh, which I knew had existed for a considerable length of time. Although a positive opinion could not be formed at this stage of the disease, I was inclined to consider the case as one of catarrh of the larynx. The next day the child's father reported to me that the cough had become loose, and that she was considerably better. I did not see the child for three days, when I found very decided stenosis of the larynx. The tonsils were covered with typical diphtheritic membrane; there was no fever. The nervous depression had greatly increased, and the case was evidently hopeless; she gradually sank and died four days afterwards.

CASE 1.—A few days before the death of the last patient her sister, a girl thirteen years of age, commenced to have symptoms of stenosis of the larynx. On examining the pharynx, I found small but characteristic diphtheritic deposits on both tonsils. This girl never appeared very ill at any time, the larynx became clear in about ten days, and her recovery was soon complete.

CASE 3.—Two weeks subsequent to the recovery of the last patient, I was sent for to see an infant eight months old, in the same family. This child had no fever, was bright and cheerful, and had a perfectly healthy pharynx; the breathing did not seem more difficult than might be expected from a slight catarrh of the larynx and trachea. In a day or two the cough became soft, and the case appeared to be pursuing the course of a mild catarrh when signs of laryngeal obstruction suddenly made their appearance, some false membrane was expelled, and the child died from complete obstruction of the larynx a few hours afterwards.

CASE 4.—Nine months after the occurrence of these cases, I was asked to see, in consultation, a child living in a house adjoining, occupied by them. This was a boy six years of age, of whom the following history was given by his mother: The boy had been suffering from croup for two weeks, but as he had been frequently affected in the same way, his parents were not particularly anxious about him. The day before my visit an infant in the family, about eighteen months old, suddenly sickened and died in a few hours, apparently of suffocation; the family then became alarmed, and sent for a physician to see the boy.

When I saw this patient his temperature was 101, pulse quick; there were symptoms of laryngeal obstruction, but the cough was softer than that usually present in croup; there was a slight nasal catarrh; the pharynx was perfectly healthy. The obstruction of the larynx increased; tracheotomy was performed and gave relief for several hours; some pieces of false membrane were expelled through the tube; the obstruction formed below the opening in the trachea, and death, resulting from asphyxia, occurred the second day after the operation.

CASE 5.—Several months after the occurrence of the last case, I was called to see a child three years old, living on the same street, and on the same side of the street, and about a block distant, from the house where the last case occurred. This child was slightly feverish, had a croupy cough,

and evidently felt very sick; on the pharynx there was a slight but characteristic exudation of pharyngitis. The next day the cough was somewhat softer, but there was evidence of laryngeal obstruction which continued for two days, when a large piece of false membrane was expelled, and the child appeared quite well. Two days afterwards, however, the membrane formed again, and complete obstruction of the larynx occurred.

CASE 6.—Six months after the death of last patient I was asked to see, in consultation, a child who lived in a house adjoining those in which the children first mentioned were sick. This patient was a boy four years old who had been ill for about a week at the time of my visit. There was an exudation in the pharynx, evidently belonging to a catarrhal pharyngitis. There was acute catarrh of the larynx and trachea; the cough became soft, and the child seemed much better next day, and I did not see him again for four days. He was then dying evidently from obstruction of the larynx, although the secretion of mucous was quite free. The temperature never exceeded 101, he was sick about ten days.

Now it may be questioned whether these were all cases of diphtheria; the first three evidently were, the false membrane in two of the others, and the characteristic features of a relapse in one of them, indicated pretty clearly the nature of the complaint. There may be a doubt concerning the sixth case, as no false membrane was visible, but the extreme rarity of death from catarrhal affections of the larynx would leave little room for doubt that this child also died from laryngeal diphtheria.

All the houses in which these children were sick were close together, but the length of the intervals between the occurrence of the illness in the different houses made it improbable that the disease was contracted from the first by the succeeding cases. Although the district was rather thickly populated there was no diphtheria in the neighborhood, nor so far as I could learn, within a radius of several miles. The houses in which these children were ill, were all situated within two hundred yards from the entrance to the St. Clair tunnel, which was in process of construction at that time, and which was probably the source of emanations which at least largely contributed to the illness of the children.

The diagnosis of nasal diphtheria is usually not very difficult, even in those rare cases were the disease begins in the nares, the profound constitutional disturbance, and the inflamed lymphatic glands at the angle of the jaw indicate the nature of the nasal obstruction, although no membrane may be visible. It is not by any means easy to distinguish scarlet fever from diphtheria in all cases. The higher temperature and more uniform redness of the throat usually serve to indicate, in the earlier stages of the attack, that it is not diphtheria. In diphtheria the nervous depression is much greater than in scarlet fever, while the rash, when present in diphtheria, has much less extension than that of scarlet fever, and, as it never desquamates, can thus be distinguished from the latter in the course of a few days. Concerning the membrane that forms in the pharynx during the progress of some cases of scarlet fever although it has the same anatomical characters as the false membrane of diphtheria, it is not capable of giving rise to that disease.

Until recently it could not be said that the microscope had given any material aid in the diagnosis of diphtheria, but since the discovery that the disease has a characteristic bacillus it is not only useful but will soon be essential in the diagnosis of doubtful cases. As yet the general practitioner is not sufficiently familiar with the appearance of the bacillus or with the methods necessary for its discovery to turn to account what will, no doubt, eventually prove to be an expedient of the greatest value.

While the difficulties in the way of making a distinction between diphtheria and catarrhal diseases of the pharynx and larynx remain as formidable as at present, the duties and responsibilities of the medical practitioner will become at times exceedingly perplexing, for while the conscientious physician will use every effort to prevent the spread of such a cruel malady as diphtheria, it will frequently be found necessary to watch, and perhaps isolate, cases which he is much inclined to regard as simple pharyngitis. In order to accomplish this it may be necessary to express the opinion that the patient is probably suffering from diphtheria, or else admit that he does not know what the disease is. In the one case he will, perhaps, get the reputation of being an alarmist, or one who is trying dishonestly to make his trifling

cases of great importance; in the other he will be considered unskillful and far inferior to practitioners who find no such difficulties. But if he yields to these influences and allows a suspected case to come in contact with healthy children, he will expose himself to severe and just censure.

A point which may be properly considered with the diagnosis, and one which does not appear to have received the attention which it deserves, is this: Does one attack of diphtheria afford any protection against subsequent attacks, as is the case with nearly all infectious diseases? If this were not so, would it not frequently happen that families once attacked by diphtheria, and continuing to live under exactly the same conditions, would have the disease almost constantly with them, and would not children living in unhealthy dwellings be liable to a second or third attack at varying intervals when there had been no cleaning or disinfection of the dwellings after the disease had once occurred. It must not be forgotten that second or even third attacks do occur, though rarely in small-pox, scarlet fever and measles.

Selected Articles.

A CONTRIBUTION TO THE SUBJECT OF ACUTE PLEURISY: ITS PATHOLOGY, ETIOLOGY, SYMPTOMATOLOGY AND TREATMENT.

The proper classification of an inflammatory affection of a serous membrane, and especially of the pleura, is not an easy task. The terms "acute," "subacute," and "chronic," that have been applied to diseases of that nature, are often subjects of much criticism, no matter what pathologic lesions or symptomatic indications they may be derived from.

In considering acute pleurisy, I shall not hope to unravel these disputed questions, and only trust that I shall not fall into the common error of making them more intricate.

The term "acute" pleurisy indicates an inflammatory condition, more or less general in extent, of the serous membrane lining the pleural cavity, with sero-fibrinous or purulent deposits, primary or secondary in nature, of microbic or other origin, with a certain degree of sharpness of attack, and in which the height of the disease is attained in a few days, ten at the most, and accompanied by a rise in temperature of greater or less extent. Acute pleurisy may thus be (1) dependent on pre-existing disease or lesions, the development of which may cause sudden intense inflammatory conditions of the pleura, more or less extensive; or (2), it may be an idiopathic disease.

Pathology.—Hyperæmia, or congestion of the blood-vessels within the serous and subserous connective tissue, causing swelling, redness, and edema of the pleura, is the first noticeable lesion of acute idiopathic pleurisy. This congestion may take place in some localized spot and rapidly extend to a greater or less degree; or it may, from the first, present quite a general appearance over the surface of the pleura. Soon the smaller and weaker capillary vessels rupture, producing ecchymotic spots over the membrane, and, at the same time, infiltration of the subserous connective tissue occurs, with a proliferation and detachment of epithelial cells. As the process progresses the pleura is studded with fine granulations upon its surface, in which appear embryonic cells. These tend to organize the newly-formed connective tissue into firm fibrinous bands, which, in old cases, are often found stretching across the pleural cavity, and constitute the so-called neo-membranes. Finally, from the congested serous membrane is poured into the cavity a liquid resembling in all respects the plasma of the blood, except that it is more dilute; the degree of dilution, however, varies with the intensity of the congestion, according to the coagulability of the fibrin in the effused fluid. There are, also, red blood-corpuscles and leukocytes in the liquid; but in simple sero-fibrinous pleurisy the red globules are not in sufficient numbers to cause any marked discoloration; if such occur, the pleurisy is termed "hæmorrhagic."

"Inflammation," says M. Germain Sée, "is a struggle for life, and not a destructive process; it is essentially a vital phenomenon eminently reactionary against a morbid agent." He believes that the teachings of microbiology show that inflammation is a physiologic process strongly exaggerated; a general struggle of the organism against microbic invaders. "The first step in this process," he says, "is leukocytosis, or the exaggerated production of white corpuscles in the blood; and the second is the absorption and destruction of micro organisms by these leukocytes, showing the defensive action of the latter; this is called "phagocytosis."

Certain physiologic functions are attributed to the phagocytes:

1. They carry from the albuminates in the intestinal canal material for combustion in the tissues.

2. Owing to their ameboid movements they are capable of transporting to distant parts of the body substances in their vicinity.

3. They possess, under certain conditions, a reproductive function, and a power of collecting themselves in vast quantities in certain localities.

4. They have what is known as a chemotactic function; so that the leukocytes thus play a considerable rôle in the morphologic contexture, as well as the chemistry of the body.

The French supporters of the germ-theory of disease, believe that when microbes have penetrated the organism, the leukocytes increase; that the conditions that bring them to the blood carry them to the point of excitement, and that it is a chemical property that draws them there. This, they claim, is true of all high-grade inflammations, as pneumonia, but not of the low grade, as malarial fevers.

In pneumonia there is an increase of leukocytes during the height of the disease, but as the fever abates the number of leukocytes diminishes. It is, therefore, held that the real termination of the disease, whether by crisis or lysis, occurs at the time when the leukocytes begin to diminish in the blood.

In acute inflammation there are three stages of development: First, vascular dilatation; second, activity and proliferation of endothelium; third, exudation, with diapedesis of leukocytes. As a consequence of these three stages it is claimed that a great afflux of phagocytes takes place toward the point of attack, both in purulent as well as in catarrhal inflammations; it is less seen in serous varieties, and, perhaps, not at all in infectious processes, the reason for this being that the infectious matters destroy the phagocytic function of the leukocytes, and hence the body has no protecting element against the enemy, and becomes the prey of the microbe. Supuration is considered no exaggeration of inflammation, but is primarily due to the action of streptococci or staphylococci, and although great numbers of phagocytes may be found, yet their defensive action is much harassed by their deadly enemy, the pus-germs.

From the foregoing, if we are to accept this doctrine, we may conclude that inflammation is a physiologic process to develop phagocytes for the purpose of antagonizing microbes.

Anatomically the pleura is a great lymphatic sac, contiguous with the arterio-pulmonary system, and naturally derives its serosity from that source.

According to the eminent French teacher, M. Guerin, "pleurisy is nothing else than lymphangitis." "If one injected the pulmonary artery," says Guerin, "with colored liquid, it would be found that the liquid would appear in the polygonal ramifications of the lymphatics of the pleura."

Moreover, he has practised on the cadaver the injection of bullock's blood in the same manner, and finds that he obtains a serosity from the pleura which, if much force is used in the process of injecting, becomes bloody, or red in color. This is on the healthy lung and pleura. If, on the other hand, he injects a colored liquid in a subject that has suffered and died from pleurisy, there will be found no coloring-matter in the meshes of the lymphatics except those that have not been affected by the inflammatory process. If this be true, we can readily see that when from some cause the lymphatics of the pleura become congested and swollen, and the natural channels for the lymph impeded, an edematous condition of the subserous connective tissue will arise, due to forced diapedesis of the serous exudate, and cause the fluid to ooze from all parts into the cavity.

If, then, the lymphatic stoppage is complete enough, and the force behind is strong enough, there will also be more or less diapedesis of red blood-corpuscles, and hence the hæmorrhagic pleurisy. If we find leukocytes in the lymphatic exudation, does that not best explain the formation of pus? It has been stated that the serous exudate of acute pleurisy does not differ materially from the plasma of the blood. (The reason for this appears plain if we consider the contiguity of the arterial capillaries with the lymphatics.) If drawn off it will coagulate, often spontaneously; if beaten or whipped, it will show a deposit of fibrin; and the constituents are the same as the plasma of the blood, except in relative proportion.

It often happens, when one is practising puncture to draw off the effusion of acute pleurisy, that the needle, if small, becomes clogged with fibrinous flocculi. From this fact, it is argued by M. Lancereaux, that these flocculi, and more especially the finer ones, become obstructed in the openings of the lymphatics and produce a mechanical hindrance to the exit of the effused fluid by the thrombus thus formed. When this has occurred to such an extent that it impedes the absorption of liquid beyond the natural time required for the evolution of the disease, he avers, "one must wait for the disintegration and absorption of the clot before any diminishing of the quantity of the fluid by nature takes place."

Secondary pleurisies, or those acute attacks from some pre-existing disease which often occur, doubtless present in many cases the pathologic lesions in the pleura upon which such disease depends; but I can see no reason for the opinion of some authors, that every pleurisy is dependent on lesions of pre-existing maladies, and especially tuberculosis.

Etiology.—In considering the causation of acute pleurisy, one must of necessity admit that there are two general classifications from an etiologic point of view:

a. Those that are not due to any pre-existing disease.

b. Those that are so due.

At the present day there are many and diverse opinions held by eminent medical men as to the factors that produce primary idiopathic pleurisy. There are, indeed, those who would go so far as

to say that all pleurisies are tuberculous in origin, and, hence, secondary.

