to other causes. A number of eruptive forms are observed, but the one of especial moment is the scarlatiniform rash. At the onset it often cannot be distinguished from scarlatina. Beginning with high fever, and often with sore throat, the eruption appears upon the face, chest, and neck, and within twenty-four hours the entire surface may present a bright scarlet aspect. At the end of this period the resemblance may be made perfect by the "strawberry tongue." Up to this point the diagnosis may be impossible. Rarely, it remains so throughout the attack, especially when the ingestion of the cinchona preparation is continued. Usually, however, after thirty-six or forty-eight hours the type of normal scarlatina is departed from. The fever rapidly decreases, the angina disappears, and the rash either fades or acquires features unlike those of true scarlatina. It becomes duller, more papular, and often tends to form miliary vesicles. Eventually it may resemble ordinary "prickly heat." Sometimes, however, the scarlatinal features are preserved throughout. In either case a copious desquamation is sure to follow. This is usually lamellar. Even albuminuria has been known to add to the embarrassment of the diagnostician. These medicinal and septicæmic rashes occur in isolated instances, and may at times baffle the keenest diagnostic powers. We may conclude that unprotected persons who have suffered injury or who have undergone surgical operations are rather more liable to scarlatina than the unprotected healthy. Scarlet fever is more apt than the other exanthemata to attack such persons, because its symptoms vary within such wide limits that it often escapes the attention of those who readily detect other infectious disorders and provide against them. When an epidemic tendency of the symptoms we have been considering is shown to prevail, it may be confidently concluded that true scarlatina is present. Septicæmia is occasionally accompanied by a scarlatiniform rash which does not depend upon the scarlatinal poison. These rashes are often attributed to scarlatina.

Scarlatina Puerperalis.—While pregnant women seem to enjoy a remarkable immunity from the specific eruptive fevers, it is well known that during the *puerperium* they are especially subject to them after exposure, and that the disease is then apt to pursue a grave and often fatal course. Not only scarlatina, but measles and smallpox may affect the lying-in woman with such malignity that the symptoms may not acquire the features of the maladies to which they belong, but become indistinguishable from those of malignant septicæmia. Scarlatina is especially liable to attack the lying-in woman. It may assume the virulence referred to, or it may pursue a course in which it is difficult to determine whether its symptoms are septic or really scarlatinal, or, finally, it may appear with typical and unmistakable features. Not a few writers have thought that the scarlatinal virus may produce in the puerperal woman septicæmia, pure and simple. This view is maintained by Playfair, Braxton Hicks, Leishman, and others. They assert that in these women, after exposure to the specific contagium, symptoms of acute blood-poisoning may be developed, and not those of scarlatina. On the other hand, just as in septic conditions, independent of puerperal causes, an erythematous rash and other scarlatinal symptoms may be observed in which true scarlatina has no part, so must one guard against assigning to scarlatina every scarlatiniform rash occurring in obstetrical cases. It may be septic in origin, or it may be a medicinal eruption. When a septic, or medicinal, or other form of erythema can be excluded, and when exposure to scarlatinal influence is followed by any degree of the symptoms we are considering, are we in atypical cases to look upon the results of the infection as distinctly scarlatinal? More recent writers regard the scarlatinal nature of the disorder as preserved, and as capable of further dissemination. It has not been determined to what extent women who have already had scarlatina preserve an immunity from further attacks during their lying-in period. It would appear that the intensified predisposition of the childbed carries with it an increased liability to second or third attacks. Busey [54] has related a case in which the patient had already had scarlet fever. Other such cases are upon record.[55]

In all probability the scarlet-fever contagium evokes scarlet fever, and not septic disorder, in the puerperal woman, whose systemic condition affords peculiar susceptibility to its influence, and predisposes her to a virulence of its activity that often leads to disastrous results. The less remote the date of delivery the graver the course of the malady is apt to be. If the symptoms do not appear before the seventh day, their development is no longer to be feared. Olshausen [56] collected from the literature 141 cases, of which the scarlatina attacked, during pregnancy, 7; in 8 it immediately followed delivery; in 62 it occurred on the first and second days; in 27 on the third day; in 22 after the third day. After the fifth day none was attacked. While the puerperal woman shows intense susceptibility to scarlatina, the pregnant woman enjoys a marked immunity from it. Olshausen thinks, however, that the period of incubation may last for months during pregnancy, but only a few days during childbed. This opinion he rests upon no solid basis. Primiparæ are more often attacked than multiparæ. The mortality in puerperal scarlatina is high. In the series just alluded to it was 48 per cent. (3 cases during pregnancy and 64 in childbed).

