A Thesis
on the Complications of
Scarlet Fever

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In the space of this Thesis I shall confine myself wholly to the clinical aspect of the complications as they appeared to me during the course of three epidemics of Scarlet Fever. No hard and fast rule can be drawn from a single epidemic and Scarlet Fever is notorious for the varying severity with which it occurs at different periods. "Of all the diseases of modern times," writes an eminent physician, "Scarlatina ranks 1st as regards the number and gravity of its complications & sequelae, so that nearly as many perish from these as from the direct effect of the poison." It is difficult to draw the line where the natural fever ends and where the complication begins, for the complication is often only a more exaggerated form of an ordinary symptom which may follow imperceptibly from it. For instance, the ulcerative, necrotic, or diphtheritic pharyngitis which occurs in a bad auricular case is a more intense degree of the ordinary inflammation of the fauces which occurs in Scarlet Fever or which by spreading along the nares sets up rhinitis, by extending
up the Eustachian tube & nasal duct causes Otitis Media & Conjunctivitis; or down the larynx, laryngitis, lung complications; down the esophagus, gastroduodenal catarrh; diarrhea & jaundice; or by setting up irritation of the nearest lymphatic glands submaxillary abscess & cellulitis; or by absorption of poisonous matter from the ulcerated surfaces, septicaemia, pyamnia.

Pharyngitis.

A convenient starting point for the consideration of scarlatinal complications will be found in the extensive & deep ulceration of the pharynx with membrane or pseudomembrane often seen in anginal cases. In considering this complication a difficulty occurs at the outset as to the nature of the pharyngitis. One school of physicians maintains the process to be one of gangrene, hence they call it "gangrenous pharyngitis"; I believe it to be of a specific pathognomonic nature. On the other hand, most English observers believe the pharyngitis to be "diphtheritic". This question will be again referred to. Whatever be the nature of the membranous exudation, the morbid anatom-
real results are similar. From the 2d to the 5th to 6th day of the disease greasy-yellow patches appear on one or both of the inflamed tonsils or uvula, at first as roundish specks, which coalesce until the whole tonsil is covered by a thick sloughy-looking membrane. This spreads backwards through the fauces to the pharynx and adjacent structures. Days quantities of thick viscous mucus are secreted by the tonsillar and pharyngeal glands, making the mouth very foul. In many cases an offensive smell is given off. The submaxillary glands enlarge and a phlegmonous cellulitis may follow. In many instances the membrane extends through the nostrils setting up a foul, fetid purulent discharge from the anterior nares. The so-called Stomatitis of the nose, which excoriates the lips, cheeks, nose and runs over them, on springing the parts, gangrenous shreds and casts of the mucus come away. The membrane persists for a time which is variable. It may come away in 24 hours or last for a week. The rhinorrhea lasts for some days after all trace of the membrane has vanished. When the membrane comes off an ulcer is left on the affected parts varying in extent and depth in proportion to the severity of the necrotic process. In some
milder cases profuse purulent rhinorrhoea occurs for several days without any visible tonsillar or pharyngeal ulceration. The ulceration may spread forwards over the palate, insides of cheeks & tongue in extensive shallow ulcers producing the variety known as scarlatinoid ulcers. In a few instances the palate is perforated forming a large gaping cavity, at the bottom of which bone may be felt with a probe; in other cases the palate is fissured, a V shaped piece being eaten away, extending from the angle formed by the gums transversely across towards the middle line. Perforation & fissure occurred in 3 of my cases. In another case an abscess formed on the right side of the soft palate. The false membrane extends up the nasal & pharyngeal ducts to the conjunctiva & there set up conjunctivitis. On only a few occasions did this membrane cover the conjunctivae of these were all fatal. Hemorrhage from the throat & nose may accompany the ulcerative process from erosion of an artery or vein. Cases have been recorded of perforation of the internal carotid as it passes upwards behind the tonsil but this must be rare. I have never seen severe epistaxis or bleeding except on one occasion. A girl aged 4, with deep & extensive ulceration of pharynx & submaxillary abscesses on both sides of the neck, at night
Suddenly got such copious hemorrhage from her mouth that she was dead in 5 minutes. Post mortem examination failed to find any source of the bleeding; the carotid and ascending pharyngeal arteries were intact; no vessel was found ulcerated, nor was there any evidence of extravasation into the tissues. The stomach and oesophagus contained blood, but only a little superficial ulcer of the stomach was noted. The right submaxillary abscess was, however, extended round the pharynx, with much cellular thickening. Where the great volume of blood came from remained a mystery. In the haemorrhagic purpurae death is there is constant oozing of blood or serous discharge from the nose for years. The lips become cracked and ulcerated and bleed on opening the mouth. Owing to the inflammation and impaired mobility of the palate, fluids often return through the nose. In some anginal cases the mucus membrane of the pharynx and posterior nares becomes gangrenous and large fested sloughs separate but the appearances are like those of the membranous variety above described. To this form the term “gangrenous” should be restricted, but is the normal condition in non-diphtheritic throats.

In cities & districts where diphtheria is prevalent as
P. Lewis Smith (Peppers' system of Practical Medicine Vol. II, p. 874) observes diphtheria is grafted onto the scarlet fever throat which forms a most suitable vector for its reception. This is the variety, scarlatinal diphtheria, most commonly seen. It is taken as the basis of my description. That it is a true diphtheria can hardly be doubted, for (1) the naked eye appearance of course is that of ordinary diphtheria. (2) The histological structure of the membrane is identical, consisting of a network of coagulated lymph encasing in its meshes, epithelial, granular debris, microbes and leaving a raw ulcerated surface when removed; (3) I have seen membrane formed concurrently with and subsequently to the throat lesion on any raw surface elsewhere on the patient's body. For instance, I have seen it covering the edges of a cervical abscess, on the ulceration that forms around the finger nails of children caused by picking their noses, on cuts and burns on the surfaces of wounds in traumatic scarlatina. Diphtheria, however, is modified when it complicates scarlatina. It seems as though the scarlatinal poison has the power to weaken or rot off of its surface the diphtheritic micrococci. In scarlatinal diphtheria the following
peculiarities are noticed: (1) The anaemia & prostration so common in non-scarlatalna diphtheria is less common in the scarlatalna variety; (2) the membrane rarely extends downwards to the larynx—it generally travels upwards to the nares; (3) albuminuria is generally absent; (4) Paralysis are the exception.