Undoubtedly, many pleurisies deemed primary are in reality dependent on some complicating or preëxisting disease; but to say that a person must be weakened by constitutional maladies in order to become privileged to have pleurisy, seems as if we were carrying the causative factors beyond the point warranted by our pathologic research. I doubt not, if the chests of many persons who to-day are in good health and have never been cognizant of pleurisy were opened, we would find various traces of old adhesions and other ancient lesions of former pleurisies.

It must, therefore, be admitted, even at the present day of biologic research, that the etiology of acute pleurisy is often obscure: the microbe will not account for all the cases; neither will any other one causative factor. While it is difficult to state with certainty that pleurisy originates in perfectly healthy persons, because latent pathologic lesions cannot be appreciated, yet we know that it does occur in persons, who to all appearances, to themselves and others, are in good health. M. Sée maintains that the etiology of acute pleurisy is always microbic; in fact, that it is a bacterial disease; cold is simply a stimulus to the activity and development of the microörganisms.

M. Jaccoud is of the opinion that in the human body many kinds and many thousands of microorganisms live in peace and harmony together so long as the functions are normal, but let a disturbing element arise, such as taking cold, and their physiologic relations become altered, so that they soon are hostile to each other and cause disease. Netter claims that all forms of pleurisy are of microbic origin, but that the microbes producing them are of many different kinds.

M. Bechamy says: "Microbes do not have so much importance in acute pleurisy as some would have them." He is certain that pleurisy may exist independently of tuberculosis, from the fact that at the age of thirty he was seized with acute pleurisy, and after the usual bleeding, blistering, and purging, he now, at the age of seventy-six, is still alive, and has not developed tuberculosis. In support of this, both he and Dieulafoy state that, in many cases, persons live ten, fifteen, and more years after the operation of thoracentesis, and do not develop tuberculosis.

That exposure to cold has a tendency to excite inflammations, and with them acute pleurisy, is no doubt true. Whether its influence is exerted through the nerve-centers, so as to directly cause pleurisy, or whether it acts simply as a stimulus to organized germs through whose activity the disease originates, is still a much-mooted question. M. Tresbot does not doubt that acute pleurisy in horses is the direct result of an exposure to cold, especially when, after a long, hard drive the animal is allowed to stand unprotected and exposed to a chilling wind. He says that "ordinarily there is nothing in common between sero-fibrinous pleurisy in the horse and tuberculosis"; and also that "it is impossible to class sero-fibrinous pleurisy in the horse with an erruptive fever, or, indeed, with any periodic disease." On the other hand, M. Lancereaux asserts: "Pleurisy should be rightly classed among the infectious maladies, and exposure to cold is nothing but an occasional exciting cause, while the action of the infecting agent still escapes us."

If we should accept the pathologic views of M. Guerin, we might easily explain the causation of the congestion of the blood-vessels by peripheral irritation, and reflex action of the vasomotor nerves from exposure to cold. This, however, does not explain why such excitement should be directed particularly to the pleura.

In a paper on "The Cause of Syncope in Pleurisy," M. La Borde has illustrated by experiment on animals that it is possible to produce a sero-fibrinous pleurisy in a few hours by the action of cantharidin injected into the blood. This leads me to ask the question, Is the causation of acute primary pleurisy ever, in a measure, governed by the ingestion of certain articles of food, taken just previously to an exposure to cold, *i. e.*, is the combination of the two forces sufficient to direct the action of inflamation toward the pleura?

The cause of acute purulent pleurisy is probably of microbic nature. Purulent pleurisies either begin as such, or are secondary to other diseases. Age and debility have much to do with the formation of pus, the young and aged being more susceptible to empyema. It is doubtful if simple sero-fibrinous pleurisies are ever transformed into the purulent form without the aid of outside interference.

Secondary pleurisies occur from a variety of causes, mostly from diseases microbic in origin.

While it may be rare to have pneumonia occur without some localized extension of inflammation to the pleura, it is doubtful if a general acute pleurisy, secondary to pneumonia, can be found with numerous pneumonia-cocci. In the same manner, the causation of pleurisy during an attack of typhoid fever, influenza, malarial fever, rheumatism, and kindred diseases, is undoubtedly due to the same influences that govern the coëxisting disease.

It is well known that pleurisy of an acute type may be secondary to tuberculosis; but there is considerable difference of opinion as to the proportion of cases arising from this source. M. Sée claims that 68 per cent. of all pleurisies are due to this cause. This seems high, or else persons radically recover from tuberculous pleurisy more often than from any other form of tuberculosis. Dr. G. G. Sears reports four hundred and fifty cases of

pleurisy, of which 39 per cent. developed tuberculous disease. Others state that not more than 20 per cent. of pleurisies are tuberculous in origin. The differences, probably, lie in the particular train of cases various observers have met.

There is, however, no doubt that a certain number of cases of tuberculosis have their initinal symptom manifested by an attack of acute pleurisy.

SYMPTOMATOLOGY.—M. Lancereaux and other French observers believe acute pleurisy to be a well-defined cyclic malady, because its lesions are always found unchangeable, and its evolution is as constant as that of pneumonia or of typhoid fever. It presents regular pathologic changes after each seventh day, and this fact allows a classification of the disease into three periods of evolution.

First: Seven days represent the time occupied for the increase of the pathologic lesions, at the end of which the effusion reaches the limit of its advance.

Second: The next seven days represent the time during which the pathologie lesions appear to remain stationary, and the effusion has not undergone any preceptible alteration in amount.

Third: From the fifteenth to the twenty-first day of the disease there takes place an absorption of the inflammatory products, including the effusion. These divisions, of course, cannot be made absolute, because individual cases differ among themselves; thus we all know that fluid-accumulations often completely fill the pleural cavity before the expiration of the first seven days, and when withdrawn reaccumulate; however, we may confidently expect at the end of the first period that in uncomplicated cases there will be no further effusion of fluid.

Each of these three stages has its respective symptoms, but they pass from one to the other without any pronounced expression of change.

In a majority of cases the first period of acute pleurisy is ushered in with a chill, announcing a greater or less rise of temperature; and accompanied with more or less pain in the affected side.

The pyrexia will generally determine the intensity of the inflammation; it seldom rises above 102° or 103°, unless the pleurisy is secondary to some other disease. Pain, which appears with the pyrexia, may begin as soreness in a circumscribed spot and progressively increase to an unbearable intensity; or it may at first be sharp and lancinating, at or below the nipple, from which point it becomes more or less diffused. The pain is increased on motion, and, for this reason, respiratory efforts are, so far as possible, restrained. A dry, hacking cough is generally present, but the pain it occasions calls for efforts at its suppression. After a few hours, when the effusion appears, the pain is moderated and gradually disappears, or its intensity is greatly diminished. The pulse is increased in frequency and generally firm, the rate varying between 100 and 120. As the disease progresses, dyspnea is often developed, and this may be due to several different causes. It may indicate abundant effusion, and be due to compression of the lung or displacement of the heart; it is most commonly a result of one of these. Again, it may be due to congestion of the lungs independently of compression. Edema of the lungs, capillary bronchitis, and rheumatic difficulties sometimes occasion dypsnea when very little fluid can be found in the cavity. Encysted pleurisies occasionally cause pain and dypsnea. Irregularity of the pulse and cyanosis are grave symptoms, largely dependent on displacement or weakness of the heart. Syncope may also be occasioned in various ways; the reflex action of pain is one of its chief causes, as we may often produce it artificially by electrifying some peripheral sensory nerve. Again, when the fluid fills the pleural cavity and is suddenly drawn entirely away, the reaction that follows may cause syncope.

PHYSICAL SIGNS.—These vary with the quantity rather than quality of the effusion. In the early stages of the disease they often establish the diagnosis, and in the latter stages they determine the amount of liquid in the cavity of the chest. On inspection there is in the early stages the restricted motion of the affected side, dependent at first on the amount of pain, and later on the accumulated fluid. The comparison with the sound side is very striking. In left-sided effusions there may be displacement of the heart-beat to the right of the normal, and when large effusions occur on the right side, displacement is sometimes to the left. On palpation, vocal fremitus is lost over a collection of fluid, and if the effusion is large on the right side, the liver may be felt to be displaced. On percussion, we may early find dulness over the lower posterior portions of the chest; this dulness gives place to flatness as the effusion appears and advances; above the fluid the percussion-note remains dull, while at its level, and below, it becomes flat. It will be remembered that the level of the fluid is not a hydrostatic one; but from the elasticity of the lung the level assumes a shape resembling the letter S. Over the sound lung the vesicular resonance is more or less exaggerated. Dulness is likely to remain for a considerable time after the effusion has been absorbed. On auscultation, the respiratory murmur is enfeebled or absent, for the same reasons and in the same progressive manner as the loss of resonance on percussion occurs. Friction-sounds are sometimes heard at the beginning of the disease, but are more common at the close of the third stage. Some observers have indicated that the transmission of the whispered voice, or its absence, has direct relation to the diagnosis of serous or purulent effusions, but this is doubted by others.

No lengthy discussion of the physical signs of

acute pleurisy is necessary here; these do not differ from their description as set forth in our modern text-books.

Treatment.—In considering the treatment of acute pleurisy we must recall the classification of its etiology, viz.:

First: Those cases that are dependent on some other disease for their cause, whether influenced by microbes or not, and hence are secondary pleurisies.

Second: Those cases that are of spontaneous origin, considered idiopathic, and hence are called primary pleurisies.

Physicians may likewise be divided into classes: those who consider pleurisy to be always a microbic disease, and those who do not.

In a recent discussion on this subject the eminent French author, Hardy, said that acute pleurisy of sero-fibrinous nature was no better treated to-day than it was fifty years ago, and, except in purulent forms, no better results were obtained now than then, the death-rate at present being 10 per cent., the same as in the days of our forefathers. This statement may be astonishing to some of us who have been taught to look upon acute pleurisy as a not very fatal disease; however, some statistics would seem to bear out this opinion. Perhaps it would, therefore, be well to consider the modern methods of treatment and then compare them with those practised in the earlier part of the century.

To undertake the consideration of all the medications for pleurisy that have been launched upon us during the past few years would take more time and space than would be profitable for me to employ; suffice it to say that a majority of them have passed into disuse.

The modern medical treatment of acute pleurisy is by the following class of agents: First, by antiseptics, to combat microörganisms; second, by antipyretics, to combat fever; third, by evacuants, to eliminate the fluid.

Dr. Charles Talamon has recently called attention to the action of sodium salicylate in pleuritic effusions, claiming for it the power of promoting rapid absorption of the fluid. He thinks it has a direct action on the inflamed pleura, because by the experiments of Rosenbach it is proved that the salicylates may be found in the serous cavities of the body after their ingestion by the mouth in doses of from ten to twenty grains. Whether the beneficial action on the fluid is due to the antiseptic nature of the agent, he does not state, and whether it is due to this or its diuretic action is still an open question. That sodium salicylate may be of use when the pleurisy is secondary to rheumatism there can be no doubt, but in the primary form to depend on its success as a germicide would be hazardous. However, the salicylates may be employed as antipyretics as advantageously as other remedies; they certainly combine the indications for an antiseptic, antipyretic, and diuretic.

The practice of injecting a solution of salicylic acid or other antiseptic into the pleural cavity to combat microbe in the effusion has been suggested by some, but the treatment seems harsh and uncalled for, unless employed in connection with surgical methods for the treatment of empyemas.

Antipyretics in acute pleurisy are only indicated when the fever rises to 101° or over, and as the fever seldom attains that height for any length of time their use is greatly modified.

Quinine may be advantageously employed in pleurisy depending on malarial poisoning, and during convalescence, as a tonic. Antipyrin, or the other coal-tar derivatives, may be useful in cases accompanied by hyperpyrexia, but none of these measures are calculated to reach the cause of the disease, or to modify its pathology.

The evacuants are administered in acute pleurisy with a view to reduce the amount of effusion after its accumulation.

Under this head, the diuretics play the most important part. Digitalis may support a weak heart, but its action in reducing a pleuritic effusion is small. Milk is often used as a diuretic, but its influence over fluid in this disease is doubtful; while as a food it ought not to be neglected.

The action of purgatives, drastic or saline, and of sudorifics, with a view to reduce the quantity of liquid in the chest, is of no value; moreover, they are often dangerous.

The pleuritic effusion is not really a question of hydropsy; the liquid of general ascites furnishes a chemical analysis quite different from the effusion of pleurisy; the latter is not simply a serum from the blood, but blood-plasma.

In a recent paper on "The Treatment of Pleuritic Effusion," M. Sée draws the following conclusions: "Antiseptics, diuretics, sudorifics, and purgatives, drastic or saline, have no kind of action on the effusion. Milk, which is a powerful diuretic, has no value here, except as a food. No one of these microbic diseases derives the least benefit from venesection. All aggravate the onset of the disease. Expectation is the only rational method of treatment, for sero-fibrinous pleurisy regularly passes through its different phases in two or three weeks, and up to that point all medication is useless." In a recent paper on this subject, M. Lancereaux says: "There is no more use in trying to ward off pleuritic fever than to ward off pneumonia or typhoid fever; however, it is necessary, relying on our pathologic knowledge of the lesions of acute pleurisy, to draw attention to the coagulation in the lymphatic system, and strive to remove it; while we may not succeed, it is the best practice to try."

In discussing the subject, M. Guerin says: "This idea of pleurisy being a simple lymphan-

gitis also gives a clear explanation of the sudden re-absorption of fluid in some cases that for a long time have proved refractory to all treatment. It indicates that the coagulum in the absorbent vessels has become re-absorbed and makes it possible for the fluid to be taken up; we should therefore attempt the prompt moderation of the lymphangitis."

Of late, in the excitement of bacteriologic investigations, the profession had been content to disregard the teachings of our ancestors, and the good old methods in vogue at their time. We have regarded these diseases as cyclic in nature; maladies that must run their regular course in spite of all abortive treatment; sicknesses that are caused by micro-organisms over which we have no control; hence, we must fold our hands and content ourselves with relieving of pain, until such time as the particular germ has loosened his besieging grasp.