In the recorded cases studied by McClintock [57] the mortality was over 66 per cent. In 34 cases at the Lying-in Hospital the death rate was 30 per cent. Of 10 deaths at this hospital, 8 occurred when scarlatina had developed within thirty-six hours after delivery. Of 18 patients attacked on the first or second day, 8 died. Of those attacked on or after the third day (16 in number), all but 2 recovered. McClintock also quotes Dr. Halahan's cases, as follows, viz.: 3 patients, ill of scarlatina at the moment of delivery, died; of 5 attacked during the first twenty-four hours, but 1 recovered; of 10 attacked during the second day, but 1 recovered; of 4 attacked during the third day, but 1 recovered. The remaining 3, attacked on or after the fifth day, recovered. Braxton Hicks' [58] contributions to this subject have been most important. He believes that in one-half of the cases the usual symptoms of scarlatina are manifested, and that the disease almost always commences after the third day after delivery. The death rate will be greater the earlier after labor the symptoms develop. Though lying-in women are peculiarly liable to scarlatina, they are frequently exposed to its influence without detriment. Women have not seldom been confined in the room, even in the bed, occupied at the same time by scarlet-fever patients, without experiencing the slightest interruption of their normal convalescence; a result that is not astonishing in protected persons if the scarlatinal virus transmits only scarlatina, but which would not be expected were the virus equally competent to communicate septicæmia in these cases. While a large proportion of cases pursue a grave and anomalous course, there are many others in which a perfectly typical scarlatina is observed, without seriously endangering life. Secondary inflammations are not unknown. Metritis, cellulitis, peritonitis, or pyæmia may be developed, but whether these are direct results of scarlatina or of the puerperal condition is undetermined.

The exact nature of puerperal scarlet fever and surgical scarlet fever will remain in doubt until a specific organism of scarlet fever can be isolated. At present this is impossible. In the mean time it is interesting to note that with the development of aseptic and antiseptic methods in surgery and obstetrics, less and less is heard and seen of "puerperal" and "surgical" scarlet fever.

Relapses and Recurrences.—There are recorded numerous instances of relapse of scarlet fever within a short period after the original attack, and second or even third attacks after a more or less prolonged interval are well known to occur. By a relapse is meant a second attack of scarlatina that is evidently due to the persistent activity of the influences that excited the first attack.

Within a short period (three days after deflorescence in a case of Woldberg's [59]) after the original attack all the symptoms are repeated; the initial disturbances, the fever, the eruption, the angina, and other phenomena, with ensuing desquamation, are developed. It is held that the second attack is but the completion of the first, that it occurs after an incomplete primary attack, and that it tends to be severe in proportion to the mildness of the first, and often to affect in eruption only those parts which were originally spared,[60] imparting thus to the second eruption the appearance of scarlatina variegata. The relapse may be accompanied by complications of throat, kidney, and other disorders, that were not present in the earlier disorder, and *vice versa*. These relapses are usually very rare, but seem to be more frequent in certain epidemics.

Thomas applies the term pseudo relapse, or *reversio eruptionis*, to those cases in which the exanthem returns before the disorder has entirely completed its course. Trujawsky found the interval between the two attacks to be from seven to ten days, with an average of eight and five-eighths days. The intermissions are completely afebrile. These relapses have been explained by, (1) a recrudescence of the original contagion, and (2) the action of a newly acquired contagion from a source different from the original one. The prognosis is often graver than in the primary attacks.

Recurrences or attacks of scarlet fever occurring after a more or less protracted interval are more common, and are due to fresh infection. They may occur at almost any period. Trujawsky [61] noted, in 300 cases of scarlatina, 18 patients who had had a former attack. Of these 4 were under ten years of age, 10 were over ten years, and 3 were adults. The interval between the attacks varied from one and a half to seven years. Thomas had personal knowledge of a case in which a second attack occurred. Willan never saw one. Many years may elapse between the two attacks, as when a mother who had the disease during childhood again develops it by contagion from her child. Heyfelder himself had a second attack twenty-seven years after the first one. Trujawsky thought that immunity is greater against contagion originating at the home or in the neighborhood of the patient than when it is brought from a distance. A third attack in the same individual may be observed (as in Richardson's case), and there are reports of repeated attacks of scarlet fever. Bernouilli,[62] for example, mentions the case of a woman, fifty years of age, who experienced in rapid succession six attacks of an exanthem indistinguishable from scarlatina. Other similar cases are on record, but their consideration suggests that they may rather have been forms of medicinal eruption.

Acute exfoliative dermatitis may also be mistaken for scarlet fever, and may attack repeatedly the same person. Rashes resembling scarlatina may occur in various other affections, such as typhoid fever, smallpox, etc. Hallopeau and Tuffier [63] saw a scarlatiniform eruption in acute rheumatism, in which there were two relapses with intense erythema, followed by copious desquamation. The possibility of all such cases being mistaken for scarlatina should be remembered. It is a rather singular fact that many persons suffer from angina whenever they are brought into close personal relationship with those who have scarlet fever. This is commonly mild, but may occasion serious discomfort. Those who suffer thus from exposure to the scarlatinal influence do not communicate scarlatina to unprotected persons. Mild desquamation is said to have been noted in some such cases. This, however, would indicate a true scarlatinal infection.

Pathological Anatomy.—In most fatal cases every trace of eruption disappears after death. After a very intense exanthem, more or less redness may remain. After malignant cases blood extravasations may present the only post-mortem discoloration. The organs primarily affected are the *skin* and the *throat;* the principal complications arise in connection with the *ear* and *cervical lymph nodes;* and the chief sequela is *nephritis*. The *heart* may be affected as a result of the general septic condition, but its lesions are more frequently dependent upon the changes wrought in the kidneys.

