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S Y M P T O M A T I C or S E C O N D A R Y
P A R O T I T I S with special reference
to E T I O L O G Y.

T H E S I S.

Submitted for the degree of M.D.

by

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SYMPTOMATIC or SECONDARY PAROTITIS, with special
reference to ETIOLOGY.

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I N T R O D U C T I O N .

The present work was begun with the object of deciding if possible the mode of the spread of infection of the Parotid Gland in a case of Secondary Parotitis observed clinically.

It has so happened that within a comparatively short period the author has had opportunities of observing several other Cases of Parotitis, each following on a different primary diseased condition.

Partly for the clinical interest which attached to these cases and partly as additional evidence in favour of the duct spread theory of infection in Secondary Parotitis, these cases have been recorded in the form of a thesis.

Parotitis is still relatively common and not infrequently leads to a fatal termination and the writer believes that it is only by recognising the true source of infection that appropriate means may be taken to prevent, what is always a serious complication, the onset of Parotitis.

CASE I.

A female patient, A. A. aged 25 years, was admitted to the Royal Infirmary, Edinburgh. on November 5th, 1910.

Complaint Swelling in the left side of the face.
Pain in the left side of the chest.
Feverishness.

History. Nine days before admission patient experienced a shivering sensation, and felt very cold. She went to bed early that evening, but in the morning felt quite well and able for her work. At this time she was nursing her brother, who was suffering from Suppurative Tonsillitis, the patient herself had complained for some weeks of an ulcerative condition of her mouth.

Five days later, four days before admission, the patient having been well in the interval, the left side of her face began to swell; she consulted her doctor who told her she had a "gumboil". The following day the patient was able to go about her work, but felt "out of sorts". The swelling in the face became more marked each day and four days after its onset the doctor, who had ordered her to bed the previous day, diagnosed Pneumonia and patient was admitted to the Infirmary, nine days after the initial symptom.

State on admission

On admission patient complained of pain in the left side of the chest, and pain in the region of the left parotid.

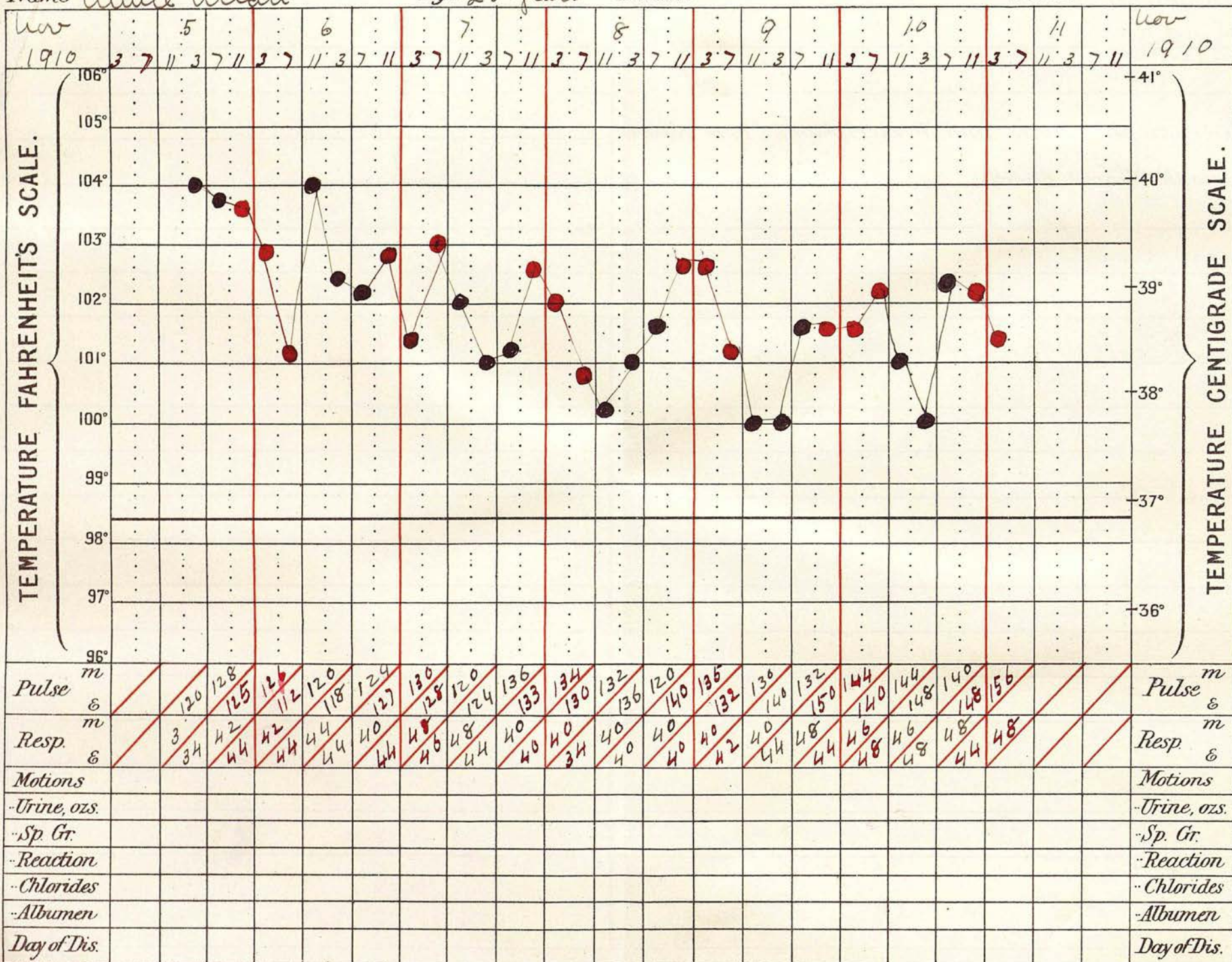
4. hourly Temperature Chart.

Case. I.

Name *Annie Allan*Age *25 years*

Disease

Result



On Examination

Her face and lips were cyanosed, the skin was moist the patient perspiring freely. Breathing was embarrassed. There was a swelling over the region of the left parotid gland, extending upwards to the zygoma, downwards to the angle of the jaw and forwards to the angle of the mouth.

Respirations 34: Temperature 104.

Pulse, 120 beats to the minute, regular but soft.

Blood Pressure, 110.

Respiratory System

Patient complained of pain in the left side of the chest. There was no cough and no sputum. Dyspnoea was not present. There was no limitation of movement of the chest.

Palpation

There was no increase of vocal Fremitus which was present in all areas.

Percussion

No dulness was revealed on percussion.

Auscultation

Anteriorly. Breath Sounds vesicular in all areas.

Posteriorly. Apices vesicular breathing on both sides. A small patch of distant tubular breathing was present at upper border of left

scapula; some rhouchi were present, heard during both inspiration and expiration.

Bases. Breath Sounds vesicular with prolonged expiration numerous rhonchi present.

Axillae Friction sounds heard low down in the left axilla.

Circulatory System

Pulse, 120 per minute, regular in time and character upstroke quick, apex fairly well sustained, no decrotism, Blood pressure 110.

Subjective No pre cordial pain or discomfort. No dyspnoea, Cynosis of the face and lips present.

Apex Beat Three and a half inches from the middle line in the fifth interspace, palpable as a feeble impulse.

Percussion

Upper border of the heart is at the third rib.

Right border of the heart three quarters of an inch from the midsternal line.

Left border of the heart three and three quarters inches from the midsternal line at fifth rib.

Auscultation.

Heart Sounds feeble, rythm tic tac, no murmurs present. Haemapoietic System. The lymphatic glands of the neck were not enlarged. There was no enlargement of the spleen or Thyroid gland.

Blood Examination

Red Blood Corpuscles. 5.000.000.
 White Blood Corpuscles. 9.000.
 Haemoglobin. 80 %.

Differential Count.

Polymorpho-Nuclear Leucocytes.	74 %.
Small Lymphocytes.	18 %.
Large Lymphocytes.	8 %.

Glycogen Reaction was negative

Alimentary System

An ulcerative Stomatitis was present involving both the gums and cheeks. Several teeth were carious and the mouth was very foul and dirty. The left parotid gland was markedly swollen, was firm and brawny. The skin over the gland was tense and shiny. There was no fluctuation.

Patient experienced great difficulty in opening the mouth, the pain caused by the attempt to do so being very severe, most marked behind the angle of the jaw.

There had been a tendency to constipation .

There was no vomiting or abdominal discomfort.

Palpation:- There was no tenderness or pain.

Abdomen moved freely.

Stomach and Liver :- Nothing to note.

Urinary System.

No pain or difficulty with micturition.

Urine Reaction acid: Specific gravity 1035.

Amber coloured: Deposit of mucus.

A trace of albumen was present. There was no blood: pus or sugar: Chlorides diminished.

Urea 16 grains per ounce.

Microscopically:- Some Granular debris, no Casts, blood or pus.

Nervous System.

There were no nervous symptoms.

Treatment and Progress.

On admission patient was put to bed and given the following prescription.

Recipe. Liguoris Ammoniae Acetatis
Spiritus Etheris Nitrosi.

Diet consisted of milk and chicken tea. 4 hourly. Castor oil was administered: Poultices applied to the back. In the evening patient was a good deal easier, was given Paraldehyde, two drachms, and slept well.

Next morning 6-XI-10.

Patient was easier. The mouth was still foul and dry. An Ichthyol Soak was applied over the parotid swelling which was firm and tense. In the evening patient had some difficulty in swallowing. Leucocytes. 8.600.

7-XI-10. Condition much the same, patient took nourishment well during the day, but the difficulty in swallowing returned in the evening. This was relieved by a Steam Kettle with compound Tincture of Benzoin, and by the application of a light linseed poultice to the throat. Patient was put on whisky half an ounce four hourly.

Leucocytes. 12.500.

8-XI-10. Bowels moved freely.

Parotid swelling has subsided. Patient is taking nourishment well and breathing quietly. Leucocytes. 10,000. Glycogen Reaction. Positive.

9-XI-10. Pulse rate increased to 140 per minute very weak. respirations. 48.

The chest is now full of moist Crepitations, There is slight increase of dulness posteriorly on the left side. Patient complains of pain over the precordia. On examination the heart sounds are feebly audible, being almost obscured by the breath sounds, no pericardial friction audible. In the evening patient is passing urine involuntarily. Pulse now weak and intermittent. Patient put on strychnine 1/60 grain four hourly alternating with the whisky : Patient was given Nucleinic acid m xxx hypodermically.

10-XI-10. Pulse stronger, less intermittent, The pain over the prae-cordia has disappeared. The parotid swelling is less marked, no definite fluctuation is to be made out. There is now some swelling of the left side of the neck extending downwards from the lower limit of the parotid

swelling. The Leucocytes under the influence of the Nucleinic Acid have risen from 12,000 to 19,200, but the Corpuscles are small and ill-formed.

II-XI-10. Patient breathing which became greatly embarrassed towards mid-night of the previous day, became shallower towards 4 a.m. Breathing became greatly laboured, patient became restless, pulse rate increased to 150 per minute. Digitalin and Strychnin were given hypodermically without result, patient died quietly at 6 a.m.

Post Mortem Report.

Body medium height, good nutrition. A swelling is present in the region of the left parotid gland. There is a brown pigmented spot in the right iliac region, the size of a penny. The muscles are dark in colour, the cervical veins are distended.

Thorax.

Left Pleural Cavity The surfaces of the pleura are adherent by fibrin; and pus is lying in pockets between the layers.

Right Pleural Cavity. A few adhesions are present at the right apex posteriorly.

II.

Pericardium - Acute fibrinous pericarditis.

Lungs. Both lungs are full of Septic infarcts and purulent foci varying in size from a pin head to infarcted areas corresponding to several lobules. Root and Tracheal glands are greatly enlarged. Thick Muco-purulent secretion is present in the bronchi. Nothing further to note in the Trachea Pharynx or Oesophagus.

Abdomen

No evidence of peritonitis.

Kidneys. Right Kidney medium size, Capsule strips easily. On section minute points of suppuration are present, scattered through the substance of the medulla. Cloudy swelling is present throughout the organ.

Left Kidney presents similar change.

Spleen Shows Acute Septic change.

Thymus - Normal

Veins Ante-mortem Thrombus is present in the External Jugular and Innominate Veins of the left side, passing downwards in a vessel from the region of the left parotid gland.

Parotid Gland.

The lower part of the left parotid gland is honeycombed with points of suppuration, these points in the early affected lobules are in the centre of the lobules.

Cultures.

Agar slope tubes were inoculated from the parotid gland, Pericardium, Pleura and Lungs. Pure cultures of Staphylococcus Pyogenes Aureus developed in each case.

Summary of Post Mortem Report.

Suppurative Parotitis. Thrombus in External Jugulae vein of same side, extending downwards to the innominate vein. Septic Emboli in the lungs - infarct and abscesses.

Minute abscesses in kidneys.

Acute septic spleen.

Acute pericarditis.

Cultures from all these sources gave pure cultures of Staphylococcus Pyogenes Aureus.

ETIOLOGY of the PAROTITIS.

The study of the antimortem record of this case in the light of the post mortem report, is extremely instructive and taken along with the bacteriological and histological examination of the various organs affords strong evidence in favour of the theory that in Symptomatic Parotitis infection takes place by way of Stenson's duct. The argument in favour of this mode of spread in this case comes under two heads.

EVIDENCE in favour of DUCT SPREAD.I. Indirect Evidence.

We wish to establish that the Parotitis relatively to the renal, Pulmonary and Cardiac conditions was a primary event, and that therefore the only available source of infection of the Parotid was the mouth. This argument will be supported by evidence obtained from the clinical Post Mortem and Histological findings.

II. Direct Evidence.

By Histological examination of the affected Parotid Gland we will submit evidence that infection could occur only by way of the duct, and not by the blood stream.

Study of the Clinical Record.

The essential points in the history of the case are as follows:-

1. The patient who had been "out of sorts" for several days was nursing a case of Suppurative Tonsillitis.
2. She was suffering at this time from an ulcerative condition of the mouth, which was dry and septic.
3. She suddenly developed a swelling of the parotid gland on one side at the time taken for a "Gumboil" Subsequently proved however to be a Parotitis.
4. Four days later she developed symptoms pointing to Pneumonia.
5. During six days in hospital her Temperature Pulse and Respirations and the Physical Signs in the chest pointed to a Broncho-Pneumonia.
6. Four days after admission she developed precordial pain which lasted for one day only.
7. Five days after admission she developed Thrombosis of Left External Jugular Vein.
8. During the course of the illness only a trace of albumen was present in the urine. Pus was never present.
9. Death occurred six days after admission patient having never rallied. Her distress being largely pulmonary, death ultimately being due to a failing circulation.

A. Conclusions from the Clinical Record.

- I. The history clearly points to the onset of the Parotitis occurring four days before the onset of the Pneumonia.
- II. The Pericarditis occurred^R only two days before death, it being evident by the onset of precordial pain, at that time.
- III. The Kidney abscesses, had they been of long duration, would have revealed themselves by the presence of pus in the urine. This was never discovered even on daily examination. The trace of albumen is explained by the condition of cloudy swelling, found to be associated Post Mortem with the recent pyemic condition.
- IV. The Septic Thrombus originating high up in the neck points to Parotid as primary source.

B. Conclusions arrived at by study of
Post Mortem findings.

The Post Mortem examination revealed a state of affairs which corroborates this view.

The Parotid Gland was in a much more advanced state of suppuration than the lungs or the kidneys.

The Pericarditis was only of a few days duration and its onset no doubt was responsible for the precordial pain experienced by the patient two days before death, the cessation of pain being explained by the developement of a large pericardial effusion, which was not recognised Ante Mortem. That infection of the blood vessels from the Parotid had occurred, was evidenced by the presence of the septic thrombus in the External Jugular Vein, the thrombus being in direct communication, by means of a smaller vein with the parotid abscess.



Fig. 1.

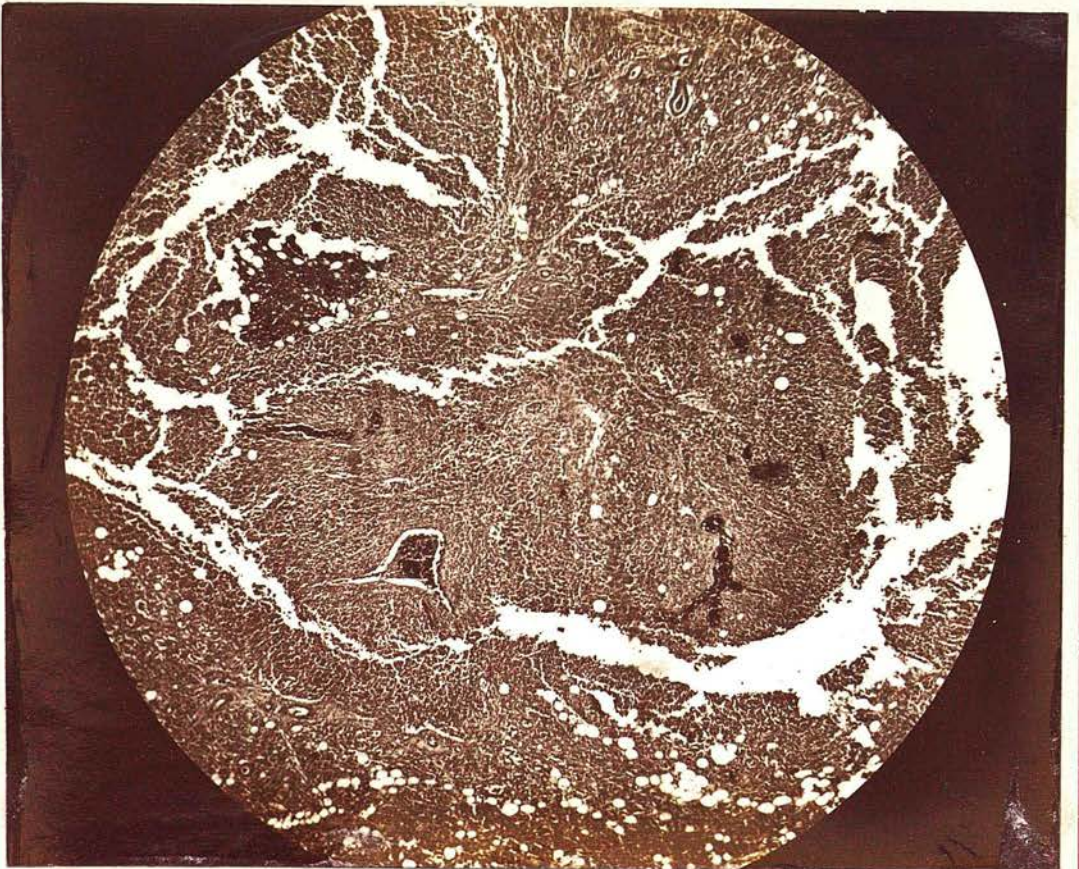


Fig. 2.