This is the "expectant treatment of to-day, and this is the treatment eminent authorities hold out to us for acute pleurisy." "The expectant treatment," says Peter, "is the do-nothing treatment, and this is what many bacteriologists practise. No physician has a right to practise inactivity in these cases when so much can be done toward curing pleurisy if energetic treatment is begun early."

To allow a patient to become weakened by pain and suffering, because pleurisy is a cyclic disease and should terminate spontaneously in two or three weeks, or, because the pathologic conditions are such that, by deferring active measures, one may with impunity puncture the chest and draw off a liter of fluid, is treating the wrong end of the disease. A physician should strive to diminish the intensity of a malady, if, indeed, he cannot shorten its duration.

What are the indications for treatment in acute pleurisy?

Briefly, they are as follows: 1. The relief of pain. 2. The reduction of fever. 3. The arrest of effusion. All of which depends on treating the cause.

It was of these indications our ancestors had a rare knowledge when they applied bleeding and localized vesications; when practised, these at once relieved the pain, reduced the fever, and there was little or no serous effusion found in their cases; moreover, if, in this they were not entirely successful, the harshness of the sickness was notably diminished. By these methods the pathologic lesions were affected, and the cause of the pain and fever reached. In 1819, Lænnec said: "If a plethoric subject had pleurisy, he required bleeding."

While it is undoubtedly true that the pendulum of exsanguination in inflammatory diseases oscillated too far in the early part of this century, and to the detriment of many, is it not also true that at the present time it has swung too far the other way, and "expectant treatment" is employed to the detriment of many? I believe the antiphlogistic treatment is indicated in just as many cases of acute pleurisy now, as in the days of our forefathers. That the treatment may be carried too far I do not deny; the judicious use of it at the right time is the essential point. Pain is almost wholly relieved by the revulsive methods in a short time; this probably being accomplished by the removal of the congestion and its baneful influences, if revulsion is practised early. At the present time, and in some form, opium is employed for the relief of pain; but does it lessen the congestion causing the pain, or simply dull the sensibility to the pain which is the result of the congestion? Revulsion lowers the fever, for which we now employ antipyretics, but will the antipyretic drugs alter the pathologic condition within the pleural cavity?

Farther on in this disease we often employ puncture of the chest to draw off the fluid, but have we not allowed the malady to reach that point by an early neglect to treat the case efficiently, and thus converted what should have been a medical case into a surgical one? This certainly is to be deplored. Peter goes so far as to say, that "By revulsive methods, early employed, one is able to prevent the serous effusion in many cases; to arrest it, if the secretion has begun to form, and finally to absorb it, if a small amount has collected." Andral and Bouilland cite thirty cases in one year, treated antiphlogistically, with one death—percentage of mortality of three. Of seventy cases reported in 1891, treated after the modern methods, there were seven deaths—a death-rate of ten per cent from acute pleurisy.

How should revulsion be employed in these cases?

Bouilland used to bleed twice and afterward apply wet cups and a large blister. This seems too energetic; yet I have recently been told by an elderly physician of my neighborhood that he once bled a woman with puerperal peritonitis until she fainted, and she had no more symptoms of the disease, and was soon about. One might employ wet cups and vesication more mildly, with equal success. "What is remarkable in these cases," says Peter, "is the toleration of the system to these large bleedings, the rapidity with which the pain ceases and the local symptoms of pleurisy diminish." On the other hand, "Bleeding," says Sée, "after doing so much harm, is springing up again insidiously in many diseases; it has no theoretic basis, and is little better than empiricism.

Can we learn anything from Nature as to the treatment of acute pleurisy?

There is pain from irritation of the intercostal

nerves, due to the evolution of pathologic processes. Nature, in consequence of this inflammatory action, evolves a fluid which acts as a sedative to the injured nerves, and, little by little, the pain subsides. The fluid comes from the blood. This fluid is found to be exactly like the plasma of the blood, except in proportion; is it, therefore, not natural to assume that Nature bleeds her patient in the early stage of pleurisy? To be sure it may be the result of a congested condition, and, hence, one of pathology rather than one of treatment; nevertheless it depletes the vascular system, which, if we anticipate Nature by doing ourselves, we thereby arrest the pathologic condition and relieve Nature.

Regarding the application of blisters, there is a diversity of opinion, although it is generally held that they are useful adjuvants to scarification.

Metschnikoff advances the idea that cantharides possesses an anti-bacterial tendency, and produces leukocytes which act as phagocytes, and proceed to destroy or change the character of the microbe. He would, therefore, inject a solution of cantharidin subcutaneously. Tresbot has no doubt that a cantharides blister is of great value in the treatment of the pleurisy of horses. La Borde, while advocating the use of cantharides blisters, calls attention to the fact that this agent is a poison, and capable of producing inflammation of the lung, bladder, or other viscus, and cautions against its use. There has been some belief that cantharides predisposes to the transforming of serous effusions into purulent ones, and especially in tuberculous patients. Potain denies this, or the possibility of its happening, and maintains that a purulent effusion always starts as such. Counter-irritation may also be effected by the tincture of iodine painted on the surface of the affected side. Little in addition can be said of the treatment of purulent pleurisies prior to surgical interference, which is almost always required.

There is no positive way to differentiate them from the serous varieties, except by explorative puncture, but the indiscriminate use of the exploring-needle, ten or a dozen times, as recommended by some, in search for pus, is to be condemned, even if no harm should chance to arise.

It is in childhood and old age that purulent pleurisy is most likely to occur. Why this is so, unless from a weaker state than is present in adults, is not easily determined; but in the treatment of this affection in children, this fact is to be borne in mind.

Thoracentesis.—This operation, though always to be deplored, is often urgent and often useful. A discussion of its history, which may be found at length in *Pepper's System of Medicine*, is unnecessary in this place. Of late there has been much criticism for and against the operation by eminent authorities. That aspiration, as first practiced by Bowditch, and later elaborated by Dieulafoy and others, is a simple, harmless operation, there can be no doubt; results will bear this statement out. All that is essential to the safety of the operation is thoroughly aseptic instruments, especially the aspirating-needle, and also some little skill in manipulation.

Thoracentesis, as practised by the majority of general practitioners, with any kind of trocar, which may have been used by them to open some abscess-cavity, or even with an aspirating aparatus that has not been perfectly cleansed, is a very dangerous operation.

It has been held by some that after the operation has been performed two or three times it produces a transformation of a sero-fibrinous effusion into a purulent one. This may be so; I do not deny that it often happens, but it is the operator and his unclean instruments that are at fault, rather than the effect of a puncture of the pleura; this is the reason we see one operator successful where another meets with failures.

As a rule, aspiration should not be performed in simple sero-fibrinous pleurisy until after the third week of the disease; and then only as the fluid tends to remain stationary and unabsorbed, unless there is urgent need of interference to save life before that time. If the cause of the non-absorption of the fluid is (according to the views of Lancereaux) a stoppage of the lymphatics of the pleura by the formation of fibrinous thrombi in their orifices, and if we must wait until a disintegration of the clot takes place before the fluid will be absorbed, then no amount of aspiration will hasten the process of natural absorption until that time has expired. Moreover, the drawing off of the fluid will, in many cases, only tend to its re-accumulation up to the point it previously reached, because, according to his theory, if the fluid remains stationary to a given level or height within the cavity of the chest, there must be stoppage of all the lymph-spaces below that level; hence, no absorption is possible. If, then, we remove a part or the whole of the fluid, we do not necessarily remove the stoppage, and the re-accumulating fluid will, in time, reach its former height.

On the other hand, if, before practising thoracentesis, we wait until the disease has reached that period when we may expect these clots to be disintegrating and being taken up by the system, we then may be of some service to Nature in hastening absorption. This period is at about the end of the twenty-first day of the disease. However, there are times previous to this period, when life is threatened by the accumulation of fluid in the pleural cavity to such an extent that it compresses vital organs. When this occurs, it becomes necessary to draw off a certain amount of fluid by

aspiration, in order to relieve distressing symptoms.

By what symptoms can we know that this danger is imminent?

When the fluid-accumulation has progressed to the extreme degree, there is dyspnea, from compression of the lung; more or less cyanosis, if the heart be displaced; flatness on percussion over most of the affected side, and sometimes accompanied with bulging of the intercostal spaces. However, if we wait for these symptoms to appear, there are strong dangers of being too late to render the assistance necessary.

There is in these cases no infallible sign by which we may discover the best time to operate.

Dyspnea, as we know, may be due to other causes than compression of the lung or displacement of the heart, and consequent twisting of the large arteries, by fluid. Cyanosis and syncope are signs that may arise from hearts weakened by excess of pain, with little mechanical interference from an effusion into the pleura.

The most reliable test is by percussion; as by it we may arrive at an approximate estimate of the quantity of fluid contained in the pleural cavity. When the line of flatness has reached the second rib on either side of the chest, the left or right, Dieulafoy estimates the quantity to be about 2000 grams, and states it is time to operate. If, with this percussion-sign, there is dyspnea and some cyanosis, it is time to hasten the operation; as, while it is not right to puncture the chest too early, it is also hazardous to postpone the operation too long.

If, as often happens after early operative interference, the fluid should re-accumulate more or less rapidly, and the symptoms of distress reappear, a second operation would be imperative, and probably later on others would necessarily follow.

Only so much of the fluid should be removed in these cases as will render the patient more comfortable from the distressing symptoms, or remove any danger of immediate collapse. Sudden death has followed the removal of the entire amount of effusion at once, death being due to the congestion occurring from the sudden return of the compressed and distorted viscus to its normal position. A symptom of this danger is said to be the albuminous expectoration observed in these cases. The fluid should, therefore, be withdrawn gradually through a fine needle, and not more than a third, or perhaps a half, of the total quantity of fluid in the chest-cavity be removed at once. It is better to perform the operation several times in this manner than to have a fatal issue from the evacuation of too great a quantity of liquid at once.

In purulent pleurisies no time should be lost in evacuating the pus, observing the same precautions necessary, if by aspiration, as in serous effusions. In children with purulent pleurisies, repeated aspirations are advisable before resorting to more severe surgical methods; but in adults, if a re-accumulation of pus occurs after one aspiration, it is usually better practice to treat the empyema as one would an abscess-cavity and establish a system of free drainage. As these several methods of drainage come strictly under the head of chronic pleurisy, I shall not occupy more time here with a discussion of them.

Finally, I wish here to suggest a method of treatment for pleuritic effusions that I must frankly state is at present a simple theory, because I have not had the time or opportunity to clinically test its value. It is the employment of electrolysis to cause absorption of pleuritic effusions, based on the same theories as when it is used in serous effusions elsewhere. In a word, the operation might be called "electrocentesis." I have not found that any literature on the subject has ever been published--or ever ought to be published; however, electrolysis, as we know, has been employed quite commonly in serous effusions of other localities, such as cysts, hydrocele, tumors, etc., with great benefit in many instances. Why should it not be used with benefit in the serous effusion of pleurisy?

Electricity is used to hasten the absorption of fluid in cysts; first, by its power to chemically transform the watery into gaseous elements; second, by its direct stimulating influence on the lining membrane of the sac.

Is there any reason to expect any different results from the use of electricity, in a similar manner, in pleuritic effusions?

The technique of the operation recommended includes the use of the electro-puncture needle, thrust into the effusion; a clay electrode attached to the negative pole of a galvanic battery, and placed on the outside of the chest-wall. A current of a strength of from 30 to 50 milliampères, and perhaps more, could be safely passed through the fluid in this manner. Care should be taken that the needle be not thrust farther than just into the fluid, so that we get only electric action on the effusion an the costal pleura, otherwise we might electrify some vital organ in a manner not pleasing. From this application of electrolysis we might reasonably expect more or less coagulation of fibrinous matter and absorption of the fluid portions of the effusion. This we might expect to be in proportion to the strength of the current, and the length of time occupied in allowing the current to pass through.

The class of cases this method of treatment would probably benefit would be those in which thoracentesis for any reason could not safely be performed those in which there has been repeated re-accumulation of fluid after aspiration, especially

those of chronic tendencies; and, finally, those of secondary nature, particularly the tuberculous.

All antiseptic precautions usually necessary in any such operation should, of course, be observed. —Frank S. Parsons, M.D., in *Med. News.*

A CASE OF SKIN SHEDDING.

Some months ago I received a letter from Dr. W. T. Bolton, of Biloxi, Miss., asking my opinion of a remarkable case of skin shedding that had come under his observation. On reading the notes that were enclosed I was surprised to find that here was a description of a disease to which I had recently given considerable study, having myself made careful notes upon two cases which are reported in the October, 1891, number of the *International Clinics*, under the title of erythema exfoliativum recurrens.

As the bibliography of this disease, and my reasons for classing it as an erythema, are there given in full, I shall not burden this paper with an unnecessary repetition.

With Dr. Bolton's permission, I here give a report of his case from notes furnished me at different times at my request.

Maggie P., aged twenty years, house-maid by occupation, but also goes to market and helps about laundry. While washing and ironing on May 6th, she perspired a great deal, and, to cool off, went out into the open air when the wind was blowing hard, thus suddenly checking her perspiration. The result was an attack of nausea and vomiting accompanied by pains in the back and limbs. A purgative was given next day, and the patient put to bed. On May 9th, Dr. Bolton was called in at 7 a.m. He noted the following symptoms: Temp. 99.6, pulse 104, nausea and vomiting. The ejected matter is yellowish (bile colored) and viscid. Patient complained of dizziness and a severe headache, the pain being in the upper and back part of the head. Skin of face swollen. Face, neck, and upper part of chest covered with an eruption (erythematous), suggesting the idea that they had been exposed to intense heat. Tongue coated.

On being questioned, patient declared that she had had a similar eruption in February, 1890, and in August, 1891. The first time she had the eruption she thought she had scarlet fever. Both previous attacks had been accompanied by slight fever. None of her relatives have suffered from a similar disease. Both parents, and two brothers, died of consumption. She has had measles, but never had diphtheria, nor scarlet fever, unless the attack of February, 1890, was this disease.

At 7 p.m., of May 9th, it was noted that the temperature was 99, and pulse 80. Patient had vomited at 6 30. Tongue coated in centre, with the edges and tip clean but red. Headache not quite so severe.

May 10, 7 a.m. Temp. 99.8, pulse 98. Slight nausea, but no vomiting since last evening. Did not rest well during night. Took wine several times and broth once. Rash has extended over whole surface of body.

