In many cases it may be necessary to use local and general electricity, or both, to hasten the recovery of the motor paresis and the secondary trophic changes, and counteract and relieve the neurasthenia that generally accompanies the chronic sciatica before the operation, or which usually follows it.

The success of nerve stretching: In chronic sciatica, in 168 cases collected from various sources 153 have been cured, 15 cases temporarily benefited. In other neuralgias, out of 189 cases, 132 have been cured, and 33 permanently benefited. Bryant gives 70 operations of nerve stretching for sciatica; statistics indicate that in 60 the patients were either cured or greatly relieved, and that in the majority of cases the relief was permanent. The writer is informed that Mr. Calendar and other English surgeons have had about 60 per cent. of recoveries from nerve stretching.

As an extreme measure and a last resort, nerve section has been done for chronic sciatica and other neuralgias. Neurectomy has been done by Sapolino, Brinton, Morton, Hodge, Vance, Golding-Bird and Wyeth with at least temporary benefit. Cures have been reported of neuralgia that was cured by resection when nerve stretching had failed.

THE ORIGIN OF PUS.

Read in the Section of Dental and Oral Surgery at the Fortieth Annual Meeting of the American Medical Association, June, 1889.

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OF NEW YORK.

It is the gift of genius to foresee discoveries even for centuries. Such a genius was John Hunter, of London, who toward the end of the last century, merely upon the ground of speculation and ratiocination, made the assertion that inflammation is nothing more than a return of the tissues to embryonic condition. And our present knowledge of the process of inflammation and suppuration is a strong proof, supported by careful researches of good pathologists of Germany and England, that Hunter's theory was the correct one.

Since microscopy became a science (a period covered by half a century) the views concerning the intimate nature of the inflammatory process have been greatly at variance. These views largely depend upon the general ground taken by pathologists in reference to the pathological processes at large. We have three marked phases in the development of pathology within the last fifty years. The first is the standard of humoral pathology promulgated by the late Rokitsansky, of Vienna. The second is the phase of cellular pathology established mainly by Virchow, of Berlin. The third phase, still in vogue with many pathologists, is the doctrine of emigration of colorless

blood corpuscles or leucocytes, as propounded by the late Cohnheim, of Leipsic. I personally went through all these phases and am willing to admit that each meant progress, and still I have grown old enough to convince myself that neither of the views mentioned contained the whole truth. I have witnessed researches going far to prove that it is mainly a combination of the previous views that makes us approach the truth. I say approach, for there is no reason to deny that coming decades will bring us forward still more in our understanding of the intricate nature of the process termed inflammation and suppuration. During the past five years bacteriology has held sway of the minds of the majority of the pathologists, proving what was just stated. To-day no one will be rash enough to neglect the influence of bacteria in producing suppuration. And have we not learned through excellent observers that it is not the microorganisms themselves, but rather their chemical products, the ptomaines, that play an important rôle in the causation of suppuration and possibly of inflammation. I here allude to the latest researches of Seber, of Goettingen, who maintains that it is a ptomaine, called by him *phlogestein*, that stands in causal relation to inflammation.

The task I have undertaken is to bring before you a review of the theories held in the last fifty years, closing my historical remarks with a description of what I consider to be the truth to-day. The review of such a history is instructive in many respects. It teaches us that none, be he of the most gifted talent, is able to emancipate himself from preconceived ideas and accepted notions. It furthermore teaches us that we are the subjects of general theories extant for the time in which we make our investigations. It teaches the great lesson of modesty and humility. If a man of my age must confess in the seventh decade of his career that he has been misled in his younger days by books and teachers and must try hard to unlearn what he thought he knew before, it is certainly proof of human frailty, and goes far to prove that we are obliged to consider the truth to be truth only *pro tempore*, viz.: so long as we do not know better. Our successors will surely step over our shoulders and will estimate our work only by considering the amount of time, honesty and acuteness of mind spent in bringing forth new facts and new revelations.

