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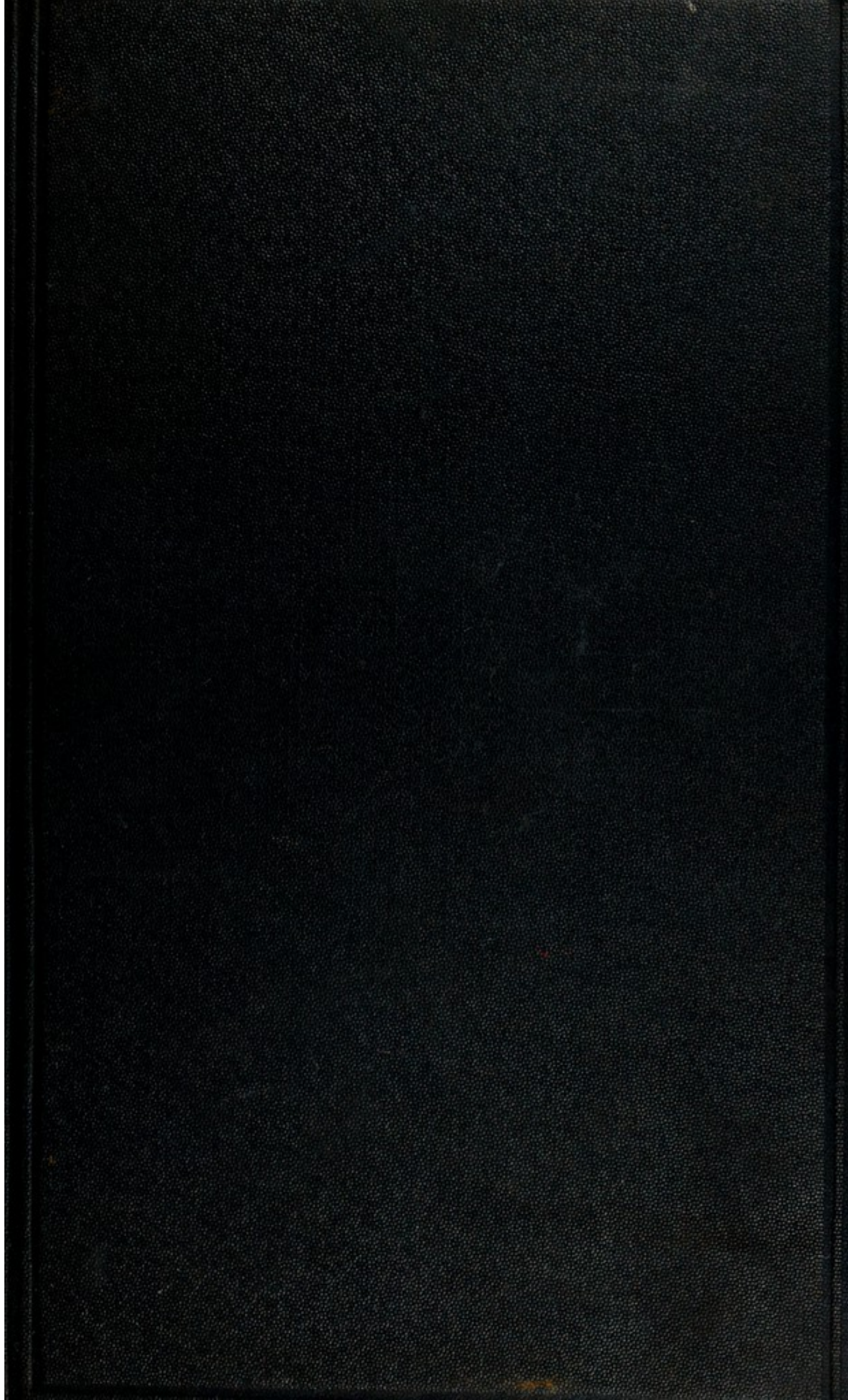## Persistent URL

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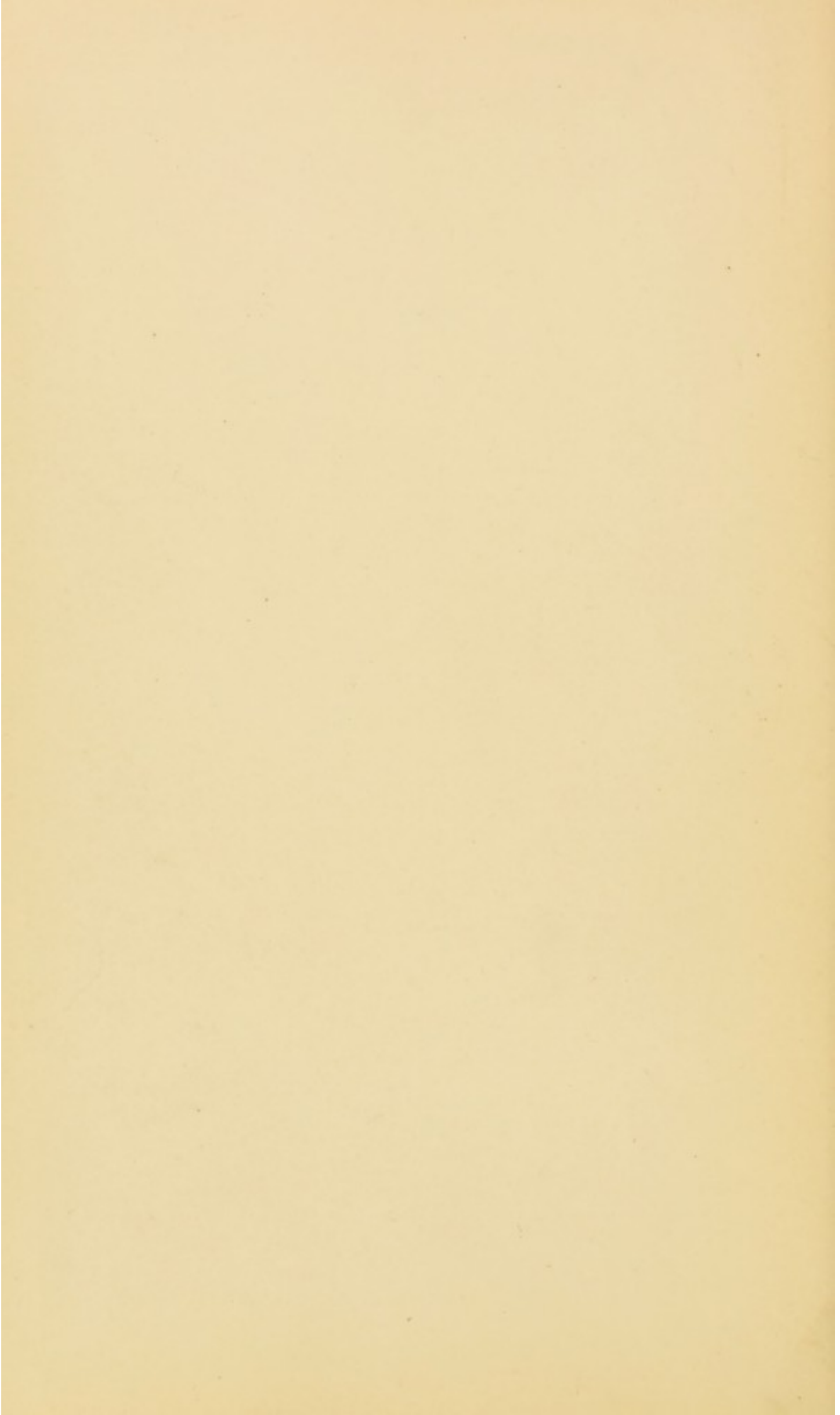
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LECTURES

UPON THE

PRINCIPLES OF SURGERY

DELIVERED AT THE UNIVERSITY OF MICHIGAN

BY

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American Academy of Medicine; Late Major
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WITH AN

APPENDIX

CONTAINING A RÉSUMÉ OF THE PRINCIPAL VIEWS HELD CONCERNING INFLAMMATION

BY

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ILLUSTRATED

Copyright, 1899,

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TO

PHINEAS SANBORN CONNER, M.D., LL.D.

Professor of Surgery in the Medical College of Ohio and in Dartmouth Medical College

THAT EMINENT SURGEON AND ELOQUENT TEACHER OF SURGERY BY
WHOSE KINDNESS THE AUTHOR WAS AFFORDED AN OP-
PORTUNITY OF TEACHING THOSE PRINCIPLES OF
HIS ART WHICH HE HAS STRIVEN TO
ELUCIDATE IN THESE LECTURES

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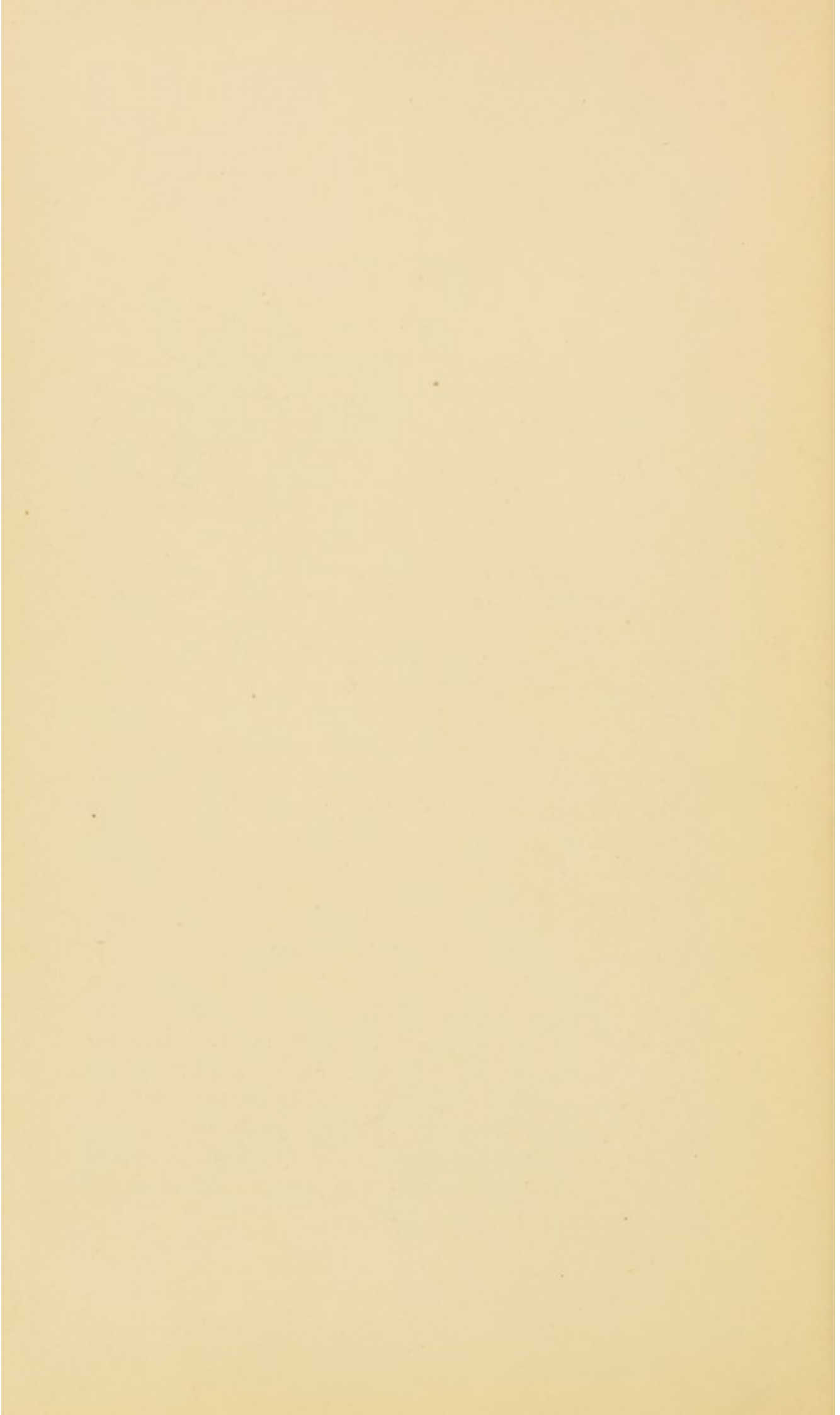
IS

GRATEFULLY DEDICATED

BY HIS FRIEND

THE AUTHOR

PREFACE.

Although many excellent works have been written treating of the Principles of Surgery, the attempt to render them too comprehensive has marred their usefulness for the undergraduate, or undue prominence has been given to the author's special methods of applying principles to practice. It must be manifest that it is impossible in the compass of a few hundreds of pages to give adequately all the essentials of surgical pathology, bacteriology, and the more or less obsolete teachings of the past, when each of these subjects requires a larger volume than any work extant on the Principles of Surgery ; yet this has been attempted by many writers.

Modern specialism in the teaching of medical science has recognized the necessity of, and has provided for, specific instruction in pathology, bacteriology, etc., so that anything beyond a mere reference to the peculiarities of form and growth of various germs, or the changes undergone during diverse pathological processes, would be a useless repetition, occupying time more properly belonging to new studies.

In the hope that some of these objections have been met, the author submits these lectures, trusting that they may be of assistance to students and, in some measure, to teachers of surgery. The author has reluctantly bowed to the steadily increasing custom of dropping the hyphen in many compound words and altering the spelling of familiar terms. All illustrations, unless otherwise indicated, are the product of the skilful pencil of his assistant, Dr. Spitzley.

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LECTURE I.

THE NECESSITY OF A KNOWLEDGE OF THE PRIN-CIPLES OF SURGERY FOR SUCCESSFUL PRACTICE OF THE ART; HYPEREMIA.

THE *principles of surgery* are those laws formulated from the combined results of experience and experiment, which when applied to special cases are found to explain the phenomena of disease and indicate the general measures best calculated to combat morbid conditions.

Because certain of the principles or laws tend to nullify one another the resultant phenomena actually observed when the former are operative seem so totally dissimilar from what should logically be expected, that they apparently disprove the truth of the alleged principles until closely scrutinized, when, if two laws are concerned, a third principle will be clearly distinguishable, which, like the resultant of two physical forces, is the diagonal —a mean as it were—between the two. When the application of many apparently conflicting principles is clearly demanded for the explanation of a pathologic condition or to warrant the enunciation of a therapeutic law, the ultimate principle may be difficult of detection. Still, the truth of every composite law can be demonstrated by careful study, because the resultant principle applicable to any given case will be found either logically, experimentally, or clinically, or often by all three methods combined, to be the result of the mutual modifications effected by the conflicting laws, and the final composite law would be incapable of proof by any of the methods applicable if any of the laws of which it is the result were false. In the present state of science our knowledge

is only partial ; hence from time to time surgical princi-
ples require revision, as advances reveal the imperfection
of the data upon which they are founded.

Although this might seem the most appropriate place to
give an illustration of the method of reconciling scientific
principles with their apparent contradictions by one
another, or by the results of their attempted practical
application so that both science and practice would profit,
yet as this presupposes information not yet given, and
would entail later repetition, any illustration must be
reserved for the chapter on the Therapeusis of Inflamma-
tion. The skill of the practitioner—*i. e.*, he who applies
principles to practice—consists in justly appreciating the
modifying influences one pathologic law must or may
exert upon another in individual cases, and the general
therapeutic principles best calculated to remove all the
morbid conditions. If this be impossible, as is the rule,
he must strive to recognize which is the most serious of
the morbid conditions and endeavor to eliminate that.
When neither of these desiderata is attainable, he must
single out that one, or those which are under the control
of his art and remove it or them, for much that is de-
sirable in theory is not feasible in practice.

For example, take a moderate-sized carbuncle. Before
the overlying skin has either lost its vitality or become
seriously infected, after preliminary freezing to render
the diseased tissues friable, an incision can be made,
all the diseased tissues be curetted or dissected away,
disinfection be effected, and the skin being sutured over
a drainage-tube, the cure will progress in many cases
as if only an aseptic wound with loss of substance was
concerned, because all the morbid conditions have been
removed—viz., the germs, their products, and the com-
pletely or partially devitalized and infected tissues. In
an acute abscess, however, all the morbid conditions
cannot be directly removed by art. The virulent infec-
tion ends in death of the cells at the focus and their con-
version into pus, and the intense surrounding hyperemia

results in such an outpouring of exudates that the still living tissues have their nutriment mechanically diminished, producing a lowering of vitality which renders them an easy prey to the multiplying germs. The pressure under which the pus exists forces into the surrounding tissues toxic substances destructive to the cells or so lowering their vitality that fresh soil for new crops of micro-organisms is prepared. Clearly, then, evacuation of the pus will remove only a portion of the germs and their toxic products, but the relief from pressure also effected will prevent the dissemination of toxins, and will relieve the strangulation of the tissues which prevents proper nutrition, thus enabling them to cope with the germs left behind. Thus, although the germs in the still living tissues—*i. e.*, the chief morbid condition—cannot directly be attacked, the evacuation of many of the germs contained in the pus with their toxic products and the relief of tension will remove many of their worst effects, and enable the tissues not only to protect themselves against further invasion, but also to destroy those germs already present.

Inflammation is the topic usually first considered when lecturing upon the principles of surgery, because it is the most common foe the surgeon has to contend against in some form and at some stage of the majority of the ailments for which he is consulted. Moreover, inflammation may complicate any wound, operative or accidental, and frustrate the best directed efforts of operative skill. I shall not pursue the usual plan because I believe that inflammation, in any true sense, is not a necessary process occurring during the repair of injuries. I believe and trust that I shall demonstrate that there is only one process of repair, which, when progressing undisturbed, has been mistermed "*aseptic inflammation.*" When microbic invasion has been successful, certain modifications of the processes of repair occur owing to interference with the normal processes, which modifications all confess to be inflammatory and are essentially destructive.

The withdrawal of the perturbing influences, whether this be effected by an actual physical removal of the germs, an acquired immunity to them and their toxic products, or a vital obstacle, formed by the erection of a barrier of sound granulations, prevent their entrance into the tissues matters not. When the disturbance is removed, the original reparative processes are promptly resumed just where they were interrupted, and they proceed in all essentials exactly as if nothing abnormal had occurred. Hence there is no longer room for such terms as "aseptic" and "infective inflammation," but only for "repair" and "inflammation," because the latter is always due to the action of germs or their products. While it is true that in the laboratory certain aseptic irritant substances may, under specially favorable circumstances, cause the production of a pyoid fluid, this is not true pus, either microscopically or clinically, and can with impunity be injected into other animals without producing any of the results following the injection of pus, provided the injected material either contains none of the original irritant, or this in a much diluted form. Still further, clinically such conditions never confront the surgeon as are possible to the laboratory experimenter.

The position that I assume is, I contend, the only logical as well as scientific one—viz., that inflammation is never anything but an infective process, yet pyogenic germs do not necessarily mean pus. What is seen during normal repair should not be termed inflammation, and while at certain stages presenting clinical and histological phenomena similar to those seen in inflammation, they are not identical processes due to the same cause. Up to a certain point some of the phenomena are indeed due to the same conditions brought about by analogous causes. Others are similar conditions brought about by different causes, and because the conditions are the same, the blunder of assigning them one causation has led to much confusion. I, in common with many other teachers,

have felt that our theories and statements did not hang together ; but the trammels of custom and the dif- ficulty of overcoming the dead weight of authority have hitherto prevented us from stating certain facts frankly.

If the microbic causation of inflammation be true, many of the statements usually made are irreconcilable with this theory, and the germ theory is in fact ex- ploded, and should be abandoned. If it is a fact, not a theory, that microbes are the real cause of inflammation, let us be consistent in our teachings and honestly true to our theory in all—not a few—of its details.

Hyperemia.—Before beginning to study the proc- esses of repair I must pass rapidly in review certain facts relative to " hyperemia " or " congestion."

A study of the phenomena of both acute and chronic hyperemia will demonstrate that, by direct or indirect lowering of the vitality of the tissues, these conditions predispose to the localization and efficient action of pathogenic organisms. Close scrutiny will show that these conditions are predisposing, never efficient, causes of inflammation. It will become possible next to recon- cile the conflicting statements that germs are the sole cause of inflammation, and the demonstrable fact that conditions resulting in marked permanent tissue-changes exist, closely resembling many of those seen during or after genuine—*i. e.*, microbic—inflammations, which yet are *not* due to microbic action. Again, the otherwise inexplicable fact that certain so-called chronic inflam- mations will persist for years, producing mere " hyper- plasia " of some of the tissue-elements, while other cases apparently identical in nature will rather suddenly term- inate in suppuration, are readily explainable by a study of the phenomena of the two conditions mentioned, when viewed in the light of modern bacteriologic knowl- edge. This study of hyperemia is an imperative duty, if for no other reason than because the reaction of tissues to injury during the processes of repair and the primary

symptoms of microbic action present, for a time, the same appearances.

The *arrangement of the vasomotor system* of nerves must first be passed in review. Starting from the center located near the calamus scriptorius two courses are pursued by the nerve-fibers. The majority leave the spinal nerves with the rami communicantes, passing upward and downward to merge with the sympathetic or splanchnic nerves. Some branches return from the sympathetic to the spinal nerves by the communicating branches, to supply the skin, muscles, and bones. A minority of the filaments form an integral part of the spinal nerves without any connection with the sympathetic system of nerves, forming the direct vasomotor supply, while the first set of nerves constitute the indirect vasomotor nerves, as they may be termed. Certain experimental facts, for the details of which physiological text-books must be consulted, prove that branches passing out with the sensory (posterior) roots of the spinal nerves, when irritated, cause vascular dilatation, either by their direct action or by an inhibitory action on the peripheral vasomotor apparatus. The remaining branches are vasoconstrictors. The existence of a peripheral vasomotor apparatus cannot be ignored, that is to say, the presence of perivascular ganglia with a communicating plexus of nerve-fibrils.

The vasoconstrictor nerves and the perivascular mechanism are believed to be "continuous in action," while the dilators act "exceptionally."

A restatement of the facts just related, in a different order and manner, will enable us to draw certain conclusions which will explain important clinical facts often imperfectly understood. If the vasodilators pass out from the spinal cord through the sensory roots of the spinal nerves, by irritation of the dilator nerves a hyperemia should result, which would produce pain in the area supplied by the spinal nerve having its origin in the irritated root or roots, while stimulation of the

periphery of the sensory nerve should produce dilatation of the vessels in the area supplied by this nerve.

What is the clinical answer to this theoretical conclusion? Quite often in trifacial neuralgias—the result of hyperemic conditions of the sensory root—a congested conjunctiva, lacrimation, salivation, edema, even desquamation of the epithelium has been noted. A still better illustration is that form of herpes zoster resulting from congestion of the posterior spinal nerve-roots or their ganglia, where pain thus produced is followed by nutritional changes in the skin, reaching even vesication. In turn, the early pain of inflammation is in part due to the congestion of the posterior nerve-roots resulting from the peripheral irritation of the sensory nerve. With a vasomotor apparatus as described, irritation of a sensory nerve of sufficient intensity to reach the nerve-center should be capable of propagation to other centers, exciting their action so that the circulation of a more or less distant part shall be modified.

What have experiment and clinical observation to say for or against this deduction? Lombard and Brown-Séquard have demonstrated that irritation of the skin-nerves by pricking with a pin causes elevation of temperature on the side of irritation, while the temperature is lowered upon the opposite side of the body. Again, immersion of one hand in cold water will produce lowering of the temperature of the other hand. Clinically this reflex vasomotor action is made use of because demonstrably efficient when the moderate cold of an ice-bag to the head produces diminution of the smaller vessels within the cranium, when enlarged during intra-cranial inflammation. I know well that the presence of vasomotor nerves to the cerebral vessels has been denied, but G. C. Huber's experiments unquestionably prove their existence, as I have personally seen under the microscope. Every resort to the common expedient of applying cold to the nape of the neck or over the forehead to arrest nose-bleed is founded, unconsciously

usually, upon the existence of this reflex action produced by an impression upon a sensory nerve.

What phenomena should manifest themselves in vaso-motor paralysis, including perhaps also the suspension of the activity of the perivascular ganglia? The arterioles and capillaries should dilate to the maximum extent so that arterial blood, or at least the cardiac impulse, will probably reach the veins.

After division of the sciatic nerve in the dog, increased tension can be demonstrated to exist in the corresponding femoral vein, which, if tied, shows a pseudo-arterial pulsation; the smallest arterioles, if compressed, can also be felt to pulsate, while the veins contain blood of an arterial hue. Microscopically, dilatation of the capillaries is manifest. Clinically, all of these phenomena are observable after the application of an Esmarch bandage, or an irritating, not destructive lesion of a nerve of an extremity. Finally, should the part be cut off from the vasomotor center, leaving the perivascular apparatus intact, would it not be reasonable to expect that after an inevitable temporary dilatation of the vessels, a more or less perfect readjustment would take place, in virtue of the increased power acquired in the course of time by the perivascular apparatus, provided no extraneous influences intervened? Assuredly; and this is seen in the course of time in the transplanted flaps of a plastic operation after division of their pedicles, and in the rabbit's ear after section of the cervical sympathetic. Clinically it is well known that in a limb whose main nerves have been divided, or in the area supplied by a divided nerve, although the local vasomotor apparatus is equal eventually to the regulation of the circulation under favorable circumstances, the most trivial changes in temperature may produce the most intense hyperemia, leading perhaps to vesication, so that immersion in cold water causes a neuroparalytic congestion resulting in almost identical appearances with those of a scald severe enough to produce isolated vesication.

LECTURE II.

HYPEREMIA (CONTINUED); MICROSCOPIC APPEAR-
ANCES; EXUDATES; RESULTS; TREATMENT OF
HYPEREMIA.

REGARDED from the clinical standpoint, hyperemia or
congestion means excess of blood in a part. If this
results from a too free access of arterial blood to the
parts the resultant hyperemia is termed "active hyper-
emia." When the exit of venous blood is interfered
with, the congestion which follows is appropriately called
"passive hyperemia." The element of the time con-
sumed before the phenomena reach their acme also partly
determines the application of these terms. Thus, in an
acute hyperemia all the phenomena rapidly supervene,
the process soon presenting a sharply defined condi-
tion, each contributing element promptly reaching the
highest point attainable, from which retrocession must
follow, or a different set of phenomena must result.
Acute hyperemias are usually excited by decided "me-
chanical disturbances," and the essential causative fac-
tors must be, directly or indirectly, an increased vis-a-
tergo of the circulation and such an altered condition of
the part as will permit the accommodation of an increased
bulk of blood.

On the other hand, chronic hyperemias are of slower
development and commonly result from persistent, slight
irritations, aided nearly always by mechanical inter-
ference with the exit of venous blood, although this
latter condition is not always easy of detection. *Ischemia*
is another term the significance of which should be
understood. It means an abnormally small amount of

blood in a part, or a reduced supply to a part, while sometimes it is incorrectly made to include the idea that whatever blood is present in, or supplied to, the part is abnormal *in quality*.

From what has been said, **active hyperemia** results from disturbances of balance of the vasomotor system of nerves and may be "*neuroparalytic*," from paralysis of the vasoconstrictor fibers, as in blushing, after division of the cervical sympathetic, injury of a nerve, as its partial division, or the pressure of an Esmarch bandage. While sometimes otherwise explainable, the congestions following the rapid removal of fluid by tapping the pleural or abdominal cavities, or the sudden withdrawal of urine from an overdistended bladder —this last sometimes producing death from the consequent congestion of the kidneys, causing suppression of urine—are "neuroparalytic," and the recognition of the mechanism of these congestions teaches us their therapeusis—viz., the avoidance of the removal of pressure from vessels whose constrictor nerves have been long unaccustomed to maintain, unaided, their normal caliber. This is one of the best illustrations of the advantages accruing from an acquaintance with minute, and apparently trivial, scientific facts. But the usual vasomotor balance existing by virtue of the equal action of the vasodilators and vasoconstrictors may be disturbed by irritation of the dilator fibers, the same excess of blood in the part—*i. e.*, congestion—resulting, not from paralysis but from active dilatation, a "*neurotonic*" congestion is induced. These hyperemias are more comprehensible, if it is remembered that they must be essentially reflex— *i. e.*, an afferent impression causing an efferent impulse. These reflex hyperemias are nearly always preceded by pain. This is due either to irritation of the peripheral branches of a sensory nerve, or more often to hyperemia of the sensory spinal roots caused by the action of the reflexly excited vasodilators. If the causes of a hyperemia are temporary, the effects are equally so; but

when the former are persistent, certain more permanent changes take place. The smaller arteries and arterioles may at first show acceleration of their current; but this condition is soon reversed, the blood moves more slowly, and upon purely physical principles the intravascular pressure is increased, this abnormal pressure being exerted upon vessel-walls thinned by distention, and less capable of restraining the escape of their contents, which normally pass out, though in less quantity, during the processes of nutrition. In consequence, the watery con-
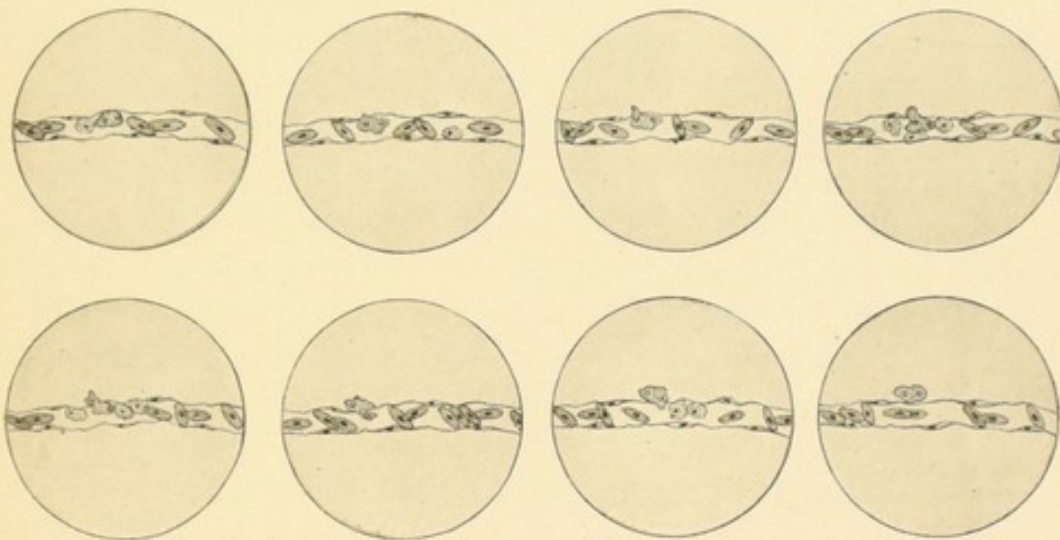
FIG. 1.—Showing diapedesis of a white blood-cell from a capillary. Observations at intervals of eight minutes.

stituents escape freely, producing an increase in bulk from the edema, as it is termed. When a cavity is concerned, such as a joint, this collection of serum is called an effusion, and is occasionally seen in an articulation after irritation or injury of one of the main nerve-trunks of a limb. The hyperemia persisting, the organized elements of the blood, such as the white corpuscles, begin to pass out by diapedesis through the walls of the venules and capillaries, still further increasing the bulk of the parts; even the red cells may also pass out into the tissues by either diapedesis or rhexis. The student should note well these symptoms, which are, of course, accompanied with heat and, as has been said, with pain,

for here are, as will be mentioned later again, the "calor," "rubor," "dolor," "tumor," and not uncommonly, as in the joint-effusion just described, the "functio laesa" —*i. e.*, the same symptoms which mark true (microbic) inflammation.

In marked contradistinction to this acute hyperemia is **passive hyperemia,** resulting from mechanical obstacles to the exit of blood, a diminished vis-a-tergo from feeble heart-action with or without loss of elasticity of the arterial walls, or a combination of both these factors. When obstruction of the main or only vein of a part is the cause of the hyperemia, serious consequences may follow, as gangrene. In passive hyperemia the parts instead of being too warm are colder than normal, of a bluish, cyanotic tint ; marked serous transudation takes place, with escape later of liquor sanguinis and the corpuscular blood-elements. When the process has been of long duration, the repeated escape of red cells into the tissues and the subsequent changes undergone by the hemoglobin produce marked discolorations—*i. e.*, pigmentation of the integument or other deeper parts.

The **results of hyperemia** vary according to the extent and duration of the hyperemia. Thus, in the acute form it may promptly disappear when the cause is removed, as a foreign body from the eye ; this corresponds, in many instances, to the "*delitescence*" of the older writers. Persisting longer it produces an increase in the bulk of the parts due to effusion of serum into the interstices of the tissues—*i. e.*, edema occurs—or where a cavity is concerned, the fluid accumulating in this forms an effusion. If it lasts still longer, the corpuscular elements escape and the tissue-cells are stimulated to division, until a considerable cellular exudate is formed, which if vascularized will develop into a permanent tissue—viz., scar-tissue. As only fluid exudate can be quickly removed by the lymphatics or by absorption by the bloodvessels, if many cells be present, a much more complex and longer set of processes is requisite than the former,

which chiefly entails the return of the vessels to a normal caliber. A term indicating a change preceding absorption has long been employed—viz., **resolution** (*re-solutio*). Although this process may, and often does, occur in its more marked forms after a mild infective process, yet many cases of resolution of inflammation really mean a subsidence of an acute hyperemia which has resulted in considerable cell-exudate.

The sudden accumulation of blood, fluid exudate, and such cells as may migrate, resulting from a rapidly developed hyperemia in parts whose blood-supply is damaged, especially if it normally be poor, will so reduce the remaining blood-supply that cellular or more massive death of the tissues often occurs, that is to say, **gangrene** results. This is because the veins and lymphatics cannot drain away the exudates which compress the plasma-channels by which nutriment reaches the cells, or still further, compresses the veins, interfering with the return circulation so that stasis occurs, preventing all access of arterial blood ; hence the death of the tissues. The active forms of hyperemia more often cause *ulceration* or *sloughing* than what is termed gangrene, but the essential processes are the same in both. Passive hyperemia is a more common cause of the massive death we call gangrene and for the following reasons. Death of the cells composing a part means death of the part. Death of a cell must result either from physical disintegration, chemical change, or starvation. Death of cells, or small masses of them may, and often does, follow from cutting off of nutriment by mechanical pressure, from what is called "strangulation" of tissue ; but in "passive hyperemia" the exit of venous blood being in a great measure prevented, the arterial supply is first diminished, then more or less completely arrested by a combination of pressure in the venous system and exudation into and increased solidarity of the tissues, until death of all the cells of a whole limb follows from starvation of cells, aided per-

haps by the effects of substances elaborated by the cells
and retained in them, the venous blood, or the exudates.
Pressure-sores, although often ascribed solely to passive
hyperemia more often are due to a primary ischemia of
the part, followed by a passive hyperemia from vascular
paresis. Thus the cells are first directly starved by
mechanical pressure preventing access of nutrition, and
later, indirectly, by the exclusion of arterial blood by
the venous congestion. Infection is promptly super-
added, being here the essential cause of tissue-destruc-
tion.

If the hyperemia persist—*i. e.*, becomes chronic hyper-
nutrition—**hypertrophy** will result, that is to say, in-
crease in bulk from numerical increase of cells with,
perhaps, increase in size of the individual elements.
The presence in the tissues of migrated white blood-
cells doubtless adds somewhat to the bulk of the part.
It is rarely true that the enlargement results from an
equal numerical increase of all the cells of a part, more
often those of the connective tissue being chiefly multi-
plied. From the subsequent contraction of the neo-
plastic connective tissue the true tissue-elements have
their nutrition so diminished that many disappear—*i. e.*,
atrophy results, although the physical bulk of the part
may remain increased in these "chronic indurations,"
miscalled "hypertrophies." Sometimes, with no mani-
fest connective-tissue hyperplasia, diminution in bulk—
atrophy—seems directly to follow from nutritional
changes induced by a prolonged hyperemia. It may be
well to call attention to the fact that hypernutrition and
a consequent form of hypertrophy may result from inter-
ference with the exit of lymph from a part.

Exudates.—The meaning of certain terms already
employed or yet to be used can best be explained now.
An "*exudate*" is the material, chiefly fluid, which
passes out from the vessels either during hyperemia or
inflammation, mingled with the products of cell-prolifer-
ation. Exudates receive names either from their ana-

tomical relations or on account of some special, distinguishing quality. Thus, a "free" exudate is one poured out upon a surface or into a cavity, the latter often being termed a "dropsy." An "interstitial" exudate is one located between the layers of the tissues. A "parenchymatous" exudate is one located in the essential, functionating part of an organ, such as the secreting portion of a gland. An exudate composed only of the serum of the blood is called "serous;" when pus-cells are present in sufficient numbers to render the fluid opaque, it is now a "seropurulent" exudate. If mucus is the predominating element, it is called "mucous" or "catarrhal;" but if pus be mingled with the mucus, "mucopurulent" is now the proper term; while if red blood-cells are sufficiently numerous to color the exudate, it is called "sanguinolent" or "hemorrhagic." Serous exudates, poor in fibrin-forming elements and showing flocculi of coagulated fibrin, are classed as "serofibrinous." A "fibrinous exudate" is one which, containing large amounts of fibrin-forming elements, promptly coagulates in the interstices of the tissue or upon the surface of a membrane, usually a mucous one—in which latter event it is called a "croupous" exudate. Although micro-organisms probably have much to do with this croupous coagulation, it does occur in their absence. The term "diphtheritic" should be restricted to a microbic coagulation-necrosis which involves the epithelium, basement membrane, and other structures of the mucous membrane, even extending to the submucous tissues. In addition, some true fibrinous exudate is often present. When this apparent membrane is torn away, the true nature of the process is revealed, showing an actual loss of substance, not the removal of a surface addition to the part, as is a "membrane."

The "results" or "terminations" of hyperemia have thus been shown to be a rapid increase in bulk, following acute congestion; a slower increase from multiplication

of some of the tissue-elements, resulting from a chronic hyperemia; death of a part—*i. e.*, gangrene—the result of a passive hyperemia, although the primary process may be acute, the stagnation of the venous current physically preventing the entrance of arterial blood, and maintaining poisonous substances in contact with the cells; and certain nutritional changes which must now be considered.

Hyperplasia—*i. e.*, a numerical increase of cells—is what produces true hypertrophy, if all tissue-elements be concerned. If only the cells of one of the tissues of a part or organ are increased, then only hyperplasia of that tissue has taken place, although this is often incorrectly called hypertrophy. With the numerical increase of the cells there may be a greater bulk attained by the cell-elements themselves. True hypertrophy may result from increase of function or from increase of nutrition. Thus, the heart-muscle compelled to overcome undue resistance grows thicker and stronger; the set of muscles constantly employed to perform certain movements become abnormally developed, and the fibula, grafted upon the remains of a tibia whose shaft has been removed, having to sustain the whole weight of the body, markedly increases in thickness. While it is true that the exercise of function—in the case of muscle, *contraction*—necessarily attracts more blood, and hence increased nutrition of the part results, yet in the absence of all functional action, such manipulations as will bring more blood to the tissues—*e. g.*, massage—will retard the wasting of paralyzed muscles or restore power to those of a long disused limb, because an increased amount of pabulum is available. Both these forms of increase of bulk are physiological and probably include an increase in number and size of all the cell-elements of the limb, those of bone and blood-vessels as well as muscles. Congenital excesses of blood-supply cause the *congenital hypertrophies*, such as those of the female breast; although others, as gigantism of a limb or segment of a limb, are

often as much due to interference with the exit of lymph as to the excessive vascular supply. Indeed, such conditions as macrocheilia are purely lymphangiectases, or result from obstruction to the exit of lymph.

The hypertrophies of pathologic origin result, for the most part, from so-called '' chronic inflammation,'' which really means a chronic hyperemia initiated by traumatism and maintained by mechanical or chemical irritants, such as uric acid, altered nerve-influence, etc. All these act in one and the same way, maintaining a constant excess of pabulum in contact with the tissue-cells. Of these, the connective-tissue cells are most readily incited to proliferation, in consequence of which there results, not a true *hypertrophy*, but a *hyperplasia* of the connective tissue. All such young connective tissue tends to occupy less space as it develops—*i. e.*, contraction occurs—the cells diminishing in numbers, and fibrillation taking place, until the tissue-elements, as those of a gland, are compressed, and their nutrition so interfered with that they numerically *decrease*. The bulk of the organ or part may, instead of being diminished, be actually increased, although its functioning power is markedly decreased. This tendency of hyperplastic connective tissue must always be borne in mind as the most disastrous effect of chronic hyperemia. On the other hand, this property is made use of in the treatment of certain diseased conditions, as will be mentioned later.

Perversions of nutrition resulting in the undue maintenance or increase of certain elements of an organ occur in advanced years, such as the senile hypertrophy of the prostate gland.

From a diminished blood-supply, which is equivalent to diminished pabulum for the tissues, results the reverse of hypertrophy—that is to say, **atrophy.** This may follow from disuse, as seen in the thin-walled, light, and fragile long bones of a paralyzed limb, or those of an extremity with a chronically diseased joint, although the muscles and fat are the tissues which suffer most. Partly

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from the diminished vis-a-tergo of a weak heart, but more from the lessened dilatability and elasticity of the arteries in old age, senile atrophy of the muscles occurs despite their functional activity, while the hair-follicles undergo similar changes. Under this head writers, such as Park, have considered another form of diminution in bulk, which is effected not by lack of pabulum, but results from the actual removal of tissue by phagocytic action. This is best seen around a pus-focus, or better still, physiologically in Howship's lacunæ of bone, the result of resorption effected by osteoclasts—*i. e.*, phagocytic cells. This view seems incorrect, the process not being atrophy as commonly understood, although this process cannot be ignored, because it is the method by which the excess of osseous bone-callus, for instance, is removed, and the manner in which room is made both in this and in softer tissues for the proliferating tissue-cells needed for repair. In contradistinction to this is true atrophy, which causes a permanent diminution in the number, or in both the number and size of the cells of a part, which reaching a certain point ceases to progress, and the future nutrition merely maintains the tissues where the atrophying process left them ; or this process may be slowly progressive during the life of the individual. The diminution in bulk following diminished blood-supply is best seen around rapidly growing non-infiltrating tumors, notably cysts, or the muscles near a joint over which an elastic bandage has been long worn, or the atrophied line where the woman's tight garter presses.

Certain atrophies, such as those following disease of an important articulation, are due to a complexus of causes. Disuse and the absence of the normal reflex dilatations of the muscle-vessels, resulting from the impressions made upon the articular branches by movements, account for the muscular atrophy following joint-affections. The removal of restraining pressure upon the end of a bone, together with the absence of stimulus to the growth of the muscles from lack of use and ten-

sion, with the persistent, though not fully normal growth of the bone, accounts for the so-called "atrophic elongation" of the bones.

What are the general indications, the laws of therapeusis, the "principles" applicable to the treatment of hypertrophy and atrophy? For hypertrophy the blood-supply, particularly the arterial, *must be diminished*, while for atrophy, the *circulation must be increased*. How this should be effected belongs rather to the Practice of Surgery than to the Principles of Surgery, but will be touched upon when considering the treatment of inflammation.

The **symptoms of hyperemia** or congestion are, as we have seen, redness (*rubor*), due to the excess of arterial blood and possibly minute hemorrhages ; heat (*calor*), resulting from the excess of blood ; swelling (*tumor*), due to collection of fluid and leukocytes in the tissues, and slightly to excess of blood ; pain (*dolor*), from the pressure exercised by the exudates upon, and stretching of the nerves by separation of the various layers of tissue through which they pass. The pain of congestion may, like that of inflammation, not be felt at the actual point of hyperemia, but is often referred to the terminal distribution of the nerve, or to other branches of the same nerve, or even other branches of the plexus from which the irritated nerve arises, or, reaching the cord, the irritation may be apparently felt upon the opposite side of the body. Thus, the pain produced by the dying nerve-pulp of a tooth may be referred to another tooth or to all the teeth ; the pain complained of in all the branches of the brachial plexus when only one nerve is actually diseased, and the reference of pain to the right kidney when there is a renal calculus in the left kidney are additional examples. Throbbing pain rarely occurs in mere hyperemia ; but, if it does, can only be explained by the hypersensitive nerves recognizing the slightly increased pressure produced by the pulsation in the much enlarged arteries.

Park's explanation is that it is caused by "the added heart-pressure of systole upon sensitive nerves." While this is true, it is not the whole truth. The pressure would not be recognizable if the vessels were of normal caliber, and that which I have taught for many years is, I think, preferable—viz., that the congested, hypersensitive nerves recognize the increased pressure induced by the pulsation of the *enlarged* vessels, when the cardiac impulse would remain unnoticed in normal-sized vessels. The impaired function (*functio læsa*) results from the pain following the hyperemia of the nerves; besides this, in the instances quoted the serous transudation into the cavity of the joint interferes with its mobility, the increased secretion of tears and mucus from hyperemia of the conjunctiva embarrasses vision, or the abnormal sensibility to light, resulting from congestion of the retinal and choroidal vessels produced by excessive use of myopic eyes, prevents their use.

These symptoms are the classical ones given by Celsus nearly two thousand years ago as indubitable proof of inflammation. While they are more marked in a genuine (microbic) inflammation, they are, as we have seen, all present in pure hyperemia; in fact, during the earlier stages of inflammation (microbic) the phenomena *are* identical, and in many traumatisms *are due to the same causes*, the microbes later interfering with the reparative processes and undoing the good effected by the circulatory changes which are a necessary prelude to repair.

I will ask you to take upon faith some of the succeeding statements, promising explanation and demonstration later.

All the phenomena of hyperemia are, at the outset, conservative. The increased rapidity of the blood-current serves to remove tissue-waste and brings abundant pabulum and numerous phagocytes. To employ a simile often used, the women and children and the sick are sent away from the besieged city, stores of provisions

are accumulated, and the number of the defenders is increased.

Why then interfere? Because the hyperemia may really be owing to slight infection, or this will be favored if germs subsequently reach the congested area from a distant infection-atrium. This is because uncontrolled hyperemia is followed by slowing of the blood-current, thus favoring the dropping out and accumulation of germs in such numbers that the tissues cannot cope with them. The excess of pabulum favors their growth, while the exudates mechanically interfere with the nutrition of the tissue-cells and with the excretion of the results of their metabolic activity, producing a lowered vitality—*i. e.*, diminished tissue-resistance.

Treatment of Hyperemia.—What are the general indications, the suggestions resulting from this study as to treatment—*i. e.*, the removal of the conditions—in other words the Principles of Surgical Therapeutics involved? In acute hyperemia, the access of arterial blood should be diminished, the return of venous blood and lymph should be favored. To do this, the caliber of the arteries must be lessened, which upon purely physical principles will increase the rapidity of the current, thus sweeping out the accumulated, cohering leukocytes, preventing mural implantation of germs if any be present, and favoring reabsorption of effused products. The conditions of the intra- and extra-vascular pressure will be reversed, which will aid in the removal of exudates and metabolic products—not interfere with this as they did before—and hence increase the resistance of the tissues and their power of destroying germs, thus preventing any serious hindrance to the reparative processes. In *chronic hyperemia*, the access of arterial blood should be increased and the exit of venous blood and lymph favored. How this can best be effected in any given instance belongs to the Practice of Surgery. I have striven to demonstrate thus far that the reaction of tissues after injury leads to hyperemia,

which is conservative and a necessary prelude to repair. It is productive of many of the conditions caused at the outset, by the action of microbes, even to the migration of leukocytes (due to chemotaxis, a term later explained), tissue-cell proliferation, and the clinical signs of swelling, redness, pain, heat, and disturbed function.

LECTURE III.

IF the tendencies are toward repair after every traumatism, whether infection has occurred or not; if without infection repair would always succeed, unless prevented by purely physical disturbances; if infection produces merely a temporary interruption of these processes, repair being resumed at essentially the point where arrested just as soon as infection ceases to be operative, then the best way to understand the modifying influence exerted upon the normal processes by microbic (true) inflammation, its evil tendencies, how these are operative, and the best way to remove or prevent their efficient action, will be to study the normal reparative processes in the various tissues.

