to the patient's efforts and to the ascent of the soft palate. During the second stage of deglutition a negative pressure is avoided, because the primary increase in pressure and the bulging of the elastic lateral walls of the nose supply a sufficient amount of air reserve to enable the descent of the soft palate to occur without creating a partial vacuum in the nasopharynx.

Until familiar with this method of inflation it is well to take a full breath preliminary to its performance. It would be unwise to prescribe this treatment for some patients, as it is subject to abuse and harm might result from faulty execution.

The author's automatic ear inflator.

To avoid all danger, I have used an instrument, here illustrated, so constructed that, no matter how forcibly the patient blows into it, the pressure in the instrument, and, therefore, in the nose during treatment, can not exceed a certain number of millimeters of mercury—the latter to be determined by the surgeon when prescribing the device. It consists of a rubber balloon attached to a nosepiece. If one nostril is closed and the nosepiece is fitted tightly into the other nostril, the patient may inflate the balloon by blowing into it through the nose. In the size I usually employ, a pressure of about 10 mm. of mercury is required to inflate the air bag, and, no matter how much it is distended, the pressure within it will remain between 5 and 10 mm. of mercury. After inflating the balloon, the patient, without removing the nosepiece or releasing the opposite nostril, swallows, and repeats this act several times; or, until the balloon has completely collapsed. At every deglutition both middle ears will be inflated. During deglutition the air pressure in the balloon remains at about 6 or 8 mm. of mercury until the bag is deflated, when the pressure drops suddenly to zero.

So far as I can see, this method is simple, safe and efficient, and enables patients to obtain every day treatment which can not but benefit the diseased conditions it is adapted to correct.

I have made many modifications of this instrument, but the one described is the simplest and cheapest. The balloon may be made in various sizes and strengths, depending on the amount of air and the pressures it is desired to obtain.

The experiments here detailed were devised by me and executed first on myself and subsequently on many other individuals, and with one or two exceptions were confirmed by competent observers. To carry out the more delicate experiments correctly much practice and patience are required.

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ABSTRACT OF DISCUSSION

Dr. J. Holinger, Chicago: The instrument Dr. Fowler shows is of decided advantage, because of its psychic effect, if for no other reason, so long as the patient has something which requires some special preparation it is an inducement for him to carry out directions more accurately. I did not hear Dr. Fowler mention the name of Professor Bezold, who has done so much along this line. A point which I think is often overlooked in affections of the tube is the difference in size of the cells in the mastoid process. If it is true that these cells have the function of a reserve air tank, it is evident that the difference in size of the tank can not be without influence on the ventilation and function of the middle ear. It seems to me that together with the adenoids this smallness of the reserve air tank in children explains the frequency of affection of the tube much better. In adults, too, after destruction of the cells by mastoid operation, I have often seen affections of the Eustachian tube which necessitated treatment extending over many weeks.

Dr. George E. Shambaugh, Chicago: I do not wish to pass unfavorable criticism on a piece of work on which so much time and patience has been spent as this paper by Dr. Fowler. I should like to suggest, however, that the one object in writing a paper is to convince other people of conclusions which our work has led us to believe are correct. It is a point that we should always keep in mind in preparing a paper. An article that covers so much as this paper by Dr. Fowler is much less likely to accomplish this object than a paper in which not so much is attempted.

Dr. Edmund P. Fowler, New York: Probably Dr. Shambaugh is right in saying that I have tried to give in one paper too many experiments pertaining to the Eustachian tube, and it would have been wiser perhaps to have presented one phase of the matter at a time. My object was to obtain criticism from men who have investigated the experiments before the meeting, to the end that my work could be substantiated. The little instrument I have shown I have used in the form of a penny whistle for some time with apparently happy results. We must be cautious in our opinion because every new treatment is apt to show remarkable results at first, but I think the method outlined has benefited a great many people and especially those who could not spare the time for regular treatment. This treatment, so far as I can see, is harmless and enables patients to maintain the drum membrane in a better condition than if they carried out no treatment at home.