In the fifth decade of our century humoral pathology was thought to hold the full truth in the explanation of inflammation. The older of us will remember which facts the humoral pathology was based upon. The web of the foot of a living frog was expanded over a cork ring and the web touched with an irritating agent, such as a drop of ammonia, an acid, or with a pointed, red-hot iron, and the subsequent changes observed under the microscope with the comparatively low powers

at the disposal of investigators of the time. They saw around the irritated portion of the web an undulation of the currents of the blood within the vessels, shortly afterwards a slacking of the current, and still later a standstill of the stream. The last phenomena they called "stasis," and this stasis was thought to be the essential feature of the inflammatory process. Many and animated controversies arose over the question, what is the stasis due to? Most observers agreed that a paralysis of the capillary blood-vessels, after a few preceding contractions, was to be considered the cause of stagnation. At the same time an inundation was seen to take place in the affected tissues with a liquid which of necessity must have come from the general blood column, and was termed "exudate." According to the nature of the exudate different varieties of inflammation were set up, such as the "serous," the "fibrinous," the "albuminous," and should blood have been admixed with the exudate, the "hæmorrhagic." Corpuscular elements seen in the affected territories and apparently suspended in the *exudate* were thought to have originated from the exudate itself, therefore the pus corpuscles would have originated from the exudate, the latter from the blood, hence the definition of pus, "dead blood." No stress was laid upon the structural changes of the affected tissue itself, except so far as the exudate saturating this tissue was concerned. The ultimate cause of inflammation and suppuration was sought in chemical mixtures of the blood, termed dyscrasia, which means bad mixture of the blood. If a person became affected with lobar pneumonia the cause surely was surplus of fibrin in the blood, or fibrinous dyscrasia. If a person produced a number of abscesses in his organism the cause was denominated purulent dyscrasia. All diseases were, in this dyscrasic view, essentially diseases of the blood. The blood in its mixture caused the diseases simply by being overloaded with obnoxious or effete material.

The man who dug the grave of humoral pathology was Virchow, in the beginning of the sixth decade of the present century. To him the phenomena of alterations in the circulation of the blood were of secondary importance. The stasis he would not admit as a cardinal symptom of inflammation for it would mean rather death of the tissue, gangrene or necrosis. The exudate also played but a secondary rôle, serving only as a pabulum to the living corpuscular elements of the tissues, the so-called *cells*. Inflammation was, in Virchow's opinion, a structural change of the affected tissue, mainly morphological changes of the cells themselves. The cells being the seats of life would attract and, as it were, imbibe the exudate, swell up, divide and come to a state of proliferation, in which a number of cells would arise from an original single cell, and the large number of newly-formed cells would replace the intercellular

substance lost by liquefaction. A formation of cells out of a previous liquid or semi-solid exudate was declared to be impossible, since all newly-formed cells must have arisen from preëxisting cells. Dyscrasias were done away with. The main causes of inflammation were peculiarities of the tissue itself, and a certain weakness of the tissue was proposed to explain the predisposition to inflammatory processes. The word suggested for this supposed local weakness of the tissue was "diathesis." If a man suffered from articular rheumatism the cause was said to be "rheumatic diathesis." Tuberculous persons were predisposed to cheesy degeneration simply because they were afflicted with *tuberculous* diathesis. In the same sense hæmorrhagic purulent diatheses were spoken of. The pus corpuscles were without exception an offspring of the previous cells of the tissue, and in this sense pus was considered dead tissue. Cellular pathology has made a very strong impression on the minds of observers. Even the stalwart humoral pathologist Rokitsansky yielded to the novel views and accepted the doctrine that no cell will grow in an exudate unless previous cells be there. Cellular pathology even in our day governs the minds of most pathologists over the civilized world. Proliferation of the cells accounts for all tissue changes, either newly-formative, the so-called hyperplasia, or destructive, that is, supuration. Proliferated cells being present in either of these instances, cellular pathology is at a loss to explain why a certain tissue, owing to proliferation of its cells, becomes augmented and hyperplastic in one instance and is destroyed by supuration in the other. Still, in spite of all weaknesses of this doctrine, we must consider it an advance in comparison with the teachings of humoral pathology. The cell being supposed to be the only center of life, inflammation and supuration were for the first time considered as the phenomena of life, and the idea began to dawn in the minds of pathologists that inflammation and suppuration were morbid processes occurring in tissue elements endowed with life, the so-called cells.