Let me start with the positive statement that repair is effected by the same processes in the hard and the soft, the vascular and the avascular tissues, any apparent differences being temporary, non-essential, and chiefly dependent upon physical conditions. Thus the physical obstacle presented by the mineral salts in bone temporarily prevents the rapid accumulation of reparative cells; but the same changes are effected as in the softest cellular tissue, though more slowly.

Two forms of repair have been erroneously described, because in reality there is only one process of repair, that attended by suppuration being merely a modification, the resultant of perturbing influences; when the perturbations cease, repair at once tends to proceed as at its in-

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ception, any variations of type observed being accidental and non-essential.

In normal repair the processes commence as soon as the physical disturbance of the parts ceases and the bleeding is checked. When the divided tissues can be accurately apposed, the wound heals with the minimum expenditure of material by what used to be called the "first intention," by "simple adhesion," or, incorrectly as I contend, by "aseptic inflammation." Such healing is only possible *in the absence of infection*, mark you, *not* the *absence* of germs, because as we shall learn, microbes may be present, but unable to multiply and interfere with the normal processes, for reasons which will be studied later.

When, however, the normal processes are interfered with by *infection—i. e.*, germs in such numbers that they can overpower the vital resistance of the cells, and which do multiply—repair is temporarily thwarted, resulting in vast loss of reparative material, tissue-destruction, and the formation of an excess of germinal tissue which, after conversion into scar-tissue, too often exerts most pernicious, atrophying pressure on the parenchyma of glandular organs, etc. So soon as the tissue-cells gain the upper hand of the microbes, a reversion to the normal reparative processes commences, which finally proceed, in essence, as if no disturbance had occurred. I have said "in essence," because there is great excess of material needed for the repair, producing at first sight some real differences ; but after the destruction of tissue by either ulceration or sloughing, repair is effected by the same processes which produce healing in an aseptic wound with loss of substance—viz., by the formation and organization of granulation-tissue, or, employing the old term, by the "second intention." Finally, in any method of healing, whether by immediate union or by the "second intention," this takes place by the formation and organization of granulation-tissue. In one, only a microscopic amount is formed ; in the other it is readily

seen by the unaided eye, but the end-processes are the same. If two aseptic granulation-covered surfaces are held in contact, they will fuse, and healing will occur, as it is sometimes called in the older books, by the "third intention."

It is useful to know both the meaning of these terms and the minute processes involved to understand the older authors. It is interesting to notice how the older authors recognized that however diverse the phenomena presented after traumatism, all evinced an "intention" to effect repair, and that their first choice is what we elect, because it is pure repair, while when this is impossible we avail ourselves of the second method, often thus being able to utilize the third, and in all—provided suppuration is avoided—in the end securing really the same kind of repair.

Healing of Incised Wounds.—Let us now examine the minute processes observed during the healing of an incised wound. *Hemostasis* is effected by the formation of thrombi in the blood- and lymph-vessels. The small amount of blood between the closely apposed wound-surfaces, together with leukocytes from the lymph-spaces and the fibrin-forming sustances contained in the effused liquor sanguinis, forms a clot, which extending into the interstices of the tissues forms a mechanical bond uniting the divided surfaces ; this is the "coagulable lymph" which the older authors regarded as the chief agent in the union of wounds. Wandering cells soon crowd the wound-borders, whence the leukocytes migrate into the fibrinosangineous coagulum, until by the third day this has about disappeared, here and there remains of clots alone being detectable, the bulk consisting of a mass of leukocytes. By the sixth day large epithelioid cells, descendants of the fixed connective-tissue cells and the endothelial cells of the small vessels—*i. e.*, the so-called "formative cells" or "fibroblasts"—invade and gradually replace the leukocytes, which are consumed as food by the former cells, although some leuko-

cytes doubtless wander back into the lymph-spaces or possibly into the blood-vessels. Some authorities contend that the cells resembling leukocytes, seen during this and the next stages of healing, really serve as formative cells or provide the endothelium for the new lymph-spaces. The fibroblasts promptly assume various shapes, becoming clubbed, spindle-shaped or multipolar and branched, the processes of contiguous cells anastomosing. As development proceeds, a portion of the protoplasm with the intercellular substance becomes fibrillated, leaving little beyond the original nucleus and a small portion of the protoplasm, forming fixed connective-tissue cells embedded in a fibrillated structure, which later assumes all the characteristics of fibrous tissue. Rendering possible this development, special provision for abundant pabulum is made. At first the cells, being nourished through the medium of the plasma-channels, obtain just sufficient pabulum to maintain their vitality and to stimulate them to multiplication, but not enough to admit of their development into a permanent tissue. About the fifth to the sixth day new vessels are formed from the pre-existing vessels at the borders of the wound, which gradually grow into the mass of cells, supplying them with such ample nutriment, that the development into fiber-cells passing across from one side of the wound to the other is very rapid, so that as early as the sixth day a *vital* bond of union, replacing the mechanical one, is well advanced in vascular structures, such as those of the face.

The first steps in *new-vessel formation* consist in an accumulation of granular protoplasm on the exterior of a pre-existing capillary loop, which gradually forms a solid, nucleated filament. This may be simple or branched, and fuses with another vessel, with another bud from a neighboring capillary loop; or, again, the filament may arch back and become connected with the vessel from which it sprang. The young connective-tissue cells (fibroblasts) near the vascular outgrowths

arrange themselves alongside them, or as bundles form solid continuations ; again, they are said to form channels which later communicate with the lumen of some capillary. Occasionally a protoplasmic vascular filament will join a process of one of the branched formative cells. These solid protoplasmic processes liquefy in their centers, a lumen forming continuous with that of the parent vessel.

FIG. 2.—Showing various ways in which new capillaries are formed in organizing granulation-tissue.

The protoplasmic outgrowths may also be from the outset hollow, admitting blood ; but even then they terminate by filamentous prolongations, and develop after one of the methods already described. Although at first the new capillaries possess only homogeneous walls, later they display the ordinary appearances.

Let me call your attention to the fact that " **healing by a blood-clot**," as it is termed, is merely an extension of the process just studied. Instead of a minute linear

space to be bridged by cells, there is a gap requiring a scaffolding to enable the cells to reach across from one side to the other and one which must remain *in situ* until vascularized—*i. e.*, until they have developed into granulation-tissue. The leukocytes penetrate and chan-
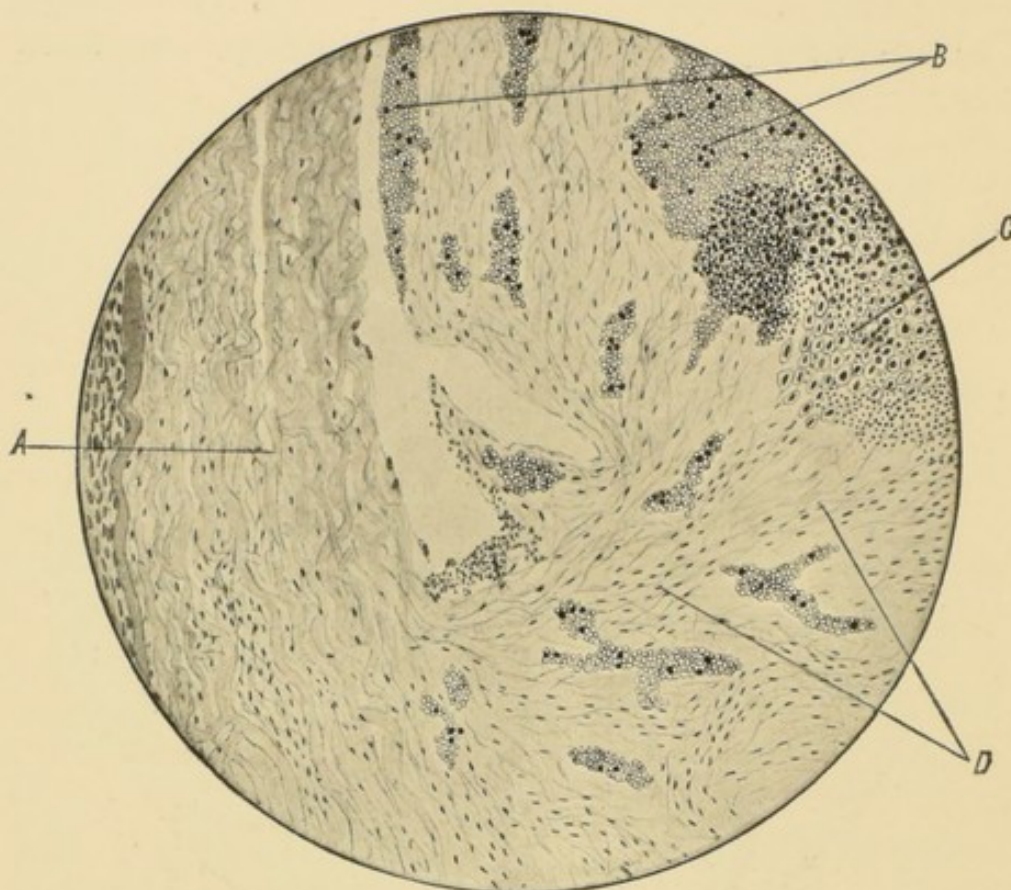
FIG. 3.—Showing organization of thrombus in femoral artery: *A*, vessel-wall; *B*, remnants of blood of thrombus; *C*, epithelioid cells; *D*, recently developed connective-tissue fibers.

nel the clot in various directions, preparing pathways for the formative cells, which follow along these tracks, until all clot disappears and vascularization is effected. Wherein does this differ from the manner in which the physical bond between the wound-surfaces, composed of blood- and fibrin-clot is substituted by a vascularized cellular mass? Not one iota. The only difference is that of degree and not of kind, again illustrating what I am contending for, that there is, in essence, but one kind of healing, modified by physical conditions. This,

however, is not always the fate of blood-clots, for they may disappear by the ingrowth of processes of already vascularized masses containing formative cells—viz., granulations, the constituents of the clot disappearing by absorption effected by the phagocytic cells. This illustrates how, when a physical obstacle interferes with the filling in of a gap by granulations, this physical obstacle is removed, whether it be the compact tissue of bone, an ivory peg, a fragment of devitalized soft tissue, or a blood-clot, which last, for some reason, acts as a physical obstacle, not as a useful scaffolding.

The term "**filling up of a wound by granulations**" is a misnomer. The actual depth of a layer of healing granulations is slight. The deeper layers—those nearest to the blood-supply—are rendered capable of development into adult tissue, which "contracting," as it is termed, draws down the margins of the wound, pulls in the surrounding tissues, somewhat elevates its base, and markedly diminishes its superficial area, so that the defect is largely effaced ; but it is not done by the "organization" of cubic inches of granulation-tissue, which would often be requisite if a defect was really "filled in by granulations."

What is this *granulation-tissue* which has been so frequently mentioned? Superficially it consists of numerous multi- and mono-nucleated leukocytes, with many delicate blood-vessels running more or less vertically to the surface. Deeper, epithelioid cells abound, and, still deeper, spindle-cells arranged in bundles can be seen. In this layer, in old wounds the majority of cells become converted into fibrous tissue, with the blood-vessels forming a horizontal network. The classical capillary loops capped with cells, which are said to account for the granular forms of the granulations, do not exist, parallel vessels, ascending more or less vertically, as has just been said, being alone detected in the superficial granular layer of the granulations.

All the essential facts concerning the aseptic forma-

tion of granulations, by which healing is effected in every tissue, have been already stated, although in certain highly specialized ones, such as spinal nerves, regeneration of nerve-tubules takes place. If there has been loss of substance, either mechanically produced or from sloughing in an aseptic wound, the healing will be by granulation in the ordinary acceptation of the term, although it has been shown that even primary union is effected by the formation and organization of vascularized formative cellular masses—viz., granulations. The chief reason why rapid repair is possible in aseptic wounds when compared with infected ones is not any difference in the processes, but because no peptonizing ferment is present, as is the case during infection, which, dissolving the intercellular cement, disassociates the cells one from another, thus mechanically preventing them from receiving the pabulum necessary for development into a permanent tissue. Unquestionably the vitality of the cells is impaired or absolutely destroyed by other toxic products of germ-growth ; but were the cells absolutely normal, they could not develop when separated from one another and floating free in the wound-fluids.

In the same way, aseptic **separation of a slough** and infective separation of a slough are identical processes. In the former, at the border where the dead and living tissues are in contact, as in the processes of repair, leukocytes and then formative cells accumulate, until nothing but a layer of cohering, vascularized cells holds the two together. Those cells farthest from the blood-supply—viz., those in contact with the dead parts—lose their vitality, their intercellular cement is dissolved, and the "slough separates"—*i. e.*, the layer of granulation-cells between it and the better nourished granulations in contact with the living tissue becomes liquefied ; hence separation ensues. Precisely the same is effected much more rapidly by the peptonizing ferments of microbic origin. In the aseptic separation of a slough the mini-

mum conversion of the border-line between the dead and living tissues into granulation-tissue occurs, and the minimum liquefaction of cells to permit separation is requisite, only a small amount of cloudy serum or lymph being produced, repair being secured with the least possible waste of material and development of scar-tissue. Infection of an incised wound, for instance, promptly causes solution of any mechanical bond of union and disintegration of all coagula ; hence any scaffolding enabling formative cells to bridge gaps is destroyed. Extensive conversion of the wound-margins into granulation-tissue occurs, and much of these densely infiltrated tissues perishes from destruction and separation of the component cells. Many living cells are washed away by the freely exuded wound-fluids, although far too many remain to develop into a dense, dangerously contractile scar. Sloughing in an infected wound is well known clinically to produce worse cicatrices than even prolonged suppuration. This is because the prolonged presence of the dead, intensely irritating, infected slough excites the most lively proliferations of the cells *in the depths* of the tissue too far removed to be destructible by the microbic ferments and toxins, thus leaving more cells than mere suppuration does to develop into scar-tissue.

The longer maintained and more intense hyperemia following true inflammation (microbic) enables the much larger number of cells resulting from this condition to form a denser and thicker cicatrix, but the same end-processes take place as in aseptic healing.

Epidermization.—Although everything has been described pertaining to all healing in the depths of a wound, the method by which the superficial layers of organized granulations are covered in by epithelium has not been described. The process is the same whether the minute line between the edges of an incised wound healing by primary union is concerned, or the wider area left where loss of substance has occurred. Epidermization,

or the final covering in of the surface-granulations by the formation of epidermic cells, results from multiplication of the deeper layers of the rete Malpighii. When the whole thickness of the skin has not been destroyed, regeneration of the epithelium takes place in part, by multiplication of the epithelial elements of the remnants of the sebaceous and sweat glands, and those of the hair-follicles, little islets of new epithelium being seen scattered over the granulating surface. To secure this development of the young epithelial cells they must be retained sufficiently long in contact with the older cells to undergo some corneous change. Toward the end of every variety of cicatrization, the drying of the exudate over the last part to heal forms a "scab"—*i. e.*, desiccation of the more superficial layers of cells retains the deeper layer of young epithelial cells mechanically in position, until the necessary developmental changes can take place. This *healing under a scab* sometimes occurs upon a larger scale, and can often be successfully imitated in aseptic wounds, by means of an artificial scab formed by the application of shreds of absorbent cotton, which becoming soaked with the blood or serum dries. The use of nitrate of silver, lightly applied around the margins of a granulating surface, acts in the same way; the layer of coagulated albumin and cells retains the deeper epidemic cells in contact with the wound-margins, enabling the corneous change to occur. Comparatively few epithelial cells are requisite to cover in defects that were originally very large, because, as has been said, the superficial area of a wound is very much diminished by contraction of the original granulations.

Union by Adhesion of Two Granulating Surfaces.— Two aseptic surfaces if maintained in contact will fuse, as has already been stated, and definitive healing will take little if any longer time than if the wound had been primarily coaptated. This is because the most superficial formative cells are as capable of development as those formed in a perfectly

coaptated wound, or those of the deepest layers of an infected granulating wound, and for the same reasons—viz., their vitality is unimpaired since no germ-products can reach them in either case, and no accumulation of fluids occurs effecting mechanical separation. Upon this fact depends the success of "secondary suturing," when, for instance, tamponade of a wound becomes requisite for the arrest of hemorrhage. Here the passage of sutures five or more days after the infliction of a wound will often secure prompt union. This is because all the same preliminary changes must first be effected in the coaptated as well as the uncoaptated wound ; but if physical fusion of the two layers of cells is then produced by pressure, the development of fiber-cells takes place, uniting the wound-surfaces just about as quickly as if the bond had been originally formed of one mass of cells.

The vitality of a severed part, as the tip of a nose or finger, or skin (not epidermic) -grafts, is maintained according to Thiersch and Tillmanns, by communications formed between the vessels of the granulations and those of the graft or severed part, through the medium of the intercellular plasma-channels. Later, all the phenomena described as pertaining to union by primary adhesion occur. The detached part is passive until after the third day, when it commences to be vascularized by outgrowths from the vessels of the surface of the wound. Despite the two or three days' interruption of the direct blood-supply in the case of a skin-graft, only the epidermal layer, a portion of the rete Malpighii, and most of the vessels perish, the last by atrophy preceded by hyaline degeneration. In from three to four days the epithelial cells of the sebaceous and glandular follicles proliferate and penetrate the mass of newly-formed cells infiltrating the wound-surface and contiguous portions of the graft, and in two weeks, according to Garré, the conversion of the granulations into connective tissue is completed.

The mechanism of the **contraction of scars** requires

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explanation, because, as commonly understood, there is a vague idea that an active process is concerned, analogous to the shortening produced by contraction of muscular tissue. The primary mass of soft succulent granulation-tissue composed of innumerable cells, in the
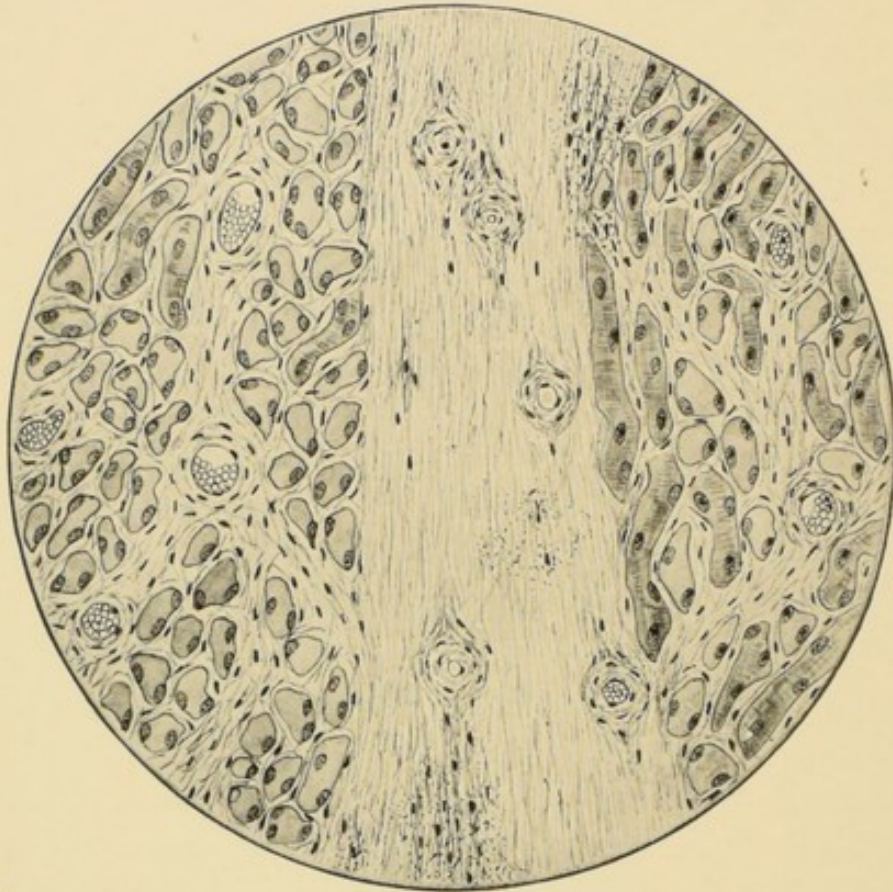
FIG. 4.—Showing scar in muscle. The scar shows its dense fibrous structure, with few nuclei; the vessels are almost completely obliterated by the condensation of the scar-tissue.

process of organization into a permanent tissue, undergoes a species of condensation. Many cells disintegrate from lack of pabulum, and the detritus is absorbed. The greater part of the protoplasm of those which remain undergoes fibrillation—*i. e.*, becomes converted into a dense, interwoven mass of fibers containing but little fluid. As this physical condensation progresses, the approximating fibrous bundles so compress many of the capillaries that they atrophy and disappear, still further lessening the bulk of the cicatrix. The diminution of

the blood-supply leads to the atrophy of many more cells, until the previously succulent, bulky, cellular mass is now composed of very few vessels and cells, and consists almost solely of desiccated fibers with a few nuclei, all occupying a comparatively small space. A comprehension of these processes explains the long continued, progressive character of this " contraction of a scar," the duration of the contracting period being in proportion to the number of cells maintaining their vitality.

Repair of Non-vascular Tissues.—Only a few words need be devoted to a description of the modifications of the ordinary processes of repair observed in non-vascular tissues, such as cornea and cartilage. In the former its anastomosing intercellular plasma-channels readily admit wandering cells coming from the vessels of the related vascular structures (sclera and conjunctiva), and, later, proliferation of the fixed cells and their vascularization occurs. It has been recently alleged that some of the cells seen in corneal wounds are corneal cells squeezed out by the elastic laminæ from the edges of the wound which slip down, as it were, into and aid in filling up the wound. In a similar manner wandering cells pass along the plasma-channels of the cartilage, the fixed cells of which later proliferate, the blood-supply reaching them by outgrowths from the vessels of the surrounding vascular tissues. The cicatrix in cartilage after aseptic healing remains long, if not always, fibrous; "if a severe inflammatory (microbic) reaction takes place, the cicatrix will rapidly become hyaline, like normal hyaline cartilage."

LECTURE IV.

REGENERATION OF TISSUES.

Do tissues actually become regenerated? In only a few tissues does repair progress beyond the formation of scartissue. Where regeneration is possible its perfection will be in proportion to the apposition effected and the asepsis secured. Surface epithelium and all connective-tissue structures, such as fascia, bone, or tendons, can be completely regenerated.

I shall briefly describe the extent of regeneration and the special modifications of the typical processes of repair observed in the various tissues or organs, recapitulating in part what has already been said.

Epidermis.—The epidermis, including the epithelium of the gastro-intestinal tract and of other mucous membranes, is completely re-formed, the new cells being descendants of the old epithelial cells of the margins of the wound, or, after partial destruction of the skin, as has been already explained, originating by division of the epithelium of the various cutaneous structures whose extremities lie in the deeper portions of the skin, or actually in the cellular tissue beneath. The same is also true for the remains of glandular epithelial structures after partial destruction of mucous membrane.

Skin.—The fibrous portion is completely regenerated, although the arrangement of the bundles is more irregular, and it is long before elastic tissue is developed in the scar ; but the hair, sebaceous and sweat follicles, with the true rete Malpighii, are not re-formed. Lymphatics are also absent, and an old scar is so much less vascular than normal skin—from obliteration of most of its vessels,

effected as described when explaining the contraction of a scar—that the cicatrix is liable to break down from slight causes. This avascularity of scars is a matter of common observation. At first redder than the surrounding parts from excess of blood-vessels, as the scar condenses most of these atrophy, rendering the scar in time whiter than the environing structures.

Fasciæ, tendon-sheaths, and **tendons** are practically regenerated. After division of a tendon the proximal end retracts, and the method of repair varies somewhat according to whether a blood-clot is or is not present. In the rabbit, when but little blood is effused, emigration of leukocytes occurs, followed in from two to three days by rapid proliferation of the cells of the sheath. Many of the cells of the tendon-stumps rapidly degenerate, but about the fourth or fifth day some take part in the formation of the granulation-tissue. The exudate extends some distance above and below the extremities of the softened, succulent, and freshly vascularized tendon-ends. Warren contends that when the sheath is filled with blood, the clot is removed by ingrowths of vascular granulation-tissue springing from the sheath without a primary infiltration with leukocytes ; other observers deny this, and probably both are right at times. The grayish cellular mass becomes pink from vascularization (fifth day, Paget ; tenth to fourteenth day, Warren), the new vessels in the granulations joining those of the tendon-ends. Thus a spindle-shaped cellular bond of union usually fills the gap by the fifth day. If no blood-clot be present, the sheath collapses and becomes adherent to the tendon-stumps, regeneration being effected by proliferation of the cells of the sheath and of the cut tendon. The new tissue gradually approximates that of normal tendon until microscopically no difference can be observed. The sheath usually remains slightly adherent at the point of section.

Muscular Tissue.—Defects in muscular tissue are

only repaired by scar-tissue formed from the granulation-tissue produced by the connective-tissue elements. Regeneration is observed to a limited extent after slight injuries or contusions, and even near a cicatrix, commencing by enlargement and proliferation of the muscular nuclei, resulting in the formation of large mono- and poly-nucleated cells, occupying the place of the destroyed fibers. These develop into spindle-cells lying side by side, which promptly become longitudinally
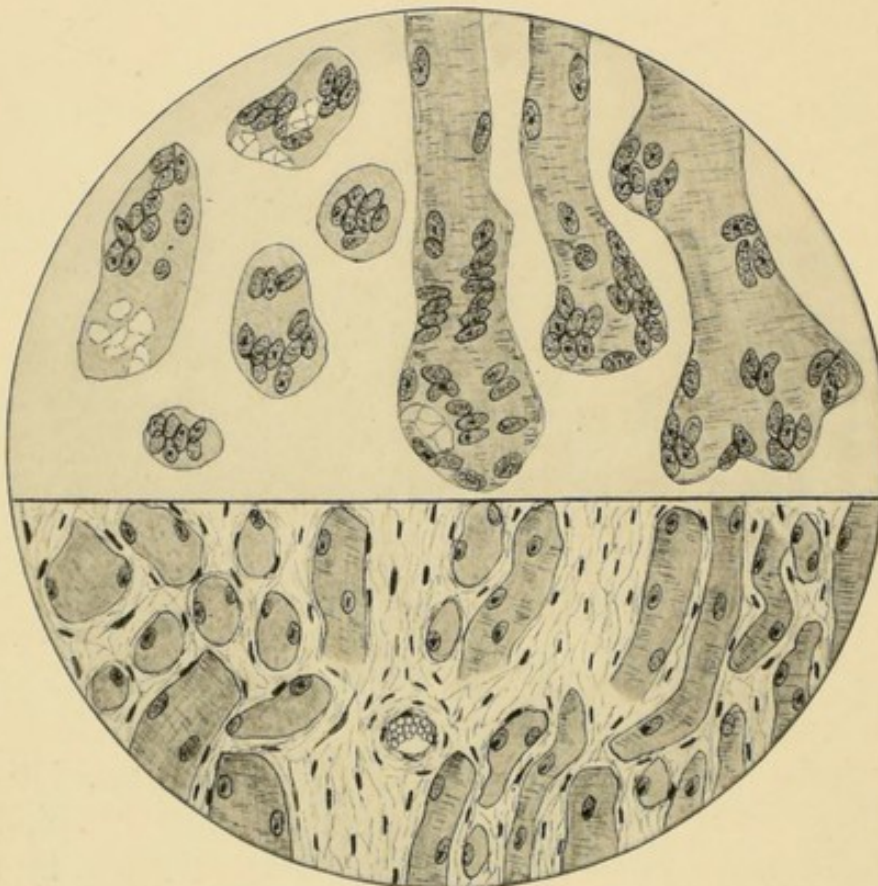

FIG. 5.—Showing, in upper half of cut, longitudinal and cross-sections of regenerating muscle-fibers; in lower half, normal muscle-fibers.

fibrillated and during the third week show commencing transverse striation. Another view is that granulation-tissue forms among and around the necrosed muscle-fragments. The extremities of the injured muscle-cells split up into spindle-shaped fragments, which fattily degenerate and are absorbed. The nuclei of the living

muscle-cells around the injured area form bundles of muscular fibrils which totally disappear by the third week. The disappearing fibers are replaced by bundles of longitudinally striated fibers and spindle-cells formed by splitting up of the living muscle-fibers and proliferation of the nuclei. Growth of new muscle-fibers into the granulation-tissue and disappearing mass of muscular débris, commences about the sixth day by small, multinucleated protoplasmic fibers, springing from the stumps of non-degenerated fibers or from those longitudinally split. These outgrowths may be bifurcate, with club-shaped or fusiform extremities which contain many nuclei. Longitudinal striation occurs early, transverse striation at the close of the second week. The new muscular filaments interlace, lateral budding being not uncommon. Many of the fibers fail to develop, fattily degenerate, and are absorbed. Those which persist, increase in bulk, acquire transverse striation, interlacing with others from the opposite side of the gap, until the connective-tissue scar may, in very slight wounds, practically disappear. Some irregularity always remains, but the interlacement of the fibers is to a great extent gradually replaced by a more normal arrangement.

Blood-vessels.—Vascular repair depends upon the formation and "organization of thrombi." Injury to or destruction of the vascular endothelium, and partial or complete arrest of the blood-current are necessary for the production of thrombi. A thrombus in a living vessel materially differs from a mere blood-clot, owing to the part played by blood-plaques and white cells. Vascular thrombi are indeed often entirely white, although ante- or post-mortem accretions of genuine clot may occur. Unless only a portion of the circumference of a vessel is injured, the thrombus usually extends in time to the collateral branch above, or much farther if a vein be concerned. A small parietal wound may become blocked by a thrombus limited to the wound and its immediate neighborhood, which may

organize, leaving only a localized thickening. This is quite common in veins, but may also occur in arteries of any caliber. The thrombus when once formed must either become organized, soften, disappear by absorption in whole or in part, or calcify. The minute processes are exactly the same seen when "healing by a clot" occurs, if the including vessel-walls are understood to take the place of the surfaces of the wound (see Fig. 3, p. 44, showing organization of thrombus). The vascular walls—like the wound-surfaces—first become infiltrated by leukocytes, next the thrombus is invaded, penetration taking place along many routes, thus breaking it up into numerous isolated masses. The endothelium proliferates where injured, and the thrombus gradually becomes replaced by the "formative cells" thence derived, which penetrate along the tracks prepared by the previous invasion of leukocytes (Ballance and Edmunds). Vascularization of the germinal tissue is effected here as elsewhere, all traces of the thrombus—the superseded scaffolding—gradually being removed. As the formative cells can enter only through the portions of thrombus in contact with the vascular wall, organization of a thrombus which does not entirely occlude a vessel is a slower process than where complete blocking has occurred. One or more of the new vessels may persist or even enlarge, restoring in a measure the continuity of the vascular lumen, but usually the occluded segment of the vessel is converted into a fibrous cord. Sinus-degeneration and other rare changes in thrombi do not concern us here. Occasionally, where tense surrounding tissue interferes with the egress of blood, a small wound of a large artery may become occluded by a clot commencing outside the coats of the vessel, extending thence through the gap in the wall and causing an intravascular thrombus limited to the immediate vicinity of the wound, leaving a portion of the lumen free. Granulation-tissue grows into and replaces the portion of thrombus occupying the vascular wound, and a con-

nective-tissue—*i. e.*, an inelastic—scar results. Intra-vascular pressure causes yielding of this scar, an aneurysm forming, or perhaps the cicatrix suddenly ruptures giving rise to an arterial hematoma. Repair after ligature of a vessel proceeds upon the same lines. To summarize, a protective thrombus forms, proliferation of intimal and connective-tissue cells takes place, substitution of the clot by these formative cells occurs, to be followed by vascularization of this germinal tissue, its development into scar-tissue, and the conversion of the vessel up to the next collateral branch into a fibrous cord.

Warren insists that at the site of ligature the vessel-walls become, as we have seen, converted into a mass of granulation-tissue, but that later the vessel-ends separate, expand, and the granulations freely penetrate the thrombus carrying in new vessels. Between the irregular masses of granulations spaces are left, which after absorption of the clot form blood-spaces, opening on one side into the lumen of the vessel, on the other communicating with the capillaries of the granulation-tissue external to the vessel. Eventually a regeneration of the vessel-walls is effected, the cicatrix being lined internally with intima, unstriped muscular tissue forming the new middle coat, and most externally is fibrous tissue. The frequent persistence of a small central vessel opening into the lumen above and communicating "with the capillaries surrounding the arterial stumps" is also described. As I have elsewhere said, while Warren's observations stand alone with regard to the formation of a muscular scar, they are deserving of further investigation, as explaining —if confirmed—why aneurysmal dilatations so rarely result from the scar of a ligation in continuity.

Nerves.—Under favorable circumstances repair is here so complete that the peripheral end of one sensorimotor nerve has been united with the central end of another, with restoration of peripheral function. The alleged "immediate union" of nerves with restoration of their conducting power, no degeneration of the peripheral one

occurring, *appears* to have been established clinically. The vast bulk of both clinical and experimental results demonstrating the apparent impossibility of this method of union, the accuracy of the observations as to cases of supposed immediate union are to be viewed with grave doubt. Anastomoses between nerves—not always those mentioned in the anatomical text-books—which are subject to wide variation, *supplemental or vicarious sensibility*, and individual differences in the distribution of a given nerve probably explain the supposed " primary union." Degeneration of the whole of the distal segment with a portion of the proximal is the rule, a downward growth of embryonic fibers from the proximal end taking the chief part in the repair ; these originate from pre-existing fibers. The embryonic fibers penetrate the granulation-tissue by means of which the *physical* union of the nerve-trunk is effected. From Howell and Huber's experiments we learn that in the dog, four days after section of the nerve, segmentation of the myelin-sheath and fragmentation of the axis-cylinder takes place in the peripheral portion of the nerve. Active nuclear proliferation in the neurilemma is distinct by the seventh day, with migration of the new cells, several often occupying one internodal space. During the following week the segmented myelin and fragmented axis-cylinder disappear by absorption, complete removal being effected in fourteen days. Next, the nuclei acquire an investment of protoplasm, which increases until " a single solid protoplasmic fiber with embedded nuclei occupies the old sheath." When physical union is effected with the central end this is the rule, but if union is not made one or more fibers may arise within an old sheath by longitudinal cleavage (Howell and Huber). These amyelinic " embryonic fibers " later acquire a myelin sheath, the old sheath probably becoming, according to these authors, part of the endoneural connective tissue. In the dog, return of function commences as early as the twenty-first day and is complete in eighty days, a much

shorter time probably than is occupied in the restorative processes in man. Nerve-impulses can be conveyed in the embryonic-fiber stage when the newly-formed fibers are united with normal fibers of the central end. Immediate suture gives the best results, even an hour's delay producing a recognizable difference. Experiment corroborates clinical experience that although complete degeneration of the peripheral end apparently always occurs, yet *regeneration is more rapid when immediate suture is done.* Because granulation-tissue opposes a physical obstacle to the penetration of the newly-formed nerve-fibers, pyogenic infection, which invariably produces an excess of this tissue, is to be avoided, aseptic healing with the formation of the minimum of granulation-tissue affording the most perfect results. The greater the length of the distal portion, the longer will be the time requisite for cure. The same remarks are true as to the time and perfection of result if a segment of a nerve is actually removed. Arrest at the "embryonic stage" occurs where suture is not done, a bulbous enlargement usually forming on the proximal end, composed chiefly of fibrous tissue; exceptionally the distal end undergoes the same changes. The statements I have made, embodying as they do the results of modern research, must be accepted as true, however much they may invalidate some of the earlier, cruder observations.

Bone.—Union may take place by the same processes seen in primary union of soft parts, or it may be secondary—*i. e.*, by granulation-tissue—the bond usually being genuine osseous tissue. When a long bone is fractured, considerable blood will be effused from the ruptured medullary and Haversian vessels, as well as from those of the periosteum and contiguous lacerated soft parts. Even in the rare event of the periosteum remaining untorn, it is more or less stripped off the broken extremities. The injured tissues infiltrated with blood are soon invaded by leukocytes and effused blood-plasma, and, fibrinous coagulation taking place, the extremities

of the broken bone lie embedded in a dense, ill-defined mass of firm cellular exudate, involving periosteum, connective tissue, and possibly contiguous muscle. In from fourteen to twenty-one days the blood is completely absorbed, leaving a firm, dense, cellular, vascularized, partially organized mass. This "callus," as it is called, is most abundant in and beneath the periosteum and extends between the ends of the fragments ; it is sometimes even at this stage partly cartilaginous. In from seven to fourteen days—*i. e.,* during the fourth or fifth week— ossification occurs in the callus, which forms a spindle-shaped ferrule of porous bone around the extremities of the broken bone, provided these are not much displaced, in which latter event the reparative tissue is more irregularly disposed. Meanwhile, similar changes have also been taking place in the medullary tissue ; the blood-clot with the contiguous soft parts of the medulla has become infiltrated with leukocytes. The blood is next absorbed, the fat disappears as the connective tissue and endothelial cells proliferate, and granulation-tissue forms from both bone-fragments, which soon fuses and develops into porous bone blocking the medullary canal.

Owing to the physical obstacle presented by the dense osseous tissue, the proliferation of the connective and vascular tissues occupying the Haversian canals in the compact tissue contiguous to the fracture progresses but slowly, the lime salts gradually disappearing, so that it is comparatively late before the granulation-tissue thus produced ossifies, definitely uniting the fragments. When union has been finally completed, the excess of "external" and "internal" callus is absorbed, the medullary canal in the course of years becomes restored, and in time the site of the fracture may be hard to detect if the reduction has been perfect.

The restoration of the medullary canal as well as the removal of excess of callus results from the phagocytic action of certain cells—"osteoclasts"—which in the former process accumulate in the small spaces of the comparatively

spongy callus, enlarging these until the bone becomes can-
cellous, the partitions between cancelli disappear, large
rarified spaces form, these finally coalescing, until the
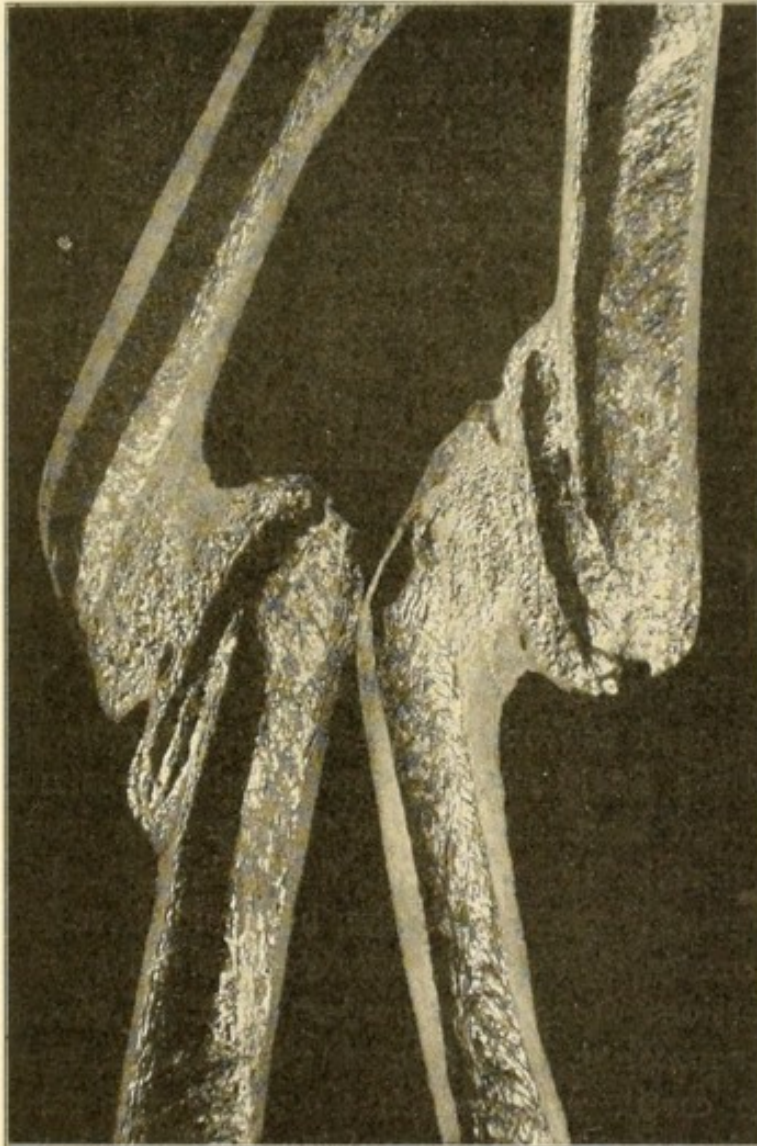medullary canal is re-formed. If overlapping of the

FIG. 6.—Showing action of osteoclasts causing absorption of dense cortical
bone and callus.

fragments persists, the open ends of the medullary canal
become closed off by bone, and its lumen is usually only
imperfectly restored by a gradual conversion of the over-
lapping and fused portions of cortical bone into cancel-
lous bone. (Fig. 6.)

To impress upon your minds the identity of the re-

parative processes in bone as well as in the softest of tissues, let me briefly rehearse the various steps. The lacerated periosteum, muscles, etc., and blood-clot are infiltrated by leukocytes; the cells of the soft parts of the bone, especially those of the deeper, osteogenetic layer of the periosteum some little distance from the fracture begin to proliferate, numerous angular and stellate cells—osteoblasts—appearing, which originate chiefly from the deeper periosteal cells. As time progresses all the osseous soft parts—*i. e.*, the periosteum, bone-cells, medullary tissue and contents of the Haversian canals, which are all continuous structures—undergo similar changes. The dense bone opposes a temporary, physical obstacle to cell-proliferation, because the earthy salts and matrix can be only gradually removed; but this is only a question of time, being exactly what is seen in the soft parts where the phagocytic cells remove the tissue-cells, as the granulation-tissue appears. The osteoblasts, probably formed by division of the fixed connective-tissue and endothelial cells of the bony soft parts, as well as from the deep periosteal cells, are separated by a finely striated intercellular substance, each surrounded by a halo somewhat like that seen around cartilage-cells, the more highly differentiated portions forming interlacing trabeculæ, here and there infiltrated with lime salts. This is *osteoid tissue.* The more central portions of callus show less striation of the intercellular material, later assuming the hyaline appearance of true cartilage, of which tissue a large part of the internal portions of the provisional callus often consists during the early period of consolidation. Direct ossification of the osteoid tissue may take place, the cells growing smaller and some becoming branched, occupying spaces in the calcareous matrix—*i. e.*, they are now bone-corpuscles. Trabeculæ of bone soon form by the deposition of the lime salts at numerous points. This commences in from ten to fourteen days, while newly formed blood-vessels spring from those in neighboring

Haversian canals. These ramify between the bone-plates, and run at right angles to those of the old bone.

Ossification of the external callus usually commences in the angles formed between the separated periosteum and bone, and extending thence, the two buttresses meet and fuse at the middle of the spindle-shaped mass of provisional callus. This bony callus is composed of a network of bony trabeculæ, the interstices of which are filled by masses of young cells that have not yet ossified ; the periphery of these masses is steadily converted, layer by layer into bone. Although most of the cartilage sometimes found in callus disappears before advancing vascular ingrowths containing osteoblasts, some is converted directly into bone by deposition of lime salts in the matrix, a portion of the cells remaining as bone-corpuscles. In the medullary canal similar changes are effected, the osteoid tissue commencing to form at the periphery of the canal, whence it spreads concentrically until it is occluded. Hyaline cartilage is rarely seen in this "internal callus." Finally, the Haversian canals of the compact tissue of the ends of the fragments become choked with a round-celled infiltrate ; the lime salts are dissolved and removed with the ground substance, the resultant large cancellous-like spaces becoming filled with osteogenetic cells. The germinal tissue thus formed between the ends of the fragments fuses with the contiguous portions of the cellular mass forming the "internal" and "external callus," and union of the cortical bone takes place by ossification of this "definitive callus."

From this description it is clearly seen that callus passes through a stage resembling the formation of fibrous tissue and that where cartilage is developed it is often immature fibrocartilage ; hence, "fibrous union" of bones is often a mere arrest of osseous union at a certain stage of the process. If the disturbing causes can be removed in time—*i. e.*, before development into an adult tissue has taken place—nature (as we know clini-

cally she often does) takes up the process where it was left off and completes bony union. This explains the benefits resulting from rubbing the fragments together, blistering over the fracture, and partial use of the limb when fixed by apparatus, sufficient hyperemia being thus induced to lead to further development of the callus and deposition of lime salts, in these cases of delayed union.

Healing of bone may thus occur by a process identical in all essentials with primary union of soft tissues ; but it may also take place by the "second intention," by granulations—indeed, must do so in open fractures, where either loss or death of bone has occurred. Where bone has been primarily lost, the periosteum having usually been destroyed over the osseous defect, proliferation of the fixed and endothelial cells of all the surrounding soft tissues, including those of the subjacent bone, takes place, the granulation-tissue thus formed becoming converted into bone by one or more of the methods described, as the superficial soft parts close over and cicatrize.

When death (necrosis of bone) occurs, at the border-line between the dead and living parts, lively proliferation of the cells of the periosteum, medulla, and Haversian canals produces a mass of germinal tissue, some of whose constituent cells (osteoclasts) cause absorption of the bone-substance, until the continuity of the dead and living bone is interrupted by a layer of cells. The bacterial peptonizing ferments, in suppurating wounds, with their other toxic products dissolve the intercellular cement and destroy the vitality of many of the cells, reducing them to a fluid tissue—*pus*—thus detaching the dead fragment. In aseptic wounds the same results follow from a more gradual loss of vitality, solution of cement and cells producing disintegration of the cellular bond of union. When the dead bone is removed the remaining granulations go on to cicatrization—*i. e.*, ossification. I need hardly point out how exactly alike are this "sloughing of bone" and the sloughing off of a piece of dead cellular tissue.

LECTURE V.

INFLAMMATION A TRUE MICROBIC PROCESS.

I HAVE now put you in possession of all the facts requisite for a clear understanding of the phenomena of inflammation, which may be defined to be the series of results caused by microbic interference with the normal processes of repair in injured, living tissues. These results usually for a time lead to the destruction of tissue, but are ultimately conservative in their tendencies, the end-results being the removal or neutralization of the microbic cause.

Microbes produce their injurious effects indirectly; first by bacterial proteids, possibly liberated from the germs only during their degeneration; and second, the presence of these proteids leads to the production of toxic substances—toxalbumins, etc., in the tissues themselves from perverted metabolism, hence the presence of germs themselves is not essential for the evolution of inflammatory phenomena, but substances containing ptomains, toxalbumins, etc., will produce all these, until the toxic substances have exhausted their power to harm, when the processes will cease, unlike what is seen where living germs are present, which continually produce new supplies of toxic substances.

It is far too generally accepted as the teaching of modern science that the presence of germs invariably means inflammation, or the disease which the microbe is the cause of, for instance, tubercle. This misapprehension is the foundation of much of the confusion existing in the minds of students in their comprehension of pathologic phenomena and methods of wound-treatment,

and must be abandoned at the outset of the study of inflammation.

To produce their characteristic effects germs must be present *in sufficient numbers to overcome the vital resistance of the tissue.*

Immunity.—The relations borne by micro-organisms to the production of inflammation can be best ascertained by first studying how the tissues safeguard themselves under ordinary and extraordinary circumstances, in other words how they dispose of germs or render them inert. Only a few words can be devoted to the question of immunity, as it bears upon tissue-immunity and resistance. Hankin's definition, aided by a few illustrations, will suffice for our purposes.

"Immunity, whether natural or acquired, is due to the presence of substances which are formed by the metabolism of the animal rather than that of the microbe, and which have the power of destroying the microbes against which immunity is possible, or the products on which their pathogenic action depends." Observers have extracted certain substances—"defensive proteids" —from the livers and spleens of animals, capable of destroying bacteria. These are never found in normal blood ; but when the febrile state has supervened, these substances, in an active state, are detectable in the circulating fluid. Blood-serum is well known to be germicidal in virtue of the nucleinic acid it contains, dissolved out of, or resulting from the disintegration of the phagocytic leukocytes.