SYMPTOMS OF INTRACRANIAL COMPLICATIONS OF PURULENT OTITIS *

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The subject of intracranial lesions arising from suppurative otitis has occupied the foreground in otologic discussions for many years. The brilliant achievements of Macewen in this difficult surgery were already well known in the 80's and his classical work "Pyogenic Diseases of the Brain and Spinal Cord," published in 1892, stands, with few parallels in surgical literature, as a monument to his genius.

The impetus exerted by this publication in extending the otologist's field has been tremendous, and has resulted in the cure of hundreds of cases which previously had been considered hopeless.

Added knowledge of the function of certain areas in the brain, improved methods of study of the blood, and cerebrospinal fluid, and accurate data regarding the static equilibrium, have all contributed in recent years, to an earlier and more certain diagnosis of the character and localization of intracranial lesions.

Certain clinical data have now come to be recognized as definite proof of lesions which formerly were considered doubtful. Nevertheless it must be admitted that, with all the evidence which can be accumulated, we are too often in grave doubt as to the nature or

* Read in the Section on Laryngology and Otology of the American Medical Association, at the Sixtieth Annual Session, held at Atlantic City, June, 1909.
location of a lesion which presents symptoms serious enough to force us into an exploratory operation. Under these circumstances one can not escape a feeling bordering on helplessness in the presence of such a desperate surgical condition.

In view of the numerous well-known works and monographs on this subject, it would be presumptuous on my part to attempt to do any more than bring up for consideration, the more important views of others, presenting them in the way they have been most useful in my own experience.

It is not within the province of this paper to discuss the pathology of extension of suppuration of the middle ear to the endocranial structures. Nevertheless it is important, to an understanding of the symptoms which arise, that the methods of invasion should be borne in mind.

Infection may take place on the one hand directly through dehiscences in the bone, to the middle or posterior fossa either the result of erosion, or developmental defects; or, secondarily, from operative work; on the other hand, the infection may travel through the lymph sheath of blood vessels, or nerves, or in the blood-current, from the tympanum, mastoid antrum, or mastoid cells, to the dura mater of either fossa, or to the sigmoid sinus. Even further you are aware that supplicative processes, having invaded the labyrinth, are in direct communication, through the duc tus endolymphaticus, with a pocket between two layers of the dura; and, through the aquaeductus vestibuli, directly with the subarachnoid space.

Naturally, symptoms of endocranial involvement must vary greatly with the method of invasion and the virulence of the organism causing it.

Endocranial lesions resulting from acute supplicative otitis are likely to be very rapid, from a most virulent infection, and very general in this invasion, owing to the fact that the infection has entered the cavernous through lymph sheaths of the blood vessels, or through the vessels themselves, rather than by erosion, or disease of the bone, as in chronic cases; and, as a result of the rapidity of the invasion, there has been no opportunity for resisting barriers to be thrown out. Symptoms of endocranial involvement of acute supplicative otitis, then, are more likely to be those of acute leptomeningitis or sinus thrombosis than of temporoparietal or cerebellar abscesses.

On the other hand, when intracranial invasion has resulted from chronic purulent otitis, the infection being slow and milder in character, due to necrosis and erosion of the inner plate, the early symptoms are likely to be those of a very local involvement of the dura (external pachymeningitis) with the production of an extradural abscess.

Should this persist, a fibrinous exudate may form on the internal surface of the dura, cementing it to the subjacent arachnoid and pia mater, thus preventing a diffuse leptomeningitis, as happens in the acute cases from the rapidity of the invasion. This barrier may also prevent further extension into the brain, but, continuing, a necrosis of the cortex may take place leading to the formation of an intradural abscess. Still continuing, the suppuration may extend to the deeper parts with the formation of a cerebral, or cerebellar, abscesses, according to the position of the primary invasion.

