not occur. It does so occasionally, however. Lewis Smith reports cases, with necroscopic examination, in which diphtheritic membranes extended along the trachea into the bronchial tubes. In the most severe cases, however, the deposit does invade the posterior nares, and the ordinary slight catarrhal discharge is supplanted by an offensive sanio-serous or sanio-purulent discharge from the nostrils, which causes excoriation of the upper lip. At the same time the nares become obstructed, and the little patient is driven to breathe almost entirely through the mouth. In many cases of extreme throat involvement the strength rapidly fails, pallor replaces the prematurely receding eruption, except at certain spots where this may persist as circumscribed patches of dusky redness; the temperature remains stationary or falls, the pulse becomes more and more feeble and rapid, and death ensues within a few hours, as if from blood-poisoning. Death either follows a slow spread of gangrene to the soft palate and tongue behind the sinus pyriformis, and to the walls of the throat, or occurs through diphtheritic inflammation of the lymph nodes and connective tissue of the neck, or through œdema of the glottis. At other times the clinical appearances during the first week may not be alarming, danger becoming imminent about the beginning of the second week. The fever may remain elevated, the eruption brilliant and intense, until a short while before death. In the rapidly fatal cases the throat and neck may not appear very much swollen. At other times the neck and submaxillary region are greatly swollen, principally from the inflammation of the glands and periglandular tissue. These parts become hard and brawny, and from the pressure upon the great veins acquire a livid appearance, which may also be communicated to the face and head. The tonsils and soft palate may be swollen until the throat will appear quite closed. The mucous membrane will be deeply congested, and covered here and there with diphtheritic exudation and with ulcers caused by the separation of sloughs. The posterior wall of the pharynx may be bathed in mucopus. Retropharyngeal abscess is sometimes formed, and may precede the fatal termination. Dyspnœa may result from swelling of the fauces caused by inflammatory exudation into the parts, from œdema glottidis, or from extension of diphtheria to the larynx and trachea, or it may be a result of the imperfect oxidation of the blood. The term "diphtheritic" is here used in a clinical sense, to designate a condition of coagulation necrosis in the tissues involved, and has no reference to a pathogenetic relationship with true diphtheria. This necrosis occurs simply as a result of the intensity of the accompanying inflammatory changes. There is no evidence that true diphtheria may not coexist with scarlatina, but that the commonly observed membranous pharyngitis of scarlatina represents this combination is most improbable. Many of these membranes are undoubtedly due to the action of the streptococcus and staphylococcus infections. Their differentiation from true diphtheria is now made easy by the bacteriological examination in reference to the presence or absence of the Klebs-Loeffler bacillus. J. Lewis Smith has seen four instances in which the diphtheria became dissociated from the scarlatina, and attacked other persons as idiopathic diphtheria. Such observations are exceedingly uncommon. The scarlatinal diphtheritic membrane is indeed essentially identical with that of idiopathic diphtheria in structure. The differences are etiological. The diphtheritic poison and the scarlatinal poison, differing in their specific natures, possess in common the power to excite such violent inflammatory changes in the tissues that a coagulation necrosis results. The diphtheritic membrane of scarlatina, then, is purely scarlatinal in its origin. This view has received solid indorsement. It has been adopted by Henoch. Heubner considers scarlatinal diphtheria to differ from primary diphtheria both clinically and histologically. It begins with a simple catarrhal affection, and, following his observations, changes from catarrhal to diphtheritic inflammation on the fourth day. Koven also thinks that the throat affections of more severe grade are characterized by necrosis from direct intensity of the scarlatinal process, and are not of a truly diphtheritic nature. He observes that while two acute diseases rarely coexist, of 426 cases of scarlatina 125 had necrosis faucium, although at the period of observation there was not a single case of primary diphtheria in Christiania. He further declares that diphtheritic paralysis never occurs after scarlatina, and that while true diphtheria shows the membrane at once, the scarlatinal slough usually appears after several days of increasing angina, and does not extend to the larynx. Henoch has never seen a single case of accommodation paralysis of the eye or of the soft palate, nor of the neck, nor of the extremities, after scarlatinal diphtheria. The inflammation may, often does, extend along the Eustachian tube to the middle ear, and excites changes that give scarlet fever one of its principal terrors, resulting often in more or less complete permanent deafness. These changes will be considered with the complications and sequelæ of scarlatina. In a number of these cases cervical adenitis and periadenitis occur, and prolong the fever beyond the eruptive stage indefinitely, frequently resulting in suppuration. Occasionally the pus burrows deeply among the tissues of the neck, and extensive gangrene may follow. Williams has reported a case of extensive sloughing in the left anterior triangle of the neck, with exposure of vessels, followed by recovery. Other similar cases have been recorded. In most cases in which death does not speedily occur after suppuration and evacuation of pus recovery will take place, but the patient may ultimately succumb under blood-poisoning and protracted fever. Occasionally, also, parenchymatous tonsillitis may cause rapid and enormous enlargement of the tonsil, with the formation of pus, a condition of extreme gravity, especially if associated with retropharyngeal abscess and œdema of the glottis. In favorable cases the sloughs in the fauces will cease to extend, the œdema and dusky redness will slowly subside, and the diphtheritic ulcers begin to granulate.