The reaction of tissues to germs is never exactly the same in any two individuals. The differences clinically observed are probably explainable as follows:

From the analogies presented by the differing vulnerability of the lower animals to certain infections, and our knowledge of the relative immunity present in certain peoples to some forms of disease, we may certainly infer that there is such a thing as inherited racial immunity. It is also a matter of common observation that some in-

dividuals, although frequently exposed to an infection, escape again and again, but finally do succumb. Probably in such cases the individual's tissues, having triumphed over previous slight infections, continue to produce substances inimical to the growth of the same germs if again implanted in large numbers—*i. e.*, an *antitoxin* or antidote to the toxins produced by the germs is formed. This power may gradually cease in time or may suddenly be destroyed. Thus, among the lower animals the adult white rat is naturally immune to anthrax. Change, however, the conditions of cell-nutrition in the same rat, by inducing extreme fatigue, and the blood becomes overloaded with the products of retrograde tissue-change which so depress cell-vitality either by directly poisoning the cells, or by interfering with physiological metabolism, that the normal defensive proteids are no longer formed and the animal can now acquire anthrax. This is frequently paralleled in the human subject. It is a matter of common remark how troops resist disease in the earlier stages of a campaign when later, after the extreme physical exertions of forced marches, etc., they readily yield to an infective exposure which is comparatively slight. Lowering of tissue-resistance may then be brought about first, by deficiency in the amount of pabulum available for the cells, so that sufficient defensive proteids are not formed ; and second, by the presence of substances in the blood, in the tissues, or in both, which are capable either of actually destroying the cells or so altering metabolism that their products, far from supplying defensive materials against germs and toxic substances, rather add new poisons to those already present.

Certain tissues are normally deficient in pabulum from their poor blood-supply, and hence are peculiarly vulnerable to infection. Anatomical structure sometimes favors infection. The almost sinus-like arrangement of the blood-vessels of the epiphyses of growing bones favors the accumulation of germs, because the velocity of the

current is lessened. This explains the frequency with which such portions of the bones are attacked in acute infectious and tubercular osteomyelitis. In contrast with the lack of resistance shown by normally avascular tissues is the well-known resistance offered to severe infection by very vascular parts, such as those of the face. The tissues then can only maintain their normal resistance by having an abundant blood-supply ; but this must move at a normal rate, in vessels of a certain caliber—although these conditions may vary within somewhat wide limits —otherwise germs will, for purely physical reasons, accumulate in overwhelming numbers. Still further, if this blood does not move at a proper rate it will not promptly carry away the poisonous products of cell-metabolism, which will otherwise directly injure the cells. Again, this poison-laden, because sluggishly moving blood may incite the tissue-cells to abnormal metabolism productive of toxic substances, even in the absence of germs, which when absorbed will produce most serious constitutional effects.

Although when the tissue-cells, the hemopoietic organs, the blood, and excretory organs do their whole duty, complete tissue-immunity is secured under ordinary circumstances, are there no reinforcements which they can call upon in an emergency? Yes. Some of the leukocytes, which, as we have seen, soon crowd into any injured part, the mono- and poly-nucleated ones, are capable and eagerly embrace the opportunity of encapsulating dead or dying cells, detritus of the same, and germs or spores. The phagocytes, as they are called, are often unable to cope with the adult germs, dying in their efforts, but can encapsulate, destroy, and digest, or mechanically remove spores by virtue of their ameboid power. The wandering tissue-cells and the tissue-cells themselves also exert this phagocytic action, especially the descendents of the fixed connective-tissue cells (the fibroblasts), and the endothelia of blood- and lymph-vessels, the latter being less active. This accounts for

the mural implantation of germs seen during infection from germs circulating with the blood, which, although unfortunate, is a protective effort upon the part of the vessel-cells. The degree to which the tissue-cells can exert this protective power depends upon the degree of perfection of the nutrition of the cells themselves. It is believed that phagocytes exercise a selective power as to the forms of organism they attack. Thus the polynuclear cells will take up and digest streptococci and gonococci, neither of which are attacked by the mononuclear forms (Park).

Chemotaxis.—Why do phagocytic cells accumulate in a part attacked, rushing like soldiers summoned to beat off an assault? Because of chemotaxis. What then is chemotaxis? It is the mutual attraction or repulsion possessed by animal or vegetable cells for one another, or of an animal cell for a vegetable one. When two cells tend to approach one another, or one immobile cell causes a mobile cell to move toward it, "positive chemotaxis" is said to exist; when a mobile cell is repulsed—*i. e.*, moves away from another cell or substance—"negative chemotaxis" is said to have been operative. Thus the vegetable cell, the bacterium, attracts an animal cell, a leukocyte. Mobile bacteria move toward pabulum, and phagocytes are peculiarly attracted by the albuminous material composing or set free from bacteria. The disintegration of tissue following aseptic wounds or other traumatisms sets free proteids which attract phagocytes. These remove the detritus as well as effect the solution of living tissues where reparative cellular exudate is forming.

I must here again impress upon you the possible disadvantages of the hyperemia of repair if excessive, and explain how it may be possible to prevent the successful secondary implantation of germs when such hyperemia exists, thus converting what would inevitably become an inflammatory into a purely reparative process. This is an appropriate place to

introduce in advance certain statements, because the therapeusis of inflammation depends largely upon a recognition and application of similar facts, and also because hyperemia is just as much a part of inflammation as it is of repair. Although hyperemia, however excited, at first serves to remove detritus, poisonous metabolic products, and to bring phagocytes, thus improving tissue-nutrition, eventually a slowing of the blood-current must ensue, and any germs reaching the circulation through some distant infection-atrium will tend to drop out.

The exudate supplies abundant pabulum for the germs while it compresses the plasma-channels, thus interfering with the nutrition of the cells; moreover, metabolic products are now retained in the part. In chronic hyperemia even varicosity of the vessels has been observed, than which no better trap could be devised for germs. If we can secure diminution of the caliber of the blood-vessels and thus increase the rapidity of the current, germs will not be so able to collect, those present will be swept away, exudate will be removed, and tissue-resistance increased from the improved nutrition.

You will recall that when considering hyperemia, the minute processes were first described and then the clinical symptoms. I shall reverse this plan and consider first the clinical signs presented by inflammation of a superficial part, and later explain the variations observed in the minute processes of repair, produced by the injurious action of germs and their products.

In the first place never forget that, just as repair is effected by the same processes in every tissue, so is inflammation the same in the hardest as well as in the softest tissue—that most vascular and that without any direct blood-supply.

Symptoms of Inflammation.—The first symptom of inflammation of a superficial part usually recognized is redness (*rubor*). This is at first uniform in tint, disap-

pears upon pressure, and fades out into the hue of that of the surrounding tissues. Later it is darker at spots— *i. e.*, it is mottled—these darker spots not disappearing upon pressure. Surrounding the periphery of the inflammatory focus there is an area of the tissues which steady pressure by the finger-tip will indent; they "pit" from *edema* as it is called. What causes the uniform redness? Hyperemia—*i. e.*, the blood-vessels are dilated and contain more blood than normal. How is the mottling to be explained? By the escape of red cells into the tissues by rhexis from the capillaries, or by the rupture of some of the vessels. Why do the surrounding tissues pit upon pressure? Because of the early, free escape of serum from the distended blood-vessels and later of liquor sanguinis. The lymph-nodes through which lymphatics draining the inflamed area pass are enlarged and tender, while the lymph-vessels, if cut, give exit to a much larger amount of fluid than can be obtained from those of a corresponding normal part. Do these phenomena vary in kind from those observed during the hyperemia of repair? Not in any important particular. It is true that escape of red cells is infrequent in hyperemia, and that any edema noticed is rather from the escape of blood-serum than liquor sanguinis; but all this can and does occur, the difference being only in degree and not in kind, every phenomenon reaching its maximum in inflammation.

Both to the hand and to the thermometer an inflamed part is warmer than normal (*calor*), but it is never hotter than the blood in the left ventricle of the patient, in fact is not so high. Owing to the increased registering power of the nerves, the sensation of heat as experienced by the patient is far in excess of the actual elevation of the temperature. This increased heat results not from any chemical changes in the part, but from the rapid flow of blood through the dilated vessels bringing caloric more rapidly than it can be dissipated by radiation.

The bulk of the part is increased, and swelling (*tumor*)

has taken place. This is due in a slight degree to excess of blood, but in the main to escape of fluids from the blood into the tissues and the migration of the corpuscular elements during the earlier stages, while, later, proliferation of tissue-cells adds another increment. This accumulation of materials separates the different tissue-layers, thus stretching the nerves, which are also compressed by the exudate, so that pain (*dolor*) becomes a prominent symptom. The pain is doubtless increased by the chemical irritation of the toxic substances produced by the germs, and the exalted sensibility of the nerves themselves, which are also hyperemic—*i. e.*, rendered abnormally capable of recognizing impressions, which in this case means pain.

These physical alterations of the tissues, or organs composed of them, of necessity interferes with the easy performance of the functions of the parts or actually arrests them (*functio læsa*).

Again, except in degree, has anything yet been mentioned which cannot be matched by the hyperemia of repair? Assuredly not; and up to this point the two processes are identical, whether excited by efforts at repair or by agents destructive of the tissues. From this point the two processes diverge and the phenomena peculiar to inflammation supervene—*i. e.*, those which interfere with and thwart nature's reparative efforts.

Microscopic Changes.—A few words can be profitably devoted to a consideration of the microscopic changes involved in the production of the coarse phenomena just detailed, because I wish to emphasize again the folly of the artificial distinction made between "the hyperemia of repair" and that of "inflammation." Thus, when inflammation is excited there is first acceleration of the blood-flow, then slowing of this; the leukocytes drop out of the axial current, roll lazily along, collecting until a peripheral layer of leukocytes forms in the venules and capillaries. Leukocytes crowd into the tissues, serum and liquor sanguinis also escap-

ing, and finally stasis occurs with coagulation of the exudate, including the blood in the vessels where circulation has been arrested. In the aseptic healing of a wound where none claim the presence of germs, have I not described all this, especially the crowding of the wound-margins, clots, and dead tissue with leukocytes, and even coagulation of the exudate? Because it is impossible to follow these minute processes continuously by the microscope for many days, an artificial inflamma-
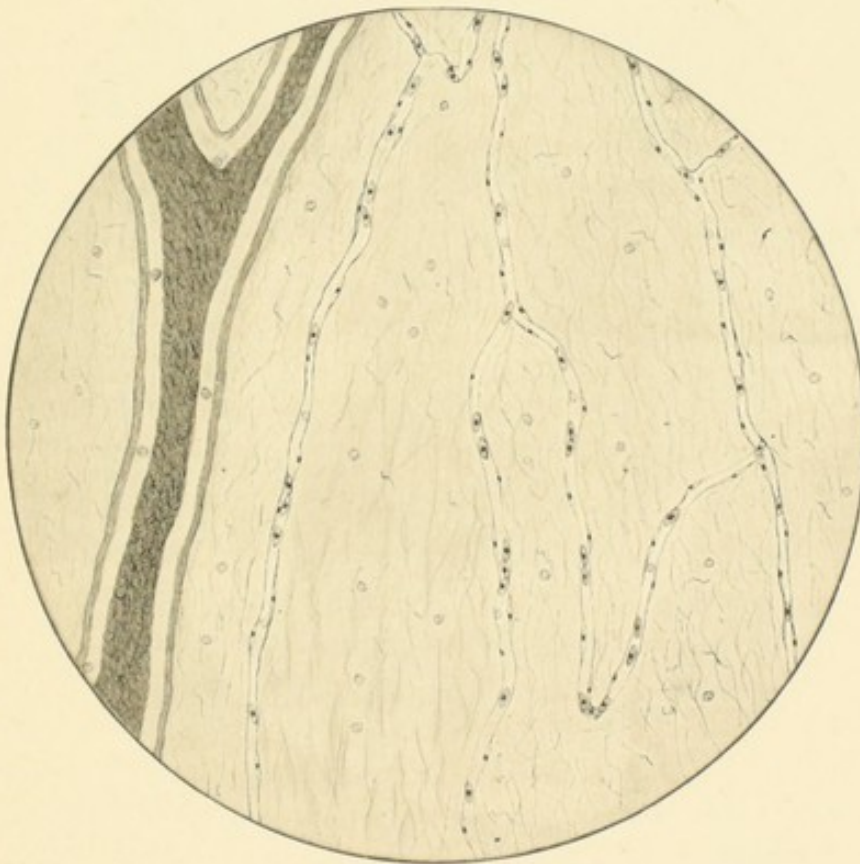

FIG. 7.—Showing normal capillary circulation in mesentery of frog.

tion can be observed only for a few hours at most. Despite what has been seen when examinations have been made at intervals of hours and days, it is still insisted upon by some, that as similar appearances are presented during infective processes, both should be considered inflammation, although one is purely conservative, the other destructive. Still further, because during

the earlier stages of experiments where eventually true (infective) inflammation does occur, leukocytes migrate into the tissues, it is contended that leukocytes only escape from the vessels during true (infective) inflammation ; this is entire forgetfulness of the laws of chemotaxis, and an ignoring of the conditions actually seen during the aseptic healing of a wound in which these
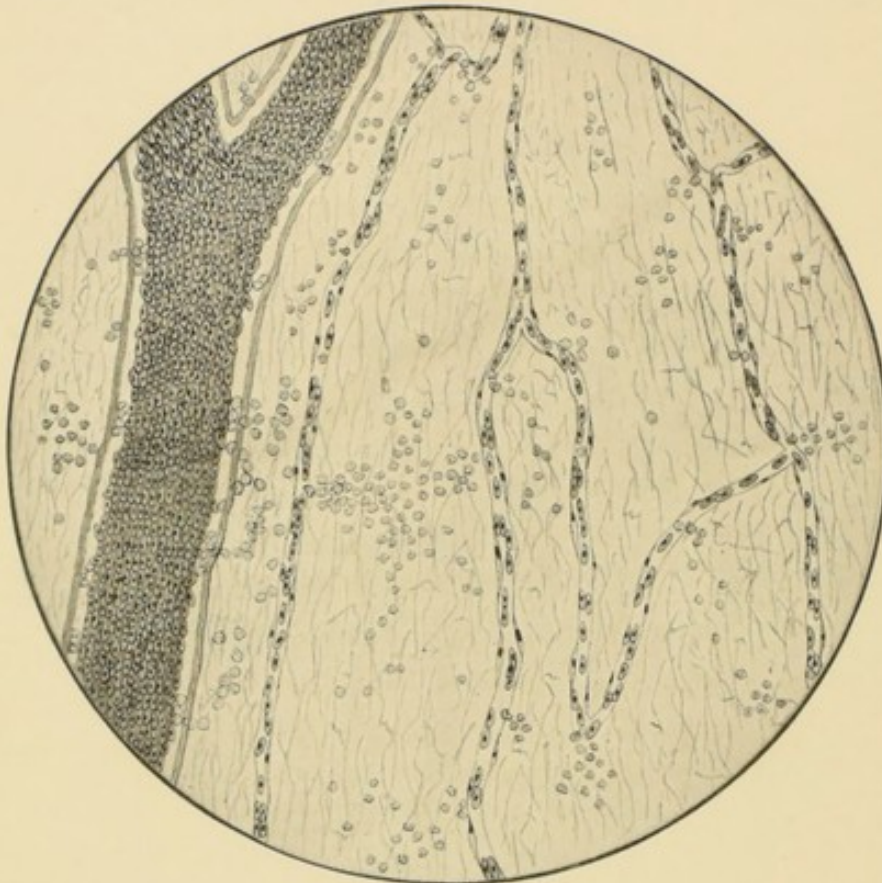
FIG. 8.—Showing acute hyperemia, induced by mechanical irritation, in mesentery of frog. Stage of dilatation of vessels, retardation of blood-current, and exudation with diapedesis of white cells.

same observers declare no germs are present. Moreover, the simple expedient of securing perfect asepsis, damaging a tissue, thus inducing hyperemia, seeing the white cells migrate into the surrounding tissues, and proving that no germs were present by culture experiments, has been entirely neglected, until at my suggestion Dr. Spitzley recently did all this. All this iteration and reiteration in my judgment is demanded, because a

clear comprehension of my position can alone reconcile
the confusing statements of many of our books and
teachers, which when carefully sifted amount to this :
germs are the sole excitants of inflammation ; migration
of leukocytes is seen only during true inflammation,
which has just been declared to be a germ-process ; yet
wounds are said to heal by aseptic (non-microbic) in-
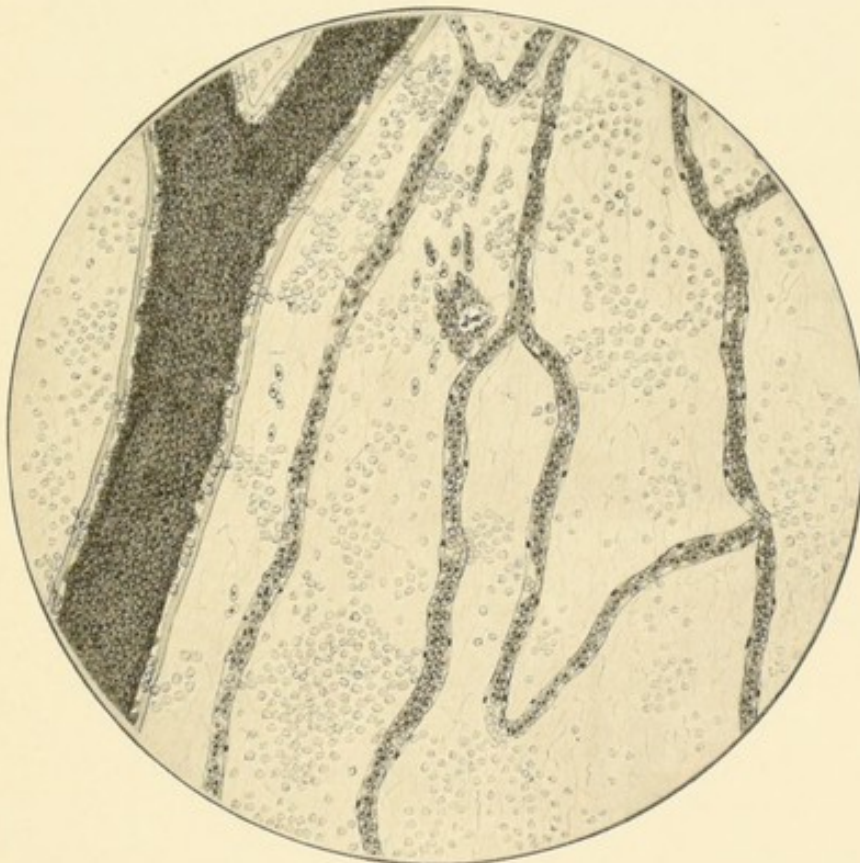
FIG. 9.—Showing inflammation (infective) in mesentery of frog.   Stage of
stasis with exudation and diapedesis of both red and white cells.

flammation, and further it is insisted upon that the
migration of leukocytes with all the processes seen in
a microbic inflammation, except the destructive ones, *do
occur in the aseptic repair of wounds.* Migration of
white cells then does occur, nay must occur during the
hyperemia of repair ; but owing to the immensely
greater chemotactic power of bacterial proteids over
those of dead or dying tissues, this phenomenon is much

more marked in genuine (microbic) inflammation, while the later destructive processes really constitute the true essence of inflammation. The pseudo-neoplastic growths induced by the tubercle bacillus, the poison of syphilis, etc., said to be the products of "inflammation," need not be considered here, since I am now only contending for the truth of the proposition that aseptic wound-repair should not be called "inflammation," and that the essence of microbic inflammation is the interference exerted by it upon the processes of repair.

LECTURE VI.

INFLAMMATION (CONTINUED): DIAPEDESIS OF WHITE CELLS, ETC.

LET us partially retrace our steps and recapitulate the causes of the phenomena of so-called "aseptic inflammation" and "aseptic fever," the former of which I have shown you is not inflammation but repair, so that we may better understand the resemblances presented by the two processes and the point where divergence commences. I will take for illustration the tissues of a sprained joint, where no question of microbic action can be dreamt of, and shall consider only those details essential for the comprehension of certain results which have not yet been dealt with. Thus, while the stasis does not necessarily terminate in thrombosis of the vessels unless the stasis is maintained for some time, yet thrombosis with coagulation of the exudates in the tissues will occur unless the circulation recommences. The effused serum and liquor sanguinis are drained away by the lymphatic vessels, which pass through lymph-nodes. These latter almost never become enlarged, as in microbic processes, and never suppurate, but may in rare instances be tender.

The nucleins and tissue-detritus removed by the lymphatics and poured into the venous blood produce elevation of temperature and some increased frequency of pulse—"aseptic fever;" but these symptoms are usually only detectable because sought for, the tongue being not dry, often but slightly furred, and the secretions of the skin, intestines, and kidneys not diminished in quantity or materially altered in quality, none of which can be affirmed of them in true inflammation. The extent to which temperature and pulse are increased

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is dependent upon the quantity of aseptic pyrogenous substances that are absorbed, hence cannot be pronounced after a slight injury, require a considerable surface for their absorption, and the symptoms commence just as soon as any shock present passes off.

A "restitutio ad integram" is brought about in the areas around the lesion which is being repaired; the so-called inflammation "terminates" in one of two ways. When the stasis is very recent and of limited extent all the circulatory and parenchymatous changes are reversed, the central mass of red cells begins to oscillate, the range of oscillation increases, the current recommences, the peripherally located white cells mingle with the central column of red cells, the migrated cells wander into the lymph-spaces (possibly into the blood-vessels) or become disintegrated and are absorbed together with the excess of fluids, leaving the part normal. When very rapidly effected this was what was formerly called "delitescence," because all the induration rapidly melted away, as it were. When, however, extensive removal of tissue by phagocytosis, with its substitution by numerous young formative cells, must of necessity follow thrombosis of the vessels with coagulation of the exudate in the tissue, the removal of the excess of cells, with the development of those which remain into permanent tissue, is a much slower process, requiring disintegration of the cells—a *re-solution* of the exudate; this termination of inflammation is appropriately called "resolution." Resolution may be perfect or imperfect, in which latter event the tissues are rendered denser, by the development of some of the cellular exudate into young connective tissue— *i. e.*, scar-tissue. Induration, inflammatory thickening, cirrhosis, according to the stage of the process or the organ attacked, is now said to have followed the inflammation, or as I should say, the excessive and prolonged hyperemia of repair. No doubt, under the term "resolution," there is often included not only conditions where, with temporary stasis, there has formed too much

cellular exudate for the majority of the cells to migrate and most have to be slowly disintegrated, but also in some instances, where mild genuine (microbic) inflammation has been present, the tissues so quickly gaining the upper hand that the processes of repair are promptly instituted.

I shall once more explain the minute processes taking place in the depths of the tissues during "resolution," because it may seem that something different takes place from that which is seen upon the surface ; but in future my statements about repair must be taken without explanation.

The stasis becomes permanent, the blood coagulates in the vessels, and thrombosis follows. The fibrin-forming constituents present in the effused liquor sanguinis and cellular exudate combine, coagulation occurring, rendering a return to the normal impossible after the manner of delitescence. The cells of the involved area, from the original injury, plugging of the vessels, and accumulation of fluid and cellular exudate, are deprived of their vitality or starved, so that they perish, and their solid detritus is removed by the phagocytes, leaving, instead of the tissue, a mass of densely packed formative cells, descendants of the connective tissue, endothelial cells, and perhaps some of the later appearing leukocytes. Vascularization is next effected as has been described for surface-wounds, and now we have "granulation-tissue" in the depths of an organ, for the granulation form is not the essence of granulation-tissue but an accident, granulation-tissue really meaning a mass of vascularized, young, formative cells, which if undisturbed will inevitably develop into cicatricial tissue. If disseminated fibroid thickening results, and if this occurs at one spot only, a subcutaneous scar is formed, capable of all the untoward consequences already described as following the contraction of a surface-scar.

No difference then exists between what is seen during normal repair and infection up to the point we have

reached ; but if pyogenic infection has caused the changes, or if this now occurs, all the processes are rapidly completed; peptonizing ferments are formed which dissolve the intercellular cement, separating the cells one from another, thus preventing nutrition reaching them.   Other toxic substances aid in destroying the cells, or coagulation-necrosis occurs in many instances, fatty degeneration and other retrograde changes follow, and what remains of the exudate, reinforced by dead phagocytes with a few living leukocytes, forms a fluid mass—*i. e.*, *pus*.   The physical change from a mutual relation of the cells to their separation one from another, prevents their vascularization ; but even could this be effected, the dead and dying cells could not avail themselves of the pabulum. The destructive processes manifest themselves by certain clinical phenomena to be described later.

" *Infective inflammation* " has been the term employed of late years to express the conditions resulting from the presence in the tissues of germs—or their products—in overwhelming amounts, but I have shown you two facts —viz., that when these are both absent after traumatism, repair and not inflammation results, and that, even if present, they must be in sufficient numbers to overcome the vital resistance of the tissue-cells.   It is true, as I have said before, that the introduction into the tissues of a number of germ-free, irritant substances may be followed by the collection of a *puruloid* fluid ; but this is not *pus*, as proven by its injection into an animal, neither general infection nor any of the destructive effects of true pus following.   This fluid consists of an accumulation of leukocytes and phagocytes resulting from chemotactic action.   Moreover, even if pus could thus be produced, the laboratory conditions requisite for success are never present clinically.   Do not forget that what really causes the destruction during inflammation are the *germ-products*, not the *germs*[1] themselves.   The presence of these substances, although the germs pro-

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[1] Pyogenic organisms are alone referred to.

ducing them are absent, will initiate the phenomena of inflammation. Stings of insects, bites of venomous reptiles, etc., may later present all the evidences of true inflammation from the effects of toxic alkaloids, ptomains, etc., analogous to those formed by germs, from the simultaneous inoculation with pathogenic germs, or from secondary infection ; but at the outset the conditions are often identical with those resulting from the presence of aseptic turpentine in the tissues.

Although this would seem to be an appropriate place in which to describe the varieties of germs, their culture methods, and the means of distinguishing between them, yet since most of those who will read these pages have been carefully instructed in bacteriology, it will be unnecessary. Moreover, in these lectures upon the Principles of Surgery, I can deal only with the general laws governing the action of germs, mentioning special instances for illustrative purposes only.

The characteristic effects of germ-products upon the cells of the exudate and those of the tissues are the prevention of the former from undergoing conversion into permanent tissue of a higher grade, and the destruction of the latter. This is true of the effects of the tubercle bacillus as well as of those of pyogenic organisms. In the former, coagulation-necrosis, fatty degeneration, caseation, followed perhaps by liquefaction into a puruloid fluid, take place ; in the latter, disassociation of cells by peptonizing ferments occurs—often preceded by a coagulation-necrosis, fatty and retrograde changes in the cells take place, and true pus is formed. These changes affect the majority of the cells, although some remain immature for long periods, but later they develop into scar-tissue. Although the most common result of pyogenic infection is suppuration, yet from what is sometimes observed clinically, it would seem possible that very small quantities of pus may be formed as the result of a limited infection, the germs be removed by phagocytes or destroyed by nucleins, and the dead cells be absorbed. This would be

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difficult of proof, but it is directly in line with some of the facts I have just related.

Pus consists first of a liquid portion (liquor puris), composed of the liquefied cells, intercellular cement and liquid exudate, and second, of pus-cells—*i. e.*, dead or dying leukocytes and germinal cells—which have perished in attacks upon the germs or have been killed by the germ-products. Suppuration occurring on a free surface, *with loss of substance*, is called *ulceration ;* when occurring in the depths of a tissue, and *circumscribed*, it forms a "purulent collection," or "abscess."

The acuteness of an "inflammation "—*i. e.*, the rapidity with which the pathological processes reach their acme—depends upon three things—viz., the character of the germs, the number of germs present, and the resistance of the tissues. Thus, the micro-organisms of suppuration—*aptly termed pyogenic*—rapidly induce the formation of pus. The inoculation of the micro-organism of glanders results in a subacute process—possibly a chronic one—while the bacillus of tuberculosis usually sets up chronic changes.

Approaching the defensive power of the tissues from another standpoint, we shall see that the portion of the old view which maintained that a form—the so-called "healthy inflammation "—was conservative, an effort to cast out or destroy something injurious, was correct. Premising that the stages of inflammation are arbitrary divisions, it must be admitted that the early hyperemia of a pyogenic inflammation, with the increased quantity and rapidity of the blood-flow, must tend to sweep out accumulating germs and prevent their implantation ; hence the good results which follow, at this stage only, the use of those therapeutic agents which increase the rapidity of the blood-current. During this period the result is in doubt, for the tissues may, unaided, conquer, and when intelligently assisted, not thwarted, often do triumph over the germs. The second stage, during which the multiplying germs overcome the enfeebled resistance of

the tissues, terminates in destruction of the central cells, with peripheral extension of the cellular infiltration and conversion of the tissues into embryonal tissue, until the exciting cause ceases to be operative. The lack of resistance of the embryonal cells is characteristic of immature tissue, because they must develop and also form protective proteids—a double task which the most trivial physical or vital disturbance renders impossible. An increasing number of phagocytes now crowd into the peripheral zone; the uninterrupted and abundant vascular supply here present enables some of the cells to progress toward development, and vital resistance is thus gradually increased, until the growth of germs is so inhibited and their toxins so neutralized that repair commences. Of course, evacuation of a purulent collection loaded with micro-organisms is one of the most important means of arresting pyogenic processes; but even after this has been done, the germs and their products in the tissues at the periphery must be disposed of, and they are dealt with after the manner just described. The cells around the pus-focus, having thus maintained their vitality and being properly supplied with blood, form a boundary layer of granulation-tissue ready for repair.

During the final (third) stage of "inflammation," the microbes having been entirely disposed of, or being in process of removal by phagocytes, all toxic substances being now rendered inert by the exalted resistance of the tissues, repair is rapidly effected, a superabundance of nutriment uninterruptedly reaching the cells from the many newly formed vessels. In certain chronic infective processes, as in tubercle, under favorable circumstances this tendency toward repair is so strong, that when retrograde degenerative processes are prevented, the tubercular granulation-tissue in whole or in part becomes converted into scar-tissue.

Severe injuries do not tend to produce localization of germs present in the circulation, owing to the large number of the phagocytic leukocytes which collect in the

injured part, and the amount of nucleins present. In bone-tubercle or acute infectious osteomyelitis, in both of which ailments the germs unquestionably reach the osseous tissue solely by way of the circulation, a slight blow or wrench, by lowering the vitality of a few cells, and the slight hyperemia following thrombosis of a few minute blood-vessels which have been ruptured, will enable the microbes to drop out and multiply in this locus minoris resistentiæ. This is because the hyperemia is too limited to admit of the prompt arrival and accumulation of phagocytes. Mark the difference in the case of an extensive infected wound. Here multitudes of germs are implanted, and despite the intense hyperemia induced, phagocytes in sufficient numbers cannot accumulate in time ; it requires the lapse of days or weeks before the defenders can even prevent the further increase of their foes, much less overwhelm and destroy them. Chronic infective inflammations, such as those produced by tubercle and syphilis, by preparing a favorable soil—*i. e.*, a low grade granulation-tissue—predispose to the implantation of pyogenic organisms, which secondary infection accounts for much of what is ignorantly set down to the uncomplicated effects of those diseases.

As yet, little beyond the local defensive power of the cells has been considered. In a general way the germicidal power of the blood-serum and the phagocytic blood-leukocytes has been mentioned ; but it must not be thought that many, much less all, the germs reaching the blood-current are thus disposed of. Large numbers of pyogenic organisms when introduced into the circulation promptly disappear therefrom, lodging in such organs as the liver, spleen, and kidneys, where some are destroyed, by methods already described, and others are eliminated with the urine, probably with the bile, and certainly from the intestinal tract. Whether the germs eliminated in the intestinal discharges are directly excreted by the intestines themselves, or escape with the bile and pancreatic secretions, is at present in doubt.

The allegation that microbes are excreted by the sweat and salivary glands, while likely to be true, would seem hardly susceptible of proof, because pyogenic organisms are so constantly present in health between the superficial layers of the epithelium of the skin and upon the surface of mucous membranes. Unquestionably the toxic materials formed by germs can be eliminated by the kidneys, skin, and intestines, and possibly by other emunctories. This emphasizes what I have been contending for during the last twenty-five years—viz., that during infective processes much may be done by stimulation of these various emunctories, death of the patient often resulting because these organs cannot be made equal to the double task of excreting toxic substances as well as the products of tissue-metamorphosis. Too often the practitioner fails to recall the advice of his much scorned "old-time" predecessors, to "clear out the primæ viæ, to stimulate the emunctories, and to maintain their secretions free," the scientific basis of which advice is now freely conceded.

LECTURE VII.

INFLAMMATION (CONTINUED): PREDISPOSING CAUSES OF INFLAMMATION.

IF my previous statements have been founded upon facts, not theories, as I have contended, inflammation can only terminate by either suppuration, ulceration or resolution ; gangrene, or mortification, as will later be shown, being not a "termination," but an accidental consequence or result of inflammation.

The essential cause of the abnormal penetrability of the vascular walls by fluids and cells probably is a molecular change effected in the vessel-walls by the products of germ-growth.

Have I yet put you in possession of all the preliminary knowledge requisite for an intelligent study of the treatment of inflammation? Certainly not. Why is it that of two men who have each suffered from a similar injury one recovers without any unpleasant consequences, and the other has an abscess form? Still better, study the effects of contusion of a bone before and after an attack of typhoid fever, pyogenic organisms being accidentally present in the blood on both occasions. Before the fever nothing beyond a temporary soreness follows even a severe contusion ; after the fever, an osteomyelitis results, which has no relation to the typhoid bacillus. Clearly, if pyogenic organisms were the causative factors in the second instance, and they were equally present on both occasions, why, when the local impairment of nutrition was greater because of the severe bruising, did no localization of germs follow? Something else besides micro-organisms, and something which was present before the traumatism which co-operated with and re-

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inforced the effect of the germs, is requisite to explain the results.

Certain conditions which favor the implantation and development of germs are called **"predisposing causes of inflammation."** In the supposed instance the tissues were incapable of normal metabolism and the formation of defensive proteids, from having been supplied with blood containing the toxins of typhoid fever. Even when these ceased to be formed, the amount of the nutriment in the blood was deficient ; hence malnutrition of the cells resulted, and finally this poor blood was propelled by a feeble heart, so that anything favoring local hyperemia would become effective in favoring or causing such a slowing of the circulation as would render certain the accumulation of pyogenic microbes. A slight blow which would be incapable of exerting any such effects in health will be quite sufficient to initiate stasis aided by such predisposing causes as have just been detailed. Let this illustration suffice, for in future I must confine myself to general statements with regard to the modus operandi of "predisposing causes." In order then to treat inflammation properly, it is absolutely requisite not only to understand that any interference with access of the ultimate nutriment to the cells diminishes the resistance of the tissues to germs, but that general conditions influence the distribution of the pabulum which the blood contains to the cells, and also that the quality of the food influences these conditions. Therefore we cannot ignore the general state of the patient and address ourselves solely to improving the local conditions, because this may either be impossible, or all may have been secured that can be effected by local measures, while the local nutrition can yet be modified by changes in the force of the current and bulk of the blood supplied to a part, and by the quality of that blood.

Predisposing causes may, for convenience of description, be classed under two heads, although there is no sharply dividing line.

1. **Deficiency in Quantity of the Blood.**—This results from causes which physically induce such a dilatation of the blood-vessels as will favor a slowing of the current, less blood passing during a given time through the part ; hence its nutrition suffers, and with this the resistance to germs. This slowing of the current permits the accumulation in the damaged part of any germs which may be circulating in the blood, just as mud is deposited by the rapid mountain torrent when it reaches the valley and forms a broad, languidly moving stream.

Deficiency in the quantity of blood reaching a tissue results from a vis-a-tergo which is below the normal— *i. e.,* the rate and quantity of blood passing through the tissues are diminished. This results from weakness of the heart which may follow hemorrhage, lack of general nutrition, disease of the organ, or senile changes, the last of which, together with diminished elasticity of the vessels, account for the greater part of the bad repute of age, as predisposing to slow repair, inflammation, and gangrene. Sometimes loss of elasticity of the vessels from atheroma or calcification is the sole or chief cause ; sometimes it is a fibroid heart, though more often both coincide ; but if neither condition is present, advanced years do not produce malnutrition of the cells, hence do not predispose to inflammation, while in comparatively young individuals, when these conditions exist, the tissues are ill-nourished and incapable of resisting the attacks of germs. It is a most important point to keep constantly in mind that it is certain conditions, usually obtaining in old age, which produce imperfect nutrition of the tissues, not the mere years numbered by the patient, and that similar changes can occur during comparative youth. Thus, one of the best marked examples of atheroma and calcification of the vessels I have ever seen, was in a man of thirty-nine years of age whose tissues, in consequence, resembled those of an aged man. Other conditions favor poor nutrition in the aged, notably imperfect digestion ; but I must insist that age, per

se, does not necessarily mean imperfect tissue-nutrition. The importance of recognizing the rôle played by a lessened vis-a-tergo is that this condition is capable of betterment by the administration of food, stimulants, etc.

Defects in the quality of the pabulum are due to insufficient or improper food, to anemia however induced, and to the presence of toxic materials. Thus the laborer who, owing to a strike, for instance, actually has too little food, and the individual who has enough food, but of a kind that his digestive organs cannot convert and appropriate, both have their tissue-cells insufficiently nourished, because the blood does not contain the requisite amount of pabulum. In both also, the heart-muscle, in common with other organs, is so weakened that the normal vis-a-tergo is diminished, and thus, in addition, a deficient supply of blood to the tissues results. Again, fatty foods may be indicated, as for instance, cod-liver oil in tubercular affections, to increase the assimilability of albuminous foods and supply hydrocarbons, thus improving nutrition ; yet this remedy often arrests digestion and, causing diarrhea, instead of improving the patient's condition, institutes a new drain upon him. To illustrate the importance of recognizing such causes of imperfect tissue-nutrition, let me tell you that an occasional dose of calomel will render it possible to administer the oil, or, if, failing by this method, inunction is employed, *suppuration will diminish or cease*, because the cell-nutrition has been increased, which is the same thing as saying *that resistance to germs has been increased*. The injurious effect upon the tissues of retained excrementitious substances, which have either been imperfectly elaborated or are in excess, although possibly normally present in the blood, is seen in the deposits of sodium urate so common in gout. In like manner the tendency to inflammation so often seen in serous membranes as an end-result of chronic renal—or even cardiac—affections, equally de-

monstrates the unseen, but tangible evil effects upon cell-nutrition of retained excreta.

The presence of certain chemical substances in the circulating fluids creates conditions unfavorable for tissue-nutrition, hence plumbism, mercurialism, and phosphorus poisoning are well-known predisposing causes of inflammation. Reasoning by the analogy presented by diabetes, a disease in which pyogenic infection is so common, we may infer that the presence of certain substances renders the pabulum more suitable for germs, as has been demonstrated for some species by showing how much more luxuriant the growth of pyogenic organisms is after the addition of diabetic sugar to the culture medium.

Trophic changes, which are alleged to be capable of initiating inflammation by themselves, are really primarily alterations of the vascular tonus through the medium of perverted nerve-influence, or as the result of the withdrawal of all nerve-influence. This prepares the tissues in such a way for the implantation of germs, that infection so readily occurs that the process seems almost the spontaneous resultant of altered nerve-influence. Very little scrutiny, however, is necessary to show that vasomotor changes, insensibility to pain, and pyogenic infection, account for bed-sores, etc. The sluggishly propelled blood in a paralyzed part is readily expressed by the pressure it is subjected to, which is possible because, no pain being felt, the patient is content to permit injurious pressure to continue indefinitely. When this is now removed, any vascular tonus which previously existed, or would be instituted by the perivascular ganglia, will be found to have been destroyed by the pressure, hence partial or complete stasis, thrombosis, gangrene, at least malnourished tissue results, which readily succumbs to even the feebly infective germs normally resident in the skin-epithelium, or to the more virulent ones accidentally present.

I must here devote a few words to the purely mechanical effects of acute hyperemia, which, although already

mentioned, have not been emphasized as I wish. Although conservative at the outset because tending to sweep out germs, prevent their accumulation, remove metabolic and microbic toxins, and bring phagocytes and abundant nutriment, yet all these advantages of hyperemia are soon reversed by the inevitable changes which follow. As the circulation becomes retarded, the increased amount of blood with the exudates compresses the plasma-channels, preventing the proper access of pabulum to the cells ; the blood becomes surcharged with metabolic and microbic toxins, which means that the pabulum which does reach the cells is actually harmful to them, while the failure to remove the injurious metabolic products of cell-activity is a physical result of the saturation of the tissue-fluids and the blood. This, combined with the diminished or actually arrested current, interferes with osmosis. All these conditions, consequent upon a persisting hyperemia, mechanically result in a concentration of microbic and metabolic poisons in direct contact with the cells, instead of diminishing this as the hyperemia at first did.

Chronic hyperemia can never be said to be conservative, but is obnoxious to similar charges, as has been already particularly pointed out.

Although it has been more than once explicitly stated that all the alleged "exciting causes" of inflammation were only "predisposing causes," favoring inflammation by either locally or generally depressing cell-vitality, or producing vascular conditions favorable to microbic lodgement, accumulation, and multiplication, yet the importance of the fact that germs alone can excite genuine inflammation needs iteration and reiteration. If this belief does not become second nature, slovenly practice and incalculable evils will follow. All predisposing and exciting (germ) causes can in theory, and in most instances in practice, be removed or avoided.

Exciting Causes of Inflammation.—Bacteria.— While the sole exciting causes of inflammation are path-

ogenic organisms or their products, yet it will be profitable to study how the different classes act, and how non-pathogenic organisms can reinforce the action of pathogenic ones, thus explaining many clinical facts which seem to be stumbling-blocks in the path not only of beginners, but also of those who should know better. Nearly all the bacteria of putrefaction are non-pathogenic, but they are of grave interest to the surgeon, because they predispose to the multiplication of pathogenic germs by preparing the soil. This preparation of the soil is brought about by the prejudicial action on cell-vitality of the chemical products of the non-pathogenic germs. Moreover, by preventing coagulation of the wound-fluids and dissolving coagula, the lymph-spaces are kept patent, thus permitting the free and rapid introduction into the circulation of large amounts of the toxic materials. These produce a chemical, systemic, and often fatal form of poisoning, termed sapremia or septic intoxication, which will be studied later in its appropriate place.

This is not the place to teach bacteriology, but I must emphasize certain general facts to show their bearing upon our own special studies. Again I wish to impress upon you that the local conditions cannot alone furnish a complete explanation of germ activity. First, a few words as to germs themselves. All are not pathogenic, as you must have already learned from my previous remarks ; yet it must not be thought that even the non-pathogenic organisms are always harmless, because some of them cause the putrefactive changes mentioned, while others become actually pathogenic if the local conditions, the general conditions, or both combined *predispose* to their multiplication. Again, one variety of germs may produce substances which either enhance the virulence of action of another species, or so alter the cell-resistance or metabolism that the true pathogenic organisms can usurp full sway over the tissues. Thus the Bacillus prodigiosus increases the effect of the toxins produced by the streptococcus of erysipelas. Still further, the growth

of one form of micro-organism may originate an un-favorable environment for a most virulent germ, thus *attenuating* its effects, as does the erysipelas coccus when inoculated at the same time with the micro-organism of anthrax. Certain temporary and often controllable circumstances which diminish natural or acquired immunity have been already described, but others must also be mentioned as pertinent to the point we have now reached in the study of the causation of inflammation. The decided effects of altered diet, the absence of fresh air and sunshine, and the humidity of the atmosphere are most potent as depressing influences. Surely, if feeding a rat upon bread will destroy its immunity against anthrax, the matter of diet is worthy of a careful study in man. Again, when we know that a relatively short exposure of tubercle bacilli to direct sunshine will kill them, and that moisture favors the growth of all micro-organisms, the importance of a proper environment for the maintenance of healthy tissue-resistance, and the destruction of tubercle germs at least, becomes at once apparent. Improper or wet clothing, because permitting or causing sudden alterations of the surface temperature of the body, as well as actual pertubations of the atmospheric temperature, favor the production of internal congestions, and should be included here as predisposing causes of germ-growth. Innumerable germs are to be found upon or in the skin and mucous membranes, ready to penetrate so as to reach the circulation by any avenue accidentally opened to them ; hence the enforcement of cleanliness of the cutaneous and mucous surfaces, proper changes of clothing, etc., etc., are not only not beneath the notice of the practical surgeon, but it becomes his duty to see that the importance of all these things is recognized.

The surgeon who expresses exaggerated fears of the evil effects of various drugs, of germs, of bad hygienic surroundings, etc., will often not appreciate how prejudicial to cell-nutrition are retained excreta, and so fails

to gain the advantages secured by the old-time doctor who got rid of these by stimulating the secretions of the skin, liver, kidneys, and intestines. Others fully understand the importance of all these and even the effects of diabetes, cholemia, lithemia, chronic renal disease, and scurvy, yet they fail to recognize how dangerous is that malassimilation evidenced by the presence of free uric acid, oxalates, and lactates in the urine.

The conditions induced by recent attacks of certain diseases, such as typhoid fever and the exanthemata, should be well understood, because clinical experience has long demonstrated that after these affections, bone and joint tuberculosis, acute infectious osteomyelitis, etc., are not infrequent. This is partly because of the lowered tissue-nutrition produced by the prolonged presence of poisons in the blood and tissues, partly because of the impoverishment of the blood, reducing both its capacity for nutrition and its germicidal properties, and partly because unexcreted toxins or a few remaining specific germs reinforce the action of pyogenic or other microbes which have accidentally gained access to the circulation.

The possibility of the fetus becoming infected (other than by syphilis) through the placenta, by microbes circulating in the maternal blood, has been proved, resulting in various forms of sepsis.

Although some local predisposing conditions have already been lightly touched upon, they require more extended notice. Thus, while the normally sluggish circulation of the epiphyseal regions of growing bones has been spoken of as accounting for the ready localization of germs at these points, yet the large, slightly collapsible, and therefore patent, veins of adult bones, in which large hemostatic thrombi must form, have not been mentioned as explanatory of the readiness with which pyogenic infection occurs after open osseous traumatisms. Granulations also have been stated to be a protective barrier against the entrance of microbes ; but the reason

for this has not been distinctly pointed out—viz., the presence in them of large numbers of phagocytic cells, *in the more superficial layers.* This protective barrier may be weakened or destroyed by its chemical disintegration by caustic germ-products, or mechanically by rough handling, or the unrest of a part. This is not a new observation, because many years ago Billroth showed that putrid meat-infusion, which when injected into the cellular tissue promptly caused the death of the animal from sepsis, was innocuous when maintained in contact with healthy granulations. Moreover when, actuated by the knowledge of this fact, he put up his open fractures in a fixed dressing, he was rewarded by having to contend with markedly fewer evidences of either local or general infection. Therefore carefully avoid all injury to this protective layer of granulations when opening abscesses and handling granulating wounds, although this rule has some notable, *apparent* exceptions, which will be found stated elsewhere, especially when treating of the Practice of Surgery.

One more fact of general importance and we shall be ready to consider another division of our subject. Septic germs—*i. e.*, those which induce putrefaction and produce alkaloidal poisons—can flourish only in dead or dying tissues, while the true infective micro-organisms *can* multiply in the living tissues, wherever found being capable of producing their characteristic poisons. This is a matter of prime importance, because septic micro-organisms can form their dangerous alkaloids *only in the wound*, which must be large enough to permit both the manufacture and absorption of enough poison to cause symptoms. Once this laboratory is destroyed by efficient disinfection or by physical removal, further danger ceases, and recovery ensues, if a fatal dose has not been already absorbed. But of what avail is it to remove a limb, the original source of true infection, if every organ of the body, including the blood, be swarming with germs which continue to produce, wherever present,

their poisons in ever-increasing amounts? The impor-
tance, the applications, and the exceptions to these facts
will become more apparent as we proceed.