In 506 cases of scarlatalna, diphtheria was present in 23; only once were any paralytic symptoms observed. In the case of Ernest G., aged 7, during the 6th week the legs became weak; gait ataxic; so that the boy could not walk unaided; sensation unimpaired; hands flabby & weak; arms weak; fluids regurgitated through the nose; no paralysis of ocular muscles. Patient completely recovered in a month; (5) Nasal diphtheria is frequent & not so fatal as the non-scarlatalna form; (6) It is less contagious, when a case occurs it rarely spreads although the material is plentiful. But it must be remembered that weighty authorities are opposed to this view; they believe the membrane to be non-diphtheritic & a true necrosis. They point out that similar membraneous exudations
occur elsewhere, as on the surfaces of scalp, in dysenteric colitis, in membranous conjunctivitis (Dis. of Childr., Vol. II p. 214) says “one strong argument in its favour is that this form of scarlet fever does not prevent the patient being attacked by diphtheria soon after.” Thus he adds, “I have seen a boy of 2 years die of croup who had recovered from scarlet fever with severe gangrenous pharyngitis; become affected with diphtheria 4 weeks later.

Adenitis & Cellulitis

The submaxillary glands are enlarged during the 1st few days in almost every case. In mild cases they are hard and tender to the touch, but in severe cases considerably swollen, painful, hard or soft, the surrounding tissues are thickened & inflamed. The temperature rises often assumes an intermittent type with evening exacerbations to 101°-102° F. days. The glands either resolve in a few weeks or break down into pus & discharge externally. The pus is often extremely fetid as if the abscess had communicated with external air in the pharynx. If neglected the pus may burrow subcutaneously or in the sheath of the sternomastoid downwards to the clavicle.
A more active & extensive infiltration of the corium-glandular tissue produce the so-called "Brawny Neck". The sides of the neck become hard, doughy or greatly swollen. The skin has a dark red erythematous blush, is glazed & pits on deep pressure. All the tissues are involved from the middle line back wards to behind the ear. It begins on one side but before long both sides of the neck are implicated. The face has a peculiar look & the lower part of the face becomes enormously swollen giving the head a conical appearance & forcing it back on the shoulders. The swelling may become so tense that the larynx is pressed upon, causing considerable dysphonia, which may render tracheotomy necessary. Pressure on the jugular vein prevents the return of blood to the heart & produces cyanosis. Depletion may become absolutely stopped. If incisions are not made, the tissues break down, become gangrenous & form a huge slough. The subsequent ulcer has boggy, irregular, dark red or livid sides & takes a long time to heal. Thrombi of the jugular veins thrombosis, from this reason have occurred. This is a very fatal complication. In 4 consecutive cases, there were 3 deaths. I have never seen a case of cellulitis of
both sides of the neck recover. In 506 cases, cellulitis complicated with one of the mylo-hyoid region bilaterally became burning hard, red, and broken down into an abscess.

In 2 other cases, abscesses formed in the same place.

Cervical cellulitis is most common in young children from 3 to 6 years of age, with short but acute. It comes on about the beginning of 12th week. In one case, phlegmonous cellulitis attacked the right cheek and gave rise to a large diffuse abscess requiring free incisions. The child recovered.

A peculiar variety of cellulitis — *Haematoma Scrotalisum* — has been described in which an enormous swelling of a reddish, hard, colour extends over one or both sides of the neck and into the chest, nearly pure blood. It is the product of a hemorrhagic inflammation of the cellular tissues. Have never seen this complication nor heard of its occurrence in this country.

The most common complication is *Otitis Media* due to the extension of inflammation through the mastoidal cells from the pharynx. Otorrhea occurred in 36 cases out of 506. Both ears were affected in 24 cases, the right ear alone in 17 and the left in 15. This affection comes on
at various periods of the fever but usually during the 2nd week. It may however begin as early as the 3rd day. In about 1% of cases a delayed otitis, not due to direct extension from the pharynx (as that has long since healed) declares itself from the 6th to 18th week or later when the patient is apparently quite convalescent. This otitis is infective as we have undoubtedly traced some cases of Scarlet fever to this source alone in children who, in the course of Scarletina, have suffered from an auricular discharge those infected their brothers or sisters. In the majority of cases otitis media appears without pain or warning, partly because it occurs in young children who cannot express this pain and partly because it is masked by the other symptoms. The first indication of the complication is made known by finding the concha full of pus. In a few cases especially in older children and in the later forms of otitis media, there is ranulae, redness and swelling of the external ear and a rise of temperature. There is usually tenderness on pressure at the meatus. When the pharyngeal ulceration is severe, the discharge is thin, ichorous, ulcerating the adjacent skin, sometimes bloody, and often has a sc Kennedy.
Swell. Under treatment the discharge ceases in a week or two, except in sinusous children when it may last four months. The tympanum generally heals up with no permanent deafness remaining. A slight amount of deafness is often present, though rarely marked. It may be due to swelling and blockage of the mastoid orifice or its perforation of the tympanum. Still it is startling how little deafness there is in cases of double otitis media, considering how disorganized the middle ear and its appendages must be from suppuration. In 3 cases the inflammation spread to the mastoid cells, stripping off the periosteum giving rise to a post-aural abscess; in one case requiring the removal of a sequestrum 3 months after. In a few cases transient paralysis of the facial nerve was present. These were marked by much pain at the onset. After of otitis media may lead to permanent deafness, meningitis, abscess of brain, thrombosis of lateral sinus with subsequent meningitis and other fatal complications, but with proper treatment this is not a complication which need give much anxiety.

About the 5th or 6th week sometimes earlier or later a secondary inflammation of the discharge from the nose
occurs. Desquamation is nearly completed, the patient has begun to feel strong and everything seems favorable to a rapid convalescence when, without warning, rise of temperature or any symptom whatever, a purulent discharge oozes from the nostrils. The anterior nurses become excoriated & crusted with hardened pus, an erosive motionless eruption appears on the upper lip & around the nose & a secondary thorax may follow. This secondary Rhinitis is very infectious. It is mostly seen in weak, anemic and strumous children under 10. It seldom occurs in adults or adolescents. It is a troublesome complication & may necessitate treatment for weeks.

