"Some Etiological Factors of Health"

A Clinical Study

by

Gerald Septimus Samuelson M.B. C. M. Ed. 1888
Member of the Medico-Psychological Assn.
Surgeon, District Hospital, Gundagai, N.S.W.
Government Medical Officer,

Thesis for the Degree of Doctor of Medicine of the
University of Edinburgh.

Gundagai, N.S.W.
March 19th 1893.
Classification.

I propose to deal with the subject of immunity
natural or acquired under the following sections:

Section A. Tissue conditions, normal or abnormal.
   No. 1. The mouth.
   No. 2. The Stomach.
   No. 3. The Intestines.
   No. 4. Cell Infection and spread.

Section B. Specific immunity of tissues.

Section C. Acquired immunity of organisms.
Introduction.

Dr. Pfe-Swift in a lecture delivered before the Royal College of Physicians of London has remarked that "disease is as natural as physiological as health — the existence of disease in general needs no further explanation than the existence of life."

To render the subject of my thesis more explicit, I will show the latter sentence in the light in which my more thoughts have made it appear.

"Life" I define as "priestess", animate or not. I take it that that which is, is "life" within the meaning of the writer quoted.

Dr. Pfe-Swift dwells upon the natural occurrence of decay and death and on the weakness of considering disease to be abnormal. "Life is a constant struggle," he says, "against surrounding forces in which each individual organism succeeds after a while by handing on its life to its successor, when it has secured the victory of the race."

The modifications of health by the aetiology now from is by disease have always been the study of the physician. How seldom is thought given to those causes which in the race between health and disease prevent the latter and preserve the former! Health is an ideal realised but comparatively. The strong
and whether have in and around these potential diseases. What are the factors that prevent disease as a disease agent?

In each individual organism the predominance of the attributes of the one over those of the other, determined the fall of the balance, where these attributes are general in their methods of mutual attack and resistance.

The attention of the "naturalized physician" is commonly directed to the removal or lessening of a disease which may threaten an organism, or a constituent part thereof, rather than to the consideration of those conditions which assist that organism or its part to combat the disease — in other words, the physiology of immunity or continued health in spite of potential disease.

There are many considerations indicated by the title of my thesis which belong to the hygiene, but I will restrict myself to the record of observations at the bedside and laboratory, and the less happy notes from the deathhouse. My endeavor will be to show some physiological factors of health of which some are dependent on physical health, whilst some are not. I will from these considerations draw a few practical deductions.
Section A.

101 The teeth are prone to certain caries due to early decay and original root development; this is not always the case, for in other words, conditions the teeth are protected from a caries disease by one of its manifestations containing another.

Dowce dwells upon the necessity of a suitable condition for the production of Caries, viz. a malnutrition and an acidity of the surrounding fluids. These conditions may be due to systemic change lowering the vitality of the tissues generally and probably rendering the saliva acids, or to an especial local change due to systemic derangement or frequently, as the latter conditions, to an acid fermentation of debris between and in cavities of the teeth themselves. Still though this fermentation is due to bacteria, it is not the bacteria but the products that make the attack. De Magill tells us that caries results from a purely chemical alteration due either to the products of acid fermentations in the saliva, or by active agents introduced directly into the mouth.

Miller while experimenting upon the action upon teeth by bread paste cultures, was forced to renew his cultures whenever they became alkaline.

When a systemic change lowers the vitality of the gum, it usually renders the saliva acid and caries usually appears. Of a period of general
medical patients, I found the saliva of but one to be alkaline, as against 49 whose saliva was acid, whilst of 25 local surgical patients, the saliva was acid in but two cases. It may be noted that the debility of starch foods are much prone to collect in the interstices, where the acidity of the saliva has weakened the action of the pericline.

When a special local change due to systemic derangement occurs, caries is not an inevitable sequel, far from this, caries is not more usual than in average health. With salivation the gums become loose; the teeth are then altered in nutrition by the cause of the salivation (a systemic change) and by its effect i.e. sponginess (a special local change). Coincident with this the saliva may become acid.

A short experience of dentistry will demonstrate that a profound alteration in nutrition need not produce caries. The lingual surfaces of the lower incisors are the sites of the deposit of tartar, which in time loosen the tooth and hence interfere with their nutrition. The origin of tartar is the saliva. The ducts from the sub-lingual glands open into the lower incisors. I have frequently seen months toothless jaw for the lower incisors for caries is infrequent in this set, by reason of the relative above quoted. I have however frequently seen these teeth, though free from caries, loosened by actual recent heaps of large masses of tartar. Mr. C. E. Lester of Sydney has shown me a lower incisor on which was a deposit of tartar twice the size
of the organs. This tooth was finally included in about one eighth of an inch of gum, but it was altogether free from caries.

I have now under treatment a patient who some years ago became merrcurialised. This malady was sustained by constant application to his aeration, which was that of an Assayer and Mining Manager. He has now increasing symptoms resembling those of angular pectrois. During all this time he has been salivated, and during the six months I have observed here his saliva has been alkaline. He has lost two teeth in three years which he says caused him no pain, and which he extracted with tailor's thread. The remaining teeth are sound though loose, his gums are weeping.

In many cases of syphilis and primary merrcurial dental looseness, I have observed that the teeth are singularly exempt from nosophic changes; with them occurs salivation. I believe this coincident condition protects the teeth.

Suppose the saliva when in process to become acid or not, whatever this is salivation, there is increased flushing of the teeth. The primary cause of (of caries) is unquestionably the product of fermentation (acid) of particles of food. How are these particles of food to collect or remain in a mouth through which there is a constant flow of saliva? How is such an acid fermentation to occur when the saliva remains alkaline and is constantly bathing the fermentable matter?

Should the saliva when in merrcurial process remain alkaline, one after another desiderata is negated.
If it change in chemical reaction, the predominance of the aqueous change modifies the acidity.

Remembering that dental caries is due to an alteration from many causes in the furred tissues, and that in salivation one of the essentials is most manifest, it is apparent that a pathological change to hyperacemia of the tissues by detaching hydrogen the teeth alters their nutrition largely, whilst the same pathological hyperacemia leading to excessive salivations combats the effect of the alteration of nutrition. In other words, there is an abnormal tissue condition rendering an organ prone to a particular disease, whilst the same alteration prevents the disease in that particular organ. (My friend Dr. Leckie, writing on "hypertrophy of the gums in syphilis causes functional rearrangement of pain, and consequent falling out of teeth, but no caries").
The stomach may be said to have a natural immunity from pathological disease dependent upon the conditions of its tissues; if this is the case an exaggeration of one of these conditions though its self-working need still further ensure immunity.

I fully recognize that the constant recurrence of this disease, its periodic exacerbations and flushings prevent bacterial disease.

There is one other important factor – the acidity of the secretion.