The third period arose in the middle of the seventh decade of our century, when Cohnheim observed a migration of colorless blood-corpuscles through the walls of the capillaries and small veins of an exposed and expanded mesentery of a frog. Several years before S. Stricker, in Vienna, observed the red corpuscles pass through the walls of the capillaries of the nictant membranes of the live frog, so-called *diepiesis*. Even in 1848 the emigration of leucocytes had been seen in England by Walker and Wallace. The facility with which the emigration of leucocytes could be seen under the microscope has induced many German pathologists to accept the view of Cohnheim, that inflammation and suppuration are but an emigration of colorless blood-corpuscles.

These accumulating in a tissue whose cells would remain inert and whose intercellular substance would become liquefied and destroyed, furnish a representation of both inflammation and suppuration. With this view there was nothing alive in the body but the leucocytes. At first Cohnheim denied the participation of the so-called stable cells in the process of inflammation, but later he admitted that in reparative inflammation the tissue cells do proliferate and furnish their share for the benefit of newly-forming tissue, exactly in the sense of cellular pathology. That pus corpuscles should be emigrated colorless blood-corpuscles was intelligible, as were isolated corpuscles nearly identical in appearance. How new tissue could form from leucocytes remained a deeply-shrouded mystery, as no observation has as yet proved that through coalescence of leucocytes new protoplasmic masses and new tissues can arise. The observation of Zeigler, of Tübingen, that multinuclear bodies, so-called giant cells, are invisible between thin glass plates introduced under the skin of an animal lacks the proof that such giant-cells arose by coalescence of leucocytes. On the contrary, later observations made especially upon deciduous, replanted and implanted teeth, go far in proving the giant-cells to be an offspring of the myxomatous granulation tissue, sprouting in all directions, filling the bay-like excavations on the deciduous and other teeth and carrying blood-vessels into places where there had been none before, leading to the vascularization of a new tissue, seen, for instance, in sponge grafting.

S. Stricker immediately sought to overthrow the teachings of Cohnheim. In his investigations he used almost exclusively the cornea (chiefly of frogs, cats and rabbits); in which he brought inflammation by introducing a thread. The changes were faithfully watched up to the formation of abscess around the foreign body. The observations enabled him to prove the established views of cellular pathology to be correct, namely, that these so-called cells divide and subdivide, but also that the coarser offshoots of the cornea corpuscles split up, and by division produce new corpuscular elements. Stricker also held, up to 1874, (Ashurst's International Surgical Cyclopædia), that pus corpuscles were products of proliferation of the cornea corpuscles and their coarser offshoots. Unfortunately he calls all the products of cells originating from proliferation of previous cells pus corpuscles. Indeed it was impossible to discriminate between inflammatory corpuscles and pus corpuscles, since all of these appeared isolated under the microscope. Clinically it is well established that every inflammation does not terminate in suppuration. Particularly do we know of a termination which instead of causing destruction of an affected tissue, as suppuration does, brings about a new formation, an increase

of the bulk of the tissue, the so-called hyperplasia. How shall we explain such marked differences by the theory of cellular pathology, which claims all corpuscular elements are isolated from the start? However, since 1880 Stricker is a convert to the views of C. Heitzmann, established in 1873, which doctrine I advocate, having studied specimens, illustrative of inflammation and suppuration, under the microscope in that investigator's laboratory.