Ulcereive Stomatitis. Stomatitis is of frequent occurrence. On the 3rd or 4th day the gums may be seen covered with patches of thin, white, epithelial membrane, easily detached & leaving a shallow, red, bleeding ulcer. The membrane consists of an extensive desquamation of the pavement epithelium of the gums & granular matter, & swarms with bacilli & spicules. The edges & backs of tongue & inside of the cheeks & lips, & the angles of the mouth are next affected & ragged
Shallow ulcers form which readily bleed. The breath is foetid. The gums swell, become spongy & deep red & recede from the teeth, leaving the crusta petroica exposed & covered with whitish-yellow debris. In more severe cases, the teeth loosen, pus wells up around the alveolar walls on pressure; deep ulcers with spongy floors form on the adjacent sides of cheek & tongue; the breath is foetid; a thick viscous & abundant flow of saliva takes place; the submaxillary glands soften & swell. The tonsils and palate often become inflamed. The bowels are constipated & the temperature, rising to 101°-102°, assumes an intercurrent type. This variety is found mainly in weak, nervous constitutions, though I have seen it in strong & apparently healthy men. The teeth are nearly always decayed, & the sharp edges tend to produce, & prevent, the healing of the lingual ulcers.

Henry H., aged 17, on 13th day, right tonsil became enlarged & swollen; gums, lips & insides of cheeks ulcerated; tongue flabby & furred; breath foetid; obstinate constipation & pain in shoulders. Temperature 102.8-100°. During this sixth week the same state of affairs
recovered. Both tonsils met in the middle line and a faint blush was seen over the skin.

Tonsillitis. As Scarlatina has a peculiar affinity for the throat and tonsils, it is natural to expect it will leave these organs weak and susceptible to inflammation. As a matter of fact, secondary throat affections are common. Many cases of Chronic Tonsillitis date from a previous attack of Scarlet Fever and vice versa. Scarlet Fever almost invariably brings on acute relapses in cases of previous Chronic Tonsillitis. Several cases have had 2 or even 3 attacks during my attendance upon them, many of whom never had a sore throat before. Tonsillitis, single or double, occurs at variable periods from a fortnight to 6 or 7 weeks after the initial symptoms of fever. The attack is generally severe accompanied by headache, furrowed tongue, fever (temperature 100-103°) quick pulse, perhaps rigors, in a few cases with a rose blush or erythematous eruption on the chest and shoulders, pointing to an auto-inoculation of the Scarlatinal poison, or recurrence of the disease.
Spots of membrane may appear on the tonsils or meet in the middle line; the palate participates in the inflammation and there may be considerable dysphagia or even dysphonia. As before pointed out, constipation and ulcerative stomatitis often run concurrently with the throat affection.

In another set of cases, the tonsils has a rheumatic origin, the patient suffers from pains in various groups of muscles and joints.

**Gastro-intestinal lesions** are uncommon.

In malignant cases vomiting is nearly always present and persistent, but this is cerebral, due to poisoning of the nerve centres. In original cases the stomach is very irritable, rejecting all nourishment for a time. In nephritis the same irritability exists, and in uraemia emesis may become so serious that death quickly ensues. In these cases the vomited matter consists of a green slimy fluid becoming brownish and clear.

At the end of the 1st week swallowing the purulent secretions of the throat often sets up a troublesome diarrhoea with lymphangitis and abdominal tenderness.
The stools are offensive, yellow or greenish and sometimes contain blood.

Gangrenous ulceration of the gallbladder, from extension downwards of the ulcer processes in the pharynx, has been noticed. Swelling of Peyron patches, solitary mesenteric glands, stimulating the lesions of typhoid fever is fairly common. Jaundice is also an uncommon symptom with a favourable prognosis. It occurred in 5 of my cases and was always transient, the yellow staining was never deep, nor was bile ever absent from the stools, the liver was enlarged and tender in most cases. It usually is present towards the end of 1st week, in 2 cases appearing on the 4th day, but in other cases it is delayed the 3rd week. Congestion of the liver is present at the earlier date but later, it is rarely present.

Next to Otitis Media ranks Scarlet shooters Rheumatism as regards frequency. During the 1st few days purulent pains attack the joints, more especially the carpals and
phalanx or of one or both hands. The ankle, tarsal, metatarsal, the large articulations, the elbow, knees, shoulders, hips are attacked in the above order of frequency, together or in succession. The joints become swollen and flexion is painful. The intense pain caused by the least jar and movement seen in rheumatic fever is rarely or never seen in scarlatina. The pain and swelling may rapidly fly from one joint to another. Pain on flexion is usually the only symptom there being no swelling, especially when it occurs during the 1st 2nd or 3rd days. The muscles of the calf, back and neck are often at the same time painful. A moderate degree of any meer from stiffness and pain of the stern mastoid and trapezius was present in some cases. The arthritic variety is extremely amenable to the Salicylates, pain and stiffness rarely lasting longer than 24-48 hours after treatment.

The question arises. Is Scarlatinal Rheumatism a distinct affection as maintained by some writers who call it Arthritis, or is it true Rheumatic Fever
modified by scarlet fever just as diphtheria is modified when it complicates scarlet fever? It all depends upon what is understood by the term 'rheumatism'. Most writers believe them to be one and the same disease. Personally, I am inclined to believe that it is a peculiar affection of the joints themselves caused by the scarlatinal poison analogous to the rheumatic or arthritic complications of dengue and epidemic influenza, having no connection with rheumatic fever, except the common point that both affect the joints. Scarlatinal rheumatism has the following peculiarities: (1) It is very rapidly and permanently influenced by salicylates of soda. On stopping of this treatment, has not the tendency of ordinary rheumatism to relapse. This argument has been adopted by some writers as a proof that it is true scarlatinal rheumatism. But if one compares the action of the salicylates on the scarlatinal affection with their effect on rheumatism, the difference is very striking. Thus in Jaggie's medicine (vol ii p. 827) a table is given showing...
The results of the saline bath treatment in acute rheumatic fever at St. Thomas' Hospital during 1876-80. Of 383 cases, only 83 were benefited under 7 days; 70 of these 27 relapsed. 95% of my cases were completely cured in the same time; 90% in 18 hours.

(1) Synovial effusion into the joints is very slight, never purulent, & adema around the articulation is very rare.

(2) The heart's serous membranes are very rarely affected. In my experience not a single case of cardiac valvular disease or of pericarditis occurred after scarlatinal rheumatism.

(3) Hyperpyrexia very rarely occurs.

(4) Very few cases. Generally in young adults, with a previous history of rheumatic fever, suffer a fresh attack after scarlet fever & at least only present a subacute type with moderate swelling & pain of joints.

Thus Rose, aged 16 years, had rheumatic fever 2 years ago, had a couple of relapses since she suffered from pains in her joints whenever the weather became cold. During nearly whole period of convalescence from scarlet fever she suffered from subacute rheumatism, knees...
Ankle, wrist, hands swollen one after the other. Temperature 99°-100°; appetite good, no sweating, no vascular lesions. So my knowledge only one case of rheumatic fever a case of purpura rheumatica has occurred during scarlatinal convalescence.