In experimental inoculations Meir{a}, Patink, Robb found but poor, if any, results following the direct introduction into the stomach of the Spirituus cholerae (Robb) The former have juices injected direct into the discharges, the latter preparatory to injections, alkalized the gastric contents.

I was aware of the constant presence of sarineae in this secretion. Sarineae are not pathogenic, and found in stomachs of healthy people, their presence depends upon either the causes menit irritated.

The immunity of the stomach through the above cause will be again referred to at page 15.

Upon the consideration of the above it is apparent that a hyper-acidity of the gastric contents and precursors must not only militate against the location in is of pathogenic bacteria, but must also weaken the attaching force to the wall that it is less noxious to the intestinal surface in contact with which it must be thought.
I had a patient suffering from melancholia in the course of which disease he lost weight and, apparently, at irregular times became febrile; he thus developed a cough. His mental condition was such as to prevent examination: he was violent and pitable; he was swallowing large quantities of phlegm and never expectorated any. I examined all available secretions and secreta for bacilli without finding any. Very shortly after he rapidly coughed up a highly offensive and acid material. It resembled the vomiting mucus for tuberculosis. At his physical condition deteriorated his mind improved, and shortly before death he became all but sane; thus I was enabled to thoroughly examine him and found much feasting down of the lung substance. Owing to swallowing his phlegm, I discovered therein large quantity of tuberculosis bacilli. Of these he surely must have swallowed very many for a long time. At the necropsy made six hours after death by my colleague Dr Edward Blake and myself, we found extensive decay of both lungs in which there were cavities filled with purulent phlegm, a foul omen of a highly grey colour containing an excess of necrosed secretion and marked just nothing change of color. Careful examination failed to show the presence of tuberculosis elsewhere than in the thorax.

I have had the opportunity of examining the intestines of many persons dead from pulmonary tuberculosis, and one of the following opinions

1. That there is among forms of animal disease, independent of intestinal tuberculosis, one
which depends upon gastric indigestion, or the indigesterility of large quantities of swallowed opium. And that the determining factors of intestinal ptosis, from the swallowing of opium, are the quantity swallowed and the period of the disease during which it is swallowed.

Observing that the ideas found at necropsy are more frequently old than recent, I believe that show once, as the last stage of the primary disease, gastric or intestinal catarrh is marked, that, so far as the pulmonary source is concerned, that the infective of the bowel ceases.

For these reasons I am of opinion that the greater morbid effect the disease has upon the stomach, and the larger the quantity of opium swallowed, the more quickly and thoroughly does the disease poison the alimentary mucous surface from itself.
Mucho is heard of a vicious circle in pathology and put little of a similar circle in therapeutics, though there are many instances of one phenomenon occurring in an organ in the course of a disease to the relief of the organ primarily affected. "The effect of nature to get rid of the disease" is faithfully considered in the study of such diseases as Typhoid and Cholera. I have found that with the frequency of compensatory acts undertaken by the intestines in congested conditions of the kidneys and liver, this has led me to utilise the organs usually relieved by intestinal flux as a means of relieving an intestinal flux itself.

The chill from sudden exposure is apt to produce in some people a fluid diarrhea. A certain number of such cases exhibit another phenomenon upon the cessation, or just before it, of the flux; namely, a copious diuresis of a pale urine of low specific gravity. The physiology, or rather the pathology, of the above depends upon a renal inactivity, and not upon an intestina] activity, the latter being the product of the former. These pyrexias are rare frequent in those whose intestinal functions are irregularly performed, and, in such cases, I have found that a full dose of nitrous ether, with or without a warm bath, produces when diuresis is established an abatement of the diarrheal symptoms. I have found many diarrheas, especially those occurring from the exhibition of purgatives in susceptible intestines, which are equally amenable to this line of treatment. Apparently a chill acting upon the kidneys through the skin, calls for a compensatory action from
the bowels, the lithuric condition being relieved, this demand ceases. I have not observed diaphoresis occur in these cases. I noticed this relief in my early cases when I was more frequent in the use of morphia; I remember how in a severe and painful case immediate diuresis followed a hypodermic injection of morphia. Possibly some of these latter classes are due to a nervous condition. Physiologically I look to a stimulation of the neural nerves as the primary cause of the anuria, in which case the diuresis following the use of morphia is due to a lessening of the stimulations; that following that following the use of nitrites often depends upon the power of dilating the vessels which the nervous stimulation has previously constricted. I have seen other cases in which a compensatory action elsewhere is not readily established, in these the diminished secretion of urine has led to much discomfort and after depressions.

To emphasize my point—a cataurial condition, or a tendency to it, or irritability in any way of the intestinal canal, must result in a ready answer to the demands for compensation by the kidney to the preservation of the health of that latter organ. I have never found this function subserved by the intestines in such cases apart from such abnormalities in them.

In these cases then the abnormality of the one organ re-establishes the health of the other.

To one other such condition will I refer. If, as Breckin says, the exciting causes of pile are habitual constipation, accumulation— in the large intestines, the want of due secretion from the mucous surface, obstruction to the
proper action of the liver, and consequent congestion of that organ will not as abnormally large aqueous secretion counteract the tendency of a congested liver? Juices reflects upon these points I do not now invariably ask my patients who suffers from flux and sometimes bleed with it "if they have piles.

Woodhead says that acids are a deadly poison to the cholera bacillus and further remarks "It is a fact generally recognized that any disturbance of the digestive function is the principal disposing cause of the disease in a cholera outbreak — a fact that is explained by the absence of the ordinary amount of acid from the gastric juices in those cases; such gastric derangement is specially met with after a bout of drinking," and he quotes Wheel to corroborate this view. This fact, gentlemen speaking of sailors as infected ports pays among the sailors a night aches is the precursor of the disease and commonly that indicates a large consumption of liquor — occasionally men who have not been aches are attacked.

I submit that Dr. Woodhead and Wheel have drawn a "novum" pequirium. I further submit that Dr. Wheel does not corroborate Dr. Woodhead. In the first place a large consumption of liquor coincident with a night ache is not a causal necessity for those who stop abounds may also be affected. Secondly gastric derangement following a carouseal more frequently produces an preco, than a diminution of gastric acidity. It purports me that Dr. Woodhead having noted the experiments of Nock, Kestle, and Peisch, should not have mere closely examined this evidence.

Mostly a night ache has much to do with the determin-
ning of an attack, though not from the consumption of liquors, so much as that of grievous food-plagues and fruit in which Jack takes such especial delight after a voyage or regulation diet. However as Dr. Woodhead suggests, Saturday night's carouses and Sunday dyspepsia, with the weakened vitality due to abstinence from food, are doubtless potent factors, but certainly not the mere hyperrhebic from the week-end. I have criticised these statements for I anticipate that is long that I shall show that as in tuberculosis, so in other syphilitic diseases whose chief localisation is the intestinal canal, a gastric hyperacidity is at times a safeguard against the effects of the oral infection of the disease.

