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and
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Recent Theories of the formation of Pus, Considered with reference to dental operations.

The ancient and time honored theory that the formation of pus is an act of the tissues themselves, ~~or is a mechanical effect following~~ ~~the~~ ~~molecular death~~ ~~simply~~ has, within the last few years, been questioned by the strictest experimental inquiry and apparently has been disproven. The surgeon of the past has seen the formation of pus occur in open wounds with such promptness and regularity ~~within a few days following an accident or an operation~~ that it necessarily ~~became~~ came to be regarded as a part of the series of phenomena taking place during the process of repair. That this formation of pus was an act of the inflamed tissue was not ~~not~~ ~~questioned~~ ~~than that inflammation was~~ ~~such~~. This was unquestionably the opinion of the medical world down to a very

recent date, and is perhaps yet-held by the masses of the medical profession, The fluid portion of pus was regarded as liquefied portions of tissue and ^{escaped} serum in which was commingled large numbers of so called pus corpuscles. These latter were very properly held, by almost all observers, to be nothing more than amoeboid cells which had been so unfavorably placed that they had died and were thrown off with the fluids, or had wandered out from the tissues and were carried away with the secretions. In this view of the case pus came to be regarded ~~as~~ generally ~~constituted~~ ^{as} a normal secretion, and was looked for as an essential result in any ^{considerable} wound which involved the integrity of superficial parts.

While this was looked for as an essential occurrence surgeons had leaned ~~to~~ to regard the character of puss with interest in each particular case, and when this product was thick creamy and inodorous it was regarded as a good omen, and

This was called laudable pus, healthy pus, &c. and as so long as this character was maintained the wound was regarded as in good condition. No. 4 If, however, the pus departed from this condition it was known to portend evil for the patient. ~~This state~~ (above)

~~This attitude was first disturbed by the brilliant experiments of Joseph Lister.~~

It had long been observed, however, that wounds in which the integrity of the skin was not disturbed very generally healed without suppuration. A bone might be broken and even comminuted and if not complicated by an opening upon the surface the wound would generally heal without the formation of pus. The case might present as much inflammatory movement as the open wound, the extent of injury might be great and the case present a grave aspect but yet it would very ^{generally} run through the course of repair without the formation of pus.

These facts gave rise to much inquiry

as to why this clinical difference, and the conjecture ~~was~~ that it was in some way brought about by exposure to the air, ^{was entertained} ~~as~~ usual when anything extraordinary occurs in the way of chemical change that meddling element Opium was regarded as "probably" the mischief maker.

These theories received the first severe discredit by the ~~x~~ brilliant experiments of Joseph Lister of Glasgow Scotland. This surgeon by what became known later as the Lister Method, a method which had for its object - the exclusion of micro-organisms from the wounds, demonstrated that open wounds might be so treated that the process of repair would be essentially the same as that of internal injuries. He arrived at the idea that ~~in the main~~ the formation of pus was in some way ^{influenced} ~~connected~~ ^{by} the life and growth of micro-organisms, but how, seems not to have been very clearly made. ~~At the same time~~ However the ancient idea that the formation of pus was a vital act was not

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questioned by him. He seemed rather to look upon the micro-organisms as having the effect of further depressing the vital energies of tissue already inflamed or of increasing the inflammatory movement already present in such a way as to increase the ^{suppuration} pus formation or to produce it - in cases in which, it would not have occurred without their presence, But down to the time of the worlds medical congress which met at London in 1881 he certainly had no idea of questioning the general opinion that the formation of pus was a vital act of inflamed tissue. At that time Prof Volkmann of Halle, ^{and some others} held the opinion that without microorganisms there was no pus formation. He says "In the worst conditions, And with the most disordered state of health no suppuration takes place if septic infection is prevented" (London Congress Vol II p-862)

This was regarded by Mr Lister and others as a very extreme view.

> ~~M. S. Hance of Paris in a series of~~

The answer to the first of these questions
'What visible phenomena are ~~then~~ found
to be present which had not been formerly
noted and considered?' is purely a matter
of observation, and can be settled in no
other than the experimental way. Many phenomena
may be present during such changes as that we are
considering that are purely accidental and not
necessary to the process. Therefore to establish
that a certain phenomenon is essential it is
necessary to show that it is always present, and
that the process cannot proceed without it -