Jane E., aged 15 years. Previous history obscure had mild acute measles. On Aug. 20th (24th day) vomiting, temperature rose to 102°. On 27th day vomiting had 2 days; eruptions of purpura spots on face, limbs; hemorrhagic exanthemation into right ocular conjunctiva; photophobia; headache; temperature 102°. Pulse 108. Weakness, pains in wrists, elbows, knees, ankles, slight swelling.

Urine Sp. gr. 1020 acid, dark, slight cloud of albumen.

On 30th day purpura fading; pains in arms; tongue dry and brown; temperature 101°. Pulse 114.

Double stools. 34-35th day swelling tendency of left hand and arm; temperature 100°. 39th day temperature 102°, pulse 140, no change. Pains lasted a few days longer. Treatment: Jodin Sulphate 50c, t. every four hours, alcalies afterwards. This
Complication occurred about 18 times in 560 cases.

Parotitis is rare. I have never seen the complication except in a case of pyaemia. I doubt if it ever follows directly from scarlatina, rheumatism or synovitis.

The respiratory organs are not often attacked in scarlatina fever, lesions being confined chiefly to the large organs. The pulmonary ulcer, on the other hand, is complication. Laryngitis may be due to different pathological causes. The commonest in scarlatina is a catarrhal inflammation of the laryngeal mucous membrane coming on at the end of the 1st week. This may occur in the mildest cases, but it is more frequent in anginal threats with much congestion or a fibrinous exudation indistinguishable from aphthous but probably due to more intense inflammation of the mucous membrane than that which sets up catarrh or again aphthous of the pharynx by extension may produce laryngitis, or it may be excited by external pressure from cellulitis of the neck. The
Symptoms vary much in degree from a mere hoarseness to extreme dyspnea. At the end of the 1st week or during the 2nd, the voice becomes husky, the respirations metastic and increased in frequency, the alae nasi expand and pressure over the larynx makes the patient moan. This condition with appropriate treatment usually passes off. In young children, however, in whom the larynx is small any catarrhal swelling is serious. In severe cases instead of improving, the symptoms grow worse. The voice becomes more husky or is lost. The respirations are prolonged and harsh, although not at 1st much increased in number. Later, they number 60-80 per minute, the alae nasi widely dilate with each inspiration, the sternum, mastoids and other muscles are brought into contact, the head is thrown back, the mouth opened, the child becomes cyanosed and restless, tossing about, clutching at its throat, the veins swell and a cold sweat breaks out upon the forehead. The lower ribs are sucked in during inspiration; in a
word the dysphonia becomes so urgent that, unless relief is speedily given by tracheotomy death is imminent. Unfortunately, most of these cases occur in children already exhausted by the severity of the fever with bad throats, rhinorrhoea, or with cellulitis, or lung complications, so that tracheotomy is useless, it may only hasten the fatal end. Tracheotomy is not justified in most cases of scarlatina laryngitis. In strong children after mild attacks, an operation may have effect but these are just the class of cases which never need operation.

Adema of the larynx occurred once with general droopy after nephritis.

Bronchitis is not an uncommon complication. It occurs in most of the cases of scarlatina gravior to a slight extent from extension downwards but whenever severe, it is always associated with Broncho-pneumonia. In fatal cases of S. angina are met with in which there is not the latter lesion. Bronchitis caught before infection frequently develops into Broncho-pneumonia during the 1st week of the case be any
Croupous Pneumonia, though not so frequent as catarrhal, is a fatal common complication. It is often seen after nephritis or such will be considered hereafter; but it is also fairly common without any kidney lesions. My cases fell into 2 well marked groups as regards the period of occurrence. In adolescents and adults, chiefly males, it came on during the 2nd to 3rd weeks, as desquamation was established. Though in nearly every case the fever was mild, desquamation slight. On the other hand when it attacked children it rarely occurred before the 4th week, in the majority during the 5th 6th week; curiously enough in 75-80% in girls. Nephritic pneumonia is seen about the 4th week or the most fatal variety. Out of 506 cases pneumonia complicated 10% and in more than half the cases, the right case

W.T. aged 17. Rash papular & dark, throat deeply congested, no ulceration; violet delirium for 3 days followed by much prostration, temperature 100-2°F. On 22nd day pain right side. Temperature 103.4°F Res.
section 32. Wooden dulness & fine crepitations over right base, during next 5 days temperature 104-5, vomiting, low muttering delirium, typhoid state, marked dulness over whole right lung, tubular breathing, rusty sputum. Very prostrate & died on 7th day.

T. Page, aged 27. S. Simplex. On 11th day temperature 104-5, 105-5°, precordial pain, double basal pneumonia, rusty sputa. Temperature ranged 104-105-5. On the 10th day of pneumonia there was still delirium, temperature 104, pulse 136, crepitations over right base, expectoration bloody, great prostration, involuntary motions. On 12th day pain in right leg much swelling, high circumference 4 inches larger than left, colour livid, purple mottlings, tenderness along saphena & femoral veins. Temperature 98-8, pulse 104. On 17th day there was considerable & dematous swelling of right side, the integument standing out in a fold reaching from flank to axilla. Sputum still viscid, dark & bloody. Temperature on the 52nd day of scarlet fever he developed phlebitis of the short saphena vein of left leg with painful swelling of ankle and calf.
Pleurisy. It is commonly stated that Scarlatina is characterised by a tendency to attack the serous membranes, var measles the mucous. This is correct as regards measles, but pleurisy & pericarditis inflammations of serous membranes are by no means infrequent. But as regards Scarlet fever, the statement is only partially true, for the mucous membranes are much more frequently the seat of complications, serous inflammations being nearly always secondary to either pneumonia or nephritis. Omit nephritis & its complications & the serous membranes are seldom attacked. True, synovitis is at times observed, but pleurisy & pericarditis are not common as I hope to show.

Pleurisy was detected twice in 506. In neither of my cases was it primary or purulent, the first being secondary to pneumonia, the second to pericarditis.

Equally rare as pleurisy after Scarlet fever is pericarditis, which occurred in only 3 cases out of 506.
(1st case) A. G., aged 12 years had mild anginal attack. On evening of 8th day had troublesome cough, loud friction sound heard over whole precordial region, best over sternum; after beat diffused & displaced downwards no thrill. Pulse 168 & weak. Temperature 100.8, two days later heart's impulse was best felt above level of nipple 1 to the right, friction faint, heart regular pulse 90 - made excellent recovery.