Further than this Dr. Brindon's opinion may be strengthened in one particular with altered in another, believing now (as he is quoted, I find no such opinion in his first edition) that diarrhoea is neither more or less likely to pass into cholera when the latter is epidemio, whether it be left alone or encouraged by purgatives, he may at some time show that those with an acute caunch are better left alone though their after care would necessitate zealous attention. I assume a trust with fairness that diarrhoea, intestinal or unintestinal, more frequently involves an increase of the secretion than the perforation of the intestines, also that constipation involves more frequently a decrease of the latter than the former. I assume further that intestinal fever, whilst dependent upon increased secretion is not accompanied by proportionate increased absorption; this I believe for as in health there is sufficient
absorption to dry to an extent the intestinal contents in their passage from the duodenum to the ileum, no known intestinal action would produce a fluid shot if its action did not affect the ratio of secretion to absorption. In diarrhoea there is a greater secretion than in health, and in constipation. My experience shows me that excess of secretion there is intestinal: flaring or flushing and with flushing there must be lessened absorption. With the amount and rate of absorption depends the effect of a given quantity of choleraic ingesta. With constipation there is a slower downward movement of the food; consequently and less frequent flushing; hence whatever absorptive power there may be a longer opportunity of taking up choleraic materials. The material also has a longer time in which to attack itself.

Toch had not only to alter the chemical condition of the bowel, but also to infect dogs fed upon choleraic geese; he had also to paralyse intestinal muscle.

The New South Wales public of Europe was repulsed with cholera, a circular was sent to various authorities by the Board of Health for public exhibition and instruction, which advocated the use of aperients and the ciderising (I must hardly claim the paternity of this word, but it suits the instructions well) of the bowels of the colony. Regard it as an order to lessen secretion, and to lengthen the chances of absorption. I believe in the reverse; I believe that any intestinal laxity short of the production of weakness to be safer than other states and that the health reacted condition of the tissues to be a safeguard to the subject.
Rock's bacilli has in times of epidemics been found in the dejecta of new choleriac subjects. I am anxious to know if any of these subjects were suffering from constipation. I suspect not.

(Since writing the above I have read that the physicians of the Karachi Hospital have treated cholera by administering citric acid whilst the stools remain alkaline.)

Reference XIV

In a paper by Dr. Sandwith on four hundred cases of Phthisis, a valuable collection of facts is exhibited. Dr. Sandwith says "one urinai had tubercle on the outer surface of his trachea. I only mention it on account of its rarity." He makes no reference to any case of tubercle in the trachea.

The further says in discussing the same series of cases "in our Soudanese I found tubercle inside the bladder."

Reference XV

Plagge says that the spread of military tuberculosis is by "the dissemination through the blood stream — in the form of bacilli or their spores. The probability now seems to be that the real starting point is often a very recent patch of tubercle which happens to be so situated that an injection of the blood stream is a necessary result of its presence."

Reference XVI

In chronic tubercular affection the mode of spreading seems to be the gradual invasion of the lymphatic channels from point to point. Some observers think — the ileum (is affected) by the spura which are swallowed (in pulmonary phthisis). Apparently then the spread of tubercle from the lung to the intestines can be causes by first the spura; secondly dissemination by the blood stream. The majority of evidence, however, goes to show that the first method is the more frequent in pulmonary phthisis. Dr. Sandwith noted that
where there were many intestinal ulcers there were often no gland changes. If the infection in such cases were due to the second cause it is difficult to explain that facillities, via the heart and with a choice of any organ select the lower end, unless. Under what circumstances will such infection of the intestines occur? I believe when the gastric juice is not very active. By this I mean when there is a deficiency of acid secretion, or so I have explained before, now there is not an acid cathartic or fermentative change in the viscera. I have somewhere read of the difficulty of infecting dogs with a tuberculoustier. Such a carnivorous animal requires a higher degree of acidity to digest its food. The hereforbough cow, with its weaker acid secretion and its less alkaline food, is easily infected.

The elective power of tubercle is demonstrated in usual cases in which a spread from one kidney down the ureter, is accompanied by the infection of the other. But I doubt whether there is an elective power to be considered for this cause which affected the first kidney has probably affected the second, and the means of contaminating the first should be sufficient for the contamination of the second also.

Primary tuberculosis of the kidney is not frequent, and it commences usually in the cortex and not in the pelvis, and chiefly in the interstitial stroma where it occurs as a manifestation of tuberculosis, and of several specimens examined by James and Steiner no tubercle was found in the tubes. Struve also says that they appear first in the cortex and from his description of the tuberculous cavities bursting into the pelvis, I assume that he too finds that the process...
commences in the intestinal worms. Ehrlich finds its commencement in a similar position. In a specimen now before me, I find such a focus clearly defined.

Knowing commenced as such, tuberculosis spreads by direct continuity along the mucous surfaces of the urinary passages, from the ureters. It is noticeable, however, that when tubercle grows on the renal mucous surfaces, the pyojepticous changes are those of suppuration of the part affected. A secretion of pus formed in this way, unless to a great extent protected by bacilli from the action of the urine's acidity. Again, the more severe the tuberculosis of the kidney, the poorer does the secretory function of the kidney cease. I have seen one such case in a patient upon whom I made a necropsy in the Devonshire (Wells) Cottage Hospital in 1889, on behalf of my friend Dr. Alfred Carr, now of Bologna.

Tuberculosis of the bladder is but seldom primary, and more frequently follows a similar condition in the higher urinary tracts, or still more frequently in the prostate and vesicles, the secretions of both of which are alkaline. Spreading as above, a condition similar to that of cystitis quickly appears shown by alkaline urine, and the formation of pus.

From this study of tuberculosis of the urinary tracts, I have learnt that the process almost invariably commences away from the acid urine—being in the renal cortex or the alkaline genital tracts—and that it spreads by direct continuity, forming a protective alkaline (pH) fluid as it spreads, also that the phenomena of not alkalinity of the urine, is an important condition, without which the progress of the disease would be but meagre. I believe that the urinary tracts
proper are specifically immune owing to the acidity of their fluid contents.

Remembering that the bladder is a Tidal organ, I have considered as to work for its restlessness and abstinences, are the factors of its freedom. Tuberculosis in the bladder has never been found in the female, though it has been found in the disease has been found in the bladder wall following primary in an adjacent organ, such as the uterus. Women secrete less urine and urinate less often than men, hence although the Tidal variations are less frequent, and the total amount for flushing purposes is also less, the female bladder is relatively immune owing to it more frequently containing urine.
Section B.