Skin.—Remy [64] and Neumann [65] have investigated the histology of the skin in scarlatina. Remy found the capillaries of the papillary layer dilated and hyperæmic, and filled with leucocytes which were enlarged and of different sizes, but not so large as in leukæmia. The vascular wall was not altered. The epidermis was thickened by increase of its cylinder-cell layer. The horny layer, sebaceous glands, and hairs were unchanged. The sweat glands were empty and shrunken. Neumann found the cells of the rete swollen. In many specimens the prickle cells were elongated, and here and there formed interspaces in which exudation cells were embedded, and into which small blood extravasations often occurred. Exudation cells extended abundantly as far as the horny layer, and at the orifices of the follicles they were very numerous. In measles this epidermal layer presents no marked changes; hence it is not difficult to understand why the epidermal cells are so much more liable to carry the contagium in scarlet fever than in measles. The corium was swollen, the fibres were thickened, partly separated by proliferation, partly by enormously dilated vessels that were at times bulbous. It is this exudation into the epidermal layers that causes the loosening of the horny layer from its bed, and the characteristic desquamation. Löschner and Fenwick have also noted this infiltration of the rete. The latter writer found the basement membrane of the sweat glands also thickened and the lining membrane gone in places, but in other places it was increased so as to occlude the sweat glands. The deeper layers were normal throughout. The scarlet-fever exanthem, then, consists of hyperæmia with exudation. Remy found the changes he describes regularly and uniformly distributed.

Throat.—The throat symptoms, as constant as are those of the skin, are due to lesions that are always recognizable after death. The milder alterations offer nothing characteristic; they are identical with those of pharyngeal catarrhal inflammation. In more intense degree follicular inflammation, with suppuration and ulceration, is superadded, and œdema becomes more prominent. The inflammatory changes extend beyond the pharynx into the buccal and nasal cavities, while parenchymatous tonsillitis and inflammation of the cellular tissues of the throat and neck develop, with, sometimes, extensive gangrene.

According to Härlin (Thomas), scarlatinous angina is specific, and is marked by "a deep, bluish-red injection of the mucous membrane of the tonsils and neighborhood, of the uvula, of the posterior portion of the tongue in the neighborhood of the highly swollen papillæ, of the posterior portion of the region of the cricoid cartilage, and of that portion of the pharynx which includes these different parts, and measures about two inches in breadth." This coloring is said to be sharply outlined in the direction of its transverse diameter.

Among the earlier writers on scarlet fever, and as late as the years 1883 and 1884 when the Klebs-Loeffler bacillus was discovered and demonstrated as the specific cause of diphtheria, much space was devoted to the nature of the diphtheritic membrane so often formed in scarlatinous angina. The weight of opinion was that the scarlatinal virus, acting upon the virus of the throat, caused a coagulation necrosis that resulted in the production of the membrane, although it was held that occasionally the scarlatinal process might be complicated by a true diphtheria. The result of modern bacteriological researches has shown that the diphtheritic membrane is in all probability the result of the activity of streptococci and staphylococci or of a mixed infection. There may be, of course, a Klebs-Loeffler infection as well. The influence exerted by the scarlatinal virus in the production of the pharyngeal inflammation cannot be determined in the present state of our knowledge of the specific cause of the disease.

Kidneys.—Bacteriological examinations of the tissues in scarlet fever frequently show general streptococcus

infections, probably having their origin in the lesions in the throat. In a number of these cases there may be found in the kidneys extensive lesions which bear no relation to the presence or absence of streptococci; on the other hand, streptococci may be found in the kidney without any lesion of the kidney.[77] We may assume, therefore, that at times the pathological changes are a result of soluble chemical poisons produced by the virus of scarlet fever or by the activity of the associated secondary infections by streptococci and staphylococci, and at times the lesions are due to direct infection of the kidney by organisms conveyed to it by means of the blood.

Councilman, who has made recent and extensive studies in the pathology of scarlet fever, divides the kidney lesions into two classes: (1) Representing simple degeneration of the epithelium; and (2) representing marked changes in the tissues of the kidney. The lesions of the second class he divides according to their anatomical distribution into (*a*) *interstitial*, in which there is marked proliferation of the interstitial tissue of the kidney; and (*b*) *capsular glomerular*, in which the lesions are chiefly confined to the glomerulus and its capsule.[77]

The first, the purely degenerative form, appears at the beginning of the exanthem, or a few days later, and disappears in a few days or weeks. It rarely excites œdema, and hardly ever kills. It is analogous to the alterations productive of the febrile albuminuria of many infectious diseases. Cloudy swelling and proliferation of the tubular epithelium, and, later, fatty degeneration, are shown. Within the tubular lumen are hyaline and granular cylinders, round cells, and desquamated epithelium.

The second type, the *interstitial*, was first described by Wagner as the lymphoid kidney.[77] The kidney is swollen, the capsule is easily stripped from the cortex, and the surface is moist, whitish, and opaque. Usually there is no hemorrhage, although in some cases punctiform areas may be found in the cortex and intermediate zone. The epithelium may show the changes characteristic of the first or purely degenerative class. The most marked feature of this class of cases is the thickening of the interstitial tissue and the abundant infiltration with round cells, most of which are plasma cells, with some lymphoid cells and polynuclear leucocytes. This form of nephritis may be markedly developed and yet not give rise to clinical symptoms any more pronounced than are seen in the first or purely degenerative class. It is not confined to scarlet fever, but may occur in the course of diphtheria, measles, and other infectious diseases of children, but is not commonly produced in adults in the same class of diseases.

