less not without value. He found that with the expectant plan of treatment the mortality was 35 per cent.; that in patients who received two cold baths daily, after the specifications of Brand, it was 33⅓ per cent., and that among those who received six cold baths daily the mortality was reduced to 16.5 per cent. Curschmann advocates the use of hydrotherapy. He speaks favorably of the water-bed method. The patient is allowed to remain for hours, even all day, in water whose temperature is never carried below 68° or 70° F. The water can be conveniently changed from time to time without disturbing the patient. Curschmann employs the Brand method only in the most severe cases.

Among other hydrotherapeutic measures may be mentioned the cold sponge bath, cold packs and half packs, lukewarm baths, and the graduated full bath. For headache and psychic symptoms the ice cap will be found of great value. Whichever method of treatment is decided upon it must be carried out with great regularity.

The cold baths influence not only the fever, but all the resultant cerebral, circulatory, and respiratory symptoms. At times little decrease in the temperature is observed after the bath, but improvement in the general condition is always seen.

The open-air treatment has already been referred to under prophylaxis, and has much to recommend it. Special nervous, circulatory, and pulmonary symptoms are managed the same as in typhoid fever.

Specific Treatment.—Little has been accomplished in the way of specific treatment. Legrain[23] feels certain of the beneficial effects of the serum treatment. He treated with success a number of cases in a prison by the injection of serum from convalescent typhus-fever patients. Chantemesse also reports success with this method. Because of the limited number of patients treated, little importance can be attached to these reports. It is unfortunate that more work along this line has not been done. It offers another field of research that has probabilities of fruitfulness.

Methylene blue has been considered to have some specific action in typhus fever. Nefedieff,[24] however, reports unfavorable results with this method of treatment. Other so-called specific methods of treatment, as by carbolic acid, sulphocarbolates, and the sulphides, have proved to be of no value.

David Murray Cowie.

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UKIAH VICHY SPRINGS.—Mendocino County, California.

Access.—Viâ San Francisco and Northern Pacific Railroad to Ukiah, thence a drive of three miles to the springs.

This pleasant resort, otherwise known as Doolan's Vichy Springs, lies nestled among enchanting hills, which fringe the boundary line of Lake and Mendocino Counties. This region has a combination of advantages which make it a natural sanitarium. From April to November the climate is delightfully balmy and the atmosphere is pure, clear, and invigorating. The scenery is of a pleasing and attractive character, and the neighborhood of the springs affords excellent fishing and gunning. The waters belong to the alkaline-carbonated class, and are clear and sparkling, with an agreeably pungent taste. Their chemical composition is not unlike that of the celebrated Vichy Springs of France (Grande Grille), and their physiological action on the human economy is practically identical with that exercised by those waters. Following is Anderson's analysis: One United States gallon contains (solids): Sodium chloride, gr. 28.60; sodium carbonate, gr. 195.52; sodium sulphate, gr. 0.36; potassium chloride, gr. 0.09; magnesium carbonate, gr. 19.75; calcium carbonate, gr. 18.14; ferrous carbonate, 0.07; silica, gr. 5.92; and traces of potassium carbonate, potassium sulphate, strontium carbonate, barium carbonate, lithium carbonate, borates, arseniates, and salts of alumina. Total solids, 268.45 grains. Carbonic-acid gas, 224.75 cubic inches. Temperature of water, 93° F.

The French Vichy contains 408.95 grains per United States gallon. As an antacid, tonic, aperient, diuretic, and alterative mineral water the Ukiah Vichy ranks among the best in the country. It has proved highly beneficial in irritable states of the gastro-intestinal mucous membrane, dyspepsia, torpidity of the bowels, sluggish action of the liver, etc. Excellent results have also been observed in Bright's disease, acid states of the blood and urine, rheumatism, and gout. The waters are soon to be used commercially. There are numerous other springs on the premises, but their waters have not yet been analyzed. Good accommodations and bathing facilities are provided for visitors.

James K. Crook.

ULCER, ULCERATION.—The proper definition of the word ulcer, and exactly what limitations and restrictions should be placed on the term, have always been matters of considerable difference of opinion. Warren,[1] Curtis,[2] Tillmann,[3] and others follow with slight modifications the definition of Billroth, who defines an ulcer as "a loss of substance with no tendency to heal." Warren even goes further and says: "The term implies that the wound or granulation surface is stationary or enlarging, and that it has developed by death of the part piecemeal."

He further states: "An open granulating wound is not an ulcer, but it may become one if the granulations begin to break down and the edges begin to melt away." On the other hand, Park[4] defines an ulcer as "a surface which is or ought to be granulating," thus agreeing with Golding Bird,[5] whose definition is "a limited area of granulation tissue on the surface of the body."