No. 1. I have spoken frequently in the earlier parts of this paper of the immunity of certain organs, whose contents are acid, from disease of bacterial origin. I will now deal more fully with the evidence I have gathered from authority as to the efficiency of acid and acid solutions as a bacterial deterrent.

It is frequently observed that pathogenic bacteria which form lactic or lactic acids do so at the expense of their own vitality, though many a pathogenic bacteria are certainly acid dwellers. A noticeable process to this rule is shown in the bacteria causing dental caries; if doubt can be deduced from Miller's experiments, and also according to Woodhead (see page 34) in the case of thoracic bacteria.

I propose to consider a little more the following table which is in itself instructive.

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Habitat</th>
<th>Reaction of habitat</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Staph. Pyog. E.g. (Rothbach)</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>3. Staph. Spyga. (Rothbach)</td>
<td>Lymphatics</td>
<td>&quot;</td>
</tr>
<tr>
<td>6. Pneumococcus (Biedlander)</td>
<td>Paracoccic Spatium.</td>
<td></td>
</tr>
<tr>
<td>7. Spic. Chol. (Koch)</td>
<td>Deficient</td>
<td></td>
</tr>
<tr>
<td>8. Bac. Pyles. (Closs Gaffey)</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>9. Bac. Tuberculosis (Koch)</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>13. Bac. Bacis Urose. (Verneuil)</td>
<td>Urine</td>
<td>&quot;</td>
</tr>
</tbody>
</table>
Nov 1. 2. 3. succumb rapidly when submitted to the action of a weak acid or the acidity of the gastric juice.

Nov 5 96, together with 7, I have not found other than in alkaline solutions.

Reference XXXII

Reference XXXIII

Reference XXXIV

Reference XXXV

Reference XXXVI

Reference XXXVII

Reference XXXVIII

Reference XXXIX

The gonorrhea has been found chiefly in the female urethra, the mucous surface of the cervix, the male urethra and the pus produced from these surfaces. Its special habitat is columnar epithelium not connective tissue or squamous epithelium hence it is cervical, uterine, tubal, and urethral. It is not strictly speaking vaginal or peritoneal usually. The vagina is lined with thick stratified pavement epithelium. The bladder with stratified transitional epithelium, the male urethra with greater portion with columnar epithelium and the female urethra with stratified transitional epithelium according to some authorities, whilst others say that its lower part is covered with squamous and higher up with transitional epithelium. The uterus is lined with ciliated columnar cells in a single layer.

From the above facts can be drawn interesting deductions, but consideration must first be given to the specific nature of gonorrhea before discussing the immunity of tissues from it.

In the Book of Genesis, in chapter fifteen, a strong indication is given of an early acquaintance with the condition of urethral "issues," at times ten day every menstruation is given as a cause of such an issue in the male.

Without tracing the history of pathological opinions of this disease through the ages intervening, I will resume it at a more recent date.

In 1840, in 1840, Leistin gave the most common cause of gonorrhea in the application of irritating matters to the lining membranes - from leucorrhoea or menstrual discharges.
but gonorrhoea makes its specific virus — and the most
common cause of inflammation.

Reference XL.

Record opined in speaking of the characteristic causes
of the disease, that a virus is about in the majority of cases.
He maintained that gonorrhoea often arises from intercourse
with women who themselves have not the disease.

Brodsky says emphatically that from the very first of a
woman having a discharge, no matter what its origin, she is
liable to give a discharge to a man.

Poirier conceives that gonorrhoea is much less
frequently contracted from contagion than from using
toilette.

Reference XLII.

Monsieur speaks of gonorrhoea passing over
the influence of connection with a menstruous woman or
with a woman who has been in contact with infected
epidemics. He has few described and systematic cases
recorded.

In 1888, Weiss wrote of a specific microbe which
he found in 1879. He was corroborated in 1885 by Brodhart.

In 1884, Eichhson differentiated clearly between a
specific and simple urethritis, and dwells upon the
difficulty in reaching this differentiation in practice. He
distinguishes the one from the other by the greater intensity
and the presence of peculiar, but immediately continuous
infections (of simple urethritis) is extremely difficult,
especially from the subacute forms of gonorrhoea that are
common in London. I cannot accept this differentiation
in the strict way in which Eichhson proposes. He speaks
of gonorrhoea as an infectious urethritis, whereas he assigns
causes of simple urethritis, pneumonia and other.
Reference XLVII

Diatheses, persons in course, excessive fear drinking, the use of instruments &c. However, he continues to pay, that it is impossible to tell where urethritis ends and gonorrhoea begins. This in my opinion forecloses the differentiation of any validity, especially since he is of opinion that gonorrhoea may arise de novo in females and that it may be generated by either impure, or indiscriminate intercourse. He does not accept in toto the auxiliary nature of the virus, paying that the actual nature "has not been certainly ascertained." Finally, Ehrlich expresses himself as follows: "it is probable therefore that the gonococcus -- is either the actual virus or in some way connected with its development."

Before concluding my retrospect I will dwell further upon matters suggested by the two pages quoted above.

Ehrlich says that gonorrhoea is an infectious urethritis. -- In January, 1890, I became acquainted with a lady and her husband who lived in congenial harmony, until now the ailments of any kind cast other, other those of the groom and Mrs. Ehrlich went on as in the one case, and the female partner of a married couple, advancing in years and honesty in the other. On the 15th of that month, W. A. consulted me about a patient which was apparently influenced by a genitul diathesis; for a month he was constantly on his back being compelled to use a bed pan. On February 20th, W. A. consulted me about a leukorrhoeal discharge. I found an eroded os cervix and a retrodisplacement of the uterus; the discharge was uncorrosive. Among other remedies the drugs of the drachm Per-Ellen was used. On Feb'y 28th, W. T. appeared with an inflammation of the urethra, this condition lasted for
fourteen days and closely simulated an ordinary specific
gonorrhoea. On April 3rd 1870 A was again suffering from
gonorrhoea, the discharge being heavy and ushered
were preceded
than before. He also had a distinct urethritic discharge. I
believe that the urethritic shown by W. A. was due to a combina-
tion of W. A.'s gonorrhoea and his diathesis and that he in
his turn infected her.

Eberth's paper that purulent urethritis can only be distin-
guished from the specific form by its lesser virulence and
its freedom from sequelae. By this he apparently means the
so-called "gonorrhoeal rheumatism" and inflammation of the eye.