The third form of nephritis, the capsular glomerular, is a more frequent accompaniment of scarlet fever than of any other infectious disease, but is not so common as the acute interstitial form. The kidney is swollen and hyperæmic, the markings of the cortex are obscured or effaced, while the hemorrhagic areas give to the kidneys a mottled appearance. Histologically the chief lesion of this form consists of a proliferation of the capsular epithelium combined with hyperplasia of the connective tissue. The proliferation of the capsular epithelium increases the pressure upon the glomerulus so much as to interfere with the blood supply and hence with the secretion of urine. It is this form of nephritis which is especially liable to give rise to dropsy, to greatly diminished amounts of urine, or to uræmia. The clinical appearance of the urine is that of an acute diffuse nephritis, the amount of blood, albumin, and casts depending largely upon the severity of the infection. Recovery may take place even from the severe forms of capsular glomerular nephritis, but chronic nephritis is a sequela in a certain number of cases. Friedländer holds this to be the only characteristic scarlatinal nephritis.

These three forms of nephritis—the simple degenerative, the interstitial, and the capsular glomerular—may usually be recognized histologically, but transition changes occur. It is not always practicable to make sharp definitions between early and late changes. The first set of changes are chiefly limited to the cortex. They are: 1. Increase of nuclei (probably epithelial) covering the glomeruli. 2. Hyaline degeneration of the elastic intima of minute arteries, especially of the afferent arterioles of the Malpighian tufts. The intima of these vessels is swollen here and there into spindle-shaped hyaline masses, causing narrowness of the lumen. There is similar hyaline degeneration of the capillaries of the glomeruli, rendering them often impermeable. These degenerated parts become fibrous in appearance, and Bowman's capsule becomes thickened. 3. A third change is multiplication of the nuclei of the muscularis of the minute arteries, with increased thickness of their walls. This is greatest at the point of entrance into the glomeruli, but is also distinct in other arteries of the cortex and in the base of the pyramids. There are also swelling of the epithelia of the convoluted tubules and proliferation of their nuclei, especially of the tubules close to the afferent arterioles of the glomeruli. In some cases the epithelium of the large tubules of the pyramids is detached. Klein's[66] observations, (1) that the hyaline changes readily affect the arteries near their point of branching, and (2) that the hyaline substance is of the nature of elastic tissue, agree with the conclusions of Neilson concerning the arteries in various cerebral disorders and in many infectious diseases. He does not think that the anuria and uræmic poisoning in scarlatina, when the kidney does not show conspicuous change, are due to compression of the vessels of the glomerulus by the nuclear germination, as claimed by Klebs,[67] but rather to the changed state of the arterioles, and suggests that the increased formation of arterial muscular fibres, under the stimulus supplied by the disease, may cause a contractility that obliterates the calibre of the arterioles and shuts out the glomerulus from the circulation, and thus, so far as it operates, suppresses the secretion of urine. The parenchymatous changes found in the early stages are slight and difficult to detect, the cloudy swelling and granular degeneration being limited to small portions of convoluted tubules. The second order of changes begins about the ninth or tenth day. They are interstitial as well as parenchymatous. Round cells are found around the larger vascular trunks, spreading into the bases of the pyramids and into the cortex. This process begins about the end of the first week, and gradually increases until portions of the cortex, rarely portions of the bases of the pyramids, are converted into pale, firm, round-cell tissue, in which the tubules become compressed and obliterated. The parenchymatous element of the nephritis consists in crowding of urinary tubules with lymphoid cells and various kinds of tube casts, and fatty degeneration of the epithelium of the tubules. This grows more marked with the advance of interstitial changes. The round-cell infiltration of the cortex begins at the roots of the interlobular vessels, spreading rapidly toward the capsule of the kidney, and laterally among the convoluted tubes around the glomeruli, at first between the medullary rays, but later encroaching upon them. Portions of the cortex may be converted into firm, pale, bloodless cellular masses in which Malpighian tufts and urinary tubules become more or less destroyed. In one case renal embolism was encountered; both interstitial and parenchymatous inflammation was very intense. The kidney was markedly enlarged. Klein also noted deposition of lime in the epithelium and lumina of the tubules, first of the cortex and then of the pyramids, at an early stage of scarlatina, when only slight changes are otherwise shown. He concludes that cases of scarlatina which are fatal after the ninth or tenth day usually show more or less well-marked interstitial nephritis.

Lymph Nodes.—Peculiar changes have been noted in the lymph nodes by Klein. In addition to the ordinary inflammatory infiltrations which he describes as occurring in the lymphatic follicles connected with the organs of the throat and in the lymph nodes of the neck, the ordinary lymphoid cells are greatly diminished in number, and are replaced by large granular cells containing numbers of germinating nuclei.