Thus we have two absolutely contradictory ideas. If we accept the first definition, we must exclude all those ulcers which have begun to granulate in a healthy manner, and to which the name "healing ulcer" has been given; while if we accept the definition of Golding Bird, we leave out all those ulcers which are not granulating, such as rapidly spreading phagedenic ulcers, or those whose bases are formed by the underlying tissues (raw ulcer), or that condition known as croupous ulcer. Park, however, overcomes this difficulty by saying "is or *ought to be* granulating." Moreover, although a granulating wound cannot be distinguished pathologically from a healing ulcer, the term ulcer conveys a different impression; a definition which includes all cases would be: An ulcer is a solution of continuity or superficial loss of substance of the skin or mucous membrane which is, or has at some time been, progressively enlarging by inflammation. This definition would include all of the above-mentioned cases, and exclude healthy granulating wounds which have been the result of traumatism.

Ulceration is a word which by many authors is very loosely used. By some it is used synonymously with ulcer. Others employ the term where several ulcers exist, or where a considerable surface is the site of ulceration, as in ulceration of the rectum; but it seems better that the term should be restricted to the pathological process by which ulcers are formed, *i.e.*, a superficial molecular gangrene or disintegration of the skin or mucous membrane.

Varieties.—Since Bell,[6] as early as 1778, classified ulcers according to whether they were due to some local cause, or were symptomatic of some constitutional vice, his classification has with certain modifications been largely followed by subsequent writers. A satisfactory classification, however, is rendered difficult by the fact that there are often several etiological factors which may be present in any one case, as, for example, varicose ulcers where frequently traumatism, infection, phlebitis, periphlebitis, œdema, or eczema, either together or separately, may determine the formation or non-formation of an ulcer. Furthermore, an ulcer may be dependent on a certain poison acting in two different ways, either by primary infection or constitutionally, as in the initial lesion of syphilis and in tertiary syphilitic ulcers.

From an etiological standpoint ulcers may be divided into three great classes:

1. *Non-specific ulcers*, including all those cases not due to some particular infection or to malignant disease, but whose etiology depends on: (*a*) traumatism; (*b*) infection with some of the pyogenic or saprophytic bacteria; (*c*) interference with the circulation either of the arteries or of the veins or lymphatics; (*d*) interference with nutrition through the trophic nerves; (*e*) pressure from without as from splints, apparatus, or even the bedclothes, as in the case of bedsores; or from within as from benign tumors, gouty tophi, etc.; (*f*) skin diseases, as pemphigus, eczema, ecthyma, and herpes; (*g*) constitutional disease, as scurvy or diabetes; (*h*) the various causes of ulcers of the mucous membranes (excluding specific infection and malignant disease), as uræmia, mineral poisons, abdominal burns, etc.

2. *Specific ulcers*, including those due to: (*a*) syphilis; (*b*) tuberculosis; (*c*) typhoid; (*d*) diphtheria; (*e*) various forms of dysentery; (*f*) malaria; (*g*) glanders; (*h*) actinomycosis; (*i*) leprosy.

3. *Malignant ulcers*, among which are included superficial malignant new growths which break down and ulcerate, or deeper ones which involve and destroy the skin or mucous membrane overlying them. These malignant ulcers may follow the types of carcinoma, sarcoma, epithelioma, or rodent ulcer, being due to the breaking down of the primary growth, or an old chronic ulcer may become malignant by undergoing epitheliomatous or more rarely sarcomatous degeneration.

Besides this classification according to etiology, various names are given to ulcers depending on their condition at the time of examination. Thus we have:

1. Healing.
2. Spreading: (*a*) inflamed; (*b*) phagedenic; (*c*) sloughing.
3. Chronic: (*a*) with feeble, indolent or exuberant granulations; (*b*) with callous edges; (*c*) with a croupous base; (*d*) with a raw base. And in addition the terms fungating ulcer and scirrhous ulcer are sometimes used to describe ulcers due to malignant disease. By some authors these names depending on the condition of the ulcer are used as a basis of classification; but in reality they are only phases through which various ulcers may run during their course.

Etiology.

We have seen in considering the classification of ulcers that there may be several factors in the etiology of one of these lesions, or that one cause may act alone. The etiology may therefore best be studied by dividing it into:

1. Predisposing causes: (*a*) general; (*b*) local; and
2. Exciting causes.

1. Predisposing Causes.—(*a*) *General.—Age, Sex, Occupation, Social Condition, etc.* Age can hardly be considered as an important factor in the etiology of an ulcer, as there are so many other elements which have a more direct bearing on its causation. To be sure, old age is accompanied by retrogressive tissue changes, atheroma of the arteries, impaired circulation, etc., and one would therefore expect the statistics to show a greater proportion of ulcers during the later decades of life. But that ulcer is not relatively more frequent in the aged is probably due to the fact that such frequent causes as tuberculosis prevail in the early decades, and syphilis in early middle age, and also that traumatism in the early and middle decades is more frequent than among the aged. As regards sex, it has been shown by statistics that ulcer is three times more prevalent in men than in women. This is probably due to the fact that men are more exposed to traumatism, and that they are more likely to neglect a slight wound, which with infection becomes an ulcer. Also the greater prevalence of syphilis and alcoholism in men may in some measure explain why ulcers are more common among them than among women. Occupation seems to have little to do with the etiology of ulcer beyond the fact that it may predispose to traumatism or various forms of infection and that it may prevent cleanliness. It is in this latter element that we have one of the most important etiological factors in the causation of ulceration. The non-specific and non-malignant forms of ulcer are infinitely more common among the poorer classes, among whom lack of means or lack of intelligence, as well as untidy habits, will allow filth, and with it of course infection, to enter a wound the result of some slight abrasion, or the lesion of some skin disease; and the formation of an ulcer is the consequence.

Constitutional Disease.—Many of the constitutional diseases such as diabetes, lithæmia, scurvy, anæmia, tuberculosis, and syphilis, as well as the exhausting fevers, as typhoid, scarlatina, etc., lower the vitality of the tissues, while other conditions, such as valvular disease and fatty degeneration of the heart, general obesity, and atheroma, by preventing proper circulation predispose to the formation of ulcers when there is in addition some exciting cause.

(*b*) *Local Predisposing Causes*—1. *Interference with the Arterial Circulation.*—There may be a predisposition to ulceration as a result of embolism which cuts off the nutrition of a part, or the embolus may be infected and thus cause the formation of an abscess, which, if superficial, may result in the development of an ulcer. Atheroma of the blood-vessels, by interfering with nutrition, may also act as a local cause. Certain vaso-motor disturbances, such as occur in frost-bite, chronic ergotism, and Raynaud's disease, may produce small areas of localized gangrene, and these areas may become subsequently the seat of ulceration.

2. *Interference with the Venous Circulation. Varicose Veins.*—When œdema results from interference with the return of venous blood from a part, it is obvious that such a condition would predispose to the formation of ulcers. The exact relation of varicose veins to the formation of ulcers is, however, a matter of dispute. There are many people who have varicose veins even to a severe degree, who never suffer from ulcer; consequently we must look for some other element in the etiology. Schreider[7] tried to prove this other element to be the gouty diathesis, and he considers both the varicose veins and the ulcers to be the result of lithæmia. Quénu[8] found a neuritis secondary to the varicose veins, and considers the formation of the ulcer to be due to trophic disturbance. In this view he is upheld by Silvy.[9] On the other hand, Mr. A. Pearce Gould[10] writes of "those troublesome ulcers of the lower third of the leg nicknamed varicose." There is one condition, however, which certainly has a direct bearing on the relationship of varicose veins to ulcer, and that is

phlebitis. Where phlebitis and periphlebitis occur, especially in the smaller venous radicles, small abscesses often form, the adjacent skin becomes involved, and an ulcer develops; or the rupture of the diseased wall of one of the

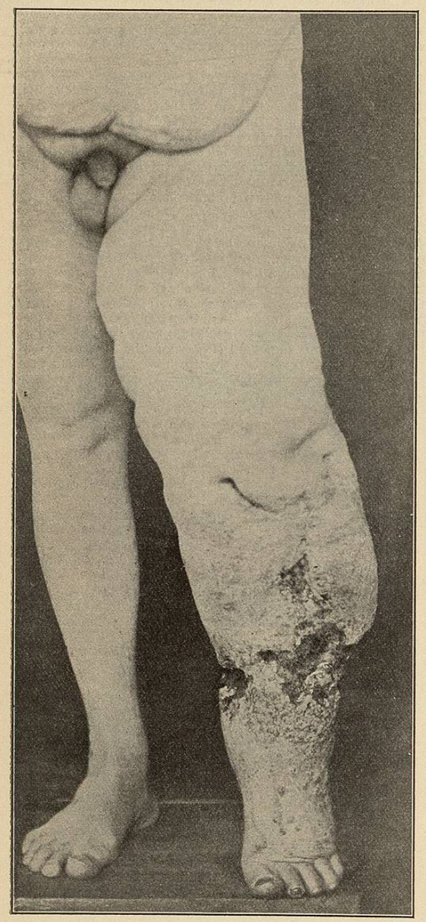

FIG. 4823.—Ulcers due to Lymphatic Obstruction. (From Dr. B. Farquhar Curtis.)

small veins may, in the case of a person of uncleanly habits, readily prove the entering point of infection. Then, under the favoring influence of an impaired circulation in and poor nutrition of the part, such a rupture is not unlikely to end in the formation of an ulcer.

3. *Interference with the Lymphatic Circulation.*—Occasionally, in the treatment of extensive cellulitis, it is found necessary to make deep incisions, and the contraction of the resulting scar tissue may interfere with the lymphatic flow and give rise to stasis and thickening of the part. Under such conditions, and especially when they occur in the leg, ulceration is very likely to take place. A similar condition is found in elephantiasis (Fig. 4823).