in the absence of micro-organisms,

This testimony drives us to the conclusion that micro-organisms are the agents of pus formation. The question as to how they act to produce pus is ~~important~~ ^{unimportant}. In the consideration of this question there are certain ~~fundamental~~ ^{fundamental} facts that are so firmly established by direct observation that they must be regarded as fundamental and no theory can be entertained that will not include them. What may be termed the anatomical or physical process of the formation of pus has been so well observed in the past, and agreed upon by so large a group of observers that it must be regarded as definitely settled, and beyond the possibility of dispute. We may properly look for factors in this process heretofore overlooked and seek different explanations of ^{the phenomena} ~~movements~~ observed but those phenomena that have heretofore been prominently apparent, are, and will

ever remain the prominent phenomena of this process. Briefly, these are, ~~the~~ inflammation with its usual phenomena of heat, Redness, pain, and Swelling as stated from the more superficial observation. But from the standpoint of histological inquiry ^{certain local disturbances of the circulation} they consist essentially of the formation within the tissues, or upon the surfaces of membranes, of a certain plastic, or coagulable exudate ~~and~~ the gathering of white blood corpuscles, wandering cells, ameboid cells, or ~~and a certain disturbance of the fixed cells of the~~ leucocytes to the spot, ~~or~~ in the focus of the inflammatory movement.

These ameboid cells are properly undeveloped ^{or cells derived from reformed connective tissues} ~~or cells derived from reformed connective tissues~~ ^{or} ~~open~~ connective tissue cells, and one of their functions in the economy of nature is the repair of injuries. In the process of ~~this repair~~ these cells are developed into the new tissue, ^{or tissue} of repair, ~~in this process~~ the plastic exudate is ~~an~~ essential ~~to~~ ^{to} ~~the~~ ^{the} ~~process~~ ^{process} All tissue of a primary

character is first developed in a homogeneous matrix, This is true of the development of the tissues of the fetus and is equally true of the development ~~to tissues~~ ^{of} tissues of repair. The plastic exudate thrown out in the process of inflammation forms the matrix in which the ameboid cells develop ~~into~~ ^{the} tissue of repair. These cells are always found imbedded within this exudate and it is essential to their growth or development into tissue.

In the formation of pus this plastic exudate is observed to liquify, and the ameboid cells are found floating in the liquid mass and are then known as pus corpuscles. ~~For~~ Indeed the cells under these circumstances ~~are~~, are no longer capable of development. The matrix in which they were lodged and in which, normally their development should proceed, has become liquid ^{and is so changed in its chemical qualities that it} and fails to support them and they die.

~~is so changed in quality that they die.~~

Furthermore this plastic exudate, as I have said, is also thrown out among the fixed cells, the older elements of the tissue in which an inflammation occurs, Liquefaction may occur, and does occur in this position also, Here we find that the ameboid cells are mingled among the fixed cells of the part and all ~~are~~ become imbedded in a matrix of plastic exudate, In the event of ^{suppuration} ~~the formation of~~ pus the plastic exudate becomes liquid and the tissue, already divided by its presence, ^{is in part rendered fluid} ~~is~~ dies, and floats away with the liquid material ~~as~~ shreds and cells together with the ameboid cells, All of these combined form ~~pus~~ what we know as pus, ~~After this process of destruction has been~~ ~~become established~~

In open wounds this process is ~~also~~ observed to begin superficially, or upon the exposed surface. In the

development of phlegmonous abscesses it may have its beginning in the midst of the tissues, In the latter case - after its establishment the further development of pus proceeds from the walls of the abscess as in the open wound it proceeds from the surface, Amoeboid cells that come to the surface tend to pile up in the form of living granulations but more or less of them float away in the lymphatic exudate that should form their matrix. In this way the ~~liquefaction of the matrix and the loss of cells~~ is constantly proceeding, the formation of the matrix ~~and the~~ filled with fresh amoeboid cells is constantly proceeding which tend to ~~go~~ develop into granulations is constantly proceeding, And the liquefaction of this exudate carrying with it the amoeboid cells in the form of pus is also ^{constantly} proceeding. If the former exceed the latter the healing of the wound is being accomplished.

~~but this is a matter~~ but if the latter exceed the former, destruction of tissue and the widening of the breach of continuity is the result. This much has been observed by histologists and pathologists for years past and may be regarded as so firmly established that our views upon these points are ^{not} likely to be essentially changed by future observations.

The ^{important} questions which present themselves for inquiry now are two, around which all others arrange themselves

1st What-visible phenomena are now found to be present which had not formerly been observed and considered.