C. Evidence of Cultures. Fig I.

Cultivations from the Parotid Gland produced pure growth of Staphylococcus. Pyogenes Aureus. This organism was obtained in pure culture from the lungs, kidneys and pericardium, This suggests a common source of infection, and is quite in keeping with a parotid origin.

D. Result of Histological Examination.

I. Examination of the Parotid Gland. Fig. 2.

All the lobules are affected by the suppurative process. In some the inflammation has been confined to a limited area, around the central ducts. Other lobules are seen to be in an advanced stage of necrosis, the destructive change having extended to the interlobular tissue. a fact which points to a process of considerable duration.

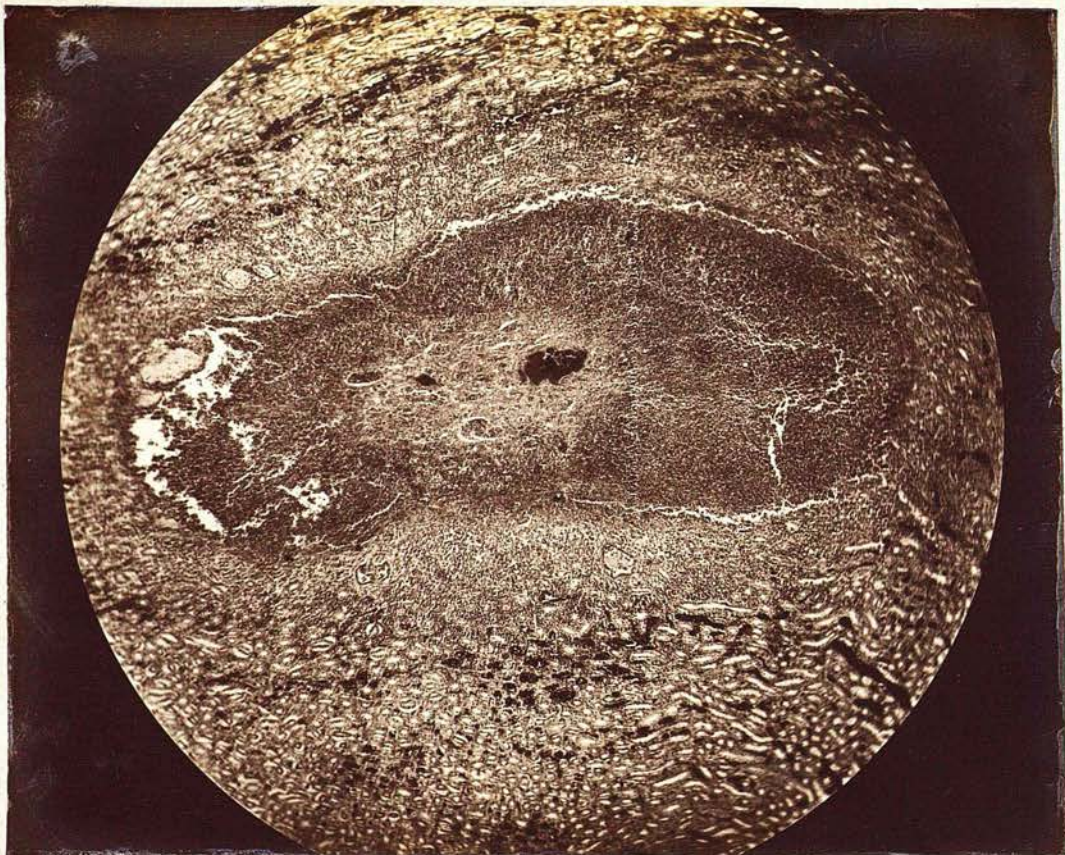


Fig III.

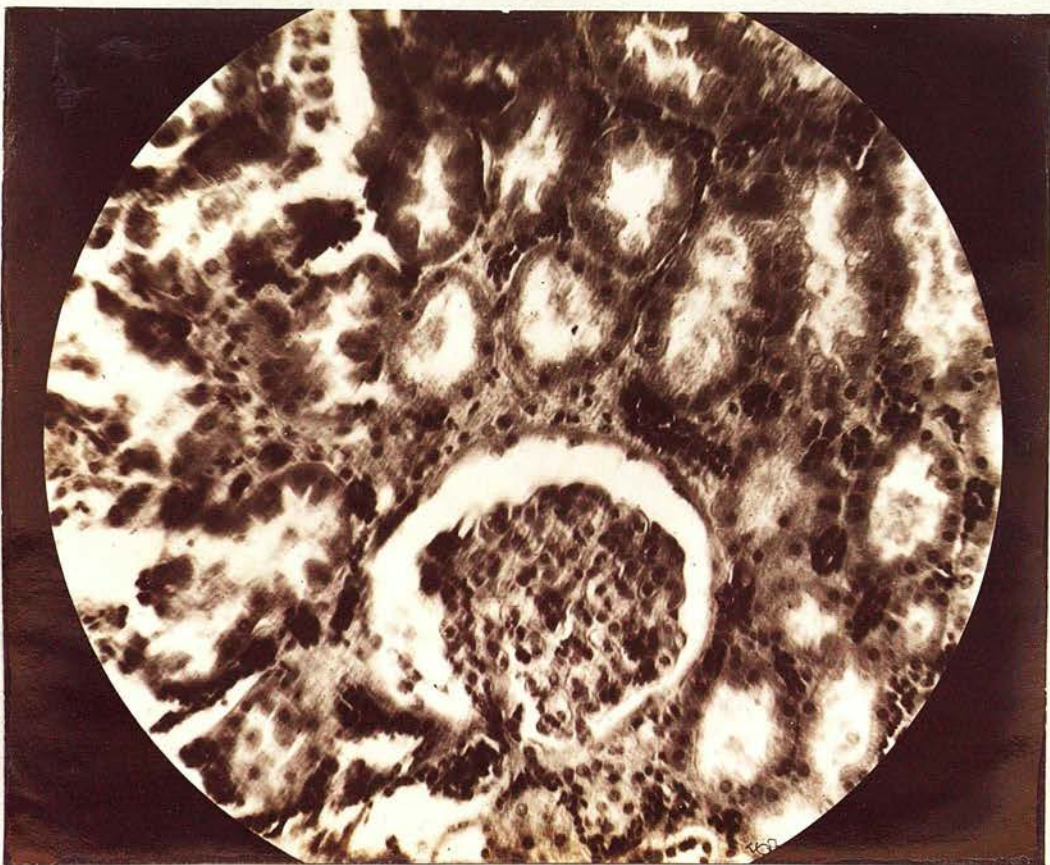


Fig IV.

II. Examination of the Kidney.

A. Pyaemic Abscess situated in the medulla.

Figure III, shows a central elongated necrotic area, containing in its midst, one large and several small plugs of Bacteria, which have retained Grams Stain. The kidney substance in this area has been destroyed and immediately surrounding it there is a ring of leucocytic infiltration.

There is cloudy swelling and catarrh of the surrounding tubules and some congestion of blood vessels.

Figure IV. High power view of cortex from the same kidney.

Glomerular Tuft.

Shows slight congestion of the glomerulus with catarrh of the cells lining Bowman's Capsule.

Tubules.

Show advanced cloudy swelling and catarrh. The change is much more marked than in the medulla. Some of the tubules show almost complete destruction of the nuclei and cell elements leading to considerable widening of the lumen

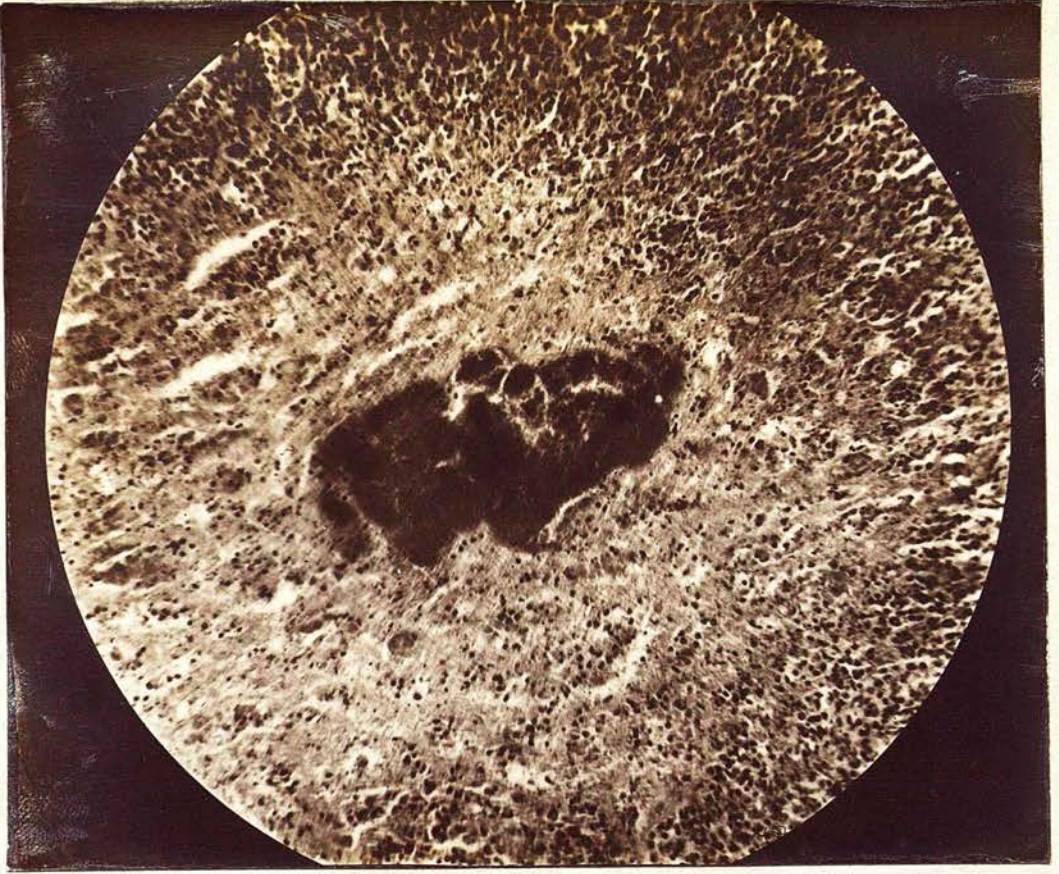


Fig. V.

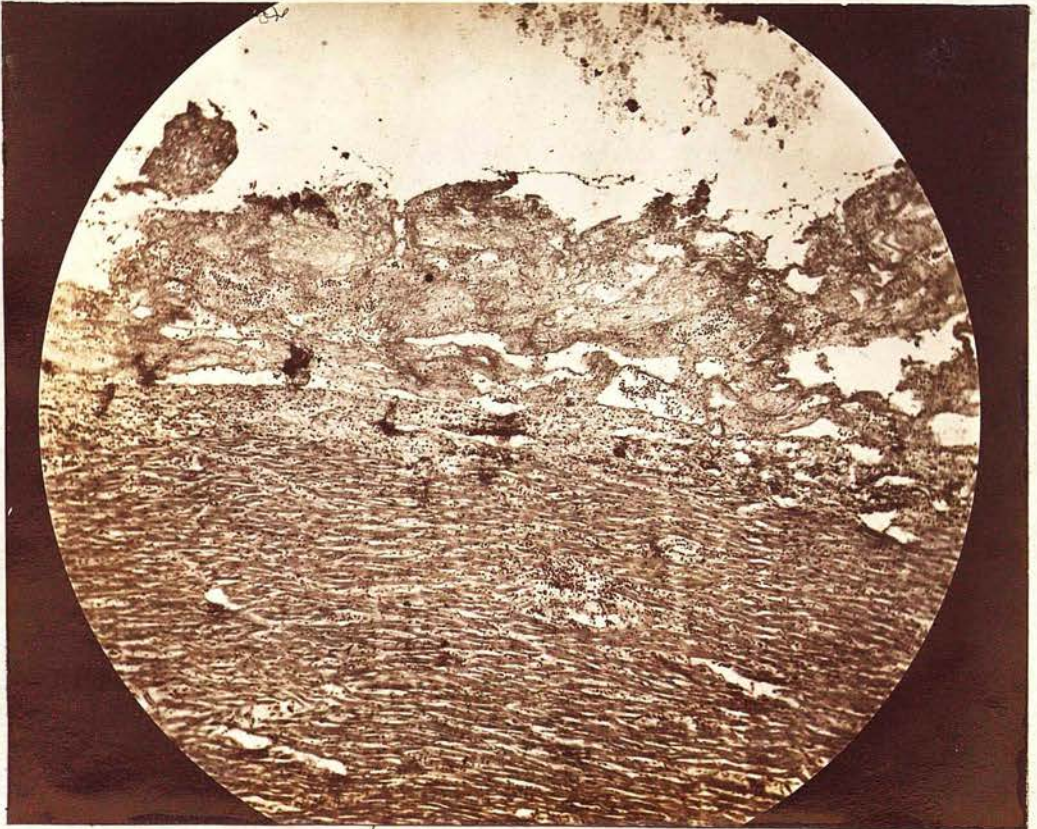


Fig. VI.

of the tubule.

Blood Vessels.

These are compressed and imperfectly filled with blood.

Pyæmic Abscess

Figure V. High power view shows central plug of bacteria surrounded by necrotic tissue, showing how completely the renal substance has been replaced by the necrotic tissue. At the periphery of the field a marked degree of leucocytic infiltration is present tailing off towards the centre of the field.

III. Examination of the Pericardium.

Figure VI. shows an early stage of pericarditis with cloudy swelling of the heart muscle.

There is an inflammatory exudate on the surface of the pericardium. The exudate consists almost entirely of fibrin arranged irregularly. Scattered throughout the substance of the fibrin are large numbers of Polymorpho-nuclear Leucocytes. Organisation has not commenced and the inflammation is of about three days duration.

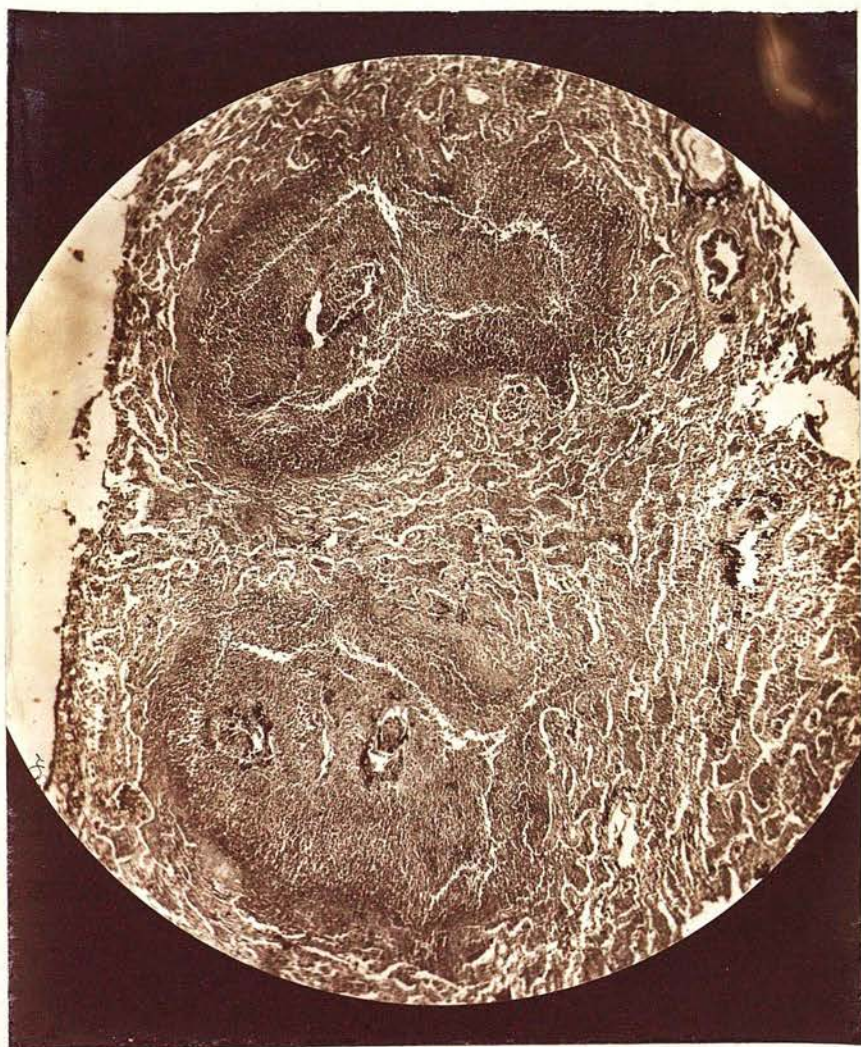


Fig. VII.