Let us recapitulate in a concise way the views held by the said C. Heitzmann concerning the normal or physiological structure of tissue. He holds that there is no isolated or individual cell in any variety of tissue in either the animal or vegetable organism. Such individual corpuscles are met with only in the fluids of the body, such as the blood, where they are named red corpuscles; in the lymph, called lymph corpuscles; in the saliva, called salivary corpuscles; in the sperm, called spermatazoids, etc. Neither the blood nor the lymph, nor any of the fluid, semi-solid secretions deserve the name of a tissue. Tissue we call a continuity of not only organized material, but a material endowed with all the properties of life, such as mobility and the capacity of reproduction. Tissues hold at certain intervals protoplasmic bodies, so-called cells, the vast majority of which are in continuity with the living matter held by those substances, previously termed "intercellular" and to-day they are known by the names of basis and cement substance. The greatest amount of living matter is present in the centers of protoplasmic bodies, where they appear as nuclei and nucleoli. The surrounding protoplasm, in full development, holds the living matter in a reticular arrangement and such a reticulum is traceable throughout the surrounding basis substance, hitherto considered as entirely inert. Thus an uninterrupted connection is established from one "cell" to another by the intervening bridges of living matter. This same arrangement being present throughout all tissues of the animal organism, a continuity of all the tissues composing such organism is established. Recent researches of botanists go far to prove that even in the plants there exist no individual cells, but the intervening cement substance, or cellulose, is traversed by minute bridges of living matter, rendering the plant an individual from the tips of the leaves to sporacles of the rootlets. Recent observers in animal and vegetable microscopy have drawn attention to the fact that by means of certain reagents the nucleus will be split up into loop-like threads, a process which they consider precedes the division of the nucleus and indirect division of the protoplasm. This process is termed karyokinesis, or mitosis—"fibrillation," "thread making." This observation is had only with certain reagents and is not visible in the fresh specimen or in one preserved in a chromic

acid solution. Suspicion naturally arises that the loop-like figures of the nucleus are artificially produced and thereby their connections are rendered invisible. The threads forming loops are called "chromatin," because readily stained by aniline dyes, whereas the intervening substance and the protoplasm itself stain but little, and therefore are "acromatin." Facts rendered conspicuous by staining appliances only are of doubtful correctness. The fact that there are threads and loops in the star-point form arrangement in the nucleus rather proves the latter to be made up largely of living matter, which is known to change shape any moment. Coarse formations of living matter readily stain with aniline dyes, whereas delicate formations of the same substance will not stain. The connections between the loops of the nucleus and the surrounding radiating reticulum of the protoplasm are plainly visible in fresh specimens, and also those preserved in liquids which we know will not alter the structure of protoplasm, such as a solution of chromic acid of one-tenth to one-half of 1 per cent. Alcohol as a preserving fluid is far inferior, owing to the shrinkage it effects.

Let us analyze the construction of dentine (most important to dentists, since it builds up the main mass of the tooth) in the light of this novel doctrine, for which the late Louis Elsberg, one of its most enthusiastic advocates, suggested the term bioplasson theory. Dentine has no cells, but is composed of a dense, firm basis substance (not cartilaginous as formerly thought but glue yielding, similar to that of bone tissue) thoroughly infiltrated with lime salts. This basis substance is traversed by the canaliculi which radiate from the surface, occluding the pulp chamber toward and join the cement and enamel. Each canaliculus holds in it a delicate fibril, the so-called Tomes fibre, around which exists a minute space filled with a liquid, obviously the carrier of nutrient and denutrient substance. Fine thorn-like offshoots were known, especially in transverse sections of dentine, to emanate from the fibril, traverse the surrounding space and fade upon approaching the wall of the canaliculus. Now in the light of the bioplasson doctrine the Tomes fibres are formations of living matter. From them arise transverse conical offshoots penetrating the walls of the canaliculi and in connection with a delicate, nearly rectangular reticulum traverse the whole of the basis substance and connect the tenant of one canaliculus with the neighboring tenants directly, and therefore all the others indirectly. The presence of this reticulum was first established by C. F. W. Bödecker, in 1878, who saw light rents in the basis substance and assumed them to hold living matter without being able to directly prove its presence.