Liver.—This viscus becomes slightly enlarged from

cloudy swelling. In one case Klein noticed, after two days' illness, acute interstitial hepatitis. The middle and internal coats of some arteries show the same alterations as in the kidneys. Wagner observed lymphoid new formations and numerous collections of cells and nuclei, especially in the interacinous connective tissue.

Spleen.—In the spleen the changes are uniform and constant. They are: 1. Enlargement of the Malpighian corpuscles. 2. Hyaline degeneration of the intima of the arteries. 3. Proliferation of the nuclei of the muscular coat of the ultimate arterioles, with increased thickness of their walls. 4. Hyaline swelling and degeneration of the adenoid tissue around the degenerated arteries. 5. In the central parts of the Malpighian corpuscles the ordinary nuclei of the lymph cells disappear, and in their stead are found large hydropic cells containing pigment (Klein). Other writers assert that there is no uniformity in the splenic changes, beyond a slight enlargement. Biermer has observed enormous enlargement of the Malpighian bodies.

Intestines.—Disorders of the alimentary canal are not frequent in scarlatina, and when they occur it is usually in grave cases. They then not infrequently constitute the principal complicating lesion. In the cases of Fleischmann, diphtheritic enteritis was the most common sequel. The peculiar "shaved-beard appearance" of Peyer's patches has been at times observed, and at times these patches and the solitary glands are prominent, reddened, and inflamed, with associated tumefaction of the mesenteric glands (Harley). Barthez and Rilliet show, however, that in cases in which the typhoid-like lesions have been discovered, the symptoms shown during life did not resemble those of typhoid fever; and conversely, cases of typhoid scarlatina cannot be expected to reveal these lesions after death. Enteritis is more often catarrhal in nature. It has been asserted that in scarlatina the exanthem invades the mucous membrane to the same degree as the skin. Post-mortem evidence of this, however, is by no means constant. The glands throughout the alimentary tract are sometimes swollen, and sometimes form small ulcers and extravasations.

Brain.—It is rare that even the most intense cerebral symptoms occurring in the course of scarlet fever are due to meningitis. Hyperæmia of the brain and meninges, with great venous engorgement, is often seen, but signs of pronounced change are extremely uncommon.

Ear.—An acute otitis media is the most common affection of the ear met with in scarlet fever. The inflammation is especially likely to result in the destruction of tissue, the formation of adhesions, and the establishment of a long-continued suppurative process with accompanying necrosis.

Periostitis and osteitis occur in connection with affections of the joints, of the nose, of the pharyngeal and aural cavities, and of other parts, but afford nothing characteristic. Neither do the general serous surfaces show peculiar lesions. The condition of the blood and blood-vessels after certain rapidly fatal cases is important. Sometimes the blood is very fluid and black. At other times clots are abundant and firm; again, it may have become diffused throughout the tissues. Remy has seen all the vessels of the papillary layer of the skin filled with coagulated blood. Thrombosis of the sinuses has been noted after scarlatinal diphtheria (Thomas).

Heart.—Fatty degeneration of the heart following cloudy swelling, with dilatation, occurring particularly in the walls of the right ventricle, is a frequent result of scarlatina, as it is of other infectious disorders.

Diagnosis.—Scarlet fever must be distinguished from measles, rubella (Rötheln), roseola variolosa, scarlatiniform rashes of septic or medicinal origin, certain idiopathic erythemata, and diphtheria. From measles it differs in its shorter incubative stage, and in the character of its prodromes. In the former affection there are symptoms of coryza and bronchitis, with photophobia, sneezing, coughing, and the appearance of Koplik spots on the mucous membranes of the cheeks and lips (see *Measles*), while in scarlatina the prodromal symptoms especially involve the throat and in children are frequently associated with vomiting. In scarlatina the eruption begins to appear during the first or second day; in measles during the third or fourth day. During the course of scarlatina there is an absence of catarrhal symptoms for the most part. There are the characteristic sore throat and enlarged papillæ on the sides and tip of the tongue, the peculiar "strawberry tongue" (after the first two or three days), the well-defined eruption, the more protracted fever, the pronounced desquamation, and the tendency toward renal complications. The eruptions differ both in their development and in their distribution in the two affections. In scarlatina the face is characteristically invaded by the eruption, which entirely spares the area about the mouth, and is nowhere copiously developed in this region; while in measles the eruption is, probably, most intense upon the face. The macules in measles are large, irregular, and mostly papular. In scarlatina the eruption is punctate and more regularly distributed, not elevated; it is scarlet in color, and generally coalescent, while in measles it is more discrete, elevated, arranged very extensively in forms of crescents and segments of circles, with greater or smaller areas of healthy skin between the lesions, and is of a darker raspberry color. In measles the stage of eruption lasts for from three to four days, and begins to decline as soon as the eruption upon the lower extremities becomes complete. It occupies about thirty-six hours in attaining its acme. In scarlatina this stage lasts for from two to six days or more. It attains its acme in about eighteen hours. In measles there is a rapid return to a normal temperature in uncomplicated cases, while in scarlatina both eruption and fever decline more slowly. The conjunctival, nasal, and bronchial catarrh of measles is absent in scarlatina. In measles the tongue remains coated throughout. Sore throat is constant in scarlatina, quite uncommon in measles, and when present is almost invariably only catarrhal. The fever in scarlatina is at once more intense and more protracted. The desquamation of scarlatina is pronounced and lamellar; that of measles insignificant and branny. The presence of leucocytosis, not otherwise to be accounted for, is evidence in favor of scarlet fever and against measles. Scarlatina is frequently complicated by diphtheritic pharyngitis and renal inflammation, measles by inflammations of the respiratory apparatus.