In many cases the faucial symptoms here described do not appear, only because life is early destroyed by the intensity of the action of the specific poison upon the blood and tissues. In such malignant cases the patients often die with the rapidity of those who succumb to narcotic poisoning; or a series of convulsions inaugurate the disease and terminate life within an hour or two. This has been called the *atactic* form of scarlet fever. At other times brief initiatory symptoms have been followed by intense fever (106°–109° F.), with uncontrollable vomiting, diarrhœa, delirium, rapidly deepening coma, and death, before the appearance of the eruption. Or, again, the disease may begin in the ordinary manner, not suggestive of a severe course, and alarming symptoms may not develop until after several days; or it may be intense from the beginning, with severe and repeated convulsions, vomiting, profound nervous depression, and the appearance of the eruption at the usual time, with steadily increasing gravity of all the symptoms, until, after a few days, death results from convulsions or coma. Finally, the malignant symptoms will appear suddenly in the midst of what has seemed a mild attack of scarlatina. An unusually protracted period of invasion is sometimes the forerunner of malignant scarlet fever, and should always be regarded with apprehension. Cases may at times exhibit at the beginning alarming symptoms. A decided apathy, in which no notice of what is passing is taken, with great apparent depression and even delirium, excites the apprehension of the attendants, yet the pulse and temperature will not show marked variation from the normal. After the second or third day such cases will very often pursue a mild course. A high temperature and very quick pulse may even be added to these symptoms and justly excite alarm, and yet the case may assume a favorable character after the development of the eruption. In such cases as these the probability of the issue in life or death seems to vary from hour to hour. All the symptoms show intensity. The fever, accompanied by more or less severe initiatory symptoms, rapidly increases, the eruption is copious and

deeply colored, the pulse beats 130, 140, 160 times, or oftener, to the minute, the respiration is proportionally accelerated, the throat duly shows more or less extensively the peculiar alterations. This course may be held throughout the first week, and even longer, without mitigation, the result remaining doubtful all the while.

In malignant scarlet fever the usual course is one of intensified general symptoms. Those of the invasion period are indicative of grave perturbation of the economy. By the time the eruption appears it is already evident that the patient is dangerously ill. He is apathetic, or perhaps extremely restless, remaining in one position not an instant. The skin is hot, dry, and pungent, the temperature very high, the features are swollen, the conjunctivæ injected, the fauces reddened and dry, the thirst is intense, but water and all ingesta are often vomited as soon as swallowed; the urine is scanty, or even suppressed, from acute renal inflammation. Diarrhœa may be present. The nervous phenomena become intensified. The eruption now appears, and may at first develop regularly, but after a while will become duskier and will not completely fade on pressure. The color tardily returns to the part whence it has been pressed. Coma or convulsions may now carry off the patient in full eruption. Often, however, the eruption will recede from certain parts in whole or partially, or it may become paler universally; or in place of the regular eruption hemorrhagic exudation will appear. Ecchymoses, from the size of a pinhead to that of the palm, or larger, will replace the usual eruption, which will in great measure disappear. Then livid spots, not fading on pressure, are found, generally upon the flanks and back, but may appear anywhere. According to Mayr, the hemorrhagic eruption may appear over a large part of the surface in children, but in adults is mostly confined to the neck, upper part of the chest, the back, and about the joints of the upper and lower extremities. This hemorrhagic variety is the most formidable form of scarlet fever, and is probably always fatal. Hemorrhages from mucous surfaces are exceedingly uncommon. Mayr has described a scarlatinal dissolution of the blood, in the gravest form of which death occurs in from twelve hours to five days. "Extreme muscular depression, with slight headache and a remarkably rapid pulse, are present from the very commencement. . . . The patient lies on his back with his eyes half open, but in an unconscious state. . . . Quivering movements of the muscles of the face and of the fingers are also commonly observed in these cases, and in children general convulsions often occur. The pupils are moderately dilated; the lips and tongue are dry, the latter being usually of a bright-red color. As the disease goes on, mucous râles are heard in the large bronchial tubes; the abdomen becomes distended, but there is seldom any enlargement of the spleen; the urine becomes scanty and of a dark-red color; the pulse continually increases in frequency, reaching as many as 200 beats a minute; the features become shrunken and the extremities cold." Death speedily follows. This form resembles the so-called typhoid scarlatina, in which drowsiness, stupor, delirium, and subsultus precede the fatal issue. The life-destroying symptoms are often connected with impairment of the heart's action, attributable to crippling of the vagus, when death occurs from heart paralysis, without widespread molecular disintegration. This failure is shown by increasing weakness, frequency and inequality of the pulse, with quickened and shallow breathing, and coldness of the hands and feet. Allbutt has classified the modes of death in scarlet fever as follows: (1) hyperpyrexia (this Jenner denies positively); (2) specific blood-poisoning; (3) special malignity of the case; (4) asthenia. In the rather uncommon event of recovery from any of the most severe forms of scarlet fever, the progress is slow, the essential symptoms, complications, and sequelæ proving all very obstinate. In those cases in which extensive diphtheritic exudation precedes a rapidly fatal course, the eruption undergoes many modifications, the integument remaining pale except for some few splotches about the joints, at other times showing only a few dark-red patches irregularly distributed, and again entirely disappearing before death. At other times the eruption persists in full efflorescence.