On the other hand, deep-seated abscesses may form without this progressive involvement of the different membranes, and the cortical substance, owing to thrombosis of small veins running in from the surface. In a similar manner thrombosis of the sigmoid sinus or jugular bulb may take place without the previous formation of a pericranial abscess.

In a general way, then, it may be said that endocranial involvement of acute purulent otitis is likely to be rapid, diffuse, and accompanied by violent systemic disturbances, in addition to the local symptoms; whereas the involvement from chronic purulent otitis is likely to be slow, distinctly localized, with slight systemic disturbances in the early stages, owing to the fact that its progress has been combated at every step by Nature's protective barriers. The final breaking down of these protective barriers, in the later stages, gives rise to violent symptoms of systemic disturbance, similar to those which occur early, in the acute cases.

The lesions to be considered are:

A. Inflammation of the meninges: (1) External pachymeningitis, or extradural abscess; (2) internal pachymeningitis or intradural abscess; (3) diffuse leptomeningitis; (4) serous meningitis.

B. Inflammation of the brain: (1) Encephalitis (a) circumscribed; (b) diffuse. (2) Brain abscess: (a) temporo-pasophenoidal; (b) cerebellar.

C. Inflammation of the sinuses of the dura: (a) lateral: jugular bulb, jugular vein; (b) superior and inferior petrosals: cavernous.

D. Erosion of internal carotid artery.

In considering any supplicative intracranial lesion it is well to bear in mind the fact that many of the symptoms arising from it are purely mechanical in origin, while others are the result of the toxemia produced by the infection.

Any meningeval inflammation has three characteristic symptoms: headache, which is severe and persistent; vomiting, which takes place without nausea and at unexpected times; constipation, without abdominal distention or pain, which can not be relieved by cathartics or purgatives. These symptoms are due largely to an alteration of adjustment of intracranial tension, although absorption of toxins has some part in them.

The accompanying symptoms, of mechanical origin —due to pressure of the effusion on the cortical centers beneath—vary according to the location, extent, and nature of the inflammation, together with the degree of irritability of the patient. Such symptoms have been classified by Ballance as follows:

1. Psychic symptoms: Irritability, change of disposition.
5. Finally, symptoms due to exhaustion and death of nerve cells: Paralyses, anesthesia, coma.

Fever, anorexia and emaciation, the other accompanying symptoms, are due to the general infection rather than to the meningeval involvement, and may be very pronounced or very mild, depending on the virulence of the organism producing them.

In a case of purulent otitis, or mastoiditis, acute or chronic, either before or after operation, the occurrence of severe and persistent headache; repeated vomiting, without nausea; and obstinate constipation, accompanied by fever, together with one or many of the detailed symptoms of cortical irritation, would point unmistakably to meningitis.
The problem then presented would be to determine whether the inflammation was of a serous, or purulent, or tuberculous type; whether localized or diffuse; whether associated with brain abscess, encephalitis, or sinus thrombosis.

Much light has been thrown on these questions by examination of the cerebrospinal fluid obtained by lumbar puncture. In diffuse purulent leptomeningitis, the fluid is under high pressure, opaque, and contains large numbers of leucocytes and abundant bacteria. In serous meningitis, it is under high pressure, contains very few leucocytes, and no bacteria. In tuberculous meningitis it is under very high pressure, and contains tubercle bacilli. At the Babies' Hospital it is not unusual to draw off 140 to 150 c.c. of fluid. In 76 cases of tuberculous meningitis the bacillus was found 74 times in the cerebrospinal fluid. In acute leptomeningitis from 30 to 40 c.c. of fluid are withdrawn, and the infective organism is always found. Diffuse purulent leptomeningitis which has an acute onset, and rapid course; while tuberculous meningitis has an insidious onset, a slower course, and a period of remission. Acute basal meningitis has a slower course than diffuse purulent leptomeningitis. It occurs in infants up to one year of age. Tuberculous meningitis occurs from 2 to 7 years, and purulent leptomeningitis at any age.