2nd What-is the cause of the liquefaction of the plastic exudate and the death of the amoeboid cells

The answer to the first of these questions must rest purely upon direct observation, and can be settled in no other than the experimental way. For many years past microbes have been observed in pus, but the idea that they were in any wise essential to pus formation was not seriously entertained until a comparatively recent date, and had not been considered. Many phenomena may be presented during the occurrence of such changes as that we are considering that are purely accidental and not necessary to the process, therefore to establish that a certain accompanying phenomenon is really essential, it is necessary to show that it is always present, and, that the process cannot proceed without it.

Observations in regard to the connection of microbes with the formation of pus have now been in progress for many years and by the most

divided into two classes ~~for~~ First, examinations of pus with the view of determining ~~the~~ whether or not microbes are uniformly present. Second, experimental production of pus under conditions introduced to illustrate the connection of microbes with the process.

The finding of microbes in pus dates back many years, for a long time nothing was thought of the matter, for it was considered to be purely accidental and of no consequence. Until comparatively recent times it was supposed that only the pus of open wounds was contaminated with these organisms, but as the means of search for them approached their present stage of perfection many individuals asserted that microbes were present in all ~~abscesses~~ pus whether ~~that~~ it ^{was} ~~be~~ from internal and previously unopened abscesses or not. These observations have now been repeated in the most painstaking way by observers in various parts of the globe separated by

Formerly, before the methods of examination had reached the perfection they have ~~now~~ acquired within the last few years observers had often failed to find micro-organisms in pus from previously unopened ~~deep~~ abscesses, but more recently this search has been more successful. I have myself examined the pus from a large number of abscesses, ~~and~~ especially alveolar abscesses, and for several years past have found cocci in the pus in all cases. Not only this but have cultivated these organisms from these abscesses in broth on gelatine and ~~upon~~ upon agar agar and noted their characters. Dr. Ernst of Boston

has also made a large number of cultivations from various classes of abscesses with similar results. This class of observation ^{is but} ~~has now~~ ^{a repetition of the observations} ~~been repeated~~ by various observers in different parts of the world separated by thousands of miles, and all give substantially the same ~~test~~ testimony. All pus no matter where found

whether in closed abscesses deep within the tissues or upon the surface contains living microorganisms that will grow when placed in suitable cultivating media.

But the more decidedly practical plan of study and one that appeals more strongly to the mind consists in the effort to produce suppuration in experimental wounds from which microorganisms are rigorously excluded.

This class of experiment has been undertaken by a considerable number of persons; In the first of these the precautions were not successful and microorganisms gained entrance vitiating the results and giving the impression that pus was produced under aseptic conditions. A very severe ~~of~~ training was found necessary to the successful performance of this class of experiment. But as the plans were improved upon success in the exclusion of microbes

was attained, The results seem to have proven that without the presence of microbes no amount of injury or irritation of the tissues will result in suppuration. These experiments have now been repeated by so many persons and varied in so many different ways, and so many irritants have been employed that the question seems no longer doubtful.

It seems unnecessary that I should now recite all of the experimental evidence that has accumulated within the last few years. This may be found in our literature and the more important portions have been collected in convenient form for reference by Dr H Knapp of New York in an article in the Archives for Ophthalmology Vol 15 (1886) page 24 and another in the New York Medical Record of December ~~1886~~ 25th 1886. I have also cited some of these in an article in the Dental Review of March 16th 1887. In addition to these the work of Dr Ernest of

Boston should not published in the transactions of the American Surgical Association for 1886 Page 58 should not be overlooked.

As illustrating the character of this work for the benefit of those who may not have followed this literature I may recite the plans of a few of these experiments. E. Schenck placed irritants, such as croton oil and turpentine in very thin glass capsules or tubes, which were sealed hermetically and sterilized. These were placed under the skin of healthy animals with antiseptic precautions and allowed to remain until the wound by which they were introduced had perfectly healed. The tubes or capsules were then broken ^{to} discharging the irritant, bringing the irritant in contact with the tissues. By this plan of experiment a hard swelling was produced but no pus was formed by except in one case and in this the experiment concluded

That the wound had not perfectly healed. Cocci were found in its track and in the pus that formed in the focus of the inflammation. This form of experimentation has been varied by a number of individuals with similar results. Dr. Rupp and Dr. Knapp have made several series of ~~experiments~~ experiments also of a similar nature except that they have injected irritants into the anterior chamber of the eye (~~into~~ (in rabbits) where both the irritant and the results of the irritation could be seen and have satisfied themselves that it was impossible to produce suppuration in the absence of microorganisms. I have myself made some experiments by passing broaches rendered aseptic by heat through the apex of the roots of teeth into the tissues beyond and tearing them up as thoroughly as was practicable. And although inflammation was sometimes produced there was no suppuration.