(2nd case) J. R., aged 13. Rheumatic fever 2 years ago followed by mitral regurgitation. Scarletina simplex. On 6th day loud friction sound, after beat 2 inches below 1/2 to left of nipple, diffused; orthopnea; temperature 100.5, pulse 144. On 10th day much dyspnea, cyanosis, developed pneumonia alveolar base, sputa rusty. 12th day, attack of cardiac asthma, respiration 54, temperature 99.5, pulse 128, heart excited. Had three more asthmatic attacks within 12 days & then rapidly got better.

(3rd case) A. C., aged 8 years on the 9th day evening temperature rose to 104, precordial pain was complained of & there was tenderness on percussion.
Soft friction sound heard over apex. The 1st Cardiac sound was obscured; heart quick, excited; apex beat not displaced. Next day temperature 99°. Friction sound almost inaudible & tenderness gone; pulse 112, regular; short dry cough. On 10th day heart sounds normal & all trace of pericarditis vanished.

The vagus nerve centres are considerably affected in Scarlet Fever. This is shown by the rapidity of the pulse, which varies from 120 to 160 in nearly every case. Nor does a rapid pulse prognosticate a fatal ending; it is found in the mildest as well as the severest type of disease.

The same remarks apply to Endocarditis which have been made with regard to pleurisy and pericarditis. In my experience endocardial complications are very rare indeed. Not a single case of valvular lesion has occurred to my knowledge & I find that Pagge (Pract ice of Medicine vol.1 p.218) corroborates my view as far as non-rheumatical cases are concerned. He writes: "This (i.e. rheumatism) may also be attended
with endocarditis it is very doubtful to be the starting point of chronic changes in the valves of the heart, but whether such changes ever arise when the joints have remained free, appears to me doubtful. 

True in a child aged 7, previously under treatment for choria, a mual systolic murmur developed a week after first seen with scarlet fever, in another girl, aged 17, with history of several attacks of choria, a loud systolic mual murmur was heard the 3rd day, the 2nd cardiac sound was inaudible; the apex beat thumping; if there was some albuminuria. In a third case Dr. B. aged 20 who had a malignant type of fever, on the 11th day a loud systolic murmur was heard at apex and over pulmonary area, it was propagated for a short distance to left of apex; at same time she complained of pains in limbs, temp. 102', pulse 120. Pains disappeared next day & murmur in 2 weeks. Was this not functional? Several of my patients had systolic mual murmurs previous to my first visit to them, due in
in every case to rheumatic fever. Of the 3 murmurs that
developed during my attendance upon the subjects
of them, I were in chronic subjects, 1 one was trans-
itory. The latter came concurrently with pain
in the limbs, but as these pains disappeared in 24
hours, it can hardly be attributed to rheumatism.
The patient was a delicate tuberculous girl with
Scars on her neck, was she had a severe type of
fever, the murmur was probably due to cardiac
dilatation and weakness.

The only other cardiac complications are ulcer-
ative endocarditis, of which some cases have been
recorded; the formation of anti-murin clots as
described by Dr. Smith; dilatation of fatty grafts,
granular degeneration of the cardiac muscle. Dilatation
of the heart is frequent in severe cases it is often seen
shortly before.

A Cardiovascular
Symptom to be remembered is syncope
It occurs in all ages but most commonly in young
boys & girls ranging in age from 12 to 17.
Sometimes it occurs about the 3rd week without
any warning, the patient becoming collapsed, ex- 
tremities cold & bluish, face cyanosed & eyes 
sunken, pulse quick & thready. Prompt meas- 
ures soon brought them round. Thus Herbert C. aged 
8. on 6th day got syncopeal attack, cold & clay- 
skin & pulse could not be felt for 2 hours, and 
Alice C. aged 14, became collapsed on 15th day 
(having 2 days had Scarlatina Simples) with green vom-
itting & epigastric tenderness, temperature 100°4, 
I have noticed these syncopeal attacks during 
the course of scarmitis also.
In severe cases when the palate, pharynx, tonsils, 
trusal passages are badly ulcerated constituting 
the variety known as Scarlatina ulcerosa, & 
especially when diphtheria is present, absorp-
tion of decomposing & poisonous substances from 
the extensive raw surfaces of the mucous 
membrane produces a state of Septicemia. 
A characteristic eruption heralds the Septic 
poisoning, appearing during the course of the
2nd week. The earliest period I have observed it was on the 5th day, but more usually it appears later, from the 8th to 12th day. Red erythematous blotches are first found over the external malleoli, on the sides of the soles of the feet, over the condyles of the humerus volarum. Next, papules appear on the forearms and tibial regions, mingled with dark reddish blotches. These, coalescing, form irregular patches of erythema over both extremities. In 24 hours these blotches have spread over the cheeks, forehead, over the chest and abdomen, being especially bright fuliginous over the iliac and hypogastric regions. The whole body ritter assumes a bright-brownish lobster-like hue, or simulates a rash indistinguishable from measles. The maculae may be raised or indistinct and disappear on pressure. If the patient has strength to overcome the blood poisoning, free desquamation follows. In a few instances I have seen the eruption passing into a condition identical with regena, assuming a vesicular nature. In another case, septicemia
manifested itself as erythema multiforme on the 7th day on the arms and legs; these spots fading in the centre, spreading at the periphery, formed large raised rings of erythema cenicata.

Other symptoms of septicaemia are an intermittent temperature, often high, but generally ranging from 100-102; a rapid pulse (120-180) at same time thready & compressible; quickened respiration; vomiting; prostration; diarrhoea. Inagination, broncho-pneumonia nearly always sets in & causes a fatal result.

In other cases of septicaemia no rash appears, but the child lingers for 3 or 4 weeks gradually becoming more emaciated until it is reduced to a mere skeleton with a thin semi-transparent, vein-intertwoven skin tightly stretched over the bony points. The head seems large in comparison to the small withered face & sunken eyes. Towards the end everything is vomiting to a colliquative diarrhoea sets in. Blood poisoning is chiefly seen in young children under 2.5.
Nervous Complications

The brain and nerve centres are much affected by the scarlet fever poison. Sleeplessness is common in adults it is no uncommon matter & finds patients who have not slept for the first 3 or 4 days & on whom hypodrines have little effect. Delirium of an active type is fairly frequent, the patient talks volubly, is constantly getting out of bed, but is rarely irritable or quarrelsome. It is usually present with a bright rash & high temperature in non-malignant cases. In malignant the rash is scanty or suppressed, the temperature not necessarily high. In this type delirium is soon succeeded by coma & collapse. Grinding of teeth & tremor of the hand are often present.