4. *Trophic Changes.*—In the discussion of the so-called varicose ulcers, it has been mentioned that Quénu and Silvy believed the ulcer formation to be due to trophic changes. There are many other conditions, however, in which the relationship of ulceration and disease of the nerves or nervous system is more apparent. In infantile paralysis, in which the nutrition of certain parts is impaired by reason of the diseased condition of their centres in the spinal cord, all that is needed for the production of an ulcer is often a slight traumatism plus infection. Perforating ulcer of the foot is a frequent complication of locomotor ataxia. Also myelitis, peripheral neuritis, and other pathological conditions of the nervous system may, either through trophic changes or by reason of impaired sensation, be directly related to the formation of an ulcer.

EXCITING CAUSES.—*Traumatism, etc.*—One of the most frequent direct causes of ulcer is an injury of one kind or another. The degree of the injury may of course vary greatly, and whether or not it will produce ulceration depends on one or more of the predisposing causes already mentioned. A severe traumatism may be necessary entirely to destroy the vitality of a part in a healthy person; while a slight injury might do so in the presence of lowered vitality, constitutional disease, impaired circulation, trophic nerve disturbance, or where the amount of subcutaneous tissue between the bone and skin is slight in amount, as over the malleoli for example. Moreover, the nature of the traumatism may vary. The skin or mucous membrane may be destroyed by a contusion, laceration, friction, or a burn, so that it must heal by granulation. If healing occurs in a healthy manner at once, without any infection, we have a clean granulating wound. If, however, infection takes place and the wound becoming unhealthy extends by a process of molecular gangrene, then we have an ulcer. Pressure from within or from without may cause ulceration, mainly by the cutting off of nutrition and thus producing localized gangrene. As illustrations of ulcers caused by pressure from without may be mentioned bedsores, as also pressure sores from too tightly applied splints and badly fitting orthopedic apparatus. Ulceration may also be caused by pressure from within outward, as in the case of the end of the bone in faulty amputations, and in that of the growth of a benign tumor. In the same manner foreign bodies, such as deposits of biurate of sodium (gouty tophi), may result in ulceration of the overlying skin. Or the ulcer may be produced by the irritation caused by the presence of a foreign body beneath the skin, as in the case of a dracuncular ulcer caused by the filaria medinensis or guinea-worm.

Infection.—Roswell Park[4] says: "The idea underlying ulceration is infection, and when limited to its proper significance, the term should never be used for a process in which infection and consequent breaking down of tissues do not virtually comprise the whole process." The development of an ulcer from a clean granulating wound or abrasion usually depends on infection with some of the ordinary saprophytic or pyogenic germs. Or, in another class of cases, infection with consequent suppuration or gangrene of the skin resulting in ulcer formation, may take place through the circulation. Rapidly spreading ulcers, such as occur in phagedena and hospital gangrene, and some of the rapidly spreading forms of tropical ulcer, are usually due to some particularly virulent septic infection, acting on a weakened subject. The chancroid is also an ulcer depending for its existence on infection, whether it be due to the ordinary pyogenic germs, or to a specific bacillus, such as has been described by Ducrey, who found a rod-shaped bacillus with rounded ends in the secretion from chancroidal ulcers. In old chronic cases the bacteriological infection is of a mild type. Bukovsky[11] examined bacteriologically one hundred cases of ulcer of the leg, and found that the number of bacteria, even in foul cases, was much less than he expected, in many cases only a single species being found. The bacillus pyocyaneus was most frequently found. Other organisms found were the staphylococci, streptococci, B. coli, B. proteus, and (in a single instance) Friedländer's pneumococcus. Less interesting were the micrococcus albus, M. sulphureus, B. lactis aërogenes, B. mesentericus, B. subtilis, B. tumescens, and sarcinæ. On the other hand, certain definite forms of ulceration depend on infection with some specific germ or virus, *e.g.*, syphilis, tuberculosis, leprosy, typhoid, glanders, etc.

Syphilis.—This disease in its various stages is one of the most frequent causes of ulcer formation. In the primary stage the chancre is usually an ulcerative lesion. The ulcerative manifestations of secondary and tertiary syphilis may develop from a tubercular, pustular, or pustulo-crustaceous syphilide. But it is in the tertiary stage which, according to the statistics of Haslund,[12] develops in twelve per cent. of all cases infected, that we find the form which is most often spoken of as a true syphilitic ulcer. In this stage gummata form in the subcutaneous tissues or deeper parts, the overlying skin becomes involved and breaks down, the gumma due to its poor nutrition and deficient blood-supply sloughs out, leaving either the typical "punched-out" syphilitic ulcer or else a deep ragged ulcer with overhanging edges. In addition to the tertiary ulcer originating from a gumma, an obliterative endarteritis due to syphilis in its late stage may, by interfering with the nutrition of a part, predispose to ulcer formation.