The eruption of rubella (Rötheln) more closely resembles that of scarlatina. It is paler, more discrete, and its lesions are larger and more distinctly papular. It is more transitory, and fades almost without desquamation, which, when present, is branny. Rötheln, moreover, has a longer incubation, almost no prodromal stage, sometimes marked catarrh, and but slight elevation of temperature. It is feebly contagious, of much shorter duration, and is hardly ever followed by nephritis and dropsy. The diagnosis is difficult only when the eruption of rubella becomes confluent. Here, however, the confluence involves certain areas. It is sharply circumscribed by normal integument, and shows in contrast the outlying characteristic lesions. It is of a pale rose-red, and not of a scarlet color, and is accompanied by the peculiar symptoms of rubella, and rarely lasts more than thirty-six hours. Both measles and rubella may at times closely resemble the milder forms of scarlatina, and from the eruption alone the diagnosis may be difficult; but a consideration of all the symptoms will usually lead to correct conclusions. The presence of leucocytosis, in the absence of other conditions to account for it, is strong evidence in favor of scarlet fever.

Roseola variolosa should excite embarrassment only when it occurs before the peculiar eruption of smallpox has appeared. It is less general, is more like a simple diffuse erythema than is scarlatina, and is so speedily followed by the characteristic vesicular eruption that doubt will soon be dissipated. Its coexistence with the essential eruption may excite suspicions of a concurrence of scarlatina and smallpox. Such an error may readily occur.

Obstetrical and surgical scarlatina have already re-

ceived attention. When erythema begins near a wound and becomes scarlatiniform in spreading, a septic origin must usually be allowed, though instances of scarlatina thus beginning have been reported; otherwise, septic erythema is more circumscribed and irregular. Scarlatina in the wounded and in lying-in women may be perfectly typical.

Medicinal eruptions have unquestionably been at the bottom of many errors of diagnosis. It has been shown that many drugs may excite eruptions and general symptoms very like those of scarlatina; but for the most part they are simple active hyperæmias, such as are produced by the action of belladonna upon the cutaneous arterioles. Such eruptions differ from that of scarlatina in the absence of prodromes, and, usually, of fever. They are also mostly partial and without the history, course, or results of scarlatina; but at times, and especially when they follow the ingestion of preparations of cinchona, the whole complex of scarlatinal symptoms may be accurately simulated. The conditions for diagnosis have already been pointed out. In second or repeated attacks of so-called scarlatina, due consideration of the possible influence of drugs as an etiological factor will doubtless convert some very puzzling cases into very simple ones.

Acute exfoliative dermatitis and desquamative scarlatiniform erythema[68] may well be mistaken for scarlatina upon their first appearance. The rash is more protracted than in the essential fever, and is less abrupt in its onset. The local symptoms are very marked, while the constitutional phenomena are usually insignificant. The desquamation may begin while the eruption is in full florescence. These affections are not contagious and have no specific sequelæ.

An erysipelatous eruption may be like that of scarlatina. It, however, differs markedly in its distribution, its evolution, and its course, being never universal, always progressive, and of indefinite duration. The subjective symptoms are quite different in the two affections; the erysipelatous eruption is painful both spontaneously and on pressure. Much œdema accompanies the latter eruption.

Diphtheria may complicate scarlatina, and the intensity of the local inflammation may induce a coagulation necrosis exactly corresponding to the membranous formations of diphtheria. Idiopathic diphtheria may especially resemble scarlet fever when it is accompanied by the erythematous exanthem that is sometimes developed, either early in the disorder or later, in cases of blood poisoning. At first it may not be possible to arrive at a correct diagnosis. According to Robinson,[69] in the early diphtheritic erythema there is no marked elevation of temperature. The rash may begin in any region, and rarely extends to the whole surface. The tongue is not affected, and there may be no special general disturbance. Desquamation does not occur. Bacteriological examination of the nasal and buccal secretions will almost always determine the presence or absence of true diphtheria. The late diphtheritic erythema is septic.

When the eruption of scarlatina is imperfectly developed, or when it does not appear at all, and when sore throat and fever are the only symptoms to attract attention, the diagnosis must rest upon the history of the patient and his surroundings, and upon the course of his illness. In not a few cases a retrospective diagnosis of scarlatina must be made, after the occurrence of desquamation or the supervention of nephritis and dropsy under conditions that indicate their scarlatinal origin.