Other low forms of vegetable life which are not
microbes initiate pathogenic processes in man, as the ray-
fungi, *the actinomycetes*, producing "madura-foot," etc.,
although their destructive lesions are chiefly due to sec-
ondary pyogenic infection. The *Amœba coli* also pro-
duces grave lesions in the intestinal tract, liver, etc.

LECTURE VIII.

INFLAMMATION (CONTINUED): PRIMARY AND SEC-
ONDARY INFECTION; THE AVENUES BY WHICH
GERMS GAIN ACCESS TO THE TISSUES; CAUSA-
TION OF SYMPTOMS; FEVER, ETC.

CERTAIN terms which have been unavoidably em-
ployed must now be defined. "**Primary infection**"
refers to the implantation of a single variety of microbe,
or to the inoculation of that form which came first in
order, if later other species were introduced.

"**Secondary infection**" means that in the soil pre-
pared by one kind of germs another variety is sown
which flourishes, often to the destruction of its prede-
cessors, as the pyogenic cocci which cause the rapid
breaking down of a caseated tubercular focus. This,
however, is not an example of destruction of the first
germs by those coming later.

A "**mixed infection**" occurs when two or more varie-
ties, or species, of germs are implanted at the same time,
the stock illustrations being the pyogenic and tetanus
germs, the gonococcus and pus-organisms.

Avenues of Infection.—The avenue whereby the
germs gain access to the circulation or a part, the "in-
fection atrium" as it is often called, is usually a "locus
minoris resistentiæ," a point of diminished resistance,
the result of normally or abnormally defective nutrition,
or a trauma. Thus a blow upon a bone, which ruptures
minute vessels, producing collateral hyperemia, inevit-
ably causes such slowing of the circulation that germs
there present will collect at the injured part in numbers
sufficient to overwhelm the tissue-resistance. Juxta-
epiphyseal strain, adding hyperemia to the normally

sluggish current and damaging a few tissue-cells, often determines an osteomyelitis. The proof of these statements has been demonstrated by introducing into the circulation large numbers of the pyogenic cocci causative of osteomyelitis, whence they quickly disappear by means such as have been already, or will be later, explained. Repeat the inoculation, however, and then contuse or fracture a bone, and promptly the microbes will be located, and osteomyelitis will develop.

The time during which a given number of germs remain in contact with the tissues also determines whether they will conquer cell-resistance or the cells conquer them. This is because their toxic products require time to reduce the tissue-vitality by means which have been frequently mentioned, thus creating a "locus minoris resistentiæ" where none previously existed. Wegner and Grawitz have shown that the same number of germs, which when introduced into the peritoneal cavity will cause no trouble, because quickly removed by absorption, will produce a septic peritonitis if sterilized water be added to the pure culture. This so increases the bulk that a longer time is requisite for absorption, and hence a longer time is provided for the toxins to act locally ; moreover, the inflammation usually starts at the point of original trauma—*i. e.*, the hypodermic needle puncture, the "locus minoris resistentiæ."

If no "locus minoris resistentiæ" exists, the germs are disposed of by the mononuclear and polynuclear leukocytes and the nucleins of the blood-serum, while some are excreted by the kidneys, others by the liver, the intestinal tract, and it is even alleged, by the saliva, and certainly microbes do pass out by the mammary secretion ; the young endothelial cells of the blood- and lymph-vessels also act as phagocytes. These protective cells accumulate where the germs are, by virtue of the chemotactic attraction exercised by the bacillary proteids.

The most common sites of "infection atria" are the

mouth, the respiratory passages, the intestinal tract, and the skin, because microbes are always found in these localities. A carious tooth, the lesions inflicted upon the gums by the tooth-brush or tooth-pick, burns, scalds, and damages of the buccal mucous membrane caused by the teeth, lesions of the nasal passages, and recently healed wounds, may any of them be the entrance point of micro-organisms productive even of fatal pyemia.

Clinical Signs of Inflammation.—We have now reached a point where the clinical signs of inflammation can be profitably considered. These are *local* and *constitutional*. The former are in their earlier stages precisely those which have been described when considering hyperemia, although all the symptoms are present only in typical cases occurring in parts accessible to the eye. One or more symptoms may be detected without inflammation being present, as pain, redness, and even swelling, the last induced by edema, observed in certain severe neuralgias ; swelling alone, as seen in hygromata of bursæ. Redness and heat may mean only vasomotor disturbance, as seen after injuries to the cervical sympathetic in the rabbit or man; while loss of function with pain on movement is well exemplified by an hysterical joint.

Redness.—The tint of redness varies in intensity, being bright in *acute* and dull in *chronic* inflammation. It is generally livid when suppuration is imminent in a superficial part. It is at first, of course, absent in non-vascular tissues, although the hyperemia is present in the nearest vascular area, as in the conjunctiva or sclera around an inflamed cornea. When the inflammation is deep-seated, the redness cannot be recognized. The redness of inflammation fades somewhat after death. The temperature of an inflamed part, like that of a hyperemic one, is never higher, indeed is usually lower than that of the blood in the left ventricle of the patient, although this fluid is of course *hotter than normal*, during an inflammation causing constitutional symptoms.

The sensations of the patient commonly indicate a much higher temperature than the thermometer reveals.

Pain.—The pain of inflammation is increased by pressure and by the dependent position, the latter of which increases the blood-pressure in the part, and by mechanically interfering with the return blood- and lymph-circulation increases the compression of the sensory nerves. This is a matter of common observation and gives an excellent hint as to the necessity of position in the treatment of inflammation.

The character of the pain is apt to vary with the tissue inflamed. Thus, it is stabbing, "stitch-like," when a serous membrane is concerned. Burning is the adjective employed to describe the pain of inflamed skin, while aching or boring pain is usually complained of during inflammation of bone. Although "throbbing pain" is commonly noted during suppuration, this is not invariably present. The pain of inflammation is most intense when the exudate is situated beneath unyielding fibrous structures, such as the sclerotic coat of the eye, the palmar fascia, and within the tunica albuginea testis. The converse is equally true, for the pain even of suppuration, when situated in such regions as the axilla, eyelids, or scrotum, is comparatively trivial. The nerves of special sense when themselves irritated by inflammation of their own structures or of contiguous parts, of course cannot express this by pain. When the optic nerve is concerned, sparks, flashes of light or colored rings are seen ; abnormal sounds, such as tinnitus, indicate auditory-nerve involvement ; disagreeable odors are often perceived when the olfactory nerve is the sufferer ; while perversion of the gustatory sense is shown by the apparent perception of unpleasant substances, such as a "metallic taste," when the nerve of taste is irritated.

Pain is of much clinical importance as a means of diagnosis, as I shall endeavor to show. What is termed "reflected" or "radiated pain" may greatly mislead if

certain facts are not remembered. Thus, the irritation of one branch of a sensory nerve may be referred to another branch of the same trunk, or to all the terminal branches of the irritated nerve-trunk. Again, the impression reaching the nerve-center may cause congestion of the gray matter of neighboring nerve-fibers which form part of a nerve of the same plexus, and the irritation thus induced may be incorrectly recognized as due to trouble in the area supplied by the latter nerve, instead of the one actually irritated. The pain produced by a stone in the pelvis of one kidney may be referred to the other kidney, still further illustrating the last condition cited, because here the irritation causes congestion of the gray matter across the cord, so that the nerves on the opposite side of the body seem to be the irritated ones. Radiated pain is well illustrated by the dying nerve-pulp of a tooth in the upper jaw. This may cause pain apparently located in one of the lower jaw teeth, or in all the teeth of both jaws. In like manner the pain of an inflamed appendix is often at first referred to the whole abdomen. Irritation of the ciliary nerves, as during iritis, is often noticed by the patient as severe pain in the side of the nose—*i. e.*, the nasal branch of the ophthalmic branch of the fifth nerve : or again, the entire distribution of the fifth nerve may recognize by pain the irritation of the ciliary nerves.

Irritation of the trunk of a nerve high up, as by the pressure of a rapidly forming abscess, may be felt as a peripheral neuralgia ; hence this fact often proves of great value in the diagnosis of certain deep-seated ailments. For instance, perinephric abscess will, by pressure upon branches of the nearly related lumbar plexus, give rise to pain in the distribution of the genito-crural, ilio-hypogastric, ilio-inguinal, or anterior crural nerves—sometimes in all of these.

Swelling varies in amount and rapidity of development with the distensibility of the tissue. It is slow and limited in extent in bone and tendon, while it is exten-

sive and rapidly supervenes in the eyelids, scrotum, and vulva.

Disturbance of function may be the first symptom noted by the patient ; for instance, inability to retain urine in the bladder for a proper length of time, or the distress produced by light (photophobia) upon an inflamed eye.

Constitutional Symptoms.—Fever.—Having now sufficiently considered the local symptoms of inflammation, those by which the circulatory, nervous, and digestive systems, and the secretory organs recognize its presence remain for study—*i. e.*, the constitutional symptoms of inflammation—in other words *fever*, with its complications must now be considered. While the conditions produced by the absorption of nucleins cause some rise of temperature with increased rapidity of the pulse, it will elsewhere be shown that this "aseptic fever" materially differs from the fever of true inflammation. Thus, after the lapse of many hours, usually forty-eight or more, the rise of temperature in true fever is usually preceded by malaise, chilliness, or rigor. The pulse becomes increased in frequency, the secretions of the skin, stomach, intestines, and kidneys are modified or arrested, whence follows the dry skin, furred tongue, anorexia, constipation, and scanty high-colored—*i. e.*, concentrated urine. Pains in the back and limbs and a general feeling of soreness are complained of. The sensorium may become so malnourished or poisoned by the impure blood circulating through it that sleeplessness, restlessness, or delirium may result. The degree of fever usually is in proportion to the extent and severity of the inflammation ; but this is not always the case, as for instance in a septic peritonitis where there may be none, or an insignificant rise of temperature, although in such a case the pulse usually runs high.

In sharp contrast to these symptoms is the constitutional condition presented by patients during the aseptic repair of injuries, or wounds, or after operations. In about

one-third an absolutely normal temperature prevails throughout the whole course of treatment, in another third there is a slight rise of temperature, while in the remainder quite a rise, even to 103° F. or 105° F. occurs. But this "aseptic fever" represents many distinctive peculiarities when contrasted with the description just given of true fever. To enumerate them, after the (perhaps) subnormal temperature following the injury, operation, or the nausea of anesthesia, the temperature *at once* commences to rise, *without previous chill or malaise*, and the pain of injury or operation after reaching its acme, in a few hours in a typical case steadily diminishes, as does the tenderness upon pressure. The neighboring lymph-nodes are neither swollen nor tender ; the individual complains of no general discomfort, headache, loss of appetite, nor (usually) of any sensation of increased heat ; the skin is only moderately or not at all dry, the tongue is moist and often not even furred, the urine is not diminished in quantity nor loaded with solids, and the same can be said of the intestinal secretions, hence the absence of constipation. Finally the pulse is only slightly increased in frequency. Except in those with the highest temperature, unless the thermometer be used no suspicion is excited of any elevation of temperature, while in a large proportion of the cases absolutely nothing abnormal can be detected beyond the increased temperature.

The duration of aseptic fever is from one to five days, but Volkmann has reported one case which lasted sixteen days. It is caused by the absorption of fibrin-ferment, nucleins, and the pyrogenous substances resulting from the retrograde products of tissue-necrosis. Whether these stimulate heat-production, or diminish heat-elimination or both, does not concern us now.

All other varieties of fever are due to the absorption of chemical pyrogenous substances (ptomains, toxins, or toxalbumins) in addition to the ordinary aseptic pyrogenous substances which cause aseptic fever ; in other

words, true surgical fever follows *infection*. The clinical combination of aseptic and septic fevers has been long observed and was explained thus : fever always followed wounds or injuries and was called traumatic, inflammatory, or symptomatic fever ; these terms were employed because constitutional symptoms invariably followed any considerable trauma, but it ordinarily ceased by the fifth day at the latest. When this favorable termination did not occur, it was said to have merged into surgical or septic traumatic fever ; which if it in turn did not disappear in the course of about a week longer—*i. e.*, shortly after suppuration was fully established—was taught to be the commencement of septicemia ; or at least it was supposed that there were good grounds for fearing the existence of this dread condition. Now we know that a rise of temperature, etc., can commence as soon as the shock is past, from the absorption of aseptic substances long before infective germs could produce enough toxins to cause constitutional symptoms ; but that if slight infection has occurred at the time of accident or operation, it will begin to show its effects somewhere from the third to the fifth day, true (septic) fever being engrafted upon and superseding the disappearing pyrexia due to the absorption of aseptic substances. This sequence of events might have been inferred from your laboratory studies which show that it requires two to three days to develop such an abundant crop of pyogenic organisms as will be able to manufacture enough poisons to produce systemic effects.

The systemic intoxication leading to fever is nearly always preceded by increasing local changes in the wound indicative of disturbances of the healing process ; the part becomes tender, then painful, the redness extends in area and deepens in hue, the wound becomes swollen with pouting edges, a purulent discharge forces its way between the margins, and breaking down of all repair, causing gaping of the wound, follows unless the mechanical restraint of stitches maintains apposition.

The lymph-nodes are enlarged and tender. Fever continues until the wound either ceases to be septic (I do not say, ceases to suppurate) or at least until it no longer permits absorption of toxins.

As the fevers following surgical injuries and wounds present either temporarily or throughout their course certain types, for convenience of description I shall describe them under certain heads. Be it remembered, however, that this is an artificial division and that the distinctions cannot always be sharply drawn.

LECTURE IX.

THE TREATMENT OF INFLAMMATION.

If germs alone cause inflammations and other surgical affections, what is the use of considering the therapeutics of inflammation? Why not merely state what preventive measures can be most successfully employed, and when these either fail or cannot be adopted, say that of course the surgeon is at the end of his resources and must abandon any attempts to control or guide the inflammatory process? This is the question actually asked by some who should know better, and when not put in words is really the principle governing others. Although I have already explained much in essence, or specifically, that should demonstrate to you the folly of such views, yet I will restate the main points, thus leaving no room for misunderstanding.

None will deny that if we can modify the hyperemia at the periphery of the focus of inflammation, we can either prevent or at least limit microbic lodgement and multiplication. In many instances the germs are so few in number *at the outset*, that the tissues can gain the mastery if fresh hordes of microbes are prevented from arriving and being detained. If in addition, some of those present can be removed, the chances of victory for the tissue-cells are still further improved. If all local conditions unfavorable to cell-nutrition can be ameliorated or removed, everything which is demanded both by theory and in practice will have been effected, and all or most of this can often be achieved.

The time during which the germs are arrested in the tissues, as shown by the experiments of Grawitz just

quoted, is of prime importance and emphasizes the necessity of instituting effective measures against hyperemia due to infection at the earliest possible moment. The general measures which at the outset are effective at the focus are useful later on at the periphery. A word of caution, however, is requisite here. Although cold, for instance, will tend to diminish the caliber of the enlarged vessels in the peripheral hyperemic area, thus obviating the dangers of a slowed circulation, yet it exerts a depressing influence upon the vitality of the cells, and somewhat condenses the tissues. It thus lessens the size of the plasma-channels, and certainly diminishes the ameboid powers of the leukocytes, both inside and outside the circulation, hence favoring stasis and thrombosis of the vessels, with gorging of the tissues by cellular exudate *at the focus of inflammation*. Judgment is therefore requisite to decide whether the evil at the focus will not outweigh the good exerted at the periphery. This question will be considered again later.

The various means by which the lodgement, multiplication, and damaging effects of germs on the tissues can be modified or checked are manifestly to be exerted though the medium of the vascular system. They will be treated of under certain heads as most conducive to a clear understanding of the matter.

First, those will be considered which influence the circulation chiefly upon the proximal side.

The indications are clearly then to lessen sufficiently the amount of, and force with which, the blood enters an inflamed part, that the veins may carry it away just as it arrives, otherwise the tendency to slowing of the circulation and stasis and exudation, interfering with nutrition and determining the accumulation of germs, must all remain unchecked ; in other words, the vis-a-tergo, if excessive, must be diminished.

The measures adapted to secure this end are those which act upon the heart itself. The one always safe to employ and of far greater power than is generally sup-

posed, is the interdiction of all muscular action, secured by rest in bed. This will materially reduce the number of heart-beats, and thus the amount of blood passing through an inflamed part in a given time. Guy showed that there was upon the average 15 beats more per minute in the upright posture as compared with the recumbent one, and that this was caused almost entirely by the muscular effort required to maintain the upright position. Nitrogenous food is the most permanent of all heart-stimulants, maintaining the force of the heart's action better than any drug. Herein lay whatever of good belonged to the employment of the so-called "antiphlogistic diet," a fact which unfortunately many practitioners ignore who reject the small, but important truth with the mass of error. Unquestionably restriction of the amount of nitrogenous animal food, or its total withdrawal, will often favorably influence an inflammation by reducing the force and frequency of the heart-action. The use of such drugs as aconite, etc., which depress the power of the heart may at times do good, but they must be used with caution and only when the excess of vis-a-tergo is so pronounced as to require more prompt 'and decided results than can be secured by recumbency and the restriction of animal food. Venesection is of very doubtful value in any surgical affection and I cannot countenance it, although I am a firm believer in local blood-letting, which will be mentioned in its appropriate place.

The same tendency to all the unfavorable conditions mentioned may be produced by the exact reverse of a too powerful action of the heart—viz., by a weak heart-action.

The measures best calculated to combat this are those drugs which increase the force of the heart-beat, and by rendering its contractions more efficient, as well as in other ways, decrease the number of contractions per minute. Strychnin, digitalis, nitrogenous food, alcohol, ammonia, etc., are the remedies usually employed.

Again, the caliber of the arteries which supply an inflamed area, as well as that of the arteries *in* the part, can be influenced. For instance, if their caliber be lessened, the rapidity of the current must be increased, ensuring the favorable results upon tissue-nutrition as well as those detrimental to germ-development, which have been so often mentioned.

The measures commonly employed, involving the use of remedies which belong to the class of drugs called *astringents*, are of doubtful value even *at the outset of an inflammation*, but they are still used for inflammations of the skin and mucous membranes. I gravely question whether the real benefit which sometimes accrues from their use is not rather due to the germicidal action of many of them, as for instance nitrate of silver, than to any narrowing of the vessels produced. In any event they can be of benefit only at the outset, before stasis has commenced, or in the later stages, when the reparative processes are hampered by the sluggish circulation incident to passive hyperemia. *Cold*, however, is a most potent remedy when judiciously employed, yet it must not be so intense as to act on the vessels directly through the medium of the overlying tissues, but by impressing the cutaneous nerves and reflexly causing contraction of the vessels. For instance, no amount of cold which could be endured would directly influence the intracranial circulation, hence a moderate degree of cold is all that is requisite. Cold so applied as directly to abstract enough caloric to influence deep-seated vessels, or those at the center of a more superficial inflammation, would lower the cell-vitality of the part, favor the cohesion of the leukocytes, and diminish ameboid activity. It should therefore always be employed with caution in parts whose vascularity is normally poor and where decided strangulation of tissue has resulted from extensive and rapid exudation, lest stasis be precipitated and gangrene result. When employed in proper cases, cold relieves pain by reducing the hyperemia of the nerves

and thus their increased registering power. The highest degree of *heat* compatible with the safety of the tissues will also produce contraction of the vessels, but it is rarely applicable except where very superficial parts are inflamed. The vascular contraction induced by heat is well seen when hot water is used to check oozing after the removal of the Esmarch bandage applied for operative purposes.

Increase in the caliber of the vessels may occasionally prove directly beneficial in the treatment of inflammation, when the vis-a-tergo is deficient, by serving to clear out the vascular areas which have become engorged with slowly moving blood, especially if the force of the heart can at the same time be increased. The increased rate of the circulation removes pressure from the cells by favoring absorption of the fluid exudates and débris, brings numerous phagocytes, and hurries away those which have seized upon germs. The means available to increase the caliber of the vessels also render the tissues more distensible, again removing pressure upon the tissue-elements and widening the narrowed plasma-channels. In addition, the veins are dilated, thus emptying the part of blood, which again favors all the conservative changes just described.

The only means which will produce this local increase in size of the veins is *heat*, and a moderate degree of this, such as can be comfortably endured. Pain is also relieved by heat when the favorable changes I have mentioned take place.

Inflammation being chiefly a vascular process can be starved, as it were, by interfering with the access of arterial blood by means of *position*—*i. e.*, elevation—which while lessening pressure from the cardiac side, will favor the return of the venous blood and lymph, thus aiding in the re-establishment of the circulation through the part, and at the same time improving the nutrition of the tissues and unfavorably affecting the environment of the germs. Ligation of the main artery

of a limb for secondary hemorrhage has so often favorably influenced septic processes that this procedure has been warmly advocated *to starve* the inflammation. While this may have been permissible in the past when antisepsis was unknown, it is merely mentioned here to impress upon you how much effect can be produced upon inflammation by diminishing the access of arterial blood to an inflamed part. Compression of the carotids has been also advocated and asserted to modify favorably acute traumatic intracranial inflammation. This I cannot but think is somewhat doubtful, yet it may possibly prove beneficial.

But inflammation should be capable of modification by measures which influence the circulation upon the distal—the venous—side of the inflammatory area, and this can be done, as will be shown. If the freedom and rapidity of the venous current can be increased, impending stasis with engorgement of the tissues, and interference with their nutrition will be prevented. Prompt removal of injurious substances and germs will be favored even at the focus, much more at the periphery—*i. e.*, the spreading margin of the inflammatory area. To favor the exit of venous blood the size of the vessels must be increased, but at the same time the blood-flow must be rendered more rapid. To secure these results first, *warmth* may be employed to increase the caliber of the vessels, and second, the velocity of the current can be accelerated by calling in the aid of gravity, by elevating a dependent part. Where it is possible directly to reach the vein, or its radicals, leading from the inflamed part, *local blood-letting* will often be of benefit, employed preferably before stasis has commenced, certainly before thrombosis has occurred. Gennsmer and I have both demonstrated the utility of local blood-letting in relieving an experimentally induced hyperemia, and clinically I have seen the same many times. A moment's reflection upon the anatomy of the encephalon will show that in inflammation of this organ venous blood can be directly ab-

stracted from its vessels, hence the proved utility of leeching and cupping in its ailments. The free communication of the superficial veins of the back of the neck with the large veins communicating with the lateral and superior longitudinal sinuses enables us to understand the effects of abstraction of blood from this region. The pain of an otitis media, which morphin hardly influences, is often relievable by removing blood by means of a leech from the external auditory meatus, and with the relief from pain, the hyperemia caused by irritation of sensory nerves in the area they supply will be sensibly abated.

The relief of tension by incisions, position, and by supporting pressure are all important adjuvants to the therapeusis of inflammation.

Pain—*i. e.*, irritation of the sensory nerves—as we have just stated, induces congestion, hence this must be relieved to reduce the hyperemia, or at least prevent its extension.

Rest to be effective must be physiological. Of what value would it be to confine a patient to bed with an inflamed eye, if this be exposed to the irritation of light? Yet an injured joint is too often in like manner not benefited, as it should be, because rest in bed or the employment of crutches is not supplemented by means calculated to prevent all motion of the joint—*i. e.*, physiological rest has not been secured. As the exercise of function always means an increased flow of blood to and through an organ—a temporary hyperemia—in the event of a previous slight infection this physiological congestion often proves most disastrous. Still further, the mechanical advantage of rest, by preventing the inoculation of fresh surfaces with germs or their products, is well exemplified by the experiments of Billroth, quoted during a previous lecture.

The bodily temperature, when excessive, often proves exhausting to the heart directly, or by increasing the frequency of its contractions. Again, heat is sometimes

directly productive of restlessness, delirium, etc. For any or all of these reasons the temperature must be reduced in *hyperpyrexia*. Antipyretic drugs are very rarely to be employed, cold, in the form of baths, sponging with cold water, etc., being both safer and more easily regulated.

The elimination of bacteria and bacterial products, together with the poisonous metabolic products of tissue-metamorphosis by the bowels, kidneys, liver, and skin, directly improves the condition of the tissues, besides often averting the death of the patient from toxemia.

I have now given the general "indications" for the therapeusis of inflammation, with a few illustrations of the manner in which some can be carried out. It now remains for me to show what are the various ways by which these ends can be reached—*i. e.*, how these "indications can be fulfilled," as it was wont to be said.

I shall now briefly indicate the methods of employing the various classes of remedial agents already considered.

Cold may be used either *dry* or *moist;* each form possesses its advantages and disadvantages, but both must be continuous in action to prevent "reaction," as it is vaguely termed, meaning either a paretic dilatation of the vessels, which usually follows only the direct application of too intense a degree of cold, or, more commonly, the return of the vessels to their original hyperemic caliber, an interval of time existing during which they may still further dilate.

Moist cold is often preferable, especially when there is much discharge, because germicidal agents can be combined with the fluid. The danger from absorption in poisonous amounts of such drugs as carbolic acid or corrosive sublimate, following the prolonged use of irrigating fluids containing these agents, must never be forgotten. Moist cold is also more generally applicable, because more attainable, since water even at ordinary temperatures, if allowed to evaporate rapidly, will abstract much caloric. Other more rapidly evaporating fluids can also

be employed, as alcohol and water, or mixtures can be made of various chemicals in solution which are actually frigorific. Evaporation is, however, the main reliance, hence the folly of applying bulky wet dressings and covering them up closely, when endeavoring to utilize the therapeutic powers possessed by cold. Moist cold can be most simply employed by cloths, constantly changed, wrung out of water or some frigorific mixture, by compresses frequently changed, which are kept lying upon a piece of ice, or by irrigation in some one of its many forms. The simplest method of irrigation is to place under the part a piece of oil-cloth or rubber-cloth so disposed on the bed and hanging over its edge as to form a gutter which will empty the fluid into a receptacle placed upon the floor. This arrangement is as requisite for the most elaborate irrigating apparatus as for the simplest. Next hang above the part any vessel capable of containing enough fluid. The ends of several lengths of plain wicking, or long narrow pieces of any cotton material should be dropped into the vessel, while the other ends rest upon a single or double layer of absorbent material smoothly covering the part to be irrigated. By capillarity a constant flow will be maintained, the fluid of the receptacle being kept at any temperature desired or securable, evaporation from the wetted covering of the inflamed part enhancing the refrigorating effect. A large bottle with the bottom broken out and whose neck has been fitted with a cork perforated by a quill may be suspended above the bed, and will allow a constant drip of fluid to fall on any given spot. There should always be at least one layer of absorbent dressing interposed between the inflamed part and the falling water, to prevent pain from the impact. This latter method is more difficult to arrange and not a whit more efficient than the one first described.

The disadvantages of moist cold are that it is more depressing to both local and general vitality ; it is difficult to manage so as not to wet the patient's clothing or

bedding ; it macerates the skin, sometimes making it painful for the part to rest even upon a pillow, and it favors the development of pressure-sores (decubitus).

Dry cold possesses none of these disadvantages and is therefore more generally applicable, especially when a very limited area is to be treated. As before said, cold is an agent which should act only reflexly upon the circulation, hence some non-conductor of heat must be interposed between an ice-bag and the subjacent parts. Dry cold can be applied by means of ice-bags, bladders, Leiter's block-tin coils, or by several yards of small rubber-tubing coiled and secured in proper shape by interweaving three or four pieces of wire, or sewing the turns upon a properly shaped piece of thin flannel. Through these tubes, water at any temperature can be passed from a vessel above into a receptacle below. Any of these devices will cause deposition of moisture upon their surfaces, necessitating care lest the clothing become wetted ; indeed, the nurse often insists that the bag or bladder must leak. Special emphasis must now be laid upon certain indications and contra-indications, despite their mention on a previous occasion. Cold acts best before stasis has occurred by preventing such dilatation of vessels as will favor this ; but later, cold will do the same at the periphery, and, as has already been pointed out, the surgeon must decide whether the good done here will be outbalanced by the possible harm done at the focus. Cold in any form is distinctly contraindicated in the later stages of inflammation, especially where there is much strangulation of tissue, because it may determine gangrene, diminishing the already feeble arterial pressure through a lessening of the volume of blood carried in the afferent vessels by narrowing their caliber.

LECTURE X.

TREATMENT OF INFLAMMATION (CONTINUED).

Heat is to be used either as *moist heat* or *dry heat*. Moist heat is usually applied in the form of fomentations or poultices, either of which may be medicated by the addition of opium, morphin, or antiseptics. Hot fomentations consist in the application and frequent renewal of flannels or cloths wrung out of as hot water as can be borne by the patient, these then being covered by dry flannel or cloths.

A **poultice** consists essentially in the constant application of heat and moisture. The supposed virtues of hops, bread, or bread and milk, for poultices are purely imaginary, while the two last soon undergo fermentation and become positively irritating. Sometimes the material of which a poultice is composed is a matter of moment.

When it is desirable to maintain a degree of heat in the applications higher than that of the part itself, it will require far less frequent changes of poultices if corn meal be used as the material, because this substance retains its temperature longer than anything else that can be used. It must be sewn up in bags, otherwise it will crumble as soon as it begins to dry. The best material for poultices in general is ground flaxseed, since it can be made thin and light and requires changing less frequently because it coheres, and, containing much oily matter, does not dry readily. The practice of pouring oil or melting lard over the surface of a poultice, or covering this with a piece of gauze to prevent it sticking to an irregular surface, making it hard to cleanse, is no

longer necessary, because it is impossible with poultices to maintain effective asepsis, and they are no longer employed for open wounds.

For the same reason, *carrot poultices* and the ferment-ing poultice, useful as they were in the past, should no longer be employed. An exception may possibly be made in favor of the *charcoal poultice*, especially for such a con-dition as gangræna oris, where it is practically impossible to secure anything beyond relative asepsis. A thick layer of recently sterilized, powdered charcoal should be spread over a moderately thick flaxseed-meal poultice, which has been sterilized by heat, as can readily be done by keeping the vessel in which it is mixed over the fire for a few minutes, after mixing the meal with *boiling* water, as should always be done when making any flax-seed poultice. No poultice is properly applied unless it is covered in with some substance relatively impermeable to heat and moisture, and which extends some distance in all directions beyond the margins of the poultice, otherwise it will soon first become cold, then dry. Unless high temperature be indicated, so long as a poultice thus covered remains moist it need not be renewed. Wet absorbent cotton, gauze, moss, etc., when thus covered constitute a poultice.

Stout paper, well-greased paper, paraffin or waxed paper, oiled silk or calico, mackintosh or thin oil-cloth are suit-able materials to place outside a poultice. Never employ anything but a wet aseptic or, better, antiseptic dressing —*i. e.,* an *antiseptic poultice*—for any open wound, or even for an abscess, *after it has been opened.* Before an abscess has been incised or has ruptured spontaneously, it is both permissible and often advisable to use any material suitable for a poultice which is available ; but when spontaneous rupture is possible, an antiseptic poultice should always be substituted, lest the abscess unexpectedly evacuate itself into a non-sterilized dress-ing, such as a poultice almost necessarily must be.

Dry heat can be employed by the use of Leiter's tubes,

coils of rubber tubing, hot-water bags, hot sand- or salt-bags, hot stove-lids, irons, etc., which never should actually touch the bare skin of a patient, a flannel cloth, or if nothing else is securable, several thickness of paper intervening lest accidental burning of the integument take place.

Heat in extreme form is indicated during the very earliest stages of inflammation, as was explained when speaking of the use of cold in the dry form and also hot fomentations. Later, especially when there is much engorgement producing strangulation of the tissues, moist heat in the form of warm poultices is better than dry heat ; it relaxes the tissues more, and favors ameboid movement of the cells, which, if suppuration be avoidable, hastens the departure of phagocytes which have incorporated germs into their substances, brings more leukocytes to the defence of the tissues, and favors the clearing away of excessive cellular exudate. If suppuration is inevitable, the same processes favor the rapid accumulation of cells at the inflammatory focus, resulting in the most rapid disposition of all germs which can be destroyed by phagocytes, the promptest breaking down of tissue at this point, and the erection of an efficient barrier of phagocytic cells outside the focus, which will prevent undue spread of the infective process. When the inflammation is somewhat deeply seated, derivation of blood to the skin and more superficial tissues is produced by a poultice, as can easily be proved by seeing the exact outline of the poultice indicated upon the skin by a reddened area ; this derivative action may possibly favorably influence the peripheral hyperemia of the inflammation. By some or all of these means the inflammation is limited and brought to a focus, and the abscess "points," if suppurative inflammation be the condition present, thus giving the explanation of the well-known good effects of moist heat noted in practice.

Heat is usually contraindicated in the earlier stages of inflammation, cold being generally preferable. Certain

exceptions to this rule have been already mentioned in a previous lecture.

Starvation of an inflammation by the temporary or permanent arrest of the arterial supply to the part is rarely, if ever, considered nowadays ; but it is somewhat of a question in my mind whether in very rare instances, when a septic process involves the leg and knee-joint, a ligation of the femoral artery low down might not be properly attempted before amputation is resorted to. The temporary arrest of arterial circulation could often under similar conditions be harmlessly employed by digital compression of the main vessel or the application of a horseshoe tourniquet for a number of hours.

Diminution of the contents of the veins may be effected by means of leeches or wet-cups, always remembering that to be useful the blood must be drawn from the vein—or the radicals thereof—which drains the inflamed area, the instance cited before of drawing blood from the mastoid region being sufficient. Multiple, small incisions or punctures may also be beneficial by virtue of the blood withdrawn, but probably prove more useful by the relief of tension afforded.

Relief of tension is a most potent means of diminishing the hyperemia and other phenomena of inflammation for the following reasons. Extravascular pressure will, as has been experimentally shown, increase the rapidity or actually determine the absorption of poisonous alkaloids.

Tension also produces compression of the plasma-channels and the cells, thus mechanically interfering with nutrition and reducing tissue-resistance. The means employed to relieve tension, notably incisions, besides permitting stretching of the tense skin and fascia, abstract blood from the vessels, and the bulk of their contents is thereby reduced. The improved circulation following this increases tissue-resistance. Exit is also given to numerous germs and bacterial products, their withdrawal reducing the stock available for absorption,

and altering the osmotic conditions upon which their former rapid absorption depended. When I state that a small card can be so cut as to form a ring capable of encircling the waist of an adult, it will be admitted that the manner of making the incisions for the relief of tension
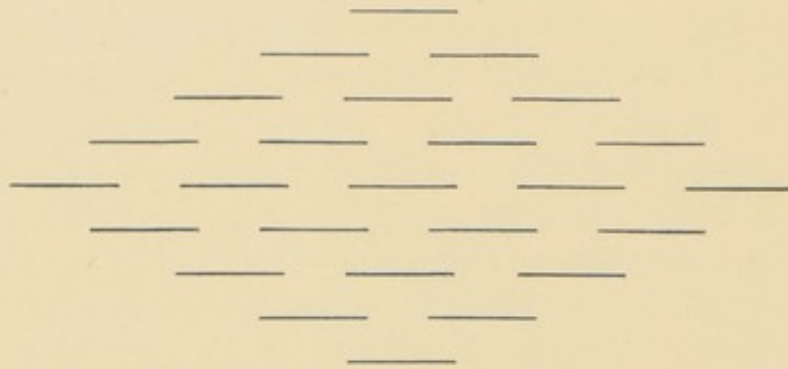
FIG. 10.—Showing how marked relief of tension can be secured by numerous small incisions, if properly planned.

is a matter of moment, in order to secure the maximum of extensibility with the minimum damage to the tissues. The method shown in the illustration is the best, the incisions extending through the deep fascia. Elevation of a limb also reduces tension by favoring the return of venous blood and lymph, and also by rendering the entrance of arterial blood more difficult.

Control of Pain.—Pain is controllable by many of the measures already advocated, such as cold, heat, and position. Certain drugs, which except in children had better be given hypodermatically, such as opium, morphin, and codein, often prove most useful, sometimes indispensable. Codein seems not to be of much value as an analgesic except for abdominal pain, when, from its non-constipating effect it is often preferable to opium. Whether opium directly causes contraction of the peripheral vessels or does so by reducing the registering power of the sensory nerves, certainly its administration during inflammation, when pain is a prominent symptom, does diminish the hyperemia, as witness the prompt healing of the congested, so-called painful ulcer of the leg under

the administration of full doses of opium when everything else has failed. Under *very* exceptional circumstances, in an exceedingly plethoric individual, phlebotomy, when everything else has failed, will sometimes relieve pain in a wonderful way. For instance, I have known of a case of vesical stone, where, after a long journey such intense inflammation of the bladder was excited that morphin in large doses hypodermatically utterly failed to give relief, the patient being forced to attempt to void urine every few minutes. Finally, the patient was bled from a large vein by a large orifice *in the standing posture*, until he was very faint, thus securing the most marked effect on the circulation with the minimum loss of blood. Immediately after this, the patient slept for a number of hours and had no return of his excessive dysuria. This is an exceptional case, but it is also worth remembering because it demonstrates that a marked diminution in the force of the vis-a-tergo will powerfully affect the hyperemia of inflammation.

Rest.—I cannot refrain from returning to the subject of rest during inflammation—rest complete and physiological, especially where a wound is concerned. Rest sometimes means restraint by a splint, at others restraint of a function, as by the exclusion of light from an inflamed eye by a bandage, permanent drainage of an inflamed bladder preventing its recurring distention and contraction. Drainage is often the most potent means for securing rest, for how can a wound be at rest if alternately filling with fluid, and, when the distention becomes sufficient to separate its edges, collapsing, to refill again? Position, making the drainage-opening dependent, compresses and other dressings so applied as to efface cavities, that separation of wound-surfaces by accumulation of fluids is impossible, are important means for securing rest. The infrequent dressings rendered possible by modern methods of wound-treatment likewise secure the same end. Removal of a foreign body may, by relieving irritation, diminish hyperemia and the secretion incident

to its presence, hence the "unrest" produced by this cause. The foreign body may be really something introduced from without, or a piece of dead bone or other tissue, which sometimes also acts prejudicially by blocking the exit of discharge. Affording free exit for extravasated urine and collections of pus would perhaps come equally under the head of relief of tension, but the physiological rest thus afforded to the parts requires emphasis here.

Lowering of the general temperature in a surgical case can rarely be done by immersing the whole body in a bath. By placing a rubber sheet, a piece of mackintosh, or oil-cloth beneath the patient and elevating the edges, bathing with cold water, ice water, or rubbing with ice can be done, freely exposing the wetted surfaces during the bath to ensure the refrigerating effect of evaporation. Such measures will prove more efficient than would be conceded at first sight by those insisting that the cold bath is the only really efficient method of lowering temperature. These measures are those only which are available in country practice, and with an army in the field.

The attempt to **eliminate bacterial and metabolic products,** although long out of fashion, is now established upon a firm scientific basis of facts. Increasing the secretions of the intestines, kidneys, and skin are the means employed to secure this result. Whatever good "revulsion"—*i. e.*, the temporary accumulation of blood in one part to its relative exclusion in another—can effect, is likewise probably secured when we reflect upon the many square feet of intestinal mucous membrane which must be congested after the exhibition of a hydragogue cathartic. Again, the increased evaporation from the skin-surface produced by free diaphoresis will lower temperature, revulse and also cause excretion of considerable amounts of solids. The **steam bath** is especially effective in securing free secretion from the skin and probably by revulsion aids in the re-establishment of the

renal secretion when this is partially or completely suppressed. A steam bath is always possible in any place where hot water and a few bottles, ears of corn, or corn-cobs are obtainable. Thus, lay the patient upon a blanket and cover him with another supported by barrel hoops, or any other device which will secure an air-space around the patient. Now place inside, carefully avoiding laying them in contact with the patient's skin, six or eight large bottles or preserve jars filled with hot water, some of which are wrapped in towels loosely wrung out of hot water. If ears of corn or corn-cobs be used, boil a dozen or more of these for a sufficient time and place them as directed for the bottles, except that no wet towels will be requisite, sufficient moisture being contained in the porous cobs.

Diuretics increase the number of germs excreted by the kidneys as well as eliminate large amounts of toxic products.

The special drugs to be employed to produce catharsis, diuresis, or diaphoresis need not be specified, because they must vary with the case, and you have elsewhere been taught the varieties and properties each of the remedies belonging to these classes of drugs.

Special remedies, such as colchicum, salicylate of sodium, mercury, etc., are of benefit because preventing the formation, directly or indirectly, of the injurious substances produced in gouty, rheumatic, or syphilitic subjects, or aiding in their elimination. Iron and cod-liver oil, by improving general nutrition are occasionally useful, while iodin often proves beneficial in tubercular and other conditions, as well as in syphilis.

Pain exhausts the old and the young, hence increases the frequency of the contractions of the heart while its force is decreased ; this is largely the result of loss of sleep and inability to take enough nourishment. For all these reasons sleep must be secured, oftentimes by the use of such drugs as sulphonal, chloral combined with bromid of potassium, codein and opium. When dealing with an

individual addicted to the use of alcohol in any form—I do not say one who frequently, but even one who never has, been intoxicated—regard restlessness or sleeplessness with grave suspicion, lest delirium tremens be imminent, and to avoid any possibility of this secure a proper amount of sleep and the ingestion of sufficient nourishment. The sudden withdrawal of all stimulants in men accustomed to their use is sometimes most prejudicial in its effects, precipitating an attack of delirium tremens. I have more than once seen a few good-sized doses of whiskey, administered at intervals of a couple of hours, put a case of delirium tremens promptly to sleep, when very large doses of opium had completely failed to produce even drowsiness. Elsewhere this subject will be more thoroughly considered, and the limitations of the exhibition of alcohol will be discussed.

If, despite all efforts scientifically directed toward the removal of all the microbic and those secondary causes which so often maintain inflammation, the conditions do not improve ; or when it is manifest that the causes cannot possibly be removed or their effects restrained within safe limits, the aim must then be to prevent molecular death or the death of the patient, even at the cost of destruction of the part, as by using the hot iron in a case of gangræna oris or amputating a limb for a hopelessly destructive septic process.

While devoting a proper amount of attention to all that has been mentioned, the practitioner must remember that he is dealing with a patient not a machine, and see that proper food in adequate amounts be not only supplied but ingested ; that bed-sores are not permitted to occur; that the bladder does not become distended, because never entirely emptied at each act of micturition, or because no urine can be passed in the recumbent posture by many individuals ; that proper sleep is secured, and that in a drunkard delirium tremens is thus warded off ; and finally, that elimination is properly carried out by attention to the secretions of the skin, bowels, and kidneys.

Treatment of Chronic Hyperemia.—Because chronic hyperemia is so often incorrectly considered to be "chronic inflammation," the general principles governing its treatment will here be considered.

The congestion being a passive one, either directly or indirectly due to difficulties presented to the free egress of venous blood, this determines a constant escape of leukocytes and liquid pabulum into the tissues; still further, the retention of an excess of venous blood in a part prevents the access of a proper amount of arterial blood. The objects to be attained are the bringing more arterial blood to the tissues, thereby increasing the vis-a-tergo, improving nutrition, and hastening the return of the venous blood. As secondary objects the removal of fluid exudate and the disintegration and absorption of the low-grade, neoplastic, fibroid tissue are to be earnestly striven for. The sluggish circulation in the part itself results from the dilatation of the vessels from causes which are often no longer operative. Under these circumstances, when accessible to the action of such agents, **astringents** are unquestionably of value. So-called **counterirritants,** such as tincture of iodin, sometimes do good, but not, as usually believed, by effecting revulsion. As commonly applied—viz., directly over the diseased area—they produce not only hyperemia of the skin but of the deeper parts—*i. e.*, direct irritation, not revulsion, occurs. Nevertheless, from dilatation of the vessels of the hyperemic area more arterial blood reaches the part, hence, when this is a desideratum, good follows irritants thus applied. The shape of a blister over the thorax will be outlined upon the pleura by a hyperemic area. Furneaux-Jordan long ago insisted that when iodin, the actual cautery, and blisters were applied over a superficial joint, direct irritation of the joint-tissues resulted instead of a lessening in the amount of blood contained in the hyperemic articulation. Counterirritation can then only be effected when congestion of vascular areas near by, but not continuous with those of the diseased

part, is produced, as the vascular areas above and below a joint. These are the results which must of necessity follow if the fact that irritation of a sensory nerve produces congestion in the area supplied by it be true, and this has experimentally been proven.

Elevation of a dependent hyperemic part is our most potent means of draining off the excess of venous blood. The indirect measures which bring more arterial blood and hasten the return of the venous blood and lymph also aid in the disintegration and removal of cellular exudate. Supporting pressure by means of an ordinary bandage, applied over an elastic substance such as cotton or oakum, as well as elastic bandages or stockings, act by compressing and narrowing the veins and also the other vessels to a lesser extent, thus increasing the velocity of the blood-current. Pressure also interferes with the access of pabulum to the neoplastic connective tissue, producing degeneration and absorption of this. Massage is of benefit when applicable, because it breaks down exudate and diffuses it over a wider and often healthier area for its absorption, empties the part of lymph and venous blood, and brings more arterial blood to the tissues. Electricity is also sometimes useful, because capable of producing the favorable vascular changes which I have stated are indicated.

Constitutional Treatment.—The pertinent question now arises, is there any such thing as a "constitutional treatment" for inflammation? Is there any special remedy or remedies that should be exhibited because the patient has an inflammatory process involving, for instance, the cellular tissue of a limb, which will directly influence the inflammation? Certainly not. Treatment must be guided by the indications present, and a purgative is not to be given because the patient has a cellulitis of a limb, but because the bowels are confined, or the eliminative powers of the intestines must be called into play, to tide the patient over a critical period induced by a toxemia, perhaps an enterosepsis. Patients who have been

eating too much or indulging in an excess of one kind of food, as meat, especially those who have led a sedentary life, should have their diet restricted, not because of any antiphlogistic effect upon the inflammation by restriction of the diet, but because the eliminative organs will have all they can do to get rid of toxins and the results of metabolism and must not be required to excrete large amounts of nitrogen, carbon, etc., which have been unnecessarily ingested. Moreover, if fever be present, fermentative changes in the food are apt to occur, which result in the production of poisonous substances, whose absorption will either directly cause grave symptoms, or being eliminated by certain organs will prevent them from performing their depurative duties, thus allowing the accumulation of bacillary proteids and tissue-waste in the blood. Patients whose habits are known, or who present the appearance of those who eat and drink too much, in whom the intestinal digestion and evacuations are imperfect, where, as used to be said, there "is congestion of all the chylopoietic viscera," are often benefited by laxatives, notably calomel. This drug acts well because acceptable to an often irritable stomach and because it is an intestinal antiseptic. In addition, restriction of—not always total abstinence from—certain articles of food and drink must be insisted upon. The intestines being freed from retained contents and stimulated to proper action, cease to be a source whence such large amounts of poisons of intestinal origin can be absorbed as will embarrass the liver when it is required to do extra eliminative work, as is often the case during a serious inflammation. Copious draughts of water will often be advantageous because of the free flushing out of the kidneys produced. Remember, however, never to alter the patient's habits radically, *without a distinct indication.*

Get the histories of your patients, find out what they eat and how much. If underfed—and this is sometimes difficult to ascertain, owing to false pride on their part—

feed them up ; if anemic, strive to ascertain the probable
cause and remedy this if possible. When alcohol has
been used, ascertain the form, quantity, etc.—for whis-
key will not do for a beer drinker—and get along with as
little as is safe of the variety habitually imbibed, lest
delirium tremens result from a too sudden withdrawal
of the accustomed stimulant. If practising where mala-
ria is prevalent, or when the patient has recently come
from such a locality, quinin may be used as a precau-
tionary measure, but is better exhibited after an exami-
nation of the blood has shown the certain or probable
presence of malarial organisms. In young vigorous
patients, at the outset, it is wise to restrict the quantity
somewhat and to be careful as to the quality of the food, for
the reasons already given. If the patient be either old
or young with a poor circulation and indifferently nour-
ished, do not restrict the quantity, but see to it that the
food is easily digestible. If the patient at any age be
asthenic, give as large amounts of easily digestible food
as is compatible with comfortable assimilation and the
ability of the excretory organs. Meat-broths, oysters,
scraped raw beef, eggs, and milk alone or in combination
with eggs should be given, *in measured amounts*, and at
as regular intervals as possible. From 2 to 6 pints of
liquid food is about as much as the average patient
can assimilate ; some can manage much less. In ex-
ceptional instances semi-solid food agrees better than
liquid. Water is rarely necessary when liquid diet is
employed ; but if for any reason indicated, and it dis-
tresses the patient when taken by the mouth, it may be
administered in the form of rectal enemata—4 to 8 ounces
every four, six, or eight hours. Farinaceous food may be
given to supplement the liquid diet, and later, as the
patient's digestion permits, vegetables and meats may be
added. Great judgment is sometimes demanded to hit
the happy mean between the administration of insufficient
nourishment and overtaxing the digestive and eliminative
organs, a pernicious blunder which is not uncommonly

made and is perhaps worse for the patient than a somewhat scanty diet.