During the year — 1886 — in which I worked at the bark
wards of the Royal Infirmary, Edinburgh, I remember for no
cause of gonorrhoeal rheumatism, I have never seen this condition
since. Gonorrhoeal pyelitis, I have seen but twice in
adults. In November 1886 I was consulted by W. B., noted for
a professional gentleman of undoubted character, a keen sufferer
of nodular and flesh pyelitis, and so marked urethral discharge; he
was suffering from pyelitis of the right to which condition he had
been prone as the result of an old accident to that joint. In
a few days the urethra had recovered, and he had developed
urethritis which lasted over a fortnight. With it he had a
purulent discharge, vesical tenderness, tenderness of the
glutes, enlargement and tenderness of the inguinal glands, and
for three months afterwards a fever. Early in October
I delivered W. B. of a healthy girl. I quote this case
believing in the moral integrity of the parties concerned, as
one of so-called "simple urethritis" which was certainly as
severe and as unamenable to treatment, if not more so,
than many cases of ordinary gonorrhoea.
To continue my retrospect - in 1870 it is said "According to history, smallpox is either pimple or gonorrhoeal. Apart from the history we cannot for certain distinguish between these two. In other words the one cannot be distinguished from the other by the standard of virulence, or by the occurrence of sequelae. It is also said that a pimple varicelous may produce most of the symptoms of one due to gonorrhoea.

Reviewing this retrospect I see how well after 1870 that pimple and specific urethritids were not differentiated from each other by cause or effect. For a while afterwards, the presence or absence of the gonorrhoea, possibly searched for by the medical amateur microscope, was the means of a hard and fast differentiation. At a still later period doubt is thrown upon the reality of such a plan. With the passage of a few years following the introduction of the gonorrhoea to the favourable notice of the public, the distinction, if any, was made by applying the test of mortality. If the patient were cured, we had gonorrhoea, if worse we had urethritis. There is force in this method, for the incurable suffer more severely, and the contagious variety is usually more severe.

In spite of more recent information I believe that gonorrhoea may be due to other causes than specific infection from specific gonorrhoeal matter, or it may be due to the latter. I believe therefore that an infective gonorrhoea may be specific.

Specific gonorrhoea (specific contagious gonorrhoea) is usually severe and is caused by specific infection only, either primarily or by addition to urethritis. If it is more specific (a bacterial contagious gonorrhoea) it may or may not reproduce itself. The compelling factors being
general disease or local conditions. If it do not arise from or produce primary chancroid, that it is mild in its course, (simple urethritis) but that though it do not arise from, it may produce a contagious gonorrhoea.

The following scheme will demonstrate this relation:

\[ \text{Gonorrhoea} \]

\[ \text{Specific} \quad ? \quad \text{Non-Specific} \]

\[ \text{Associated} \quad \text{Unassociated} \]

\[ \text{Contagious} \quad \text{Non-Contagious} \]

\[ \text{Contaminated} \quad \text{Associated} \]

\[ \text{Non-Contagious} \quad \text{Contagious} \]

(I need hardly say that I assert that the fearful gynaecological results of latent gonorrhoea in the male to occur when the disease has been specific only.)

Gonorrhoea in the female is but seldom uremic. If specific the germ will have but a temporary hold on the vaginal subcutaneous epithelium or in a menstruating uterus. In the male however the gonococcus will have a far longer grasp of the urethral columnar epithelium. Hence we hear much of latent gonorrhoea in the male, and hence females have a comparative immunity from this condition, and their male cohabiters from an acute attack of gonorrhoea from such a cause, though in males such a cause frequently infects and killing the
female.

After thus dealing with the biology of gonorrhea I will briefly discuss the immunity of these organs usually brought into contact with the virus.

A. The female bladder and urethra. In this section I will deal with the male bladder also. There is nothing yet proved to prove that gonorrheal coitus is primarily gonorrheal, and not due to receptacles. This may also partially account for the relative immunity of the female bladder which is normally less often infected.

In both sexes this organ's immunity may be due to the causes given at page 58. The urethra is not directly immune because of its anatomical condition only. A straight tube from a male bladder might be safe but not from a female bladder. The rare case of gonorrheal urethritis that I have seen in a woman was coincident with old Bright's disease.

B. The vagina. I advance a plea for this organ for comparative immunity because of its histological formation and the acidity of its secretion.

C. The uterus. The claims of this organ have few discussed.

References LV

References LVI

Returning again to Dr. Woodhouse's valuable work I learn that the diphtheria poison is always most active when alkaline, and that during an acid period to which he refers, that it is less virulent.

I beg here to be allowed to digress for a while to draw attention to some deductions that have influenced my practice successfully.
In 1888 it is alleged that "it is now agreed that the membranous
membrane of the tonsils and palate should never be forcibly removed."
I do not perhaps understand what is meant by "forcefully,"
but I would certainly remove the membranes whenever practicable
short of removing anything else with them. Such precaution is
follows the operation, I believe to do no harm. The membranous
membrane independently of the membrane, for they can be found in
the pharynx deep after the teeth have "cleared." For many
other reasons also I believe that the destructive process resulting
in the membranous formation which takes away carries the
process by protecting it against local applications and the
action of the "vis Medicativa naturalis," shown by the history
of the affected part to slough away.

I have quoted an extract from the Australian
Medical Gazette as an explanation of what I believe to be
a separate disease stimulating croupous.

Referring to Dr. Lewes' article in your issue of the 14th insta
(March 1890) upon membranous pharyngitis, I can strongly
endorse his every word and trust that you will allow me to
assist in drawing more attention to this condition. To his
remarks I would add the following, gained from an
experience of eighteen cases:

That when first seen the disease had nearly, if not
quite, reached its acme, at which it will frequently
remain for seven days. I therefore follow it to its
sudden in cases.

That successful local treatment of one tonsil is
frequently followed—by us means as a result—by a
similar eruption elsewhere in the throat; and so long
as the interval (in one case of nine days) between the
disappearance of the one, and the re-appearance of the other, that apparent relapses are observed.

That the decline is sudden; in some cases, extraordinarily so.

That no nervous symptoms follow.

That it does not depend upon pneumonias.

That in the western district, whence my cases are drawn, it occurs more commonly in hot, dry weather, all my cases being preceded in the summer, and worse of them during or after rain, at which times dysentery becomes more common.

I may remark, that the consideration of the above, and the points noted, by Dr. Lewes, especially its hematuria or purulent nature determines my diagnosis.

My last six cases have been treated with a paint of Bierio, mixed with a mixture of a saline opium, and occasionally quinine or potassium; but I believe the general treatment to be the best auxiliary.

I believe that Bierio acts upon some of these discharges in a good few years ago, marking its influence from the properties of gastric and gallbladder origin, its old time hospital treatment, and its congestion in those of pneumonias.

I have not seen suppuration in these cases nor evidence of contagion. The temperature varies between 100° F and 102° F being rapidly reduced under treatment. The shortest duration was two, the longest three days. The results have always been satisfactory. I have seen it associated with a watery diarrhoea, and found it to be catarrhal, though I am open to persuasion that it is a local clysis disease.