This proof has been quite recently furnished by Wm. Carr, who after decalcification of the dentine

by means of a 6 per cent. solution of acetic acid rendered the reticulum visible by staining with a chloride of gold solution, and also osmic acid. These last results have not as yet been published by their observer. I have seen his own specimens through lenses of 1,000 to 1,200 diameters with good immersion, and am convinced of the presence of the reticulum throughout the dentine.

Thus we understand how dentine can grow and be nourished, and realize it is a tissue endowed with sensibility prominently at those places which abound with living matter, such as the periphery toward the enamel and the cementum at the neck of the tooth. Therefore we can appreciate that living dentine, if irritated by a foreign body, such as a mass of gold filling, or chemically by acids, will react upon this injury, become inflamed and produce a new tissue, called osteo-dentine, which sometimes is more compact than the original. This process of the inflammation of the dentine, called "eburitis," was carefully studied by Dr. Bödecker a few years ago, and although he has not yet completed his studies upon teeth which had been filled with different materials for months and years, every intelligent dentist knows that such a reaction exists. This fact is utilized by all of us in introducing highly-irritant agents, such as oxyphosphate of zinc, into otherwise poorly calcified, so-called soft, teeth. The result after some months is a compact wall of the cavity made up of osteo-dentine, or secondary dentine, and better fitted to tolerate a gold filling than it was before this temporary filling. How can we understand these changes and the toleration of highly conductive filling, unless the tooth substance be a veritable living tissue throughout its extent? Whenever irritation is brought to bear upon a living tissue reaction will follow, and this is inflammatory process. The first that occurs is liquefaction of the basis—or cement—substance, probably induced by the presence of an acid, mainly lactic, thus the living matter previously concealed (held) in the basis—or cement—substance becomes liberated and the protoplasmic form of the basis substance reappears. This condition has directly been observed by S. Stricker on the cornea of frogs, as before stated. He saw the basis substance in motion, changing the configuration of its living matter, under the microscope, much like clouds changing on the face of the sky. Next the protoplasm furnishing the substratum of previous basis substance becomes split up into small bodies, known as medullary, or embryonal, or inflammatory corpuscles. Any portion of living matter of such indifferent bodies may grow to the size of a nucleus, owing to the presence of an excess of pabulum beyond the physiological requirements of these bodies, an excess essential to the inflammation. Not only the original "cells," but the intervening basis substance will participate in the formation of new

elements or inflammatory corpuscles. Here is the distinguishing feature between modern and antique pathological views, for according to the latter only the "cells" themselves were considered active and capable of proliferation. The sum total of the newly-formed inflammatory corpuscles is known under the term inflammatory infiltration, which means that a certain amount of a tissue, be it connective, muscular or nerve tissue, is replaced by and transformed into newly-appearing protoplasmic bodies in the stage of indifference, that is, being purely protoplasmic tissue bodies without any distinctive character either as to origin or destiny. So long as the inflammatory corpuscles remain in continuity by their delicate offshoots they represent a tissue, though in a condition of indifference, or embryonal state. Such a tissue by new formation of a basis substance will either return to the previous normal state, terminating the inflammatory condition in "resolution," or, being considerably augmented itself, will produce a tissue much greater in bulk than the one originally inflamed and we will have hyperplasia. In neither instance will the inflamed tissue cease even for a moment to be tissue. The tissue which is the bearer of the blood and lymph vessels is the connective tissue, and connective tissue is the only bearer of these vessels. And here is the starting point of inflammation in almost every instance. This we understand from the fact of the rather low dignity and activity of the connective tissue. We look for the greatest reaction on irritation at the sources of nutrition, obviously the blood-vessels. Muscle, nerve and epithelial tissue react in rather a secondary manner on inflammation of connective tissue. Virchow proposed the name "parenchymatous inflammation," for the designation of the inflammatory process, which term may be retained if restricted always to a secondary manifestation upon the primary inflammation of the "interstitial" connective tissue. A gland, for instance, is a compound organ composed of parenchyma according to Virchow, which is the epithelial glandular tissue. This is surrounded with and accompanied by connective tissue bearing many blood-vessels. It is impossible that the glandular tissue could be the primary seat of the inflammatory process. This process will be present in the connective tissue first and more or less rapidly invade the glandular epithelial tissue. If the connections of the inflammatory corpuscles be severed we shall have a certain number of isolated medullary or embryonal corpuscles suspended in an albuminous liquid.