Optic neuritis is an early symptom in leptomeningitis and a late one in tuberculous meningitis. Blood counts show a high leucocytosis (30,000 to 40,000) and high polymorphonuclear count (75 to 90 per cent.) in diffuse leptomeningitis, while in tuberculous meningitis, it is likely to show a low leucocytosis (10,000 to 15,000) and a low polymorphonuclear count (60 to 70 per cent.) Meara has reported 37 cases in which 81 per cent. showed a leucocytosis of over 13,000, ranging as high as 38,000 in clinically uncomplicated cases. It is evident from this that the blood count is not as valuable a differential factor as microscopic examination of the cerebrospinal fluid for the tubercle bacillus.

**EXTRADURAL ABSCESS, INTRADURAL ABSCESS, CEREBRAL ABSCESS, CEEREBELLAR ABSCESS**

The symptoms arising from localized collections of pus within the cranium, like those from meningeal inflammation, are best appreciated when considered from the points of view of (a) the systemic disturbances due to the toxin; (b) the mechanical disturbances resulting from increased intracranial tension; and (c) those resulting from pressure on or destruction of definite areas of the brain.

Thus, the mere presence of pus, depending on the virulence of the organism producing it, and the amount of poison taken into the circulation, might produce moderate or high temperature, chills and vomiting. The mechanical pressure might alter the intracranial tension sufficiently to cause slow cerebration, slowness of pulse, vomiting of a cerebral type (at unexpected intervals, and without nausea), and obstinate constipation. Pressure on or destruction of centers in the motor area might cause convulsions, on the one hand, or paralysis on the other; the same action in other areas might cause a paresis or loss of certain special senses. Thus it frequently happens that large collections of pus may at times take place extradurally, or intradurally, or in an area of the brain not occupied by important centers or tracts, and, being well walled off, may remain for long periods without giving rise to any characteristic symptoms. Such a collection of pus is not infrequently encountered during the course of a mastoid operation, the symptoms, previous to operation, having been so slight that the abscess was unsuspected.

In other cases, the suspicion of an epidural abscess or intradural abscess with connecting fistula is aroused by the unusual and persistent discharge of a quantity of pus from the tympanum which seems too large to have come from the middle ear and mastoid cells.

Many cases, however, in which there is no fistulous outlet, present characteristic symptoms. High fever, definitely localized pain and tenderness (often far back in the region of the mastoid emissary vein) and stiffness of the neck, are indications of extradural pus under tension. Further symptoms of intracranial pressure may be present from the pressure of extradural abscess only, and all or a part of the graver symptoms of leptomeningitis, meningitis or brain abscess may appear.

It has frequently happened that graver symptoms have cleared up after the evacuation of an epidural collection of pus. There is no certainty from the symptoms, however, that such a case is not accompanied by an intradural abscess, or a serous or purulent leptomeningitis, or brain abscess, or sinus thrombosis. In such cases, lumbar puncture, and a blood count are of value in differentiation, in the manner pointed out.

Unless a brain abscess has actually destroyed important areas, or is large enough to interfere with their function, by compression, the location can only be conjectured from attending circumstances. It must be borne in mind that even a large abscess may lie in certain parts, without giving rise to localizing symptoms, owing to the fact that the space for the pus has been made by actual destruction of brain tissue. The abscess formed by the disseminated brain substance occupies the same space, and therefore gives no evidence of its presence by pressure symptoms. Such accumulations have often become encapsulated and remained for years without sufficient symptoms to bring them to the attention of a surgeon. So-called latent abscess, however, is latent only in the sense that no violent symptoms are present, and careful examination of pulse, temperature, ocular fundi, the functional power of groups of muscles, reflexes, static equilibrium, etc., would have brought to light sufficient evidence, in many of these cases for a diagnosis.

Remembering that brain abscess is a disease of an organ of most complex functions; that it is secondary to many serious conditions which themselves present a variety of symptoms which might be due either to the primary disease, or some other complication; that wide variations in the virulence of the invasion are possible; it is easy to understand that the clinical picture will be a variable one.