Myocardium.

The muscle fibres are swollen, the nuclei in some cases have become indistinct. There is a considerable degree of leucocytic infiltration between the muscle fibres. There are several dilated blood vessels, the contents of which have not been preserved, and almost in the centre of the field is a large collection of leucocytes.

IV. Examination of the Lungs.

The lungs shows changes of Acute Septic Broncho-pneumonia.

Figure VII.

Presents two small abscesses under fairly low power. The lung substance in these areas has been largely replaced by necrotic tissue in which are collections of Gram + Organisms. The surrounding alveoli are filled with inflammatory products and there is a considerable degree of small celled infiltration. The Bronchi show advanced catarrhal changes, the whole Mucous Membrane being swollen and partially detached in places.

The sum of the " Indirect Evidence" goes to prove that the parotitis was a primary condition relative to the Cardiac, Renal, and Pulmonary conditions. The sequence of events, therefore, would appear to be as follows:-

Firstly.

Parotitis.

Secondly. to this.

- A. Infection of the blood stream.
- B. Invasion by septic material, carried by blood stream of:-
 - I. The Lungs.
 - II. The Kidneys.
 - III. The Pericardium.
- C. Septic Thrombosis of External Jugular Vein.

Having established this " indirect evidence " and having excluded a Pyaemic source of infection in relation to the abdomen or thorax, we may deal with the only remaining source - namely the mouth.

State of the Mouth.CASE I.

In this case the mouth was in a particularly bad state. A condition of ulcerative stomatitis had been present for some time before the onset of the Parotitis, and was probably aggravated by the constant exposure to infection necessarily associated with the nursing of a suppurative tonsillitis.

The secretion of the saliva was diminished and the mouth dry.

The patient was not in a good general state of health and had "felt out of sorts" for some time.

We have therefore the three factors present which when present in animals always enabled Claisse and Duplay, experimentally to produce an infection of Stenson's Duct, which lead invariably to an inflammation of the Parotid Gland.

Namely:-

1. Marked oral sepsis.
2. A dry mouth.
3. A patient in a state of depressed vitality.

In the light of the above experimental result (later on referred to in detail) with such an obvious source of infection as the mouth, we can

only conclude that infection occurred by direct spread from the mouth, along Stenson's Duct.

B. Direct evidence in favour of duct spread.

Before dealing with the direct evidence afforded by the histological study of the affected parotid gland, a short description of the structure of the normal Parotid Gland will be given.

Histological structure of the normal Parotid Gland (Quain).

The parotid gland is a compound acinous gland consisting of innumerable lobules composed of multitude of alveoli opening in clusters into the extremities of the branched excretory duct. The alveoli are filled rather than lined by secreting cells and are arranged round the commencing branches of the duct, with which their cavities are continuous. The ultimate branches of the duct open into larger branches, these again into larger still until they terminate in the main excretory duct, by which the secretion is poured into the mouth.

The lobules are held together by the branches of the duct to which they are appended and by interlobular connective tissue which also supports the blood vessels, nerves and lymphatics in their ramifications. The larger lobules are made up of smaller ones, these of still smaller and so on for several successive stages. The smallest lobules consists of a single group of alveoli collected round a small duct which issues from the lobule.

Structure of the Duct.

(Quain), "The duct is constructed of a basement membrane and lining of Epithelium, and in its smaller divisions there is nothing else: but in the larger branches and trunks a coat composed of Connective tissue with which in some cases involuntary muscles fibres, are introduced is added.

In the more recent German works on Histology in addition to the above, there is described running in the connective tissue coat of the main duct a number of elastic fibres.

Symonowicz describes the main duct as consisting of a double layer of cells arranged on the basement membrane supported by a connective tissue

coat containing involuntary muscular fibres and numerous elastic fibres.

As the duct becomes smaller the double layer of cells is replaced by a single layer, and the connective tissue coat becomes thinner.

He makes no reference to the presence or absence of elastic fibres in the smaller branches of the duct.

In order to demonstrate the exact distribution of the elastic tissue within the gland sections of normal parotid glands were made and stained especially for elastic tissue by Weigerts process as follows:-

Resorcin fuchsin stain. one hour.

Acid alcohol one minute.

Wash thoroughly in water.

Counterstain with:-

Saffranin I ϕ . one minute.

Wash in water.

Dehydrate and mount in Canada Balsam.

Figures VIII & IX. show respectively low and high power view of one of the resulting sections all of which show the same distribution of the elastic tissue.



Fig. VIII.

Figure VIII. Low power.

Shows the division of this portion of gland substance by interlobular septa, into several lobules. Running in the septa are several blood vessels of different size, the elastic laminae of which stand out prominently stained black. One large and some smaller ducts with elastic fibres in the connective tissue of their walls, stained black

In the interior of the lobules,

- I. No blood vessels sufficiently large to show an elastic laminae.
- II. No ducts containing elastic tissue.
- III. Numerous small ducts some of which are branching in a manner similar to that displayed in Fig. X.
- IV. Numerous fat spaces.
- V. Gland substance arranged round the termination of the ducts.

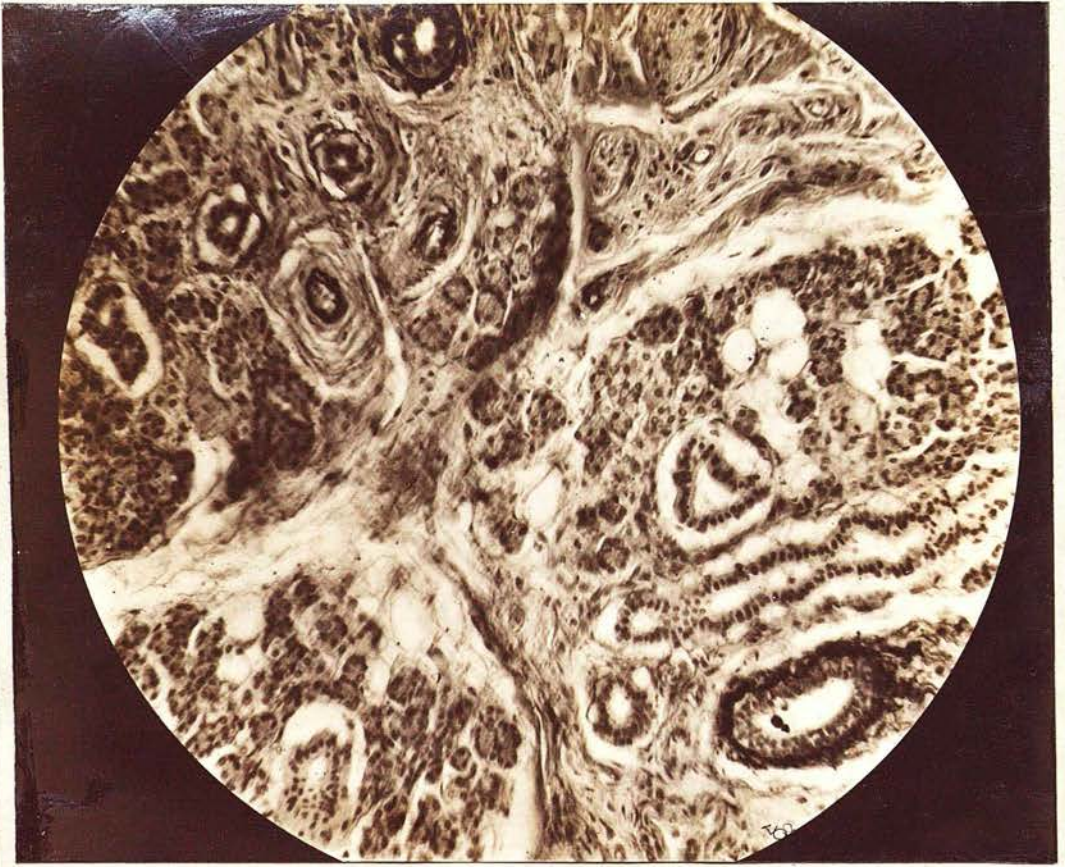


Fig. IX.

Figure IX. High power.

Shows parts of several lobules separated by inter lobular septa.

Chiefly of importance are :

I. The large duct almost in the inter lobular septum below and to the right; which shows very clearly.

- A. Elastic fibres in its Connective tissue coat stained black by Weigerts method.
- B. A double layer of cells arranged on basement membrane.

A similar duct is seen towards the upper limit of the figure.

2. The duct cut longitudinally immediately above the large duct but situated completely intra lobularly and almost certainly derived from division of the larger duct.

- A. It shows entire absence of elastic tissue in its wall.
 - B. A single layer of secreting cells.
3. A triangular duct of similar structure adjacent to 2.
4. The entire absence of a blood vessel of sufficient size to contain an elastic laminae or to be mistaken for a duct.



Fig. 1Xa. Shows the distribution of the elastic tissue in the normal Parotid gland, as described in the text.

I. One concludes therefore regarding the structure of a normal parotid gland, that the elastic tissue of the ducts is continued into the branches of the ducts until these become intra lobular or partly intra lobular and that when they become completely intra lobular in addition to losing the second layer of secreting cells the ducts lose the elastic tissue from their walls.

II. That no arteries except minute Capillaries which contain no elastic tissue are present within the lobules of the parotid gland.

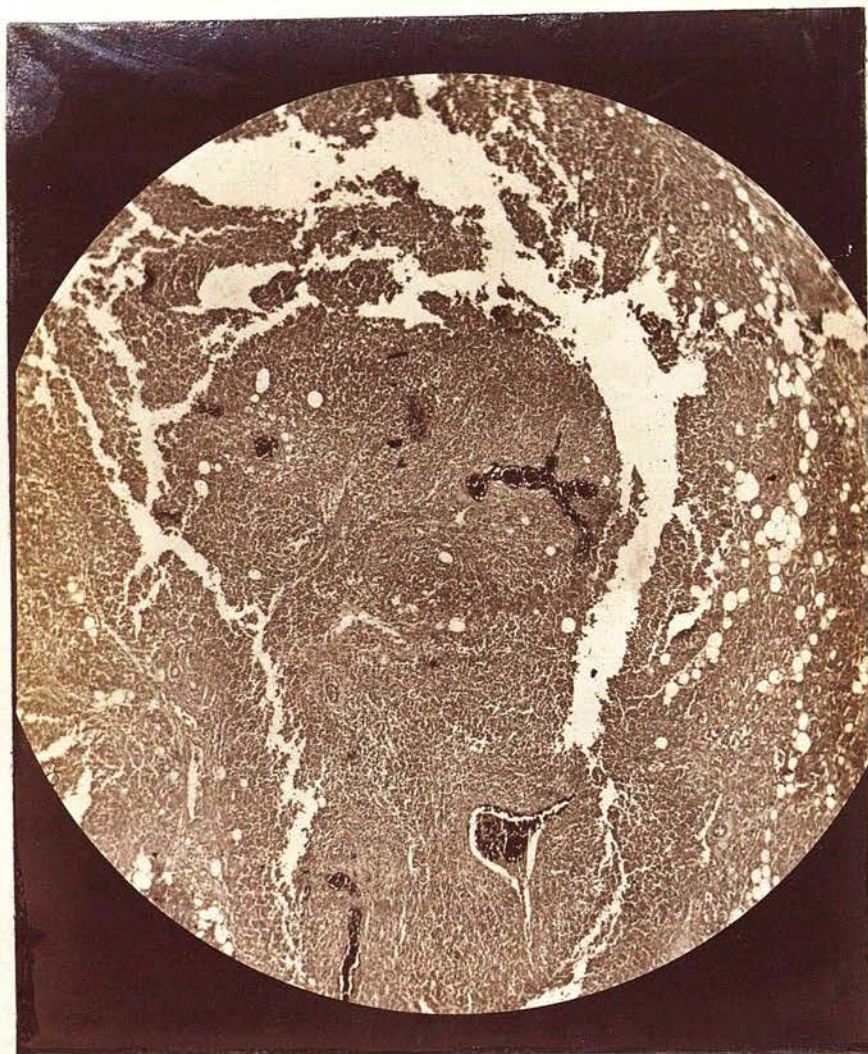


Fig. X.

B. Direct evidence in favour of duct spread.Histological examination of the affected Parotid Gland in Case I.

Figure X. Shows an affected lobule of the gland.

The specimen was stained by Weigert Resorcin Fuchsin method to show up the elastic tissue. Then by Gram's method to stain the organisms and counterstained with Saffranin.

Staining method.

Resorcin Fuchsin- - - -one hour.
 Acid alcohol - - - -one minute.
 Wash thoroughly.
 Carbol Gentian Violet - five minutes.
 Grams Iodine Solution - half minute.
 Aniline oil - - - two - three minutes.
 Alcohol - - - - half minute.
 Wash in water.
 Saffranin I % sol - - one minute.
 Wash.
 Dehydrate and mount in Canada Balsam.

The central part of the lobule is undergoing necrosis. Scattered throughout the figure are

several darkly stained patches. These are the intralobular ducts choked with organisms, which are Gram positive and under the oil immersion are easily recognised as Staphylococci.

The periphery of the lobule is comparatively unaffected there being a slight degree of cellular infiltration however, which extends into the inter-lobular septa. The clear round spaces throughout the figure contained fat, which has been dissolved out by the xylol in the staining process.

The branching form they assume suggest that the spaces we are dealing with, which are plugged with organisms, are the intra-lobular ducts. In order however conclusively to prove the nature of these spaces the specimen was stained by Weigert Resorcin Fuchsin stain for elastic tissue as described above

Reference to the description of the structure of the normal parotid gland stained by this method will show that,

I. The blood vessels except minute capillaries are all extra lobular and that when stained as above, the elastic tissue of the inner coat of the blood vessels stains a deep mahogany brown.



Fig. XI. Oil immersion view of an affected lobule
Shows the organisms mixed up with the desqua-
mated epithelial cells, occupying the lumen of
a duct.

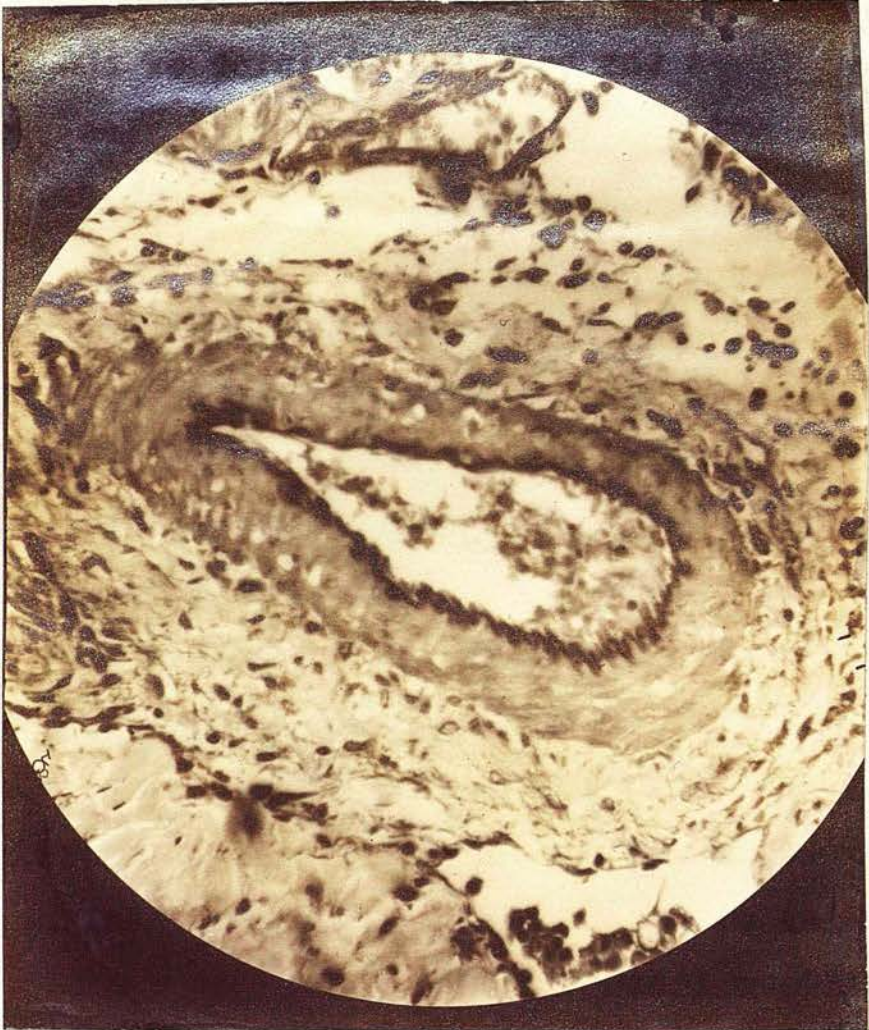


Fig. XII.