Changes in the Blood.—The number of the red corpuscles in general is but slightly altered, more, however, in scarlet fever than in measles. The same may be said concerning the character of the red cells and the amount of hæmoglobin; in scarlet fever poikilocytes and normoblasts are occasionally found. The state of the leucocytes is much more characteristic. In measles there is either a normal number or a diminution of leucocytes; in scarlet fever there is hyperleucocytosis. In the former, as the temperature falls the leucocytes gradually increase; in the latter the temperature and leucocyte curve run parallel. In both diseases, but especially in measles, is the number of polynuclear neutrophilic cells diminished, while lymphocytes, both large and small, are relatively increased. The eosinophilic cells are diminished in number in measles, reaching their normal state long after recovery; in scarlet fever they are constantly increased.

Complications.—*Nephritis.*—Derangements of the kidneys are the most important complications of scarlet fever. Indeed, a number of recent writers assert that these organs are always affected in this disorder. Among these may be mentioned Frerichs, Reinhardt, Begbie, Newbigging, Holder, Böning, and Stevenson Thompson. Steiner states that evidences of kidney disorder are always present in those who die of scarlet fever. Thomas' clinical observations do not bear out this statement, and Friedländer, who examined the bodies of two hundred and twenty-nine persons dead of scarlatina, found kidney disorder in less than one-half. Though renal inflammation is not shown as yet to be a constant accompaniment of scarlet fever, it occurs much more often than is commonly supposed. Renal catarrh, which Eisenschitz declares to be as much a feature of scarlet fever as bronchial catarrh is of measles, is indeed an extremely common complication. It usually escapes detection from the general neglect duly to examine the urine. Thomas, in denying that this catarrh is at all constant, shows that it also occurs in measles, croupous pneumonia, etc., and is often only an expression of the febrile condition. Yet the catarrh is relatively so common in scarlatina that he cannot avoid concluding that the specific influence of the disease is often concerned in its production. In many cases, from the very beginning, cylinder-like masses of renal epithelium may be detected. In milder cases the urine will contain mucous casts with increased quantity of mucus, but no albumin. In more severe cases the urinary sediment will contain hyaline masses with epithelium and epithelial *débris*, and red and white blood corpuscles. Slight albuminuria will also be present. This catarrh is usually insignificant, and but rarely serves as the starting-point for the graver and characteristic forms of nephritis scarlatinosa, though doubtless many milder forms of nephritis and dropsy originate in it. Thomas concluded, however, that the cases of scarlatinal nephritis not developing from preceding catarrh, but arising suddenly, usually end fatally. Scarlatinal nephritis varies greatly in the relative frequency of its occurrence, involving from five to seventy per cent. of cases in different epidemics. In the Children's Hospital, Hillier noted its occurrence in about half of the cases. Dickinson[16] considered this rather below than above the average. Fleischmann[17] noted 95 cases of Bright's disease in 472 observations. During 1861 every third child with scarlatina had dropsy, while in 1862 it affected only one case in ten. Thomas asserts that renal alterations develop in about one-half of all cases of scarlet fever. It has been shown that there are those who assert that the renal alterations are constant. On the other hand, Jaccoud[18] declares that for fifteen years he has never had a case of nephritis among his scarlet-fever patients, a result that he attributes to his treatment. Albuminuria may appear at any time during the attack of scarlatina, though its most common occurrence is during the second and third weeks. Dropsy should not be taken as marking the beginning of the nephritis, the signs of which may

be present in the urine sometimes for days before this occurs. In Fleischmann's cases dropsy occurred 9 times during the first week, 30 times during the second week, 23 times during the third week, 20 times during the fourth week, and 5 times after the fourth week. Of 60 cases at the Children's Hospital, 42 began between the end of the first week and the end of the fourth week; 5 became dropsical during the first week.