Prognosis.—The mortality from scarlet fever varies widely in different epidemics. From the affection that in Sydenham's time "hardly deserved the name of disease" to a pestilence of intense malignity, all degrees of fatality have been, and continue to be, observed. Epidemics have been recorded in which no deaths have occurred. Recently Whitla[70] has recorded but a single death in 133 cases of scarlatina treated in hospital. Such results are, unhappily, exceptional. The mortality has been known to reach 30 and 40 per cent. An excessively high rate of mortality is, in great part, attributable to epidemic tendencies toward grave complications, diphtheria, nephritis, etc. In private practice the death rate will not often exceed 10 per cent. In hospitals the percentage of deaths is usually much higher, the result being due to the fact that milder cases are kept at home for the most part, and not to differences in social condition, except in so far as neglect and exposure previous to admission may have aggravated an attack or have excited a complication. The death rate will be high or low in accordance with the type of the prevailing epidemic, and the average mortality of the disease should always be considered with reference to this. Neither season nor atmospheric condition appears to exert any influence upon the epidemic type. Likewise, telluric conditions do not modify it. Benign and malignant epidemics follow each other without evident cause. The mortality at the beginning and during the height of an epidemic is greater than during its decline. Barring the effects of extreme poverty and exposure, scarlet fever affects the rich and poor impartially. The sexes are almost equally attacked, but age exerts a striking influence upon the result. Children under one year of age, though less apt to be attacked, are especially liable to fatal forms of the disease. According to Fleischmann, the mortality at St. Joseph's Hospital was: under one year of age, 75 per cent. (8 cases, 6 deaths); from one to four years of age, 43 per cent.; from five to twelve years of age, 19.6 per cent.; the total mortality being 10 per cent. The majority of deaths occur under the sixth year of age; with increasing years the prognosis becomes more favorable. Fleischmann's records show a higher mortality than those of some other writers. For example, Kraus gives 4 deaths in 13 cases less than one year of age; 29 deaths in 113 cases from the close of the first to the close of the fifth year of life; 10 deaths in 106 cases from the end of the fifth to the close of the twelfth year of age; and 2 deaths in 40 cases from the twelfth to the twentieth year of age. Voit reported 1 death in 5 cases less than one year of age; 24 deaths in 166 cases from the first to the close of the sixth year of age; 10 deaths in 109 cases from the sixth to the twelfth year of age. Roset reported 16 deaths in 43 cases less than one year of age; 31 deaths in 156 cases from the first to the close of the fifth year of age; and 3 deaths in 88 cases over five years of age.[71] An exception must be noted to the favorableness of the prognosis in persons of maturer years, in the case of puerperal women, in whom scarlatina has already been shown to be especially malignant. No case can appear to be so mild as to justify a prognosis unqualifiedly favorable. From the beginning until the termination in recovery there is no period when a sudden change may not place the life of the patient in jeopardy, whether by an aggravation of the essential symptoms of the disease, or by the supervention of complications. The prognosis, however, is generally favorable if the disease pursues a regular course; if the eruption follows a brief prodromal stage and is regularly developed; if the fever, more or less intense from the first, does not exceed at the height of the eruption 40° C. (104° F.), and, steadily falling, reaches the normal on the sixth, seventh, or eighth day; if the angina do not assume a diphtheritic character, and is not complicated by parenchymatous tonsillitis, retropharyngeal abscess, or cellulitis of the throat or neck; if the kidneys remain unaffected or show only slight evidences of disorder. On the other hand, the prognosis is more grave when the eruption appears after a prolonged prodromal stage, or when the attack is ushered in by convulsions or other profound nervous disturbance; or when the temperature reaches a high degree, 40.6° to 41° C. (105° to 106° F.), at once; or when intractable vomiting is present; or when diarrhœa is a prominent feature; or when the pulse beats more than one hundred and twenty times to the minute, and is feeble, unequal, and irregular; or when the throat is ulcerated and develops diphtheritic inflammation; or when suppurative, parenchymatous, or gangrenous inflammation of the tonsils, or retropharyngeal abscess supervenes; or when the neck becomes swollen, brawny, and livid from glandular, peri-