5.30 p.m. Temp. 99.2, pulse 108. General appearance better. Expression of face not so anxious. Skin very red. Wine and broth several times during day.

May 11th, 8 a.m. Temp. 98.8, pulse 90. Rested well, and slept greater portion of the night. Took wine and broth several times. Tongue clean, but not red; its coating, together with a thin enveloping membrane, is coming off. Epidermis of upper eyelids beginning to desquamate.

1.30 p.m. Condition about same as at 8 a.m.

May 12, 8 a.m, Temp. 98.8, pulse 88. Had a severe headache during last evening and early part of night. Return of nausea, but no vomiting.

The mucous membrane of roof of mouth exfoliated in a solid mass. On May 12, and two following days, the epidermis of the entire body was thrown off. That of the trunk and limbs coming off in pieces one to two inches wide, and three to five inches long.

The epidermis of the hands and feet, particularly of the former, came off almost without a break, resembling gloves and moccasins. The nails were loose but did not come off at this time.

The malady gave no further trouble, and the patient was soon up and out, and spending a week in New Orleans. However, on May 31st, just twenty-five days after the beginning of the last attack, she was again suddenly taken sick with the same symptoms, only in a much milder way, the rash being less intense and extensive. The face, mouth, and upper extremities peeled, as before, and on June 4th, the fifth day of the attack, a piece of epidermis a foot long was removed from the arm and elbow.

During the second attack there was slight sore throat, and some pain over the part of the right lung on coughing. On June 4th, the urine was examined and found free from albumen. Specific gravity, 1008.

The nails of fingers and toes fell off after the first attack, but the time is not stated. They also fell off after the two previous attacks. There was very little therapeutic interference in the course of the disease. Oxalate of cerium was given for the vomiting at the outset, and later on calomel and ipecac, combined in powders, were administered.

One dose of quinine and phenacetine, two grains and a half each, was given in the course of the disease, and several times the skin was anointed with vaseline and quinine.

I would respectfully call attention to what I

deem the peculiar characteristics of this case and those already reported by me.

1. *Recurrence.*—In the case here reported there were four attacks in the space of two years and four months, coming on at irregular intervals.

One of my patients was averaging two attacks a year, and at the time his case was reported (1891), had had the exfoliative erythema twenty-three times.

The cause of the recurrence, like the cause of the disease, is unknown, and the accidents precipating the attacks differ with each case.

Dr. Bolton's statement that his patient had had a check of perspiration, suggests that this may have been the immediate or exciting cause of her eruption. If this be so, then the skin and nervous (vaso-motor) system must have been peculiarly sensitive and liable to disarrangement by impressions from external temperature.

That a marked general erythema (hyperæmia) may be produced by cold I can attest from a case that came under my observation last summer. One of the students of the University of the South, 18 years of age, complained to me of a sense of malaise and nausea on going out into the weather on damp and chilly days, saying that large surfaces of his skin would turn red and burn. Furthermore, he said that when he bathed in cold water the part of the skin touched by the water would turn red.

As he was otherwise in good health he was greatly annoyed by this physical peculiarity. Anxious that my eyes should confirm his statements, he requested me to go with him to the general bathing pool of the students and watch the result. He then removed his clothes and showed a good muscular development and quite a white skin. After diving into the water which had a temperature of 56° Fah., and after remaining there four minutes he came out to be inspected. His condition was now quite changed, as he had declared it would be. The face was flushed, but the trunk, arms and thighs had turned a bright red—a smooth, diffused erythema, feeling quite warm under the fingers. He said that it itched him and burned slightly. I then made him come out and put on his clothes, taking his temperature, which had fallen a degree and a half during the bath. The redness remained for an hour and then passed off.

Here was a case of almost universal erythema, which had been directly produced hy a known cause—cold. I have mentioned this case which is unique in my experience, as an illustration of the fact that erythematous disease is not necessarily dependent upon a germ or poison within the system, and that recurrences may be produced by the removal of certain known causes.

2. *Low pulse rate.* I have already called attention (loc. cit.) to the lack of resemblance of the pulse in recurrent exfoliative erythema to that of scarlet fever, a disease which French writers have supposed it to resemble, for the rapid pulse of scarlet fever is characteristic, and in marked contrast with the disease in question. The case here reported had a faster pulse than any that I have noted, and this averaged only 94⅗ beats per minute.

3. *Absence of marked febrile symptoms.* The disease is not characterized by marked febrile symptoms. The history of the cases goes to show that the first attack is the one most likely to be accompanied by high fever. Subsequent attacks may or may not be accompanied by elevated temperature.

The highest temperature attained by Dr. Bolton's case was 99.8.

4. *Extensive desquamation.* I know of no disease where the desquamation is as great and as complete.

Even *dermatitis exfoliativa* (of which Brocq believes this to be a variety), though accompanied by general scaling of the epidermis, does not shed in so short a space of time, scales as thick and as large as this disease. It differs from *dermatitis exfoliativa* by being of shorter duration, by not appearing in patches at the beginning, and by not having evening exacerbations.

6. *Resemblance to Scarlatina.* For a short time in the history of a case the resemblance to scarlatina is so marked that it seems to me desirable that this article should be concluded by contrasting the main points of difference in parallel columns.

Scarlatina.	Erythema Exfoliativa Recurrence.
1. Occurs once.	1. Recurs frequently.
2. Contagious.	2. Non-contagious.
3. Marked pyrexia.	3. Pyrexia very slight.
4. Fauces much swollen.	4. Fauces red.
5. Pulse very rapid.	5. Pulse not rapid.
6. Eruption lasts five days.	6. Eruption lasts 3 days.
7. Desquamation sometimes considerable.	7. Desquamation always excessive.
8. Albuminuria common.	8. No albuminuria.

—Henry Wm. Blanc, B. S., M.D., in *Jour. of Cutaneous and Genito-Urinary Diseases.*

THE TREATMENT OF DIABETES MELLITUS BY MEANS OF PANCREATIC JUICE.

In the *British Medical Journal* for January 7th Dr. Mansell-Jones suggests that as the juice of the thyroid gland appears to be almost a specific in myxœdema, pancreatic juice administered before or after meals should be given a fair trial in diabetes, as this disease, he adds, in most cases, appears to be due to disease or disordered function of the pancreas.

Neither pathology nor physiology, however, lend much encouragement to the hope that diabetes mellitus will prove tractable in such a simple way. In the first place, the pathogenesis of this disease is much more complex than that of myxœdema, and disease of the pancreas accounts for probably only a fraction of the cases of this malady. In the second place, even if it were a fact that in most cases diabetes was due to disease or disorder of the pancreas, the analogy of this doubly active gland, both excreting and secreting, with the ductless thyroid gland is not a very close one. There is some reason, however, on theoretical grounds, for the belief that pancreatic juice might have some beneficial effect even in non-pancreatic diabetes.

The recent researches into the pathology of the pancreatic form of diabetes mellitus, of which a most interesting account was given by Dr. Vaughan Harley in the *British Medical Journal* for August 27th, 1892, make it very probable that, in addition to the well-known tryptic, diastatic, fat-splitting, and milk-curdling ferments, a glycolytic ferment is also produced by the pancreas. Assuming the existence in the normal pancreas of this latter ferment, I thought it possible that the administration of a pancreatic extract by the mouth might have some beneficial action in diabetes mellitus by assisting to destroy the sugar in the blood. Acting on this idea, therefore, I anticipated Dr. Mansell-Jones's suggestion, and for some time past have been treating in a tentative way two pronounced cases of diabetes mellitus under my care at the Royal Free Hospital, by the administration of liquor pancreaticus in half-ounce doses given three times a day immediately after food. It is the generally received opinion that, when given in this way, the liquor has no appreciable digestive power, so that we may put the latter effect on one side. No other medicine was given after this treatment was started, and in every respect the patients remained under the same conditions as before.

In both cases the patients have assured me they have experienced benefit from the treatment. I should not have attached so much importance to their statements had it not been that, without any suggestion on my part or collusion on the part of the patients, who attended on different days, there was a remarkable agreement in the accounts they gave of this beneficial effect. They both said they had lost to a great extent their feeling of lassitude and languor, and felt stronger in every way. Their thirst, moreover, had considerably lessened, and they had passed a smaller quantity of urine. These beneficial effects, moreover, have continued. The specific gravity of the urine and the relative amount of sugar have, on the other hand, not been affected.

In an in-patient under the care of my colleague, Dr. Samuel West, his house-physician, Dr. Rendel, informs me that since the administration of liquor pancreaticus, the amount of fluid imbibed during the twenty-four hours, which had previously averaged 12 pints, has fallen to 6 pints, with a similar decrease in the amount of urine passed,

In a disease like diabetes we must be thankful for even small mercies. For myself I would rather find an improvement in the general condition of the patient, increased strength, diminished thirst, and diminished quantity of urine as a result of treatment than a mere diminution of the amount of sugar in the urine without such improvement. I should have preferred, of course, to have found both results. It is evident that liquor pancreaticus is no specific, but the effects in these cases are encouraging enough to induce me to make further trial of it, and it is possible that in cases of true pancreatic diabetes the benefit might be greater. H. W. G. Mackenzie, M.D., F.R.C.P., in *Br. Med. Jour.*

CASES CONTRASTING GOUT AND RHEUMATOID ARTHRITIS.—A CLINICAL LECTURE.

Gentlemen,—I present to you to-day two patients whose diseases I wish to contrast, and thus more clearly bring out a differential diagnosis between them. Both are men about forty years of age, and from their family histories we can elicit no information that has any bearing on their present diseases. This man to my right says that he is by occupation a butler, and that he has been sick for the last twelve years. He had had no serious illness previous to that time. His habits were those of a man accustomed to the best of food, and he indulged to excess in fermented wines, mostly sherry and Madeira. Twelve years ago he had an attack of gout, beginning in the first joint of the great toe of the right foot. This attack lasted six days, and then entirely disappeared. Six months later he had a second attack extending to the same joint of the other foot. This second attack lasted five weeks and then disappeared, to be followed in nine months by a third attack, which involved the knee and finger joints, lasting in its severity one month, when it assumed a chronic phase which resulted in the deformed condition of fingers and toes which he now presents.

Gout is a blood disease, characterized by a deposit of urate of soda in the joints, and by constitutional symptoms, and arthritis. The first arthritic symptom is manifested in the metatarsophalangeal articulation of the great toe, usually of the right side; why this is so I cannot say. This is the history of this patient, and if his memory were sufficiently reliable we could get from in addition premonitory symptoms that preceded his attack of twelve years ago. There are many con-

ditions that indicate a gouty diathesis; these are biliousness, headache, arterio-sclerosis, indigestion, etc. A man who is a high liver and presents such symptoms as these, epecially on the morning after an acute debauch, is on the direct highway to gout. Every man with hard arteries, or these other symptoms, has not gout, but, I repeat, is on the gouty line. Such a man, can, by a timely correction of his habits, avoid an attack of gout; but a man in whom an attack has once developed, even though it be a slight one, has a gouty prospect that will require his utmost solicitude to counteract.

Gout once developed is extremely prone to recur. Upon examination we find that this patient has extremely hard arteries, a pulse of high tension and an hypertrophied left ventricle, but no murmur. These hard, whipcord arteries are due to a general condition of fibrosis; we should find such a condition if we examined his heart, liver and kidneys.

You noticed that in getting the patient's history, I dwelt considerably on his appetite. He said that he was a hearty eater and lived on rich food. This alone, without the use of wine, was cause enough to produce gout. Excessive eating of rich food is a frequent cause of gout in females who lead a luxurious life. They eat often and take large quantities of food, but are absolutely averse to exercise. Walking, with them, is one of the lost arts. Horseback-riding is good, but if you induce them to practice it you perform a miracle. They get gout which brings on a chain of other disorders, particularly at the menopause. It requires patience and tact to treat such cases.

This second patient is a man with diseased joints somewhat similar to that of the gouty patient. You will notice, however, that while the patient with gout complains most of pain in his fingers and toes, that this patient complains of most pain in his elbows. There is also an absence of the deformity in the phalanges here, which we see so plainly marked in the first patient. This man has chronic arthritis in his joints. He says that some years ago he noticed a tenderness, redness, and swelling of the right elbow, then of the left, and next of the ankles. This is a prominent feature of rheumatoid arthritis; it attacks a joint and damages it to such an extent that it often leaves it permanently crippled; this done it attacks another joint with the same result. It appears first in the large joints, while gout appears first in the small joints. This is an important diagnostic point, as we can often tell which disease it is by knowing the joint first affected. Chronic arthritis attacks small joints as well as the large, and may leave them in a more crippled state than will gout. While gout is a disease due to an excess of uric acid in the blood, arthritis is supposed to be an affection of the nervous system. Arthritis is not a rheumatic trouble, and anti-rheumatic drugs like salicylate of soda and iodide of potash have no effect on the disease. There is no drug that will cure it, but I have found that while it is progressing it may be delayed by the judicious use of cod liver oil and arsenic.

Now gout, unlike arthritis, will yield to medicinal remedies. Colchicum is almost a specific in gout. It may be given with or without alkalies; of its various preparations I have found the acetic extract to be the most efficient. I think that it is much better than the tincture or the wine of colchicum seed. If the acute pain of gout does not yield to it, combine a little morphia. After the patient has been relieved of his acute attack we should forcibly impress upon him the necessity of a reformation in his habits; unless he does this he is sure to have another attack in a few months. Gout demands scientific and practical treatment. It is a disease that makes a man prematurely old. The general fibrosis which it gives rise to will, in a short time, make an old man out of a young man.—Dr. A. L. Loomis, in *Med. Fortnightly.*

MEDICAL NOTES.

Prof. Senn recently stated that catgut is the only suture necessary to approximate and maintain any *Fracture of the Patella* until the occurrence of union.

As a *Substitute for Tincture of Iodine* the following is recommended *(Pharm. Era):* Dissolve 1 part of iodine in ether, add 20 parts ol. paraffine, and then allow the ether to evaporate.

Federow Charkon obtained a rapid and complete cure of the *Vomiting of Pregnancy* by the use of fluid extract of hydrastis canadensis, twenty drops four times a day *(Boston Med. and Surg. Journal.)* He believes that this drug reduces the blood pressure, diminishes the hyperæmia of the uterus, and calms the irritated vaso-motor centres of the digestive apparatus.