Pus is therefore the resultant of destroyed tissue, but quite unfit for the production of any form of tissue. How much the emigrated blood corpuscles contribute to the formation of pus may not yet be positively stated. However, we do know positively that upon the approach of suppuration

in a certain tissue its blood-vessels are destroyed without exception in the territory involved. The blood-vessels, by outgrowth of their endothelia, become at first solidified and afterwards split up into medullary and pus corpuscles, the same as all the other elemental constituents. Even the smooth muscles of an artery will partake in this pus-forming process. The amount of migrated leucocytes cannot be great, considering the loss of the vessels which have supplied them. Pus is destroyed tissue, first originating in a closed cavity bearing the name of "abscess," or, secondly, coming from the walls of physiologically shut cavities, termed "empyema," or, thirdly, springing from exposed tissue surfaces, viz., such as granulating surfaces of wounds, termed "pyorrhœa." In the two latter instances, namely, empyema and pyorrhœa, the emigration of colorless blood corpuscles plays a far more important rôle than in the case of "abscess." In granulating surfaces of wounds, especially, the source of the pus corpuscles must be sought mainly in the capillaries, which abound in the myxomatous granulation tissue, producing loops therein, upon the grouping of which depends the raspberry look of the so-called "proud flesh." Around an abscess a dense layer of fibrous connective tissue is formed very soon—beautifully displayed in alveolar abscess at the apices of diseased roots of the teeth. This newly-formed layer represents the productive activity of inflammation, being hyperplastic, fibrous connective tissue, more or less well supplied with blood-vessels. Old pathologists termed this layer "membrana pyogena," which signifies that the membrane itself produces the pus. This view was abandoned long since, and to-day the membrana pyogena is known to be a secondary formation in the appearance of an abscess, as it were, a protective wall to the healthy tissues. After the evacuation of the pus from an abscess or an empyema, either by spontaneous rupture of the covering layer toward the surface, or artificially by knife or caustic, the membrana pyogena changes its character and becomes the seat of an acute inflammation, which leads to the formation of a freely-vascularized myxomatous tissue. This is what is called "proud flesh," or granulation tissue. In all instances in loss of tissue by suppuration the final result is the formation of a scar. This consists of a dense fibrous connective tissue whose bundles freely interlace and which is, as a rule, scantily supplied with blood-vessels. It is an outcome of myxomatous granulation tissue and is covered with epithelium whenever the pus has been discharged through the skin or a mucous membrane, which themselves appear in a normal condition with an epithelial layer.

Pus, therefore, is a tissue disintegrated and broken up into indifferent or medullary corpuscles which are suspended in a liquid more or less