Artery from the parotid abscess.

Figure X// This figure shows an inter lobular vessel running in the inter lobular tissue adjacent to the lobule containing the infected ducts, figured in the previous photograph.

It shows the positive staining of the elastic laminae and in addition it is quite free from infected material of any kind and possesses a healthy endothelial lining.

The position is therefore that on the one hand we have the ducts in the interior of the lobules crammed full of infected material, with a process of suppuration most marked around these ducts and gradually becoming less intense as the periphery of the lobule is reached.

On the other hand we have healthy arteries none after examination of many sections was found to be infected - containing no infected material of any kind, situated in a healthy connective tissue matrix.

This can be explained only by assuming infection to take place by way of the ducts and not by the blood stream.

If infection occurred by the blood stream the blood vessels must show traces of infection and

we should expect the suppurative process to be most marked where the blood vessels run in the inter lobular tissue, also we could not account for the marked involvement of the ducts upon this theory.

Microscopic evidence proves conclusively therefore that spread in Case D, from which the ~~secretions~~ ^{sections} were obtained, occurred by way of the blood stream.

Features of general interestCASE I.

Apart from the question as to the mode of spread of the infection, this case is one of considerable interest and importance.

It illustrates one of the many possibilities in those cases of Parotitis which go on to suppuration:

Namely, spread of infection to the jugular vein, with resultant secondary Embolic Pyaemia.

Also it illustrates the gravity of the condition and the necessity, always of giving in cases of Parotitis, a guarded prognosis.

The girl was treated in the early stages for a gumboil and it was only when she had developed secondary pyaemic abscesses in the lungs, giving symptoms of Broncho-pneumonia, that she was sent into hospital.

The question of treatment also comes up for discussion.

In the light of the post mortem examination, incision of the gland however freely carried out would probably have been without avail, as secondary spread had already taken place and death was the result of the complications and not of the Parotitis. Still it is interesting to find that no fluctuation was ever elicited, in fact the swelling was tense and firm and that it subsided under the local

application of ichthyol in glycerine 10 %, suppuration not having been suspected . The naked eye appearance of the gland at the post mortem examination explained why no evidence of suppuration existed. There was no generalised abscess cavity. There existed throughout the gland numerous small collections of pus situated for the most part in the centres of the lobules and arranged around the ducts.

One can understand how in this case even a free incision would fail to give any marked relief owing to the tenseness of the parotid fascia it is almost impossible to arrive at a satisfactory conclusion regarding the presence or absence of pus formation within the gland and if incision is to be carried out, it must if it is to avail not only be accomplished early but be carried out freely.

A therapeutic point of interest was the use in this case of nucleinic acid, with a view to establishing a leucocytosis.

Within 12 hours of a hypodermic injection of thirty minims of nucleinic acid the leucocyte count, which had never been above 12,000 since admission, rose from 10,000 to 19,200. The increase

in the number however was made up of illformed and undersized corpuscles, whose value in combating disease one would not be inclined to estimate at a high value, especially as the increased leucocyte count was not accompanied by any change for the better in the patients condition.

CASE 2.

ACUTE HAEMORRHAGIC PURPURA complicated by
PAROTITIS.

A. K. aged 17. Single.

Occupation. stonebreaker.

Complaint Pain in his side and vomiting.

Admitted Oct. 10. 1910.

History.

On Friday Sept 30th, 10 days before admission patient suddenly began to shiver and complained of pain in his sides. He vomited that night the vomiting continuing for 3 days, being brought on when the patient attempted to drink milk or water He improved for the next few days and his doctor allowed him some gruel which he vomited, On the

Saturday before admission the vomiting became worse and pain more severe and with a tendency to be more acute in the right iliac fossa. The bowels had moved quite well and regularly with the aid of purgative medicines until the Thursday before admission. As the pain continued he was admitted to the surgical house on Sunday. On admission patient seemed dull mentally and slow in answering questions.

Condition on admission.

Patient was flushed Temp 98" Pulse 88.
Respirations 22. Leucocytes 28,000.
Blood pressure raised : Second sound accentuated at base of heart. There was tenderness on deep palpation over the Kidneys especially on the right. Abdomen moved freely on respiration, no pain, no tenderness over Mc Burney's point.

Urine contained considerable quantity of blood and albumin.

Patient had an enema on Sunday evening and the bowels moved slightly and on Monday he passed a good deal of flatus. His condition was thought to be one of Acute Nephritis and he was transferred to the medical side on Monday Oct 10. 1910.

Temperature Chart.

Case. II.

Name *Alexander Kirkhof* Age *17*

Clin. Clerk.

No.

Date *October*
F. 107°

C. 41° 106°

105°

40° 104°

103°

39° 102°

101°

38° 100°

37° 99°

98°

36° 97°

96°

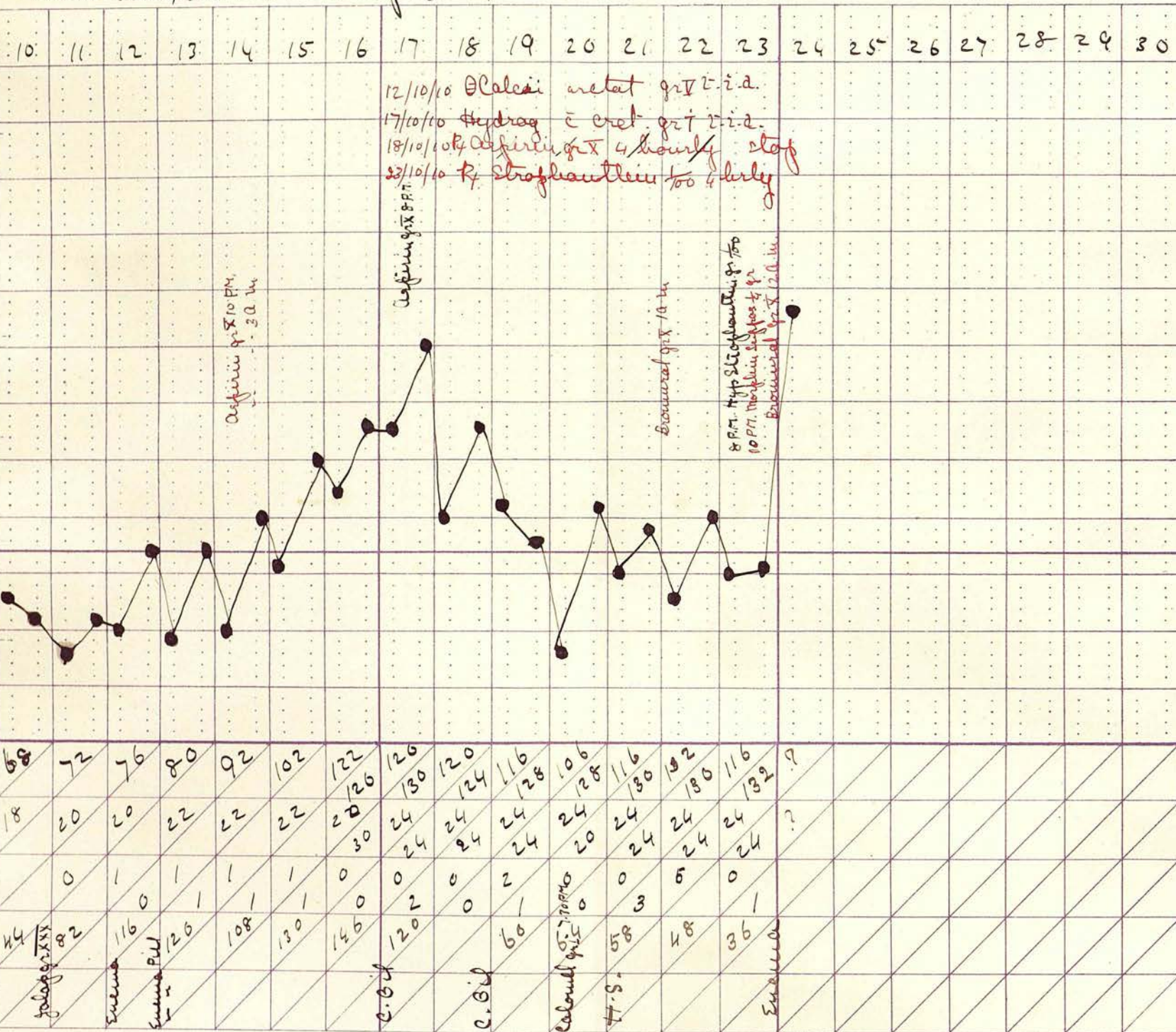
35° 95°

Pulse.

Resp.

Bowels.

Urine of.

October
1916Hb. Red Cells
100% 5,000,000

90%

80% 4,000,000

70%

60% 3,000,000

50%

40% 2,000,000

30%

20% 1,000,000

10% 500,000

Previous Illnesses.

Congestion of the Kidneys, two months ago, which lasted five days.

Family history.

Satisfactory.

State on admission to Medical Ward.

Patient was very flushed, Tongue dry and coated with a white fur, Temperature 97.6, Pulse, 68, Respirations 18, Patient seemed rather dull and lethargic. Pulse full regular in rate and rhythm. Blood pressure high. Patient complained of pain in the abdomen. There was a slight rigidity and tenderness in the right iliac fossa, and also on palpation over the kidneys.

Patient complained of headache. There was no oedema. Patient began to vomit about 8 p.m. the vomit was watery and bile stained. Patient was given Pulv Jalapae Co gr xxx, which however he vomited up. He continued to vomit at intervals and was unable to sleep.

Oct 11th. Temperature 96.6, Pulse 72, Respirations 20, Dull and lethargic: flushed: tongue dry, mouth very dirty, continues to vomit. Has been given sips of hot water without any relief. There is

very little movement over the lower part of the abdomen and marked tenderness on palpation.

Patients bowels have not moved but he has passed flatus.

The lungs are quite healthy.

The urine contains a considerable quantity of blood and albumen. Leucocytes 41.500.

Polymorphs 98 %.

Patients stomach was washed out with Bicarbonate of soda and ultimately the fluid returned clear.

Oct 12. Patient had a good night; but vomited early in the morning; vomit was watery and bile-stained. Patient has developed a marked purpuric rash over the extensor surfaces of the knees, elbows, forearms and hands and over the dorsum of each foot, there is no eruption on the body. Widal reaction negative.

Heart.

A apex Beat in fifth left interspace, three and a half inches out.

Abdomen.

Somewhat Tumid and Tympanitic, no peristaltic waves: Tender all over. Enema- Result watery and blood stained and very foul smelling.

I4-X-10, Patient much better : no abdominal pain no sickness: given peptonised milk: Rash still marked.

I5-X-10. Temperature rose to 102, Pulse 100, Respirations 23, Patient complains of pain all over the body ,given aspirin gr,x. Is now getting five ounces of peptonised milk 4 hourly.

I7-X-10, Temperature still up. Condition much the same.

I8-X-10. Temperature down:vomited curdled milk. Breath very foul: Mouth dirty, Tongue still dry. Stomach washed out - gave relief.

20-X-10. Vomited several times, complains of great pain, condition less satisfactory.

21-X-10. Patient has developed a swelling of the left parotid gland which is very painful. Hot fermentations. Belladonna Glycerine applied.

22-X-10. Parotid swelling still marked:Pain has been relieved by the Belladonna.

Patient again complained of severe abdominal pain relieved by hot fermentations. Urine contained a large amount of blood and only a trace of albumin. Motions loose, watery and blood stained very offensive smell. Patient was given Bromural gr x. The purpuric rash is fading.

23-X-10. Parotid swelling slightly less and patient can open his mouth without so much pain. Patient began to vomit in the afternoon and vomited several times - vomit green and foul smelling.

8, p. m. Pulse weak and irregular. Patient put on Strophanthin 1/100 gr 4 hourly.

10. p. m. Patient very weak, was transfused with very slight benefit.

5. a. m. Bowels moved very freely. Motion consisting of almost pure blood and of very offensive odour. Patient became very pale and almost pulseless was stimulated freely. Respirations became laboured patient appeared to be in considerable pain which came on in spasms.

6. a. m. Bowels moved again. Motion similar in character.

8. a. m. Patient gradually sank and died quietly. After death there was a great flow of blood from the bowels which collected in a huge pool in the bed and was extremely foul smelling.

POST MORTEM REPORT.

Rigor mortis present, marked haemorrhagic eruption over front of both knees. left forearm, hand and a few over dorsum of foot and toes, both elbows, lips. and right ear. Swelling over left parotid. Gums markedly exsanguine. Greenish discolouration of decomposition over abdomen. Very little subcutaneous fat.

THORAX.

Left pleural cavity contains 15 ozs blood stained fluid. Right pleural cavity contains about 6 ounces. Pericardium contains no excess of fluid.

ABDOMEN.

Omentum very little fat. Small intestine-serous covering injected and shows slight diffuse haemorrhage. This is well seen towards the lower end.

Mesenteric glands are of a pinkish grey colour. Small amount of bloody fluid in the pelvis, appendix healthy. Whole of small intestine shows dark discolouration, also seen over the caecum. No perforation of the bowel at any part. Large intestine contains fluid altered by blood.

URETERS.

Intensely congested with haemorrhages into the mucous membrane. Contents of a dark brownish grey color.

RIGHT KIDNEY.

Shows older changes.

PANCREAS.

Haemorrhage over surface, not in substance, no softening.

STOMACH.

Contains creamy bile stained fluid. Some purpuric haemorrhages in the wall, particularly in the greater curvature. In lesser curvature are dark grey lines along the ridges. Near the pylorus, surface is covered all over with little haemorrhagic patches and on this congested area extending for two inches from pylorus are numerous ulcerated areas, majority of which are superficial but one in the upper part the size of a three-penny piece extends to the deeper coats and the base of the ulcer is haemorrhagic and necrotic.

DUODENUM.

Surface is covered over with blood stained mucus, but no necrosis can be made out on the general surface. The papilla is infiltrated firm and shows haemorrhages into mucous and sub-

mucous coats. A short way down duodenum haemorrhage and mucus become small in amount. There is a little hollow immediately to the anterior side of the papilla which resembles a superficial erosion. Below it numerous haemorrhages into the mucous membrane. Further down mucus is lessened and occasional haemorrhages are present. There is some thickening of the wall of the bile duct as it passes through the pancreas.

JEJUNUM

Appearances similar.

ILEUM.

In upper part occasional small haemorrhages Wall thin and a little lower down distinct erosions near attachment of the mesentary. Lower down the erosions are specially on the ridges. These erosions lower down still - 3 feet above ileocaecal valve - become very extensive and large areas of mucous membrane are removed, or necrotic. Extreme lower end is very markedly congested.

LARGE INTESTINE.

Is full of slightly altered blood.

CAECUM.

Numerous small erosions, and the ridges are intensely congested with haemorrhages into their substance. They are few erosions in the lower half of the large intestine.

ESOPHAGUS.

Numerous small submucous haemorrhages tending to run in a longitudinal direction.

TRACHEA & AORTA.

Nothing to note.

LEFT LUNG.

Subpleural haemorrhages on the lower lobe posteriorly and on the diaphragmatic surface adjacent. On section. Lower lobe, congestion and emphysema only, posteriorly, numerous small haemorrhages Upper lobe very emphysematous. Numerous small haemorrhages. Early acutelobar pneumonia, great infiltration of the walls of the vesicles with polymorphs and proliferated cells.

RIGHT LUNG.

Pale, emphysema throughout. Few small subpleural haemorrhages over the lower lobe.

HEART.

Some thickening of the Epicardium. Myocardium pale. Left auricle dilated, walls thin. Mitral valve segments thickened at their margins, on the inner cuspa small patch of haemorrhage. Aorta valve healthy.

LEFT KIDNEY.

Supra renals healthy. Pelvis shows numerous haemorrhages and a large necrotic patch. Capsule adherent, strips in layers. Cortex swollen. Patchy haemorrhage in cortex. Subacute Parenchymatous Nephritis with some interstitial change. Marked cartarrh in tubules.

SPIEEN.

Enlarged, soft, haemorrhages into the substance

LIVER.

Extreme pallor. Small haemorrhagic spots throughout. Cloudy swelling.

CULTURES.

No growth was obtained from cultures made from the spleen.

Urine Chart.

Case. II.