The paralyzing effect on the vagus centres has already been noticed. Convulsions are rarely observed, even at the onset in young children. In this point scarlatina contrasts strongly with
other febrile states. Acute convulsions will be considered later.

Meningitis is common. In only 5 cases was it noted. Young children suffer more than adults; out of 5, 4 were under 12 years of age. In young children it is difficult to clinically differentiate meningitis, as its symptoms are simulated or obscured by the severity of the fever.

The most characteristic, one of the most frequent, it at the same time, one of the most puzzling complications of scarlet fever is Nephritis. I shall not attempt to consider its etiology or pathology, but shall confine myself to its clinical aspects. One thing, however, is certain, cold or a chill is not the cause. Nearly all my cases occurred before the patient left bed, in a temperature maintained as nearly as possible at 60, with every precaution taken to prevent exposure. Nephritis shows itself in so many varieties that any classification is well nigh useless. As a rule it occurs during the 3rd week, usually from the 17th to the 24th days most often later, but it
may come on at any period during convalescence. In some cases, temporary albuminuria may be detected during the acute period of fever, as in any other fever with high fever temperature. This is merely symptomatic and differs from the latent inflammation. In scarlatinine nephrites, there are a few fairly marked variations observed which may be roughly stated as follows: - In a small number of cases, albumin appears in the urine about the 18th or 21st day without any symptoms whatever, lasts a few days, then passes off or the urine may be somewhat seamy and thus attract attention. This non-symptomatic variety, however, is sometimes seen later, in one case during the 65th week, in another another during the 41st. In another set of cases, nephritis is ushered in with fever (103° in 2 instances), restlessness, headache, with quick pulse—other words with the symptoms of an acute congestion of the kidneys; the urine is reddish or smoky in colour, contains blood in a variable quantity of albumen with blood casts and corpuscles. In
less than a week all traces of albumen will have disappeared from the urine.

Chas. J., aged 7 yrs. on 30th day complained of sore throat, temperature 100 to 103°, pulse 128, urine sesty, bloody, Sp. Gr. 1022, flocculent brown deposit. Two days later there was no trace of blood or albumen, urine, Sp. Gr. 1018, acid, temperature 98, pulse 84.

This transitory albuminuria is exceptional, but another insidious form lasting 2 weeks or longer more frequently occurs which, if not detected and treated, may lead to serious consequences. He premonitory symptoms draw attention to the kidney lesion; the child seems perfectly well, except that there may be a little fulness or puffiness about the eyelids or some loss of appetite or uneasiness, not noticeable to any inexperienced observer. The urine, however, may be scanty and albuminous, loaded with urates, or of a dull ochre colour, Sp. Gr. varying from 1020 to 1022. More often the urine is abundant Sp. Gr. 1005-1010, pale, clear, and deposits no sediment.
Only a slight cloud of albumen is obtained on heating which, on standing, will not measure more than 1/10 - 1/20 of a column in the test tube. Both temperature changes are unaffected. The 3 types above mentioned are moderately common, but the majority of cases of nephritis have characteristic symptoms. A dimness of both pupils, vomiting, irregularity of respiration, irritability of the stomach, furrowed tongue, restlessness, anorexia, head ache, may occur together or separately. The urine is scanty as a rule; high coloured & deeply coloured water. Not more than a few ounces may be passed during the first 24 hours. The amount of albumen varies considerably, but it is usually copious & on boiling the urine may almost solidify in the test tube. The urine soon becomes more abundant, clear & watery at the Sp. gr. falls. The usual tubular, epithelial & corpuscular elements are found microscopically. Blood is present at some period in about 1/2 the cases. It may be found on the 1st day, or the clouded urine may be one of the 1st intimation of the onset of nephritis, or it may not appear till some days later. The colour of the urine varies from a bright straw red to a
dark turbid liquid of different shades of red-brown, brown & ochre. The blood disappears in a week or two & all traces of albumen in 3 or 4 weeks. The temperature varies considerably; it may remain normal throughout, or it may rise to 100°-104° at the commencement & then fall to normal or, again, it may assume an intermittent form with evening exacerbations. The pulse is generally quickened, the heart beating from 100 & 120 times per minute; but it soon slows to 50-70 if the tension increases.

Numerous complications crop up during the course of kidney disease. The commonest is an effusion of serum into the connective tissues, first appearing about the eyelids, then about the ankles & dorsa of feet. The effusion may increase so as to constitute a general and pustula of the body with dropsy in the dependent, loosely-woven connective tissues as the scrotum & penis. The face becomes pale, pasty & swollen; the skin harsh & dry, fitting readily on pressure; the eyelids may swell so much
that they cannot be opened; the pelvis become large, tense and rounded. The scrotum is enormously distended, thin & translucent, causing the lips to be divericated, whilst the penis is bladder-like, shapeless & twisted. At any moment fluid may accumulate in the peritoneal cavity, pleura, pericardium, or the interlobular & interalveolar tissues of the lungs. This serous effusion comes on suddenly & rapidly increases, so that in 24 hours the adema is general & the condition dropsical. Dropsy was present in 2 out of 19 consecutive cases of nephritis.
A.P. aged 4 yrs. Mild Scarlet Fever.

On 15th day general anaesthesia, urine ¾ column of albumen, temperature 93°, pulse 90, full & thready.
On 21st day, ascites, urine 16 oz. dark, 1014, ¼ column of albumen.
On 26th day, adema of lungs, 3½ urine only.
Temperature 101°, pulse 92, State of Orthopnea.
On 40th day, adema less, none in scrotum; urine good quantity, bloody, temperature 99, pulse 96. Res-
On 68th day, urine still bright red, alkaline, much blood.

On 92nd day, blood more abundant in urine, deep red colour.

On 108th day, urine quite clear, no albumen.

A.S. aged 6 yrs. Scarletina angina with doubtful diarrhoea.

18th day, temperature 103°. Serotal dropsy, no adema of legs, urine good quantity, haer of blood, 1/3 column of albumen.

21st day, urine 3 oz. Albumen 1/2 column, acid, adema general, ascites; temperature 98.4°, pulse 72 irregular.

29 1/2 day, urine 50 oz. pale, 1008, 1/5 column of albumen, reddish sediment with casts, cellular dropsy nearly gone.

5-5 1/4 day, no albumen.

Pericarditis occurred twice twice on both occasions followed by pneumonia, but clear.

only one. In this last nephritis did not
supernova till 16th week of Scarletfever. Temperature was normal, urine good quantity, no column of albumin, no edema. On 14th day of the nephritis pleurisy developed.