"I would like to hear as to the presence or absence of micro-

hological products of life." - Gerald S. Samuelson
The success with which my treatment herein shown was accom-
plished led me to use the same in diptheria. At that time I
believed in the co-admissibility of the local application of
strong acids which method was employed by Marchand de
Garavillet who was copied by Belinoueau and not con-
denmed by Bartheau. Having however a case of diptheria
in which the membrane was especially resistant to pilots, I
removed it and painted the exposed surfaces with Glye: Ac.
Carbon.: The next case I treated with phlegion and I asked
one part of the throat with carbolic acid the other with Hy-
chonic acid. The after appearance of the parts showed no
superiority of the one method over the other. However I gave
Carbolic Acid because of its anaesthetic properties. Preparad
Carbolic Acid with HClitide of mercury but had a poor
result from the former. These experimental contrasts were made
in the two cases quoted above, both of whom recovered with
(perhaps not through) the acid treatment, and including three
I have had forty cases since. Of these, but one was over
eight years of age, and death occurred in four cases all of
which had pronounced laryngeal symptoms.

The method I now adopt in all possible cases is as follows
- Introduce into the pharynx a half CP. lamp mounted
on a holder which acts as a tongue depressor, and leaves the
other hand free for the forceps or hook. I strip and even
perforate the affected part of to which I then apply Glye:
Ac: Carb:.

I believe in the efficiency of the Carbolic Acid treatment
not only because it is Carbolic but also since it is acid.

Why does not diptheria from the over constant
swallowing of discharge spread via the aerophagos ?
The peculiar anomaly of the successful use of lime water can be explained thus — the lime is precipitated on the secretion (degeneration) of the membrane, this lime precipitation and local applications have an immediate action on the precariously surfaces thereby exposed.

The Dakin's acid treatment, described by Parrot, has a far greater effect apparently, than treatment with its soda compound.

References LIX

In speaking of the microbes of stale or alkaline urine, refer to the bacterium urea (urea anaerobe of Bish) and the facultative urea of Leclat.

These microbes are not excited by the bacteria with a pathogenic power, but the clinician has spoken of them as a cause of catarrh, by their production of an alkaline formation in the urine when introduced into the bladder.

The question as to whether these organisms produce or depend upon alkalinity is not clearly shown by a review of current literature. Woodhead speaks of them (bacillus and micrococci) as occurring with, and Baggaw bringing about an alkaline formation. The latter also gives information as to a non-peptic and a-bacterial alkaline urine; whilst Roberts has recorded cases where a "non pathogenic" — the supposition is mine — bacterium, has been found in freshly voided acid urine.

More recently Wiure, Raben and Sanada have opined that urine facilli maintain the acidity of the urine, and do not depend upon pepsin.

I will not use time by quoting the various authorities who believe that the bacterias anaerobe were, or are
dependant upon it. day.

It has often been observed in pre-natal periods that
intrauterine catheterization was followed by ammoniacal fermentation of the
urine. I see no reason to doubt it, but I believe that the accident
occurred when cystitis, cataract, dilatation with its concomitant
alkalinity or neutrality necessitated the catheterization. Under such
conditions I can readily believe in the pathogenic power of the one
or both micro-organisms.

It is not easy to see how the bacteria can otherwise gain
access to the bladder in cases of cystitis or dilatation. For with
the exception of Sir W. Roberts article, there finds no evidence
of their occurrence in the healthy bladder. The theory that
abnormal rupture accounts for their presence requires evidence
that they are found in pus or blood elsewhere.

In a recent number of the *London Medical*
I have read that certain observers believe that the urine
bacillus is a pleomorphic variety of the colon bacillus. Indeed
the whole matter requires much more attention.

I have endeavoured to elucidate point of the points
thus left under question, and some observations shown in
accompanying Table I are of opinion—

1st. That the analysis of urine is independent of
bacterial agency.

2nd. That the bacteria depend upon and do
not cause alkalinity.

3rd. That they depend upon urine as the source
of their nutrition in urine from a healthy subject
4th. That the bacteria appear in the analysis
of urine.

I have considered the primary production by bacteria.
of an alkaline fermentation of the urine in vivo, but cannot
conceive it to be possible. That (vide Tew I) my first view
is true accounts for the occasional bad results of catheterization,
but I believe such a result to be impossible apart from
urinary infection.

Apparently then the bacteria are facultatively
pathogenic. Introduced by a catheter when used for
slight early or transitory infection, the bladder is immune
from their results—introduced by a catheter when used
for prostatic dis ease or anything that has produced
cystitis, dilatation, or chronic infection, the bacteria are
pathogenic.
Observations with blue litmus paper, not dipped in A. 6f and Microscope.

<table>
<thead>
<tr>
<th>No</th>
<th>Treatment</th>
<th>12 hours</th>
<th>24 hours</th>
<th>36 hours</th>
<th>48 hours</th>
<th>60 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>—</td>
<td>Neutral</td>
<td>Neutral</td>
<td>Alkaline</td>
<td>Alkaline</td>
<td>Alkaline</td>
</tr>
<tr>
<td>B</td>
<td>(Addition of per aqua.</td>
<td>Neutral</td>
<td>Neutral</td>
<td>Alkaline</td>
<td>Alkaline</td>
<td>Alkaline</td>
</tr>
<tr>
<td></td>
<td>Hyd: Seck. 1:1000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>* Dito. cito*</td>
<td>Neutral</td>
<td>Neutral</td>
<td>Alkaline</td>
<td>Alkaline</td>
<td>Alkaline</td>
</tr>
<tr>
<td></td>
<td>1:2000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>* Dito. cito*</td>
<td>Neutral</td>
<td>Neutral</td>
<td>Alkaline</td>
<td>Alkaline</td>
<td>Alkaline</td>
</tr>
<tr>
<td></td>
<td>As B. bei Bacilli Introduced</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>—</td>
<td>Neutral</td>
<td>Neutral</td>
<td>Alkaline</td>
<td>Alkaline</td>
<td>Alkaline</td>
</tr>
<tr>
<td></td>
<td>As B. bei Bacilli Introduced</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>As B. bei Bacilli Introduced</td>
<td>Neutral</td>
<td>Neutral</td>
<td>Alkaline</td>
<td>Alkaline</td>
<td>Alkaline</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>As B. bei Bacilli Introduced</td>
<td>Neutral</td>
<td>Neutral</td>
<td>Alkaline</td>
<td>Alkaline</td>
<td>Alkaline</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H</td>
<td>* As B. bei Bacilli Introduced*</td>
<td>Neutral</td>
<td>Neutral</td>
<td>Alkaline</td>
<td>Alkaline</td>
<td>Alkaline</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day</td>
<td>Tube A</td>
<td>Tube B</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>---------------------------------</td>
<td>---------------------------------</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alkaline</td>
<td>Alkaline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cloudy</td>
<td>Cloudy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>In three days</td>
<td>In three days</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Third day:</td>
<td>Third day:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sterilized in steam chamber</td>
<td>Sterilized in steam chamber</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Filtrated.</td>
<td>Filtrated.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inoculated.</td>
<td>Inoculated.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>As Neutral</td>
<td>As Neutral</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Phosphoric acid added to</td>
<td>Phosphoric acid added to</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>slight acid reaction</td>
<td>slight acid reaction</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fourth day</td>
<td>Fourth day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neutral</td>
<td>Neutral</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Percentage of water decreased</td>
<td>Percentage of water decreased</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fifth day</td>
<td>Fifth day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alkaline</td>
<td>Alkaline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bacteria</td>
<td>Bacteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Percentage of water decreased</td>
<td>Percentage of water decreased</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sixth day</td>
<td>Sixth day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alkaline</td>
<td>Alkaline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cloudy</td>
<td>Cloudy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bacteria</td>
<td>Bacteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Percentage of water decreased</td>
<td>Percentage of water decreased</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Seventh day</td>
<td>Seventh day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alkaline</td>
<td>Alkaline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bacteria</td>
<td>Bacteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Percentage of water decreased</td>
<td>Percentage of water decreased</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Eighth day</td>
<td>Eighth day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alkaline</td>
<td>Alkaline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bacteria</td>
<td>Bacteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Percentage of water decreased</td>
<td>Percentage of water decreased</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**Test III**