Nephritis during the first week of scarlatina often escapes detection from the blending of its symptoms with those of the essential disease, and from the attendants' neglect to examine the urine. Dropsy will, of course, attract attention, but this does not often occur so early, and may be confounded with the œdema from the exanthem. Rarely, the fatal issue of what was, apparently, malignant scarlet fever, may really have resulted from uræmic poisoning due to a fulminating nephritis. The symptoms may be identical. Fever, vomiting, headache, delirium, amblyopia, coma, convulsions, may have been present. The convulsions are often very irregular. They may be general, partial, or unilateral, tonic or clonic. The patient may have them in rapid succession, or may pass into a *status epilepticus* from which death alone will release him. The urine will be completely or partially suppressed. If secreted it will be of high specific gravity (1.020 to 1.040), dark and smoky in appearance, loaded with albumin, and forming an abundant sediment of hyaline, granular, epithelial, and blood tube casts, with renal epithelium and white and red blood corpuscles in greater or less quantity. If the kidneys become implicated toward the end of the first week, the symptoms may delay the course of what may otherwise appear to be an ordinary case of scarlet fever. Microscopical research will often betray the onset of the changes in advance of chemical analysis; casts of the renal tubules will be observed, with epithelial deposits and detritus, before albuminuria is established. This will shortly appear, and in severe cases the nephritic symptoms will obscure those of the scarlatina. There will be no constant relation between the amount of albumin, the tube casts, and the general detritus, one variety of sedimentary matter being at one time copious, at another scanty. At this time vomiting may appear with returning headache, the appetite will again fail, and pain in the loins may become annoying; the patient may again become dejected and feeble, and his fever may cease to diminish—may even exceed its original intensity. At other times no apparent influence will be exerted upon the scarlatina, which will follow its usually mild course until dropsy and albuminuria reveal the state of the kidneys. When the renal disorder develops after defervescence, during the second, third, or fourth week, or later, the same series of symptoms may be observed, their severity being in direct ratio with the earliness of their occurrence. Cases developing after the fourth week may be expected to pursue a favorable course. Although it has been asserted that the renal disorder may arise several months after a scarlatinal attack, a patient will almost certainly escape it if he pass the sixth week in safety. The symptoms in cases arising during these weeks are not always gradually developed, and some of the most disastrous results of the disease may be encountered, during the second, third, or fourth week, in children apparently convalescing from scarlatina, and often in full desquamation, who, after indisposition for a few hours, with nausea, headache, confusion of ideas or stupor, with return of fever, rapidly pass into coma or convulsions, ending after a short interval in death, before dropsy has developed, but after partial or complete suppression of urine. Scarlatinal nephritis has usually a mild and favorable course. Dropsy is usually the first symptom observed, first appearing in the face and sometimes remaining confined to this locality; at other times becoming general speedily, and giving an appearance of plumpness, but with a wax-like translucency of skin. The face, upper and lower extremities, body wall, and prepuce may thus become dropsical. The serous cavities are also implicated, and more or less effusion into the pericardial, pleural, peritoneal, scrotal, and intracranial cavities occurs. Œdema of the lungs and of the glottis may imperil life. Desquamation is often completely arrested upon the supervention of dropsy. The temperature is more commonly but little above the normal (38.3° to 39° C.—101° to 103° F.). The pulse, sometimes feeble and accelerated, will often become remarkably slow and intermittent, and so remain throughout the attack. The child will grow dull and listless, and extremely feeble. Pain in the belly and in the back may at times prove very distressing, or, again, it may be absent. The tongue, having lost the strawberry aspect of the eruptive stage, will become pale, flabby, and coated. The appetite will fail, and the bowels become sluggish. The urine will rapidly diminish in quantity and may deposit urates abundantly, or may present a smoky and oily appearance, due to the abundant presence of epithelial cells, white and red blood corpuscles, and tube casts. The total amount may now be reduced to a few ounces. The blood corpuscles often form a thick red layer at the bottom of the test tube. This free admixture of blood may amount to pronounced hæmaturia, is generally post-scarlatinal, and, according to Schütz, occurs most frequently during the third or fourth week. Of itself it adds but little to the gravity of the case. The patient often feels fairly well, and may eat and sleep with comfort. While the pallor and œdema may be very decided, the temperature and pulse may vary but little from the normal, or may show the variations of ordinary nephritis. With the gradual improvement of the general symptoms the hæmaturia disappears. Heubner has reported a case of nephritis after scarlatina in which hæmoglobinuria was present. The urine was brownish-black; no blood corpuscles were found. Death resulted from asthenia on the fifth day after both albumin and hæmoglobin had disappeared from the urine.

The amount of albumin in the urine in scarlatinal nephritis is usually very great. The urinary sediment is abundant, and is largely composed of tube casts, the hyaline character predominating; finely and coarsely granular, epithelial, and blood casts are, however, numerous. Later, coarse fatty granules stud the casts plentifully. These casts are often almost diffluent, and differ strikingly from the firm and sharply outlined ones of more chronic nephritis. Crystalline deposits are scanty, and are mostly of uric acid and urates; on the other hand, the amorphous urates are often very abundant. The degree of albuminuria present is of less importance than the total quantity of urine secreted, rapid and pronounced diminution of this indicating the accumulation of nitrogenous waste in the blood, and consequently the danger of uræmia. According to Glax, a lessening of the proportion of urine secreted to the fluid ingested (2:3) not infrequently foreshadows the approach of uræmic symptoms, even though the urine contain no albumin. Whether the temperature remain normal throughout the attack, or whether, after an initial chill, it become elevated, and all the symptoms of acute nephritis develop, complete recovery may reasonably be expected if the patient pass safely through the earlier phases of the disorder. But although nephritis may be mild—the dropsy lasting only a few days, and, perhaps, being limited to slight puffiness about the eyes—the disorder does not usually entirely subside in less than a month. It may endure as long as three, four, or even five months; and there is good reason to believe that chronic nephritis in young people may, in rare instances, have had its beginning in antecedent scarlatinal inflammation of the kidneys. Such a result is, however, exceedingly uncommon.