In certain rare cases of head-injuries if the patient be vigorous, albumen-water, barley-water or even plain water may alone be given during the first twenty-four to even forty-eight hours, and despite all theory, these patients suffer less from headache, mental confusion, and dizziness than when placed upon a more generous diet. When, at any stage of an inflammation, the patient seems to be losing strength, more food, if it can be appropriated, strychnin, digitalis, alcohol, etc., should be exhibited, the choice or combination depending upon the condition of the digestive organs, and the deficiency or normal amount and character of the renal secretion. It is better to determine by auscultation of the heart when to commence the administration of stimulants, rather than to be directed solely by the condition of the pulse. A heart whose first sound is prolonged and strong is not in need of assistance, while if the first sound approaches in time and strength the second sound, then the organ is in urgent need of stimulation. Quite often the pulse seems fairly good when auscultation suggests a different idea, and *vice versa.*

Finally, keep track of the condition of the kidneys and intestines by frequent personal observations, to ascertain whether they are eliminating properly. Abnormally offensive stools indicate a degree of intestinal fermentation which may prove serious, and intestinal antiseptics, purgatives, or both are indicated. Deficiency in the quantity of urine and the solids contained demonstrate the necessity of increasing these if possible, and if this be impossible, of calling in the assistance of the skin and intestines.

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LECTURE XI.

CHANGES IN BLOOD; LEUKOCYTOSIS; DIMINUTION OF HEMOGLOBIN; THROMBOSIS.

AT this stage of our study of the Principles of Surgery it becomes necessary to consider certain facts relative to the blood, which have recently been shown to be important in the diagnosis and treatment of several surgical affections. While there are many other problems of interest, I desire at present to confine my remarks chiefly to leukocytosis, because of the increase of the white cells of the blood during acute, especially suppurative inflammation.

Leukocytosis means a temporary increase of the white cells of the blood as contradistinguished from their permanent increase in leukemia. The increased number and disintegration of these cells and the consequent presence of an excess of fibrin-ferment and of paraglobulin in the blood accounts for the well-known increased liability to thrombosis of the vessels during true (infective) inflammation. The different forms of cells detected and the proportion of each present, at times serve a useful purpose in the differential diagnosis of certain surgical affections.

Neither increase in the number of blood-plaques nor deficiency of the leukocytes has yet been proved to be of any certain pathological significance, hence these points will not be referred to further. The varieties of leukocytes and the proportion of each in normal blood will now receive brief notice. Taking 5,000,000 red cells per cubic mm., for men and 4,500,000 for women, 7000 to 7500 would represent the number of white cells, of all forms, present in normal blood.

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The following forms are recognizable :

Lymphocytes, small leukocytes with a single rounded nucleus and but little protoplasm ; these compose somewhat over 20 per cent. of all leukocytes.

Large mononucleated cells, with a greater amount of protoplasm than the former possess, present only in the proportion of 2 to 3 per cent.

Certain cells, forming about 2 to 4 per cent. of the total, with rounded, often lobed, but somewhat irregular nuclei, in whose protoplasm are granules staining only with acid anilin dyes, refusing to take the basic ones : from the readiness with which these stain with eosin they are called "eosinophile" leukocytes.

About 70 per cent. of all leukocytes are either actually polynucleated, or the main portions of their nuclei are united by such delicate prolongations that they appear to be two or more separate bodies. The nuclei stain readily, but the granular protoplasm feebly, unless a mixture of acid and basic anilin stains be employed, hence these cells are called "neutrophile cells." Certain transitional forms are found, which, being of no special importance, will receive no further notice.

Although the proportions of these cells may vary slightly in health, they remain fairly constant, so that decided changes, amounting to an increase of one-sixth or more, are often of great diagnostic import. Thus, some general septic conditions closely simulate typhoid fever. Again appendicitis may, from systemic infection, put on the guise of typhoid fever with exceptionally severe ulceration, hence peritoneal symptoms in the iliocecal region. In typhoid fever, however, unless there be some complication, such as pneumonia, or local or general sepsis, leukocytosis is absent. Leukocytosis is either absent or very slightly marked in non-suppurative (so-called "catarrhal") appendicitis. An exceedingly limited suppurative inflammation will cause a distinct leukocytosis, as I often observed as long as twenty years ago. Some surgeons, as Cabot, rely so

much upon the absence or presence of leukocytosis as to decline to undress an open fracture, even when general symptoms would suggest the propriety of this, if no leukocytosis exists. While unwilling to go so far as this, for some time I have placed almost implicit reliance upon leukocytosis as proving the existence of an infective inflammation, probably of a suppurative type.

Leukocytosis is absent in uncomplicated tuberculosis, while in malignant disease, especially in rapidly growing sarcomata, it is very common.

Suppurative inflammations of mucous surfaces, such as cystitis and endometritis, where free exit for all discharges exists, do not give rise to leukocytosis. On the other hand, pocketing of pus in suppurating wounds will generally give rise to it. Infective osteomyelitis shows marked leukocytosis, this symptom being thus of great value in suspected cases where a deep-seated bone is concerned, enabling the surgeon to insist upon early operation. The application of the fact that leukocytosis is an early symptom of infective and suppurative inflammation is of wide applicability, and needs no further illustration.

The **red cells** are chiefly of interest to the surgeon because a scarcity in their numbers indicates a deficiency of the oxygen-carriers, and hence the necessity of avoiding all unnecessary loss of blood during an operation. Yet sometimes when the number of cells is not seriously lessened, the material upon which their power of carrying oxygen depends may be diminished out of all proportion to their lessened number. I recently operated upon a patient whose red cells were reduced less than one-sixth, yet whose blood contained only 50 per cent. of hemoglobin.

Probably hemoglobin rarely equals 100 per cent., 95 per cent. being fully up to the average for men, with 3 to 4 per cent. less for women. Reduction of hemoglobin to less than 20 per cent. probably always means death by collapse, and just in proportion as the deficiency of hemoglobin approaches this point so is the danger. The

chance of eventual recovery can often be quite accurately determined by the rate at which the hemoglobin is regenerated. According to Osler mere regeneration of the number of cells may take place at the rate of 50,000 per c.mm. per day ; but as before mentioned, this does not always mean that the total hemoglobin of the blood is proportionately increased. Rapidity of the pulse is often explainable by deficiency in the number of red cells or the amount of hemoglobin they contain, because fewer cells having to carry the oxygen they must complete the round of the circulation oftener, doing double or treble duty, as it were ; this accounts largely for the increased frequency of the pulse noticed after severe losses of blood.

In my service at the University Hospital I have found that the hemoglobin is always markedly diminished in rapidly developing sarcomata ; in carcinomata the same probably holds good, but the change is not so marked. After removal of large sarcomata (and probably this is always more or less true for carcinomata) the hemoglobin increases, my experience in this respect confirming that of Park and others who state that a very decided, early, and persistent increase of hemoglobin indicates that complete removal has been effected ; while if this does not obtain at all, or the increase of hemoglobin is not maintained, a radical operation has not been done or visceral metastases have taken place.

The results of changes in the blood-vessel walls must now be studied, as well as certain abnormal elements which accidentally reach the blood-stream. This necessity arises because such knowledge is an essential preliminary to the study of the pathology and treatment of many wound-complications. Thus, the conservative efforts whereby natural hemostasis is secured unfortunately lay the best possible foundation for the development of pyemia if infection occurs, and hence explain why infection is especially dangerous in tissues, such as bone, where, anatomically, extensive hemostatic throm-

bosis must occur. Some vascular changes are diagnostic; moreover, the vessels are the avenues by which germs, toxic materials, and accidentally present pathogenic substances, such as fat, are disseminated throughout the organism.

Thrombosis first deserves consideration. This means the ante-mortem formation of a clot in the heart or blood-vessels. Although there are other co-operating favoring circumstances, damage to, or loss of vitality of, the vascular endothelium seems a prerequisite. This may be physical, by a cut, a tear, the compression—*i. e.*, laceration—produced by a ligature, or the chemical action of bacterial products.

The bearing on thrombosis of the presence in the blood of increased numbers of polynucleated leukocytes during inflammation demands a brief study. Since all the fibrin-ferment and most of the paraglobulin are contained in the leukocytes, so long as there is no undue disintegration of these cells there can be no increased tendency of the blood to clot. As a matter of fact this tendency is present during inflammation. Let me make a few more statements, and the explanation of the well-known clinical fact that thrombosis is very liable to occur during inflammation can be made clear.

Generations ago it was experimentally shown that the coagulation of the blood contained between two ligatures applied to a living vein was retarded for long periods, while this same blood withdrawn from the vessel soon coagulated. Again, although the blood may be maintained at complete rest in an aneurysmal sac, coagulation often fails, but when there is little or no such rest of the contents, coagulation may be started by, and this may rapidly extend from, even a slight point of damage to the endothelium, as Macewen has shown. This damage causes first the accumulation of blood-plaques and later of leukocytes. It is then by virtue of some property exercised by the healthy vascular endothelium that coagulation does not occur, either because disintegration

of the leukocytes is prevented, or any fibrin-ferment accidentally present is rendered inoperative ; this latter action has apparently been experimentally proved. If healthy endothelium can so act in health, so it will in disease ; but if there is present a large amount of material, ready to provide an excess of fibrin-forming materials, but little damage to the endothelium will be requisite ; hence the presence in the circulation of bacterial proteids resulting from inflammation may either directly damage the endothelium sufficiently, or its nutrition may be so altered as to deprive it of the power of preventing coagulation.

Among the **predisposing causes** of thrombosis, one indeed which takes first rank, is "slowing" or arrest of the circulation. The causes of this "slowing" may be central or local or both, the former being a weak heart, however produced, which is the chief factor in the production of the so-called "marasmic thrombosis," although blood-changes, notably leukocytosis, probably assist. Local predisposition arises from obstruction to the exit of venous blood, often aided by the diminished vis-a-tergo due to loss of elasticity of the arteries induced by calcareous or atheromatous changes. The pressure of a tumor will at times likewise so interfere with the exit of venous blood as to produce the "slowing" necessary for a rapid vascular thrombosis. Complete arrest of the blood-current, resulting in thrombosis, may be produced by the pressure of a tumor upon a vein, or by partial or complete division or rupture of an artery or vein. Again, embolism of an artery, even if complete blocking of the lumen is not at first induced, will soon lead to this by extension of the clotting process. Ligation of an artery will, of course, produce thrombosis of the vessel at the point tied ; but it may so reduce the force of the return venous circulation as to lead to thrombosis of the vein, especially when pressure, as of an aneurysm is exerted upon the latter vessel. Foreign bodies, such as detached fragments of tumors,

vegetations from the heart valves, etc., by multiplying the points around which leukocytes will gather, provide abundance of fibrin-ferment and paraglobulin for the rapid formation of fibrin. Infective micro-organisms lodging in the vessels of a part act both as foreign bodies multiplying the points of contact, and as a source of chemical injury of the endothelium.

Another cause productive of the necessary changes in the endothelium is aseptic death of the tissues surrounding a vessel, resulting in aseptic thrombosis, because the endothelium also dies from lack of nutriment. Bacterial products formed by the germs causing a primary tissue-infection will produce nutritive changes in the endothelium, usually by setting up a pyophlebitis, or by killing the tissues, a "primary infective thrombosis" resulting. An extension of the primary hemostatic thrombi of a wound often follows secondary infection, this form being termed "secondary infective thrombosis."

Any chronic arterial disease altering the physical or vital condition of the endothelium predisposes to thrombosis such as endarteritis in any of its forms, the atheroma so commonly following this with its calcareous changes, and notably the roughening caused by the rupture and evacuation of softened atheromatous areas— the so-called "atheromatous ulcers" of the older writers.

Thrombi then are primary—*i. e.*, of local origin—and therefore usually limited to the locality where the cause is operative ; and "propagated" or "spreading," starting indeed locally, but extending beyond the point where the primary cause is operative, or even originating at a distance, as the result of secondary influences.

Parietal, annular, partial, and obstructive or complete, as applied to thrombi, are terms which explain themselves. Although the first three conditions may remain so throughout, it is vastly more common for any one of the forms of partial thrombosis to become complete. Through late secondary changes, a complete thrombosis

sometimes presents apparent indications that it was originally only parietal, or annular.

Thrombi are more prone to form in the veins than in the arteries, because the conditions are normally more favorable. The thrombus once formed, in the case of an artery usually extends only to the next collateral branch above ; but it may reach farther upward, and possibly extend a little downward. In veins the thrombus passes far beyond the primary focus, sometimes extending as far as the vena cava when originating in the veins of the lower extremity.

The possibility that the veins of any organ may become thus blocked, especially the sinuses of the brain and the superior mesenteric vein, must never be forgotten, because arrest of the circulation in these localities is productive of such serious and often anomalous symptoms. The majority of thrombi which fill small wounds of the vascular walls are at first composed of blood-plaques and then leukocytes. These sometimes undergo organization, repairing the injured wall ; but later, from partial or complete arrest of the blood-current by the extension of the parietal thrombus, accretions of ordinary red clot may occur. When from any cause an ordinary blood-clot causes partial blocking of a vessel, the accretions consist chiefly of blood-plaques and white cells, because deposited from circulating blood.

Thrombi are classed as fibrinous, hematoblastic, leukocytic, red, hemostatic, etc., because composed principally of one or other constituent of the blood, but every thrombus contains all of the constituents in varying proportions. From the gradual manner in which thrombi are usually formed by deposition of fresh layers upon their exteriors, they present a laminated structure, and, owing to the different proportions of red cells present in the different layers, this lamellar structure is often readily distinguishable by the eye.

Changes must occur in every thrombus. While the first detectable by the eye is decolorization, invasion by leuko-

cytes and the changes mentioned on pages 56 and 57 have previously taken place. Calcification is not very uncommon, giving rise to the bodies called phleboliths. The alteration most to be dreaded is softening, which even when not due to infection may give rise to embolism, producing serious consequences if the fragments are swept away and lodged in an important organ. Softening, leading to such untoward results, is very rare, except when infection is the cause. The liquefied clot is reddish, pulpy, oily, grayish, or puriform in appearance, according to the number of red cells present, and, especially when a white one—*i. e.*, one composed chiefly of leukocytes—is concerned, macroscopically the material closely resembles pus, although the microscope at once corrects this erroneous impression. Later, when micro-organisms have actually penetrated the thrombus, or have originally been present, genuine pus does form as the thrombus with the vessel and the other environing tissues breaks down and forms an abscess.

LECTURE XII.

THROMBOPHLEBITIS; THROMBO-ARTERITIS; EMBOL-
ISM; METASTATIC PROCESSES; FAT- AND AIR-
EMBOLISM.

Thrombophlebitis or **thrombo-arteritis**—*i. e.*, a
spreading infective inflammation of the vessels, accom-
panied by an advancing thrombosis—is the usual cause of
these infective softening processes, and is a most fatal
malady, as witness the spread of thrombophlebitis from
the middle ear to the cerebral sinuses and to the brain
itself.

Embolism, a process already incidentally mentioned,
demands thoughtful consideration by the surgeon. Em-
bolism is the process by which blood- or lymph-vessels
are occluded by substances—emboli—brought to their
points of arrest by the blood- or lymph-stream. The
embolus or plug may completely occlude the lumen of the
vessel where it lodges, or at first it may only partially
block this, later additions nearly always taking place
which render the obstruction complete. The material
composing these plugs is most often fragments of thrombi
or blood-clots, although vegetations torn away from the
heart valves, portions of degenerated intima of the heart
or blood-vessels, masses of micro-organisms, fat globules,
myeloplaxes or liver cells, after traumatisms of or hemor-
rhages into the medulla of bone or the liver, aggregations
of pigment, even air may form them.

These emboli are dislodged from the site of their
formation by movements of the part, by traumatism, or
by the force of the blood-current increased by a more
powerful action of the heart, whether this be induced

by exertion, mental emotion, or otherwise. While it is possible thus to dislodge a part or the whole of an unaltered thrombus, it is extremely unlikely unless precedent softening has occurred, which rarely takes place to any dangerous degree unless infection has taken place.

Emboli having their origin from a thrombus located within the pulmonary circulation are most apt to lodge in the lungs, although they may pass the lung-capillaries and reach the systemic circulation. Those originating in the systemic circulation are most often arrested in the vessels of the lungs, brain, spleen, or kidneys. If the thrombus, by fragmentation of which the emboli are formed, be situated in a radicle of the portal vein, the emboli will lodge in the liver, unless very minute, when they may reach the right side of the heart and thence be distributed. It must not be overlooked, however, that these primary emboli may initiate a thrombosis which in turn may give rise to secondary emboli, thus accounting for some of the apparently anomalous or irregular distribution of emboli so often observed.

The importance of metastatic processes depends chiefly upon the composition of the emboli. If an embolus contains chemically active materials of microbic origin —*i. e.*, is a "septic embolus"—necrobiotic changes of the tissues of the vessel and of those which surround it must ensue. If, in addition to their products, it contains micro-organisms which are capable of multiplication, the "metastatic infectious embolus" will become a new center of microbic growth, forming a possible fresh center for metastasis. Should the embolus consist of or contain cells capable both of maintaining their vitality and of multiplying, new centers of growth for such malignant neoplasms as sarcoma or carcinoma will be formed. Some benign growths, such as the famous case of chondroma of Paget, under extraordinary circumstances have undergone metastatic dissemination ;

but such a case is unique, supposedly similar ones having always shown evidences of sarcomatous tissue.

The ease with which the vascular metastases are recognized seems to have overshadowed the equally important fact of lymphatic metastases, which, like those occurring by means of the blood-vessels, usually take place in the direction of the normal current; yet "retrograde metastasis" may occur in the lymph-circulation exactly as in the vascular, from blocking of the normal, direct route.

The term "retrograde metastasis" requires explanation. It is rendered possible in the peripheral vessels by reversal of the ordinary direction of the current because of obstruction of the direct current through distal blocking of the vessels. Arnold has shown that when artificial emboli are introduced into such large veins as the jugular and femoral, which from their size are unable to enter, much less pass, through the capillaries, they are "carried, by a current running in a reverse direction, not into the trunks but into the smallest branches of the veins in the liver, kidneys, heart, extremities, dura and pia mater, and orbits, as well as into the posterior bronchial veins."

The result of the lodgement of an aseptic embolus in a vessel of a young person, or in one of those of an older individual with dilatable vessels, is merely a temporary lack of direct blood-supply to the area nourished by the occluded vessel, the collateral circulation soon amply sufficing, unless the vessel be a "terminal" one, when most serious consequences must follow, as will now be explained. Of course, even when the artery is not a "terminal" one, if the collateral vessels do not dilate sufficiently, necrobiotic changes or gangrene of the parts supplied by the blocked vessel must follow.

When an artery is "terminal" or nearly so, the tissues supplied by it must either undergo simple coagulation-necrosis, and subsequent degenerative changes, or coagulation-necrosis followed by hemorrhagic infiltra-

tion ; in other words, "hemorrhagic infarction" is determined.

By an *infarct* or *infarction* is meant the more or less conical altered area of tissue which was formerly supplied by the artery occluded by the embolus. If there exists practically no communication with the capillaries of the surrounding tissues, simple coagulation-necrosis and certain later changes take place, including the possible occurrence of moist gangrene from infection of tissues whose vitality has been reduced to a minimum by total deprivation of pabulum. If the vessel which is blocked is not absolutely terminal, but its capillaries have some communication with those of the environing tissues, a reflux of blood takes place, and, as Rindfleisch has pointed out, a higher pressure prevailing in the vessels of the infarct than in the surrounding normal capillaries, it is not surprising that, aided by the coagulation-necrosis almost invariably present in the vessels distal to the embolus, vascular rupture should occur, a "hemorrhagic infarct" resulting. When hemorrhagic infiltration does not occur, the parts remain bloodless, an "anemic infarction," as it is termed, resulting.

Embolism of the mesenteric artery is second in importance only to embolism of the brain, indeed is a far more fatal condition, leading to necrosis of the parts involved. The symptomatology of neither condition can be pursued, the occurrence of embolism in these localities having been cited merely to show the importance of the process under consideration.

The general indications for the **treatment of thrombosis** and **embolism** are to maintain the life of the part by favoring the development of both the venous and arterial collateral circulation, and securing the freest possible return of the venous blood. Maintenance of the warmth of the part and elevation are means to secure these results when applicable. When tissue-death results, the diseased parts must be removed if accessible, as a gangrenous patch of lung, a mortified limb,

necrotic bowel, etc. If the process be a septic one, the measures indicated are those appropriate for the treatment of the same local or general conditions induced by infection otherwise produced.

Fat-embolism.—By fat-embolism is meant the occlusion by minute fat-globules of arterioles and capillaries, chiefly the latter, the embolic material having gained access to the circulation on the venous side. This accident is not uncommon after crushes of bone, especially open ones (except in children, because they have

Fig. 11.—Showing fat-emboli (stained with osmic acid) occluding a number of the lung vessels.

so little fatty medulla); extensive injuries of the panniculus adiposus; traumatism of or hemorrhage into the liver or the medulla of bone; and acute infectious processes in bone. Park makes the statement, evidently experimentally not clinically determined, "that fat-

embolism may occur when fluid fat has been passed into the heart through the thoracic duct, although more slowly.'' As the data for this statement are not given, as well as those upon which the allegation is founded that the same accident can occur from absorption of fat from a serous sac, the possibility of fat-embolism thus originating is suggested, not affirmed. It is also believed that fat may enter the opened vessels during an operation. First and chiefly the capillaries of the lungs are blocked, then, if the force of the circulation is competent to drive the fat through these vessels, the capillaries of the brain, spinal cord, choroid, liver, and kidney may one or all become blocked ; of course, the vessels of other organs may also suffer. The fat is most often finally arrested in the liver and kidneys, where it is disposed of in non-fatal cases. Oxidation and saponification effected by the alkaline salts of the blood also aid in getting rid of the fat.

Symptoms.—These are often confounded with or complicate shock. Opinions vary as to the frequency and importance of fat-embolism, but none deny the serious nature of this accident if extensive capillary areas of important organs remain for any length of time occluded. The symptoms in minor degrees of fat-embolism, where the occlusion is only temporary, however pronounced they may be at the outset, are evanescent. The primary dangers from fat-embolism and the earlier symptoms are those of pulmonary embarrassment with edema, deficient oxidation of the blood soon becoming pronounced — *i. e.*, acute asphyxia results. This is shown by restlessness, rapid respiration, increasing dyspnea, pallor, promptly succeeded by more or less decided cyanosis, and a rapid pulse. The later symptoms result from obstruction of the capillaries of the organs due to the lodgement of such fat-globules as have been forced through the lung-capillaries. In addition to the pulmonary symptoms, mental excitement, somnolence, and coma may succeed. The respiration,

rapid from the outset, later becomes stertorous, the pulse irregular and feeble. Pulmonary edema is apt to become marked, with expectoration of frothy, blood-stained mucus, or actual hemoptysis occurs. Symptoms indicative of involvement of the spinal cord are at times detectable. The temperature is usually subnormal at first, may remain so, or may become elevated if complications arise.

Diagnosis.—The occurrence of fat-embolism may at first be difficult to detect, if pronounced shock also exists. Fat-embolism usually presents symptoms only after any shock which has been present has passed away, or when this was never pronounced. This time-rule is by no means absolute, because cases have been reported where the accident occurred and death resulted inside of twelve hours. Except at the beginning, the pallor so pronounced in shock is replaced by cyanosis, and the rapid and, later, stertorous respiration differs materially from the feeble, sighing character of that observed during shock. The pulmonary and renal congestions sometimes following prolonged etherization may be confounded with fat-embolism. If free fat is detected in the urine the diagnosis will be clear, otherwise it must often remain for the time in doubt. Unless the fat comes from an infected source or sepsis supervenes, fat-embolism is unlikely to be confounded with acute septicemia, because of the gradual onset of the symptoms in the latter, with the pronounced temperature rise. Still, sepsis may later complicate fat-embolism. The previous recognition of the existence of fat-embolism, or the detection of fat in the urine should insure a correct opinion. The discrimination of fat-embolism from other acute pulmonary affections occurring independently of the former should be possible with ordinary care. The occurrence of acute suppression of urine after a serious injury or operation should always excite a suspicion of the presence of fat-embolism. Cerebral symptoms may, indeed, be due to hemorrhage or ordinary embolism. In

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fat-embolism they commence gradually with the precedent symptoms of fatty lung-embolism, or when of later development the wound-fluids contain much free fat, which can also be detected in the urine. Hemiplegia is usually absent, coma gradually supervening without distinct paralysis, while the detection of fat-globules in the urine will settle the diagnosis. In addition, the history of rapid respiration with dyspnea should suggest a lung-embolism which had escaped detection.

Prognosis.—Severe cases are nearly always fatal, while for slight fat-embolisms recovery is the rule. The most dangerous period is the first forty-eight hours; but if this be successfully passed, recovery will probably ensue. Recovery depends upon the ability of the heart to free the occluded capillaries by forcing the fat through them in time to prevent either asphyxia or grave secondary changes in the pulmonary tissues, and the subsequent escape of the brain, spinal cord, etc., from serious involvement produced by the lodgement of the fat which has passed beyond the lungs. The possibility of fresh increments of fat entering the circulation must be taken into account. Unless originating from a septic focus or secondary infection occurs, nothing beyond "hemorrhagic infarcts" follow.

Treatment.—The indications are to increase the vis-a-tergo, to dilate the arterioles and capillaries as much as possible to permit the readier passage of the fatty accumulations, and to supply enough oxygen both to maintain life and aid in the removal of the fat by increased oxidation. The administration of strychnin, ammonia, and alcohol will fulfil the first indication, while belladonna, and possibly nitrate of amyl cautiously employed, will secure the maximum dilatation of all the peripheral vessels, hence those of the lungs. Although digitalis is usually recommended, it should not be used, because it will increase the contraction of the peripheral vessels. Inhalations of oxygen should be tried. Because fresh amounts of fat may enter the circulation, absolute quiet

of the part and of the individual must be secured at any cost, lest movement set free additional fat by rupture of more fat-cells. Experiments having shown that the introduction of a laminaria tent into the medulla of a bone is competent to *cause* fat-embolism, the tension actually forcing the fat into the circulation, free drainage must also be provided for all wound-secretions.

Air-embolism.—Although air-embolism has always been one of the rarest of surgical accidents in the past, and should be still more so when general anesthesia is employed, yet the possibility of its occurence demands that the whole subject shall be thoroughly considered. The danger of air-embolism is chiefly due to the impossibility of the right heart forcing the air through the lung-capillaries, large areas of these becoming obstructed. This results both from the inherent difficulty of the task, and because the valves are not so readily closed by the pressure which a bloody froth can exert as by that of the fluid blood. The left heart, nearly empty of blood, fails to supply with blood the nerve-centers essential to life and the heart itself, hence the symptoms and death. Quite large quantities of air can be slowly introduced into the circulation of animals without dangerous consequences, the air failing to cause the lung- and heart-complications, because at no one time is there enough present to block the lung-capillaries, and it passes through them and is found in the capillaries of many organs, being there disposed of. Smaller amounts, if suddenly introduced, will cause death in the human subject. Operations in the axilla, about the base of the neck, and such as involve the cerebral sinuses are those in which this accident is most likely to happen. "Canalization" of veins predisposes to air-embolism—*i. e.*, a condition, normal or acquired, which by converting a vein into a rigid tube prevents it when cut from collapsing. The cerebral sinuses are anatomically incapable of collapsing from atmospheric pressure, and so are some veins in the "dangerous regions" just mentioned, owing to their perforating or

being attached to dense fasciæ. Inflammatory induration of a vein-wall, or a similar induration of the tissues surrounding veins also produce "canalization." Putting the vein upon a stretch just as it is cut will cause temporary "canalization." When any such favoring conditions are present, if a patient be not anesthetized, the sudden, deep inspiration following the holding of the breath induced by the pain of a cut, or while struggling to get free, renders it very likely that air will be drawn into an opened vein emptied of blood by the inspiration.

All cases where, after sudden death, air or gas is found in the veins, are not due either to air-embolism or to the entrance of gas formed during putrefactive changes in wound-fluids under pressure. Modern research has shown that this condition sometimes results from the rapid growth in the blood of aërogenetic bacilli.

Symptoms.—A hissing noise is heard, and bubbles of air are seen in the wound ; there is sudden heart-failure, with irregular respiration and dilatation of the pupils. A "churning" systolic sound is recognizable on auscultation over the heart, convulsions quickly commence, and death soon follows.

Treatment.—This should be chiefly prophylactic, because introduced in sufficient amount the time which will elapse before death takes place will not suffice for the adoption of efficient therapeutic measures. Complete anesthesia must be maintained when operating in the "dangerous region." If anesthesia for any reason be impossible, the arms should be confined to the sides and the movements of the thorax be restrained by a firm binder. The head must not be much elevated when operating within the cranium, lest air enter an open sinus. Structures which may contain large veins should not be put upon the stretch while being incised. Veins had better be tied when possible before division. Should air-bubbles be seen in the wound or a hissing sound be heard, fill the wound with water or blood by a squeeze of a sponge, compress the vein by the finger until it can

be clamped with forceps, stop further administration of the anesthetic, institute artificial respiration with lowered head to retain a functioning amount of blood in the brain-centres, give strychnin both as a cardiac and respiratory stimulant, and atropin as a respiratory one and to aid in securing the maximum dilatation of the clogged pulmonary capillaries. In a few cases life will be saved by the prompt employment of such measures.

LECTURE XIII.

SURGICAL FEVERS: SAPREMIA; SEPTICEMIA; PYEMIA.

FEVER has already been incidentally considered as a symptom of inflammation. Several of the most important wound-complications are invariably accompanied by fever, indeed their most patent symptoms being febrile, they have often been described as "fevers," practically ignoring the fact that the combination of phenomena we call fever is merely the evidence that some pathological condition exists, and not the essence of that condition. It is of first importance to understand that in all fevers observed during the course of any surgical affection the rise of temperature is due more to excess of heat-production than to lack of heat-elimination. Again, because fever is a symptom, all the possible causations of each factor must be studied lest serious practical error result. When the onset is gradual neither chill nor rigor occurs; but when there is a large dose of poisons suddenly thrown into the circulation chill is practically certain to result, unless the vital powers be overwhelmed. But an actual chill, or the statement by the patient that he has felt chilly or has shivered with cold, is not always indicative of the absorption of toxic substances, for no rise of temperature or any other symptom of fever may follow. This variety of chill has been called for lack of a better name, a "nervous chill." While this does occur, it is only by close and repeated thermometrical observations that we can be sure that this supposed "nervous chill" is not in reality a "septic" one, resulting from a small dose of poison in one with an unusually susceptible nervous

system and in whom a repetition of the dose, or a larger one, will occur later, unless the unsuspected source of infection be diligently sought for and, being found, is removed.

All the febrile symptoms occurring after any traumatism or operation are due to the presence in the blood of some pyrogenous substance usually, but not always, originating in and absorbed from the wound.[1] These pyrogenous substances belong to two very different classes—viz., the aseptic and the septic.

The aseptic pyrogenous substances are fibrin-ferment, the nucleins of the tissues, and similar proteids, the results of increased metabolism or of tissue-destruction. None of these produce much beyond a higher bodily temperature and a somewhat more rapid pulse; but the arrest of the secretions and other evidences of the toxemia of true fever are all absent, as described in Lecture VIII. when treating of "aseptic fever." As this "aseptic fever," which lacks most of the characteristics of genuine fever, is usually self-limited, subsiding spontaneously when all the original stock of pyrogenous substances has been absorbed from the wound, the question of treatment might seem of no importance; nevertheless, when very marked, it may possibly be just the slight additional depressing influence which will so lower local or somatic vitality that a primary or secondary infection, too slight to become operative in normally resistant tissues, will now prove effective. Again, "enterosepsis"[2] may complicate true "aseptic fever," and this in turn pave the way for a true septic infection; therefore, it is sometimes wise to accelerate the disappearance of aseptic fever by cold sponging, laxatives, and free diuresis, induced by drugs or preferably by the ingestion of large amounts of fluids, especially water. These latter may be administered either by the mouth or rectum, or by both avenues. Careful regulation of the diet is also advisable, both as a prophylactic against enterosepsis

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[1] See Autointoxication, page 178.   [2] See page 103.

and as relieving the emunctories of all unnecessary labor.

Sapremia.—The name sapremia should theoretically be applied only to the constitutional effects produced by the absorption of the chemical products of the growth of saprophytic germs. In addition, however, it is probably true that at times the toxins manufactured by the pyogenic cocci are taken up in sapremia, adding to the systemic poisoning, although the germs themselves, of course, do not gain access to the circulation. The two facts that putrefactive changes are prominent in the class of cases to which the term sapremia was originally applied—viz., puerperal—the discharges in such cases being very offensive, and that just as soon as the putrefying blood-clot is removed from the uterus, and disinfection—as shown by the absence of odor—has been secured, the symptoms quickly subside, go far toward proving that the putrefactive organisms manufacture most of the substances producing sapremia. Still further, the measures which prove efficient in putting a stop to sapremic intoxication would have but little effect in arresting the development of pyogenic organisms lodged in the interior of the uterine cavity, or in preventing their effecting a successful lodgement in the uterine tissues. Again, laboratory investigations have taught us that while saprophytic microbes grow with such rapidity that in a few hours they can manufacture poisonous amounts of ptomains, yet pyogenic cocci require a longer time to develop in sufficient numbers to produce toxins in dangerous quantities. This is not a fine theoretical point, but has an important practical bearing upon the therapeusis of septic conditions. Chemical substances are with difficulty absorbed when in contact with a healthy granulating surface, while if in solution in the wound and tissue-fluids of a recent traumatism, or in the peritoneal cavity—*i. e.*, an enormous lymph-space—are in direct osmotic relation with the blood and lymph, only the requisite animal mem-

brane intervening. A consideration of these latter facts must convince us of two things—first, that sapremia nearly always must commence soon after the traumatism has been inflicted; and second, that given a normal granulating surface throughout[1] any operative or accidental traumatism—*i. e.*, a number of days must have elapsed since the primary infection occurred—if systemic intoxication now takes place, it must be from germs manufacturing their poisons within the economy, not developing in the wound alone.

Sapremia cannot develop from a small point of infection, because there must be sufficient space for the accumulation of enough wound-fluids to undergo changes which will supply large amounts of chemical poisons, and also a surface extensive enough for their rapid absorption. It has been estimated that it requires the absorption of from 1 to 2 ounces of fluids saturated with the chemical products of germ-growth to produce sapremia. Sapremia, at one time well termed "septic intoxication," was formerly quite common after intra-abdominal operations, the abdominal cavity being in reality an enormous lymph-space, presenting unparalleled opportunities for the absorption of unlimited quantities of toxic substances. The rapidity of absorption is so great that the resulting death is even now sometimes, and formerly was frequently, considered to be the result of "shock."

To epitomize the conditions most favorable for the development of sapremia, and thus provide data for a differential diagnosis between this and septicemia; sapremia most often follows recent, extensive wounds which are kept neither aseptic nor properly drained; wounds of serous and synovial membranes which are not kept aseptic and efficiently drained; abscesses and granulating wounds in which septic discharges are retained under pressure, because free drainage is pre-

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[1] Of course, destruction of the granulations over any extensive surface by caustic microbic products or mechanically would invalidate this statement.

vented by too small an opening, the pressure mechanic-
ally rendering the protective barrier of granulation-
tissue ineffective.

Post-mortem examination shows, as would be ex-
pected from the gastrointestinal and nervous symptoms,
congestion of the stomach, intestines, and nervous
centers; the kidneys, through which the poisons are
chiefly eliminated, are also hyperemic. Marked dis-
integration of the red blood-cells is found, accounting
for the staining of the tissues and vessels, the detritus
often producing blocking of the vessels, causing their
rupture, and hence the petechiæ more or less generally
present throughout the body, but seen more especially
beneath the serous membranes. No germs are found in
the blood or internal organs, because saprophytic
microbes cannot flourish in normally vitalized tissues.

Prognosis.—In a typical case, because the poisoning is
a chemical one, unless the amount of the poisons ab-
sorbed either in one dose or continuously be necessarily
fatal, the prevention of the absorption of fresh incre-
ments, and the arrest of any further formation of poison-
ous alkaloids, should cut short the sapremia. This is
often both theoretically and practically possible by
affording free drainage and securing efficient disinfection
of a wound, by the removal of a limb, etc. Of course,
even when all these things have been promptly and
effectively done, a fatal dose of poisons may have already
been absorbed; yet sometimes, by securing free action
of the emunctories, what would otherwise unquestion-
ably prove a lethal dose is recovered from. Unfortunately
pyogenic cocci often coexist in the wound with the
saprophytic micro-organisms. The former only too
frequently gain a firm hold on tissues partially devital-
ized by the chemical products of putrefaction, penetrat-
ing whence through the lymph-spaces they soon reach
the blood, and now germs, capable of living, flourishing,
and manufacturing toxins wherever arrested, become
located in every part of the organism, true septicemia

being now engrafted upon and having been rendered more possible by the previous sapremia. Nevertheless, although a fatal termination is too often the outcome of this aggravated condition, it is theoretically and sometimes clinically possible to prevent fatal septicemia by ridding the economy of the poisonous alkaloids, thus enabling the blood and tissues to render inert or destroy a number of germs which otherwise would prove fatal.

Symptoms.—General malaise begins soon after shock has passed off, and the attack is often—but far from always—ushered in by a chill or severe rigor, with a sudden elevation of the temperature to 103° or 104° F., and with all the other manifestations of fever. Headache and nausea, possibly with vomiting, soon appear. Mental disturbance, frequently culminating in delirium, soon shows itself. The pulse becomes rapid and feeble and the tongue dry and coated. If a lethal dose of poison has been absorbed or a continuous absorption of ptomains obtains, rapid collapse occurs with subnormal temperature ; coma takes the place of any previous delirium, and death ensues. Sometimes an overwhelming dose produces collapse, and death takes place in coma without any of the symptoms first detailed. In contrast with this, after a medium dose of poisons or repeated non-lethal doses, the fever and nausea with occasional vomiting persist, diarrhea or purging appears, and the patient becomes anemic, icteroid, or actually jaundiced. Either death occurs from exhaustion, the tongue becoming dry, brown, and fissured, sordes accumulating on the lips and around the teeth, and the pulse becoming frequent and feeble, septicemia or pyemia sometimes terminating life, or recovery takes place gradually.

Hectic Fever.—Although not altogether fashionable to speak of "hectic fever" and fully recognizing that the condition so called is only a modified sapremia, yet it is so common a clinical condition as to demand special recognition. It gradually supervenes upon more acute forms of sepsis, the original fever declining in severity

as the system becomes partially habituated to the poisons, these being slowly introduced in smaller amounts, a sufficient daily elimination of toxic substances occurring during the sweating stage to secure a daily apyretic period. Hectic will show itself in any case of chronic suppuration where free exit for the pus cannot be secured, and is a danger-signal to be heeded, for unless its causes can be removed, in a marked case, death will eventually ensue from exhaustion or from amyloid disease of the liver, kidneys, or intestines.

Symptoms.—Every afternoon, usually without even chilliness, there is a rapid rise of temperature, the cheeks present a circumscribed flush, the palms of the hands often show similar appearances, the eyes are brilliant, the pupils large, and the pulse rapid and feeble. During the evening or night, profuse sweating occurs, usually during sleep. In the morning the temperature is normal; but the patient is pale and exhausted, the tongue is red and dry at the sides and tip, the pulse is rapid, small, and weak. The appetite steadily decreases, diarrhea begins, emaciation is rapid and marked, and death results from exhaustion, although with all the tissues so depressed in vitality secondary infection readily occurs, so that some hectic cases die rather suddenly of acute infective complications.

Treatment of Hectic Fever.—This demands that the source of the infection be radically attacked, and preferably by methods which will ensure complete removal of the infecting focus, or if this cannot be done, the most rapid healing possible must be secured, to save further drain; for instance, amputate a limb rather than resect or erase a diseased joint. Fresh air, sunshine, plenty of good food, stimulants, and tonics are indicated. In all other respects the advice given for sapremia is applicable here, modified to meet the altered circumstances.

Treatment of Sapremia.—This has been outlined by the explanations already given. Removal of the cause by free drainage and disinfection must be secured by

placing the part, when possible, in the continuous warm aseptic or antiseptic bath ; by the cautery even, as in hospital gangrene ; by amputation possibly ; by laying open a suppurating joint ; by amputation of a hopelessly infected puerperal uterus, etc. In the severe cases, secure free watery movements of the bowels by the exhibition of salines preceded by calomel, as is so successfully done for septic peritonitis. Increase the elimination of the kidneys by fluids and drugs, if the renal congestion will not thereby be increased, or relieve this and secure a freer secretion of urine by calling in the aid of the vicarious action of the skin, employing to this end hot-air or steam-baths; these last measures will sometimes be followed by free diuresis, when diuretics would only increase the suppression. Wash the economy free of poisons by saline transfusion—hematoclysis. Administer drugs capable of sustaining the power of the heart, such as strychnin, digitalis, etc. See that sufficient nutritious, easily digestible food is ingested. In the more chronic cases, when the diarrhea seems in excess of the requirements for elimination, endeavor to diminish the frequency of the evacuations by the use of intestinal antiseptics and the administration of mineral acids. Secure sleep, if possible, without the use of opium. By some or all of these measures most unpromising cases are occasionally saved, while in those less severe the scale is often turned toward recovery. There can be no question that proper measures will sometimes prevent the conversion of a sapremia into a septicemia, despite the pessimism of some writers. Tonics and nutritious food will prove useful during convalescence. I hardly need to point out that antistreptococcus serum is not only utterly useless, but probably will prove harmful in pure sapremia—a fact which presents an additional reason for attempting to distinguish between the various forms of so-called "blood-poisoning." Even delay by a resort to a bacteriological examination of the blood is warranted.

LECTURE XIV.

SURGICAL FEVERS (CONCLUDED); SEPTICEMIA.

WHILE fully admitting that sapremia may pave the way for septicemia by favoring the access of micro-organisms into the circulation, yet it is clinically necessary to describe two separate diseases, because first, there is a chemical infection entirely controllable by art, and second, if this be controlled or modified, either the genuine infective process may be prevented, or if not actually arrested, may be confined within limits compatible with final recovery.

In sharp contrast with the necessary conditions of a sufficiently large wound and absorbing surface for the production and entrance of enough alkaloids to produce sapremia, uncomplicated primary septicemia often starts from some trivial wound, as, for instance, a slight cut or a needle-puncture received during a post-mortem examination of a body of a patient who has died of septicemia, a wound manifestly too small to permit either the accumulation of enough wound-fluids to generate the chemical poisons of sapremia, or even if formed—which is impossible—to admit of their absorption rapidly enough and in sufficient quantity to produce sapremia; hence, an incubation period of from two to three days must elapse before the germs can become diffused, located, and multiply sufficiently to form enough toxins to produce the systemic effects of septicemia.

Symptoms of Septicemia.—When occurring after a wound or operation there is usually some slight fever, which is first observed soon after reaction has taken place. This may, at the outset, be genuine aseptic fever, or slight

chemical intoxication—*i. e.*, sapremia—and it lasts during
the incubation period of the septicemia, merging with the
symptoms of that disease; or this may suddenly evidence
its onset by a chill. If resulting from a small focus of
infection, such as a dissecting wound, there are usually
no perceptible symptoms until an initial chill announces
the onset of some grave malady. The marked prostra-
tion preceding any chill, or this symptom if no chill
occurs, when present with fever continuing from the
time of reaction, or with a fever commencing after the
third day, is almost pathognomonic of septicemia. When
gradually superseding the non-infective fevers just men-
tioned, marked prostration soon develops with gastro-
intestinal symptoms, such as anorexia, diarrhea, etc., the
fever and general condition rapidly assuming the ty-
phoidal type. The fever is a continued one, but usually
shows slight morning remissions ; later, the temperature-
curve becomes more irregular, the perturbations being
sometimes clearly dependent upon changes in the condi-
tion of the wound, when there is much local septic dis-
turbance. A sudden rise just before death is not very
unusual. Slight hematogenous jaundice is usually ob-
served, the spleen is enlarged and tender, and the lymph-
nodes nearest the lesion, if accessible, will also be found
in the same condition. Septic lymphangitis is a variable
symptom and is dependent largely upon the character of
the wound—*i. e.*, whether the local infection is marked
and the infection be single or mixed. It is an error to
lay too much stress upon the local conditions found in
septicemia, such as sloughing, gangrene, etc., for they
really result from inflammatory strangulation, or from the
locally destructive effects of bacterial alkaloids or proteids,
just as in any other infected wound not causing septice-
mia, and are not directly due to the general toxemia.
Doubtless the resistance of the wounded tissues to the
invasion of germs is seriously impaired by the constitu-
tional poisoning as well as by the development of purely
saprophytic organisms. It has been too readily assumed

from the small extent of the local lesions often discoverable in diphtheria that in surgical toxemias also much of the poison is manufactured in the wound and its immediate surroundings. The diphtheria bacillus like that of tetanus seems capable of developing specific poisons capable in minute doses of producing tremendous effects, but this is not true for the toxins of the pyogenic cocci. It is not correct to state that septicemia is a " progressive invasion of tissues by continuity," because with a most trivial "infection-atrium," with no local lesion capable of producing appreciable amounts of toxins, the most marked constitutional symptoms often develop, and again, marked local infection of the tissues does not necessarily produce septicemia. Still further, the post-mortem findings demonstrate that the toxins are not manufactured in the wound alone but everywhere throughout the economy. Unquestionably, if septicemia attack a patient with a large infected wound, marked local changes spreading far and wide by continuity will almost necessarily occur, especially if saprophytic organisms are present. This, however, is an accidental accompaniment of septicemia, not an essential part of the disease, because some of the most typical instances of septicemia develop in the absence of marked local trouble, proving that the widespread presence and multiplication of the germs in any and every part of the body form the essence of this disease.

As would be expected leukocytosis is marked, and cultures made from the blood will often demonstrate the presence of viable pyogenic organisms.

The heart early shows the depressing effects of the toxins, becoming weak, frequent, and irregular in action. The skin at first hot and dry, later is leaky, or free sweating may alternate with dryness. Different forms of skin-eruptions are sometimes observed, usually tending to be pustular or purpuric, although erythematous conditions often simulate scarlatina, etc. Hoffa teaches that these eruptions, other than erythematous, are due to capillary

thrombosis. He contends that the micro-organisms he has obtained from the skins of patients with such lesions are non-pathogenic, but either mechanically, or through action on the fibrin-forming constituents of the blood stand in a causative relation to the thrombosis. Microbes which are non-pathogenic in health may be pathogenic when the vitality of the individual infected is lowered, and since the blood has an abnormal tendency to coagulate in septic conditions, even non-pathogenic germs might readily serve at least as mechanical starting-points.

The nervous system early shows the action of the poisons by restlessness, delirium succeeded by apathy, stupor, coma and death in this state.

The renal secretion is diminished or suppressed, the urine containing albumin in most instances.

The distaste for food increases, the earlier diarrhea becomes more pronounced, the evacuations become offensive, the icterus deepens, and there are bronchial symptoms, shown by quickened respiration, cough, etc., the fatal ending resulting from exhaustion and collapse in the less acute cases.