Ballance adopts the five types of clinical evolution described by Brissaud and Souques:

1. A subacute evolution, in which three stages are evident: (a) The initial stage of septic infection characterized by headache, vomiting and fever, lasting 4 or 5 days; (b) the second stage of remission in which there is a sudden or gradual abatement of the active symptoms; this period of calm may run over a very long course; (c) the third, or paralytic stage, comes on suddenly with or without a convulsion. Come and death may ensue in a few hours, from rupture of the abscess or, there may be recovery from the apoplectic seizure, followed by symptoms of rapid extension.
2. The evolution with severe general infection which is rapidly fatal. High fever with acute delirium are prominent symptoms, marking the more characteristic symptoms of brain abscess. This is usually mistaken for a malignant form of some specific fever unless other conditions have previously pointed to possible brain abscess.

3. Evolution with complete latency until the final attack of coma: In this variety, sudden death occurs without previous suspicion of brain abscess until revealed at the autopsy. It is in this "latent stage" that the suggestion was made that some symptoms might have been found if carefully sought after.

4. In the fourth type the evolution is just like that of a brain tumor. Abscess produced by infection of low virulence, and symptoms are similar to a tumor of the same size in the same region.

5. The fifth type of evolution is the remittent type: Here the clinical evolution is in two acts, separated by an "interlude" of greater or lesser duration. The first act is marked sometimes by an attack of mania, sometimes by acute delirium. Then all quiet down and the patient seems cured. But after a few weeks, a few months, or even a year, follows the second act, which is commonly quickly fatal.

It is perfectly evident that the array of symptoms of suppuration; local headache, tenderness on percussion, local increase in temperature, etc., while they have some bearing on the location of the abscess, are not characteristic of any particular type of collection of pus. One can not say whether it is extradural, intradural, or in the brain substance. So, too, the symptoms of pressure, headache, dizziness, vomitting, loss of memory, delirium, slow pulse, optic neuritis, change in size of pupils, apathy, somnolence, etc., while indicative of intracranial lesion, are not characteristic of any particular type or location. Localizing symptoms when present are frequently most definite in pointing out the position of an abscess. Lack of time will prevent more than a brief allusion to some of the more important of these.

Balance calls attention to the following facts:

1. The cortical center for hearing may be in part or wholly involved, causing tinnitus, hyperacusia, or absolute deafness of the opposite (healthy) ear.

2. The cortical centers for taste and smell may be affected. Alteration or suppression of the sense of smell may occur in abscesses, involving the anterior extremity of the temporal lobes.

3. Sensory aphasia often occurs in abscess of the left temporo-sphenoidal lobe in consequence of the cortical centers for the mechanism of speech being on the left side of the brain. The auditory word center and the visual word center are the opposite of temporo-sphenoidal abscess. A temporo-sphenoidal abscess on the left side is therefore commonly more easy to recognize than on the right.

4. Paralysis of the opposite side of the body may be of cortical or internal capsule type. The march of the paralysis is different in the two cases. This paralysis is a frequent occurrence from pressure on the posterior end of the internal capsule, and may be associated, as might be expected, with hemianesthesia.

5. Paralysis of the third nerve on the side of the abscess. This is important. The paralysis is rarely complete. A stable pupil on the side of the suspected abscess clinches the diagnosis.

6. Paralysis of the "naming center." Certain clinical and pathologic observations point to the conclusion that the nervous mechanism by which the ideas of objects are correlated with their names is located in the left temporo-sphenoidal lobe.

Few of the early symptoms of cerebellar abscess recorded by various writers can be considered as pathognomonic of that condition alone, since they are so frequently found in other intracranial lesions. In later stages, when the abscess is large, or meningitis of the cerebellar fossa has developed, pressure symptoms appear which point toward that region. Yawning, slow opening and shutting of the mouth, and rigidity of the masseters have been frequently observed. Optic neuritis, while not characteristic, may come on early and be very marked. Pressure on the medulla at a low level produces a brachio-cephalic paralysis on the same side. If pressure be exerted on the pons on the same side as the abscess, a crossed paralysis would result; pressure on both sides of medulla and pons would produce a bilateral paralysis.