Tuberculosis.—Ulcerative lesions due to infection with the tubercle bacillus are common both in the skin and in mucous membranes. In the skin they may be due to direct infection by the tubercle bacillus and they then take one of three forms: tuberculosis cutis, which is due to a local infection, but is part of a general tuberculosis; tuberculosis verrucosa cutis, which is frequently spoken of as anatomical tubercles or post-mortem warts, and is usually found in dead-house attendants, butchers, and those who have to do with animals; and lupus vulgaris. The fourth and most common form of tuberculous ulcer of the skin, known as the scrofuloderm, is due to indirect infection of the skin. It occurs usually in young subjects, most frequently in the neck, or over some group of lymphatic glands, or over a joint which has been the seat of tuberculous inflammation. Suppuration occurs in the infected part and the pus is discharged through a sinus leading to the surface, where a typical tuberculous ulcer develops. The same form of ulcer may develop from a deposit of tuberculous material in the subcutaneous tissues, quite independently of any gland or bone involvement, the tuberculous tissue breaking down and involving the overlying skin. There is still another ulcerative skin lesion which has been attributed to tuberculosis, but its exact etiology is still a matter of dispute; I refer to erythema induratum or Bazin's disease. Thibierge and Rayaut,[13] Colcot Fox,[14] and Philipson[15] believe it to be of tuberculous origin. Tenneson,[16] Leredde,[17] Audry,[18] and Johnson[19] came to the opposite conclusion. Whitfield[20] suggests that there are two diseases included under the same heading. On the mucous membranes tuberculous ulcers may occur as a primary lesion, but most frequently they are secondary to tuberculosis in some other part of the body, usually the lungs.

Other Forms of Specific Infection.—There are, besides tuberculosis and syphilis, several other forms of ulcerative lesions of the skin and mucous membranes due to specific infection. Glanders or farcy due to the bacillus mallei may be contracted by human beings from horses or mules. Soft nodules form in the mucous membranes (usually of the nose) or in the skin, and these rapidly break down, leaving ragged ulcers. The lesions of actinomycosis, or "lumpy jaw," due to the ray fungus, usually at an early stage become infected with pyogenic bacteria, suppurate and form abscesses which discharge by fistulæ, and if close to the surface form ulcers. A form of chronic perforating ulcer of the foot, known as "Madura foot," is caused by a fungus closely resembling the actinomyces. Leprosy due to the lepra bacillus, discovered by Hansen in 1874, is also an ulcerative disease, the ulcers being due to the breaking down of the leprous tubercles or to trophic nerve changes, or else they are caused by the fact that the impaired sensation prevents recognition of an injury.

There are many other specific infections which result in ulceration of the mucous membranes. Thus, for example, ulceration may be produced by the typhoid bacillus, the diphtheria bacillus, the amœba coli, the dysenteric bacillus of Shiga,[21] and, as has been recently reported by Haller,[22] the malarial plasmodium. (For further details with regard to these the reader is referred to the special articles on these subjects.) Two of the forms of intestinal ulceration are, however, of especial interest from an etiological point of view: the ulcers of the duodenum secondary to cutaneous burns and those described by Mathieu and Roux,[23] which are due to uræmia.

Skin Diseases.—Certain forms of skin diseases, such as herpes, especially when it occurs on the genitals, ecthyma, and pemphigus, where infection of the primary lesion occurs, and there is a lack of cleanliness, may result in the formation of ulcers. This is particularly so in eczema, where the inflamed, thickened, serum-infiltrated skin is easily abraded and apt to break down and form ulcers. The ruptured vesicles of eczema also make an excellent point for entrance of infection, and when eczema accompanies varicose veins of the leg, it is often very difficult to prevent ulcer formation.

Action of Drugs.—Besides the local escharotic effects of certain drugs, as an etiological factor in producing ulcers, some drugs produce ulceration of the mucous membranes by their elimination. Mercury by its elimination by the salivary glands may cause an ulcerative stomatitis; by elimination in the colon, an ulcerative colitis. Phosphorus, by causing a necrosis of the maxilla, may result in ulceration of the buccal mucous membrane.

Malignant Disease.—Primary new growths of the skin usually take the form of epithelioma, which may develop on any surface of the body where there is squamous epithelium. Nearly three-fourths of these neoplasms occur on the face and lips. Rodent ulcer, a form of epithelioma, nearly always is found on the upper half of the face. These lesions are ulcerative from the beginning. Sarcoma of the skin—viz., the melanotic form—does occur, but it is rare in comparison. Carcinoma and sarcoma may also produce ulceration by secondary involvement, as when by rapid growth and subsequent breaking down of growths lying close to the skin, the latter is destroyed, and an ulcer is established. In addition to these primary and secondary ulcerating neoplasms a chronic ulcer in an old person may undergo epitheliomatous or, more rarely, sarcomatous changes and become malignant.