~~in the absence of microorganisms,~~
 accumulation of

This testimony drives us to the conclusion that microorganisms are ^{the} active agents in pus formation. The question as to how these act to produce pus is the next important consideration and leads to the consideration of the second question viz. What is the cause of the liquefaction of the plastic exudate and the death of the ameboid cells.

This question had been discussed before the discovery that microorganisms were in any wise related to the suppurative process. And the general idea of pathologists may be expressed in the phrases "over stimulation of the part"

"Intensity of the inflammatory process"

"Failure of nutrition" "Death ^{of the} cells from abnormal exposure" &c On these points there was no very ~~clear and~~ satisfactory opinion, the general idea being that these cells unfavorably placed either by the intensity of the inflammatory movement, by the partial cutting off of the supply of arterial blood, or by a generally impoverished condition of the ~~systemic~~ system, died, ~~on that account~~, and ^{that} the liquefaction of the matrix or exudate was the result of this death of the cells. All of these suppositions, except the last, are probably ^{and should still have due consideration} very nearly correct, but further study of the subject seems to show that the liquefaction of the exudate is dependent upon other ^{and is the primary lesion looking to the formation of pus} agencies. It may be stated as an axiomatic principle that no portion of the animal body is capable of self destruction. The changes that take place are under

Therefore the liquefaction of the exudate cannot be regarded as the act of the tissues nor can the amoeboid cells destroy themselves.

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the influence of life and life is not self destroying. One part of the body may destroy another - as is seen in the solution or absorption of the roots of the temporary teeth ^{odontoblasts, or the absorption of bone by the} by the osteoclasts, but the roots of these ^{teeth have no part, except a passive} teeth ^{in the process,} have no part, except a passive one. One cell may destroy another in the same way, or one group may destroy another group. But in no case can a cell or any individual component of the body ~~Therefore the liquefaction of the exudate is not an act of the tissues~~ destroy itself. Any component of the body may die from want of nutritive support ~~for other cause~~, or may be destroyed by traumatism but in this event the material elements, the chemical forms, remain. These dead forms have no power in themselves to change. Long ago it was supposed that organic matter left to itself fell into a state of molecular motion in consequence of which its chemical form was changed, as

7 This is continually witnessed in various parts of the body - Blood clots resulting from the ligation of arteries. Empyema, Infarction in which considerable areas of tissue is destroyed ~~and~~ &c &c rarely suppurate. In these cases it is seen that a tissue that simply loses vitality, dies does not tend to change form except - as it is acted upon by the living tissues about it, and being shut off from the approach of microbes does not take on degenerative changes.

The cause of suppuration, when it does occur in these, will be explained later.

was taught by Liebig. We have since that
 time learned, however, that all such trans-
 formations of matter are results of the life
 force exhibited in certain micro-organisms
 and that matter in ~~and of the~~ itself and
 by the powers inherent in it as matter has
 no power to change, except as one element
 or compound is presented to another, And
 that in each instance the action of chemical
 affinity thus aroused tends to self-satisfaction
 and a condition of permanent quiet, until
 again disturbed by forces extraneous to itself. Now
 tissue that has died is left to itself and its
 chemical affinities are satisfied. It is therefore
 incapable of self-movement or of change of
 chemical form. ~~Is it otherwise with the~~
 forces inherent in matter, as such, certainly the
 theory of spontaneous generation might be
 maintained

That the liquefaction of the plastic

exudate for the formation of pus is not
 an effect of inflamed tissues ^{in the first instance} is a matter
 of experimental inquiry rather than for
 theoretic consideration. This inquiry has
 been had as related above and this
 theoretic consideration is ^{properly} the result
 of that inquiry. In the years that have
 past since the discovery of the yeast plant by
 Schwann in 1838, discovery after dis-
 covery have combined to show that all
 of those changes that ~~go~~ had been observed
~~to go on in~~ in the various forms of fermenta-
 tion ^{and putrefaction} were the results of the life and growth
 of microorganisms, and ~~that~~ in no case
 has matter been found capable of spontaneous
 change. Now it is found by the strictest
 experiment that those changes which
 result in the formation of pus are not
 due to the vital principle residing in
 the tissues of the animal, but to the vital
 (nor to physical agencies -

I had been supposed that contact with dead tissue was sufficient ~~for the dead~~ to destroy the vitality of the amoeboid cells but it has been found that they may wander into the dead tissues of infarcted regions ~~regions destroyed by traumatism~~ or dead tissues placed in the body experimentally and retain their vitality