rich in albumen. How much the emigrated leucocytes share in the formation of pus corpuscles may not be determined. The pus corpuscles remain alive as long as the liquid surrounding them is sufficient for the support of their nutrition. Even fatty degeneration of the pus corpuscles, as seen in chronic abscesses, will not altogether deprive them of their vital phenomena, such corpuscles, if transferred upon a slide, remain amoeboid, and they die only by exposure to a low temperature, or after the addition of chemical reagents, or within the body by being deprived of their nourishing liquid. The last condition is noticed in cheesy degeneration of the pus so often met with in tuberculosis. A question now arises. What is the cause of the formation of pus? Ever since the famous experiments of Cohnheim and Counselman, who introduced vials filled with croton oil under the skin of rabbits, with anti-septic precautions, and after healing was complete cracked the vials, subcutaneously, thus producing an abscess, this is a much mooted question. A host of experimenters who have repeated these trials have taken views pro and con of the question. I consider the experiments of P. Grawitz and W. de Bary, published in Virchow's Archives, 1887, the most valuable testimony towards settling this question. They found that the subcutaneous injection of concentrated solution of chloride of sodium in rabbits and dogs was followed by œdema, a swelling of the facia, but no suppuration. It makes no difference whether the solution be mixed with a large number of staphylococci. Staphylococcus, with its three varieties, the staphylococcus pyogenes, aureo-albus and citreus, first discovered by Rozenbach, was thought to be essential to the production of pus. And even H. Knapp, of New York, maintained that a small quantity of Croton oil mixed with olive oil will not produce suppuration if introduced into the anterior chamber of the eye of the rabbit, whereas the same mixture if contaminated with pure culture of staphylococcus aureus will invariably be followed by suppuration under like circumstances. Grawitz and de Bary, on the contrary, have proven that not only the coccus named, but certain irritating reagents and different products of microorganisms, not necessarily the staphylococcus, different alkaloids, or ptomaines, are productive of pus. Solutions of nitrate of silver, if injected under the skin of dogs, rabbits, rats and mice, will be harmless if weak, not stronger than .005 of 1 per cent., but when 5 per cent. in strength will invariably be followed by abscess in dogs. Neither acids nor alkalies cause suppuration, except the liquor of caustic ammonia, which if introduced in full strength is followed invariably by suppuration. In neither of these instances were there any microorganisms discoverable in the pus freshly removed from the abscess, or transferred upon nutritive gelatine as a culture

medium. Oil of turpentine in rabbits and guinea pigs did not induce pus, even though injected in large quantities, but only inflammation; whereas in dogs oil of turpentine, which is a strong germicide, after subcutaneous injection was always followed by suppuration.

From these experiments it follows that chemical substances entirely free from bacteria and cocci tend to induce the suppurative process in different animals. On the other hand it was experimentally proven that in dogs and rabbits the injection of cultures of staphylococcus into the normal subcutaneous tissues will not induce inflammation or suppuration. It is necessary that a tissue first be irritated to a condition of inflammation, by traumatism or by chemical irritants, to have a soil favorable for the development of staphylococcus, which under these circumstances will produce an abscess. Lately P. Grawitz has shown that the subcutaneous injection of a ptomaine derived from putrescent organic material, called cadaverine, will always lead to the formation of an abscess. From the practical standpoint it is of the utmost importance to work aseptically, or in other words, with such degree of cleanliness that the introduction of microorganisms or their ptomaines becomes impossible. It is well established that even open fresh wounds if aseptically dressed with gauze saturated with a 1:1000 solution of corrosive sublimate and left alone for a few weeks will kindly heal without one single droplet of pus. Corrosive sublimate and carbolic acid are still most reliable germicides. We are not yet prepared to assert that microorganisms alone cause suppuration, since there are irritating chemical substances whose introduction into the body may likewise be followed by this process. Recent observers have maintained that there is a marked difference between a mere accumulation of leucocytes and an abscess proper. In the first instance there may be present a serous or fibrinous exudate entangled with a number of emigrated leucocytes, without a loss of tissue, without, therefore, suppuration. In the latter instance a certain amount of tissue is destroyed and directly transformed into pus. If this view be correct, it certainly strongly supports our present notions of the process of suppuration herewith laid before you in this résumé of my paper.

1. Inflammation is a disturbance of nutrition of a tissue causing a recurrence of the embryonal condition of the tissue involved.

2. The embryonal condition is established by the breaking up of the tissue into those medullary or indifferent corpuscles which, at an early stage of normal development, have built up the tissue.

3. The medullary corpuscles arise not only from the protoplasmic bodies of the tissue, the so-called "cells," but also the intercellular or basis substance is productive of such corpuscles,

as these have shared in the formation of basis substance in the process of normal development.

4. The medullary or indifferent corpuscles will still represent a tissue so long as they remain interconnected and continuous. By a simple reappearance of basis substance the most favorable termination is established, so-called "resolution."