On the heart, kidney disease has a weakening effect. The pulse at first is quick, full & compressible, but afterwards may become slow and tense, which is however, rather the exception than the rule. Syncopeal attacks & Collapse sometimes come on & may be fatal unless promptly treated. The heart is nearly always hypertrophied & dilated.

Pneumonia is fairly frequent & generally follows some other serious complication. Four out of seven cases were fatal. General dropsy was present in 5, pericarditis in 2, & 2 had convulsions. I have never seen a case of Adenoma of the glottis follow nephritis. I am aware of only one case of 2 cases. A case of nephritis may apparently be
progressing favourably the albumen almost or quite disappeared from the urine, when the kidney inflammation suddenly lightens up, a fresh relapse, the temperature rises the previously clear urine becomes bloody. This symptom is less serious than it looks, in the patient, I think unvariably improves in a few days. The following are a few examples of relapse:

Fred. J. aged 8 yrs. Scarletina Simplex
25th day Adema of eyelids, urine Sp. gr. 1022.

Moderate cloud of albumen.
37th day Urinal trace of albumen only detected.
38th day temperature rose 103, pulse 120, vomiting, pain in abdomen & neck. Urine: dark, large amount of blood, albumen 1/3 column. Sp. gr. 1014, dark amorphous sediment.

39th day, temperature normal.
41st day, trace of blood, a week later urine quite free from albumen.

Seth M. aged 7 year.
24th day. Adema of eyelids, vomiting, headache,
Temperature 100°; 1/10 column of albumen.
33rd day. No albumen.
36th day. Urine contained much blood; temperature rose to 100.4°.
45th day. Albumen disappeared.

Adam aged 7 years. Scarletina simplex.
20th day. Diffuse erythema appeared on legs and arms; edema of eyelids; urine, clear; sp. gr. 1025; plentiful, acid, 1/2 column albumen. Temperature 98.4°.
26th day. Urine clear; sp. gr. 1013; 1/2 column albumen.
28th - urine plentiful, dark red, sp. gr. 1020; 1/2 column of albumen; blood casts, cells & epithelium.
31st - urine dark; 1/2 column albumen.
42nd day - urine normal.

We now come to the most characteristic set of symptoms of nephritis, namely those due to uraemia. They are briefly: an eruption, vomiting, suppression of urine, convulsions & coma.

A bromic rash appeared in only 4% of cases. Its appearance varies, but it usually simulates
The eruption of measles, being formed over both body & limbs.

Frank S. aged 5 years

30th day- Without any premonitory symptoms, the abundant urine became dark red from blood.

33rd day- Only trace of blood lost.

39th - 49th day of nephritis, a bright rose measles looking rash came out over whole body, quickly becoming scarlatiniform. Temperature 99°.

Pulse 104.

5th 30th day. Urine normal.

Fanny P. aged 14 years


31st day. 4 short convulsions. Temperature 105°.

Pulse 104, retention of urine.

37th day. Macula raised rash appeared on body & limbs lasted for 36 hours. Urine plentiful.

Sp. gr. 1010. Acid, clear, red.

5th 2nd day. Blood still traceable.

Fred M.

Adam M., aged 7 years.
Nephritis was ushered in by a diffuse erythema of extremities & puffy eyelids. Relapse on the 10th day, when much blood was present in urine. Its colour was dark & porto-like, 1/6 column albumen.
The gravest form of uraemia is undoubted by suppression of urine. Fortunately complete suppression is rare, but partial suppression is common it gives rise to much anxiety. The kidneys fail entirely to excrete in spite of all treatment, or at best excrete only an ounce or two daily. Death takes place in about a week. At first in most cases the symptoms do not seem grave, the patient is fairly bright, but soon drowsiness comes on. The tongue & mouth become dry, muscular weakness is marked, twitching of the limbs or convulsions occur. The pupils contract & the patient dies either of convulsions or coma. Only 1 case of total anuria occurred in my cases.

Joseph L., aged 9 years.

On 17th day. Urine: - Scent: dark, bloody.
3/4 column of albumen.
22nd day. Temperature 100.6°. Pulse 120.
Tongue dry; pains in abdomen, legs, arms, & back; diarrhea; vomiting of bright green mucous fluid.

24th day. Amenorrhea; constant green vomiting. Tongue dry & brown. Delirious. Temperature 101°, pulse 120, weak & irregular. Takes fluid plentifully; sweats freely. No diarrhea but motions are watery.

25th - no change.

26th - Heavy & drowsy. Constant green vomiting with effort immediately after injection. Skin acts well after hot packs. Temperature 100°.

27th day. Vomit has become dark brown & bloody. Tongue dry & brown; temperature 100°; pulse 100. Very weak. Knew & urine screted for last 4 days. Death.

Treatment: Metaphasic, pilocarpin, cotton oil &c. In this case no convulsion ushered in the end, nor did coma. Asthma was the cause of death.

Another fatal uraemic symptom is vomiting. The stomach rejects everything fluid if swallowed, immediately regurgitates, mixed with greenish mucous, or a few
ounces of a brownish green glairy fluid is ejected at intervals without effort. No treatment has any effect and the patient soon dies of exhaustion. It is a very fatal complication as diarrhea is generally present at the time and water are not retained. This kind of vomiting which is purely central due to poisoning of the nerve centres, must be distinguished from that due to irritability of the stomach present in many cases of nephritis especially at the onset. During the first few days the stomach may reject almost everything then quickly become tolerant of food. The vomit is seldom green, but consists of food with a brownish glairy fluid and is not serious. The commonest and most characteristic symptom of uremia is convulsions, which may occur at any period of nephritis. Often the onset of kidney trouble is heralded by sudden convulsions or they may constitute the beginning of a relapse, when the hitherto to non-bloody urine becomes loaded with blood. The first symptom may be headache, vertigo or vomiting, or a peculiar strange-ness or curious start in the eyes; the child is stupid, drowsy; if spoken to, takes no
notice or trembles; or on the contrary may be restless. Then the orbicular eye muscles twitch and rapidly other groups of muscles are involved chiefly those of the extremities. At the commencement of the attack, the tonic symptoms are usually limited to one side of the body, to one or both extremities, but before long the whole body participates. The head is retracted, the eyes are widely opened, turned upwards, fixed or staring; the pupils are widely dilated (in one case considerably contracted) they may or may not react to light; the tongue is protruded from the mouth; it is liable to be bitten; the respiration is stertorous. All consciousness is lost. The attack lasts from a few minutes to several hours. Generally there is only one fit, but sometimes the attacks are multiple.