Milder cases are observed, where a moderate febrile septic condition may persist for weeks, with nothing very distinctive except a decided enlargement of the spleen. Recovery often occurs in this class of cases, which are unquestionably mild septicemias. Indeed, from the slighter febrile attacks lasting for from seven to ten or more days after operation, formerly called "traumatic" or "septic traumatic" fever, which were either slight sapremias, or sapremias combined with, or followed by, slight septicemias, up to the most malignant septicemia, there are all conceivable grades of severity.

Although, as has been explained, there can be no "characteristic changes in the wound" beyond those seen in an infected one in a patient whose vitality has been seriously impaired, undoubtedly in the more malignant septicemias there will be marked evidences of local infection, shown by edema and congestion of the wound-

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margins and surrounding tissues, watery, ichorous, and offensive discharges, possibly sloughing and gangrene, with lymphangitis and local bacterial infiltration; but all this can be said of many infected wounds which may be followed only by slight sapremia if drainage be free, or by septicemia, or pyemia, according to whether certain local conditions do or do not prevail.

After death, examination shows the blood to be tarry in consistence, with little power of coagulating, and the various internal organs contain cocci, streptococci being perhaps more common than staphylococci, although both may be found. The pia mater shows extravasations, and punctiform hemorrhages may also be seen in the deeper portions of the nerve-centers. Beyond a brownish discoloration of the muscles, these present no changes. In the more chronic cases the endocardium is thickened, but any ulcerative lesions of this membrane are rare in pure septicemia. Slight pleural and pericardial effusions are often found. There are evidences of a distinct gastro-intestinal catarrah, especially marked in the duodenum and rectum by punctiform hemorrhages. The spleen and the lymph-nodes, especially those of the mesentery, are enlarged. The kidneys are congested, and the glomeruli are many of them blocked by germs, evidently brought there for excretion. The liver is often "emphysematous" from early decomposition, indeed the bodies of those dead of septicemia show a marked tendency to early decomposition. Post-mortem examination of these bodies, *when fresh*, are especially dangerous; after putrefaction they become less so, because the saprophytic germs supersede and apparently destroy the infective micro-organisms. In and around the wound numerous bacteria will be found, with infected thrombi, originally merely hemostatic, extending some distance along the course of the vessels. A similar condition exists in the lymph-spaces and vessels. Phlebitis and thrombophlebitis, although vastly more common in wounds giving rise to pyemia, are conditions not incompatible with

septicemia. As has been stated many times, while septi-
cemia most commonly originates from some surface-lesion,
or one which is readily detectable, yet there are many
cases whose origin is most obscure. Warren and other
writers have pointed out that many of these cases start
from infective processes involving the appendix, the
nasal cavities, the tonsils, the urethra, rectum, the teeth,
or the middle ear. I have seen typical septicopyemia
originate from a trivial skin-lesion.

Treatment.—It is imperative to adopt energetic and
effective local disinfection, because thereby any further
sapremic complication can be prevented, and fresh in-
crements of germs also may possibly be prevented from
reaching the circulation from the wound. Certain
forms of wound-infection, which certainly sometimes
destroy life by instituting genuine septicemia, seem to
be controllable, as hospital gangrene, by destruction of
the diseased parts with a zone of the surrounding appar-
ently healthy tissues. The actual cautery, because the
radiated heat is germicidal far beyond the point of
application, and pure bromin—as Goldsmith showed
during the Civil War—from the penetration of its vapor
into the deepest recesses of the wound are the best when
applicable. Pure carbolic and nitric acids are also effic-
ient ; potassium permanganate, one dram to the ounce
of water, may also be employed. At other times the
knife or curette is preferable to any form of caustic, but
this is true only when the infected parts can be removed
entirely, and through healthy tissue. Sometimes none
of these measures can be adopted, when the continuous
warm bath or antiseptic irrigation should be tried, re-
membering the risk of absorption of poisonous anti-
septics. Powdered charcoal mixed with one-third its
bulk of sugar containing 5 per cent. of naphthalin is
extolled by Park as an application in malodorous cases,
and may be tried in default of the possibility or desira-
bility of employing the measures already mentioned.
Increasing the eliminative action of the emunctories to

get rid of chemical and bacterial poisons, sustaining the circulation by large doses of alcohol and appropriate drugs, and maintaining the nutrition by rectal as well as oral feeding, and the restraint of the temperature within safe bounds by the use of cold, have already been sufficiently dwelt upon when describing the treatment of sapremia or will be considered under Pyemia. The diarrhea, when in excess of any useful purpose in eliminating, must be kept within bounds by such intestinal antiseptics as salol, occasional doses of calomel, or mercury and chalk, bismuth, beta-naphthol, etc. Intestinal antiseptics also lessen the chances of enterosepsis. When the action of the bowels is irregular and inadequate, the occasional use of small doses of calomel and soda, followed by a laxative is indicated, mechanically removing much poison : indeed, purgation is sometimes indicated, especially in peritonitis, as the most efficient method of evacuating germ-products. If the original focus or any suppurative lesion shows a pure streptococcus infection, or if this is reasonably probable because of the extensive lymphatic involvement,[1] the hypodermatic use of Marmorek's antistreptococcus serum will often completely revolutionize the course of the disease. If staphylococci are the sole or the predominating organisms, but little if anything can be expected from the use of the serum, which it must be remembered is not without danger.

[1] Streptococci seem to be peculiarly prone to attack the lymphatic system.

LECTURE XV.

PYEMIA.

PYEMIA is a misnomer, the liquefied thrombi having been mistaken for pus-collections occupying the lumen of the vessels. Pyemia is a disease resulting from the lodgement of septic or infective emboli, which are in turn the result of septic or infective thrombophlebitis; possibly it sometimes originates from septic or infective lymph-emboli, due to septic or infective lymphangitis; in rare instances infective emboli have been found in the capillaries which have originated in an infective thrombo-arteritis. Although the presence of pus in the blood is not the cause of pyemia, yet now and then the rupture into a large vein of a quantity of infected pus, or the introduction of the same through the lymphatics, may give rise to infective embolism and hence to pyemia. Let there be no mistake, however; pyemia thus produced is not due to pus—either cells or liquor puris— but, just as in the case of an embolus of blood-origin, it is due to the contained micro-organisms, which, entangled in masses of pus-cells, form emboli exactly as cinnabar, fragments of sterilized pith, or potato will secure the retention of the germs where these masses lodge, damaging the endothelium and rendering implantation of the germs a certainty. Thus a postperitoneal abscess has caused pyemia by rupturing into the ascending vena cava, as Schuh has reported. The many ways in which infective emboli can originate require study. The hemostatic thrombi formed in every wound may either become soaked with the ptomains of decomposition, or with bacillary products, or pyogenic cocci may infiltrate them. The same remarks apply to

lymph-thrombi. Lodgement of germs in the arteries will set up coagulation-necrosis of the vessel-walls and thrombosis followed by suppuration, just exactly as if an infected embolus had been arrested. Although mycotic endocarditis may be secondary to septicemia and pyemia, it may originate the latter disease ; emboli composed of fibrin loaded with germs, or of practically nothing but masses of micro-organisms being swept away from the heart-valves, lodge in the arteries and set up thrombo-arteritis with coagulation-necrosis and suppuration in the surrounding tissues. The microbes causing the endocarditis enter the circulation at some distant infection-atrium and accumulate upon a previously roughened valve, or themselves so damage the endothelium as to gain a permanent lodgement and produce a local thrombosis. The microbes causative of endocarditis, or of infection of the thrombi elsewhere formed which by their disintegration give rise to infective emboli, may indeed come from a wound ; but sometimes an unsuspected appendicitis, dysenteric ulcer, gonorrhea, nasal catarrh, an ulcerated tooth, slight pyosalpinx, osteo-myelitis, or the mere abrasion from the adhesive plaster employed for extension after fracture of the thigh may be the real cause of the miscalled ''spontaneous pyemia.''

Pyemia has even first shown itself after the wound giving rise to it has healed, as I have seen.

A definition of pyemia often accepted is incorrect—viz., ''pyemia may be described as septicemia plus thrombotic and embolic accidents which lead to distribution of infectious material to all parts of the body,'' unless this statement can be shown to be true of all varieties of pyemia, which it cannot. Thus, as is well-known, the disease may originate from a trivial lesion, from a focus in which neither alkaloids nor toxins can be developed in sufficient amount to produce any systemic intoxication, which is conspicuously absent until thrombo-phlebitis occurs or saturation with and softening of a

hemostatic thrombus by chemical poisons takes place, when infective or septic embolism occurring—as it may and often does—then, as every surgeon knows, systemic poisoning promptly shows itself. Instances of this have been already given and could be multiplied indefinitely. Unquestionably many cases of pyemia are preceded by a mild septicemia, the majority of cases observed in practice being really instances of "septicopyemia"; but this condition differs materially from the one pictured to us by the definition, which incorrectly states the causation and pathology. Bearing in mind the statements made concerning metastasis in Lecture XII. it is easy to realize how one infected thrombus may provide numerous emboli, which, carried to the lungs, will establish secondary foci of infective disease, each of which in turn may supply dozens of emboli. In the infective form of pyemia, germs are present in the blood, and they can usually be cultivated from this as well as from the other fluids of the body; but where the disease originates from the distribution of emboli, free from germs though *soaked* with *ptomains* and *toxins*, diligent search fails to detect any.

Fatigue and starvation, or an insufficient supply of food, are usually believed to predispose to pyemia, but this is no more than stating that all mycotic processes are favored by anything causing general lowering of the vital powers; hence, deprivation of food plus overloading of the blood with the retrograde products resulting from excessive tissue-waste is a peculiarly prejudicial combination. Children rarely suffer from pyemia, because in them elimination and assimilation are apt to be more perfect than in those of more advanced age.

Open wounds of the medulla of long bones, of the diploë of the flat bones of the skull, because large hemostatic thrombi must form in their relatively non-collapsible veins; infections of a puerperal uterus for similar reasons; infective inflammations, such as carbuncle situated on the lips or neck, are especially prone to be

followed by pyemia, as well as many of the so-called phlegmonous inflammations of the cellular tissue.

Symptoms.—The germs require quite as long a time as those producing septicemia to multiply into numbers sufficient to infect the hemostatic thrombi in a wound, or to cause thrombophlebitis ; but, in addition, more time must elapse before they can effect such secondary soften-ing of the thrombi as will permit fragments to be washed away by the blood- or lymph-stream ; hence the symp-toms of pyemia rarely appear before the end of ten days after a wound or operation, although precedent sapremia or septicemia may have been in evidence all along. From what has been already said it will also be clear that pyemia may appear much later, indeed at any time when the local conditions are favorable. There are probably two distinct forms of pyemia, although clinically they can be only inferentially distinguished by the relative mildness of the local manifestations and the greater fre-quency of recovery. In one, the' thrombi are plainly only soaked with ptomains or bacterial toxins, hence, when the locally destructive effects of these substances are exhausted, unless a genuine infection occurs second-arily, or a fatal *septic*—mark, not *infective*—embolism takes origin from the primary embolic centers, recovery may readily ensue, if the necrotic tissues can be success-fully evacuated. In the other, the infective form, the emboli contain infective micro-organisms as well as their poisonous products ; hence, wherever these emboli are arrested, their germs multiply, extending the destruction of tissue continuously, and almost certainly become new centers for the distribution of infective emboli. This is not a nice distinction, but upon the possibility of the occurrence of the first variety depends a more hopeful prognosis, which encourages the surgeon to adopt active measures. Typical cases of pyemia differ from sapremia and septicemia chiefly by the repeated chills followed by abrupt rises of temperature, which are in turn succeeded by profuse sweating, the temperature

rapidly approximating but rarely reaching the normal point. Occasionally even in acute cases the temperature may remain normal for twenty-four hours, possibly longer, but this is very rare. Sometimes no chill precedes the first abrupt temperature rise and sweat, but the rule is that the onset of the disease is marked by a rigor. It is also rare to have more than one chill a day, yet this is possible. The temperature-chart of a case of pyemia indicates an irregular remittent type. When combined with septicemia the irregularities of the temperature-curve are still greater, fluctuating from hour to hour, even in the intervals between the chills. The temperature is lowest toward the close of the colliquative sweating. Although it is generally believed that each recurrence of chills results from the lodgement of fresh emboli, this is not invariably the case, while one primary embolic focus has been known to give rise to paroxysms of chill, fever, and sweat, recurring with such regularity as to simulate intermittent fever closely, yet recovery ensued without any evidence of other embolic foci.[1] Later in the disease sweats occur independently of chills, but preceded by accesses of temperature. When the emboli are sufficiently disseminated throughout the lung and produce distinct edema, or the secondary changes set up by them become pronounced, dyspnea and cough are noticeable, with or without sputum, the latter possibly discolored or even bloody. Small emboli deeply situated, unless very numerous, may give rise to no very definite pulmonary symptoms, but if lodged near the pleural surface they will give rise to pleuritic pain; auscultation will then usually reveal a friction-sound or effusion, and evidences of basal pneumonia involving one or both lungs may likewise be detected. Too much stress has been laid upon the "hay-like" odor of the breath, described by Braidwood. Tenderness on pressure over the liver or in the epigastrium, soon followed by icterus and pain on deep inspiration, next show that embolism of the liver

[1] Personal experience.

has occurred. Embolism of the liver is usually the first symptom of visceral involvement where the infective thrombus has originated in one of the tributaries of the portal vein, but conversely is a late symptom when the primary source of the emboli is located in a systemic vessel. Enlargement of the liver can usually be detected by the time liver-tenderness is unquestionable, and may even be present without this. Pain on deep inspiration will be detected only when the emboli are near the peritoneal covering, or an abscess is extending toward the hepatic surface. Friction-sounds produced by the lymph-roughened surfaces can sometimes be heard on auscultation. Small multiple abscesses are the rule in the liver and in the lungs, but in the former occasionally large collections are found resulting from the confluence of several smaller ones lying close together. Owing to the fact that the embolic processes are often confined to small portions of a few organs, the amount of poisons present in the blood at any one time is not so great as in septicemia, where the germs are more widely diffused, hence the nervous symptoms such as delirium, etc., are absent during the early stages of pyemia, unless it is complicated by septicemia. Of course, at any period embolism of the central nervous system may occur, giving rise to symptoms indicative of the part involved. Late in the disease delirium and coma occur. Erythematous, papular, pustular, and purpuric skin-lesions are occasionally seen in pyemia. With or without any or all of the visceral lesions, a sudden implication of one or more joints, or the tendon-sheaths around a large articulation sometimes occurs. Other joints become involved, those first attacked in a few days improving to relapse, or possibly to go on to permanent recovery. The pain is slight if the parts are kept at rest, but is intense if the joint be moved. There is often neither fluctuation nor redness in the early stages. Owing to edema of the soft parts over the joint, the swelling does not assume the characteristic form the joint would show if only the synovial

membrane was distended, but is rounded and ill-defined. The improvement seen in most of the joints after the first few days results from the manner of the deposition of the germs. Small embolic masses of micro-organisms are found in the slighter cases lodged in the synovial fringes and the tissues surrounding the articulation. The tissues gain the victory, nothing but a serous effusion being found in the joint-cavity, or at worst one containing many phagocytic cells loaded with microbes, giving the appearance of seropus. The tissues having won the victory, the serous fluid is absorbed, and the phagocytic cells also disintegrate and are absorbed, or they migrate into the lymphatics. A similar explanation holds good for the painful indurations so often seen in the cellular tissue, so many of which are evanescent. The one or more joints which do not improve gradually present the ordinary appearances and symptoms of articulations being destroyed by suppuration, the cartilages becoming eroded and the bones carious. Emaciation is rapid and marked, in this respect decidedly differing from septicemia. Gastro-intestinal disturbances are not marked in pure pyemia, although, later, diarrhea may set in. The tongue is heavily coated at the tip and sides, brown and dry in the middle, and later covered with sordes, as are the teeth, lips, etc. There is a general hyperesthesia. The spleen is not enlarged unless the subject of metastases, presenting in this respect a marked contrast to the almost constant enlargement of this organ observed in septicemia. The pulse is at first fairly strong, although it is quite rapid, later it becomes very feeble, and subsultus tendinum is common. The blood shows a marked leukocytosis with diminution of the number of red cells, while germs can usually be detected, except in the purely septic, non-infective variety. The urine is scanty, high colored, and contains both albumin, peptone, and germs. Prostration now becomes marked in these latter stages of pyemia, presenting in this respect a marked contrast to the *early* exhaustion characteristic of septicemia. The

duration of pyemia varies according to whether it is acute or chronic, cases of the former rarely lasting more than ten to fifteen days, instances of the latter continuing for many weeks or months before either death or recovery takes place. If there be a wound it will cease to give exit to normal pus, the discharge becoming watery, and if sloughing or gangrene be present, it will be free and malodorous, while any granulations which may have formed will become dry, glazed and will either melt or slough away. It is astonishing to see how rapidly long amputation-flaps will disappear, leaving the bones protruding. The surrounding soft parts are swollen, reddened, and edematous. Evidences of thrombosis of the veins and inflammation of the lymphatics leading from the wound are often detectable.

On post-mortem examination, uncomplicated pyemia shows metastatic processes, infarctions, and suppurations in the viscera, joints, parotid gland, and cellular tissue, in one or all, in addition to the conditions usually found in septicemia, except that the spleen is not enlarged, unless it contains emboli. In septicopyemia the spleen is, of course, enlarged as it is in pure septicemia. Germs are to be found in the infective form, which is the only one the surgeon usually has to examine post mortem.

Prognosis.—Acute cases of the infective variety are practically always fatal, because the vital organs contain numerous emboli, and even if the systemic intoxication does not prove fatal, the destructive effects of the consequent suppuration in organs essential to life will cause death. Where the brunt falls chiefly or entirely upon the joints and cellular tissue, recovery is not very infrequent. Some patients in whom sapremia exists, if this can be relieved, provided the viscera are not seriously involved, and especially if the emboli are entirely germ-free—only *septic*, not *infective*—may possibly be saved. In a few cases the pus resulting from the lodgement of one or more emboli in an accessible organ or an external part may be successfully evacuated, the pros-

pect being more promising if the source of the sapremia can be removed, as by amputation of a limb. While the outlook is always gloomy for any case of pyemia, it is a great mistake to consider the result a foregone conclusion and to abandon all serious effort, as is too often done. Cases of chronic pyemia, where the viscera usually escape, recover more frequently than do those attacked by the acute disease.

Treatment.—Remove the primary source of the emboli when possible, but if this cannot be done, disinfect the thrombotic focus. All blood-flow through the thrombosed vein beyond the obstruction should, when feasible, be checked by ligature of the vein. Macewen freely opens the thrombosed lateral sinus, for instance— in cases of middle-ear disease where the infection has spread to the membranes of the brain—scrapes away the clot, packs it full of boric acid and iodoform powder, and plugs with gauze, also tying the jugular vein upon the affected side when considered advisable, low down in the neck. Aspiration or incision of liver-abscesses, opening and draining of joint, parotid, and other metastatic abscesses, the continuous hot bath for appropriately located lesions, and the other measures advocated for the treatment of septicemia are to be adopted if possible. For the medicinal and dietetic treatment, what was said under the same head must suffice.

LECTURE XVI.

TOXEMIC CONDITIONS PRODUCED BY DRUGS; DIF-FERENTIAL DIAGNOSIS BETWEEN SAPREMIA, SEPTICEMIA, AND PYEMIA; AUTO-INTOXICATION.

BEFORE considering the differential diagnosis between the various infective and septic diseases and the more innocent wound-complications, certain preliminary information is requisite.

The local or constitutional **effects of certain drugs** may give rise to errors in diagnosis. Thus, iodoform, carbolic acid, and corrosive sublimate sometimes produce a most marked and often puzzling erythema and even dermatitis. The internal use of the iodids, quinin, antipyrin, copaiba, etc., may give rise, some to papular, others to urticarious skin-eruptions; more rarely, still different drugs give rise to pustular, bullous, purpuric, or nodular skin-lesions. Eruptions caused by the local action of any drug are either strictly limited to the area to which they have been applied, or spread but a short distance beyond, while those resulting from the internal use of drugs are seen in parts far distant from any traumatism. The erythema and edema, suspected to be due to the local effects of iodoform, may be quickly proved to have this origin by the application of iodoform to some distant part, closely confining it by the outer dressings, when if the suspicion be well founded, the healthy skin will soon present the same appearance as that around the wound. Although, in a general way, in any given case of suspected sepsis, it can be affirmed that the appearance of skin-lesions which are pustular or hemorrhagic is confirmatory evidence that ''derma-

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titis medicamentosa" does not exist, this cannot be relied upon if one or more of the drugs mentioned below have been exhibited. If, on the other hand, no such drugs have been administered, and pustular and hemorrhagic eruptions do appear in a case of suspected sepsis, then they may be considered as almost pathognomonic. Although the prolonged use of drugs is usually necessary for the production of pustular or hemorrhagic skin-lesions, this is not always the case, a single dose of 2.5 grains of potassium iodid having produced a fatal purpuric eruption in an infant. These purpuric eruptions produced by iodin usually appear soon after the first administration of the drug and cease when it is withdrawn, but are often reproduced by even minute doses. Again, the toxins produced by some microbes give rise to scarlatiniform eruptions which may readily be confounded with drug-erythemas or with genuine scarlatina. Scarlatina was recognized many years ago as apt to appear in young patients who, previously exposed, showed no evidences of succumbing to the ailment until their vital resistance was lowered by a severe operation or injury. The discrimination of "dermatitis medicamentosa" from septic or scarlatinous eruptions depends upon the exaggeration of the features of the disease simulated, the absence of the throat-lesions of scarlet fever, the head- and backache with the fever of small-pox, etc., and the effect of withdrawal of all internal and external drug medication. The stress laid upon the fact that skin-eruptions not uncommonly follow the use of copaiba, quinin, iodin, and bromin seems to have diverted attention from the rarer, but possible, skin-lesions caused by the administration of such common drugs as chloral, opium, and mercury. Skin-eruptions have followed the exhibition of arsenic, antipyrin, belladonna, atropin, bitter almonds, bromin, borax, cannabis indica, carbolic acid, chloral, copaiba, creasote, cubebs, digitalis, duboisin, hyoscyamus, iodin, iodoform, mercury, opium, pilocarpin, phosphoric acid, petroleum,

quinin, resin, salicylic acid, santonin, tar, and turpentine. Of course, these eruptions are nearly always the result of idiosyncrasy. The toxemia produced by the absorption of either iodoform or carbolic acid is liable to lead astray the inexperienced, who may continue the local use, or increase the amount of these drugs, hoping thereby to diminish the sepsis apparently indicated by symptoms which in reality are produced by the drugs themselves. Hence, in all cases where instead of sepsis the poisonous effects of these drugs might explain the symptoms, chemical evidence of the presence of carbolic acid or of iodin should be sought for in the urine, even if the use of these remedies has been suspended. It is therefore of the highest importance to be acquainted with the toxic symptoms resulting from the absorption of these drugs.

Iodoform poisoning may be preceded for a day or more by general malaise, succeeded in the worst cases by somnolence deepening into coma, with contracted, immobile pupils. In slighter cases marked restlessness is first noticed, which soon develops into active delirium, the temperature in these two form of poisoning being always normal and the pulse very rapid. In the more severe cases removal of the dressings fails to avert a fatal termination ; indeed, sometimes the evidences of poisoning have not been detected until after the use of iodoform has been suspended. Schede describes five other types of iodoform poisoning from absorption, besides those already mentioned—viz., '' 1. High fever, without other phenomena. 2. Fever, with mild gastro-intestinal irritation, depression of spirits, and rapid pulse ; recovery almost invariable. 3. Very rapid, soft pulse, 150–180, no fever ; great danger. 4. Very rapid pulse, with high fever ; death almost invariable. 5. After severe operations, rapid collapse and death. A form of poisoning with melancholia, dilated pupils, and hallucinations is also described ;'' recovery from this is slow. A dark-red, roseola-like condition of the skin has been not

uncommon in some cases. Iodin in large amounts is always to be found in the urine.

Two forms of **carbolic-acid poisoning** are said to be recognizable. One, chronic, is evidenced by headache, anorexia, bronchial irritation, severe pain in the renal regions, cutaneous pruritus, and "various paresthesiæ, and loss of power in the legs." The acute forms show dizziness, delirium, and unconsciousness. The urine is of an olive-green or blackish hue, this usually being detected only after exposure to the air. One of the earliest symptoms of poisoning is declared to be the disappearance of the sulphates from the urine. Proper chemical manipulations will show the presence of large amounts of carbolic acid in the urine. After two hours' exposure to the carbolic-acid spray formerly employed during operations, one surgeon reports that he recovered 30 grains of the acid from his urine.

Differential Diagnosis.—While freely admitting that many cases commence as sapremia, then are succeeded by septicemia, and later perhaps have engrafted upon them pyemia, so that the condition present in any given instance is often a most complex one, unquestionably there are cases where each ailment pursues its course uncomplicated by any other. It is theoretically possible to distinguish between them, and while their therapeusis is practically identical, the prospects of success depend largely upon which of the three diseases is the sole one, or the chief one to be dealt with.

Sapremia follows large recent wounds or incision of a large abscess ; the discharges are offensive ; there is early chill or profound collapse ; prostration occurs early, and recovery follows when the cause is removed and disinfection secured. The symptoms may recur if the conditions productive of sapremia are reproduced, to recede again when they are removed.

Septicemia occurs later. It may result from an insignificant wound, such as a needle-prick ; it is often engrafted upon a previous wound-fever ; is an irregular

remittent fever. It promptly produces exhaustion out of all proportion to the amount of the fever, etc. The spleen is enlarged. Chill, if present, is early and is not repeated. Metastatic processes are not present.

Pyemia.—Suppuration is a necessary predecessor and usual accompaniment of pyemia, although it sometimes occurs with very small lesions. The necessary formation of infected thrombi and their softening takes time, hence uncomplicated pyemia commences after the first week or ten days, sometimes much later. Chills, repeated at irregular intervals, followed by a sharp rise of temperature and profuse sweating, always occur. Evidences of visceral or articular metastatic processes are detectable ; the spleen is not distinctly enlarged except when the seat of embolic processes.

Although many other minor differences are distinguishable, they need no repetition, having already been mentioned when describing the symptoms of the three affections.

Auto-intoxication.—Much has been said concerning the chemical poisons which result from bacterial activity in dead or dying tissues and even living tissues ; but are these the only sources of the toxemias observed after accident or operation ? Nearly thirty years ago I had my first clinical lesson, which unmistakably taught me that my predecessors in the art of healing during many centuries were right in regarding attention to the condition of the "primæ viæ," as they quaintly termed the skin, intestines, and kidneys, as of equal importance with diagnostic and operative skill in surgery—nay, as of more importance, because both these might be frustrated by failing to get rid of all "peccant materials" both before and after operation. The scientific explanation and enforcement of the clinical fact so often insisted upon in the past have but recently been given. This fact of the possibility of auto-intoxication has been too often ridiculed until recently, because the old explanation was not in accord with modern teaching, therefore

(strangely enough) the *fact* could not be admitted. Thanks to Bouchard's forcible teaching we now recognize that when we have perfectly appreciated the rôle played by germs in the causation of disease, we are still far from comprehending all about pathogenic processes, even those in which germs are the real agents. Perhaps more important still, we have learned that oftentimes no germs could develop, or if multiplying unaided they could not cause death, and that even in their absence from the blood and tissues death may occur from auto-intoxication. Bouchard with a few of his predecessors and followers have reconciled modern science and ancient clinical observation.

A few words must be devoted to a recapitulation of some facts which are familiar, but are commonly overlooked. Much of what follows will be found in the pages of Bouchard, Vaughan and Novy, and in text-books of physiology. Various alkaloidal substances are normally found in the tissues—the result of metabolism and retrograde metamorphosis. These are believed by some to be formed as the result of the growth of germs in the intestines, whence they are absorbed, being either eliminated or retained ("stored up") in the tissues. Some of them are the predecessors, perhaps necessary steps in the formation of urea, for instance. Thus, adenin and guanin from vital or putrefactive changes give rise to ammonia, and this in turn serves to form urea. All leukomains are probably poisonous when in sufficient amount. The true leukomains probably result only from the metabolism of the cells and protoplasm.

To what end have these statements been made? To impress the fact that substances elaborated by the cells are injurious to these same cells, "if the products of their activity accumulate about them." To give a few illustrations of the poisons produced, the hydrocyanic molecule is a frequent constituent of leukomains. The peptones and albumoses of digestion, which are actively poisonous, should not reach the circulation, yet there are

evidences that these do, or at least are not retained in an inert form by the leukocytes, as maintained by Hoff-meister, peptonuria and albumosuria evidencing this possibility. Unquestionably all the phenomena of fever do result from the overformation of poisons from pro-longed mental or physical exertion, or from both, pro-ducing chilliness, a rise of 1° to 3° F., anorexia, wakeful-ness, an increased frequency of pulse. All this is explained by the conception that the presence in the blood of an excess of leukomains resulting from retro-grade tissue-changes causes disintegration of the leuko-cytes and a setting free of fibrin-ferment and nucleinic acid. "Exhaustion fever," as it is called, is a more severe form of "fatigue fever;" and has actually been mistaken for typhoid fever; but rest and proper food, the lack of which caused it, will soon clear up the diagnosis. This form of fever is not uncommon among young troops during an active campaign when sufficient food is rarely obtainable, and the men are unaccustomed to the severe, prolonged exertion of marching, trenching, etc.

Fever from retention of excreta is more common than either of the preceding forms, especially in those con-fined to bed who have previously lived an active life. Purgation will commonly relieve this fever, because dehydration of the tissues will enable the fluids to take up the poisons which in turn, reaching the blood, are eliminated into the intestines or pass off by the kidneys. The elimination of these substances is also advantageous because it permits the liver to perform its own functions properly instead of being compelled to attempt the transformation of poisonous into inert substances, and their excretion with the bile. For instance, pepsin or trypsin absorbed unchanged are very poisonous. These the liver arrests and transforms. It will be profitable to consider still further "the dangers threatened by the imperfect performance of the functions of the kidneys, liver, intestines, and skin, by which both poisons nor-mal to the economy and also those of extraneous origin

are evacuated.'' Only the salient points need mention, because further information can be secured by consultation of any physiological text-book, and the works of Bouchard, and Vaughan and Novy.

If time permitted, there are many interesting questions upon which I should like to dwell, but I can mention only those which distinctly bear upon symptomatology and treatment. As has been said, ''man escapes intoxication by the intestinal, cutaneous, pulmonary, and renal emunctories,'' as well as by the arrest and destruction of poisons by the liver.[1] Although some of the poisons eliminated by these organs are not preformed, some are, and each has precedent substances, out of which they are formed, that are injurious if not transformed and eliminated. The importance of the renal secretion as a means of elimination of poisons has always been appreciated, and we have in modern times learned that it contains convulsive, pyrogenous, and narcotic substances, even in health. When possible, in all surgical cases, the quantity of urine must be ascertained, and to what substances its specific gravity is due. Urea, too commonly regarded as a deadly poison, is far from being this, and is, while injurious if accumulated in enormous quantity, a most useful agent whereby the depurative action of the kidneys is increased. It would take sixteen days for a man to form enough urea to kill him, while in the presence of anuria, so-called uremic accidents appear within the first three days. Urea is eliminated fifty times as rapidly as water by the kidneys, being the most powerful diuretic known, and thus these organs are enabled to carry off in solution large quantities of other more poisonous materials, for instance the coloring matters, which are infinitely more toxic than urea. The evidences of one or more defective links in the chain of disassimilation as shown by the deficiency of certain normal urinary ingredients and their replacement by oxalates, lactates, etc., although the specific

[1] Bouchard.

gravity may be normal, should warn the surgeon of possible cellular malnutrition, which may render efficient an otherwise inadequate number of germs, accidentally present in the economy ; or, in the absence of germs, auto-infection may be imminent.

LECTURE XVII.

AUTO-INTOXICATION (CONTINUED).

THE kidneys may be ready to do their whole duty, while the liver is unable to entirely convert natural, much less excessive amounts of retrograde materials into urea and normal biliary excreta. Thus, while an excess of uric acid probably results from "a metabolism slightly diverging from that of urea," it is most likely that the "turn to uric acid rather than to urea is given in the liver," although the spleen may play some part. Again, substances which should be converted into normal constituents of the bile or urine remain unchanged, or the bile is reabsorbed, producing poisoning. The coloring matters contained in the bile are ten times more poisonous than the biliary salts usually credited with so much toxicity. A man secretes enough bile in twenty-four hours to kill three men of the same weight as himself. The poisons of the bile are known to be six times more toxic than those of the urine and lead to destruction of the red-cells of the blood and those of other tissues, notably the hepatic cells themselves. There are also many poisonous materials absorbed from the intestines which may fail in the liver to undergo conversion into innocent ones. That such changes do take place in the liver has been proven by the experimentally determined greater toxicity of the blood of the portal vein as compared with that of the hepatic vein. Still further, germs normally present in the intestines which aid digestion produce poisons that should be almost entirely excreted by the feces, but which may be absorbed in dangerous amounts under favoring circum-

stances, when present in excess. The steady diminution in the amounts of solids excreted in the urine is a serious menace in any surgical condition, but is peculiarly so when both the liver and kidney are crippled. As the author so often quoted insists, when the urine has lost the toxicity due to substances either absorbed from the intestines, elaborated by the liver, or excreted by the tissues into the blood, then systemic poisoning is imminent. The sources of this toxemia are disassimilation, the substances which should form the solids of the urine and bile, the food, and the products of intestinal putrefaction. Small amounts of the intestinal poisons are constantly being absorbed, but are as constantly excreted by the urine, no harm thus resulting. Any excessive bacterial action which would lead to the formation of dangerous amounts of toxins is probably restrained in health by the hydrochloric acid of the gastric juice. In many, although not all, cases of fever, this acid is reduced in amount, and large quantities of indol, phenol, and cresol are formed, as shown by the increase of such substances in the urine as the "conjugate sulphates." It is quite possible that leucin is formed in the presence of certain germs from peptones, which (leucin), if absorbed in large amount, it will embarrass the liver to dispose of by conversion into urea, thus interfering with the occurrence of the proper metabolism of this organ. The amount of indol detectable in the urine is regarded by many as an index of the amount of intestinal putrefaction and absorption. It is asserted that in simple constipation no increase of the indican can be detected, while intestinal obstruction shows the opposite condition. Although the skin does not normally eliminate nearly as much excrementitious material as the other emunctories, yet it does enough, or can be made to do enough in disease to make its assistance worth invoking. While the poisons eliminated by the skin seem to be few in number and small in amount, it is believed that much more of the imperfectly metabolized

substances out of which the toxic substances are formed
are thus got rid of.

Treatment of Auto-intoxication. —What can be
done in the way of prevention and cure of auto-intoxi-
cation? The importance of the avoidance of excretory
strain being put upon damaged or even healthy kidneys,
by compelling them to eliminate autopoisons in addition to
bacterial ones, must be apparent to all, because congestion
may be thereby induced and functional activity be dimin-
ished or arrested. The question of general anesthesia,
prolongation of the anesthesia, and the anesthetic se-
lected should be influenced by such considerations,
especially if examinations of the urine reveal lessened
solids or solids in abnormal forms. Relieve the liver
from all unnecessary labor by diminution in the quan-
tity and discrimination as to the quality of the food,
which will also lessen the chances of the formation and
absorption of intestinal poisons. Disinfect the feces by
charcoal for instance, which by fixing the coloring mat-
ters and biliary salts reduces the toxicity of the urine
from one-half to two-thirds, the amount of dangerous
substances in this fluid being a fair index of the quanti-
ties of intestinal poisons formed and absorbed. Give
calomel, salol, charcoal with bismuth, bismuth, betanaph-
thol, etc., to so inhibit germ-growth that less toxic matter
will be formed in the intestines. The use of intestinal
antiseptics becomes an imperative duty when the dejecta
are especially offensive, and proper mechanical removal
of the fermenting intestinal contents by laxatives and
enemata are equally demanded. Free purgation by
dehydrating the tissues, etc., may not only permit the
solution and direct removal of leukomains, but also by
their removal favor proper cell-metabolism and elimina-
tion. Ingestion of fluid by the mouth, rectum, or by
hypodermatoclysis, must follow purgation, lest the kid-
neys be not provided with enough fluid to enable them
to dissolve and eliminate the proper amount of solids.
Use judgment in calling upon the skin to remove ex-

creta, lest, as has just been said, the water of the urine being diminished, renal excretion is interfered with ; but when renal congestion exists, causing partial or complete suppression of urine, the revulsion effected by securing free diaphoresis will often relieve the congestion of the kidneys and start the secretion of urine.

In the presence of deficient renal activity or actual anuria, cold enemata introduced into the colon[1] have been successful by contracting the abdominal vessels, thus expelling from the veins of the spleen and those of the chylopoietic viscera a large amount of blood loaded with poisons, while arterial tension is also increased. Dilution of the poisons and increased vascular tension, securing increased renal elimination, may be secured by venous saline transfusion, or by hypodermatoclysis, these measures sometimes being followed by chill and a decided rise in temperature, both being due to the increased amounts of poisons dissolved out of the tissues and suddenly added to the blood.[2]

If time permits, before every operation the old-time practice of securing a normally acting skin by baths, friction, and the ingestion of water ; proper regulation of food, sufficient in amount to nourish, yet not to embarrass the action of the liver ; improvement of the hepatic circulation by diminishing the work of the liver by the restriction of food, and alcohol, and the employment of mechanical cleansing and chemical intestinal antiseptics (calomel combining both of these requirements in a notable degree), must one and all be attended to. Do not always be satisfied with one mechanical cleansing of the bowels, but administer laxatives upon several successive days in all cases where circumstances seem to indicate the necessity of this measure. Determine whether the kidneys are excreting enough solids, and these properly elaborated. If this is not being done, overcome the incompetency by free ingestion of water, and attention to stomachic,

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[1] Bouchard.

[2] Possibly also to disintegration of blood-cells setting free nucleinic acid.

intestinal, and hepatic digestion. Should it appear probable that the function of the kidneys is likely to be inadequately performed because of lowered vascular tone, employ means to improve the force of the circulation. Insist upon fresh air and sunlight when possible, both before and after operation, securing this by free ventilation and by placing the bed where the sun can fall upon the patient for a few hours each day, except during the height of summer. Abaki and others have observed the favorable influence of these agents on the presence of albumin, sugar, and lactic acid in the urine.

To sum up for practical purposes—excessive amounts of poisons are produced in the intestinal tract in the relative absence of hydrochloric acid during febrile disturbances and other less well understood conditions, which poisons must pass through the liver. Of these, the greater part in health are arrested and re-excreted with the bile, or modified, the residue passing out by the urine. In disease the following are the results: The overtasked liver cannot perform its own metabolism properly, thus adding to the poisons present in the blood ; the kidneys fail to get rid of all that which passes beyond the liver, partly because of sheer inability, partly because of the diminished formation by the liver of the normal diuretic urea ; and finally, either dangerous systemic auto-intoxication results, or the auto-intoxication removes the restraint exercised upon the development of germs by normally nourished tissues, thus often rendering fatal an amount of mycotic infection which would otherwise have proved trivial in its effects. Such considerations are of special importance when we reflect how often in hepatic surgery jaundice is present, symptomatic of improper liver-metabolism and excretion. Under such circumstances if the kidneys are fully equal to their duty, there is no special danger ; but if they are inadequate, unless the formation of the autopoisons can be checked, death will occur irrespective of mycotic infection. Still

more certainly will this take place if germs are present, although but few in number.

Intestinal sepsis being more often mistaken for sapremia than for anything else, a few words are still requisite concerning auto-intoxication. Nothing specific has been said concerning diagnosis because it has seemed more important to impress the fact of the possible occurrence of conditions outside of the wound causing serious intoxications, believing that with this information and a description of the conditions productive of toxemia this would seldom be permitted to arise, and when present would be eliminated by the measures which would naturally be taken during the treatment of surgical ailments, if the advice given, born of experience, be taken. If the skin, urinary, and intestinal secretions are scanty, and altered as already described, especially if there be no adequate explanation of the systemic condition in the wound, suspect the presence of auto-intoxication, and if the restoration of these secretions to their normal amount and character be followed by a decided change for the better, auto-intoxication may be safely affirmed to have existed. Even in the absence of any apparent deficiency in the alvine and urinary secretion, it is wise to assume that in disease undue amounts of deleterious substances are formed, requiring exceptional freedom of excretion to maintain health, hence an occasional laxative will often be followed by surprisingly large and offensive stools. Free ingestion of water or the exhibition of diuretics will likewise be followed by the passage of large quantities of urine whose specific gravity is not diminished in proportion to the increase in the quantity of fluid, while the systemic intoxication diminishes *pari passu* with the successful maintenance of this excess of secretion.

LECTURE XVIII.

SUPPURATION; ABSCESS; SINUS; FISTULA.

Suppuration.—The subject of acute abscess and its possible consequences, sinus and fistula, must now be considered. True (microbic) inflammation resulting from infection with pyogenic organisms must result in suppuration, microscopic or macroscopic. When this occurs in the tissues, not upon a free surface, it presents general characteristics only slightly modified by the anatomical and physical surroundings of each case. In like manner the general principles applicable to the diagnosis and treatment of acute suppuration require in their application to special cases only the modifications enforced by the anatomical and physical peculiarities of the location. When suppuration is circumscribed, forming a well-defined collection, the environing tissues being rendered more dense and easy to differentiate from the central liquefied portions of the tissue, an "abscess" is said to have formed. When the circumscription is less well defined, the surrounding parts tending to break down into pus, no distinct boundary of normally vitalized and vascularized cells existing, this is often called a "purulent collection." If throughout the tissues innumerable foci of pus exist, varying in size from microscopic collections to large accumulations, with boundaries between the pus and the tissues so ill defined as to defy accurate delimitation, this condition is termed "purulent infiltration." This is clearly a misnomer, the pus not originating from some one or more points and thence permeating the tissue; but the germs, located by embolic processes simultaneously at numerous points or spreading

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through the lymph-spaces to become arrested in greater numbers at certain points, really account for this dissemination. These three conditions are questions of degree merely, depending upon the virulence of the infecting agents and the resistance of the tissues. The central portion of any infected focus undergoes coagulation-necrosis, the intercellular cement and many of the cells are dissolved, others are loaded with bacteria—*i. e.*, they are dead phagocytes—these constituting pus-cells; moreover, the phagocytic action of the leukocytes serves to remove some tissue. The soakage into the tissues of germ-products containing peptonizing agents disassociates the new cellular exudate, mechanically preventing the vascularization and conversion of the cells into tissue while also actively attacking their vitality and rendering them an easy prey for the germs. This process continues to extend peripherally until the virulence of the infection is less potent than the resistance of the tissues, or more generally, until nature or art evacuates the pus; hence one of the dangers of delay in giving exit to pus.

Symptoms.—The inflammatory process becomes more localized, the pain usually assumes a throbbing character, and a chill frequently accompanies the formation of pus if the focus be a considerable one. When superficial, the circumscription of the inflammatory redness and swelling is readily discernible. Palpation soon reveals central softening which in turn passes into "fluctuation," detectable by pressing alternately with a finger of each hand. The softened area becomes more acuminated— *i. e.*, "pointing" takes place. The skin overlying the pus, having its return blood-supply through the subjacent parts destroyed, becomes purplish, thinned, glazed by distending pressure, and finally gives way either by the detachment of a small slough or by ulceration, in either case this being usually preceded by the formation of a "bleb" of epithelium elevated by exuded serum.

When suppuration is deeply seated, edema of the superjacent parts, mottling of the skin from an interference

with the venous return less marked than in superficial abscess, tenderness upon pressure, a localized induration or sense of increased resistance of the deeper parts, later softening of the previously indurated area, and possibly obscure fluctuation, indicate acute abscess, especially if fever with preceding rigors has occurred. If situated in certain localities, from pressure upon important veins, edema of the distal parts is produced *pari passu* with the development and spread of the localized tumor. Pains felt in the distribution of a nerve or many branches of a plexus are often significant when conjoined with local tenderness and constitutional symptoms; this is notably the case in a perinephric abscess. Percussion will often map out a dull mass which palpation may not locate with much certainty. This is, of course, applicable only to regions where normally resonance should be detected, as the loin, abdomen, and thorax. Sweats with irregular temperature are frequently present in deep-seated pyogenic processes; in other words, a variety of sapremia or possibly true septicemia results. In such cases as would entail operative procedures to reach a supposed focus of deeply-seated pus, when there is such reasonable doubt as cannot otherwise be resolved, unless the anatomical relations forbid, the use of the exploring or aspirating needle should be employed. This must never be done if it be a question of an intra-abdominal abscess, careful incision being not only a much safer, but more certain procedure, because coils of thickened intestine, matted together with lymph, may readily be punctured by the needle, while the pus-cavity may not be struck or the fluid may be too thick to flow through the instrument. Again, if pus be found by the needle, immediate incision should follow lest the exploratory track become infected.

In many localities nothing but good can follow a properly executed incision into an infected inflammatory focus, even if no pus be found, but this cannot be averred of the abdomen, liver, pleura, etc.; hence the qualified

advice given above as to the use of the needle for the liver, pleura, kidney, or deeper structures of the limbs and trunk. The time element and the absence of systemic symptoms should exclude any probability of error arising from cysts, malignant tumors, or aneurysms, the differential diagnosis between such conditions and abscess being rarely, if ever, requisite in acute suppurations. When a chronic or ''cold abscess'' is concerned, the reverse is true, and under this head the differential diagnosis will be mentioned.

Diagnosis.—To recapitulate, in a supposed superficial abscess, circumscription of induration, softening, then fluctuation, the portion of the inflamed area showing the last two signs becoming more acuminated—*i. e.*, pointing—preceded by fever and often chill, indicate pus-formation. In deep-seated abscess, the same or more severe constitutional symptoms, edema, and venous or nerve pressure-symptoms, possibly evidences of compression of the esophagus, trachea, rectum, or urethra, when all of short duration and accompanied by a rapid increase in area of the induration, tumor, or area of dulness upon percussion, with lessened resistance over the swelling, still more fluctuation, are reliable signs of deep-seated pus. Of course, in both superficial and deep abscesses, all these signs may not be present in every case, but there are enough detectable to admit of a correct diagnosis being made.

Treatment.—Until rupture is imminent, heat and moisture will both hasten and limit the suppurative process, as has already been explained. When it is possible that pus may evacuate itself before the surgeon's knife is permitted to do so, antiseptic poultices must alone be used. Whenever feasible, pus must be evacuated at the earliest possible moment by a free incision made at the most dependent point when this is anatomically safe. This injunction applies to pus wherever situated, in brain, bone, or the skin. Tension and accumulation of pus must be obviated by counter-openings

if necessary, by drainage-tubes, compresses, and sometimes by bandaging. The incision should be free enough to permit spontaneous evacuation of the pus, doing away with any necessity for pressure which might damage the protective barrier of granulations and thus spread the infection. For similar reasons, irrigation is often harmful ; but if employed, free exit must be provided for the fluid, lest overdistention force infective materials into the surrounding tissues. Pus is always destructive, travelling in the direction of least resistance, hence the danger of leaving accumulations of pus unopened when located near the peritoneum, a joint or tendon-sheath. Beneath dense fasciæ, pent-up pus not only gives rise to intense pain but also may exercise dangerous or occlusive pressure upon a canal, such as the urethra, trachea, etc., while in other regions it may burrow extensively, producing widespread destruction of tissues. The scar left by an incision through intact skin is always less unsightly than that following spontaneous evacuation by ulceration.

Where a free incision might compromise the integrity of important vessels or nerves, after cutting parallel to their course only through the skin and deep fascia, a grooved director can be bored down into the cavity, pus then passing out by the groove along which a closed pair of dressing forceps can be passed and, being withdrawn opened, a sufficiently large orifice will be made, the vessels and nerves being safely pushed aside. This is Hilton's method for opening deep-seated abscesses.