Dr. W. L. Symes (*Dublin Jour. of Med. Science* in *Med. Record*, Oct. 1, 1892), reports a case of *Heart Failure* in which there was no pulse, no sound over his heart, no respiration; his eyes were glassy and fixed; he could not swallow; and severe slapping of the epigastrium had no effect. Having exhausted all available stimulants, and recollecting that he had read somewhere of the value of a hot spoon applied to the epigastrium in states of asphyxia or syncope, Dr. Symes took a hot lump of coal from the fire with the tongs, and applying it over the xiphoid cartilage once or twice, was gratified to find it produce powerful contractions of the inspiratory muscles. This was continued for some time, applications being

made around the insertion of the diaphragm, until, by degrees, respiration returned, the patient's eyes opened, his heart beat, and the color came back to his face. He then took some brandy, and finally rallied.

As an *Antiseptic Mouth Wash*, Müller (*Med. Record*) recommends :—

R.—Acid. thymic., ... gr. iv.
Acid. benzoic., ... gr. xl.
Tinct. eucalypti, ... fʒss.
Spirit. vini rectificat., . . . fʒiij.
Essent. menth. piperit., . . gtt. x.—M.

Sig.—Drop enough into a glass of water to cause turbidity, and rinse the mouth morning and night.

An excellent cleansing and disinfecting *Solution for Free Use in the Nasal Cavities*, by means of the spray apparatus, douche, or syringe, is prepared as follows :—

R.—Acidi borici, ...
Sodii borat., ... āā ʒj.
Sodii chloridi., ... ʒss.
Listerine, ... fʒij.
Aquæ puræ, ... fʒvj.—M.

Prof. Hare says that an ordinary cup of coffee contains from twenty to thirty minims of the oil of caffeine.

Prof. Keen says that in some few cases the bacteria of *Erysipelas* injected in a cancerous tumor have at first produced erysipelas in the patient, but later on have effected a cure of the tumor.

Prof. Keen prefers not administering any stimulants in the earlier stages of *Erysipelas;* but thinks that they should be administered in the later stages, if the patient's condition calls for them.

Prof. Parvin says that antipyrine will be found very useful for the relief of pain in *Dysmenorrhœa;* and, moreover, it is free from the many objections that are raised against opium for the relief of pain.

Prof. Hare says that in *Persistent Attacks of Vomiting* best results will be obtained from rectal injections of the following :

R—Tinct. of opium. deod., . . ♏ xxx.
Bromide of potassium, . . grs. xxx-xl.
Starch water, ... q. s.—M.

Dr. Eschner says that in cases of *Disease of the Heart*, calling for a treatment by digitalis, the best results will be obtained by administering it for three or four weeks and then substituting some other heart tonic for the same length of time.

If the stockings are soaked in a saturated solution of boric acid and allowed to dry, and be then worn, Prof. Hare says that cases of *Sweating of the Feet* will often be cured, and in most cases if not cured, the odor arising from the sweating feet will often disappear.

Prof. Brinton gave his class the following prescription for a case of *Gonorrhœa:*

R—Plumbi acetat., ... gr. xxx.
Zince sulph., ... gr. xv.
Hydrastin sulph., . . . gr. xij.
Extract. ergotæ fluid, . . fʒ iv.
Tinct. oppi, ... fʒ iij.
Aquæ, ... fʒ vj.—M.

Sig.—Use as an injection. Shake well before using.

The following constitutional treatment is recommended by Prof. Keen in cases of *Septicæmia, Pyœmia*, and *Puerperal Fever:* Good, easily digested food, and alcoholic liquor in as large a quantity as the patient will be able to bear without becoming intoxicated.

Dr. Albert Brubaker says that *Spasmodic Croup* in children, coming on suddenly at night, is often due to impaired digestion brought on by eating some heavy food just before retiring. If the stomach in these cases be emptied by an emetic, it will be found that the croup will also disappear.

—*Col. and Clin. Record.*

CANNABIS INDICA.

As a non opiate anodyne this drug ranks next in importance to the coal-tar compounds. It is less used at present than it should be. It seems to have fallen into disuse through the fact that preparations are often found in the shops comparatively inert, and also through a fear of its toxic power. Indian hemp, however, is not a poison. Not a case of death from the drug is on record. It may in very large doses cause alarming symtoms but never death. Regarding the preparation of the drug used, one can by a careful trial with various specimens secure a reliable preparation and establish its dose.

There can be no doubt as to its analgesic power. Stillé says : " Its curative powers are unquestionable in spasmodic and painful affections." Its anæsthetic virtue is shown in allaying the intense itching of eczema so as to permit sleep. Its most important use is probably in the treatment of miggraine. As this affection often leads to the establishment of the morphine habit, we should always give this drug a thorough trial before resorting to opiates. Ringer considers it the most useful drug, and lays great stress upon its power of preventing the attack when given over an extended period. It is most effective in attacks due

to fatigue, anxiety or climatic changes. For the relief of a severe attack in the young from gr. ¼-½ of a good extract may be given and repeated in two hours if it does not produce sleep.

It has been found of marked value in the epileptic head-pain and unrest; in neuralgia and neuritis even of long standing; in the constant all day headache not dependent on anæmia or peripheral irritation; in cardiac tumult, senile delirium and the night unrest of paresis or the delirium of celebral softening. In dysmenorrhœa it is sometimes signally successful. In genito-urinary disorders it often acts kindly; in vesical spasm, chordee, retention of urine and the renal pain of Bright's disease. It calms the pain of clap, relieves the distress of gastric ulcer and gastrodynia. In incurable diseases, such as advanced phthisis, it brings euthanasia by allaying pain and producing sleep without the disagreeable after effects of some other drugs.

Failure with the drug is often due to the use of a poor article. At times 10-12 grains of the extract may be given without result, and again a third of a grain may be efficient. Hence the importance of knowing the character of the preparation used. Potter says that if the precipitate formed when the alcoholic preparation is added to water be of a brownish hue, a dirty yellow brown, the sample will be almost inert, but if of a decided olive-green color the preparation will be active.—Dr. Dodge in *Pacific Med. Law.*

ACCIDENT UNDER CHLOROFORM; RECOVERY.—A few brief notes on a case that might be called "apparent death from insufficient chloroform" may be useful. The patient, a strong man of about thirty, had a very large sarcoma of the testicle. Chloroform was at once given, according to our usual practice with new patients from a distance, although he had had a full meal. Chloroform was given slowly by a native assistant. The patient struggled when the first incisions were made and almost immediately began to retch and brought up a little rice. The stomach probably remained full. Up to this time he had taken about five drachms of chloroform, but after vomiting no more was put on the cap by the assistant. I was assisting Dr. Ernest Neve with the operation. A clamp was put on the cord and the tumor cut away. I had previously noticed great dilatation of the pupils. The patient now became rigid, almost in a state of opisthotonos; the face was blanched, no pulse or cardiac impulse could be felt and the breathing stopped. He was lifted at once off the table and put on the floor with legs raised. Artificial respiration was begun and was assisted by irregular gasping inspirations. The wound had ceased bleeding and the heart's action appearded to have stopped; several minutes passed. The clamp on the cord was loosened; dilute ammonia was injected into the arm and half a pint of hot water into the rectum. A slight color now returned, though the pupils continued widely dilated and a feeble pulse could be felt. The operation was rapidly proceeded with and the wound stitched; sensibility began to return. Suddenly smart bleeding began, owing to retraction of the artery above the ligature on the cord. The wound had to be reopened. I then took charge of the chloroform myself and rapidly administered two drachms; the breathing became quiet and regular, the artery was found and ligatured, and the operation completed without any difficulty. I would call attention to a few points: (1) Although a good deal of chloroform was administered, it was obviously insufficient; (2) clamping the cord in a semi-anæsthetised patient who had just been vomiting produced shock which in the higher nervous organization of a European might have proved fatal; (3) the patient bore the rapid administration of another full dose of chloroform when properly administered perfectly well and showed no signs of shock when the cord was religatured.

For over twenty-five years the extensive surgical work of this medical mission has been carried on without a single death under chloroform, which is the only anæsthetic employed. To what do we owe our immunity? Not to climate, for the temperature of the operating room from October to March is never above 50° and often below 40°; partly perhaps to the low nervous organization of the natives and their perfect confidence in taking chloroform; chiefly, it would appear, to the sound principles on which it is administered, according to the rules laid down by Syme, although the actual administrators are always unqualified native assistants.—Arthur Neve, F.R.C.S. E., *in Lancet.*

APPENDICITIS: MEDICAL AND SURGICAL MANAGEMENT.—At the September meeting of the Missouri Valley Medical Society, Dr. A. F. Jonas, read a most interesting paper on the above subject. Notwithstanding the fact that the topic is almost worn threadbare, it is one of such importance that the physician should be alive to the necessity of being ever ready to meet it; especially does this apply to those physicians practicing in rural districts where they do not have time nor opportunity to call counsel.

While surgery is making such rapid strides followed by most brilliant results, he advises the younger practitioners not to be carried away with the idea that medicinal means are of no avail, in the treatment of appendicitis, but urges a more thorough study of the disease, and accuracy in diagnosis. Good sound judgment and anatomical knowledge of the parts are absolutely necessary, before the physician can be sure of just when to call surgery to his aid.

He gives a table of a series of thirty cases,

which have occured in his practice, or have come under his notice. The treatment was largely surgical; a few cases apparently recovered under medicinal measures. After carefully considering the various means, usually employed in the treatment of appendicitis he summarizes as follows:

1. When called early, insist on absolute rest in bed.

2. Fomentations, if pain is severe, and give codeine if necessary.

3. Salines (liberal doses) every half hour until four to six fluid stools are produced.

4. If the salines, after having produced free catharsis, fail to relieve, or aggravate the pain and fever; *operate.*

5. If salines fail to produce free catharsis; *operate.*

6. Temporizing with salines, or any other form of medicinal treatment, is worse than useless. When there is the slightest evidence of the presence of pus, an operation must be done at the earliest possible moment.

7. Guard against over-zealousness in search of the appendix, in large abscess cavities, lest the limiting intestinal agglutination be broken down and general peritoneal infection follows.

8. Always remove the appendix when it can be safely done.

9. Elevate the pelvis, as in Trendelenburg's position, in cases of small abscess limited to the lumen of the appendix or its mesenterium, or in any case when the peritoneal cavity is entered and where the intestinal distention is such as to make it difficult to find the caput-coli, or the appendix.—*Med. and Surg. Rep.*

URIC ACID PATHOLOGY.—It needs a man possessed of no small amount of skill and perseverance to undertake, *à fortiori* to carry out, investigations bearing on the pathology of uric acid, the more so because the results of years of patient research may prove inconsistent or fail to resist hostile criticisms. Dr. Haig, some nine years ago, was mesmerised by the uric acid problems, and ever since he has unintermittingly read contribution after contribution, every time extending the sphere of evil influences until he has eventually come to regard uric acid as the source of many, if not most, of the ills to which flesh is heir to. Nevertheless, and in spite of the fact that his facts do not always concord with clinical observation, his work has indisputably added very materially to our knowledge of a very recondite problem in pathology. A high intra-arterial tension is the forerunner of a whole series of morbid changes in tissues and organs subjected to the continuous strain, and an exaggeration of vascular tension, he shows us to be intimately associated with the pressure of uric acid, in one form or another, in the blood. It acts by causing irritative construction of the arteries, thus increasing the perpetual resistance, and *pari passu* the pressure on the larger arteries. This, however, is only the case provided the heart responds to the greater resistance by increased propulsive energy. If the resistance exceed a certain ratio, or if the heart, from any cause, prove unequal to the occasion, a fall, and not a rise of blood pressure follows. Having established to his satisfaction the fact that vascular tension is the effect of a surplus of uric acid, Dr. Haig proceeds to show that it, and its various sequelæ, may be conjured by measures that facilitate the elimination of the acid, notably by alkalies and the iodides. He professes, indeed, to have substantiated this action by numerous—shall we say—numberless—clinical and analytical observations. Everything turns on the reliableness of the test employed by him to ascertain the presence of, and variations in the quantity of, uric acid, and grave doubts have been expressed on this point, doubts of the value of which we will at once admit our incompetence to decide. We cannot, however, lose sight of the facts brought forward by Sir William Roberts at the last meeting of the Royal Medical and Chirurgical Society. This observer, basing his remarks on laboratory experiments, categorically denies that either alkalies or the iodides have any effect in mitigating the extreme insolubility of the salt of uric acid deposited in the cartilages in gouty arthritis. This salt, the biurate, is radically insoluble, while the uric acid in the blood exists in the form of quadriurates, which are strikingly soluble. The iodides have an action in reducing exaggerated blood-pressure as indisputable as it is immediate, and Dr. Haig maintains that this is because it favors the excretion of uric acid. To this it was objected that nitrite of amyl and kindred substanees bring about a similar fall of vascular tension in a space of time measured by seconds, hence that Dr. Haig's explanation is not admissible. The debate did not turn on questions of detail, for the observers are at variance on the most elementary points. Sir William Roberts, for example, is disposed to regard uric acid in a soluble form as a tolerably harmless impurity, and he points out that it is only when thrown out of solution inside the body as the biurate is outside the body (so to speak) as calculi or gravel, that it is productive of trouble. The question, as will be seen, is full of interest and is highly suggestive. It is just possible that our acceptance of the uric acid pathology of gout has blinded us to the real scope and nature of the production of gout, and that the acid is after all but an incident in the evolution of this protean disorder. Uric acid was formerly regarded as an intermediate, or imperfectly developed product in the formation of urea, but the more modern view is in favor of the two being essentially distinct

We cannot convert uric acid into urea, or urea into uric acid, and certain animals excrete one or the other in virtue of laws still unknown to us. Sir Dyce Duckworth mentioned, at the Clinical Society, that most of the errors that had crept into the pathology of gout were based on the results of investigations carried out in the dead-house and this ought to stimulate observers to pay more attention to its clinical manifestations.—*Hos. Gaz.*

THERAPY OF PHENACETINE.—Dr. John V. Shoemaker writes as follows in his work on Materia Medica, Pharmacology and Therapeutics (Vol. II): Phenacetine was originally introduced into medical practice as an antipyretic and subsequently was found to possess analgesic powers. In diseases attended by hyperpyrexia, such as rheumatism, pneumonia, typhoid fever, and phthisis pulmonalis, phenacetine exerts a very happy effect in about half the dose of antipyrine, the ordinary dose being from 3 to 8 grains. The mortality of the typhoid fever of children has been very materially reduced by the employment of phenacetine. The fall of temperature does not occur until half an hour after the drug has been taken, and the effect continues from four to eight hours. As an antipyretic, phenacetine is considered by many good authorities as the safest and most efficient member of the analine group. In epidemic influenza, phenacetine rapidly relieves the muscular pains and favors diaphoresis; the catarrhal symptoms subsequently require other remedies.