Etiology of Regional Ulcers.—In addition to the varieties of ulcer already spoken of, we may mention here briefly the various forms of ulcerative stomatitis due to traumatism, infection, gastritis, and sprue, as well as to mercurial poisoning. Ulceration of the rectum also deserves to be mentioned here. Although it may be due to several causes, in a large number of instances it is due to tertiary syphilis, and especially to the breaking down of gummata. Tuberculosis, either primary or secondary to tuberculous deposits in other parts of the body, dysentery, acute proctitis, traumatism from hardened fæces or foreign bodies in the fæces, pressure from a polypus or from a displaced uterus in women—all these may be causes of ulceration of the rectum.

Fissures, or those forms of ulcer which occur at the junction of the skin and mucous membrane of the nose, mouth, or anus, and on the nipples of nursing women, may also be considered under this heading. Fissures at the angle of the mouth, due to tuberculosis, have been already mentioned under the name of tuberculosis cutis. In infants suffering from hereditary syphilis, fissures are frequently found about the mouth and anus. The usual

form of fissura in ano, a small, extremely painful linear ulcer, ordinarily found near the posterior commissure, is due to abrasion with hardened fæces or to infection after scratching, or to the breaking down of a herpetic vesicle. Ball considers anal fissure to be due to a broken-down anal valve. The lesion is prevented from healing by the movement of the sphincter at defecation and the turning in of the edges of the ulcer. Fissures of the nipples are due to infection following an abrasion or injury caused by the infant's nursing, and are prevented from healing by motion of the part.

Pathology.

The pathology will vary somewhat with the conditions causing the ulcer, although in the non-specific forms of ulcer we shall find the phenomena of congestion, exudative and necrotic inflammation, together with reparative inflammation or granulation, making up the whole process. In the stage of development of the ulcer the degenerative process predominates, in the healing stage the reparative.

When the ulcer develops from without, as when infection enters the skin through an abrasion, or through a wound of a different character, congestion first occurs. This is rapidly followed by the emigration of leucocytes, by a diapedesis of red blood cells which rapidly disintegrate, and by an exudation of serum and fibrin. At the same time there is a proliferation of the epithelial cells of the rete mucosum lying on the papillary layer of the corium, and also a proliferation of the connective-tissue cells of the corium. The tissues next become softened by the exudate between the cells. Then, as a result of the pressure of the exuded serum, of the crowding by the leucocytes, and of the cutting off of the blood supply, and also in some measure through the effects of the toxins furnished by the bacteria, there occurs necrosis or death of the cells, which are thrown off from the surface with the products of exudation, until there is formed an ulcer with its base consisting of spheroidal and a few epithelioid cells developed from the connective-tissue cells by proliferation.

The process of molecular gangrene just described does not represent the only way in which an ulcer may form; it may develop from a blister, by the sloughing away of an area of localized gangrene, from an infected wound with loss of substance, by the destruction of the skin over a superficial abscess, or by pressure from within. After its formation it may heal, continue to spread, or become chronic.

If an ulcer in its complete stage of development be examined, it will show the following pathological condition: The surface is covered with a layer formed by the overproduction of new round cells, together with the exudate of serum, fibrin, and the cellular elements of the blood. When the discharge from the ulcer is profuse, this may be constantly washed away. When the ulcer is sluggish, it may be in a condition of coagulation necrosis. In this condition a croupous material covers the base of the ulcer, and below this is a more or less distinct layer largely composed of cellular elements, with very little intercellular substance, the cells being spheroidal and epithelioid in character and mingled with polynuclear leucocytes. As we go deeper, the amount of intercellular substance increases, and we find a number of transparent fibres and fusiform cells. In this layer of granulation tissue are also the newly formed blood-vessels, the most superficial branches being vertical to the surface, and developing by a process of budding from the endothelial cells of the capillaries deeper down. This layer is paler in color than the layer made up of the cellular elements, but may contain pigment from the disintegration of the red blood cells. It gradually merges into a layer of cicatricial connective tissue which lies beneath the ulcer. A section of the edge of a chronic ulcer would show an enlargement and prolongation of the papillæ, with a marked proliferation of the epithelial cells covering them. This is most marked in the condition known as callous ulcer, where the edges may by this proliferation be considerably raised above the level of the surrounding skin, and often overhang the base of the ulcer, as if nature were trying to bridge it over.