27 ^{by the operation of microbes} Liquefaction of the plastic exudate death of amoeboid cells from ~~loss of their~~ and the changed chemical character of their ^{matrix} support These combined form pus. These processes produce irritation which extends the inflammatory movement. Hence the suppurative and destruction of tissue

principle as exhibited in microorganisms.
 It is in fact a species of fermentation of the
 plastic exudate thrown out in the act
 of inflammation. ~~As is found to be the case~~
^{Strict experimental inquiry shows that}
~~with all fermentations, this~~ This exudate
^{by a fermentative process}
 is rendered fluid, and is so changed in its
 chemical qualities that it no longer supports
 the ameboid cells. These cells then die, and
 in some of the forms of suppuration many
 of these are broken up and liquefied also.
 This then is the explanation of ^{the} suppurative process
 and is in strict agreement with other forms of ^{anaerobic} fermentation.
 This power of micro-organisms of
^{to} ~~changing~~ gelatinous material into the
 liquid form is well illustrated by artificial
 cultivations. A considerable number of
 varieties ~~of these~~ when grown in ordinary
 sterilized gelatin in ~~test~~ test tubes produce
 liquefaction quite rapidly at ordinary
 summer temperature. One variety that
 I have recently been cultivating with
 a little brownish red cocci will

When planted upon the surface of gelatin in a test tube liquify it ~~at the rate~~ progressively from top to bottom at the rate of about $(\frac{1}{8})$ one eighth inch per day, at a ^{and much more rapidly as the temperature} temperature of eighty degrees, Fahr. Many others are found that liquify gelatin, ^{but with less rapidity} similarly.

It is ~~this~~ ^{that gives} power of liquefying substances upon certain species which they grow that ~~the power of~~ certain microorganisms the power to produce pus rather than any quality of producing irritation or inflammation. Most of them indeed produce irritation also and some of them do so in a very marked degree, especially the ordinary Streptococcus pyogenes so generally found in phlegmonous abscesses. In this respect there is great difference According to my own observation among the different varieties of pus producing microbes. This has been so marked in my experience that I am able generally to say in advance

(in certain qualities of abscess
 that certain microbes are absent or present.
 For instance I have never found the *Streptococcus pyogenes* in a white creamy
 pus in an abscess that had arisen
 with little pain or in a wound that had
 not taken on a decidedly inflammatory
 condition &c. However it is not now
 the intention to discuss the characters of
 individual species or varieties of microbes
 that are active agents in pus formation.
 There are now about twelve varieties recog-
 nized that are capable of producing pus
 the greater part of which I have myself observed
 and cultivated artificially. It is probable
 that additions will be made to this number
 in the near future,

> To the dental profession it is a fact
 of great interest that ^{most} ~~all~~ of these fungi
 grow fairly well in the ^{human} saliva, and are
 found in it very frequently, ~~this being~~

27 ~~22~~
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have recently

I made a considerable number of ~~cultures~~
plate cultivations from the saliva of dif-
ferent individuals for the purpose of
determining how frequently these fungi
might be found in ~~this~~ the mouth of
healthy ^{persons} ~~individuals~~. The number of
cultivations ^{have} ~~were~~ ^{been} ~~not~~ sufficient to be
satisfactory. In the examination of ten
cases I found the ~~staph~~ Staphylococcus
^{pyogenus} ~~several~~ ^{seven} times as determined by its form
& growth in broth, upon gelatin and
upon Agar Agar. Staphylococcus pyogenus
albus four times, Streptococcus pyogenus
three times, ~~Other forms are found only~~
~~occasionally in the mouth.~~ These trials
were made by simply scraping the tongue or
some portion of the mucous membrane of the
mouth ~~with~~ once with a platinum wire
previously brought to a glow. These three
forms are the most wide spread and

prevalent of the pyogenic fungi and my supposition is that they would be found in a vast majority of healthy mouths if the search was carefully and persistently made.

In a few cases that I have examined repeatedly I have found the Staphylococci ~~Streptococci~~ every where in the mouth. In other cases I have been unable to find them at all after repeated efforts.

The streptococcus pyogenes is so similar to the ordinary streptococci of the mouth that it cannot be distinguished by microscopic examination except by one who has had much observation of these species together and separate; but its colony, and its surface growth on gelatine afford a means of distinguishing them after its manner of growth ^(see learned) has once.