A number of cases have been recorded in which pressure on the medullary respiratory center has been so marked that respiration ceased while the heart continued to beat. I have seen two such cases in which artificial respiration prolonged life for hours. Evacuation of the abscess in such a case might be followed by restoration of the function of the respiratory center, and recovery.

Two symptoms which may appear early are of great significance, viz: rotatory nystagmus, and cerebellar ataxia. The disturbances of equilibrium may not be differentiated from those arising from suppurative labyrinthitis, but Neumann has pointed out that there is a marked distinction in the nystagmus. The nystagmus from progressive labyrinthitis grows weaker as the disease progresses and finally disappears, while that from a cerebellar lesion increases as the disease progresses and reaches a degree never attained in labyrinthitis.

In addition to this, a point of great value appears from the fact that, in a case in which at first the nystagmus was more pronounced when the eyes were turned toward the well side, if the phenomenon later was suddenly reversed a diagnosis of cerebellar lesion could be made with certainty, and a labyrinthine cause excluded.

INFECTIVE THROMBOSIS OF THE SIGMOID SINUSES; JUGULAR BULB; JUGULAR VEIN

As has been pointed out, an infective thrombosis may occur in the sigmoid or jugular bulb by direct infection through disease of the overlying bone cells, and lining membrane, or by thrombosis of small vessels within the tympanum or mastoid process which lead directly into the sinus or bulb.

The first type is more likely to occur in chronic or subacute purulent otitis, the second from acute purulent otitis, of a virulent character.

Naturally, the chronic cases are more likely to be associated with other intracranial lesions—epidural abscesses; cerebral or cerebellar abscesses; encephalitis, or meningitis—which would increase the difficulty of diagnosis.

In a typical case without other cerebral complications the symptoms are very characteristic. A chilly sensation or distinct rigor, lasting for a few minutes, followed by a rapid rise in temperature to 104 or 106 F. The pulse is small and rapid; the facial expression is anxious; there is intense local pain, there is complete anorexia, great thirst, and sometimes frequent vomiting. The tongue is thickly coated, and the breath has the characteristic fetid odor of sepsis. The patient is perfectly rational, perhaps somewhat irritable. The
rapid rise in temperature is succeeded by almost as rapid a fall to normal, or often subnormal, accompanied by profuse sweating and prostration. According to the virulence of the infection, this is repeated in a few hours, or possibly not for a day or two. Irregularity has seemed to me to be a strong diagnostic sign in contradiction to malarial or typhoid conditions, though at times there has been a diurnal vacillation from the first. Usually the chills and fever, followed by the sudden remissions, appear at increasingly shorter intervals, with more marked rigors and wider variations in temperature. Death may occur in a few days from acute septicemia, the patient being perfectly rational up to the last minute.

Left alone, however, signs of metastatic processes in the lungs usually manifest themselves within a week or ten days. Pain in one lung is followed within twenty-four hours by rapid, superficial breathing, accompanied by cough and expectoration of rusty sputum. The temperature may remain persistently high and death may supervene from septic pneumonia, or gangrene of the lungs may take place and death occur at a much later period from pyemia. Should this occur, the constipation, which has usually been present in the first stages, will have given place to a foul diarrhea. Metastatic abscesses in the joints are likely also to appear in the cases which assume the pyemic character.

A certain number of cases will assume a typhoid character very early; there will be no wide vacillations in temperature, and no chills, except possibly at the very beginning. In such cases the mentality is not clear as in the first type. Diarrhea, abdominal pain, and meteorism are present, and skin is likely to be broken by a chill red rash. The presence of otitis or mastoiditis may be of great value in determining a diagnosis. In such a case, an absence Widal reaction, high leucocytosis and polymorphonuclear count; and, presence of streptococci would be of great value differentially.