The dropsy indicates the degree of renal derangement, except in the most acute cases, and sometimes attains enormous proportions. As the urine increases in quantity the albuminuria proportionately diminishes, and the dropsy disappears. The skin, which until now has been dry and inactive, becomes softer, more elastic, and resumes its proper functions. The appetite improves, the

spirits, strength, and mental activity return, and good health becomes gradually restored. Just as the microscope reveals the earliest evidence of renal derangement, so does it continue to expose the results of pathological action after chemical tests fail to do so. Tube casts continue to appear in the urinary sediment, sometimes for weeks after the cessation of albuminuria, the blood casts, epithelial, coarsely granular, and fatty casts gradually giving place to finely granular, hyaline, and mucous ones, which in turn finally disappear. When the disorder terminates fatally, the symptoms will be those of acute nephritis; suppression of urine may be followed by cerebral disturbance, headache of violent character, during which blindness may occur, with or without dilatation of the pupil, vomiting, and convulsions, partial or general, coma, and sometimes paralysis; or the fatal termination may be slowly reached through constantly increasing asthenia; or, what is more frequent, complications may arise which cannot always be definitely ascribed to the nephritis or to the scarlatina itself. Such are inflammations of the pleuræ, of the pericardium and endocardium, the peritoneum, the cerebral meninges, etc. Pneumonia, acute articular rheumatism, or enteritis, may also hasten the fatal issue.

Cases are occasionally observed in which dropsy follows scarlatina, but without albuminuria. Indeed, a tendency toward non-albuminuric dropsy after scarlatina has been associated with certain epidemics. Scarlatinal dropsy without albuminuria has been observed by Guersaut, Rilliet and Barthez, Noirot, Bouchus, Löschner, Duckworth, and others. Quincke[19] tries to explain such cases of non-albuminuric dropsy as not depending upon nephritis, but as a consequence of the scarlatinal irritation exerting some peculiar influence upon the connective tissue. Cases occur probably in the experience of most practitioners. One should be cautious, however, in deciding against a nephritic origin of these dropsies, except where they can be definitely attributed to anæmia and debility. Henoch[20] has asserted that nephritis may occur without albuminuria up to the time of death. He reports a case in which anasarca was present for three weeks after scarlatina, without tube casts or albuminuria, until convulsions occurred, death resulting from œdema of the lungs. The necropsy revealed the presence of acute nephritis. He also reports the case of a child, dead on the thirteenth day, of malignant scarlet fever, in whom repeated tests during life had not shown albuminuria, and yet whose kidneys showed indubitable evidence of hemorrhagic nephritis. Steiner has seen nephritis without dropsy, but never dropsy without nephritis, after scarlatina. It is altogether probable, however, that in many cases the dropsy following scarlatina without albuminuria is secondary to concomitant anæmia. This is the view adopted by Henoch. Whatever be their explanation, such cases usually run no remarkable course. The general health is not much reduced. The urine is in normal amount, the various functions are fairly performed. With the disappearance of dropsy convalescence is established.

Scarlatinal nephritis is not associated with any especial phase or type of scarlatina. It is as frequent after mild as after severe attacks; indeed, it is possible that the care exercised over those who have grave attacks of the fever, in proper nursing and surroundings, may furnish a safeguard against renal complications. At all events, there is a widespread belief that the milder cases are more apt to be followed by nephritis and dropsy. Violent nephritis may certainly follow a scarlatina so mild as to have escaped observation. Individual predisposition and epidemic type are probably the most important etiological factors, though at present enough is not known to justify dogmatic statement. The nephritis and dropsy may occur without antecedent symptoms of scarlatina. Instances of this are not uncommon. Several members of a family or of a school or asylum in which scarlatina has been known to prevail may exhibit dropsy and albuminuria characteristic of scarlatina, without having manifested any other symptom of the disease. Such cases pursue an ordinary course generally, but at times develop a severity altogether unexpected.

Inflammation of the Lymph Nodes and Connective Tissue of the Neck.—Although Barthez and Rilliet, and others, have observed cases of scarlatina in which there was no angina, in one form or another it is nearly always present. More or less hyperplasia of the neighboring lymph nodes also constitutes part of the ordinary phenomena of scarlatina. The infection is supposed to originate in the throat, the hyperplasia being due to reflex irritation from the scarlet-fever contagium, or to the secondary streptococcus or staphylococcus or mixed infections. It has already been shown that the inflammation sometimes leads to suppuration and even gangrene of the glandular and periglandular structures. This especially occurs in scrofulous and rhachitic children, but is probably a result of septic absorption. The active symptoms become prolonged beyond those of simple scarlatina into the second, third, or fourth week, and even later, and merit some especial notice. They may not develop until as late as the third or fourth week, thus constituting true sequelæ rather than complications. Usually the fever continues after the subsidence of the eruption, the pain and stiffness of the neck increase, and deglutition continues painful and difficult, or even almost impossible. The mouth may be held open and saliva constantly dribble from it. The neck becomes hard, brawny, and swollen; the integument tense, smooth, and shining. The outline of the neck sometimes stands in line with that of the head and underjaw, and it becomes impossible to distinguish the enlarged nodes in the mass of inflammatory exudation. The patient is unable to find repose, or to swallow food or fluids, unless in small quantities and with great pain. Rest is broken and unrefreshing. Suppuration reveals itself by dark-red, livid spots which soon fluctuate; or it may be deep-seated and difficult to detect, or may point and discharge internally. The parotid gland and periglandular tissue often become involved. At times more or less widespread necrosis may lay bare important muscles, vessels, and nerves, and involve large areas of tissue. These diphtheritic and gangrenous inflammations may give rise to phlebitis or arteritis with thrombosis, and embolism with metastatic inflammation. Compression of the larynx, of the trachea, or of the jugular veins may also result. At times pus may burrow into the deeper cervical structures. Hemorrhage may also occur from exposed vessels. Baader[21] reported two cases of death from hemorrhage thus occurring. The extent of these phlegmonous inflammations of the neck varies greatly. In most cases, after the evacuation of pus, recovery follows, though slowly. In more severe cases death may result from exhaustion or from blood-poisoning. In healing, the scars may be insignificant, or, where granulation involves a large surface and is protracted, the resulting cicatrix may occasion deformity by its contraction. Retropharyngeal abscess, which has already been described, is not common. Schmitz, in the Child's Hospital in St. Petersburg, did not observe it once in 450 cases of scarlatina. Cases, however, have been reported. Bokai reported it as occurring seven times in 664 cases; of these 2 were fatal. Lewandowsky[22] reported 2 cases, both resulting in recovery.