Disease *Acute Haemorrhagic Purpura*Name *Alexander Kiehlhofe* Age Occupation

Admitted

Discharged

DATE.	No. of Motions.	3 of Urine.	CHARACTERS OF URINE.		Reaction.	Specific Gravity.	ALBUMIN.		UREA.		GLUCOSE.		Blood.	Bile.	TREATMENT.	
			Naked Eye.	Microscopic.			Grain per oz.	Grains per diem	Grain per oz.	Grains per diem	Grain per oz.	Grains per diem			Dietetic.	Medicinal.
<i>1910</i> <i>October</i>																
<i>11</i>	<i>0</i>	<i>82</i>	<i>Smoky.</i>	<i>Blood: 20 Casts. Granular debris.</i>	<i>Ac</i>	<i>1015</i>	<i>7</i>		<i>7.5</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>12</i>	<i>1</i>	<i>116</i>	<i>"</i>	<i>Blood: Blood Casts.</i>	<i>"</i>	<i>1016</i>	<i>9</i>		<i>6.5</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>13</i>	<i>2</i>	<i>120</i>	<i>"</i>	<i>" " "</i>	<i>"</i>	<i>1012</i>	<i>1.8</i>		<i>6</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>14</i>	<i>2</i>	<i>108</i>	<i>"</i>	<i>Blood & Epithelial Casts.</i>	<i>"</i>	<i>1012</i>	<i>9</i>		<i>7.5</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>15</i>	<i>2</i>	<i>130</i>	<i>Smoky: Copious Deposit</i>	<i>" " "</i>	<i>"</i>	<i>1015</i>	<i>7</i>		<i>6.5</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>16</i>	<i>0</i>	<i>146</i>	<i>" "</i>	<i>" " "</i>	<i>"</i>	<i>1010</i>	<i>1.3</i>		<i>8</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>17</i>	<i>2</i>	<i>120</i>	<i>" "</i>	<i>" " "</i>	<i>"</i>	<i>1010</i>	<i>1</i>		<i>6.5</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>18</i>	<i>0</i>		<i>" "</i>	<i>Polyph and Pus Casts</i>	<i>"</i>	<i>1012</i>	<i>.5</i>		<i>7</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>19</i>	<i>3</i>	<i>60</i>	<i>" "</i>	<i>" " "</i>	<i>"</i>	<i>1013</i>	<i>times only</i>		<i>8.5</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>20</i>	<i>0</i>	<i>54</i>	<i>" "</i>	<i>Blood, pus and granular Casts.</i>	<i>"</i>	<i>1016</i>	<i>"</i>		<i>8</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>21</i>	<i>3</i>	<i>58</i>	<i>" "</i>	<i>" " " "</i>	<i>"</i>	<i>1017</i>	<i>"</i>		<i>6</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>22</i>	<i>5</i>	<i>48</i>	<i>" "</i>	<i>" " " "</i>	<i>"</i>	<i>1013</i>	<i>"</i>		<i>8</i>		<i>-</i>	<i>+</i>	<i>-</i>			
<i>23</i>	<i>1</i>		<i>" "</i>	<i>" " " "</i>	<i>"</i>	<i>1012</i>	<i>"</i>		<i>8</i>		<i>-</i>	<i>+</i>	<i>-</i>			

SUMMARY of CASE II.

This case presented considerable difficulty in diagnosis in the early stage.

Admitted to the medical wards with the diagnosis of acute nephritis, appendicitis having been excluded while under observation on the surgical side, the illness being of 10 days duration. The urine chart (no. 1.) page.) is of considerable interest under such circumstances. Reference to it brings out the interesting fact that for a week after admission the patient passed over 100 ounces of urine per diem, the record for the first day being in-complete, and that on one occasion the amount reached as high as 146 ounces. The presence of albumen, blood, casts and epithelial casts was sufficient evidence of acute renal mischief, but the large amount of blood and the large amount of urine passed in 24 hours indicated that the diagnosis of acute nephritis far from covered the case, the small amount of albumin seldom above one grain per ounce is also of interest.

The persistent attacks of vomiting, the presence of pain in the abdomen, at times severe and the marked leucocytosis 28,000 on admission rising on the second day to 41,000, with a differential count of 98 % polymorphs pointed in

the absence of physical signs and symptoms relative to the chest, to severe abdominal mischief. Until shortly before death there was no evidence of any lung mischief. The leucocytosis excluded the diagnosis of Enteric Fever, but a Widal reaction was done, which was reported negative.

The vomit was exceedingly offensive and the stomach was washed out several times, with relief to the patient. The mouth was foul and dry the teeth very dirty, and many carious.

On the third day of admission a purpuric rash developed and the diagnosis of acute haemorrhagic Purpura was made, a disease the Etiology of which is so obscure, although over 30 % of recorded cases are associated with previous renal mischief, as in the present case.

The patient was treated medicinally with Calcium Acetate gr v. T. I. D.

Hydrarg Cum Creta gr. i. T. I. D.
and occasionally aspirin was given. He made no headway, in fact became weaker. although in view of the suggested Rheumatic origin of Haemorrhagic Purpura it is interesting to note the result on the patients condition of the administration of aspirin grains x, four hourly

on October 17th when he was complaining of considerable abdominal pain and the temperature had risen to 102 f. The temperature dropped within 12 hours the pain was relieved temporarily to return however the following day, but the temperature after an initial rise never again reached above 99".

On the 21st October 3 weeks after admission patient developed a left sided parotitis. Ever since admission his mouth had been in a filthy state and at times very dry. The application of hot fomentations, belladonna and glycerine, relieved the pain in the parotid swelling, so that the patient had great difficulty in opening the mouth, on account of the pain occasioned by the movement. The patients general condition was now very unsatisfactory. The vomiting continued very offensive smelling, material green in colour. He now began to have loose motions, his bowels moved three times on the 20th, five times on the 21st. The motions were watery and blood stained and exceedingly offensive.

The purpuric rash which had lasted for 10 days had now almost faded, and was quickly disappearing. The parotid condition on the

23rd, 3 days after its appearance, was less marked. The pain was less severe and the swelling had subsided slightly, patient being able to open his mouth without marked pain.

The temperature seemed to be unaffected by the onset of the parotitis, continuing to oscillate between 98 in the morning and 99 in the evening. On the 23rd the patients condition became much worse and during the day he had considerable pain, vomited several times and his bowels moved very freely shortly before death on the morning of the 24th he passed per rectum a huge amount of almost pure blood

POST MORTEM REPORT.

The post mortem report gives a detailed account of the appearances found in the various organs. A culture from the spleen failed to give any growth.

ORIGIN of the PAROTITIS.

From the point of view of the origin of the parotitis P.M examination revealed no source of infection save the alimentary tract, There was no focus of suppuration in any part of the body from

which an embolus could have started. The entire absence of any growth in the culture taken from the spleen puts out of count a generalised blood infection arising from the ulcerative condition of the bowel, as the source of infection. Here however, we have the three factors present, in an extreme degree, which were associated with case I.

- I. A foul septic mouth.
- II. A dry mouth with diminished salivary secretion.
- III. A greatly lowered vitality.

CONCLUSION.

Therefore we conclude that the parotitis in this case also took its origin in an infection which spread from the mouth by way of the duct.

The importance of the third element in the causation of parotitis is emphasised in this case.

On admission patient was in a comparatively good state of vitality, but 10 days after admission and 3 weeks after the onset of symptoms, when the constant vomiting and purging has greatly reduced his strength we have the onset of the parotitis followed in 3 days by a fatal issue.

So far as the writer has been able to discover from the literature on parotitis, this is the first case of Acute Haemorrhagic Purpura complicated by the onset of parotitis, which has been placed on record.

CASE III.ACUTE NEPHRITIS complicated by PAROTITIS.D. W. aged 30. Newsvendor.Admitted. Oct 7th 1910.Complaint.

Swelling of face and legs. Passing little water.

Duration 3 days.History.

Patient was at Empire on Wednesday night. It was very hot there and he perspired a great deal & took off his neckerchief, when he came out forgot to put it on again and thinks he must have caught a chill on the way home. On Thursday morning when he got up his feet were swelled so that he could scarcely get on his boots. However he managed to get them on and went out and sold papers. Thursday night his face swelled. He went out yesterday (Friday) and managed to sell his papers as usual but last night he had severe pains in his legs and his face and feet were very much

Temperature Chart. No. I.

Case. III.

Name David Wilson

Age 30

Clin. Clerk

No.

Date Oct 1910

October 1910

C 41° 106°

Hb. Red Cells 100% 5,000,000

105°

90%

40° 104°

80% 4,000,000

103°

70%

39° 102°

60% 3,000,000

101°

50%

38° 100°

40% 2,000,000

100°

30%

37° 99°

20% 1,000,000

98°

10% 500,000

36° 97°

96°

35° 95°

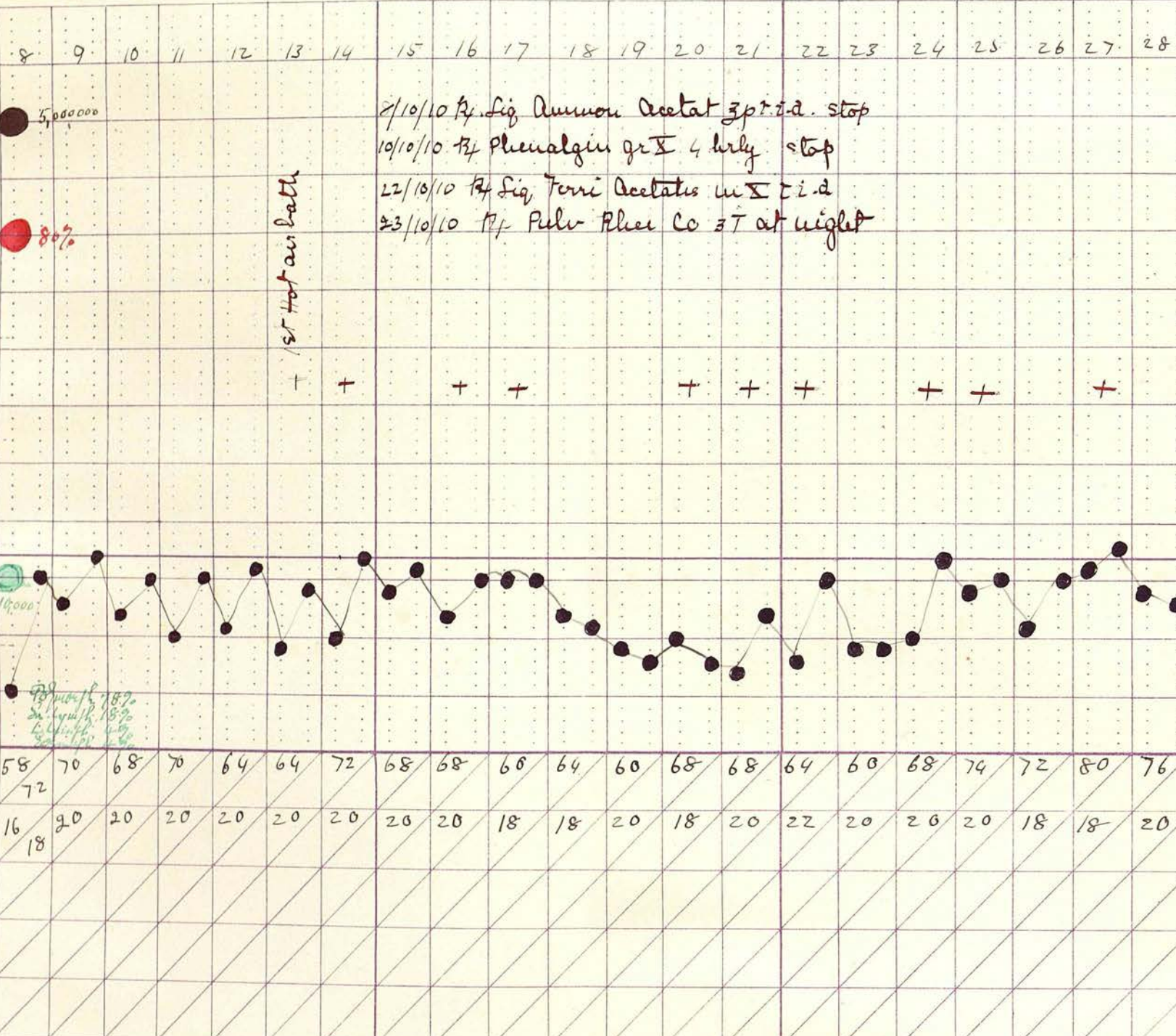
1st hot air bath

8/10/10 Rx Sig. Ammon. Acetat 3p i.d. stop

10/10/10 Rx Phenalgin grs 4 hrly stop

22/10/10 Rx Sig. Ferri Acetatus m 2 i.d

23/10/10 Rx Pulv. Plur. Co 3T at night



10000
 78.7
 18.7
 18.7
 18.7

swollen. This morning when he got up his eyes were so swollen he could see only with difficulty. He came to hospital and was admitted to Ward 31.

Previous Illnesses

Gonorrhoea and Soft Sores. Nothing else remembered.

Family History.

Nothing to note.

Social Conditions and Habits.

Patient sells papers in St Andrew St. Exposed to all weathers, Lives in lodgings, gets plenty to eat, non-smoker, does not drink to excess. Seldom the worse of drink.

On admission.

Patient is well developed man, marked dropsy of the face, slight dropsy of hands and legs . Temperature 96. Pulse 58. Respirations 16. Patient complains of headache: no drowsiness. Patient is very rough and illmanered and belongs to lowest class of the social scale, very surly and discontented.

Temperature Chart. No. II.

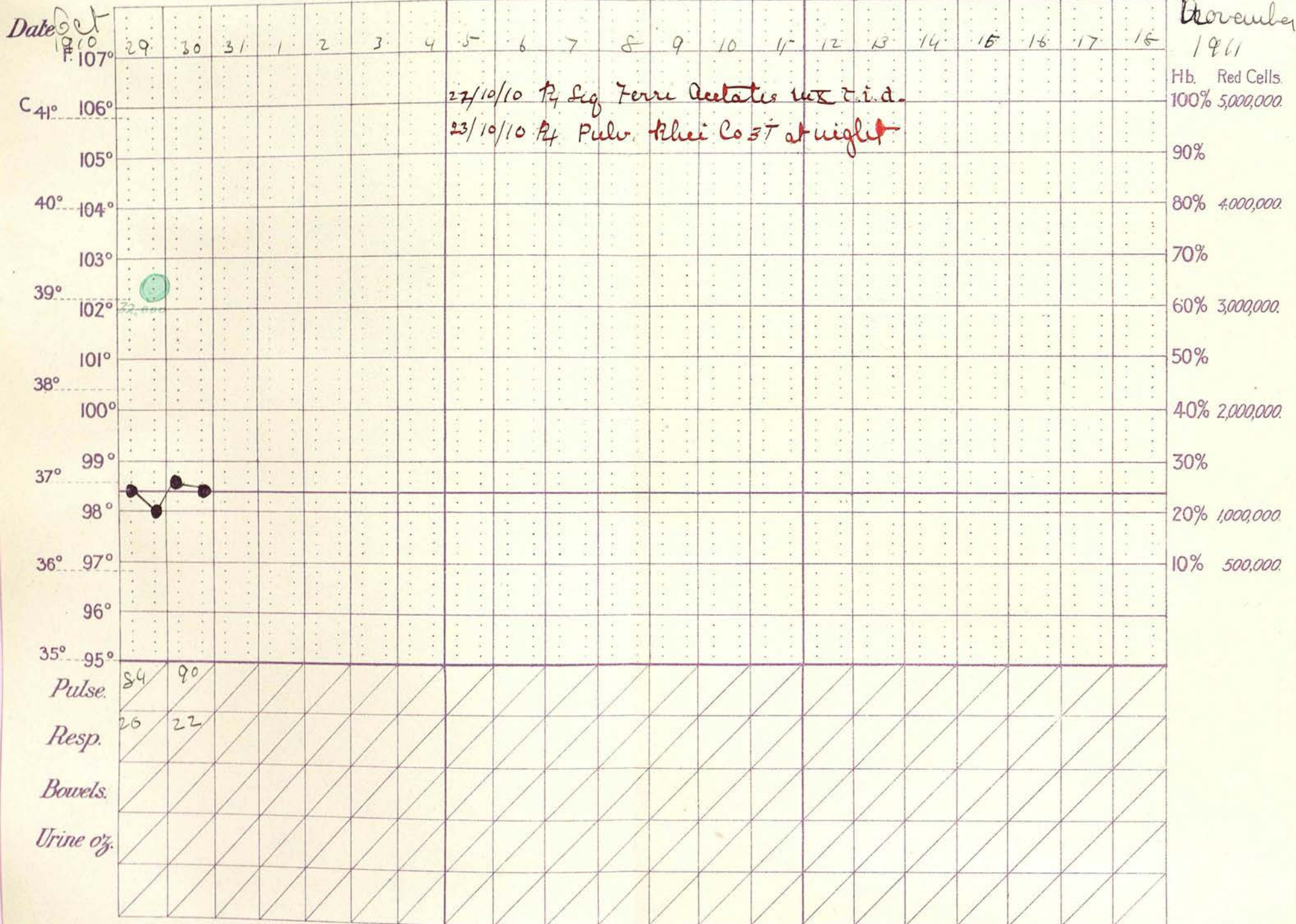
Case. III.

Name David Wilson

Age

Clin. Clerk.

No



URINARY SYSTEM.

Patient is not passing much water. No pain or difficulty on micturition, but urine comes away slowly.

URINE.

S. G. 1016. Alk 1016.

Albumin. X 6 grs per oz.

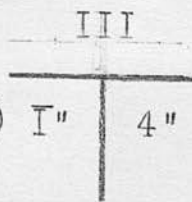
Urea. 4.5 grs per oz.

Microscopic Phosph. R. B. C. No casts.

CIRCULATORY SYSTEM.

No dyspnoea: Slight pain over praecordia on deep inspiration. Chest well formed. No precordial bulging.

Apex beat not visible: Palpable as a faint impulse in the fifth interspace 4 inches from mid-line.

Auscultation

M. A. 1st prolonged & muffled.

3rd loud & accent no murmurs.

A. A. 1st loud & prolonged.

2nd markedly accentuated.

P. A. & T. A. both closed, no murmurs.

2nd accent

Pulse 64, Regular in rate and rythm. Volume full; slow and forcible; slow rise, apex sustained, Fall gradual B.P. 135. Arterial wall not thickened.

RESPIRATORY SYSTEM.

No Subjective Symptoms.