In ordinary colds, one or two 5 grain pills of phenacetine remove all symptoms. The combination of salol (or salophen) with phenacetine is especially useful in influenza and rheumatism. The analgesic effects of phenacetine are very marked in various forms of headache, including migraine and the headaches from eye-strain, having the advantage over antipyrine in not so frequently causing a rash. In the neuralgic pains of tabes dorsalis, in herpes zoster, and intercostal neuralgia, 5 grain doses, given every hour for three or four hours, usually afford complete relief and cause sleep. Phenacetine is extremely useful in chronic neuritis, and, according to Kater, is unsurpassed in the treatment of cerebral disorder due to excessive indulgence in alcoholic drinks. In whooping-cough $\frac{1}{2}$ grain doses dissolved in 10 drops of glycerine are readily taken by children, and afford prompt relief, permitting sleep and ameliorating the attacks. In delirium, a dose of 10 grains of phenacetine will usually afford a quiet night. Mahnert considers phenacetine a specific in acute articular rheumatism, as it reduces fever, relieves pain, and lessens the duration of the attacks. It has been found useful in some cases of gonorrheal rheumatism, and is worthy of more extended trial in this rebellious affection. Given several hours before the time of the paroxysm of intermittent fever, it prevents the chill. In insomnia from simple exhaustion phenacetine acts admirably.—*Med. Rev.*

ANTISYPHILITIC TREATMENT: PROPER DURATION.—This is one of the most interesting and, as is well known, most disputed questions of medical practice. The physician is often puzzled in making a proper choice, and in the end, and after much hesitation, generally decides upon what in his experience has been the mean duration of treatment. But how often do we see patients who, after having been under medical supervision for a length of time, abandon the doctor to treat themselves—believing to have a sufficient experience to permit them to do so, and thus destroy their constitutions by a too-prolonged use of mercurials when the latter are no longer indispensable, yea, often even harmful!

In a communication to the Paris Société de Thérapeutique, Dr. Bontemps, of Saumuz, supported the doctrine of Prof. Fournier, who asserts that in no case can the duration of the antisyphilitic treatment be fixed at less than three or four years. As, at the same time, the patients ought to observe in the medication certain alterations and periods of repose or dishabituation, Dr. B. has arranged the following table, a convenient *vade-mecum* for the practitioner:

First year:
- 6 months of mercurial treatment.
- 3 months of potassium iodide.
- 3 months of repose.

Second year:
- 2 months of mercury.
- 5 months of iodide.
- 5 months of repose.

Third year:
- 2 months of mercury.
- 5 months of iodide.
- 5 months of repose and sulphur baths.

Fourth year:
- No mercury.
- Potassium iodide, with intervals of repose and sulphur baths.—*Merck's Bulletin.*

TREATMENT OF CHLOROFORM COLLAPSE.—Prof. Koenig, of Gottingen, Germany (*La Semaine Medicale*), treats it by rapid compression of the cardiac region. The physican stands at the patient's left, and brings energetic pressure to bear between the ball of his right thumb, at the apex of the heart, and his left hand placed upon the patient's sternum. These are repeated 120 times a minute. This compression disgorges the blood from the dilated and over-filled right ventricle, and causes an artificial carotid pulse to begin. The pupils, always dilated in such a case, contract, and then, after a certain time, spontaneous

respiratory movements begin. Then one may suspend this treatment as long as the pupils remain contracted.—*Lancet-Clinic.*

ECLAMPSIA.—The general opinion now, in Germany, is, that eclampsia should be regarded as a result of poisoning of the blood by substances which circulate in the latter, causing symptoms, such as convulsions, rise in temperature, etc. It is not yet possible to decide whether the inflammation of the kidneys, always present, is primary, and the retention of substances which have been formed in the body itself is caused by this, or whether the poisonous substances are external invaders and give rise to nephritis, as they are excreted. In the most recent times, the opinion has gradually gained ground that the last theory is the true one, and an attempt has been made to seek in bacteria the noxious cause of eclampsia in pregnant women; nevertheless, up to the present time, these are only investigations which do not even give an appearance of probability. Ten years ago, Lochlein estimated the mortality at thirty-two per cent.; he now gives it at nineteen per cent.; Olshausen lost twenty per cent.; the Charité, eighteen and seven-tenths per cent.; Goldberg, twenty-four and seven-tenths per cent; so that the mortality can be fixed at about twenty per cent. The prognosis becomes worse as the number of convulsions increase. If more than fifteen convulsions, the prognosis is unfavorable. With frequent attacks, the prognosis is only favorable if the course of the affection is slow. The more severe each convulsion is, the worse is the prognosis. Eclampsia, with a temperature above 39° C. (101⅘° F.) has double the mortality of that without fever; in all cases with a fatal termination, the pulse is greatly increased in frequency, even after the first convulsion. The prognosis for children is, according to Lochlein, a mortality of thirty-seven and three-tenths per cent.; according to Olshausen, after subtraction of unviable children, twenty-eight per cent.; according to Goldberg, thirty seven and six-tenths per cent. The chief cause of this mortality is the number and quick succession of the convulsions; after twelve or fifteen attacks, the fœtus is usually dead. The unfavorable influence of large doses of narcotics, especially morphine, used for controlling the convulsions, is often observed. The first dose of morphine should be 0.03 grammes (½-grain). It is seldom that more than 0.06 grammes (1 grain) is used, altogether. Olshausen uses little chloral, and recommends chloroform only in those cases where it is desirable to control convulsions immediately.—*Annals of Gynæcology and Pædriatry.*

MASSAGE IN MEGRIM.—Mr. A. Symons Eccles has treated fourteen cases of severe megrim which were under his constant observation. So far as the drug treatment has availed he has found that largely diluted doses of hydrochloric acid given with or directly before food, followed by a capsule containing *b*-naphthol shortly after food, are useful in preventing the dyspeptic conditions which he believes are causative of paroxysmal hemicrania. If it is true that the occurrence of megrim is due to disordered digestion, in the sense that, coupled with sluggish portal circulation, there is an interference with the quantity or quality, or both, of the gastric secretion, that with this insufficiency of secretion there is also inactivity of the gastro-intestinal motor apparatus, permitting the generation, accumulation, and absorption of toxic matters in excess, so that the liver is unable to cope with them either by arrest, combination, or secretion with bile, it seems that the indications for treatment are to improve the circulation through the chylo-poietic viscera, and at the same time to aid the activity of the liver in its function as a policeman in guarding the system against the intrusion of evil-doing leucomaïnes and ptomaïnes, while such a diet is prescribed as shall be easily assimilated, demanding as little activity and richness of secretion as possible. These indications are met by: 1, rest in the recumbent position; 2, massage of the abdomen; 3, general massage of the whole body at least once daily; 4, the dietetic treatment—milk, green vegetables, etc.—*The Practitioner.*

ROSSINI'S MALADY.—The French and Italian medical press have recently taken much interest in the life and ailments of the great composer. The question of special import is: Was Rossini neurasthenic? Two facts are certain about his career. In the first place, he overworked himself. Within nineteen years he wrote thirty-six operas. This implies not only great mental work, but also severe anxiety, due to thirst for public favor and dread of adverse criticism. At the same time he went freely into society; he was also a very gallant gentleman, and in no way abstemious. Secondly, he suffered from a violent shock when 55 years old, for in 1848 he happened to be at Bologna, where he saw people shot down in the streets. This shock affected him for years. He had inclinations towards suicide; he no longer cared for meals, though his appetite and digestion did not fail, and he could not sleep. He complained of intolerable coldness of the hands. His physician prescribed opium, and he—perhaps wisely—would not take that drug. Professional jealousy, especially the increasing popularity of Meyerbeer, aggravated by the ill reception of the *Semiramide* at Florence, increased his ill-health. He often exclaimed: "What a spiritless, ignorant, filthy age we live in!" In respect to his malady, he said: "I suffer from all the ills women complain of—*il ne me*

manque que l' uterus" In age he recovered from his nervour symptoms, and composed a fine Mass in 1869. In fact, he reached an age when he ceased to trouble about the world and his rivals, and so fitted himself for a little more good work. —*Med. Rev.*

PERITYPHLITIS.—Dr. Saundby records a series of fifteen cases of perityphlitis, only one of which was subjected to operation, and this was the only fatal case. A large majority was males, and in six there was a tuberculosis history. The duration varied much, but in several a cure was established in less than three weeks. The treatment adopted was rest, free evacuation of the bowels, and hot formentations or the ice bag, with the addition in chronic cases of repeated blistering over the tumor. In one case spontaneous purging effected a cure without the aid of drugs. The remedies which Dr. Saundby mostly used were calomel, hot Seidlitz powders, and enemata. He does not believe that it is possible to distinquish between cases in which the appendix is really the seat of inflammation and those in which it is not; nor does he think it of the least practical importance. The occurrence of high temporature is no bar to successful medical treatment, as was evidenced by two cases.—*Birmingham Medical Review.*

IS EVOLUTION TRYING TO DO WITHOUT THE CLITORIS?—Dr. Robert T. Morris, in considering the above subject, advanced the following proposition:

1. The prepuce and the glans clitoridis are bound together by adhesions partly or completely in about 80 per cent. of all Aryan and American women.

2. Preputial adhesions are rare among negresses, and seem to occur only in a few of the individuals possessing a large admixture of white blood.

3. Highly domesticated animals do not present examples of the degeneration so far as the author's observations have gone.

4. When preputial adhesions are extensive, the glans clitoridis and the imprisoned mucous glands remain undeveloped, but they may develop later when the physician has separated the adhesions.

5. The failure of the embryonic genital eminence to properly develop the prepuce and glans clitoridis for perfect cleavage propably means that nature is trying to abolish the clitoris as civilization advances.

6. The degenerative process represented by preputial adhesions is characterized by the civilized types of homo-sapiens, in which we find decaying teeth, early falling hair, and imperfect cornea and eye-muscles.

7. Preputial adhesions, which involve small portions of the glans clitoridis, are of interest simply as anatomical curiosities.

8. Preputial adhesions involving a large part or the whole of the glans clitoridis cause profound disturbance, and are among the most pronounced of the peropheral irritations. They cause desire for masturbation, which leads to neurasthenia, and they are responsible for grave reflex neuroses.

9. Preputial adhesions probably form the most common single factor in invalidism in women. The clitoris is an electric button which, pressed by adhesions, rings up the whole nervous system.

10. The physician who fails to examine the female child for preputial adhesions neglects the most important single duty of his professional life.—*Annals of Gynæcology and Pædiatry.*

TREATMENT OF STRUMOUS CICATRICES OF THE NECK.—Calot (*Rev. de Chir.*, May 10th) reports two cases in which he has obtained good results by operation. In the first case the whole of the scarred skin, which measured four centimeters by three, was dissected up, together with about one to two millimeters of the surrounding normal integument. This was removed, and the edges of the wound were freed for a short distance, and then completely united with sutures, no drainage of any kind being employed. The result was very satisfactory, the original scarred surface being replaced by a fine cicatrical line, which was scarcely visible. In the second case the same method was adopted with a similar good result. Calot proposes the same plan for scrofulous ulcers, and also for ulcers which are kept up by the presence of a suppurating gland. He advises total removal of the suppurating gland, along with the surrounding ulcerating tissues and skin, and then close suture of the wound and no drainage.—*Br. Med. Jour.*

DOBELL'S SOLUTION.— The following is the formula of this popular mixture:

℞—Acid carbolic pur., . . . ʒss.
Sodi bi-carbonat.,
Sodi borat., ... aa ʒj.
Glycerini, . . . ʒj.
Aquæ, . . . q. s. ad ʒxvj.—M.

It is a most excellent wash for the throat and nasal passages, and as a spray for diphtheritic throat it is very much used.—*Med. Rev.*

CHRONIC RHEUMATISM.—

℞—Liq. potassi arsenitis,. . . . ʒ ss.
Potassi acetatis, ... ʒ iij.
Vini colchi rad. . . . . ʒ ij.
Ext. cimicifuga, ... ʒ iij.
Ext. phytolacca, ... ʒ iss.
Aquæ menth pip. . . . . ʒ iij.—M.

Sig.—Two teaspoonfuls in water every four hours.—*Med. Summary.*

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DYSPEPSIA.

The general practitioner is constantly faced by patients complaining of this symptom, and it is probable that the most of us have each his own routine in the treatment of it. No symptom-complex calls for more careful discrimination on the physician's part, at any rate if he is going to meet with any uniform success in treatment based upon a clear conception of its pathology. Of course the first duty is diagnosis of the cause, and the second, closely associated with the first, is the recognition of associated conditions, such as anæmia in young persons, especially females, or malignant disease in those of advanced years, or the dietetic or hygienic errors, excess in eating, drinking, or smoking, which lie at the root of most cases. Into the question of diagnosis and pathology the scope of this article does not permit us to enter. As regards treatment, cases fall into two large classes: first, those in which flatulency is the main symptom, frequently most annoying at night, and accompanied usually by more or less dilatation of stomach, gaseous and acid eructations, and, especially in women, distention of the intestines with gas, "bloating of the stomach" as they call it. The cases not so characterized may be relegated to the second group.

The main difference in the treatment of these two classes lies in the prescribing of diet suitable to each case, and a rudimentary knowledge of our *fin-de-siècle* physiology should suggest the right dietary. Starchy, fatty, and other carbohydrate and hydrocarbon articles, if insoluble when swallowed, must lie unchanged in the stomach till passed on into the intestines. Proteid food stuffs are liquefied and made osmotic in the stomach. Flatulency indicates that one if not all three of the stomach functions is in abeyance, these three being abscrption, motion on of its contents into duodenum, and digestion of proteids. If the power of motion onwards of its contents be impaired, time is allowed for abnormal fermentation processes, the gas evolved still further dilates the stomach, the free hydrochloric acid, the natural antiseptic of the stomach, is secreted in less amount just at the time it is most needed, and so the pernicious process acts and re-acts in its own favor unless nature be intelligently relieved.