Disorders of the Auditory Apparatus.—These are very important complications of scarlatina. Probably most cases of deafness acquired in early life are results of scarlatina. Of 85 cases of affection of the middle ear following this disorder, 18 had lost the sense of hearing in one or both ears, and 3 were deaf-mutes.[23] Milder degrees of middle-ear inflammation arise by extension from the throat, and are simply catarrhal; but the severer forms are preceded by croupous-diphtheritic inflammation of the fauces. The milder form of otitis media will cause the patient some earache, of which, if he is old enough, he will bitterly complain. Infants will indicate their sufferings by cries, by raising their hands to the ears, by rolling the head toward the affected side. If the Eustachian canal remain pervious, all inflammatory exudation may escape, and no symptoms, other than those

mentioned, and slight and transitory deafness, may occur. This latter symptom may result from the pressure of an enlarged parotid gland upon the external auditory canal. But in the severer forms the pain may be excruciating, the deafness more or less complete, and the fever high. The Eustachian canal becomes occluded from inflammatory swelling, and exudation accumulates in the cavity of the tympanum. Headache may be violent. The drum membrane will be bulged outward from internal pressure, and will be reddened and swollen. The pent-up fluid, unless released by puncture of the drum membrane, finds an exit for itself by perforation. Extreme pain is often produced by pressure upon the tragus and over the mastoid process. Rarely, delirium may be followed by signs of meningitis from extension of the inflammation from the middle ear to the dura mater, along the course of the middle meningeal artery. In mild cases the inflammation will subside, with or without perforation of the drum, and hearing may be perfectly restored. In severer cases, timely tapping of this membrane may yet preserve the sense of hearing—but, unfortunately, it but too often happens that the ossicles of the ear and the tympanic membrane are destroyed; the bony walls, even of the middle ear, become carious, and irreparable damage is done. The severer inflammations involve a croupous-diphtheritic process that often entails wholesale destruction. According to Green, disease of the labyrinth, involving absolute deafness, may occur within a day or two. In such cases the watch held to the skull, the ear, or between the teeth, may not be heard. Green[24] thinks that when loud "clashing," "ringing of small bells," or "musical notes" are heard during scarlet fever or cerebro-spinal meningitis, these are apt to be immediate premonitions of labyrinthine disease; whereas the subjective sounds always accompanying acute purulent inflammation of the tympanum are described as "hissing," "singing," "buzzing," or "throbbing." He also suspects that the fluid secreted in immense quantity—a clear, limpid serum—differing from the wine-yellow serum of tympanic inflammation, may be labyrinthine peri- and endolymph. Pus may form in the mastoid cells. These changes may occur either as complications or as sequelæ. Caries sometimes appears quite early, and the chronic otorrhœa thus set up may last for years, occasioning widespread disorder of both soft parts and bone.[25] In necrosis following middle-ear disease, the facial nerve may become involved with subsequent paralysis. Fatal hemorrhage from the ear may occur after scarlatina, from exposure of vessels from the diphtheritic processes.[26]

Chronic posterior nasal catarrh, and necrosis of the bones of the nasal cavity, constituting various degrees of ozæna, sometimes follow the extension of the pharyngeal inflammation to the nasopharynx. The eye may likewise be implicated in scarlet fever. Conjunctivitis may develop as a complication, or diphtheritic inflammation may extend along the lachrymal canal and involve the conjunctivæ. It may produce keratomalacia and even destruction of the eyeball. Retinitis after scarlatinal nephritis has been observed by Schrötter. Its course is favorable. Temporary blindness may be due to uræmia. Acute amaurosis after scarlatinal nephritis has been noted.[27] Transitory blindness, lasting for from twenty to sixty hours, has been observed by Ebert,[28] Henoch,[29] Tolmachew.[30] In a case of Förster's it lasted eighteen days.