Other pyogenic fungi than these three I have only occasionally found in the mouth. I have also found them much less frequently in abscesses and in pus from wounds and must suppose that they are much less

frequent in the air. It should be understood that any microbes in the air we breathe which are capable of growing in the saliva are likely to be found there from time to time. Besides these there are certain species that are peculiar to the saliva and are found in the mouth of almost every individual. These latter are comparatively few in number and ~~are soon~~ become familiar to one who spends much time in making cultivations from the saliva or mucous membranes of the mouth.

As dentists we must consider that these pyogenic fungi are ^{generally} ~~constantly~~ present in the mouth, and that every wound we inflict is in danger of becoming infected by them. Under these circumstances a study of the consequences of such infection becomes important to us, ^{and our patients}. In the first place the tissues upon which we operate - except it be the ~~teeth~~ hard tissues of the teeth themselves - are the best

supplied with blood and nerves of any in the
 body and are proportionately more resistant
 to evil influences of all kinds, on that account
 This power of resistance applies to the onset ^{evils resulting from}
 of microbes as effectively as to any
 other form of injury. Very few of the pyogenic
 fungi ~~if any~~ ^{ordinarily} have any considerable power
 to produce inflammatory conditions, and
 they have practically no power of mischief in
 the absence of inflammation in any part of
 the body and necessarily the least about
 the mouth and face where the power of
 resistance is greatest. When inflammation
 gives them the opportunity some of the varieties
 cause a very rapid liquefaction ^{lymph deposits in} ~~the~~ the inflam-
 matory focus, with increase of the inflam-
 matory movement, until the growing fungi
 are surrounded by an almost solid wall
 of living ameboid cells - The so-called
 pyogenic membrane of older writers -

Another reason for the abridgement of the damage is found in the weakening of the fungi from the accumulation of their own waste products. If we plant pyogenic fungi in peptonized beef broth they will make a more or less vigorous growth but after a time they sink to the bottom of the vessel and all growth ceases. In case of the acid forming fungi we have learned by direct experiment that this cessation of growth is not on account of the exhaustion of the peptone but on account of the accumulation of the waste product, the acid, and that if this is neutralized by the addition of an proper amount of an alkaline base forming a salt, answering the end of elimination of this product, growth ^{the} will again proceed, ~~just this~~. This accumulation of waste products of the pyogenic fungi occurs in the pus of abscesses rendering it unfit for the continued growth of the fungi which produced it; the operations of the fungus becomes limited to the fresh exudates thrown in from the abscess wall.

This is in turn limited as the walls 31 ~~of~~
become more solidly packed with ameboid cells,
living matter, which is not so readily attacked.

This increase of the living element in the wall
of the abscess, has the effect of limiting the growth
of the fungi and in the end ^{they are} expelled
followed by the healing of the abscess.
This ~~course~~ ^{sharp rise and subsidence of suppuration} may be said to be the common
course of events when a focus of inflam-
mation ^{arising from direct traumatism or other cause} is infected with pyogenic fungi
alone, and is varied by two factors. First-
the vital powers of the tissues 2nd the activity
or vital ^{energy} ~~power~~ of the fungus. According as
the one or the other is greater or less will
the course of suppuration be prolonged
or abbreviated. If, however, the focus of
inflammation be contaminated ^{also} with
those species of fungi which produce
active inflammatory conditions such
as the cocci of *Dyscyspilus*, or others ^{of similar nature}, the
injury may be wide-spread and grave.
Happily such is but rarely the case in the
class of lesions which we are called upon to
treat.

This far we have considered open wounds only, or abscesses which may possibly have been infected from the surface. But microbes are found in all pus whether the locality of its formation has had the opportunity of infection from the surface or not.

The question now comes as to the possibility of infection by other channels, or what I shall designate as indirect-infection.

The ~~apt~~ ablest experimentation on the subject of indirect-infection has been performed perhaps by Becker and Krause, it having been observed that pus was occasionally formed in case of simple fracture of the limbs, in which the wound was in a position which rendered direct infection impossible, and yet that this pus contained the usual cocci. These gentlemen instituted experiments for the purpose of determining whether or not microbes could be conveyed to the spot by the blood stream, or by the lymph that had been

together with others of a 33 like nature 26

widely entertained

soon after the introduction of the Lister dressings. This had lead to the search of healthy tissues for microorganisms and the results satisfied bacterologists that such tissues did not contain them. Still the question as to whether microorganisms gaining access to the blood by accident might be so carried was an open one. In this view of the case the gentlemen named began their experiments by breaking the limbs of healthy rabbits, and found them to heal readily without the formation of pus. Then the experiment was varied by injecting into a vein of the ear a fluid containing Staphylococci after the ~~pr~~ breaking of the bone of the leg. In this case it was found that suppuration followed regular and that the same species of microorganisms appeared in the pus. These experiments established the fact that microbes may be carried by the blood to a focus of inflammation and there set up suppuration.