Treatment should in all such cases be at least partially explained to the patient, that his own self-denial may be enlisted. He should be told that medicine in his case takes third place and that the dietetic and hygienic treatment are more important than the medicinal.

As to dietetic treatment, the main indication is to limit the foods not digested in the stomach, *i.e.*, the starches. Reduce to a minimum the quantity, especially, of bread and potatoes consumed; allow vegetables green and fresh, if not of a too bulky character, but forbid cabbage in any form except shredded fine and raw. Prohibit salt or warmed-over meats, fish and fish-balls, cheese, high game, pastry, pickles, strong condiments, sweets, and fats; in short, as far as possible, reduce the patient to the diet of half-cooked tender beefsteak and plain water. If the necessity exists, pre-digest even the proteids with papoid, or with pepsin and hydrochloric acid after the approved method we have learned of late for making beef-tea. If the patient can afford it, order peptonized beef-powder, or beef-cacao, or liquid peptonoids; or if necessary, even resort to partial alimentation by the rectum, supplying carbohydrates in this way, as described in a recent number of this journal. There are probably few cases, except in a consultant's practice, that do not present themselves for treatment before such a stage is reached as to make these latter measures necessary, unless it be as a consequence of acute gastritis.

As regards fluids, the difficulty is to find a suitable substitute for tea and coffee at meals. One may

mention milk of course, diluted if necessary, cocoa and chocolate, beef-cacao, *black* coffee (the harm being done by the adding of milk to a scalding decoction of the bean, which makes the compound more indigestible), and after the process of digestion is well over, say three hours after a meal, any of the ærated waters now so readily obtainable, such as Vichy, or Apollinaris, the carbonic acid given off being grateful to the usually irritable gastric *mucosa*. And one must not omit to direct, first thing in the morning, before the night dress is removed, a tumblerful of either ærated or plain cold water. The sipping of hot water is frequently felt by the patient himself to be painful to the stomach, and therefore harmful. The cold water is of service only at this one period in the twenty-four hours. Its rationale seems to be, that the stomach is on rising in the morning as empty and well-contracted as it can be, and the entrance of the cold water excites vigorous peristalsis, causes excellent *lavage*, the only kind that patients as a rule can be in this country induced to submit to, and after flushing away the collected mucus and *debris* from the last meal, goes on to prepare the intestines for a normal evacuation in an hour or two after, aided by the peristalsis set up by the ingestion of breakfast. The simple measure of setting a tumbler of ice-water by the bedside, from which a sip might be taken when the patient rises wakeful and uneasy on his elbow during the night to give vent to the relieving and reverberating "yawp," will be found by many a sufferer most soothing to the heated stomach. And though it is rather out of its proper order, ice-cream must be mentioned as an excellent article of diet for such stomachs.

2. The medicinal treatment is simple. And here it may be said, in the face no doubt of disapproval from many an old practitioner, that an alkaline stomachic is bad treatment in every case except that of acute indigestion when the stomach imperatively calls for relief from the acid fermenting mass it contains, which may be chemically neutralized if alkalies are given in sufficiently large doses, then evacuated thoroughly if emesis have not already occurred. The apparent success of alkaline stomachics, in trivial cases of pain after eating due in strong adults to dietary indiscretions, is due to their chemical effect upon the acid irritating contents, the *vis medicatrix naturæ* being capable of restoring function without further aid. But rational treatment calls for the temporary artificial supply of the stomach's own antiseptic, hydrochloric acid, which though acid, diminishes the hyper-acidity of which these patients complain, by preventing the abnormal fermentation which produces lactic and acetic acids, and even alcohol, in the stomach. In addition to hydrochloric acid, strychnine has a well-known action upon unstriped muscle through the nerves, and the motor functions of the stomach can be by it much improved. Then a simple or aromatic bitter is of proved value. So that for a routine prescription we know of nothing that produces better results, both upon appetite and function of stomach and intestines alike, than the following:

℞—Liq. strychniæ, ... ℥ jss.
Ac. mur. dil., ... ℥ v.
Tr. cinchonæ co.,
Syr. limonis, ... āā ℥ j.
Aq., ... ad. ℥ vj.—M.
Sig.—℥ ij. ex aq. p. c.

Constipation of course will probably, at any rate at first, need pil. aloin. bellad. et strychn. or cascara in addition to the cold water in the morning, and the strychnine after meals; but with the return to fair health and digestion the constipation, too, will in most cases be much improved. In many cases one drop of creasote with two or three drops of hydrocyanic acid, in water, has proved most effective in controlling the abnormal fermentation and flatulency. We do not think that creasote is used in this condition so often as it should be.

PROPOSED AMENDMENT TO THE PHARMACY ACT.

Our readers will be interested in noting the proposed changes in the Ontario Pharmacy Act, which affect the medical profession. The new Bill will, if it become law, make a serious change, and not for the better in the condition of a good many medical men in Ontario. The idea seems to be that a practising physician should not be the proprietor of a drug store. At any rate the proposed amendments will, we think, effectually shut out physicians from that business.

Section 31 of the old Act gives doctors the right

to carry on the business of a pharmaceutical chemist, *without passing the examination prescribed by the College of Pharmacy.*

The proposed amendment strikes out from this section the words "he shall not be required to pass the examination prescribed by the College of Pharmacy," and inserts in their place:

"He shall be at liberty to do so in places other "than incorporated Cities or Towns in the Prov-"ince of Ontario, so long as he employs an assist-"ant to manage or have charge of such business "who is a legally qualified Pharmaceutical Chem-"ist, and he shall be required to pass the final "examination for the degree of Phm. B. of the "Toronto University."

There are also three sections added to the old amended Act, one of which is as follows:

"36. No physician or medical practitioner in "any incorporated City or Town can become regis-"tered as a Pharmaceutical Chemist and carry on "business as a Chemist and Druggist unless he "ceases to practise as a Physician and Surgeon, "and unless he passes the final examination for "the degree of Phm. B. of the Toronto University, "and has in all other respects complied with the "requirements of this Act; provided, also, that "any medical practitioner who at the time of the "passing of this Act is lawfully engaged in carry-"ing on the business of a Pharmaceutical Chemist "under the provisions of this Section, may con-"tinue so to do by registering and complying with "all the other requirements of this Act."

These changes and additions need no comment. The profession will at once see that if they become law, physicians are practically debarred from carrying on the business of chemist and druggist.

Fancy a doctor who has been in the drug business, and in practice for twenty years, commencing to fag up for the final examination of "Phm. B. of the Toronto University." It goes without saying that he must go out of business. Then comes the generous offer that he may remain in business in places other than incorporated towns and villages, IF he keep a Phm. B. to look after it, and also becomes a Phm. B. himself. The conditions are too hard and he must in nine cases out of ten go out of business. It seems to us that some concerted action should be taken to see that the vested rights of medical men shall not be interfered with in this matter.

GOLDEN RULES OF SURGICAL PRACTICE—*(Times and Register)—Continued.*

THROAT..—In cut throats where the trachea has been opened, never neglect to remove all small fragments which hang loose in the trachea, or they will swell and eventually stop respiration.

Never leave a scald of the glottis a minute without tracheotomy tubes and knife placed at hand.

Do not neglect to warn your patient that the food may run away after tracheotomy through the tube for the first few hours.

Never neglect or think lightly of stab wounds of the neck.

In œdema of glottis due to syphilis, erysipelas, wounds of glottis, scalds, always have the tracheotomy instruments by the bedside.

Remember that in stab wounds of the upper part of the neck with arterial bleeding, there is an impossibility in many cases of distinguishing the exact source of the hæmorrhage, so numerous are the great vessels in that region. Apply a ligature to common carotid or external carotid if excessive.

Remember that tracheotomy and insertion of tube is especially necessary in wounded epiglottis or arytenoid cartilages.

Always secure your tracheotomy tube by knotting the tape. Little patients are apt to drag at a loop.

Remember diffuse cellulitis of the neck is very fatal.

Avoid sutures in cut throat, when the windpipe is opened.

Never put silk or silver ligatures into a wounded œsophagus; only use catgut.

Never forget that fractures of the laryngeal cartilages are of serious importance; the nearer the cords, the acuter the symptoms, the more decisive must be the treatment. If the fragments are displaced and the mucous membrane lacerated or perforated by the fragments (as testified by emphysema and blood spitting), tracheotomy must immediately be performed.

Never neglect in all sudden dyspnœa in a child to pass your finger into the upper part of the larynx to search for a foreign body.

Sanction no delay in removing a foreign body known to be in the larynx.—Invert.

Never hesitate in foreign bodies in trachea to invert the patient after the tracheal incision has

been made for the extraction of the foreign body. Never use forceps, rather invert the patient, or use a hook, bent probe, or wire snare, inversion, succussion.

But never invert unless you have your tracheotomy instruments ready, for the danger of instant suffocation, through lodging of the foreign body in the glottis, is great.

Never forget that lung disease invariably ensues on the retention of a foreign body in the bronchus.

WARNINGS TO PATIENTS AND THEIR FRIENDS.—Never forget to warn your patient that a Colles' fracture, even when treated with the greatest care, leaves some deformity.

Never forget to warn a case of fracture of the patella, that the fragments tend to separate.

Always warn your patient that there may be loss of power of deltoid after dislocation of shoulder if much pain is experienced, *i. e.*, the nerves have been pressed upon.

Always warn the patient or his friends of the possibility of suspension of growth, in injury to an epiphyseal cartilage.

Never forget to warn the parents of a hare-lip that the operation is usually inadequate.

Never forget to warn your patient that the loose cutaneous anal tags swell after an operation for piles, or he may suppose you have overlooked them.

Never forget to warn your patient that a Meibomian cyst fills with blood after being scooped out, or he will think that the operation has been performed in a slovenly manner.

Always warn the patient's friends that fluid taken by the mouth may run out through a tracheotomy wound for the first few hours, and that such is not due to a wound of the gullet.

WOUNDS.—Never forget that the surgeon who neglects to suture a nerve or tendon commits the same mistake as he who neglects to reduce a fracture.

Never forget the tripod of successful healing of wounds has three legs—ascepticism—rest—coaptation of edges.

Never forget that if an operation wound suppurates the fault lies with the operator or his assistants.

A SCHOOLBOY ON BONES.—The following essay on bones was actually written for a school exercise y a boy. It may be found helpful as an introduction to the science of osteology *(The Hospital Gazette)*. "Bones are the framework of the body. If I had no bones in me I should not have so much motion, and grandmother would be glad; but I like to have motion. Bones give me motion because they are something hard for motion to cling to. If I had no bones, my brains, lungs, heart, and large blood-vessels would be lying around in me, and would get hurted, but now the bones get hurted, but not much, unless it is a hard hit. If my bones were burned I should be brittle, because it would take the animal out of me. If I were soaked in acid I should be limber. Teacher showed us a bone that had been soaked. I could bend it easily. I would rather be soaked than burned. Some of my bones don't grow close to my body, snug, like the branches of a tree, and I am glad they don't, for if they did I could not play leap-frog and other nice games I know. The reason they don't grow snug to my body is because they have joints. Joints is good things to have in bones. There are two kinds. The ball and socket, like my shoulder, is best. Teacher showed it to me, only it was the thigh-bone of an ox. One end was round, smooth, and whitish. That is the ball end. The other end was hollowed in deep. That is the socket, and it oils itself. It is the only machine that oils itself. Another joint is the hinge joint, like my elbow. It swings back and forth, and oils itself. It never creaks like the school-room door. There is another joint that don't seem like a joint. That is in the skull. It don't have no motion. All my bones put together in their right places make a skeleton. If I leave any out, or put any in their wrong places, it aint no skeleton. Cripples and deformed people don't have no skeletons. Some animals have their skeletons on their outside. I am glad I aint them animals; for my skeleton, like it is on the chart, would not look well on my outside.

CHLORAL IN OSTEOMALACIA.—Dr. M. Petrome, of Naples *(Wiener Med. Presse—Cincinnati Lancet-Clin.)*, knowing that this disease is produced by a nitrate-forming bacteria, which is rapidly killed by chloroform, conceived the idea to administer chloral internally in the treatment of the affection, as this drug produces chloroform as a by-product after its introduction into the system. The writer thus treated a fifty-year-old vii-para, who had always been well and healthy,

and who, after a contusion of the left lower limb, was seized with violent pains in that extremity. A few months later, during pregnancy, she was attacked with pains in the pelvic bones, the ribs and vertebræ, which increased in intensity and were followed by osseous deformity. As he first saw her her trunk formed a shapeless mass, the vertebral column was inclined forwards. She was not as tall as before, the pelvis showed the characteristic changes of osteomalacia, only the cranial and facial bones being uninvolved. The spontaneous pains from which the patient suffered constantly were increased on pressure. Walking or standing was impossible, even the slightest movement in bed caused her terrific pain. Besides this, she was tortured by attacks of coughing and hiccough. The urine contained neither sugar nor albumen, but traces of propeptone, and quite a large quantity of nitrous acid. He commenced treatment by giving two grammes (30 grains) of chloral hydrate, per diem. After three days the pains had greatly decreased in intensity, the urine was free from propeptones, and showed a decrease in nitrous acid. On the fifth day the urine was normal. At the end of the first week the patient was able to leave her bed, clothe herself and take a few steps, without assistance. The spontaneous pains had nearly entirely disappeared, as well as the coughing and the hiccough. In fourteen days she could be regarded as cured. The treatment was continued for eight days longer, three weeks in all. At present the patient is well, has no pains, ascends stairs and makes all sorts of movements without the slightest difficulty. The writer ascribes the curing of this case to chloral, respectively, to the chloroform alone, and in those cases which have been successfully treated by castration, he attributes the result to the chloroform, used anæsthetically, rather than to the operation itself.