Inflammation of Joints.—Not very infrequently inflammation of the synovial membrane of the joints appears as a complication or as a sequel of scarlet fever. The usual date of its occurrence is during the second week or later. It is often only indicated by pain without swelling, and may be limited to a single joint. In other cases a number of joints are involved, usually the ankles and wrists, knees and elbows. The hip-joints may be affected, and also the smaller joints of the extremities. The inflammation may betray all the features of acute rheumatism—the fugitive character of the inflammation, the metastases, the sweating, the fever, even the tendency to implicate the other serous surfaces, the pleuræ, the endo- and pericardium, and the meninges. Mahomed's[31] studies showed that, as the urine increases in quantity from the seventh to the fourteenth day, it loses its deposit of lithates, and often its albumin (if this has been present). It is highly acid, and uric acid is abundantly thrown down by the nitric-acid floating test. It was at this period that he found the rheumatism most apt to occur. This rheumatism seems identical with ordinary acute rheumatism, but follows a less protracted course. Numerous writers have seen purulent arthritis as a sequel of scarlatina. It commonly occurs during the second or third week, and in most instances is monoarticular. Pyæmic arthritis usually results in suppuration, erosion, and destruction of the cartilage of the joint. According to Spender,[34] the wrist-joint is most often attacked, next in frequency the knee and hip. Recovery may take place, but usually death follows the discharge of pus and the formation of fistulous openings, from exhaustion, or from the further progress of the pyæmia. The approach of these complications, which are fortunately rare, may be recognized through the thermometer.

Affections of the Heart and Pericardium.—Cheadle[32] quotes fifteen cases from West, in which endo- or pericarditis, or both, supervened upon scarlatina. These did not occur during the acute stage, but during desquamation. He, however, considered them rather the result of uræmia and nephritis than of rheumatism. Henoch[33] relates two cases in which acute arthritis appeared during the first week of scarlatina, followed by severe chorea and loud mitral murmur. As to the cardiac symptoms, Cheadle concludes that they occur in scarlatina as results both of scarlatina and of nephritis. He also thought that "scarlatina would appear to have a special influence in causing dilatation and hypertrophy without accompanying valvular disease." Endocarditis, which not uncommonly arises, may be very insidious, and may even pass undetected if not looked for. Probably not a few old valvular affections have originated in attacks of scarlet fever. Acute pleuritis or pericarditis may accompany joint inflammation, or may occur independently. In severe cases they may result in purulent exudation and ultimately terminate fatally. Sometimes the serous inflammations are pyæmic. Endocarditis ulcerosa may begin in this manner.

In the most severe and malignant cases of scarlatina the heart muscle undergoes, first, cloudy swelling, and later, fatty degeneration, especially on the right side. This is the occasion of death from heart failure in many malignant cases.

Affections of the Respiratory and Alimentary Tracts.—Inflammation of the respiratory tract is decidedly uncommon in milder scarlatina. Bronchial catarrh is apt to complicate serious cases. Pneumonia is seen sometimes as a secondary complication following nephritis, diphtheria, etc. Disorders of the intestines are also uncommon. Diarrhœa, when present, is usually associated with severer forms. Diphtheritic enteritis was the most frequent sequel in the cases observed by Fleischmann. Peritonitis may occur as a rare complication. Henoch has seen bedsores complicate scarlatina.

Sequelæ.—The affections that constitute true sequelæ usually are disorders that persist after scarlet fever has completed its course, having begun as complications. Thus are encountered chronic buccal, pharyngeal, nasal, and aural inflammations, nephritis (which, as a rule, ultimately entirely disappears), or inflammation of the various serous membranes. In some cases, marked by severe eclamptic seizures, there results contraction of different groups of muscles, giving rise to permanent deformity. Chorea may develop in connection with the arthritis and endocarditis. Progressive involvement of the limbs with paralysis and wasting may rarely be met with, showing the clinical features of an ascending spinal paralysis. Cerebral thrombosis and hemiplegia rarely occur. Mania has been known to follow scarlatina.[35] Gangrene, apart from that resulting from diphtheria of the throat, is infrequent. Noma has been observed by a number of writers (Barthez and Rilliet, Heyfelder, Bön-

ing, and others), but it is notably less common than after measles. Necrosis of the nasal cartilage was observed by Henoch during convalescence.

Concurrence with Other Specific Affections.—Scarlet fever may be complicated by, or may complicate, other acute exanthemata, not to the extent, however, that many writers believe. Mayr and Hebra, indeed, taught that scarlatina never coexists with measles or smallpox. This question is involved in much obscurity. Scarlatina may be simulated by a variety of affections that may in fact coexist with the exanthemata, by various erythematous eruptions, by the roseola that often precedes and accompanies the eruption of smallpox, by certain anomalous forms of measles, and by various medicinal rashes—those caused by belladonna, copaiba, chloral, and especially cinchona and its preparations. These considerations and faulty methods of observation and recording lead to the rejection of much of the evidence adduced in favor of these coexistences. After all faulty observations are thrown out, however, there still remains strong proof that scarlet fever may coexist with other exanthems. It will be everywhere admitted that one exanthem may follow close upon the heel of another. Prior[36] noted a case in which scarlatina developed on November 18th, varicella on December 2d, and measles on December 13th. When the two exanthems develop simultaneously, there will often remain much doubt, in the absence of evidence of the double exposure of the unprotected individual and of his subsequent double protection. Where one precedes the other by a few days, the difficulties are not so great. Scarlet fever has been observed as complicating, or complicated by, other exanthemata by Steiner,[37] Monti,[38] Thomas,[39] Fleischmann,[40] Fabore,[41] Stillen,[42] Zechmeister,[43] Backer,[44] Dornig,[45] Lewis Smith,[46] Murchison,[47] and many others. The combinations and the order of occurrence have been noted as follows, viz.:

Scarlatina and measles.
Measles and scarlatina.
Scarlatina and smallpox.
Smallpox and scarlatina.
Scarlatina and vaccinia.
Scarlatina and varicella.
Varicella and scarlatina.
Scarlatina and typhoid fever.