New York Medical Record ^{performing} December 25 1886

Dr Knapp's experiments, also show that
 microbes ~~can~~ gain entrance to the blood
~~after~~ from wounds that are infected exper-
 imentally, for in his experiments with
 double operations on the eyes of rabbits
 (see New York Medical Record of ^{Dec.} ~~Jan.~~ 25 1886)
 one of which was infected and the other not
 he ^{occasionally} found the same order of microbes
 used in the infection in the eye that was
 not infected, showing that direct injection
 into the blood stream is not absolutely
 necessary to the infection of an internal
 focus of inflammation. This teaches us that
 an internal focus of inflammation may ~~be~~
 become infected from a remote focus
 of ^{suppuration} ~~pus formation~~ upon the surface
 or upon the mucous membranes. It therefore
 behooves the surgeon to examine his patient
 critically for minor points of suppuration
 before performing any ~~capital~~ important
 surgical operation.

The time, in the course of a suppuration at which the blood is most likely to contain microbes is important, I do not know that there has been any direct experimentation upon this point but it is reasonable to suppose that after the formation of granulations or the limitation of an abscess by the formation of a so called pyogenic membrane the danger of internal infections will be greatly diminished. This is ^{also} in harmony with what is known of the occurrence of septicemia from wounds,

Such being the teaching of the facts now at
 our disposal it becomes important that we
 inquire into the circumstances and conditions
 under which infection with these fungi
~~is~~ ^{is} liable to occur in dental operations
 and the circumstances ^{and localities} which favor the
 continued activity of the fungi after
 infection has occurred, ~~and the~~ The great
 prevalence of alveolar abscess, and the frequency
 with which the dentist is called upon to
 operate under conditions which favor the infec-
 tion of the apical space ^{the best example for the illustration of the principles} renders it ~~important~~
^{taught by the facts ~~unavoidably~~ recited} ~~that it be considered~~. Since we have learned
 that we can only have ^{alveolar} alveolar abscess after
 infection of the apical space with pus forming
 organisms we have an incentive to the perfor-
 mance of aseptic operations which we never
 felt before, we have also learned that the
 enemy is in the saliva of our patients
 and the facts at our disposal point to this
 as not only the most probable source of

infection, but as surely the almost universal ^{except-} source, ~~of~~ infected instruments ~~accepted~~

It therefore follows that this source of infection should always be eliminated before a pulp chamber is opened. ~~It might~~ To do this it is necessary to put on the rubber dam and disinfect the tooth or teeth included and to disinfect all instruments used in the operation. For this purpose suitable disinfectants should always be on the operating table. It may appear to some that in case of a pulp chamber that is already open and infected this precaution will be of little avail - Not so, for if the canals are already infected the first object will be to thoroughly disinfect them, and to do this the source of infection should first be eliminated. Then and not until then can we expect to successfully disinfect the canals. The fact that many root canals

have been successfully disinfected without
 this precaution only shows the power of
 the disinfectants employed, It does not
 seem to recommend such procedures since
 the source of infection has become so
 certainly known, Very foul roots should
 be cleaned and disinfected with the rubber
 dam in position and the last of the operation
 should be done with freshly disinfected
 instruments. The cavity should then be
 perfectly sealed with a temporary filling of
 gutta percha or other suitable material
~~and the patient~~
~~wait the next sitting~~
 In no case should
 this filling be removed ^{at a subsequent sitting} before the readjustment
 of the rubber dam ^{and disinfection of the included parts}. In those cases in which
 the pulp chamber is opened for the first time,
 as in the removal of a living pulp, or a pulp
 destroyed by the operator, we should never
 or at least very rarely have abscess occur
 Indeed it should be impossible except by
 incorrect infection. This also is a matter

that should always receive our attention, Unless the exigencies of the case especially demand it - we should not open a pulp chamber at a time when there is an active focus of suppuration in progress any where in the body of the patient, In this statement I do not include chronic discharges, though these must, in every case, be regarded as unfavorable.