5. If the inflammatory or medullary corpuscles have largely augmented, a number thereby remaining in original connection, the result will be productive, viz.: with a newly formed tissue of increased size, a so-called "hyperplasia."

6. If the inflammatory corpuscles springing from previous "cells," basis substance and blood-vessels break asunder and become isolated, they will be suspended in an albuminous liquid, they will henceforth represent pus corpuscles.

7. Pus, therefore, is a destroyed tissue broken up into its constituent elements, and as such unfit for production of a new tissue, although the single pus corpuscle will remain alive and amœboid almost indefinitely as long as they are sufficiently nourished.

8. The emigration of colorless blood corpuscles certainly participates in the formation of pus and in the purulent discharge of proud flesh or granulation tissue, and is probably the main source of the pus.

9. Suppuration is caused by the presence of certain microbes, mainly the three varieties of staphylococcus, only when a previous inflammation be present in the tissue, furnishing a favorable soil for the development of the before mentioned microbes.

10. Staphylococcus is not the only antecedent of suppuration, it having been proved by experiments that the introduction of certain chemical agents, unfavorable to the development of microbes, may likewise be followed by suppuration.

THE CLIMATE OF SOUTHERN ALBERTA AND ITS RELATION TO HEALTH AND DISEASE.

Abstract of a Paper read at the Annual Meeting of the Canada Medical Association, Banff, Alberta, August 16, 1889.

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It appears to me that no apology is necessary in introducing the subject of this paper. It is only a few years since the opening of our great National highway brought the Northwest Territories into touch with the rest of the world; and only a few years further back since these vast plains and mountains, which are now so quickly becoming the homes of civilized man, were regarded as an inhospitable desert, fit only for the buffaloes which roamed their solitudes and the Indians subsisting on the chase. The past five years have been epoch-making so far as our great

West is concerned. Coincident with, and following on the building of the Canadian Pacific Railway, a flood of light began to illumine the Eastern intelligence, and as a consequence settlement has steadily and in a gradually increasing stream poured into the Territories.

It will be readily understood that a country almost equal in area to Russia must have many diversities of climate. As I cannot undertake to speak for the whole of the Northwest, I have limited my remarks to that part with which I am best acquainted, viz.: that strip of country lying along the eastern base of the mountains, and more particularly the southern part of this—bounded, say, on the north by the C. P. Ry. I make the eastern limit a line drawn north and south through Lethbridge, although for all practical purposes this line can be extended as far east as Medicine Hat. On the south is the International boundary line, and to the west the summit line of the Rockies and British Columbia. This Southern Alberta comprises an area of 150 miles square, of mountains, foothills and prairie. It is intersected every few miles by mountain streams as yet unpolluted by the filth and garbage of more thickly settled communities. Its general character is treeless save along the valleys of these streams, which are fringed by the willow and cottonwood, and on the sides and bases of the mountains. To the outside world this is known as Canada's grazing country, whence England will draw a large part of its future beef supply. As a sort of corollary to this, it has also been known in a general way that it was reputed to have a milder climate than the rest of the Northwest Territories. To most of you the following description will be not only interesting but necessary to a proper understanding of what follows. It is taken from an admirable paper by Mr. C. C. McCaul, of Lethbridge, published in the August number of the *American Meteorological Review*. After noticing that winter only really sets in about the middle of December, he goes on to say:

It is characterized by a maximum of bright, still, cloudless days, a scanty snow fall, and frequent and prolonged breaks of warm weather, heralded by the Chinook wind, of which more hereafter. Occasionally a bad snow storm will cover the prairie and hill to a depth of 18 or 20 inches. This, however, is very exceptional. The winter generally breaks up in February by a grand blow from the west, followed by a period of from one to three weeks of warm, bright weather, which may fairly be called the beginning of spring. Spring, here as elsewhere, is the most variable and capricious season of the year. On the whole it may, perhaps, be described as cold and damp, with frequent rainfalls, varied by bursts of the most gloriously bright warm weather, lasting sometimes a fortnight or three weeks.

May is generally fine, warm and bright; June