For example:

Emily S aged 13

July 23rd One fit lasting 3 minutes
" 27th " 3 fits in one hour
" 29th " 30th Convulsions very frequent
31 distinct fits occurred in 19 hours
Urine was bloodstained throughout
Hanny G. aged 13 years.
January 2nd 4 short seizures each lasting 4½ ½ minutes.
Sydney A. aged 8 years.
Feb. 17 & 18th 6 attacks.

During the interval between the fits consciousness is restored and the child takes an interest in its surroundings. In the case of Emily G. above mentioned, the girl knew when the seizures were coming on; she exhibited extreme terror, cried out, fought desperately against the spasmodic movements of her hands, reminding one of tetanus. The temperature rises during the attack & the pulse quickens. Collapse may supervene with a quickening pulse, cold limbs, & much exhaustion; but in most of my cases which were treated under chloroform followed by chloral & the bromides, when the anaesthetic was pushed the child fell asleep & remained in a quiet sleep for some hours. A curious feature of tonic convulsions is the occurrence of cyanosis; it was seen in 3 or 4 of my cases but never lasted for any lengthened period.
Convulsions came on in 14 out of 19 consecutive cases of nephritis, in 3 of which the result was fatal, though never directly, one dying of a later attack of croupous pneumonia and one of amputation, whilst the 3rd had renal vomiting and diarrhea for 6 days which reduced the child to a state of collapse, just before death convulsions set in, most marked on the right side.

Daisy C. aged 7 years. Mild Scarlet斑
17th day. Vomiting of milk curds and blood
-sheared mucous. Urine. 10 c.c.
light colour. sp. gr. 1.023, 1/2 column albumen, temperature 98°, pulse 104.
20th day. Frequent greenish vomiting.
urine, very scanty, 3/11 daily.
Morning temperature 98.4, evening temperature 100-101. Same day pulse 120.
124 weak—green diarrhea.
23rd day. Collapse.
24th day. Convulsions—temperature 99°.
Heart 200. Death.

Emily J. aged 13 years. Scarletina Agizia.
On 24th day green vomiting. Urine, scanty
1/2 column albumen, temperature 98.4, pulse 100.
31st b 34th day - vomited now and then urine scanty 1/6 1/4 column albumen
35th - headache, eyes puffy, urine: 3xxx
1/5 column albumen. Convulsions for minutes beginning in right arm. In the evening had persistent green vomiting. Temperature 98.4. Blood appeared in urine.
36th & 37th day - no convulsions, urine pleasant bloody. Hb 90. B. 1006 1/2 1/3 column albumen.
38th & 39th day, 31 convulsions - no vomiting.
40th day. Restless, urine: 3xxx, dark bloody, albumen opaque, temperature 101.4° pulse 134.
43rd day - developed left basal pneumonia.
48th day. Death.

Skin Diseases. Numerous skin diseases appear during the course of con
Balescence from scarlet fever but perhaps with the exception of herpes, regula, purpurca, this fever does not seem to predispose to any cutaneous
Urticaria is common in spring and
Autumn & may be observed running
A course concurrently with the scarlatinal rash or immediately after it fades. The raised wheals standing out on the rose ground of scarlatina rash. As urticaria is an erythematosus disease it would naturally be found concomitantly with an exanthematosus disease like scarlet fever.

Eczema is very common, affecting the scalp, face, ears & nose. Acute general eczema was noticed several times. Scarletina undoubtedly brings out this dermatitis in those predisposed to it as most cases occur during the 3rd week.

Herpetic eruptions of the lips, cheeks, nostrils & chin during the onset of the fever appear in about 5% or 10% of all cases. Sometimes the herpes is extensive & frequently bilateral.

Eosinophy is fairly common amongst strumous ill-nourished children.

Lichenous eruptions are less common.

In a boy of 4 a papular rash came out on extensor aspects of hips, buttocks, face
Chin, arms & back of fingers. Vaccinations quickly took place in them giving rise to dark purple, large, irregular papules. Pustules were also observed on legs & feet, temperature 98.40°, pulse 84. About 14th day a scalpel formed on tip of pimple, came off with the cut adjacent adherent blood clot, leaving a deep pit. Vesicles was observed about 12 times, the distribution not always being characteristic.

Purpuric eruptions, ecchymoses & pustules constantly occur in malignant cases, & in weak anaemic children, are often observed on the limbs particularly the legs during convalescence. In a youth of 17 on getting up large purplish patches appeared on the insteps, round the ankles & extended up both legs to the knees, accompanied by pain & swelling. A subcutaneous purple staining was present in another man, who, at the same time had phlebitis of the saphena vein. In the latter case the appearance of the leg resembled gangrene.
In severe cases, the nails become deeply furrowed, and in a few cases were shed. Abscesses are liable to occur in the subcutaneous and deeper tissues of the body. I have seen them form from no apparent cause, in the buttock, over sacrum, in the axilla, about the elbow and shoulder joints, and other parts of the body. In Else W., a large diffuse abscess was opened in the right calf, there was thickening and tenderness along the Saphena Vena.

Other symptoms may complicate Scarlatina. In most cases the symptoms of the complicating fever are intensified.

In Röthela, the tonsils often ulcerate and there is a copious purulent rhinorrhea. The lungs are attacked, broncho-pneumonia being common. Large sites common and fatal. The rash was in several cases dark, bright, temperature high, pulse rapid and the cervical, axillary and inguinal glands invariably enlarged. I have seen 42 cases. Ten had broncho-pneumonia.
Six laryngitis, 9 fatal double otitis, 7 ulcerative stomatitis, 2 melaena, 1 pleurisy, 4 died. All the children attacked were under seven except two, the average age being 2-4. Only a few cases of measles occurred with the death.

Conclusion.
I have tried in this thesis to show that too much stress has been laid on the occurrence of endocardial lesions, inflammations of the serous membranes, too little on those of the mucous membranes; that scarlacial rheumatism is peculiar to this fever, not to be confounded with acute rheumatism; that synovial effusions are rare; that diphtheritic pharyngitis is common; that most cases of the so-called "necrotic" pharyngitis are in reality, diphtheritic; that the latter disease is modified by scarlet fever to a large extent; that septicemia is accompanied, in nearly every instance, by a pathognomonic eruption, which I have not seen.
described elsewhere.

I have drawn attention to the frequent occurrence of secondary lesions of the nose, ears + throat, which are very frequent. In dealing with the complicated subject of *Nephritis*, I have tried to classify this protein disease as it appeared at the bedside, for fortunately, a pathological classification cannot well be made, unless much is the great majority of cases recover, the kidney lesion in its early stages cannot be investigated.
I hereby certify that this thesis has been composed by myself and is solely and entirely my own.

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