Under proper treatment the reparative process proceeds faster than the degeneration of the cells and the ulcer begins to heal by granulation. Small sprouts or buds of protoplasm protrude from the capillaries below or in the base of the ulcer, developing from the cells in their walls. These are hollowed out by the blood pressure and form new blood-vessels which anastomose with others. Nuclei form in the protoplasm and thus endothelial cells develop. At the same time small spheroidal cells developing from the connective-tissue cells become grouped around the blood-vessels. These at first are closely crowded together, being only separated by a small amount of fluid intercellular substance. Some of the round cells then become larger and fusiform or branched. The larger cells are known as epithelioid cells. Some of the fusiform and branched cells, called fibroblasts, develop the new delicate fibrillar intercellular substance, while others form the connective-tissue cells. Gradually the fibrous intercellular substance increases in amount, while the cells become fewer and flattened, and cicatricial tissue is formed. The contraction of this cicatricial tissue constitutes an important element in the healing of an ulcer.

During the process of granulation, more of the round cells are produced than is necessary. These die and are thrown off in the discharge. Healthy granulations should be small, even, and of a reddish-pink color. Where the growth of the blood-vessels proceeds more rapidly than the development of the cells and the formation of connective tissue there is produced a soft, pale, flabby condition known as exuberant granulations. On the other hand, both the cells and the blood-vessels may develop very slowly, forming indolent or sluggish granulations.

In order that the ulcer may heal it must eventually become covered with epithelium, and this (unless grafting is practised) can only develop from the epithelium at the edges of the ulcer. Under favorable conditions, when the granulations reach the level of the surrounding skin, the epithelium begins to spread in a thin bluish-white line from the edges, out over the surface, until it is entirely covered, when the ulcer is healed. The rapidity of this process differs in different cases. Deutsch[24] measured one hundred healing ulcers, exclusive of traumatic cases, and found the average growth of epithelium to be from 2 to 3.5 mm. per week. The influence of contraction of the base of the ulcer, however, must also be taken into account.

The scar left by the healing of an ulcer is always considerably smaller than the original lesion, due to contraction of the cicatricial tissue, and this contraction, especially when the ulcer has occurred on the face, may lead to considerable deformity. The healed surface differs from normal skin in the absence of hair follicles and of sebaceous and sweat glands. In the mucous membranes, on the other hand, regeneration to a condition more nearly approaching normal occurs.

Modifications of the Course in Special Forms of Ulcer.—The pathological condition which has been described above applies to the simple forms of ulcer; but it practically forms the basis of all cases of non-specific and non-malignant ulcer with certain modifications due to the conditions present and to the etiology. These pathological changes are found not only in the ulcer itself, but also in the adjacent tissues. When an ulcer becomes acutely *inflamed*, a condition of cellulitis exists in its base and edges and in the tissues surrounding it. In a *spreading* ulcer there is little or no attempt at a reparative process, while degeneration and death of the cells proceeds rapidly. This may occur to such an extent in *phagedenic* or *sloughing* ulcers that we find, in addition to a process of molecular gangrene, considerable areas of necrotic tissue in the base and edges of the ulcer. In certain chronic cases there may be very little or no formation of granulation tissue, and the base would then be formed by the underlying body tissues instead of the layer of round cells already described. This condition is known as *raw* ulcer. In other cases the base may be smooth and shining like mucous membrane, or may be covered with a *croupous* layer formed of the material exuded. In *scorbutic* ulcers the base is covered with an adherent spongy, fœtid, dark-colored crust, and bleeds very easily.

In the case of *varicose* ulcers there is usually more or less pigmentation of the surrounding skin. Often œdema of the skin and subcutaneous tissues, and in many cases eczema, is present. When the latter occurs there is apt to be more than one ulcer. The veins are dilated, thickened, and tortuous, and show marked insufficiency of their valves. These changes are more marked in the superficial than in the deep veins, and cases vary as to whether the dilatation of the larger veins or that of the smaller radicles forms the more prominent feature. The walls of the veins become the seat of a phlebitis, which may be suppurative in nature, producing a periphlebitis, and thus frequently, by involvement of the skin, causing the formation of a new ulcer. The arteries may show an endarteritis. The nerves, as previously mentioned in connection with the etiology, show the signs of chronic neuritis. In old, chronic cases the connective tissue, muscles, and even the underlying periosteum and bone may be affected by a chronic productive inflammation.

Syphilis.—The pathology of syphilitic ulcers differs from that of the cases previously described, in that the ulcerative process is preceded by a productive one. In the chancre we have first an infiltration of the connective tissue at the site of the lesion with small spheroidal cells and an occasional giant cell, together with a proliferation of the connective-tissue cells. This breaks down and ulcerates at the surface. The superficial ulcers of the late secondary or of the tertiary stage are preceded by a round-cell infiltration, which may break down at once and form a pustular lesion from which an ulcer develops, or one or more tubercles may form which then break down. It is by the spreading and marginal growth of one of these tubercles or the progressive involvement of others, while healing is going on at one part of the ulcer, that the serpiginous ulcer forms. The deep ulcers of tertiary syphilis develop from gummata. These are variously sized deposits, largely made up of small spheroidal cells with some polyhedral and occasional giant cells. They are poorly supplied with blood-vessels and undergo coagulation necrosis, but do not tend to suppurate until infected. Sooner or later the overlying skin becomes involved, either with or without a secondary pyogenic infection, and the gumma then sloughs out, leaving the typical syphilitic ulcer. A bacillus, which has been described by Lustgarten,[25] and which is very similar to the tubercle bacillus, is found in small numbers in the lesions. Lustgarten was unable to cultivate it artificially. Van Niessen,[26, 27] however, succeeded in cultivating from the blood of syphilitics and from the discharge from syphilitic condylomata a bacillus which when inoculated on monkeys developed a primary lesion and general glandular enlargement similar to those seen in man.