SYPHILIS OF THE CEREBRAL ARTERIES.—The Lettsomian lecture delivered before the Medical Society of London, England (*Hos. Gaz.*), on Monday last, by Dr. Bristowe, takes us back to a time when the influence of syphilis in determining arterial diseases was apparently not even thought of. Case after case was observed in which the most extensive disease of arteries, large and small, was associated with a history of syphilis, but it was long before even Dr. Bristowe timidly suggested that they might possibly stand to each other in the relation of cause and effect. The lesions were often of extraordinary severity; the aorta was sometimes reduced to an almost impervious canal, in other cases the pulmonary arteries hardly admitted a bullet probe, and in a large proportion the principal arteries carrying blood to the brain had been rendered impassable. A noteworthy feature in several of the cases narrated by the lecturer is that the symptoms heralding the advent of this terrible complication supervened within a few months, is, at any rate, within a year or two of the primary infection. It is impossible to avoid the conclusion that arterial disease of the gravest description and extent, may occur at any stage of syphilis, and is not, as is often assumed to be the case, only or principally to be met with among the tertiary phenomena. Another point to which attention was called is that the virus does not act exclusively on the large vessels, for all large enough to have a distinctive name are occasionally affected, andthis fact justifies the inference that these changes may probably affect even microscopic arteries, determining atrophic lesions in the tissues supplied by them less in severity only because less in extent. We are at present hopelessly in the dark as to why in a certain proportion of the cases the arterial system is singled out for degenerative processes consequent on syphilis, and we are fain to fall back on the time-honored explanation (?) of a selective action due to the inherent liability to degenerative change on the part of certain tissues—the particular tissues varying according to the individual.

Dr. Bristowe formally repudiated the view that the tertiary lesions of syphilis are not infective. Admitting that they are less so than those of the first and second stages, he points out that this may be due to the fact that the tertiary lesions are more strictly localized, and are, moreover, usually situated in positions not lending themselves readily to the transmission of the virus. The cases quoted of infection by tertiary lesions were not very conclusive, because they involve the acceptance of patients' statements—a disturbing factor in deciding questions of this magnitude.

THE CONTAGIOUSNESS OF CANCER.—During the

last few years, physicians and surgeons *(Concours Med.)*, recognizing their inability to cope single-handed with the two great scourges of mankind, tuberculosis and cancers, have joined their forces to fight with a greater chance of success. The treatment and prophylaxis of tuberculosis have occupied the minds of the best men of the medical profession, and the causes of the disease seem to be well known, while the study of cancer is still surrounded with countless mysteries. For many years careful observers have noted the transmission of cancer by heredity. Modern histologists are extremely divided upon the microbiological question of cancer. In some cases this disease seems to be infectious; it penetrates into the organism through some solution of continuity, at first being localized and later giving rise to secondary formations by a series of successive growths. Cancer is inoculable from one animal to another of the same species. It is probably inoculable from man to an animal. Many reported facts seem to prove that cancer is contagious. The contagion may take place through direct inoculation and also by means of soiled linen and clothes. M. Dave has mentioned the history of a young man, thirty-eight years old, who died of cancer of the tongue. A few years previous to his death, his father-in-law had died of an epithelioma, which after appearing on the ala of the nose, had spread over the face and superior maxilla; contagion evidently took place through the son-in-law smoking the pipes used by his father-in-law. The incubation of cancer varies from several months to a few years. About 72 per cent. of the cases die within five years from the date of the time of infection. Contagion is not common, however, for it requires a predisposition, fortunately not very frequent.

THE TREATMENT OF PUERPERAL FEVER.—The paper on this subject by Dr. Lapthorn Smith in the *N. Y. Jour. of Gynecology and Obstetrics* is fertile in suggestive and advanced ideas. He urges that when there is the slightest rise of temperature, the clean doctor or the clean nurse should with a clean syringe give a vaginal injection of hot water with or without permanganate of potash. The vaginal douches should be repeated every eight hours. If in spite of this the temperature has not fallen within twenty-four hours the uterus must be washed out with plain hot water and an iodoform-gauze drain be lightly packed in the uterus, the end being left like a lampwick coiled up in the vagina. If the temperature still keeps high after twenty-four hours we should draw down the uterus gently and thoroughly curette, and again wash out and drain with gauze. From the very first rise of temperature until the patient has recovered we should keep the great main sewer of the body, namely, the intestine, constantly flushed by means of small doses of sulphate of magnesia so as to carry off the ptomaines as much as possible. The only drugs required for internal use are quinine and alcohol. The coal-tar antipyretics by bringing down the temperature while the disease is rapidly progressing mislead us into false security so that precious hours may pass unutilized. Besides, the coal-tar group paralyze the heart, on the endurance of which the life of the patient greatly depends. If in spite of all that we have done the temperature continues to rise, and the life of the patient begins to be clearly in danger, let us explore the abdomen and remove the cause, even if this necessitates the removal of the uterus, before pyæmia or general peritonitis ensue.

THE EFFECTS OF ACIDS ON THE FUNCTIONS OF THE STOMACH (*Centralbl. f. Klin. Med.*)—1. Acids throw down a considerable precipitate of mucus.

2. They increase the cellular elements of the gastric contents.

3. Their introduction is followed by butyric acid reaction, most marked after hydrochloric acid.

4. Larger quantities of the acids result in a considerable effusion of bile into the stomach.

5. They stimulate the secretion of pepsin, but have no influence upon the secretion of hydrochloric acid.

6. Their long-continued administration is followed by marked diminution of the secretion of hydrochloric acid.

7. Even in large quantities, hydrochloric acid produces no gastric disturbances On the contrary, a continued administration of the acid is attended with a feeling of well being.

8. The difference in the effect between acids and the alkaline salt on the gastric functions consists in the fact that the alkaline salts dissolve the mucus and decrease the secretion of pepsin, while

the acids precipitate the mucus and increase the secretion of pepsin. The disappearance of the alkaline salts from the stomach is followed by a decided increase of the hydrochloric acid secretion. This does not occur, or only to a slight degree, in the case of acids. Both the acids and salts, in large quantities in continued use, have the same effect in lowering the activity, and finally in destroying the function of the glands secreting hydrochloric acid.

BACTERIOLOGICAL NOTES.

COMPILED BY E. B. SHUTTLEWORTH.

Death Point of the Cholera Spirillum.—It has occurred to the writer that some of the information afforded by Dr. Sternberg's exhaustive paper on Disinfection at Quarantine Stations—to which allusion was made in our last number—may serve a useful purpose, for reference, if put into tabular form. The figures herewith appended give the death point of the spirillum, after exposure to the agents indicated:

DIRECT SUNLIGHT.	
Bouillon culture, in test tubes. (Sternberg)	5 hours.
Bouillon culture, on writing paper. (Wilson)	2 "
DESICCATION.	
Culture, spread on glass. (Koch)	3 "
Culture on blanket, in sun and air. (Sternberg and Wilson)	4 "
Culture on blanket, in dark closet. (Sternberg and Wilson)	48 "
MOIST HEAT.	
Bouillon on cotton, at 52° C. (125.6° F.). (Jenkins and Sternberg)	10 min.
Bouillon on cotton, at 60° C. (140° F.). (Jenkins and Sternberg)	very brief
DRY HEAT.	
Bouillon on blanket, at 60° C. (Wilson)	45 min.
Infected woolen garments, at 80° to 100° C. (Sternberg)	30 "
VARIOUS MEDIA.	
White wine. (Imp. B'd Health, Germany)	5 "
Red wine. " " "	15 "
Cider. " " "	20 "
Cold coffee. " " "	2 hours.
Pilsener beer. " " "	3 "
Tea, 4 per cent. " " "	1 "
Tea, 3 per cent. " " "	24 "
Tea, 2 per cent. " " "	96 "
Milk, unsterilized. " " "	24 "
Smoked and salted herring. " "	24 "
Confectionery, chocolates, almonds. "	24 "
Strawberries. " " "	24 "
Dried fruits. " " "	48 "
Fresh fish and shell fish. " "	48 "
Pears. " " "	5 days.
Cucumbers. " " "	7 "
Cherries, sweet. " " "	7 "
Cherries, dry. " " "	5 "
Cherries, sour. " " "	3 hours.
Bouillon, containing 50 per cent. beet sugar. (Sternberg)	48 "
Bouillon on cotton between sacks beet sugar. (Sternberg)	4 days.
Bouillon on cotton placed in beet sugar. (Sternberg)	48 hours.
Copper and silver coins. (Uffelmann)	30 min.
Dry hand. "	2 hours.
Fabrics, apparently dry. "	4 days.
Pages of printed book. " alive after	17 hours.
Writing paper in envelope. " "	23½ "
Dry hand. " "	1 "
Smoked fish. " "	4 days.
Butter, slightly acid. " "	6 "
Roasted meat, under bell jar. " "	7 "
Rye bread, under bell jar. " "	7 "
Moist fabrics. " "	12 "
Cocoa, infusion, 1 or 2 per cent. (Imp. B'd Health, Germany)	7 "
Milk, sterilized. " " "	9 "

It is needless to point out the many applications that may be made of the details afforded by the above table. One fact may, however, be emphasized—the low thermal death point of the cholera spirillum. It is definitely stated, by Drs. Jenkins and Sternberg, that the organism is destroyed by exposure, for a very brief period, to a moist heat of 140° F., and in thirty minutes by a dry heat of 176° to 212° F. Prolonged contact with steam under pressure, as commonly used, is needless, provided the heat penetrates the articles to be disinfected. A high dry temperature, which in most cases utterly destroys clothing, is also unnecessary. The employment of this agent, as commonly directed, is a waste of energy. As Dr. Sternberg says, it is like using a sledge-hammer for the purpose of killing a mosquito. The practice originated in the experiments of Koch and Wolffhugel, in 1881, who found a temperature of 284° F., maintained for three hours, to be necessary for absolute sterilization. It must, however, be remembered that these gentlemen experimented on spore forming organisms, as *B. anthracis*, *B. tuberculosis*, or *B. subtilis*, which are exceedingly resistant, though not to the same degree. The assertions in regard to these have been made generally applicable, and, apparently, unnecessarily so. The writer, when in the United States during the epidemic, last year, had the pleasure of seeing some of the experimeuts then being carried on by Drs. Sternberg and Wilson, in the Hoagland Laboratory, Brooklyn. The investigation was continued for some months, and has been very thorough. Dr. Sternberg's position, as Deputy-Surgeon General of the U. S. Army, and his high reputation as an authority on bacteriology, give additional weight to his assertions, which, though contrary to the notions entertained by some, are entitled to be received with confidence.

THROUGH the courtesy of His Honor the Lieut.-Governor of Ontario, a communication has been received by Dr. J. Gibb Wishart, Secretary of the Ontario Medical Association, covering an invitation from the Central Committee of the Eleventh International Medical Congress, meeting in Rome on the 24th of September, 1893, to the President and members of the Ontario Medical Association to appoint delegates to this Congress. Members who desire to attend this Congress should communicate with Dr Wishart.

At a meeting of the Windsor Physicians and Surgeons Association, held Feb. 13th, 1893, the following resolution was unanimously adopted:

Whereas a number of Medical Practitioners are endeavoring to secure legislation in a manner subversive to the usefulness of the Ontario Medical Council. We who daily contrast the Medical legislation of Michigan with the efficient legislation of Ontario, beg to express our entire confidence in our Medical Council. And we beg to urge that any change in legislation that we may desire, should be brought about only through the Council by the representative whom we elect.

Books and Pamphlets.

SYPHILIS AND THE NERVOUS SYSTEM. By W. R. Gowers, M.D., F.R.C.P., F.R.S. Philadelphia: P. Blakiston, Son & Co. Toronto: Carveth & Co. 1892. $1.

This is a revised reprint of the Lettsomian lectures for 1890, delivered before the Medical Society of London. The name of the author is sufficient warranty for both the scientific and practical value of the work. It deals exhaustively with the subject and will be most interesting to those engaged, either in general practice or in studying the diseases of the nervous system.

HYGIENE AND PUBLIC HEALTH. By Louis C. Parkes, M.D., D.H.P. Lond. Univ.; Lecturer on Public Health at St. George's Hospital, etc. Third edition. Illustrated. Philadelphia, 1892: P. Blakiston, Son & Co. Toronto: Carveth & Co. $2.75.

This new edition of Parkes' standard and well-known work has been thoroughly revised, and enlarged. Among new matter introduced are the subjects of Smoke prevention by mechanical appliances, Weather observations and Cyclonic systems, and Epidemic influenza. The chapter on Diphtheria has been re-written, and other chapters dealing with Etiology and Bacteriology have been brought up to date. The work is classical, and we can heartily recommend it to those engaged in the study of hygienic science.

A MANUAL OF CLINICAL OPHTHALMOLOGY. By Howard F. Hansell, M.D.; Lecturer on Ophthalmology in Jefferson Med. Coll., etc.; and James H. Bell, M.D., Ophthalmic Surgeon to the South-western Hospital, etc.; with 120 illustrations. Philadelphia: P. Blakiston, Son & Co. 1892. $1.75.

This work does not treat very fully of the subjects contained in it, being of the nature of clinical lectures, and practice. It is concise. It deals with the anatomy, physiology, refraction, and common diseases of the eye, in a pointed and practical manner, which makes it of great value to the general practitioner and student. The illustrations are good, and altogether the work undertaken is well done.

DISEASES OF CHILDREN. A manual for students and practitioners. By C. Alexander Rhodes, M.D.; Instructor on Diseases of Children, New York Post-Graduate Medical College. Philadelphia: Lea Brothers & Co. Toronto: Carveth & Co. 1892.

This is one of the student's Quiz series, compiled from Keating's Encyclopædia of Diseases of Children, J. Lewis Smith, Eustace Smith, Jacobi, Meigs, Pepper and various other authors. It is in convenient form for students and will be useful for rapid reference by practitioners.

ANATOMY. A manual for students and practitioners. By Fred. J. Brockway, M D.; Assistant Demonstrator of Anatomy, Coll. Phys. and Surgs., New York; and A. O'Malley, M.D., Instructor in Surgery, New York Polyclinic. Philadelphia: Lea Bros. & Co. Toronto: Carveth & Co. 1892. Pp. 376.

The authors have selected from the larger works of Gray, Quain, Henle, and others, the Essentials of anatomy, such as will be most useful to the student and practitioner. Illustrations have been introduced. Both English and metric measurements have been given. A useful addition is a glossary, which will certainly aid students in acquiring a correct pronunciation of anatomical terms. A useful book.