Chest well clothed and well formed, exp. good, no *increase* of V. F.. Percussion note good all over the chest: *Amplification.*

Auscultation

Breath sounds vesicular all over the chest, no accompaniment. No increase of Vocal Resonance.

ALIMENTARY SYSTEM.

Teeth bad, tongue furred, mouth very dirty, breath very foul, appetite moderate: No gastric syptoms.

LIVER. Normal.

STOMACH. Normal.

BOWELS. Constipated.

SPLEEN. Not enlarged: No enlarged lymph glands.

BLOOD. R. B. C. 5.000.000 Wh. 10.000

Progress & Treatment.

8-X-10. Patient on admission was put between blankets and given Lig Ammon Acet 1/2 oz 3 times a day. Milk only.

9-X-10. Patient had a fairly good night Has considerable oedema of face this morning. Passed 10 ounces of urine yesterday. Given Pulv Jalap Co gr LX this morning and Salts in the evening B. P. 135.

10-X-10. Passed 12 ounces of urine yesterday still a good deal of oedema of face. Bowels have moved but patient has not sweated much Phenalgin gr x 4 hourly.

12-X-10. Patient sweated profusely after the Phenalgin wh: was stopped after 6 doses. Patient a little better but not passing much urine, mouth very foul and breath sickening: complains of headache.

13-X-10. Given hot air bath - felt better Jalap at night: Passed a little more urine which contained 4 grs albumin. R. B. C. No casts.

14-X-10. Hot air bath: Felt better.

15-X-10. Patient had a good night, breath is extremely foul in spite of repeated mouth washing. Patient is passing 28 ounces of urine. Was very sick this morning. Vomit brown and evil

smelling;no hot air bath.

16-X-10. Hot air bath. Castor oil at night instead of Jalap.

17-X-10. Vomiting very foul stuff again this morning. Breathing very foul .Stomach washed out and patient felt much better,given hot air bath, only passed 12 ounces of urine which contained 6 grs per ounce of albumin. No casts. Jalap again gr LX.

18-X-10. Better. 22 ounces of urine. No hot air bath.

19-X-10. Better 30 ounces of urine. Jalap LX,

20-X-10. Vomited large amount foul black stuff this morning ;given hot air bath,27 ounces of urine;stomach washed out at night,patient felt better.

21-X-10. Better.Hot air bath,40 ounces of urine

22-X-10. 16 ounces of urine.Hot air bath; Jalap gr LX. Lig Ammon Acet stopped, put on Lig Ferri Acet gr X t. i. d.

23-X-10. Patient complains of pain below the right ear which on examination proved to be discharging pus. Ear syringed with H_2O_2 and patient puts on Pulv Rhei Co at night to try

and improve his gastric condition.

25-X-10. Right parotid gland swollen, hard and brawny.

26-X-10. Parotid swelling increasing in size, patients ear examined by Dr. Logan. Turner who discovered a small perforation on posterior part of membrane. He did not think there was any Acute Otitis Media and thought the ear condition had no connection with the parotid swelling. Belladonna and glycerine applied to the parotid without relief of symptoms. Temperature sub normal.

There was great pain and difficulty in opening the mouth.

28-X-10. Swelling has extended from the parotid region up to the eyes and down in to the neck. Right eyelid very oedematous. The parotid on the left side has commenced to swell. Patient complains of a good deal of pain. Hot bath and Opium Formentations applied without much relief.

Patient very sick tonight. Vomit brownish and contains curdled milk.

29-X-10. Swelling on right side of face very intense hard and brawny. Patient is unable to open his right eye, complains of a great deal of pain. Hot Lead and opium failed to give relief. Leucocytes. 32,000. Polymorphs 90 %.

Temperature normal. Pulse 84. Respirations 20.

30-X-10. Swelling in right side of face more intense; skin glazed and tense. Ichthyo and Glycerine (10%) applied. There is a great deal of thick tenacious mucus about the mouth and nasopharynx which patient has great difficulty in getting away. Nose bled this afternoon. Patient was very restless in the evening and had great difficulty in clearing his nose and nasopharynx, but was however breathing easily.

31-X-10. 12-30 . A. M. Bromide gr xxx to quieten him as he was very restless and appeared to have difficulty in breathing and occasionally appeared to be choking.

2-30. A. M. Patient quieter. Breathing quite free and

5-30 A. M. Patient suddenly choked, became black in the face and unable to clear his throat, and died in a few minutes.

POST MORTEM REPORT

Body small well developed and nourished, oedema in lower limbs and upper part of face and chest. Slight adhesions on lower part of left pleural cavity. None on the right side. Considerable amount of fluid in the pericardial sac.

HEART.

Enlarged ovoid and shows excess of sub - epicardial fat. Right auricle dilated, also right ventricle, whole right ventricle is somewhat hypertrophied and shows over loading with fat. Tricuspid valve slight thickening of attachments and segments. Mitral valve slight chronic thickening. Distinct hypertrophy of wall of left ventricle. Coronary arteries shows patches of atheroma. Aorta shows diffuse thickening. Aorta valves competent.

LEFT LUNG.

Distended. On section shows oedema and congestion towards the base.

RIGHT LUNG.

Similar.

SPLEEN.

Enlarged, fairly firm on section pale with some swelling of Malpighian Bodies.

LIVER.

Marked post mortem staining, pale, friable, soft, shows cloudy swelling fairly well marked.

LEFT KIDNEY.

Small. Ureter markedly dilated, Surface rough.

RIGHT. KIDNEY.

Distinctly enlarged. Markedly irregular surface. Ureter dilated. Supra renal distinctly enlarged.

BLADDER

Distended. Walls somewhat thickened. free passage into both ureters. Curious polypoid projecting close to left ureter.

EPIGLOTTIS.

Glottis and tissues round swollen, pale, oedematous. Tissues of neck and side of face, especially the right side show extreme swelling.

SUBMAXILLARY PAROTID. shows pus.

Disease Acute nephritisName D. W.Age 30

Occupation

Admitted

Discharged

DATE.	No. of Motions.	3 of Urine.	CHARACTERS OF URINE.		Reaction.	Specific Gravity.	ALBUMIN.		UREA.		GLUCOSE.		Blood.	Bile.	TREATMENT.	
			Naked Eye.	Microscopic.			Grain per oz.	Grains per diem	Grain per oz.	Grains per diem	Grain per oz.	Grains per diem			Dietetic.	Medicinal.
1910 October																
9	2	12	Yellow turbid	Blood: no casts	alkaline	1016	6	4.5		—		+				
10	1	16	Smoky	" " "	"	1018	4.4	5.5		—		+				
11	3	—	"	" " "	"	1017	8	4		—		+				
12	2	4+	"	" " "	"	1018	4.4	4		—		+				
13	3	—	"	" " "	"	1015	4.8	5		—		+				
15	1	28	"	" " "	"	1020	5.5	5		—		+				
18	2	30	"	" " "	"	1025	5.	5.5		—		+				
19	1	29	Yellow colour	Pus: granular casts	"	1015	7	5		—		+				
20	1	44	"	" " "	"	1015	6	6		—		+				
21	0	16	"	Pus: no casts	"	1012	6	5		—		+				
22	2	30	"	Blood: no pus	"	1012	5.5	7		—		+				
24	1	26	"	Blood: no casts or pus	"	1017	5.5	6		—		+				
26	1	26	"	" " " "	"	1012	6	5.5		—		+				
27	1	30	"	Blood absent	"	1015	3.5	6		—		+				
28	2	36	"	" " "	"	1013	4.4	6.5		—		—				
29	0	41	"	" " "	"	1015	3.5	6		—		—				
30	1	40	"	" " "	"	1014	4.	6		—		—				

SECTIONS of KIDNEY.

Shows considerable interstitial change
Chronic Catarrh of the tubules and some fibrosis
of the glomeruli.

Walls of dilated pelvis formed by the fibrous
bands infiltrated with inflammatory cells.
Epithelium desquamated.

SUMMARY of CASE III.

In this case a double Parotitis complicated.
A typical case of Acute Nephritis supervening in
a case of old standing kidney mischief. An
almost complete picture of the nephritic element
of the case is obtained from studying the urine
chart No II. page and comparison along these
lines with the previous case is of considerable
interest. X
interest

For several days patient passed a very small
amount of highly coloured, urine, containing a large
amount of albumin.

ORIGIN and NATURE of the PAROTITIS.

From the clinical record and post mortem
report no septic focus is to be found from which

a spread to the parotid gland by the blood stream might have occurred, and we are forced to seek the origin of the infection in the mouth.

Here again however, we have the same state of affairs which existed in the previous case, a mouth which was

- A. Dry.
- B. Very foul and septic.

The third factor which we emphasised in the previous case namely:—"A lessened state of vitality" is not so pronounced in this case, but although the patient on admission was in a fairly good state of nutrition, his fortnights residence in hospital, associated with considerable vomiting and a markedly restricted diet had greatly reduced his resistance.

Of great importance in this respect however, we would consider his renal condition, as well as the very severe acute attack of nephritis, the old standing kidney disease must have tended greatly to lessen the patients resistance to the onset of such a condition as Parotitis.

The spread to the parotid gland of the opposite side, which has often been recorded, occurred in this case on the fifth day, the time of its onset would appear to be purely accidental judging from recorded cases, where the time has

varied from 24 hours to as many days or longer. That the parotid of the opposite side should be affected in these cases is to the writers mind decidedly in favour of a duct borne infection in these cases as it is difficult to imagine a secondary spread to occur by the blood stream, without involving other organs in the body, none of which were affected in this case.

Therefore we conclude in this case as in the former, from the study of the clinical record and post mortem report, that infection of the Parotid gland occurred by way of the duct.

COURSE of the PAROTITIS.

The parotid on the right side was from the first the site of the severe inflammation which occasioned the patient considerable pain and discomfort and local applications of Belladonna Lead and opium, and Ichthyol failed to give relief. There was no local indication of suppuration, but this is exceedingly difficult to define.

The temperature: Pulse rate and Respirations were unaltered throughout the course of the disease by the onset of the Parotitis despite its severe character, and this led to the conclusion that suppuration had not occurred.

The bearing of the parotitis on the fatal issue in this particular case was probably quite indirect. Death was due to "Oedema Glottidis" but at least we may conclude that a Parotitis especially when it is double is of evil omen, as indicating a greatly lessened resistance on the part of the body tissues.

CASE IV.

GASTRIC ULCER complicated by the onset of double PAROTITIS.

R. D. aged. 25. married.

Surfaceman.

Admitted March 16 th. 1911.

Complaint. Vomiting of blood.

Duration. 4 days.

Examined. 19-3-1911.

History.

Social history and home surroundings. satisfactory.

Formerly smoked 2 ounces of tobacco per week, smoking given up 6 months ago on account of digestive disturbance.

Present Illness.

Six months ago patient began to experience a heavy feeling in the stomach especially after breakfast: He frequently had a very bad taste in his mouth, this was thought to be the result of smoking because it stopped when smoking was given up.

Sunday March 12th 1911.

Felt squeamish for breakfast, went to church after breakfast and there felt sick, came out and walked 50 yards when he had to hold on to a dyke. he fainted but was helped home to his father's house, on attempting to drink a cup of tea he vomited, vomit is a dark brown in colour, like coffee grounds, several ounces were vomited and gave relief. Able now to walk home He had tea and had a short walk, then returned home and took a dose of salts, after which he again vomited, this time greenish material, no coffee grounds, motions had been black all day. After taking some bread and cold meat he went to bed and slept well, and was now free from pain.

Monday 12th March. 1911.

Rested all day: Passed black motions Vomited three times in the afternoon and evening at first coffee grounds then thick clotted blood and again dark fluid blood. He felt very faint and weak. A doctor was called in and recommended sips of hot water and ordered medicine, to be taken 4 hourly.

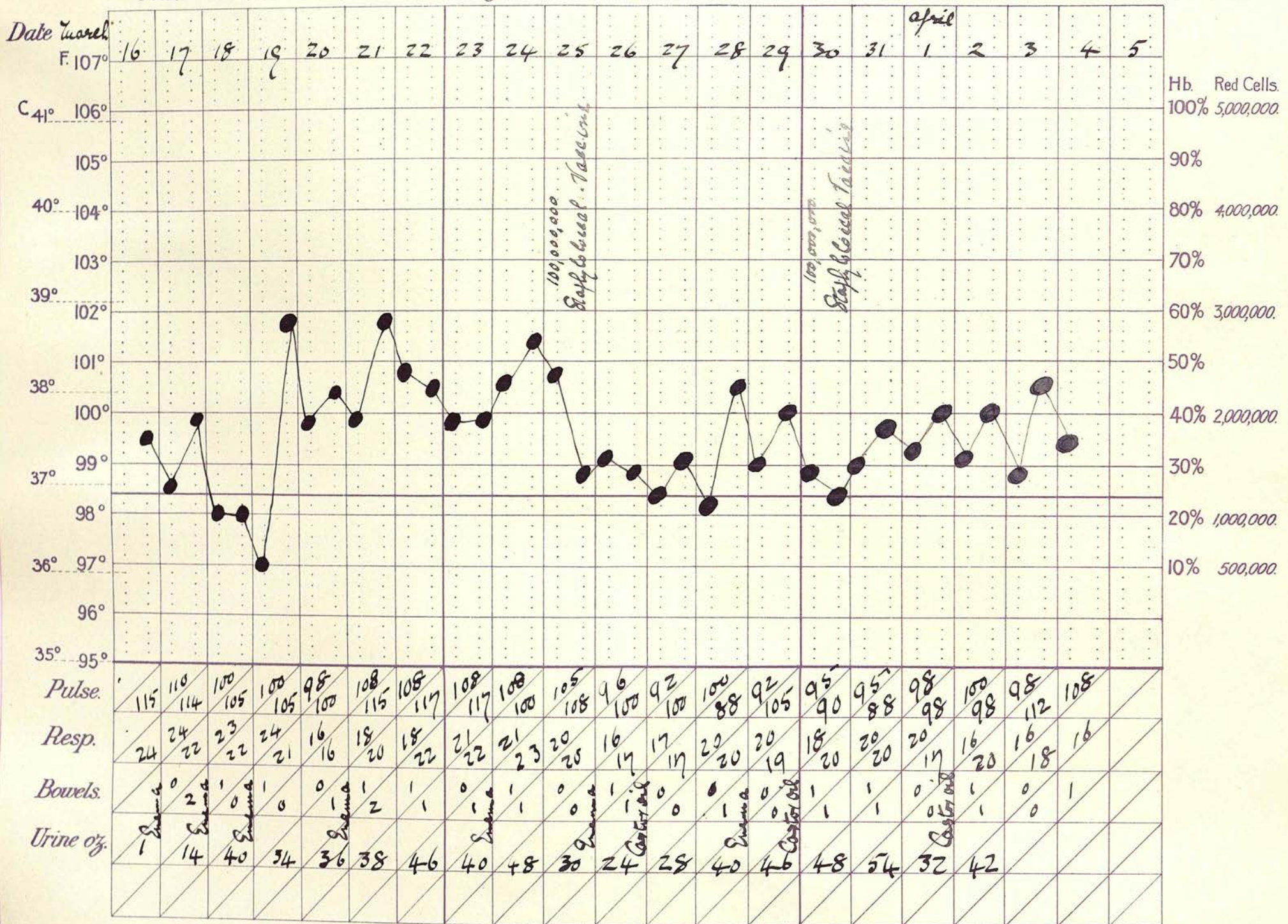
Temperature Chart.

Case. IV.

Name *R. W.*Age *25*

Clin. Clerk.

No.



Tuesday 14th.

In bed all day. 12 noon vomited a little clotted blood. 2. p. m. vomited his milk and potash soured and discoloured by coffee grounds from stomach: Black stools passed.

Wednesday 15th.

No vomiting. Black stools passed.

Thursday

Admitted to ward XXIX. R. I. E.

Throughout the illness there has been a marked absence of pain.

General Facts.

The patient is a sallow, and obviously anaemic man, with tinge of yellow in the conjunctivae. Expression is anxious, attitude, on his back as ordered but is able to lie on his side.

Temperature 99.6 On admission now risen to 102.

ALIMENTARY SYSTEM.

Lips and gums are pale and dry. Tongue pale, flabby and dry, white fur at sides, Teeth fairly good except molars, which are all carious.

Mouth excessively dry and troublesome to patient, swallows perfectly easily. Appetite poor: no special desire for food. Thirst is troublesome as patient is given no food by the mouth at present.

Between meals he is quite comfortable but after a meal he has for months complained of a feeling of weight in the stomach, no actual pain however has been felt. There is no heartburn or waterbrash. Bowels always moved once a day, stools never been black before Sunday 12th.

At present.

Stools are not formed. Are black like tar and contain blood.

ABDOMEN.

Moves freely with respiration, no local rigidity. The right upper quadrant is more prominent than the left on palpation.

There is no abnormal tenderness or resistance to be made out.

PERCUSSION.

Liver dulness in mammary line -4 inches, interrupted by a resonant area to the costal margin, giving note of colon. There is comparative dulness below the costal margin and to the right of the Umbilicus.

STOMACH.

Not dilated.

SPLEEN.

Slightly larger than usual.

CIRCULATORY SYSTEM.

Fainted with the onset of vomiting, never before. No other symptoms referable to the circulation.

INSPECTION.

Chest is well formed. Apex beat visible.
Excessive Carotid pulsation.

PALPATION.

Apex beat in fifth left interspace three & three quarter inches from midsternum. Apex forcible and well sustained Systolic thrill over the carotids.