Complications.—Owing to the removal of support, sometimes severe oozing or actual hemorrhage may follow the opening of an abscess, even when the patient is not a "bleeder." It comes from vessels in the wall of, or traversing, the cavity. Later, because of weakening of their walls by ulceration, a large vein or artery may open into the abscess, as the deep jugular vein or carotid artery in cases of abscess following scarlet fever and diphtheria. Sometimes this occurs before these abscesses are

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opened. Any of the complications common to infected wounds are possible, such as pyemia, erysipelas, etc. The method of healing has been already described in a former lecture. Moist antiseptic dressings are indicated so long as pus or much fluid is being formed, after which dry aseptic ones may be employed, although in an abscess of any magnitude moist dressings are usually requisite until the cavity has been effaced.

Sinus and Fistula.—A sinus or fistula is a consequence of the failure to heal of an abscess or wound opening into some canal, a more or less tortuous, narrow channel being left, lined with avascular granulations secreting thin pus, and opening into the midst of unhealthy and usually exuberant granulations. A "sinus" technically is a suppurating tract open at one end, while the term "fistula" strictly interpreted indicates a similar condition which is open at both ends. Either condition results from the persistence of a source of irritation and infection, as dead bone, a foreign body, the constant escape of the contents of a canal, hollow viscus, or gland, as the bowel, gall-bladder, urinary bladder, or a salivary duct. An unhealed wound of a salivary duct, intestine, etc., will give rise to a fistula, as will also sloughing—for instance, the destruction of the vesico-vaginal or vesicorectal septum by this process. Congenital failure to close on the part of an entire branchial cleft will give rise to a "congenital" or "branchial fistula." These will not be specifically considered here.

Causes.—Obliteration of an abscess-cavity or closure of a wound of a hollow viscus being effected by fusion, organization, and contraction of the granulations, if the fusion is mechanically prevented, the deeper portions will form scar-tissue, which by its contraction gradually obliterates the blood-supply to the more superficial layers of cells, rendering them incapable of fusion and definitive healing, even if their surfaces were not mechanically kept apart by wound or other secretions.

Treatment.—Healing of a sinus is prevented then by

two differing conditions, one vital, the other physical.
The first often results from the second, because attempts
at healing having been thwarted for long periods by
mechanical separation and disturbance of the parts,
organization in the deeper parts has gone on until the
blood-supply of the surface-granulations has been so di-
minished that now removal of mechanical obstacles alone
will be useless, adequate reparative material being ab-
sent.   The mechanical disturbance may result from
muscular action, as the alternate contractions and re-
laxations of the sphincter ani, the movements imparted
to the tissues of the groin during walking.   Imperfect
drainage prevents the maintenance of contact, and sepa-
rates from time to time the sinus-walls.   As discharge
must persist if a foreign body or infection remains present,
the removal of such causes is imperative.

The next question is, Is the blood-supply adequate to
enable the lining granulations to be healthy, fuse, and
develop into scar-tissue?   If this condition exist, prevent
separation of the walls by securing free drainage and
maintain them in contact with compresses, bandages,
and splints, thus securing rest of the sinus and the part
also.   The tube or gauze employed for drainage must be
shortened from day to day, permitting the granulations
to coalesce behind ; these measures will usually succeed
with a recent case where the granulations are vascular.
If it is believed or known that the lining layer of granu-
lations is incapable of securing healing from lack of
vascularity, the use of means to induce hyperemia will
be requisite, in addition to the measures already de-
scribed. Sometimes sufficient hyperemia can be secured
by packing with iodoform gauze, the application of
tincture of iodin, nitrate of silver fused upon a wire, the
galvano- or thermo-cautery, or curettage.   Again, in
certain localities the whole sinus-track with its useless
granulations can be dissected out and the tissues main-
tained in contact by layer sutures.   At other times
counter-openings will secure the necessary rest from

better drainage, while the requisite hyperemia can be induced by some of the agents mentioned. In many cases, laying the sinus open throughout its whole extent fulfils every indication, securing the drainage, rest, and reparative hyperemia. These measures are more especially adapted for the treatment of a sinus, but, in addition, it is usually requisite when dealing with a fistula either to divert entirely any secretion—as urine or bile —from passing into and separating its walls, or render it easier for this to get out of the natural canal than to escape through the fistula.

The complete diversion of any secretion is only possible by a plastic operation, securing permanent coaptation of healthy tissues, rest, and the exclusion of anything which can mechanically separate the tissues or interfere with healing. Plastic operation is practically the only method applicable for the treatment of vesicovaginal, vesicorectal, and many intestinal and other varieties of fistula, resulting from the opening of the hollow viscera by disease or accident. Gradual, and therefore partial diversion of a secretion, for instance urine, from a urethral fistula is usually all that can be done or is necessary. Just so soon as any stricture of the urethra in front of the fistula is sufficiently dilated to render it much easier for the urine to pass out by the meatus than by the fistula, the fistula begins to diminish in size and heals, if the normal caliber of the urethra is maintained. In like manner the removal of the spur of bowel in an intestinal fistula will often secure closure of an artificial anus by permitting the feces to pass along the gut more readily than—being diverted by the obstruction—through the artificial opening.

The foregoing statements embrace all the principles involved in the treatment of any sinus or fistula, requiring, of course, modification to meet the peculiarities of special cases.

LECTURE XIX.

GANGRENE, MOIST AND DRY; RAYNAUD'S DISEASE; SPREADING TRAUMATIC GANGRENE; MALIGNANT EDEMA; PERFORATING ULCER.

Gangrene (*Sphacelus*, *Necrosis*).—Gangrene is not a termination, but a result of inflammation, usually due to accidental conditions which are chiefly physical. Gangrene can occur independently of any inflammation, as in anemic gangrene from the tying or thrombotic occlusion of an artery. Sphacelus, gangrene, and necrosis, often used interchangeably, are also restricted by some to certain phases of death of tissue. *Sphacelus* has been thus employed to designate death of all the tissues of a limb in contradistinction to death of some of the tissues, as gangrene of the skin, partial gangrene of a leg. *Necrosis* is chiefly used when speaking of death of osseous tissue *en masse* as contrasted with molecular death of bone, "caries;" but it should always be qualified by the word bone—*i. e.*, "caries of bone"—because the phrase necrosis is also applied to death of other tissues. There being no rule respecting the use of these terms, the varying significance with which they are employed is mentioned to assist you in your studies.

Gangrene has usually been described as "traumatic" and "spontaneous," yet this is faulty, the essential cause of death in many instances of so-called traumatic gangrene being identical with that in the spontaneous variety—viz., deprivation of arterial blood. Indeed, except when the traumatism physically disintegrates tissues as a stone is reduced to powder, heat or strong acids physically or chemically destroy structure, or cold suspends cellular nutrition so long that when this nutri-

tion becomes a physical possibility, vital metabolism cannot be resumed, gangrene always results from total deprivation of pabulum. Tissues normally poorly vascularized, epecially if of such loose texture as readily to permit accumulation of exudates, as the scrotum, are especially prone to the occurrence of sloughing and gangrene. In whatever manner effected, the cutting off of pabulum to the cells is the determining factor, because even if bacterial poisons help to kill the cells, comparatively few would perish if all were properly nourished. But as gangrene often occurs in the absence of all germs or their products, their direct action in determining gangrene is clearly only of secondary importance. The interference with nutrition may be a gross one, as obstruction of a main artery producing direct tissue-starvation, or the return of venous blood being impossible, arterial blood cannot reach the tissue ; moreover, this overplus of blood so compresses the plasma-channels that cell-nutrition is arrested. Germs causing, by the inflammation excited, the most intense forms of hyperemia and the maximum accumulation of solid and liquid exudate in the tissues, similar interference with cell-nutrition results, so that the tissues perish, partly from starvation, partly from chemical action.

In proof of the statement that interruption of the blood-supply to the cells, or the pabulum thence derived, is the essential cause of gangrene, it is only necessary to point out that when the direct blood-supply of a limb is destroyed, no gangrene results unless the tissues are so tensely filled with exudate that the collateral blood-supply with the access of plasma to the cells is arrested, when gangrene will follow, whether the compressing material be normal blood or the most toxic of germ-products. This is well seen in injuries of limbs where the direct arterial supply is destroyed and the tissues in which the collateral circulation must develop are tensely filled with effused blood. Traumatism sometimes produces gangrene by pure physical de-

struction of tissue, with partial devitalization of a small surrounding area where the effusion of blood and reparative material so adds to the pressure that vascular stasis results. Again, trauma may produce widespread damage to the vitality of the tissues, but not their actual destruction, the effused blood and reparative exudate, as just said, by determining stasis, causing death of the part, which is nearly always hastened by infection. Finally, infected traumatisms produce such free exudation that the consequent severe strangulation, assisted by the chemically destructive action of germ-products, overwhelms the vitality of the part. Injuries of the cerebrospinal axis or the peripheral nerves often so disturb the vasomotor equilibrium that imperfect nutrition of the tissues results, rendering them vulnerable to germs. Absence of pain in such cases also permits prolonged pressure, which mechanically deprives the tissues of their blood-supply ; but, this pressure removed, vasomotor paralysis will be found to have in some measure resulted, often ending in stasis and thrombosis. These facts, with infection of the enfeebled tissues by skin germs, account for the variety of gangrene termed "decubitus."

It is a mistake to think that the terms "moist" and "dry" gangrene indicate a necessary difference of causation. It is true that, because fluids in excess are present during inflammation, when gangrene follows inflammatory strangulation and infection it is always moist. It is equally true that anemic gangrene, due to the gradual diminution and final arrest of blood-supply —the result of diseased arteries and, finally, thrombosis —is, when uncomplicated, a dry gangrene. In the first form, death of tissue results from the inflammatory exudates depriving the cells of pabulum, by compression of the small vessels and plasma-channels ; in the second, starvation occurs because all arterial blood is cut off from the limb. Yet, let accidental inflammation have preceded the final blocking of the artery, or let ligation of the main artery cause gangrene because the collateral

circulation cannot become developed, and if an aneurysmal sac is so disposed as to interfere with a free return of venous blood and lymph, this anemic gangrene will in both instances prove "moist," not "dry."

Thus it is clear that the presence or absence of fluid in the part at the time gangrene commences determines whether it shall be moist or dry, not the cause of the gangrene. Dry gangrene must always be due to arterial anemia, so gradually induced that the venous blood readily escapes from the part. Although it may follow embolism, it is far more commonly the consequence of a gradual diminution in the arterial blood-supply to the tissues, resulting from loss of elasticity of the arteries with a consecutive total, but slowly produced, blocking of the vessel by thrombosis. Because arterial atheroma and calcification are rarely pronounced except in old age, too often "senile gangrene" and "dry gangrene" are used as synonymous terms. "Anemic gangrene" is better than either of these names. The vasomotor spasm of the arterioles and smaller arteries induced by the prolonged ingestion of ergotted rye will produce a dry gangrene exactly like that which is the result of diseased arteries. The fingers, toes, and tips of the nose and ears are most commonly the parts lost by ergotism.

Symptoms of Dry Gangrene.—After a longer or shorter period of coldness and numbness of the toes and feet, even when not exposed to cold, with occasional cramps involving the calf-muscles—*i. e.*, evidences of deficient peripheral capillary blood-supply—a corn is trimmed too closely or the shoe chafes the foot, producing perhaps nothing but some non-inflammatory hyperemia. This is just enough to determine stasis and thrombosis; the hyperemic area and the tissues around the corn or chafed spot become purplish, insensitive, and cold, although quite frequently complaint is made of severe pain in the circumjacent living tissues. The dead parts become blackened, shrivelled, and offensive, while the still living parts are reddened, somewhat ede-

matous and swollen, and are promptly attacked with genuine inflammation due to the infective germs resident upon the skin.

The line of intense hyperemia at the junction of the dead and living tissues is sometimes called the "line of demarcation," while the ulcerative one which succeeds this at about the same place is also called the "line of demarcation," or, more properly, the "line of separation." This limitation of the process is often only temporary, the gangrene resuming its progress from time to time, or it is continuous, usually, however, becoming arrested just below the knee, because the popliteal artery is often blocked just above the bifurcation, and the tissues below and around the knee receive their blood-supply through anastomoses with the inferior branches of the femoral. When the gangrene is limited in extent there is no constitutional disturbance, although the pain may, by preventing sleep, damage the health and nutrition, but if the death of the parts is extensive, evidences of constitutional sepsis appear early.

Raynaud's disease, or **symmetrical gangrene,** is an anemic gangrene due to vasoconstrictor spasm of central cerebral or spinal origin, with possibly secondary changes in the peripheral nerves.

Symptoms.—First pallor, coldness and numbness; "local syncope" is noticed at intervals, affecting the fingers, toes, tip of the nose, the ears, etc. This is the stage of invasion, which lasts for from a few days to a month or more. Next, cyanosis with much pain occurs—"local asphyxia." Finally, a dry gangrene (perhaps preceded by vesication) sets in, producing characteristic conical, pointed, shrivelled, gangrenous finger-tips. This process with separation of the eschars and the subsequent healing of the granulating surfaces occupies from ten days to as many months, averaging from three to four months. Although often associated with various chronic ailments, as no apparent cause is often detectable, renal and cardiac disease, with grippe, etc., can hardly be

considered as standing in any essential causative relation to Raynaud's disease.

Symptoms of Moist Gangrene.—There are two forms —viz., that limited to the areas actually killed by a traumatism, with some surrounding tissue which dies from causes already explained ; and that which tends to spread widely, this latter usually being caused by specific micro-organisms, an intense, widespread pyogenic in- flammation resulting, involving the subcutaneous and intermuscular cellular planes, by strangulation of the vessels of which all blood-supply to the remaining soft parts is destroyed. In both forms the primary burning, stinging pain of the inflammation ceases, the skin is pale, cold, insensitive, mottled, greenish, purplish, or red and livid. Blebs containing brownish serum quickly form, beneath which the derm resembles smoked beef and has lost all sensibility. These blebs, being caused by the accumulation of serum beneath epithelium which has lost its vital connection with the derm, can be slipped around upon the surrounding true skin, the epithelium readily separating for long distances around, as in a cadaver, thus permitting their easy differentia- tion from the blebs forming after severe contusions, where the tension and discoloration of the parts often suggest the idea of gangrene to the inexperienced.

If a wound be attacked with gangrene, its surfaces becomes pulpy, yellowish or grayish, profuse offensive discharge occurs, the surrounding tissues are swollen, brawny, and then boggy, intensely hyperemic, and the skin presents in various degrees the conditions just de- scribed. The line of demarcation forms by the same septic ulcerative processes already indicated. If life is prolonged, this ulcerative process will effect complete amputation of a limb, even the bone becoming divided. Hemorrhage is uncommon, because in advance of the ulceration thrombosis of the vessels takes place. Gan- grene following injuries causing free division of all the soft parts, especially the skin, because vent is afforded

for all discharges, are less dangerous to life and limb than slighter traumatisms which mechanically favor the retention of the inflammatory and gangrenous products.

Noma, or **cancrum oris,** is a gangrenous stomatitis, is apt to attack children convalescing from scarlatina, measles, etc., is mycotic in origin—often truly diphtheritic—and produces death of the tissues by inducing capillary thrombosis.

Spreading traumatic gangrene is, as was incidentally stated, an acute infectious or septic process, or a result of a mixed septic and infective inoculation. Undoubtedly some cases result from infection with a gas-producing bacillus. This variety of gangrene advances with lightning speed, extending most rapidly along that side of the limb where the lymphatic vessels lie. Sometimes it follows infection after extensive crushes of a limb, where both the main and the collateral arterial supply is destroyed; but it often arises from a comparatively slight injury, where either a virulent mixed infection has taken place, or one by a specific aërogenetic bacillus.

Symptoms of Spreading Traumatic Gangrene.—The limb quickly becomes intensely swollen and brawny, the skin becomes bronze-colored or resembles bacon rind, streaked or marbled with greenish or purplish lines. Emphysematous crackling can be felt extending beyond the apparent limits of the disease, whose progress can be followed by the eye from hour to hour. Marked sapremic symptoms soon appear because of the rapid absorption of the chemical products of putrefaction caused by the great pressure exerted by the confined fluids and gases; of course, genuine septicemia may follow if life be sufficiently prolonged by operation or otherwise. When the cellular tissue and fat are incised they resemble spoilt fat pork.

Gangrene in Diabetes.—In glycosuria, whether genuine diabetes or occurring in conjunction with gout, arteriosclerosis and chronic neuritis are not uncommon.

Slight inflammation excited in the ill-nourished tissues of such patients, in tissues rich in sugar, a substance which has been proven to favor the development of pyogenic organisms, often determines a variety of moist gangrene. Operations in glycosuriacs are apt to do badly; but, if infection can be avoided, they are not nearly so hopeless as was formerly taught.

Perforating Ulcer.—While in the condition termed "*perforating ulcer*" the process is rather ulcerative than gangrenous, this slowly destructive process, in time involving the integrity of the foot, can best be considered here, because largely due to chronic neuritis—often coinciding with glycosuria—plus infection of the consequently lowly vitalized tissues. It is characterized by a painless, progressive destruction of the parts, the bones succumbing in time, while the ulcer is insensitive, and resembling a physical removal of tissue rather than one produced by ulceration. Cutaneous anesthesia is always present in some degree, often extending almost to the knee.

Treatment of Gangrene.—Nothing but the promptest possible amputation well above all external evidences of the disease will avail to save life in the *spreading form* of gangrene, the freest possible incisions usually failing to secure against the absorption of a fatal dose of ptomains. Less radical procedures are often possible in other forms of gangrene, at least there is more time for deliberation. Prevent as far as possible the action of, or the access of, germs from without, especially in senile gangrene; secure free drainage and the relief of tension by incisions in appropriate cases. Favor the return of venous blood and lymph, and the development of the collateral circulation. Disinfect and employ dry, absorbent dressings, because moisture favors putrefaction. Abundance of sterilized powdered charcoal, with or without chemical disinfectants, is invaluable. Amputation for localized traumatic gangrene should be done only after the line of demarcation has clearly formed, other-

wise an unnecessary sacrifice of parts will often result. When spreading rapidly do not wait for the line of demarcation to form, cutting many inches above the apparent limit of the disease. When undoubtedly the result of arterial embolism, or following the ligation of a main artery, amputate at, or slightly above, the point of lodgement or ligature, so as to secure flaps vascular-ized by vessels coming off from the artery well above the site of the embolus or ligation. As experience shows that in *senile gangrene* the superficial femoral artery is often thrombosed far up the thigh, amputation at the junction of the middle and lower thirds, or about the middle of the thigh, should be done rather than a low operation. Of course, many patients are in no condition to stand such an operation. Removal of the limb at the point designated will secure flaps nourished by branches of the profunda femoris artery, which is never throm-bosed in senile gangrene. It is true that in rare instances spontaneous amputation may take place in senile gan-grene just below the knee ; but if an operation be done, the point advised had better be chosen. Many cases of senile gangrene, if limited to the toes, are best treated by dry antiseptic dressings and removal of the dead parts as they separate, satisfactory healing often following this plan of treatment. Extra long flaps should be employed, especially when the amputation is done for gangrene fol-lowing traumatism, and they should be held in con-tact by the dressings, not by sutures ; or iodoform gauze should be placed between them, secondary suturing being resorted to later if deemed requisite.

Stretching of the nerves and free excision of the dis-eased structures will at times permanently arrest the progress of a "*perforating ulcer*," but amputation above the line of anesthesia is usually the only satisfactory procedure. The treatment of *hospital gangrene* by de-struction of the infected tissues by the cautery, bromin, nitric acid, potassium permanganate, etc., has already been mentioned and will later be again described.

The treatment of *Raynaud's disease* consists in the use of the continuous descending current, warmth, protection of the parts, local anodyne applications, massage, and the administration of nitroglycerin before gangrene has taken place. When gangrene is present, the general principles of treatment applicable to this condition should guide the surgeon.

Noma, being a microbic disease, must be treated upon the same principles which have proved applicable to hospital gangrene and sloughing phagedena, except when proven to result from the action of the diphtheria bacillus, when serum-treatment should be instituted.

Malignant Edema; Gangrene Foudroyante; Gangrene Gazeuse.—This is an acute, infectious, mycotic, primarily non-suppurative gangrenous process chiefly involving the cellular tissue, and results from inoculation with an aërogenetic bacillus having for its favorite habitat garden-loam. Clinically, the free development of offensive gases seems to be dependent upon the presence of saprophytic organisms in addition to the specific bacillus; in other words, the disease results from a "mixed infection."

Symptoms.—These do not materially differ from those just mentioned as characterizing ordinary spreading traumatic gangrene, except by the more pronounced gaseous distention, producing a crepitation like that of normal lung when handled. A thin, stinking fluid can be expressed in large quantities from any wound present, or after incision of the skin. The temperature quickly reaches a high point, where it remains with but slight fluctuations. Delirium appears early, a typhoid condition soon develops, coma appears, and death soon occurs, the whole course of the disease being often run in from eighteen to thirty-six hours. The same bronzing and mottling of the skin already mentioned on page 203 is seen, and blebs containing bloody fluid frequently form. Post mortem, the cellular tissue and the muscles are bathed in and permeated by a thin gas-containing, red-

dish, evil-smelling fluid. Softening thrombi are found in the veins, while the heart and larger vessels may contain gas set free from the decomposing blood.

Prognosis.—This is grave, for even if recovery ensues, destruction of the part or loss of the limb is the price paid.

Treatment.—While free incisions and the subsequent use of antiseptic dressings or the antiseptic bath are all that can be done locally, when the disease is so situated that the affected tissues cannot be physically removed, amputation should be performed if a limb be the site of the trouble. Appropriate constitutional treatment must be employed, as indicated by the condition of the patient.

LECTURE XX.

ULCERATION; ULCERS.

Ulceration is molecular destruction of tissue, strictly speaking, always the result of infection. An "ulcer" is a solution of continuity situated upon the skin or a mucous surface, produced by molecular loss of substance, sometimes increased by sloughing—the result of microbic inflammation. Any granulating surface left after accident or operation is also incorrectly termed an "ulcer."

The differing appearances presented by many ulcers are due solely to local conditions which may vary, from accidental causes, from day to day, obscuring any characteristic features the sore may possess. Others owe their peculiarities to specific or constitutional causes, although varying local conditions may produce in them also such changes as will prevent, at least for a time, the recognition of the true causation of the ulcer. These latter forms of ulcers will not be considered here.

As all ulcers must attain to the conditions characterizing the "simple," "healthy," or "healing ulcer" in order to cicatrize, this variety must be studied first. All variations from this type are non-specific and are caused by obstacles presented to nature's attempts to heal by organization of granulations and the covering in of this organized tissue by epidermic cells. The margins of a healthy ulcer are smooth, shelve down to a level base, which is covered with healthy granulations, moistened by a little creamy, inodorous pus, or, if asepsis has been maintained or later secured, by an opalescent fluid, rendered opaque, not by dead phagocytes—*i. e.*, pus-cells—but by living cells which have been mechanically extruded with

the outflowing serum. The surrounding skin is healthy, the edges of the sore are not sensitive, and merge gradually into the granulations by a bluish-white film, opaque and whiter upon the skin side, distinctly bluer and more translucent where joining the granulations. These appearances are due to the advancing growth of epithelium, there being many layers on the older, comparatively avascular organized granulation-tissue where white and opaque, permitting the more recently organized, hence more vascular, granulations to show through a smaller number of layers of epidermic cells where the bluish appearance is seen.

Treatment.—As the name implies, if protected from irritation and if the part is kept at rest, healing will take place. Should the surface be so extensive that the regenerative powers of the epithelium are not equal to the task, Thiersch or Reverdin skin-grafting should be employed.

Fungous Ulcer.—Let anything persistently interfere with the return circulation, even from a healing ulcer, still more from one that has not attained this condition, and the granulations will become congested, deep-red, overgrown, and ill-formed. They will project above the healthy margins, readily bleed, and give exit to a free, thin, purulent, or blood-stained discharge. Healing ceases to progress and may even retrograde slightly. The dependent position and excessive scar-production in and around the margins of the ulcer, the latter becoming pronounced because of the delay in cicatrization, are the most common causes of degeneration of a healthy ulcer into a fungous one, or of its original formation.

Treatment.—The indications are removal of the cause, as the dependent position of the limb, favoring venous return, the employment of remedies to constringe the vessels of the granulations, and mechanical support of the circulation. These indications can be carried out by applying solutions of astringent salts, as those of silver, copper, and zinc, touching the granulations with the

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solid stick of nitrate of silver or a crystal of copper sulphate, and elevation and bandaging of the limb.

Edematous Ulcer.—If persistently employed, poultices or wet dressings will convert any ulcer more or less completely into an edematous one, although a feeble venous circulation, however produced, favors edema of granulations and may very rarely be the chief cause. Sometimes maceration of the ulcer by its own discharges produces the edematous condition. If the ulcer has previously been healing, the margins may remain fairly healthy, although further progress in the formation of epidermis ceases. The granulations are swollen, flabby, pale, semi-translucent and friable, a large amount of watery pus being secreted, while the margins of the ulcer eventually become sodden and macerated.

Treatment.—More blood and that circulating at a proper rate of speed is clearly demanded. Stimulant and astringent applications, such as chloral, grs. x to aqua ʒj, resin ointment, balsam of Peru, dry dressings, elevation of the part, and mechanical support of the circulation by gentle pressure over the ulcer and bandaging of the limb distal and proximal to the ulcer, are the indications, and their fulfilment will usually soon correct the condition. Indeed, the substitution of dry dressings, in conjunction with desiccating powders such as boric acid or zereform, renewed frequently enough to prevent maceration by the discharge, will often be all that is requisite.

Inflammatory Ulcer; Inflamed Ulcer.—Traumatisms which would produce in healthy individuals no especial result, or at the worst an ordinary ulcer, will often cause in those addicted to the use of alcohol and who are ill-fed and poorly nourished, a rapidly enlarging ulcer, irregular in outline, with sharp-cut, ragged margins. The base of such an ulcer is formed not of granulations but of the red, inflamed tissues themselves, which freely secrete a serosanguinolent discharge often containing shreds of dead tissue. If the process be hyperacute, the base may be covered with yellowish sloughs. The

circumjacent skin is reddened, inflamed, edematous, and tender.

Inflamed Ulcer.—A previously healthy ulcer, if irritated, neglected, and allowed to become fouled with dirt, especially in drinkers after a spree, will often be attacked with inflammation, the granulations becoming intensely red, swollen, and sloughing ; the margins break down, and the surrounding parts become inflamed.

Treatment.—In both the inflammatory and inflamed forms of ulcer the indications are to remove irritation, diminish hyperemia, disinfect, limit the amount of the alcoholic poison, secure elimination of effete matters, improve the general nutrition, and secure sleep. Elevation of the part, the application of warm, moist, non-irritating antiseptic lotions—best of all, immersion of the part in the continuous warm bath—moderation in drink, the ingestion of more and better food, and the use of laxatives, diuretics, and diaphoretics will in time transform the unhealthy into the healing ulcer.

The **sloughing ulcer,** except when the term is applied to the worst varieties of the inflamed or inflammatory ulcer, is rarely seen except in venereal disease attacking broken-down alcoholics. The parts rapidly melt away, and as the cellular tissue is destroyed more rapidly than the skin, the latter becomes undermined, and the edges of dusky-red integument are consequently inverted. The base is covered with gray or blackened sloughs and the discharge is free and foul-smelling. Great pain is experienced and much constitutional disturbance accompanies the progressive destruction of tissue.

Treatment.—This differs in no important particulars from that appropriate for an inflamed ulcer, except for the greater need of anodynes, tonics, and stimulants. Even in syphilitics mercury is rarely indicated, and when employed must be used with great caution, because the real trouble is acute pyogenic infection, not syphilis.

Phagedenic Ulcer ; Sloughing Phagedena.—It has been contended that this results from infection by a

specific microbe. As it occurs chiefly in broken-down intemperates after chancroidal infection, this explanation is questionable. In most cases a virulent mixed pyogenic infection attacking the poorly nourished tissues of individuals whose powers of elimination are seriously impaired by their habits and whose blood is already saturated with imperfectly elaborated excreta, would account for the results without invoking the action of a specific germ.

Symptoms.—For a wide area around the ulcer, the inflamed skin presents a dusky-red, purplish hue, the margins of the sore are ragged and undermined and rapidly break down, the base is a bloody, sloughing mass, from which exudes a free, sanious, and offensive discharge. In its worst forms the tissues disappear with great rapidity by a combined process of ulceration and sloughing, until, for instance, the whole external genitals are destroyed. Great pain and constitutional disturbance attend the progress of the ulceration.

Treatment.—Because of the virulence of the mycotic infection destruction of the germs and the tissues into which they have penetrated is requisite. For this purpose—in the severe cases under anesthesia—after careful removal of all sloughs by the scissors or curet, pure bromin, nitric acid, or the actual cautery should be thoroughly applied. In milder cases after removal of sloughs thorough mopping with a 1 : 1000 mercuric chlorid solution followed by the free use of iodoform may suffice. The continuous warm antiseptic bath may be employed to supplement or supersede the methods recommended. The exhibition of opium, tonics, and stimulants, with the administration of abundance of nutritious food, and improvement in the hygienic surroundings are both desirable and usually essential.

Hospital gangrene is probably a variety of sloughing phagedena, due to a streptococcus infection ; but as it has practically disappeared, no reliable bacteriological examinations have been made. It attacks open wounds,

producing destructions of tissue which tend to assume a
rounded form. The diphtheritic form consists in a co-
agulation-necrosis of the granulations, moderate inflam-
mation of the wound-margins, diminution of the puru-
lent discharge, followed by a free watery one, and finally
separation of the sloughs takes place, leaving a "crater-
shaped" loss of substance with everted edges. An
ulcerating form occurs accompanied by a rather super-
ficial enlargement of the sore, the granulations becoming
gray and sloughing, and the discharge free. Finally, a
pulpy variety is seen, where the granulations become
edematous and necrotic, producing excessive swelling
of the wound-surfaces, which give exit to large amounts
of fetid discharge. The margins are swollen, everted,
very sensitive, and the surrounding parts are discolored
and swollen. Marked constitutional disturbance exists.
Secondary hemorrhage is common, joints may be laid
open, and death results from sapremia or septicemia,
hastened by loss of blood in many cases, even where no
very large vessel is opened; of course, recovery may occur.

Treatment.—Asepsis should prevent and isolation ar-
rest the spread of this disease, which is clearly con-
tagious. Placing the patients in tents will often serve
the double purpose of isolation and improve the condi-
tion of the patients. The local treatment recommended
for sloughing phagedena is applicable to the treatment
of hospital gangrene, adding to the list of destructive
agents permanganate of potassium and perchlorid of
iron. Amputation may become requisite in the presence
of severe hemorrhage or destruction of a joint.

Indolent or Chronic Ulcer.—Let any granulating
surface have its healing processes repeatedly thwarted
by mechanical or other irritation, and the formation of
epidermis will be prevented, while the embryonic tissue
of the margins and base will develop into dense fibrous
tissue fixing them to the subjacent bone or fascia. Aided
by a weak action of the heart and interference with the
return of venous blood, for instance that produced by

varicosity of the veins, venous hyperemia will be maintained; moreover, the arterial supply will be still further diminished by the pressure exercised upon the vessels by the condensing scar-tissue. From a combination of all these causes, the formation of healthy, organizable granulations ceases. As ulcers heal chiefly by the diminution in their superficial area, effected by the contraction of the granulation-tissue forming their bases during organization, only a comparatively small extent of the original surface requiring to be covered by epidermis, fixation of the base and margins of a chronic ulcer is one of the chief obstacles to healing, and the loosening of these parts is essential for cicatrization.

Symptoms.—The lower third of the leg is the favorite site for a chronic ulcer. The edges are smooth, rounded, elevated, and indolent, while the circumjacent skin is bronzed, purplish, and often eczematous. Very few granulations can be seen on the base of the ulcer, any present being scattered, pale, and ill-formed. The discharge is thin and puruloid or sanious, and usually very offensive. Pain is nearly always insignificant. The duration is from a few to forty years. Some undergo a pseudo-epitheliomatous change, and a few a genuine one, productive of the so-called "Marjolin's ulcer." The terms "eczematous" and "varicose ulcer" merely mean ulcers complicated with eczema or with varicose veins, the former probably largely caused by the latter on account of the chronic hyperemia and malnutrition thus induced.

Treatment.—Two distinct indications exist, the first to secure loosening of the base and indurated margins of the ulcer from the underlying tissues, and, second, to so improve the vascular supply of the ulcer that venous return and arterial supply will become normal, resulting in the formation of healthy, organizable granulations. Radiating incisions through the thickened margins reaching down to the fascia will serve the double purpose of loosening attachments and inducing hyperemia.

More or less extensively encircling incisions made down to the fascia, about one inch from the margins of the ulcer, may be employed in extreme cases, and will permit the base to contract. Pressure by strapping will stimulate absorption of exudate, thus loosening the adhesions and removing pressure from the vessels, permitting easier ingress and egress of blood. Blistering will effect the same results and also induce an acute hyperemia. Free removal of the base of the ulcer with the curet, followed by disinfection with a solution of zinc chlorid, hydrogen peroxid, etc., will often secure asepsis, which must be maintained, and with removal of thickening of the margins by incisions, pressure, etc., will often also secure the hyperemia requisite to induce repair. Rest in bed, with elevation of the limb, is a most important adjuvant and is almost always essential. If this cannot be done, and certainly for some time after healing has been secured, moderate support must be afforded to the circulation of the limb by bandages, or an elastic stocking. This support must be permanently employed if decided varicosity of the veins exists, or the veins must be ligated or excised. This latter operation is sometimes advisable as one of the curative measures. When asepsis is not attempted, some one of the measures adapted to remove the thickening of the margins with the use of stimulant applications will be best, as resin ointment, blistering, etc. Strapping with resin adhesive plaster by causing absorption from pressure, and at the same time supporting the circulation and stimulating the surface, is an old and reliable method. Although aseptic measures are best in theory, in private practice among the poorer classes they are often impracticable. Skin-grafting, when healthy granulations have been secured, may prove useful. Extensive ulcers which have destroyed much of the skin and completely encircle the limb are often incurable, and either palliation or amputation have to be resorted to.

Irritable or Painful Ulcer.—This usually occurs in women over forty years of age, is situated above and near the ankle, and is congested, very sensitive, and a constant source of suffering. Sometimes by gently going over the surface of the ulcer with a probe, a limited point or points of increased sensibility can be made out, due to a partially exposed or irritated nerve-filament or filaments of some magnitude. At other times, especially tender points cannot be detected. The congestion which prevents healing is due to irritation of the exposed sensory nerves, which, as we have learnt, will induce hyperemia in the parts supplied by them.

Treatment.—When distinctly painful points can be made out, a section of the base of the ulcer at such places with a tenotome will, by dividing the nerve-filaments, at once relieve the pain and congestion and lead to healing. If sensitive points are absent, rest, elevation, and asepsis must be tried, and full doses of opium administered, which latter often promptly relieves the congestion and causes healing by obtunding the irritability of the implicated nerve-filaments.

Syphilitic, gouty, tubercular, lupous, scorbutic, carcinomatous, and rodent ulcers, being special conditions not occurring in some one of the forms described as present after destruction of normal structures by inflammation, are not amenable to any general laws or principles, hence must be studied elsewhere.

LECTURE XXI.

ERYSIPELAS; TETANUS; TETANY.

IT will now be proper to take up the consideration of certain wound-diseases, because any wound, however slight, may serve as their starting-point, or have its course and nature of its treatment decided by the presence of the complicating ailment.

As special instruction is given in bacteriology in all medical schools, no description will be attempted of the morphology, staining qualities, or culture methods of the germs causing the diseases now to be mentioned.

Erysipelas.—This is an acute, non-suppurative, infectious inflammation of the skin or a mucous membrane, due to a streptococcus-infection which tends to travel widely along the superficial lymph-vessels but sometimes extends to the deeper parts. The germs are found in the lymph-spaces, especially at the advancing border. Erysipelas is always due to an open traumatism, but this may be most trivial, may have healed before the outbreak of the disease, or may be concealed in a cavity, as that of the nose or pharynx, either of which often contains the infection-atrium of a facial erysipelas; hence the term "idiopathic erysipelas" is a misnomer, indicating merely that the point of infection has not been detected. It has frequently appeared as an epidemic, often when puerperal fever was prevailing, both proving specially virulent, and apparently closely related to one another.

Prognosis.—This is usually good, but owing to complications, the result of a *mixed* or *secondary infection*, it must be guarded, as meningitis, peritonitis, and

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various septic or infective conditions may supervene. Thus, the comparatively innocent Bacillus prodigiosus when grown with the streptococcus of erysipelas increases by many fold the virulence of the streptococcus, which is taken advantage of in preparing the antistreptococcus serum for the treatment of malignant growths. When the inflammation attacks the subcutaneous tissues it is called "cellulocutaneous erysipelas," while if pyogenic infection is present producing suppuration the term "phlegmonous erysipelas" is applied to the disease. When all the deeper tissues of a part are attacked by a pyogenic inflammation in which the skin is involved, the process should not be considered as erysipelas, but what it really is, a diffuse spreading pyogenic infective inflammation of the cellular tissue of a part, an "infective cellulitis." Although it is quite possible to have an infective cellulitis secondary to true erysipelas, or even under certain circumstances have the erysipelas coccus cause suppuration, yet, dermatitis being a necessary result of deep cellulitis, the skin-lesion does not prove that the disease-process bears any relation to erysipelas ; moreover, suppuration in erysipelas is nearly always the result of secondary staphylococcus infection originating from the germs present upon or in the skin, or deposited from the blood in the congested, inflamed parts. Inflammations of the salivary glands in facial erysipelas and a form of pneumonia may complicate erysipelas ; hence, with all the risks incident to any pyogenic process, the prognosis must be uncertain and dependent rather upon the absence or presence and nature of the complications.

Symptoms.—These are both constitutional and local. Malaise is complained of, and sometimes the lymph-nodes nearest to the point of infection may be swollen, tender, and painful twenty-four or more hours before other symptoms are detectable, with reddened lines of inflamed lymphatic vessels leading to them. A chill usually ushers in the attack, sometimes replaced in chil-

dren by a convulsion. A prompt rise of temperature follows the chill, although an abrupt temperature elevation may precede the chill. The pyrexia is irregular, with a gradual subsidence to the norm, if no complications occur, in the course of about ten days; but in some cases, owing to the tendency to attack fresh portions of the skin—the "wandering erysipelas"—convalescence may be indefinitely delayed. Gastro-intestinal disturbance is evidenced by anorexia, a coated tongue, nausea, and possibly diarrhea, which may even be bloody from intestinal ulceration. In phlegmonous erysipelas all the evidences of constitutional infection are present in varying degrees, death taking place in a typhoid state in the worst cases. Delirium and apparent evidences of implication of the central nervous system may be present, especially in facial erysipelas, without any inflammation of the brain or its membranes, due to plugging of the cortical vessels with lymphocytes, although true infective meningitis does sometimes occur. As has already been incidentally mentioned, any or all of the complications of pyogenic infection may occur, many of which have been mentioned when speaking of prognosis; others will be described when giving the local symptoms of the phlegmonous form. The local symptoms of the cutaneous variety are due to a dermatitis resulting in decided infiltration and edema of the skin, which presents a brightly reddened, sharply defined area, with an elevated irregularly outlined border on the advancing side, which gradually fades into the surrounding healthy parts at the point first attacked. Vesication is common, the blebs rupture, and, drying, leave brownish scabs; marked desquamation of the epidermis takes place after recovery. In lax tissues as those of the eyelids and scrotum, the edema is so excessive that great disfigurement is thereby produced. From the great distention of these comparatively avascular parts, gangrene or ulceration of the skin sometimes occurs, which if reinforced by secondary pyogenic infection may give rise

to serious consequences, as, for instance, deep orbital abscess with possible involvement of the meninges by continuity of tissue. When attacking a wound, any union present breaks down, sloughing occurs, and the discharge becomes profuse and seropurulent. If a granulating surface is concerned, the granulations become dry and glazed, coagulation-necrosis takes place, an apparent pseudomembrane forming, and this in turn is succeeded by sloughing of the granulations, which next attacks the deeper parts, the disease progressing by preference along the cellular tissue. The enormous amount of exudate poured out in phlegmonous erysipelas causes extreme tension ; the parts are brawny and covered with bullæ, and the induration is followed by bogginess and later by obscure fluctuation, which becomes more distinct as time elapses. There is decided pain from the great tension, which produces death of the skin, partly by strangulation of its blood-supply, partly from actual destruction of the same, because of death of the subjacent tissues. When the skin yields from sloughing or is incised over the softened areas described, pus and sloughs, the latter composed chiefly of cellular tissue resembling wet wash-leather, will be exposed. Because the lymphatic vessels, by which the streptococcus infection travels most readily, run in the intermuscular planes and fasciæ, the muscles are often extensively dissected from one another, the pus burrowing deeply, even baring the bone of periosteum and causing necrosis, possibly producing involvement and subsequent destruction of joints. The alleged rheumatic joint-affections sometimes observed during an attack of erysipelas are either due to spreading of the inflammation to the joint structures by continuity from a neighboring streptococcus process, or are merely instances of septicemic or pyemic arthritis. If the risk of sloughing of the skin of the eyelids or scrotum from accumulation of exudate is present in ordinary cutaneous erysipelas, this becomes a practical certainty when the same parts are attacked by

phlegmonous erysipelas. It is not uncommon for considerable portions of the aponeurosis of the occipito-frontalis muscle to slough, and even necrosis of the skull may occur. Erysipelas attacking the mouth, fauces, or pharynx, is a most serious affair, because of the probability of acute edema of the glottis ; indeed, this accident is thought by many to invariably be due to erysipelatous laryngitis. The older surgeons thought that the intense hyperemia caused by an attack of erysipelas was favorable to subsequent healing ; this can hardly be true except for a chronic or indolent condition, where the vascular supply has been deficient.

Post mortem, nothing characteristic will be found except the local conditions described and the changes common to septic infections.

Diagnosis.—Only the cutaneous form is likely to be confused with any other ailment, and this only with erythema and drug-eruptions. Rashes from ptomain-poisoning are commonly generalized, and are not followed by vesication. Erythema has no elevated, sharply defined border, but the redness fades out into the hue of the surrounding healthy skin. Drug-erythemas are either confined to the areas to which the application has been made, or appear at points far distant from the wound. Still further, constitutional symptoms are absent in drug-erythemas.

Treatment.—Isolation is necessary because erysipelas is contagious, and if possible special nurses should attend to erysipelas cases to lessen the risk of contagion, while all dressings should be promptly destroyed. Proper action of the secretory organs must be secured and maintained. Avoid or remove intestinal sepsis. Secure sleep by the administration of hypnotics. Employ supporting measures and administer alcoholic stimulants freely, especially in drinkers, supplementing this with strychnin. See that the patient ingests a sufficient amount of proper food. Tincture of the

chlorid of iron, so much in vogue at one time in the treatment of erysipelas, is probably of no value beyond, perhaps, its diuretic effects, and later to relieve the anemia. Curative measures must aim to destroy or inhibit the growth of the streptococci producing the disease. This the antistreptococcus serum is supposed to do, and there seem to be good grounds for this belief; yet the whole matter is still upon trial. Maintaining germicidal substances in contact with the skin will result in their absorption by the skin lymphatics—*i. e.*, the tissues in which streptococci flourish. Thus, weak solutions of carbolic acid or corrosive sublimate sometimes exert a decided effect. Ichthyol in the form of 10 per cent. ointment is apparently still more efficacious. Park extols an ointment composed of resorcin 5 parts, ichthyol 5 parts, mercurial ointment 40 parts, and lanolin 50 parts. The use of a 15 per cent. ointment of Crede's colloid silver will probably be still more efficacious. Injections twice daily of small amounts of a 2 to 3 per cent. solution of carbolic acid into the skin or subcutaneous tissues around, or, better, in advance of the spreading borders of the disease, have seemed to arrest its progress. Recently it has been claimed that the light passage of an alcohol flame twice daily for one or two days over the inflamed surface, covered with one or two layers of moistened gauze, will destroy the germs at the expense of slight vesication of the surface; this is a radical measure and would seem not entirely devoid of danger. Free antiseptic incision made down to the bone in case of whitlow, before pus has formed, will usually abort the inflammation and prevent the sloughing and necrosis in this probably erysipelatous ailment. The suppurations and sloughings of the phlegmonous variety must be met by incisions, disinfection, and the general and local treatment adapted to similar conditions produced by pyogenic infection, such as those which have already been described, or which will later be adverted to. Amputation may become requisite to avert death

from exhaustion or infection, and to remove at the same time a hopelessly damaged joint or limb.

Intubation or tracheotomy is usually requisite to save life in edema of the larynx, but free scarification of the infiltrated tissues will sometimes suffice.

Tetanus.—This is an acute, non-suppurative, microbic disease, characterized by tonic followed by clonic contractions of the voluntary muscles. The germ is an anaërobic one, hence the danger of lacerated and punctured wounds which provide anaërobic culture-chambers. Suppuration favors tetanic infection because the pyogenic organisms consume the oxygen and thus render possible the multiplication of the tetanus bacillus and its spores, which latter are in the majority of instances the infective agents. Valagussa has apparently demonstrated that the tetanus germ is only toxic when developing anaërobically, losing its virulence when exposed to sunlight and air, although still continuing to grow. It cannot be isolated from the surface of the soil, being there killed by the agencies described—*i. e.*, sunlight and air. The aërobic form, if old and grown with common saprophytic germs anaërobically in the dark for long periods at a proper temperature, becomes again pathogenic. These observations explain the toxicity of the germs found in the excreta of some of the domestic animals, notably the horse. The microbes are found in the deeper portions of the superficial layers of black loam, particularly garden soil which has been manured with horse-dung, animals in whose alimentary canal tetanus germs flourish. Although certain races seem more prone to develop tetanus, as the negro, the Hindoo, and the South Sea islanders, this may be more apparent than real, owing to their unclad feet and bodies, the use of mud- or dung-poultices for wounds, mud for arrow-poison, a constant temperature favorable to the development of the bacillus, and their often filthy habits of life. Conditions favoring lowered vitality and congestion, as the relatively cold tropical

nights following the hot days, predispose to tetanus, as Larry found in his Egyptian campaign.

The specific microbe is never found in the tissues at any distance from the wound, hence it acts by its toxins, *tetanin*, *tetanotoxin*, and *spasmotoxin*. Idiopathic tetanus does not exist, the name being a mere confession of ignorance as to the locality of the site of infection ; thus I am cognizant of a case where the portal of entry was proved to be a carious tooth, the child constantly putting the finger, soiled with garden mould in which it played, into the painful hollow tooth. Slight traumatisms of the respiratory and alimentary tract may be the sources of the infection. Tetanus occurring in consequence of wounds of parts supplied by the trifacial nerves is apt to show predominance of spasm of the facial, pharyngeal, and cervical muscles, and a comparative absence of involvement of the abdominal muscles ; indeed, from the superficial resemblance to hydrophobia, it is often called "Tetanus Hydrophobica," and is asserted to be one of the least fatal varieties of tetanus.

Symptoms.—While in the laboratory the incubation period is about forty-eight hours, it must be clear from what has been said that clinically it must vary, because a less virulent form of microbe may acquire toxicity by long growth with saprophytes in the wound, and the necessary anaërobic conditions may supervene at different periods. Thus, although the disease usually appears in from two to four days after the reception of the wound, and rarely later than three weeks, yet it may show its first symptoms as late as between six and eight weeks, as in one reported case. The first symptoms noted by the patient are stiffness of the posterior cervical muscles, preventing the chin from being readily depressed, and difficulty in moving the jaw from "stiffness" of the temporal and masseter muscles, this condition being often described by the patient as a "sore throat."