Tuberculosis.—The gross pathological condition of ulcers due to tuberculous infection varies greatly, depending on the variety of the lesion. It may take the form of discrete, shallow ulcers as in tuberculosis cutis, or that of the ulcerative warty lesion of tuberculosis verrucosa cutis. Lupus vulgaris, characterized by its brownish-red, semi-translucent, smooth, shiny, apple-jelly-like tubercles, also due to tuberculosis, may form an ulcerative lesion, but the non-ulcerative form is more common in this country. Erythema induratum begins as a varying-sized nodule under the skin which subsequently breaks down. The forms mentioned above, together with the irregular ulcer with livid overhanging edges, known as the scrofuloderm, the origin of which has already been mentioned, show how great a variety exists in the gross pathology of tuberculous ulcerative lesions. The minute pathological examination, however, shows all of these forms to be dependent on the formation and subsequent breaking down of tubercle tissue. The latter may be localized, forming miliary tubercles, or it may be diffuse. The tubercle bacillus and its toxins cause a productive inflammation, with proliferation of the connective-tissue cells, the tubercle tissue thus being made up of small spheroidal or polyhedral cells, or of the latter together with a fibrous stroma and cells resembling leucocytes, or of giant cells. At times the tubercle tissue very closely resembles granulation tissue. There is very little production of new blood-vessels, and the tubercle may undergo coagulation necrosis and caseous degeneration. The process may cease by encapsulation of the lesion with fibrous tissue, but the most frequent course is that in which the lesion breaks down, either as a result of secondary infection or directly without the intervention of infection. The pus which is formed as a result of this breaking down finds its way to the surface, along the course of least resistence, and gives rise to the scrofulo-derm.

Neoplasms—Malignant Degeneration.—When an ulcer occurs as a result of a benign tumor, the cause being pressure, the pathological process is similar to that of the simpler forms of ulceration, although in some cases, as for example in cystadenoma of the breast, the rapid growth of the tumor may cause the tissue of the new growth to form the base of the ulcer. When, however, the ulcer is a result of malignant new growth, whether primary or secondary, the ulcerative process is due to a breaking down of the cells forming the new growth and an involvement of the adjacent structures with cancer cells. There may also be more or less production of granulation tissue. Primary malignant ulcers may be due to sarcoma, but far more frequently they are of the epithelial type—carcinoma, epithelioma, or rodent ulcer. Both sarcoma and carcinoma may secondarily involve the skin and produce ulceration. The former has a markedly less tendency to do so than the latter, but sarcomatous ulceration, when it does occur, is apt to take on the characters of the fungating variety. In all of these malignant ulcers the base and edges are made up of the cells of the neoplasm. For the minute pathology the reader is referred to the articles on *Carcinoma*, *Sarcoma*, and *Epithelioma of the Skin*.

Old chronic ulcers, especially in elderly patients, may undergo epitheliomatous degeneration and become malignant. This process, when it occurs, begins in the edge of the ulcer, taking its origin in the epithelial layer of the skin, and by its growth both base and edges of the ulcer become formed by the malignant epitheliomatous tissue, and the appearance then presented is that of a foul, hard, warty, easily bleeding ulcer. Sarcomatous degeneration of a chronic ulcer has also been reported. Here the new growth takes its origin in the connective-tissue cells instead of the epithelium.

Symptoms and Course.

Non-specific Ulcers.—The appearance and symptoms of the various non-specific ulcers will vary with the etiology of the ulcer and the pathological condition present at the time of examination. These differences will manifest themselves in the shape and size of the ulcer, in the appearance of the base and edges, in the character of the discharge, and in the condition of the surrounding tissues.

Acute Traumatic Ulcer.—When an ulcer is formed as a result of infection of an area where the skin has been destroyed by some form of traumatism, the symptoms are those of an acute inflammatory condition. The base of the ulcer is usually only slightly depressed, and is of a gray or dirty-yellow color, or sloughing in appearance. There are few or no granulations, the edges are clean-cut, soft, and inflamed, the surrounding tissue is red and congested, and the discharge is thin, sero-purulent, or blood-stained and containing débris. The shape of the ulcer will vary with the nature of the traumatism. These cases are accompanied with more or less pain. Such an ulcer may begin to granulate and heal, may spread or become chronic; and these conditions, which are more or