PERCUSSION.

	3rd rib.	
1 3/4"		3 3/4"

AUSCULTATION.

There are systolic murmurs, soft and blowing in all areas, especially well heard at the base.

Second sounds are closed.

PULSE.

100 per minute: regular in rate and rythm, rise is sudden, apex sustained, impulse small, vessels healthy, vessels easily compressed. Maximun systolic pressure. 95 m. m. Hg.

RESPIRATORY SYSTEM.

Respirations 20 per minute.

Abdomino-thoracic in type.

No abdominal sensation: No cough. Chest moves equally, no local changes except bulging on the right lower half. Vocal resonance and vocal fremitus no change.

Percussion note is clear.

AUSCULTATION.

Breath sounds are vesicular, no accompaniments.

HAEMOPOIETIC SYSTEM.

No enlarged glands. Spleen slightly larger than normal. Thyroid gland not enlarged.

BLOOD.

Red Blood Corpuscles. 2.030.000.

White Blood Corpuscles. 10.400.

Haemaglobin. 45 %.

DIFFERENTIAL COUNT.

Polymorphs.	71 %.
Lymphocytes.	25 %.
Large Mononuclears.	2 %.
Eosinophils	2 %.

The red cells are small pale and irregular. Some nucleated reds are present. All normoblasts.

URINARY SYSTEM.

Patient has never had any difficulty with the water.

URINE.

Amber coloured. Acid.
 Clear. Specific gravity. 1020.
 No abnormality.
 Urea. 12 grains per ounce.

NERVOUS SYSTEM.

Beyond a heavy dull headache at the present time, there has never been any affection causing pain in the head. No fits. Sensations perfect.

Motor. Organic reflexes intact. Skin reflexes all present and lively. Plantar reflex gives plantar flexion

Tendon jerks sluggish.

Biceps active.

Knee jerks sluggish.

Coordinated voluntary movements are carried out perfectly.

Vaso motor.

Marked pallor; all the vessels seem contracted.

Cerebration perfect, attention and memory good.

PROGRESS.

I6-3-II. On admission patient was put on Rectal alimentation and nothing whatever was given by the mouth. There has been no pain.

Black unformed stools were passed and found to contained blood. Patient is troubled by thirst and complains of a dryness of the mouth.

I8-3-II. Patient quite comfortable except for dry mouth. Constant mouth washes are being given of Potassium Permanganate and Potassium Chlorate. No food is given by the mouth. Mouth is very foul, tongue is dry and like coarse sandpaper. There is a yellow spit.

19-3-II. The right parotid gland is enlarged.

The tongue was painted with Boro glycerine.

Mouth exceedingly dry. Temperature 101.8.

In the evening the left parotid is now much swollen and very tender to the touch. The swelling of the right parotid has subsided.

20-3-II. Patient complaining of pain in the left parotid which is very much enlarged and tender.

No fluctuation is to be made out; yellow pus may be expressed from Stenson's duct.

Mouth is very dry. Albumin water with lemon juice, half an ounce at intervals given for the thirst. In the evening the left parotid is still very large.

21-3-II. Left parotid still very large. Hot fomentations with Belladonna applied. Pain relieved during the afternoon.

22-3-II. Pain much easier in every way and looking brighter. Left parotid decreasing in size. A culture was made from the mouth and one from the pus squeezed from Stenson's duct. Both gave pure growth of Staphylococcus Pyogenes Aureus.

23-3-II. Left parotid decidedly smaller and general condition much improved. Milk and lime water added to the diet.

25-3-II. An antogenous vaccine of one hundred million staphylococci given by the mouth in normal saline. Temperature dropped by the evening to 98.8 F from 100.6 F. There was no initial rise.

26-3-II. Patient keeping much easier and milk foods added to the diet.

27-3-II. Patient keeping very well, and sleeping well. Enjoys his food.

28-3-II. Temperature risen to 100.4 F. Slight tenderness over right parotid especially below the lobe of the ear.

29-3-II. Right parotid rapidly swelling and painful. Belladonna formentations applied. General symptoms show no change.

30-3-II. A second dose of 100 million Staphylococci given. Right parotid increasing in size.

3I-3-II. Right parotid now hard and brawny.
Some throbbing pain in the gland.

2-4-II. Area of parotid swelling increasing, the swelling is now softer and pus is undoubtedly present under the parotid fascia, and the overlying skin is becoming red.

3-4-II. Patient was transferred to ward I6 and the parotid swelling was incised in two places A little below and a little behind the mastoid process and a little above the angle of the jaw. Several ounces of yellowish blood-stained pus escaped. The pain was immediately relieved by this evacuation of the pus.

7-4-II. Patients general condition much improved The discharge from the incised parotid which was copious for the first two days is now subsiding and the gland has a feeling of firmness.

10-4-II. Patients general condition shows steady advance. Discharge from parotid almost stopped. Gland is now very hard and firm. The anterior

incision has been allowed to close up. The posterior wound still contains a small drainage tube the wound gives indication of rapid closing.

The urine has never contained any abnormal constituents.

SUMMARY of CASE IV.

The provisional diagnosis here is Gastric Ulcer. The excessive loss of blood both by the mouth and by the bowel is a marked feature associated as it is with entire absence of pain throughout the illness.

ETIOLOGY of the PAROTITIS.

A complete investigation of the case entirely fails to supply any septic focus, other than the mouth, as a possible source of infection of the parotid. Examination of the mouth however, reveals a condition of affairs quite in keeping with that found in the preceding cases.

I. An excessively dry mouth, salivary secretion having been completely arrested. This condition was contributed to and assisted by the fact that the patient was on Rectal alimentation.

II. Marked oral sepsis, including septic foci in connection with the molar teeth nearly all of which were carious. To the condition of the teeth, especially the molars, one would attach great importance in considering the factor of oral sepsis in its bearing upon the Etiology of Symptomatic Parotitis.

The third element on which we have laid stress - lessened resistance, due to a lowered vitality -

was present here also.

This is a factor which varies greatly in each case and in this case was dependent very largely upon the great loss of blood, which reduced the number of red blood corpuscles to one half of the normal number, the patient prior to the onset of the parotitis being extremely weak and markedly anaemic.

We have therefore good reasons to believe that in this case, as in the others infection of the gland occurred by way of Stenson's duct.

Bacteriological Evidence

In support of this theory as applied here there is the Bacteriological evidence afforded by Cultivations made from the mouth.

I. The mouth.

II. The pus expressed from the opening of Stenson's duct.

The growth in each case gave a pure culture of Staphylococcus Pyogenes Aureus.

COURSE and TREATMENT of the PAROTITIS.

The parotitis in this case was of a severe nature and failed to respond to medical measures, The right parotid gland was found to be swollen on the morning of the 19th, a week after the onset of the patients initial gastric symptoms.

The left parotid gland became affected on the evening of the same day, the interval of only 12

12 hours between the involvement of the second gland is interesting as compared with Case III. where several days intervened before the involvement of the gland of the opposite side. Reference to the literature, confirms the fact of which these two cases are an illustration, that no time limit may be laid down for the onset of inflammation in the gland of the opposite side, which as seen in cases I & 2 may not and frequently does not become involved.

The inflammation in the right parotid subsided within 24 hours, The left parotid continued to be swollen and tender for several days, and although no fluctuation could be elicited, pus could readily be expressed from the opening of Stenson's duct.

On referring to the temperature chart it is to be noted that with the onset of the Parotitis on the 19th, the temperature rose to 102 F, but there was no alteration of the pulse or respiration rate.

The temperature continued to swing until the 25 th, when patient was given an antogenous vaccine of 100 million staphylococci.

Although there was no initial rise of temperature on 4 hourly observation, the patients temperature quickly fell to 99" and the swelling of the left parotid which had remained fairly well marked quickly subsided and did not again recur.

However on the 28th return of the inflammation occurred in the right parotid evidenced by tenderness over this region and accompanied by a rise of temperature.

A second dose of vaccine caused the temperature to fall to normal but the parotid condition advanced and failed to yield to local applications. As evidence of pus formation appeared the gland was incised after which the patient made a good recovery and the parotitis has not returned, the patients general condition being much improved and there is reason to believe he will completely recover from his gastric trouble.

VALUE of VACCINE TREATMENT.

The use of the Antogenous Vaccine was of interest and from studying the temperature chart and the patients condition one would say that it had a beneficial influence upon the course of the parotitis, and that its failure to prevent the recurrence was probably due to the fact that it was not administered in a sufficiently large dose as evidenced by the absence of an initial rise of temperature.

HISTORICAL and CRITICAL REVIEW of previous work
dealing, for the most part, with the
PATHOLOGY of SYMPTOMATIC PAROTITIS.

As soon as it became recognised that Symptomatic Parotitis was a secondary condition and not a primary inflammation of the parotid gland as occurs in mumps, that it frequently went on to suppuration, and that the disease was not contagious, various theories were advanced to explain its origin

I. HEAT DEGENERATION THEORY

Liebeneister regarded the degeneration as due to hyperpyrexia, but as fever in some cases is entirely absent this theory failed to explain the disease and was soon dropped.

II. TOXIN EXCRETION THEORY.

Bouchard propounded the theory that the parotitis was due to the failure on the part of the gland to excrete the Toxins manufactured by the organisms causing the primary disease, but as parotitis occurred in cases where Toxaemia was absent this view which did not gain wide acceptance was dropped.

III. SYMPATHETIC THEORY.

This theory which was suggested by Stephen Paget as the result of observations of a large series of cases is the one which until the last few years was most generally accepted, at least in this country.

But comparatively recently Mr Paget stated that the view previously held as to the possibility of a sympathetic parotitis was now untenable.

However Paget's work still remains the chief systematic study of symptomatic parotitis in this country.

In his first paper writing on parotitis after injury or disease of the abdomen or pelvis he began by quoting Graves lecture on the connections between the diseases of various organs.

You are aware "he says" that some organs when labouring under disease are apt to implicate other organs, giving rise to various deranged conditions, which are developed sometimes simultaneously, but in general consecutively and in sequence. It is of the greatest importance to study each link in this morbid chain, and to ascertain the nature of its connections, as to have a distinct conception of the whole.

Paget then quotes his own experience of Pyaemia in acute necrosis where in 61 % of the cases abscess of the heart or kidney followed -while in pyaemia after amputation, in a slightly larger series of cases pneumonia occurred in only 1 % of cases. In this way he says disease may disclose relations between organs which are latent in health.

Paget then described a series of 101 cases of parotitis following on injury or disease to the abdomen or pelvis

In 93 % of these cases the parotitis was a solitary event.

Paget then states that parotitis following injury or disease of other parts of the body is usually part of a general Pyaemia or Septicaemia with abscesses and effusions into the joints, and he infers that parotitis in the form that follows injury or disease of the abdomen and pelvis cannot in most cases be due to any ordinary form of pyaemia.

This form of parotitis has no fixed period of incubation and even no regular course. Its onset is variable in character, rigors are exceptional and there is frequently very little

constitutional disturbance at the onset . Its course is irregular it may subside and swell up and subside again.

These facts make it impossible for us to say that this form of parotitis is due to any ordinary form of pyaemia or septicaemia.

The mouth as the source of infection was excluded because Socia Parotitis which lies so near the mouth was affected only once or twice in the series of IOI cases and because the mouths of patients with abdominal or pelvic lesions are not more parched than the mouths of other patients. Admitting that the general condition of the patient especially in cases of septicaemia and pyaemia is concerned in the productions of this form of parotitis must we not " he says" take into consideration the reflex action of the nervous system ?. He then gives some well known experiments to show that the secretion of saliva is in part controlled by the sympathetic nervous system

The influence of the nervous system is the more probable- he writes - if we consider how often retention or suppression of urine follows abdominal operations; here the influence of the

nervous system cannot be doubted. Again, attention has lately been drawn to the fact that in many cases of disease of the Thoracic or abdominal viscera, there is inequality of the pupils; they differ not only in size but also in their susceptibility to light and this difference between them may change as the disease goes on.

This inequality of pupils is it is said, most often found in those diseases which not only affect the system generally, but which like pneumonia, pleurisy and renal colic, are also definitely localised.

In the same way with regard to parotitis after abdominal or pelvic lesions we may admit that the general condition of the patient may help to cause it, without denying the local influence of the reflex nervous system.

Finally it seems probable that this change brought about in the gland is not a spasmodic closure of the duct but a change in the gland itself, not a retention but a suppression. Retention of saliva causes a mere passive dilatation of the gland which varies in a transient fashion with every meal, never suppurates, is never accompanied by disturbance of the general health and vanishes when a probe or catheter is passed up the duct.

As Paget no longer adheres to this theory it is perhaps unnecessary to discuss at length the arguments which he used in support of it, but it is interesting to think a sympathetic theory which rested very largely on negative deductions should for so long have been held a sufficient explanation of a condition, which although it does not always go on to suppuration, is nevertheless always associated with the presence in the ducts of the gland of micro-organisms, although these organisms may be found in some cases only at the extremity of the main duct.

IV. Theory of infection of the blood stream.

Osler: Benoit & Pozzi all support the theory that in many cases at least infection occurs by the blood stream but the strongest evidence has been brought forward by Tebbs in a paper read before the Medico-Chirurgical Society of London

SUMMARY of TEBBS Paper.

Tebbs says that the view most generally held is that in the absence of food from the mouth the salivary flow is not stimulated; the mouth consequently becomes dry and septic, and

an opportunity is afforded for a spread of infection to the gland by the duct.

The various observers, then, for the most part recognise a predisposing cause - the suppression of secretion, which is variously attributed to reflex nervous effects and to the absence of food from the mouth and an exciting cause infection.

The actual inflammation is always preceded by suppression of secretion - this is constant; the parotitis itself is inflammatory and septic. We have, therefore, to deal with two stages in the process. The first stage consists of the suppression of the secretion, a process which throws the gland out of its normal condition, and renders it more liable to infection, and the causes that operate to produce this suppression of secretion may be called the predisposing causes. The second stage consists of the actual inflammation and the cause at work in this process - the exciting cause - is infection. These may be considered separately.

A. The Predisposing Causes leading toSUPPRESSION of SECRETION.

That the onset of Symptomatic Parotitis is preceded by suppression of the salivary secretion is an old observation. The suppression has been ascribed to the absence of food from the mouth, it has been suggested that in default of their natural stimuli the salivary glands are not excited and cease to excrete. But although the absence of food may assist in the suppression of the secretion it is probably not the main cause.

In Mr Tebbs cases not only had many of the patients been receiving solid food by the mouth for considerable periods before the onset of the parotitis but in many cases mouth feeding had never been interrupted. Even among 15 non operative cases of gastric ulcer, six only were on Rectal alimentation at the time of the onset of the parotitis. We must "he says" therefore look to other causes for the suppression of the secretion. These causes are to be found in injury or disease to the Alimentary canal.

Apart from the direct experiment quoted in Mr Paget's paper that interference with the alimentary canal of a dog leads to a suppression

of salivary secretion, we have abundant evidence clinically of the effect of abdominal operations on the secretion.

Thirst and dryness of the mouth are always more marked after operations on the stomach. Dryness of the mouth is also especially noticeable in cases of peritonitis. Injury or disease of the gastro intestinal tract seems to have the effect in some way of inhibiting salivary secretion. This inhibition may also be marked after operations on the pelvic organs, in this case it is probable due not so much to the interference with the pelvic organs themselves as to the accompanying damage to the gastrointestinal tract or peritoneum.

The probable cause of the suppression of secretion is a reflex nervous inhibition, set up by reflex nervous impulses from the injured alimentary canal.

There is a possibility that another cause may be at work as well.

Bayliss & Starling have pointed out the "rôle" played by the body secretion as an excitant of pancreatic secretion, It is obvious that grave lesions of the stomach leading to a Temporary paralysis of that organ, or such a condition as peritonitis, would largely interfere with the formation of this body and might in that way lead to a diminution of the salivary secretion. In the absence however, of more exact knowledge on this point we must allot the chief share in the suppression of the salivary secretion to reflex nervous influence.

Suppression of the secretion of the gland- whatever way caused by throwing the gland out of play, would seem to lower its resistance and leave it liable to infection. It remains therefore to consider the method in which the gland becomes infected.

The Exciting Cause - Infection

The direct or exciting cause of parotitis is undoubtedly infection; the only question is the path by which it reaches the gland. There are three possible ways in which infection might gain access to the gland - firstly by the blood stream, secondly from the mouth along the

duct, thirdly by direct spread of an inflammatory process from surrounding structures.

My own opinion (Tebbs) is decidedly in favour of spread by the blood stream and I will endeavour to state as briefly as possible my reasons for this view.

1. Under this heading Tebbs analyses 60 cases collected by Paget; and points out that there is direct evidence of sepsis in about one half of the 60 cases.

He points out that Parotitis follows operation for gastric ulcer in 18 % of cases; whereas in cases of gastric ulcer treated without operation it occurs in 2 %, and he concludes that the septic soiling of the peritoneum in the case of a patient with perforated gastric ulcer has much to do with the production of the parotitis. As regards appendicitis, in all the cases complicated by parotitis, recorded at all in detail either abscess formation or general peritonitis was present.

2. The second point he brings out is the frequent association of parotitis with other septic complications -Tebbs places the figure for post operative cases as high as 30 %.