<table>
<thead>
<tr>
<th>Tube A</th>
<th>Tube B</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 days old</td>
<td>2 days old</td>
</tr>
</tbody>
</table>

**Sterilized.**

**Neutralized with Phosphoric Acid for three days when bacteria are few.**

**Neutralized with Phosphoric Acid for three days when bacteria are many.**

---

* Possibly this indicates something other than water had been broken up by the bacteria in Tube A before the sample was sterilized. In these tests I used my own urine. I believe it to be normal.
Typhoid Bacillus.

It is found that this bacillus can grow in an acid medium, and indeed, media are purposely acidified to prevent other micro-organisms from growing. Further than this, the bacillus secretes an acid, and not alkaline, proteolytic product. In these particulars it differs from the other bacilli I have considered. It is a question, however, as to whether the bacillus of Eberth is pathogenic. Dr. Gabriel Volle has shown in his "Le Bacillus coli Communis dans les Réseaux avec le bacille d'Eberth," that Eberth's bacillus is frequently met with in typhoid fever, though the Bacillus Coli Communis for the typhoid bacillus itself is distinctly comparatively rarely met with.

Dr. Volle concludes that the coli Communis Bacillus (Escherich) is but an intermediate form of Eberth's bacillus. He shows how Escherich's bacillus, when cultivated in fermenting fecal solutions, may become more virulent than Eberth's bacillus. Possibly it would be more correct to speak of Eberth's bacillus as a modified Escherich's bacillus. I saw however find but slight information upon this matter, and so can say but little. I may however assume the possibility of typhoid fever being due to Escherich's bacillus after treatment in fermenting feces, and not to Eberth's bacillus in which case this seeming exception that "Pathogenic bacillus prefers an acid medium and produces an acid product is explained. If however Eberth's bacillus be the pathogenic factor, an interesting analogy is seen between its acid protolytic product, from its alkaline habitat, and the alkaline protective product of the tubercol bacillus, should we be located in an acid habitat. My last digressing should be passed in order to allow me to suggest the following points — if Eberth's bacillus be the pathogenic factor, then a treatment by alkaline injection as shown by Dr. Bumont in "La Dynamique Médicale" Dec. 1872, page 8 is indicated.
Escherichia fecalis is the factor, then the Citric Acid Cholera Treatment of the Vreeland Hospital might be of avail.

It is interesting to compare the luxuriant growth of E. coli fecalis in a carbolized potato-agar medium and the saltb treatment of the disease.

It has been found that inoculation experiments with focal cultures of Escherichia fecalis have been made with success.
Having studied the partial immunity of certain tissues from the above specific diseases, it is now my duty to explain how and in what way the effect which favours them, and how general specific immunity may be explained.

A. The products of bacterial growth (alkaloids, albumoses, etc.) have been shown in certain instances to be antirncreus and specific in their action on the bacteria by which they are produced. It has also been shown that a relative excess of the product to the causative organism proves fatal to the latter. The first point was shown in 1887 by Bopp in his study of typhoid fever, and in 1889 I was shown a letter from M. Pasteur which corroborated Bopp. The second point has, I believe, been demonstrated by Koch's tuberculin treatment.

With a view to testing the effect upon culture colonies of acidifying their culture media, I made the following tests. The medium used was nutrient gelatine—I could procure agar-agar at my far away home—sterilized according to the methods recommended by Crockshank. I inoculated these tubes with a purified culture from a deep cerebral abscess. These I labelled A, B, and C in four days. I incubated with Shoch's method and glass preparations from each, and found nothing but the purified culture. The tub cultures were similar to those figured by Crockshank, save that the culture may have been paler. I then placed B and C in a sterilizer for one hour a day for six days. I then added Acetic Acid to C until I produced an acid reaction; in two days I alkalized C. I then filtered and re-sterilized A, B, C, which were finally inoculated from A. The days afterwards there was no appearance of any...

References:

LXXII

LXXIII

LXXIV

LXXV
growth in C, B was full of Pseudocoecii. The same observations were made in control rats. A tube of the original medium was chemically treated in the same way as C, sterilized, and inoculated. Its macro- and microscopic appearances were similar to those of A.

A period of 3 days with facts: Brucella suis Subtilis and other acetone and a solution from a tetanised guinea pig gave similar results; indeed I have demonstrated the variation in pathogenicity of the Pseudocoecii cultures to —. Using samples B & C after sterilization, chemical treatment, and re-irritation I injected 0.01 cc. each side of a rat. D produced a phlegmonous abscess from which I cultivated the coccius again. The effect of C was transient.

Having learned that the Stass-Otho method of deriving

Reference LXXVII, LXXVIII

purifying alkaloids depends upon the pellability of the ratio of these alkaloids as evidenced by the treatment of the material with Tannin acid, I sought an explanation of the foregoing observations in the study of the increased pellability of the acid salt produced. Having before me only the fact that alkaloids are more freely soluble in the alcohol of bodies than in Alkaline, I had further to seek a that Rocke's reference treatment depends upon a peculiar condition which I will now elucidate.