glandular, and diffuse cellular inflammation. Apprehension should always be excited if the eruption come out imperfectly or irregularly while the fever is intense; or if, once fully developed, it suddenly fade; or if the eruption assume a livid color or a distinctly hemorrhagic character. A coppery hue of the eruption is unfavorable, as is also a livid coloration of parts not invaded by the eruption. Small, scattered petechiæ in the midst of an otherwise normal eruption are unimportant. Miliary vesicles, developing in the ordinary course of the fever are insignificant; occurring later, during an attack of unusual severity, they are often the forerunners of death. Convulsions first occurring after the height of the fever are more ominous than if occurring earlier. Should the eruption, and especially the fever, continue unabated after the usual period, dangerous complications are to be apprehended. Coma is of grave augury, as indicating uræmia, œdema of the brain, or even meningitis. Nephritis is more serious the earlier it is developed. It occasionally happens that scarlet fever at first shows the symptoms of a mild attack, but, before the completion of the eruption, assumes a malignant character. If symptoms of malignancy occur after the completion of the eruption, they are usually attributable to complications. On the other hand, all the signs of malignant scarlatina may be present at the outset. High fever, rapid pulse, convulsions or coma, protracted vomiting, intense eruption, may all yield after the second or third day, the disease thenceforward pursuing a mild course; again, symptoms of malignancy may disappear upon the supervention of a delayed eruption. Mayer[72] observed a temperature of 43° C. (109.4° F.) on the evening of the second day. The temperature subsequently varied slightly until the fourth day, when, upon the appearance of the eruption, it subsided. The occurrence of scarlatinal diphtheria always increases the danger of death. Heubner regards its sudden extension to the soft palate and to the portals of the œsophagus and trachea as certainly to be followed by death within from twenty-four to forty-eight hours, the fatal issue occurring either through gradual progress of gangrene, by inflammation of the lymphatic glands and connective tissue of the throat and neck, or by œdema glottidis. When circumscribed spots are invaded and the lateral portion of one tonsil shows the first patch, from which the membrane gradually spreads, recovery may occur. Diarrhœa persisting during the attack greatly increases the danger. Nephritis is always a serious complication, though terminating favorably in most cases. The danger is usually proportionate to the earliness of its occurrence. Death may occur as in ordinary nephritic inflammation. Scarlatinal nephritis most rarely becomes chronic. Inflammation of the organs of hearing, while rarely imperilling life, often results in partial or complete deafness. This, according to Burkhardt-Merian,[73] depends upon croupous-diphtheritic inflammation primary in the throat. The prognosis is more unfavorable if the process be allowed to go untreated. Rheumatic and rheumatoid inflammations are not commonly dangerous complications. Endo- and pericarditis, pleurisy, peritonitis, meningitis, pneumonia, dysentery, parenchymatous degeneration of the heart, etc., are all complications of extreme danger. Purulent inflammations of pyæmic origin usually constitute sequelæ of scarlatina, and are of the gravest importance.

Treatment.—Mild cases of scarlatina require little more than good nursing, the regulation of the diet, and proper precautions against the spread of the disease to the other members of the household. Severe cases, on the contrary, tax the resources of the physician to his utmost.

General Principles of Prophylaxis, Hygiene, and Disinfection.—As soon as the disease is recognized the patient should be removed to a clean, well-ventilated room, preferably at the top of the house, and accessible by way of back stairs, and all persons not concerned in the care of the patient should be rigidly excluded.

The following practical rules and directions, taken in part from those issued by the New York Board of Health, for the prevention of the spread of the disease in a family in which one case exists, cover in a general way the matter of prophylaxis and disinfection. They should be followed as closely as possible, but at times may be modified to suit the requirements of the case. A copy should be given to the nurse and parents at the beginning of the quarantine, and they should be instructed to study the principles on which the directions are based in order that they may know how to meet conditions which are constantly arising in the course of the disease and which cannot be specially mentioned in directions intended for general use.

1. If possible one attendant should take the entire charge of the sick person, and no one else besides the physician and nurses should be allowed to enter the sick-room. The attendant should have no communication with the rest of the family. While in the sick-room she should wear a covering to protect her hair, and a gown, which should be removed when she leaves the room. When leaving the portion of the house which is quarantined she should change her shoes, and dress and disinfect her hands and face, and should make use of the back stairs, so far as possible in going in and out of the house. The members of the family should not receive or make visits during the illness. Other children in the family should not be allowed to go to school, if they remain in the infected house, and if sent away they should be kept from other children until the stage of incubation has passed. The physician should exercise great care not to carry the contagious elements to other patients. Disregard of such precaution by many physicians is a fault only too common, and one which deserves the most severe condemnation. A cap covering the head and back of the head, a gown reaching from the neck to the floor, and rubber overshoes should always be put on before entering the patient's room. When these are removed, they should be dipped in carbolic acid or corrosive sublimate solution, and hung up to dry in an ante-room, where they will be ready for use on the following day. Rubber gloves are also a useful addition to the articles just mentioned.

2. The discharges from the nose and mouth must be received on handkerchiefs or cloths, which should at once be immersed in a carbolic solution (made by dissolving six ounces of pure carbolic acid in one gallon of hot water, which may be diluted with an equal quantity of water). All handkerchiefs, cloths, towels, napkins, bed linen, personal clothing, night clothes, etc., that have come in contact in any way with the sick person, after use should immediately be immersed without removal from the room in the above solution. These should be soaked for two or three hours and then boiled in water or soapsuds for one hour. They should be laundried separately from the household articles.

3. Great care should be taken, in making applications to the throat and nose, that the discharges from them in the act of coughing are not thrown into the face or on the clothing of the person making the applications, as in this way the disease is likely to be caught.

4. The hands of the attendant should always be thoroughly disinfected by washing in carbolic solution, and then in soapsuds, after making applications to the throat and nose, and before eating. Rubber gloves may be worn with advantage while handling the patient.

5. Surfaces of any kind soiled by discharges should be immediately flooded with the carbolic solution.

6. Plates, cups, glasses, knives, forks, spoons, etc., used by the sick person for eating and drinking must be kept for his especial use, and under no circumstances be removed from the sick-room nor mixed with similar utensils used by others, but must be washed in the room in the carbolic solution and then in hot soapsuds. After use the soapsuds should be thrown into the water-closet, and the vessel which contained it should be washed in the carbolic solution.

7. The room occupied by the sick person should be thoroughly aired several times daily, and swept frequently, after scattering wet newspapers, sawdust, or tea leaves on the floor to prevent the dust from rising. Af-