He argues that when parotitis forms one of many secondary infections, it seems hardly reasonable to suppose that the infection in the case of the other complications occurs by the blood, while in the case of Parotitis it occurs by the duct, nor does it " he says" seem reasonable to separate out the cases of Parotitis which occur with other secondary complications and label them pyaemic and adopt a different explanation for the parotitis which occurs as a solitary event.

3. The third argument is that the severity of the parotid infection varies with the severity of the primary condition and with that of other complications, suggesting that the same organisms are at work in both processes. Where the parotitis occurred with other complications in the cases recorded, if any complications resolved the parotitis usually did the same while if the other complications were a suppurative one, the parotitis usually went on to suppuration. For example, a resolving parotitis would be associated with a simple pleurisy, a suppurative parotitis with an Emphyema.

Also there is a general though not absolute relation between the severity of the parotitis

and that of the primary condition

4. Fourthly the complications of parotitis is relatively to the number of abdominal operations far less frequent at the present time than 20 or 30 years ago this improvement Tebbs attributes to improvements in Antiseptic Surgery, for if the parotitis were due to a spread from the mouth improvements in Antiseptic Surgery would have no effect on it.

5. The fifth point is that brought out by Mr Paget, that if the spread were by way of the duct the first portion of the gland should be the *Socius Parotidis*, this was found in most cases not to be so.

6. Oral Sepsis. Tebbs could not establish any connection between Parotitis and Oral Sepsis. With a view to testing the efficiency of oral antiseptics, Tebbs analysed 300 consecutive cases of gastric ulcer. Among 68 cases in which antiseptic mouth washes were systematically employed there were four cases of parotitis, while among 232 cases treated without them, there were only two cases; and in order to exclude other factors taking the cases that were being fed by the Rectum only, parotitis was still four times as frequent

in those in whom mouth washes were used nor "he says" can it be objected that mouth washes were used in patients with particularly septic mouths, for it is the practice of some to use them as a matter of routine in all cases, while others do not. The only conclusion is that oral antiseptics has little effect in preventing complications.

Tebbs then explains the process as follows In these patients that develop Parotitis there is a species of mild septicaemia. The organisms may be absorbed from the peritoneal cavity, or in the case of gastric operation or ulceration of the gastro-intestinal tract, through some abrasion of the mucous surface and reach the parotid by the blood stream.

In the case of a healthy functional gland they might pass through it, as they pass through the kidney and be thrown off in the secretion. But in these cases the secretion has been, reflexly or otherwise, suppressed, and the organisms instead of passing the gland are retained within it and start an inflammatory process.

The suppression of secretion acts not only by lowering the resistance of the gland, but also by allowing the organisms to obtain a footing.

In fact in this production of parotitis after an abdominal operation we have an exact parallel in those cases of acute nephritis following operation on the lower urinary passages, where the distribution of the renal lesion shows the path of infection to have been by the blood stream. In each case the effect of the operation is to depress vital activity of the gland, and to allow the organisms which otherwise would pass through it and be excreted, to obtain a footing and start an inflammatory process.

The only direct evidence for the theory of a spread of infection by the duct is an inflammation of the duct, preceding that of the gland, which is said to have been observed in some cases.

Finally, if infection occurred by the duct parotitis should be a common and not a rare phenomenon, since the necessary factors for such an infection are constantly present both in post operative and febrile conditions.

III.

CRITICISM of TEBBS VIEWS.

Treating the subject purely from the clinical standpoint Tebbs has built up a very strong case for infection by the blood stream, but none of his arguments is free from objections and in their entirety they are not conclusive.

In deciding upon the Etiology of this condition the microscope must be the ultimate test, and in not one of his cases has Tebbs brought forward any histological evidence in support of his theory, and one ventures to think that had he done so he would, from this source, have been quite unable to support his argument. The histological appearance of the parotid gland in cases of Symptomatic Parotitis will be referred to in detail, in discussing the "theory of duct spread" and in describing the condition which was present in the cases brought forward by the present writer, in support of the theory of duct spread.

To return to the several arguments Tebbs advanced.

Firstly. The argument that in the majority of the cases of parotitis secondary to abdominal conditions, sepsis was a marked feature, would hold

equally well in support of the theory of duct spread, because in these cases the mouth becomes very foul and septic and therefore an ascending infection of the duct is the more likely to occur.

Secondly. That parotitis may occur as a part of a general pyaemia is admitted, but the cases are comparatively rare, in which histological examination has revealed a primary source of infection by the blood vessels.

It seems to us just as reasonable to argue that because, in some cases, a septic Broncho-Pneumonia has its origin in a blood borne infection therefore every case of septic Broncho Pneumonia must originate in this way; as to argue that because undoubted cases occur in which the parotid has become infected, in the course of a general pyaemia by organisms conveyed by the blood stream, therefore every case of Parotitis must originate in the same way and that it is unreasonable to assume a different origin for the infection.

Thirdly. In answer to the argument that the severity of the parotitis bearing a definite relation to the severity of the primary disease,

indicates that the same organism is at work and supports therefore blood spread, one would say that rather it illustrates the fundamental point in the Etiology of the condition.

Namely that parotitis is an indication of lowered vitality on the part of the tissues of the body, and in proportion to the degree of lessened resistance, so is the severity of the parotitis.

The main cause for the lessened resistance being the primary disease, our argument might therefore be given as follows: In proportion to the severity of the primary disease so is the severity of the parotitis. This can obviously have no connection with the mode of spread.

Arguments four and six may be taken together the sum of them is that oral sepsis has nothing to do with the incidence of parotitis.

In reply to this argument one would quote the conclusions arrived at by Drs. Rolleston and Oliver, after studying 34 cases of secondary parotitis in cases of gastric ulcer treated medically by oral starvation.

Their conclusions are as follows:-

1. Secondary parotitis may complicate cases of gastric ulcer treated medically by oral starvation.
2. That it occurs ten and a half times more frequently in such cases than in cases allowed

fluid by the mouth.

3. That it is an outcome of the dry condition of the mouth and that mouth washes do not prevent its occurrence.
4. That it is more often unilateral than bilateral
5. That suppuration occurs in about one fourth of the cases and that this constitutes a grave complication.

Conclusions two and three of the above have a direct bearing upon the point of oral sepsis Rolleston and Oliver agree with Tebbs in his statement that mouth washes and local antiseptic treatment of the mouth cannot prevent the occurrence of parotitis. Tebbs uses this as an argument in favour of blood spread and against the theory of a duct infection. But it is impossible even by taking the most careful precautions to render the mouth aseptic, thoroughly to accomplish this and it is quite conceivable that organisms may still be left in the recesses of the buccal mucous membrane which could find their way into the opening of Stenson's duct. Under such circumstances the statement that oral sepsis plays no part in the production of parotitis is hardly justifiable.

Rolleston and Oliver's statement that parotitis is an outcome of the dry condition of the mouth is of more importance than the speculative

statement regarding oral sepsis .

Rolleston and Oliver found that parotitis occurred ten and a half times more frequently in cases of gastric ulcer treated by oral starvation, than in those cases which were given fluid by the mouth. This can only mean that a dry mouth affords a favourable nidus for the organism, which can from this situation reach the gland only by way of the duct. A dry mouth could have no controlling influence over a septic embolus conveyed by the blood stream.

Tebbs fifth argument in favour of blood spread is not a formidable one.

It was first advanced by Paget who observed that the socia parotidis was not the part of the gland early affected although it was nearest the mouth.

This would appear a sound argument on the surface but it is not difficult to understand why the socia escapes, if we examine the anatomical distribution of the ducts in the parotid.

The main duct goes on dividing in a regular manner until its ultimate divisions are reached.

The socia is to be regarded anatomically as a separate lobe of the gland to which a special branch of the duct is given. It seems quite reasonable to assume that once the organism has gained

entrance to the duct it will follow the line of least resistance and pass to the ultimate divisions of the duct, instead of branching off going out of its way in fact to enter the branch of the socia. That this might occur in some cases is of course probable, Further as Bunts points out, clinically it must be difficult to establish an inflammation of such a small portion of the gland. Its attendant enlargement can be not slight and the pain on palpation cannot probably be so accurately localised, as to enable us to say, whether it is in this point or the anterior portion of the gland proper. Moreover as has been determined histologically and clinically inflammation of Stenson's duct precedes that of the Parotid gland. Malcolm reported two cases in which the opening of Stenson's duct was visibly inflamed, when attention was first directed to the gland, and in both cases carious teeth were situated opposite the opening of the duct. Hawthorne noted this early infection of the duct stating that from the very earliest appearance of Parotitis, pressure over the duct would cause the expulsion of a drop of pus from its orifice. For these reasons alone we believe that Tebbs and others have failed to prove spread by the blood stream, but the positive

arguments in favour of duct spread are still more convincing and prove conclusively the spread occurs by way of the duct.

V. THEORY of DUCT SPREAD.

This theory, the last to be brought forward, is still by no means generally accepted. Evidence in its favour has been adduced by various workers some approaching the subject from the clinical and histological standpoint, others from the experimental side, of the former Bunts and Bucknall in this country, Hanan and Pilliet on the continent are the chief, the experimental work has been carried out almost entirely by European workers especially by Claisse and Duplay. Hanan and Pilliet found on examination of sections of the parotid microscopically.

1. that the ducts were choked with debris containing micro-organisms.
2. that the inflammatory processes present invariably began around the ducts in the centre of each lobule and only spread later to the periphery of the lobule, and the perilobular connective tissue in which the blood vessels are situated.

They concluded therefore, that secondary parotitis could not be of embolic origin, or else

the inflammation would have originated around the vessels and that the fact that inflammation began simultaneously in the centres of many lobules at once, pointed to ascending infection of the ducts as the real source of the affection - a conclusion which was further borne out by the actual presence of micro-organisms in the ducts themselves in such cases.

Microscopical or Bacteriological evidence in favour of ascending infection has been brought forward by the following observers:

Girode, Prantois, Diog, Swan, Bosquier, Morley, Subkovski, Toupet, Testa, Fischel, Jauowski, and Lehmann.

Bucknall gives an account of six cases, where in each instance a bacteriological or microscopical examination afforded ample evidence that the affection of the Parotid was due to an ascending infection of Stenson's duct from the mouth.

Two of the cases occurred within a week after the onset of severe gastric symptoms.

Two in the course of Enteric Fever.

One in the case of Pyelo nephritis.

The sixth during an attack of Diabetic Coma.

In two of these cases, those in which complete recovery ensued, cultivations from the mouth, from the pus evacuated from Stenson's duct and from the pus obtained by incision of the gland, pure

cultures of Staphylococcus Pyogenes Aureus were obtained. Similar cultures were obtained from the same sources in each of the other four cases, but in addition histological examination of the gland revealed the following conditions:

In the early cases:-

Many of the ducts in the centres of the lobules were distended and blocked with debris.

The central portions of the affected lobules were inflamed and infiltrated with leucocytes and inflammatory cells, which however had not extended to the periphery of the lobules.

The inter lobular tissue was unaffected, the blood vessels being quite patent and free from infected material.

In a more advanced case.

Inflammation and necrosis had occurred around the ducts in the central portion of the lobules. There was slight infiltration of the inter lobular tissue, the blood vessels however in this area were still unaffected.

In the fourth case.

Where supuration had advanced to an extreme degree. Some of the lobules had completely broken down and undergone necrosis, but even in this advanced stage, although the inter lobular tissue was infiltrated to a considerable extent, no infection of the blood vessels had occurred

The same evidence has been forthcoming in all cases of Parotitis that have been submitted to Pathological and histological examination.

EXPERIMENTAL EVIDENCE.

Claisse and Duplay experimentally on animals proved:

1. That the healthy parotid duct cannot be infected either by smearing its orifice with micro-organisms, or by introducing organisms into its lumen, via the normal orifice of the duct or by an artificial fistula.

2. That an ascending infection of the duct, leading to the production of Parotitis, have been observed in secondary Parotitis in the human subject, can easily be produced by either of these methods under the following circumstances:

A. If the micro organisms introduced were excessively numerous or virulent.

B. If the general vitality of the animal had been depressed by starvation or other methods.

C. If the normal secretion of the Parotid had been altered in quantity or quality, in one or other of the following:

1. By ligaturing the duct and preventing the flow of saliva, after infection had been attempted.

2. By administering drugs, such as opium, which check and alter the salivary secretion.

All these conditions which Claisse and Duplay found to be necessary for the production of parotitis in animals are met with in cases in which parotitis occurs in the human subject, and the comparative frequency with parotitis occurs in connection with each individual disorder varies directly with the degree and frequency with which these predisposing conditions are met with in each instance.

The importance of deficient Salivary Secretion
in the causation of Parotitis.

Following on Claisse and Duplay's experiments Dr. Soltan Fenwick's observations are of considerable interest.

Writing on " the prevention of Parotitis " during renal feeding "

Fenwick says: "When I first adopted the use of large nutrient enemata of peptonised milk (15 to 20 fluid ozs), some ten years ago, and trusted to the usual methods of cleansing the mouth a very large number of cases developed Suppurative Parotitis. An effect was then made to promote the continuous secretion of saliva, with the object of irrigating the ducts, and thus of preventing an ascending infection, and with this object the patients were directed to chew horse radish Pellitory or pieces of raw meat at intervals, or

to keep a pebble constantly in the mouth.

Eventually it was found that an india rubber teat about 2 inches in length met all the requirements of the case, and that patients were quite content to suck it for hours at a time, with the result that the mouth remained quite clean and moist.

When not in use the teat is kept in a weak solution of Condys fluid. Since this simple device was adopted I have treated 300 cases of haematemesis by Rectal alimentation, lasting from ten days to seven weeks without being troubled in a single instance by parotitis.

It is only in hospital practice where the mouth is very foul at the time of admission, that the gland occasionally becomes inflamed before any measures can be taken to prevent it."

These observations afford valuable evidence in favour of the theory that spread in cases of Symptomatic Parotitis occurs by way of the duct, from an infected mouth.

Also they confirm Claisse and Duplays experimental observations that so long as the mouth is kept moist by a free flow of saliva, infection of the parotid does not readily occur.

SUMMARY of CONCLUSIONS.SYMPTOMATIC or SECONDARY PAROTITIS.

- I. Incidence.
- II. Pathology.
- III. Course.
- IV. Treatment.

I. Incidence.

Any classification of the diseases which have been complicated by Parotitis must necessarily be very artificial. As fresh cases are put on record it becomes increasingly plain that the onset of Symptomatic Parotitis is dependent upon causes, which may be associated with almost any diseased condition, whether acute or chronic, and involving almost any organ of the body. There is no definite incubation period of Symptomatic Parotitis, And the condition is not infectious, it must however be regarded as a serious complication of any disease.

II. Pathology of Symptomatic Parotitis.A. Etiology.

Infection of the parotid gland occurs by direct spread from the mouth along Stenson's duct to the gland, in all cases except those in which the parotitis is part of a generalised Pyaemia.

Exciting Cause.

This is organismal. In nearly all cases Staphylococcus Pyogenes Aureus is the cause.

Predisposing Causes.

- I. Diminished resistance due to lowered vitality on the part of the patient.
- II. Diminished salivary secretion, leading to a dry state of the mouth.

B. Anatomical appearances of the gland.I. Normal parotid gland.

The elastic tissue of the ducts is confined to the walls of the extra lobular ducts, the small intra lobular ducts contain no elastic tissue in their walls.

II. Diseased Parotid Gland.

The process of inflammation begins in the centre of the lobule, in connection with the small ducts which are primarily infected and gradually the process spreads to the periphery of the lobule.

As the parotitis advances an increasing number of lobules becomes affected and in advanced cases the whole of the gland becomes involved and may be more or less replaced by necrotic tissue.

III. Course of the Parotitis.

- A. The parotitis may resolve and the patient make a complete recovery from the primary disease.
- B. The parotitis may fail to resolve and death result.
 - I. from the parotitis, causing, as in case I general pyaemic abscess formation.
 - II. from the primary disease, the persistence *of* the parotitis being a bad prognostic sign.

The prognosis in all cases is guarded . No reliance however can be placed on the involvement or escape of the gland of the opposite side, As a guide to this matter, as many cases of double parotitis recover and frequently those in which only one gland is affected end fatally. Of the four cases specially referred to, three occurring in one ward, all ended fatally, the one in which the parotitis was most severe and in which both parotids were involved. recovered.

IV. Treatment.

A. Prophylactic.

Having regard to the duct spread theory of infection, the importance of careful and systematic attention to the mouth and especially to the state of the teeth, more particularly the upper molars to which the orifice of the duct is in such close proximity, becomes of first importance, as lessening considerably the chance of infection, in the event of the patient being reduced to the low state of vitality which predisposes to the onset of Parotitis.

The salivary secretion should be stimulated along the lines suggested by Dr. Soltan Fenwick, namely, to cause patients who are in a poor state of vitality and who are suffering from an excessively dry mouth to suck a small rubber teat. This becomes of primary importance in cases which are placed upon Rectal Alimentation.

B. After the onset of the parotitis treatment consists of:-

- I. Local applications of ichthyol 10 % in glycerine, or Belladonna and Glycerine. In mild cases this is sufficient to bring about resolution.

II. Incision of the gland.

This is necessary in cases which go on to abscess formation and should be carried out freely.

C. Vaccine Treatment.

An Antogenous Vaccine should be given. In most cases a dose the equivalent of 100 million staphylococci is safe to begin with to be repeated in 4 or 5 days, depending upon the constitutional reaction. The vaccine may best be administered in normal saline, by the mouth. This has so far not been extensively used but the result obtained in Case IV, would indicate its use in these cases.

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