Bacteria, parasites, or protozoa, or force their host to do so, in material, toxic, not only to their host, but to themselves. Their attack upon a tissue may be combated by chemical toxicity, or by a ready or rapid removal of their products. If a considerable amount of the toxin be already formed the phagocytics that removes but the organism remedies the attack but partially, and leaves the soluble toxin to carry on its work. Should the pellability of the toxin be increased

Reference LXXVIII

Reference LXXIX
by its combination with an acid, or by its treatment with glycine, one or both of the following will occur.

The osmotic processes between the bacterium and its circumsurrounding fluids will be profoundly changed; the rapidity of the excretion of the toxins through the agency of the lymphatic canal and capillaries will be increased, the bacterium is not at its best, (or worst for the patient) and its products will be allowed a shorter time to work their toxic ends. (Does the rise in temperature following Koch's tubercle treatment indicate an absorption of toxins, other than that rejected?)

I may parenthetically remark that I anticipate that so long the bacterium will hold a less acquiescent pathological position. Dr. Parke has shown, speaking of changes attendant to bacterial invasion, pages 24, 25, a suitable and nutritious medium to be produced, certain physical and chemical changes are necessary, &c. &c. Comparing this with the methods of preparation of culture medium, described by such authorities as Brockbank, Woodworth, Godwin, and Penrose, (Bigg), it is recognized that the bacterium in its natural or experimental habitat, is as the eight months foetus in an incubator. In short this will be relegated from the position of a primary, to that of a secondary factor in pathology.

Finally, let me remark that if there is weight in the consideration of the physico-chemical conditions of a medium, of these changes of the elements, antecedent to bacterial invasion, then is as much in the consideration of those conditions that defy invasion.
The immunity conferred from further attacks upon one who has suffered from a specific disease, may possibly be due to circumstances analogous to the primary immunity of a given organ to a given specific disease.

I have endeavored to show in the above how a difference in the cosmic factors relating to the bacteria, probably determines its weak or wise; I have shown how by the saturation of the bacteria in its own waste products is followed by its death.

Let us consider further the close analogy in action, site and symptoms, between the pneumoniae and remember that a attack of either does not confer immunity from the other. If it is suggested that immunity from further attacks is due to the union of pabulum, the suggestion to gain faith must show that, unlike other chemical or morphological elements in the body, that this pabulum is not renewed when used up. Considering again the principles of the Shear. Otto method, and my own researches to the effect of combusting the bacterial products with organic acid radicles, it immediately suggests itself that the alkalinity of the body fluids retards the persistance of the products, and in process of time interferes with the vitality of the bacteria, and thus results in the cure of the patient.

Still the bacterial products are not removed, and though their increase is prevented by their own presence, i.e., by their effect on their producer - yet they remain in the system and for many a year may affect a fresh infection just as they affected the former. It is suggested that acquired immunity is due to an established toleration by the morphological elements, but it is not clear whether this toleration is the property of the ever changing soluble components of the cells or of the ever changing insoluble
components that were yesterday formed, are today used and tomorrow will be replaced.

I have learnt to believe that acquired immunity is due to the cause cure by use of sterilized pure cultures. This explanation is certainly more scientific than the following which is merely a circulus in definendo. "Immunity produced by the attack of a specific disease must then be looked upon as an acquired tolerance or adaptation of the cells of our bodies to the specific poison of the special bacterium of that disease."
Index of References

Reference No 1 "Lancet." 1st Sept 1892
II "Systems of Dental Surgery." Druce. 1859
III "American Systems of Dental Surgery.
IV "Practical on Dental Caries." Magotet. 1821
V "Bacteria and their Products." Woodhead. 1842. 1890
VI "Dental Pathology & Medicine." Warren. 780
VII "Essentials of Bacteriology." Ball. T. 107. 1871
VIII "Human Physiology." Poore. P. 177. 1884
X "Bacteria &." Woodhead. 780
XI "Idem." 7180
XII "Idem." 7180
XIV "Lancet." 4th Sept. 1892
XV "Principles & Practice &." Tagge. Vol. I. P. 792
XVI "Idem." 793
XVII "Lancet." 4th Sept. 1892
XVIII "Principles & Practice &." Tagge. Vol. I. P. 794
XIX "Bacteria &." Woodhead. 7222
XX "Pathological Anatomy." Jones & Smeeking. P. 97. 1875
XXI "Idem." 779
XXII "Theory & Practice of Medicine." Bristow. 7844
XXIII "Science & Art &." Erichson. Vol. II. P. 92
XXIV "Theory & Practice &." Bristow. 7842
XXV "Idem." 7847
XXVI "Science & Art &." Erichson. Vol. II. P. 1045
XXVII "Pathological Anatomy." Jones & Smeeking. 7709
XXVIII "Theory & Practice &." Bristow. 7867
"Pathological Anatomy." Jonas Säveking. P.209
"Elements of Human Physiology." Pomer. P.155
"Beitrag zur Kenntniss der Gruenke. Quaestor. (Arch. Gym) 1827.
"Elements of Anatomy." Klein. P.21
"Elements of Anatomy." Klein. P.251
"Elements of Anatomy." Klein. P.269
"Elements of Anatomy." Klein. P.265
"Elements of Surgery." Lethow. P.371. 1844
"Science & Art of Surgery." Oskers. Vol. II. P.1068-g
"Idem." P.1068
"Idem." P.1069
"Idem." P.1068
"Idem." P.1069
"Idem." P.1069
"Idem." P.1069
"Idem." P.526
"Bacteria +" Woodhead. P.305
"Idem." P.307
| LVIII | "Clinical Medicine." Trueman. Vol. II | 1.584 |
| LX | "Manual of Bacteriology." Crookshank. | 1.377. 1890 |
| LXI | "Bacteria v2." Woodhead. | 1.424 |
| LXII | "Principles and Practice v2." Paget. Vol. II. | 5.580 |
| LXIII | Idem. | 1.582 |
| LXIV | Idem. | Loc. Cit. |
| LXVI | "Lancet." 17th Dec. 1892. | 1.1421 |
| LXVII | "Bacteria v2." Woodhead. | 1.197 |
| LXVIII | Idem. | |
| LXIX | "Lancet." 8th Oct. 1890. | 1.848 |
| LX | Idem. | 1.849 |
| LXX | "Manual of Bacteriology." Crookshank. | 1.585 |
| LXXI | "Animal Alkaloids." Ashby. | 1.88.9 |
| LXXII | "Tomatoes." Farguier. | 1.92. 1892. |
| LXXIII | "Bacteria v2." Woodhead. | 1.363. |
| LXXV | "Tomatoes." Farguier. | 1.138.9. 1891. |
| LXXVI | "Tomatoes." Farguier. | Vaught. | R.192. 1891 |
| LXXVIII | Idem. | 1.912. |
| LXXIX | "Tomatoes." Farguier. R.92.67 deg. | |
| LXXX | Idem. | 1.89.40 |
| LXXXI | "Bacteria v2." Woodhead. | 1.378. |