Concurrence of scarlatina and Rötheln has not been reported. A probable source of fallacy is the scarlatiniform rash that is often observed in smallpox, and occasionally in typhoid fever; indeed, Simon asserts that Fleischmann has even made this very error. The possibility of these rashes should always be held in mind when questions of concurrence are under consideration. When scarlet fever develops after smallpox the eruption involves the parts of the skin left free by the lesions of smallpox, more especially about the chest and abdomen. When the two exanthems appear simultaneously, their course is shortened; "the second mitigates the first and becomes shortened itself," excepting, according to Fleischmann, when severe smallpox occurs in connection with scarlatina, when death usually results. The same author asserts that if scarlatina appear at the period of maturation of smallpox, the latter, in mild cases, is shortened and mitigated. When scarlatina complicates measles, the latter is shortened, but the scarlatina thus occurring may be mild or severe. Barthez and Rilliet noted that in scarlatina-measles, when the former malady predominates, bronchitis is more marked; but when measles is most severe, faucial angina is worse. All of these statements lack such evidence as would entitle them to unqualified acceptation. Very often neither disease is well developed, and the true condition may be very difficult of recognition. In America these concurrences are more uncommon than they seem to be abroad.

Whooping-cough has been known to complicate scarlatina, and a number of non-specific affections may occur simultaneously with it. These coincidences are purely accidental and present no peculiar interest. Biart[48] has reported psoriasis as following scarlatina. Barthez and Rilliet assert that tuberculous children very rarely have scarlatina. Some chronic affections partially or entirely disappear during an attack of scarlatina. Among these may be especially mentioned certain cutaneous affections, eczema, psoriasis, etc., but they usually reappear upon the establishment of convalescence.

Surgical Scarlatina.—Sir James Paget, in 1864, and again in 1875,[49] declared that patients who have undergone surgical operations are peculiarly susceptible to the action of the scarlet-fever poison. This question has attracted a great deal of attention. In France, Trelat was the first to accept this view, though scarlatinoid rashes had been observed by Civiale, Germain Sée, Tremblay, and others. Similar rashes were reported by Hutchinson, Hilton, Bryant, Lee, Moore, Stirling, and others. They had generally been considered as of septicæmic origin. In 1879 Paley and Goodhart[50] and House[51] reported observations of endemics of scarlatina in the Evelina Hospital for Sick Children and in Guy's Hospital. The first-named authors based their report upon twenty-five cases of scarlatina occurring in surgical patients. Of these nineteen were known to have been exposed to scarlatina, and all the rest, save one, were known to have had possible sources of infection. House's paper was based upon four cases of surgical scarlatina. The epidemic tendencies ceased upon the establishment of isolation, and one cannot doubt their scarlatinal origin. These writers were careful not to assert that *all* such red rashes should be attributed to scarlatina, or that there is *not* "such a thing as a rose rash in a typical case of septicæmia"; but they believe that when occurring in groups they may nearly always rightly be attributed to scarlatina. Riedinger and Howard Marsh also agreed that there exists in wounded persons a predisposition to scarlatina. While Holmes coincided with these views, he, however, declared that many cases of "surgical scarlet fever" are really due to pyæmia and other causes. Most recent writers incline to the opinion that these eruptions are due to true scarlatina. When any epidemic tendency is shown, every one will agree with such conclusions. This cannot be granted of rashes occurring in isolated cases. Of 25 cases reported in Paley and Goodhart's paper, scarlet fever attacked 17 after operations; 7 were without any wound whatever, and 1 had only an old sinus. In many of the cases reported by other writers there was no open wound. These reporters, unfortunately, most rarely note whether their patients had ever previously had scarlatina. Most children, when first exposed to the contagion of this disease, become infected. Is it remarkable that they are unable to withstand it when it attacks them, weakened by injury or surgical operation? Apart from epidemic influence, it is probable that scarlatiniform eruptions in the wounded may justly, in a large proportion of cases, occur quite independently of scarlatina. Rashes of septicæmic origin are well known to occur. Various fugitive eruptions often result from emotional and nervous irritations, or from the ingestion of certain articles of food or medicines. It must be admitted that scarlatiniform septicæmic rashes are uncommon. But there is excellent evidence that they occur.[52] Attempts have been made to establish a differential diagnosis for the surgical scarlatiniform rash. Cheadle,[53] for example, claimed that it has specific characters in not often being universal, and in being confined to the body and parts covered by the clothing; that it rarely lasts twenty-four hours, and that it never desquamates. He also asserted that there is no tonsillar swelling, nor glandular enlargement, nor the peculiar "strawberry tongue." Such points of differentiation do not appear to be well founded. Scarlatiniform eruptions also occasionally follow the ingestion of certain drugs. They may be evoked by belladonna, copaiba, opium, chloral, mercury, and other drugs, but, above all, by cinchona bark and its derivatives. These eruptions are much more common than is generally supposed. Quinine has been frequently given to those who have been injured or submitted to surgical operations, and beyond question eruptions evoked by it are often attributed