If we have found the conditions favorable abscess ^{following} ~~after~~ the removal of the tooth pulp should be impossible, we may pass our broach through the apical foramen and wound and lacerate the tissues and possibly provoke an inflammatory movement but if the ^{root canal and the} instrument be a septic abscess cannot occur, This I have tried experimentally in a sufficient number of instances to be convinced that it is practically true as well as theoretically true, I have in several cases

(These experiments were made with broaches ^{by heat} clean but in no case did abscess occur)

While all of this is true, it is absolutely essential that ^{both the canal and ~~the~~ broach} the broach be aseptic. Few men who have not had practical experience in the cultivation of microbes can form an adequate idea of the ^{readiness with which} danger from which they may be carried ~~carrying~~ ~~microbes~~ ^{by} them into the tissues of the apical space ~~with~~ the broach, I have often passed a surgical platinum suture wire, after having brought it to a glow, to completely disinfect it; into a ^{foul} root canal and then into stiff gelatin cultivating media four five and six inches, and have seen the development of microbes along the track of the wire from end to end, ~~If this~~ ~~may be~~ If these organisms may be carried into a stiff gelatin in this way with a perfectly smooth platinum wire what may we expect from a barbed broach thrust through a foul root canal into the tissue? If this does not produce abscess it

will be because the root canal happened not to contain pus forming microbes, I have myself, ^{as I think} produced abscess in this manner many times, and from what I know of ~~what is occurring~~ in the practice of others I am satisfied that such an accident is of very frequent occurrence. In the cleaning of ~~ceptive~~ ~~can~~ contaminated canals - and all root canals to which the saliva has had access should be regarded as contaminated - No attempt should be made to reach the ~~end of the~~ apex before the introduction of a reliable disinfectant. The danger of forcing microbes through into the tissues of the apical space is so great that they should be destroyed before the attempt ^{to reach the end of the canal} is made. Then we may risk passing the broach to the very end of the canal and not ~~wait~~ before.

The rule is simple enough, To have alveolar abscess we must first have microbes in the ~~tissue~~ ^{canal}. If the canal has been irrigated

to the fluids of the mouth we must not risk forcing its content through the apex until it has been rendered aseptic, Then if this aseptic material is forced through it - may produce a transient inflammation but it will not begit abscess

What is the teaching of the facts developed in case of infection of the tissues - First - I may say that it is clear that there are many infections that are successfully resisted by the tissues, and the microbes eliminated without the formation of abscess, Second, Where the area of the abscess is small and the general condition of the patient good the tissues by their own unaided powers will expell the intruder. with the pus first formed or very soon thereafter if proper opportunity is presented. This only requires free discharge of the pus, with care that there is not continuous re-infection. It should be stated that pus itself should be

regarded in the same light - as a state
 cultivation media in which the organisms
 which produced it ~~is~~
 is incapable of growing, hence when the central
 focus of the inflammation or of the lymph
 deposit is broken down the organisms have
 become greatly depressed from the presence of their
 own products and are readily expelled by the
 young connective tissue cells which have formed
 themselves into a ^{compact} wall about the abscess.

Therefore the source of continuous reinfection
 which such exist - should be eliminated, In case
 of ^{simple} alveolar abscess this source is the pulp canal.
 In this space ~~they are~~ the microorganisms are out
 of reach of the tissues and a new growth occurs
 with a ^{every deposit of lymph} reinfection about the apex of the root.
 every time fresh pabulum has entered the
 root canal ^{and there infect any fresh lymph deposit about}
 apex of the root. Hence the abscess becomes
 chronic. ^{with its Haversian canals} Dead bone offers the same opportunity
 for growth of organisms in a harbor beyond
 the reach of the tissues. If ^{on the other hand} this bone be

aseptic ~~on the other hand~~ it - will be absorbed
 by the tissues, and removed. With the root-
 of the tooth this sometimes occurs to our
 sorrow, but ^{in case the root of the tooth becomes denuded of its tissue} ~~happily~~, this tendency is here
 largely counteracted by another which is
 the ~~strong~~ tendency to the recovering of the
 root with cementum, ~~wherever it has been~~
~~denuded of its tissues~~. But in order that
 either of these can occur the part must
 be aseptic, or free from microbes. In any
 such case the microbes have a wall to lay
 against and ~~the tissues~~ are to that extent
 protected. The active tissue is on one side
 only instead of surrounding them. Experience
 teaches us that in this case a chronic abscess
 is maintained ^{or} in many instances. This
 requires antiseptic treatment to dislodge
 the microbes but - in all ordinary cases
 of alveolar abscess the elimination of the
 source of ~~the~~ continuous reinfection

from the root canal is sufficient to cure
chronic alveolar abscess

The rule is that, ^{the} microbes of pus formation are
unable to maintain themselves continuously
when surrounded by ^{living} active soft tissues &
any kind