Then there is a class of cases in which symptoms of meningitis are present early, in connection with the signs of infective thrombosis. Rigors are unusual in the early stages, and the temperature is persistently high. Many of the pressure symptoms and symptoms of cortical irritation of meningial inflammation are present; tonic and clonic spasms of groups of muscles; mud; early optic neuritis; etc.—in fact the symptoms of meningitis may entirely mask the involvement of the sinus until metastatic processes appear. Early coma, and death, however, are most likely to supervene.

All these types are easily recognizable, but there are many cases in which the early symptoms are not so typical. The signs of infective thrombosis may be moderate vacillations in temperature, without chills and without prostrating sweats. Or in a less virulent case the temperature at first may not show much vacillation, remaining moderately high for a few days without other indications.

On the other hand, wide vacillations in temperature may be present, in infants, without the presence of infective sinus thrombosis. At the Babies' Hospital, I have repeatedly observed infants who had influenza or bronchopneumonia, in which vacillations of from six to eight degrees took place for many days; in one case for weeks. The fact that such vacillations took place in those who did not have discharging ears, as well as in those who did, deterred me from investigating the sinuses and jugular bulbs in those with the purulent ears. It is gratifying to report that these children did not die of infective bulb thrombosis. In one child, with very marked purulent discharge from both ears, the vacillations were so very wide that, in the absence of pulmonary signs, I was tempted to perform an exploratory operation. The patient finally died, and I then had the opportunity of investigating the sinuses and jugular bulbs. They were both perfectly healthy.

It seems, therefore, that there are a certain number of cases in which an early diagnosis is very difficult if not impossible. It is possible that increased knowledge from examination of the blood may enable us to make a positive diagnosis in the future at an earlier moment than now. With certain doubtful clinical symptoms present a blood culture showing the presence of streptococci would lead the physician to make a diagnosis of probable infective sinus thrombosis, providing there was no other evident cause for the streptococci. So, too, a very high or low leucocytosis, with a high polymorphonuclear percentage, taken together with other symptoms, even though not typical, might lead to a diagnosis of septic thrombosis.

It seems unnecessary for me to discuss extensions of involvement of other sinuses since they are given additional symptoms. Intervention of the jugular vein may give rise to tenderness along its course, and even to a cord-like feeling, so it is said, although I have never observed it. Infective thrombosis of the cavernous sinus occurs from suppurative otitis only secondary to that of the superior or inferior petrosal sinuses. In addition to the symptoms of septic poisoning, thrombosis of the petrosals is likely to cause epistaxis, swelling of the veins over the temple, and thrombosis of the retinal veins.

Extension to the cavernous sinus causes an engorgement of the vessels of the orbit, with consequent ophthalmie, photophobia, disturbances of vision, paralysis of the motor oculi, blepharitis, and trochlear nerves, piosis and edema of the eyelids and trigeminal neuralgia. Thrombosis of the cavernous sinus of the other side is likely to follow rapidly, accompanied by similar symptoms in the corresponding eye.

While erosion of the internal carotid artery is extremely rare, a few cases have been reported, all of them fatal. The condition can only occur in chronic cases with extensive necrosis. The diagnosis is evident from the gushing of bright red blood from the aortie with great force. This is temporarily stopped by pressure on the carotid in the neck.

In the reported cases death has occurred in from five minutes to thirteen days, even in those patients in whom ligation of the common carotid was performed.

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To Limit Use of Saccharine.—The Scientific American says that a conference was held at Brussels for requising the use of saccharine in food products or drink. While the drug has 400 times the sweetening power of sugar, it is dangerous to health and it is desired to prevent the fraudulent use of the product. Delegates were present from all parts of Europe, and it was decided to hold a new conference on the subject at Geneva during the next spring. A resolution regulating the use of saccharine was adopted. Fiscal agents will look after the manufacture and destination of the product and see where it is employed. In many countries of Europe it is brought in by contraband, and in one case there was found in a vessel in the Russian port of Riga about a ton of saccharine, which would replace 400 tons of sugar.