The mouth cannot be fully opened, and the maximum point is slowly reached with manifest effort. The mus-

cles mentioned are rigidly contracted, which condition soon renders them sore and painful. Owing to tonic contraction of the risorius muscles and the depressors of the angles of the mouth, a peculiar "sardonic grin" is noticeable. The abdominal muscles are so tensely contracted that the abdominal wall feels "as hard as a board." The extensor muscles of the trunk and limbs gradually become rigidly contracted, producing over-extension, "opisthotonos." "Pleurosthotonos" is the term employed to designate lateral flexion, while, if the abdominal muscles and flexors of the thighs gain the mastery, the body is bowed forward, "emprosthotonos" resulting. Opisthotonos is almost the rule, and is often so extremely pronounced that the patient rests upon the occiput and the heels, and can be lifted by the latter, touching the bed only with the head. Both the tonic rigidity and the clonic spasms are apt to commence in the limb injured, although there is no invariable rule. Even during the early stages of the disease, clonic spasms occur from time to time, induced by light, noise, a waft of cold air, or the slightest touch, owing to the intense excitability of all the reflexes. Tonic spasm of the sphincters produces constipation of the bowels and retention of urine. The mind remains clear, and the temperature is often not abnormal for the first few days. As the case progresses, the clonic exacerbations increase in frequency and severity, and add to the difficulty of respiration constantly present from slight tonic contraction of the diaphragm, complete spasm of this muscle with violent contraction of the accessory muscles of respiration and of the glottis, until death seems imminent from apnea. Occasionally a little air enters or escapes from the chest with a hissing sound through the tightly clinched teeth, the patient presenting a most distressing appearance, the face cyanosed, with foam-covered lips, protruding eyeballs and beaded with sweat. The violence of the paroxysm gradually diminishes, and a return to the rigid tonic contraction occurs, or death

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takes place from apnea. Pain is not so severe as would seem must result from the violent contraction of the muscles. Later in the disease, generalized sweating is not uncommon. The temperature may rise and continue to ascend even after death, touching a high point. The case may reach its acme, and death, due to apnea, occur in from a few hours to three or four days; or again the disease may commence more gradually, progress slowly, and only occasionally exhibit the violence described, when it is called "chronic tetanus." The incubation in such cases is usually much longer, and the whole course is much milder, justifying a more favorable prognosis than for acute tetanus.

Post mortem, there is nothing really characteristic to be found either in the wound or elsewhere.

Diagnosis.—Strychnin-poisoning contrasted with tetanus shows spasms of the masseters last, the convulsions are clonic not tonic, and there is often green vision. Hydrophobia has distinct mental symptoms, hallucinations and paralysis of deglutition, a thick tenacious mucus is secreted, and death takes place after apparent amelioration of the convulsive phenomena—*i. e.*, paresis or paralysis occurs, sharply contrasted with the increasing violence of the paroxysms, and death during a convulsion, as is usual in tetanus.

As *tetany*, a pure neurosis, may supervene after thyroidectomy, the chief points in which it differs from tetanus require mention. The symptoms may appear almost immediately or as late as ten days after operation. Facial muscular spasm appears late or not at all, when present sometimes producing trismus and the sardonic grin. The hands are flexed and drawn to the ulnar side, the fingers are extended at the interphalangeal joints but flexed at the metacarpophalangeal joints, the thumb being drawn into the palm of the hand; "the elbows are bent and the arms folded over the chest." This results from tonic spasm of the affected muscles, which may be partly overcome by firm traction. Pain is ex-

perienced during these tonic spasms, which last for a number of minutes, recurring at fairly regular intervals. Similar pedal spasms are also noted ; hence the name "carpopedal spasm." There may be elevation of the pulse and temperature in acute cases. Compression of the main vessels or nerves of a limb will induce the spasms, as will a sharp tap over the facial nerve as it comes out from the parotid gland. The post-operative disease is always dangerous and is frequently fatal. Its connection with dilatation of the stomach and other affections does not here concern us.

Prognosis of Tetanus.—Although the prospects of recovery are poor in an acute case, the epigrammatic French saying "tant des cas tant des morts" is not justified. The prospects are better in the subacute form, and in cases of chronic and of head tetanus. Any patient who lives over six days has a fair chance of recovery. Lambert claims that even with the older methods of treatment the mortality is for acute cases only 80 per cent., and for chronic cases 40 per cent., being an average mortality of 60 per cent. for all forms. While the antitoxin treatment has effected only a reduction of 5 per cent. in the mortality of acute tetanus, it has reduced that of the chronic variety to 16 per cent., a very material improvement. This increase in the chances of recovery is unfortunately limited to cases where the incubation period is seven days or over.

LECTURE XXII.

TREATMENT OF TETANUS; HYDROPHOBIA.

Treatment of Tetanus.—From careful observations it would seem that a peculiar affinity exists between the nerve-cells and the tetanus toxins, and their fixation by them has been seemingly demonstrated. This explains the failure of antitoxin when employed late and by the cellular tissue or by the veins, because of the weaker affinity of the antitoxin than of the toxins for the nerve-cells. A number of clinical observations apparently indicate that the serum is not strictly antidotal, but confers immunity on the nerve-cells, hence must be used chiefly as a prophylactic, although of course serviceable during an attack in protecting such nerve-cells as have not absorbed the poisons. Intravenous injections should be used to neutralize the toxins in the blood; intra-cranial ones can alone reach those fixed by the nerve-cells. Intracranial injections must be thrown in drop by drop, with the strictest of aseptic precautions.[1] Rambaud recommends doses of 3 c.c., thrown in by means of a special syringe with a piston working through the cap by a screw-thread, the fluid being forced out of the instrument by screwing the piston down. The point must be conical, to fit a trephine opening through the skull 7 mm. in diameter, the needle being connected with the syringe by rubber tubing, to prevent laceration of the brain; the needle should be grooved on one side to permit the escape of any fluid. Ten to twelve minutes should be occupied in making each injection,

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[1] A death from brain-abscess weeks after recovery from the tetanus has recently been reported.

which should be made into the brain-substance in one of the neutral zones. Several points of injection may be deemed requisite, as well as a repetition of the process. Intravenous injections should also be employed in conjunction with the intracranial ones, for reasons already mentioned. Perfect rest must be enjoined, because Lambert reports five cases of sudden death after apparent recovery, occurring during a convulsion excited by the patient being suddenly aroused out of a sound sleep. The only probable means of averting such an accident, besides quiet, is keeping the patient under the influence of chloral. As the vitality of tetanus bacilli and their spores is not affected by the antitoxin, both retaining their vitality in a wound for weeks, successive increments of toxins may be formed as the spores develop, although the effects of the primary dose may have been neutralized by the antitoxin. Repetition of the remedy in doses of 10 to 20 c.c. once a week for at least three weeks has been recommended to maintain the immunization.

What therapeutic doses of antitoxin shall be employed? From 15 to 30 c.c. repeated at intervals of six hours until improvement is noticed; then smaller doses at increasing intervals. When used by the intravenous route, either smaller doses should be employed or the intervals must be greater. But in the absence of antitoxin, or until this can be procured, can nothing be done? Such a measure of success attends the use of drugs which physiologically antagonize the symptoms induced by the tetanus toxemia, that prolongation of life is often secured until all the poison is eliminated and the tetanus spores cease to develop; indeed, some of the drugs to be mentioned, notably chloral, have frequently been used in conjunction with the antitoxin. The twofold end should always be kept in view, of antagonizing the effects of the toxemia and preventing the further formation of toxins by destruction or removal of the germs and their spores. Considering this last indication first, amputation

of a limb or excision of the tissues for some distance around the wound has been practised. Rose has seen 42 cases recover after the first procedure, and it is certainly the surest method of disinfection, because removing the source of infection. Destruction of the infected tissues by the free use of the actual or potential cautery has seemed occasionally beneficial, while in the more chronic forms, hypodermic injections of from 2 to 5 per cent. solutions of carbolic acid or weak solutions of corrosive sublimate seem to have exercised a curative effect. Neurotomy and nerve-stretching still have advocates, as Rose. As several toxins have been isolated, and as some cases of tetanus begin with pains in the wound and spasms of the adjacent muscles, while others show trismus—*i. e.,* "lock-jaw"—first, it has been suggested that different toxins are operative in these two classes; restriction of amputation and nerve-section to cases where local symptoms are the primary ones, may demonstrate the real value of these procedures. A colleague obtained a recovery after an amputation of a finger, cause and effect seeming to be distinctly related. Of the drugs which diminish reflex excitability and the tendency to clonic convulsions, chloral occupies the first place. When difficult to administer by the mouth it may be given by the rectum, and by either route acts better when combined with a bromid. Chloroform, cautiously employed, certainly relieves suffering, and its use is claimed to have reduced the death-rate by 10 per cent. Morphin is beneficial by mitigating the tonic spasm and securing sleep. Inhalations of nitrite of amyl, administered between the paroxysms, has in my experience given great relief. When one remedy ceases to benefit, another should be tried for a time, perhaps returning again to the former remedy later. Absolute quiet in a dark room, daily evacuation of the bowels, catheterization of the bladder at proper intervals, and the administration of nourishment, by a catheter passed through one nostril if food cannot otherwise be ingested,

or by the stomach-tube under anesthesia if necessary, are all imperatively demanded.

Comparing the antitoxin treatment with that by drugs, Goodrich's statements may be accepted as true in the present state of science :

(1) " The rate of recovery is higher under the antitoxin treatment in those cases having short incubation.

(2) " The supremacy of antispasmodic treatment seems most marked in cases of unknown incubation, where, in most instances, no wound was discovered.

(3) " Most of the cases treated by antitoxin have probably been reported, whereas the great majority of fatal cases treated by other methods are quite as probably withheld from publication.

"Thus it would seem that . . . . antitoxin holds a very important place in the treatment of tetanus, although whether it is established there positively is as yet a question. . . . Tizzoni's product has undoubtedly been the most successful."

The *prophylaxis* of tetanus is of great moment. Eating raw fruits or vegetables which have not been thoroughly cleansed should be avoided, especially fruit which has fallen to the ground, lest an abrasion of the mouth or a carious tooth become infected ; again, fissures of the anus, a lacerated perineum, etc., might permit infection by germs passed with the feces. Absolute asepsis of all dressings is imperative, for one surgeon relates two cases where tetanus resulted from the application of dressings which had been laid upon the floor. All wounds, however trivial, should be disinfected and protected. When well-grounded suspicion of tetanus infection exists, after the ordinary scrubbing and disinfection employed for any infected wound, such remedies as hydrogen peroxid, a 1 per cent. solution of silver nitrate, tincture of iodin or iodoform powder, should be used. Dennis states that a ½ per cent. solution of iodin trichlorid should be tried, when procurable, because this " destroys the action of the toxins in less than an hour."

If a finger or toe be the part wounded it should be at once removed, if the vulnerating object can be shown to have tetanus germs upon it ; this is, of course, often impossible to do, but immunization by antitoxin should be effected in all suspicious cases, especially in localities where tetanus is known to be rife.

Hydrophobia.—This is an acute infectious disease never originating in man. It usually results from the bite of some rabid animal, as the dog, fox, wolf, skunk, or cat, although the saliva applied to a scratch, as by a dog licking the hand, may cause the disease. Although the saliva is the usual vehicle of the contagium, yet the other fluids or tissues of an infected body may, if applied to a wound, produce hydrophobia, as has occurred during a post-mortem examination. As the teeth of the animals mentioned are not grooved or hollow, as are those of venomous serpents, they are often wiped clean from any saliva, if the wound is inflicted through the clothing. This is especially true for those last injured if a number of other animals or persons have been bitten in rapid succession ; hence the greater danger of face and hand wounds. Skunks usually bite the face or hands during sleep, and in consequence, these animals convey the disease with great certainty. From 5 to 15 per cent. of those bitten acquire the disease where the bites are received in all parts and through clothing, etc. The mere fact that typical cases occur in infants disposes of the absurd contention of some that the disease is a psychic one, the result of fear and a knowledge of the symptoms. Because of the risk of this disease being conveyed to man by pet dogs during the incubation stage, it is important to study the symptoms of hydrophobia as exhibited by the animal.

Symptoms in the Dog.—After a variable period, if a bite has been received, there is fever, which is of course rarely detected. The animal is dull, and shows distinct evidences of mental disturbance ; he is shy, restless, hides himself ; has illusions shown by snapping at in-

visible objects and listening to sounds, neither of which have any existence. When caressed he will snap at the hand, and then suddenly fawn on his master, evidently having at first failed to recognize his owner. Additional proof of the altered mental state is shown by the tendency to roam unless chained, due to a desire to avoid association with either men or animals ; indeed, the sight of another dog will often give rise to an access of fury. Perversion of appetite is shown by eating coal, gravel, excrement, his own hair or tail. Free salivation is an early symptom, the fluid being thick, tenacious, possibly frothy. Owing to congestion, with edema of the fauces and pharynx, the bark is nearly always much altered, muffled, hoarse "ending with a peculiar howl." Partial paralysis of deglutition appears, rendering it hard to swallow, but a rabid animal will often plunge his muzzle beneath water in the effort to drink ; indeed, there is no *fear of water* in the dog, although, finding that he cannot swallow readily, he may abandon the effort. In one form the "furious" fever, rapid respiration, dilated pupils, and active delirium become pronounced, the animal, if roaming away from home, attacking all objects animate, or, occasionally, inanimate in his path; or, again, he may now for the first time start on his wanderings. Gradually increasing paralysis, commencing in the hind limbs and extending forward, succeeds the "furious" stage, the hind quarters dragging helplessly ; then all power of locomotion is lost, and the animal dies exhausted. The dumb form results from early paralysis of the muscles of the jaw, which drops, permitting a constant dribbling of frothy saliva ; the animal is "dumb" because he cannot bark without control of his jaw-muscles. During the feverish stage, and for some days preceding this, the saliva is infectious, which explains the cases of hydrophobia caused by the bite of a supposedly healthy dog which unfortunately has not been allowed to live long enough to demonstrate that it is really suffering from rabies. Death is almost

invariably the result in the dog, taking place in from twenty-four hours (rare), to so late as ten days (very rare), the majority perishing in from six to seven days. A few recover. The incubation period is very variable, lasting usually in the dog about six weeks, although it has recently been alleged to have extended to two hundred and seventy-six days. The possibilities of error are so great in such exceptional cases, that, unless the animal has been caged all the time, a reasonable doubt must exist whether a later inoculation has not occurred. In man the incubation varies from eight days to as many months, averaging between fourteen and thirty days. The microbe has never been isolated, but seems to locate itself chiefly in the central nervous system, although the virus is also found in the peripheral nerves and the salivary glands. Drying in the air will destroy the activity of the spinal cord in fourteen days ; 50° C. will do the same in one hour, as well as prolonged exposure to the direct rays of the sun, or to the action of a 1 per cent. solution of either carbolic acid or corrosive sublimate. Pasteur's theory of the causation and treatment is founded upon the, as yet, unproved hypothesis that rabies is a germ disease, the microbes locating and developing chiefly in the nervous system, and that either the germs or the tissues produce an antitoxin capable both of neutralizing the bacterial poison and destroying the specific microbes.

Post mortem, the changes are not very characteristic. In dogs the mucous membrane of the mouth and fauces is congested and coated with thick, tenacious mucus or mucopus, "often mixed with dirt." The mucous membrane of the respiratory tract may present similar appearances, minus the dirt. Congestion of the lungs, with subpleural ecchymoses, is common. The stomach is usually filled with a miscellaneous collection of feces, stones, coal, etc., unless the animal has been prevented from securing anything but its food. The upper portion of the small intestines also is apt to contain similar

objects. Intense hyperemia and swelling of the mucous membrane of the stomach exists, with extravasation of blood and erosions. The same covering of thick, tenacious mucus is seen. The small intestines present minor degrees of the same conditions. Marked hyperemia of the kidneys is present, and, possibly, small ecchymoses in the floor of the fourth ventricle. Sometimes all these conditions are practically absent. In man definite, gross lesions are absent, the appearances presented being those common in an acute infectious disease ; as Biggs says, there is " possibly more marked congestion of the mucous membrane of the alimentary and respiratory tracts, and more hyperemia of the brain and spinal cord and their meninges, than is usually present in other types of infectious disease." Sections through the bulb or cervical division of the cord show microscopically decided hyperemia, with perivascular cell-proliferation and accumulation. Minute nodules, composed of embryonal cells surrounding degenerated or proliferating nerve-cells, are said to be almost diagnostic when occurring in the nuclei of the glossopharyngeal, pneumogastric, and hypoglossal nerves. Hyperemia, edema of the membranes, occasional hemorrhages around a few vessels, thrombosis or obliteration of the minute vessels of the gray matter from hyaline degeneration; blocking of the same by hyaline pigmented material or leukocytes ; proliferation of the lining cells of the central canal of the cord ; hemorrhages into the gray matter and small foci of degeneration in the same, have all been described. According to one writer, mononuclear, and less often polynuclear, lymph-cells " invade the protoplasm of the cell and fill the pericellular lymphatic spaces, dilating them to form nodes." Apparently the nerve-cells themselves proliferate, several smaller cells replacing one larger one ; or more extensive, evenly distributed degeneration takes place. These changes not only involve the gray matter of the centers of the nerves already mentioned, but also the cerebral cortex, thus accounting

for the hallucinations. The changes induced in all these centers diminish their power of control over reflex irritations, death eventually resulting from *exhaustion-paralysis* of these nerve centers—the maintenance of whose functions is so essential to life—because they are no longer capable of responding to the vital reflexes.

Symptoms.—*Prodromal Stage in Man.*—During the incubation period the healed wound may itch, become painful, swollen, and cyanotic; very rarely vesicles form. Darting neuralgic pains starting from the wound are sometimes complained of ; gastric disturbance and mental depression are the rule ; hyperesthesia is marked, and undue irritability of the special senses is shown by the distress caused by ordinary light and sounds. Chilliness may be felt, while the bodily temperature is elevated one or more degrees. A peculiar catching of the inspiratory act, due to incipient spasm of the diaphragm, next appears.

Stage of Excitement.—Stiffness and pain are experienced in swallowing, soon followed by spasmodic contractions of the musculature of the pharynx, and to this is added pronounced spasm of the diaphragm, which before existed only in its incipiency. Excited at first by actual attempts to swallow, by association of ideas aided by the increased sensibilities of all the reflexes, the sight or sound of pouring water, a sudden sound, bright light, touching the skin, etc., will now start the paroxysms of choking and dyspnea, during which attempts to hawk up and expectorate the tenacious mucus which cannot be swallowed take place, causing sounds which it is imagined resemble the bark of a dog. In the intervals irregular, spasmodic action of the diaphragm causes distressing dyspnea. Delirium is absent at first, but later is often present, especially during the paroxysms, when the patient may become maniacal, even attempting to do violence to others. Vomiting is common, and incontinence of urine and feces is not rare, the urine being apt to be albuminous. Moderate pyrexia is the rule,

although even 105° F. has been observed. This stage may last from a few hours to ten days, averaging, however, from four to five days.

Paralytic Stage.—For a variable period before death, the paroxysms diminish in frequency and severity, swallowing now often becomes possible, delirium disappears, and a delusive calm comes on ; but the pulse becomes rapid and weak, death resulting possibly in coma—the end being often preceded by a varying degree of paralysis which usually commences in the muscles of mastication and deglutition.

Prognosis.—Death is almost invariable in genuine rabies, but as only about 5 to 15 per cent. of those bitten in all parts of the body by all varieties of animals capable of conveying the disease acquire hydrophobia, the disease is a rare one.

Diagnosis.—This is far easier in practice to one familiar with tetanus, hysteria, and cerebral affections, than is generally believed. In a well-marked case of hydrophobia, the great mental excitement, the delirium, the intense hyperesthesia, the characteristic respiratory spasms steadily increasing in " frequency and intensity," the free secretion of tenacious mucus, the illusory improvement before death, with paresis or actual paralysis, and the absence, post mortem, of " gross lesions sufficient to account for the symptoms during life," and the microscopic finding already described, should suffice for a correct diagnosis. The history of a bite, especially if healed for weeks, and not anywhere in the distribution of the fifth pair of cranial nerves is important corroborative evidence, because tetanus never occurs *weeks after* the healing of a wound, and the only form likely to be confounded with hydrophobia, the " tetanus hydrophobica," never occurs excepting when the injury involves the distribution of these nerves. Subdural inoculation of rabbits with portions of the cord will settle the diagnosis.

Treatment.—This must be prophylactic. Excise when possible the infected tissues for some distance around the

wound, and preferably cauterize them afterward with the hot iron, although other caustics will serve. If excision is impracticable, use the hot iron or any other caustic available. When possible, employ Pasteur's prophylactic vaccination. As this cannot possibly be done outside of specially equipped hospitals, a minute description of the method would be here out of place. Suffice it to say that rabic virus, with a fixed period of incubation, is secured by subdural inoculation of rabbits; that the virulence is attenuated by drying over caustic potash at about 73° F.; and that daily inoculations are practised with virus of increasing potency until, by the fourteenth day, a cord dried for only three days is employed. The use of a too active cord may produce fatal paralytic rabies, as sometimes happens when treating bites about the face by the "intensive method." To be successful, the method must be inaugurated early, before the poison gains a firm foothold in the central nervous system. After the disease has commenced, palliative treatment on general principles, similar to that advised for tetanus, must be adopted.

Prophylaxis.—If proper laws be enforced, requiring the registering and muzzling of all dogs, this disease could be readily stamped out; where wolves abound, this source of hydrophobia must always exist.

The treatment of supposedly rabid dogs which have bitten men or animals must be considered. All suspected animals should be confined for at least two weeks, while those bitten must be either kept *in confinement* for at least six weeks, or killed. When an animal cannot be kept alive long enough to determine whether it is or is not rabid, it should be killed and an emulsion of its brain and spinal cord be injected beneath the dura of several rabbits, who will soon develop hydrophobia if the dog has been rabid. By a resort to this procedure, the intolerable anxiety of the patient can either be relieved or the necessity for inoculation be demonstrated.

LECTURE XXIII.

GLANDERS, FARCY ; ANTHRAX, OR MALIGNANT PUSTULE.

Glanders, Farcy.—This is a specific, infectious, mycotic disease, caused by the Bacillus mallei, which induces the rapid formation of nodules composed of a species of granulation-tissue, in the skin, subcutaneous tissue, and the mucous membrane of the nares and that of the respiratory passages. These nodules are peculiarly prone to degenerate, liquefy, and rupture, leaving ulcers with undermined edges surrounded by an extensive area of phlegmonous inflammation. Although these lesions are at first chiefly located in the skin and respiratory mucous membrane, later by continuity, by the lymph-stream and by vascular embolism, dissemination takes place, the viscera, muscles, bones and joints, becoming involved, sometimes by continuity, sometimes by metastasis. Pyemic lesions are also common late in the disease. Usually acquired from the germs contained in the nasal discharges of glandered horses, this is not invariably the case, as the source may have been another human being, possibly sheep or goats ; the disease has also been contracted in the laboratory from careless handling of cultures of the Bacillus mallei. Practically, whatever may be possible from a theoretical standpoint, glanders is commonly acquired through some open but trivial lesion of the skin or a mucous membrane, as the conjunctiva, or that of the nose or mouth. Coachmen are apt to dust harness or polish the horses' coat here and there with their handkerchiefs; hostlers and those about stables often drink out of horse buckets, thus explaining

the infection. The wind will carry the nasal discharges when a horse snorts, into the eyes and face of the driver, and in the same way infection of animals grazing in a field separated by a lane from the diseased animal has been reported. Very rare in this country, glanders was found among the army horses during the late war ; it is not uncommon in Cuba, hence must be carefully studied. It has been experimentally produced by rubbing the germs into the intact skin.

Symptoms in the Horse.—A brief account of the disease as seen in the horse is advisable as a possible aid in diagnosis and an important one for prophylaxis. The nasal secretion is first slightly increased, possibly only from one nostril, but it constantly flows, is watery, mixed with a small amount of mucus, later becoming peculiarly viscid, "gluey," as it is termed. The quantity rapidly increases, soon becomes bloody, and in the later stages offensive. In very exceptional cases, this "gluey" discharge may persist for long periods without other symptoms. The submaxillary lymphatic glands early become enlarged on the side of the diseased nostril. At first a diffused swelling is formed, but later most of this subsides, leaving one or two hardened glands firmly adherent to the jaw-bone. The nasal mucous membrane presents a cyanotic hue, with probably circular, deep ulcers with abrupt, prominent margins situated upon the cartilaginous septum. These ulcers increase, finally so obstructing the nasal cavities that a peculiar "grating" or "choking" sound is heard during respiration. Later, owing to involvement of the frontal sinuses, the skin covering them becomes thickened and tender ; the facial, cervical, and general lymphatic vessels become diseased, constituting "farcy," the enlargements produced by the valves or interposed nodes forming the so-called "farcy buds" ; these soon ulcerate. The lymphatics of the extremities are attacked, causing great swelling, heat, and disability of the limbs ; ulcerations occur here also. Loss of flesh, appetite, strength, and

urgent cough supervene, the coat is roughened and staring, and death occurs from exhaustion. When infection has taken place elsewhere than in the nasal cavities, the hardened, nodular, tender, inflamed lymph-vessels about the lips, nose, neck, axillæ, and thighs, followed by ulceration, producing rounded sores with elevated, indurated borders and pallid bases, combined with loss of appetite, flesh, and staring coat, present a characteristic picture. A temporary improvement sometimes occurs, generally to be followed by a relapse, generalization, and death. The discharges from all ulcers are quite as infective as those from the nasal cavities, and will give rise to glanders when properly inoculated. All animals presenting either of the foregoing sets of symptoms should be destroyed and their carcasses burned. The mangers, racks, and partitions of the stall occupied by the diseased animal should be scraped down, scoured with plenty of soft soap and hot water, and coated with a thick layer of chlorid of lime mixed with water. Although whitewashing of the walls is considered sufficient, chlorid of lime had better be also used for this purpose. All head gear and harness of the affected animal must be destroyed, and wooden drinking pails ; those of metal, and all iron work, can be disinfected by fire and paint.

Symptoms in Man.—The incubation period in acute cases varies from three days to eight days, although it may be longer. Malaise with febrile symptoms, then shiverings, pains in the limbs, perhaps rigors and sweats, are noticed. If internal infection has taken place, "the gastro-intestinal disturbance, the fever, and the general prostration, may cause the case for a time to simulate typhoid fever;" but the development of the external lesions will soon correct this error, assisted by the bacteriological and other tests now at our disposal for the determination of typhoid fever. When accessible to the eye, the point of primary infection gives evidence of a more or less distinct phlegmonous cellulitis ; in addi-

tion, there is probably inflammation of the lymph-vessels and nodes, and possibly there is also phlebitis. Vesicles soon form over the diseased areas, hemorrhage takes place into them, and suppuration promptly follows, phagedenic ulcers resulting. When primarily situated in the nasal cavities, extensive destruction of both the soft and the hard parts of the nose, palate, and mouth occurs. If originating in some other part, after the premonitory constitutional symptoms, secondary nodules appear, usually during the second or third week, in the nasal chambers, giving rise to, first a thin, then to a gluey, and finally to a mucopurulent, often bloody and offensive discharge from the nostrils. The conjunctivæ may also be attacked, marked edema of the eyelids resulting, causing, with that incident upon the nasal and buccal lesions, generalized facial edema and a peculiar shiny, dusky-red appearance of the face and neck, extending to the scalp. Multiple skin and mucous membrane lesions may occur within the first few days, but are more apt to be delayed until the period mentioned. As any mucous membrane may be attacked, that of the lungs or intestinal tract often becomes diseased. Severe pain in the muscles of the extremities is common. A very hard, papular or nodular generalized skin-eruption, not unlike that of small-pox, especially marked upon the face, appears in from a few days to the second or third week. This soon becomes pustular, often confluent, giving rise to irregular ulcers "encrusted with a soft, brownish, sloughy coating." Occasionally the eruption is vesicular from the outset, resembling that of varicella. In many cases "acute farcy" is superadded—*i. e.*, specifically inflamed lymphatics forming tortuous, knotted cords, with enlargements at the site of the valves. The lymph-nodes are also involved, and suppuration of these and the lymph-vessels with diffuse suppuration in the limbs is apt to follow. Distinct and early symptoms of sepsis are shown in acute cases, and in all forms, later. Delirium sets in with stupor, sooner or later, and a

typhoid state and death result, sometimes accelerated by bronchial, pulmonary or pyemic accidents. In chronic cases, the lesions develop more slowly and are less numerous. They may gradually improve, ulcers and abscesses healing, and recovery ensuing after many months. Nevertheless, chronic glanders, after progressing slowly for long periods and involving chiefly the lymphatics, sometimes terminates in acute glanders.

Diagnosis.—Early in the disease, rheumatism and typhoid fever, and, later, pyemia—which, indeed, often complicates glanders—must be excluded. The peculiar exanthem, the coincident nasal discharge, the peculiar ulcers, and the dusky-red, glossy, edematous swelling of the face, head, and neck, especially if the lymph-vessels and nodes are diseased as has been described, should render a clinical diagnosis possible. Bacteriological examination and inoculations of susceptible animals will render the diagnosis positive.

Prognosis.—Acute glanders is probably always fatal in from three days (very rarely) to several weeks. As just said, a certain number of chronic cases recover after much disfigurement and crippling.

Treatment.—Prompt destruction of diseased animals and of everything which may be soiled by the discharges is imperative as a prophylactic measure. Thorough disinfection of any accessible point of primary infection by excision and the actual cautery should be tried. Secondary nodules and abscesses must be opened, curetted, the hot iron freely used, or zinc chlorid solution, 1 part to 8, be applied ; moist antiseptic dressings should afterward be employed. In all other respects the surgeon must be guided by the general principles applicable to the treatment of any septic and infective process accompanied with exhausting discharges. Mallein has not yet been applied to the treatment of glanders in man; hence its therapeutic value, if any, is unknown.

Anthrax (*Malignant Pustule ; Charbon ; Wool-sorter's Disease*).—This is an acute, infectious, usually non-

suppurative, microbic disease, produced by the spores or adult organisms of the Bacillus anthracis. The point of entrance for the germs may be the respiratory or the alimentary tract, but most commonly is some slight external traumatism, perhaps the bite of a fly, whose proboscis serves to convey the infective material with which it has become covered by feeding upon the bodies of animals dead from anthrax ; fetal infection through the placenta has also been reported. The destructive effects of the Bacillus anthracis are both local and systemic, the large size of the organisms serving to block the capillaries of a part, thus determining gangrene ; while the toxic substances generated produce fatal systemic poisoning. The spores are among the most resistant of those produced by any pathogenic organism. Many interesting facts, related in any work on bacteriology, must be omitted as not pertinent to our present studies, but some are of importance to bear in mind because diagnostically suggestive. Thus, anthrax is prevalent in Russia, Siberia, and especially in portions of Hungary, France, and Germany. While not common in the United States, it is by no means unknown. In South American countries, whence many hides come, and also wool and horse-hair, this disease is quite prevalent. Drying does not destroy the virulence of anthrax germs, which, when desiccated, retain their vitality for years. In this country, infection usually occurs from handling the hides of infected animals, or sorting wool or horse-hair from animals who have died of anthrax, hence the origin of one of the names, "wool-sorter's disease." Therefore, butchers, wool-sorters, and curriers are the classes of patients who may reasonably be suspected of suffering from anthrax when presenting suspicious symptoms.

Symptoms.—Wool-sorter's disease belonging to internal medicine will not be considered here. In anthrax proper, after a short incubation of from one to three days, a small papule—there may be more than one point of infection—is noticed upon the face, hands, or arms,

which quickly gives rise to discomfort. Soon a vesicle forms, and the papule becomes surrounded with a constantly increasing area of cellulitis and widespread edema. The central vesicle ruptures, showing beneath a gangrenous, blackened mass, while an encircling ring of secondary vesicles forms around the primary one. In most instances cellulitis, infiltration, and sloughing continue to spread, lymphangitis and phlebitis develop, and rapid toxemia and death result. In a very few cases the infiltrated, gangrenous areas separate, leaving imperfectly granulating surfaces, which eventually heal. Pain is not a prominent symptom, and systemic symptoms are mild while the disease remains local, usually consisting of slight chills and a little fever. In fatal cases, death is usually the result of toxemia, although thrombosis of the cerebral sinuses by extension of the process through the facial vein, and various pyemic and septicemic accidents, may prove lethal. When systemic intoxication is well marked, rigors occur with high fever and great weakness, general infection is present, and these symptoms are succeeded by delirium, weak pulse, sweating, diarrhea, and more or less generalized pain. Pulmonary disturbance is evidenced by cough, dyspnea, etc. Sudden death in collapse is quite common. An edematous form of anthrax has been described, in which a diffuse edema rapidly spreads from the point of infection, the superjacent skin being of a livid color. The degree and extent of this edema is sometimes enormous. Blebs form at various points over spots of local gangrene of the skin and cellular tissue. Suppuration, as in ordinary anthrax, is unusual and the result of secondary infection.

Post mortem, the lymph- and blood-capillaries are found blocked by bacilli, which produce gangrene of the tissues partly by directly cutting off the blood-supply, partly by causing such an outpouring of exudate that the remaining vascular supply is destroyed by the strangulation thereby induced. Sepsis often having been present, the usual evidences of this will be found.

Prognosis.—With early diagnosis and energetic treatment, external anthrax is quite manageable, but if general infection has taken place, death is inevitable, and the prospects of recovery are poor indeed if treatment, however appropriate, is commenced late.

Diagnosis.—The central bleb beneath which is a gangrenous patch of skin, the ring of secondary vesicles, the firmly attached sloughs, the lack of pus primarily, the comparative absence of pain, the widespread infiltration, and later, the general toxemia, can hardly be confounded with the symptoms of any other ailment. The differentiation from a disease, already described when speaking of gangrene, will become apparent when the symptoms of "malignant edema" are reviewed; but in passing, it may be said that the rapid disintegration and liquefaction of the tissues seen in the latter disease stand in sharp contrast with the dense, adherent sloughs characteristic of anthrax. In any suspicious case the use of the microscope or inoculation of a mouse with a little of the wound-fluids will settle the question beyond all cavil.

Treatment.—When possible, the infected tissues should be removed with the knife, cutting widely through the healthy tissues, and, to prevent their infection, the fresh surfaces should be seared with the hot iron, be wiped over with pure carbolic acid, or a solution of zinc chlorid, 1 part in 8. The actual cautery, freely used, will often alone suffice, if the infected area be not too large to be readily destroyed by this means. When excision is impracticable, deep crucial incisions freely cauterized with the hot iron or pure carbolic acid have been successfully employed, these measures having been reinforced by the injection of a 3 or even 10 per cent. solution of carbolic acid by means of a hypodermic needle, so employed as to reach beneath the deeper portions of the infected area and well beyond its spreading borders. The diseased tissue should be also thoroughly infiltrated with the same solution. Subsequently, dress-

ings wet with a 1–1000 solution of mercuric chlorid should be applied, over which an ice-bag had better be placed, because the bacillus grows feebly below 20° C. (68° F.), and ceases to develop below 12° C. The injections may be repeated every six or eight hours, until the process ceases to extend or symptoms of carbolic-acid poisoning appear, an accident which must be carefully guarded against, by frequent examinations of the urine and scrutiny of the patient's condition. Taking advantage of the fact that the bacillus ceases to develop at temperatures above 42° to 45° C., hot poultices have been employed with decided benefit at 50° to 55° C., reapplied every ten minutes, in conjunction with repeated injections of a 3 per cent. solution of carbolic acid.

The strength of the patient must be maintained by the free use of alcoholic stimulants, strychnin, digitalis, abundance of nutritious food, etc. The secretions of the skin, kidneys, and intestines must be maintained and regulated, and a proper amount of sleep must be secured.

LECTURE XXIV.

ACTINOMYCOSIS ; TUBERCLE ; COLD ABSCESS.

Actinomycosis.—This is a subacute or chronic ailment caused by a variety of ray-fungus, which by its growth in the tissues produces granulation-cell tumors, often in the past mistaken for sarcoma. In man, suppuration is the rule, resulting usually from a mixed infection, while in animals the appearance of pus may be long delayed. The initial lesions in animals are usually situated in the buccal cavity, due to injuries
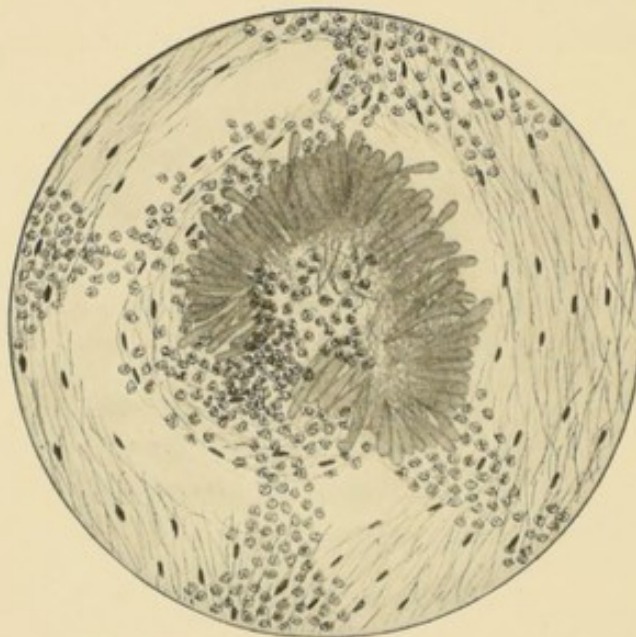
FIG. 12.—Actinomycosis, showing nodule. From a case of actinomycosis in man.

received during grazing, whence the respiratory tract may become infected by inspiration of, or the alimentary by swallowing, the actinomyces. Because the jaw is so often attacked in animals, the disease in them is popu-

larly known as "lumpy-jaw" or "swelled-head." Owing
to the habit so common among farm-laborers of chewing
pieces of grass or beards of grain, infection in man also
frequently occurs in the mouth, often through a carious
tooth. The "Madura-foot" of India is now believed to
result from a variety of actinomycis. The actinomycetis
granules found in the tissues or pus are pin-head in size,
or may be aggregated, forming masses the "size of a
pea," or they may be microscopic. They present vari-
ous appearance—*i. e.*, "grayish and translucent like
sago particles, or opaque, or white, greenish-yellow or
yellow, brownish, even black." They are soft and
gelatinous when young, but become harder later, and
"gritty" from calcareous degeneration. In suppurating
cases these bodies may be so scarce in the pus as to re-
quire long search to find any, in other cases they are
innumerable. The granules perceptible to the eye are
"colonies or clumps of colonies." These colonies are
composed of a central portion formed of rods and threads
branching dichotomously, which become densely inter-
woven more peripherally, and radiating, terminate in
bulbous enlargements ; this bulbous enlargement, which
is not always present, is, with calcification, a degenera-
tive change. Although secondary pyogenic infection
is usually the cause of suppuration, it has been claimed
that the actinomycis is itself pyogenic. As the granula-
tion-tissue formed by the action of this fungus is, as a
rule, permeated with bacilli and bacteria, this proposition
is difficult to prove. The first histological changes caused
by the presence of the parasite are " necrosis and lique-
faction of tissue with emigration of white blood-corpus-
cles," to which succeeds the formation of "vascular
granulation-tissue."

Diagnosis.—Although resembling in many respects
sarcoma, the free suppuration, with the detection of the
characteristic bodies in the discharge, should prevent
mistake. The possible simulation of sarcoma, carci-
noma, tubercle, and syphilis, by actinomycosis, especially

in those dealing with cattle, ought to suggest an examination of the discharge, all the more if investigation reveals that "lumpy-jaw" has been noticed among the animals with which the patient has been brought into contact. Again "metastases do not appear to occur in the lymphatic nodes communicating with a primary focus of actinomycosis," although pyogenic infection may simulate this; this absence of lymph-involvement is more suggestive in the cervicofacial region than elsewhere, because sarcomatous tumors are here more apt to secondarily involve the lymph-nodes, while in carcinoma of these parts, early infection of the nodes is the rule.

Prognosis.—If thoroughly accessible, the prognosis is good; if in regions where radical measures are impossible, the reverse is true, especially if secondary visceral involvements have taken place. Still, being a chronic, almost painless, ailment, destroying by slow infection producing exhaustion, much can often be done.

Treatment. — Complete extirpation by the knife, curet, and cautery will often cure. An affected jaw or tongue should be totally or partially excised. Even actinomycosis of the lung, pleura or liver, *when accessible externally* may be benefited by free removal of the diseased structures. Great advantage will accrue in some cases from the internal use of large doses of potassium iodid in conjunction with potassium iodid gargles, and the local action of iodin introduced into the tissues by cataphoresis.

Tuberculosis. — Tuberculosis is a chronic or subacute, very rarely an acute, microbic disease caused by the action of the Bacillus tuberculosis, which induces the formation of a peculiar, low-grade, granulation-tissue. So long as the bacilli and their products are active, the tubercular process extends peripherally, while in spots, usually those more centrally located, coagulation-necrosis occurs, followed by caseation and often liquefaction, although calcification may take place:

these changes are favored by the relative or absolute avascularity of the older—*i. e.*, the more central, portions of the tubercular masses, but probably directly result from the action of the germ-products. Although some patients, presenting certain anatomical peculiarities, are prone to develop tubercular lesions, and these are often said to be "strumous," I have maintained for many years that all active, so-called "scrofulous" or "strumous" manifestations are really tubercular and should be so described. A "scrofulous" or "strumous" diathesis therefore does not exist, what is so termed being either an expression of active tubercle, or is merely indicative of that anatomical structure of the tissues which is most favorable for the arrest and multiplication of tubercle bacilli. None can deny that those patients with flabby muscles, rounded limbs, thin, blue-veined skin, brilliant coloring, finely chiselled features, grey, blue, or even dark, humid eyes with sluggish pupils shaded by long lashes, fine auburn or blonde hair —possibly dark hair—and tumid upper lips, are more apt to succumb to tuberculosis, when exposed, as are also those with coarse reddish hair, thick muddy complexions, coarsely modelled features, a tendency to various eruptions and to chronic inflammations of the conjunctiva, eyelids, etc. Nevertheless, tubercle is frequently seen in those who present none of the characteristics mentioned. Tuberculosis seems to require a conjunction of the following favoring conditions : A lowering of normal cell-resistance, either peculiar to the individual or the result of an injury so slight as not to invite the accumulation of numerous phagocytes, yet severe enough to promote the development of such a hyperemia as will mechanically favor the local aggregation of germs ; these may be introduced at the point of traumatism, but more usually reach the *locus minoris resistentiæ* by the circulation, having gained access thereto at some distant infection-atrium where the germs have failed to effect a permanent lodgement. Thus gonorrhea is well known to

favor the development of tuberculosis of the testicle and female genital tract, probably by damaging or destroying the resistance of the surface-epithelium.

Such a large proportion of cases in surgical practice depend upon tubercular infection or its sequelæ that a comprehensive survey of this disease is requisite, although detailed descriptions of the many phases assumed would be out of place here, belonging to the practice, not to the principles, of surgery.

As you have elsewhere learned all that is essential concerning the tubercle bacillus and the histology of tubercle, it would be superfluous to enlarge upon these points. A study of the natural terminations of tuberculosis shows that at times nature is equal to the task of cure, and this study serves the practical purpose of providing us with valuable suggestions as to how we can best aid, not thwart her. Degenerative changes leading to massive necrosis and separation of the diseased tissue is seen in lupus, and more rarely in an ordinary tubercular focus, when, after caseation, evacuation of the liquefied tissue occurs, the thin environing layer of tubercular tissue either being destroyed by pyogenic infection producing sloughing, or the consecutive hyperemia providing sufficient phagocytes to successfully contend with the few remaining tubercle bacilli. Tubercular tissue consists of a modified granulation-tissue, and if the bacilli or their products which cause necrobiotic changes in the component cells, are either removed or the latter chemically neutralized, this granulation-tissue, like any other, will develop into scar-tissue ; this is often observed occurring either spontaneously or as the result of treatment. In the former case, the resistance of the tissues becomes increased so that the germs are destroyed and the effects of their toxins prevented; in the latter, substances inimical to the life of the bacilli are brought into contact with them, or chemical changes are effected in the toxic substances rendering them inert : probably both results are secured. It has been explained that the local spread of

a tubercular focus depends upon peripheral infiltration
of the tissues. If the infection be slight, the soil poor,
possibly because exceptional phagocytosis is excited and
thus the resistance of the environing tissues increased, a
non-tubercular envelope of granulations may form which
develops into scar-tissue encapsulating the tubercular
nodule, a temporary, possibly a permanent cure result-
ing. This encapsulation may persist indefinitely, the
tubercle becoming absorbed or converted into scar-tissue,
or it may become obsolete by one of the changes to be de-
scribed. Again, after long quiescence the tubercular
process may become active, the capsule becoming con-
verted into tubercular tissue, and the disease resumes its
interrupted march, perhaps after many years of apparent
cure. Caseation, as has already been said, results partly
from lack of blood-supply in the tubercular tissue, chiefly
from chemical changes following the coagulation-necrosis
induced by the bacillus and its poisons. Although the
bulk of the tubercular tissue is converted into a yellowish,
cheesy, friable mass, yet, at the periphery, living miliary
tubercles surround it, forming, with a low-grade young
connective tissue, the mistermed " pyogenic membrane,"
which would be more properly termed " tuberculogenic,"
because, if not destroyed, the specific process will con-
tinue to maintain itself, or spread from this " membrane "
as a center. It must not be thought that the degene-
rated caseous mass is harmless. Far from it. However
difficult it may be, at times, to detect tubercle bacilli in
liquefied tubercle by the microscope, let caseated tuber-
cle be inoculated into a susceptible animal, and no doubt
will be entertained of its virulence nor that it contains
the germs of tuberculosis. It is of great importance to
recall this fact when operating, that all possible pre-
cautions may be adopted to prevent the inoculation of
the fresh surfaces of the wound by contact with caseated
materials or with tubercular pus.

Calcification.—Under certain circumstances lime salts
are deposited in tubercular foci, the process usually com-

mencing in the central portions of the giant-cells, whence
it spreads.   Considerable masses of tubercular tissue may
be thus rendered obsolete.

Cold Abscess (*Chronic Abscess ; Congestive Abscess*).
—Abscess is nearly always a misnomer for this condition,
the fluid contents, while resembling pus, being usually
in reality only caseated tubercular material which has
become liquefied.   Although it has been contended that
under especially favorable conditions tubercle bacilli can
produce suppuration unaided by any other organisms, in
practice, when genuine suppuration occurs in a tuber-
cular focus, secondary pyogenic infection is the cause.
It is a well-known clinical fact that a cold abscess may
present not a symptom in common with acute abscess
beyond a gradually increasing accumulation of pyoid
fluid which will spontaneously evacuate itself, and that
again, a cold abscess of large dimensions may suddenly
appear, and present all the symptoms of an acute affec-
tion ; yet, when the contents are evacuated, it becomes
clear that the greater part of the process was one of long
standing, most of the fluid being not pus but liquefied
caseated tubercle.   Although cold abscesses may be found
in any organ or part, as the brain or any other viscus,
they are vastly more common in connection with
lymphatic, articular, or osseous tubercular disease.

As already stated, a cold abscess is surrounded by a
layer of ill-organized neoplastic connective tissue which
contains tubercular tissue in greater or less amount,
usually in the form of miliary nodules, some of which may
present evidences of caseation.   This "membrane"
results from the invincible tendency of granulation-
tissue, however produced, of forming scar-tissue, and, in
this instance, is developed in the surrounding layers of
tissues which, although intensely hyperemic, are not
infiltrated with tubercle or are but slightly infected.
This—so incorrectly called a "pyogenic membrane"—
if in any sense an active factor in the process, serves
merely to aid in the circumscription of the tubercular

process, and if genuine suppuration be present, to limit this. A similar membrane—except the tubercular tissue —is seen lining old sinuses, as explained when speaking of sinus and fistula, and it is incorrect to make the presence of a so-called "pyogenic membrane" a determining point as to the presence of a tubercular process, as has been unfortunately done by a recent author. Of course, in a tubercular process this "membrane" is more marked than in non-specific conditions, yet it cannot be considered a diagnostic point, but merely a cause for suspicion when strongly marked, especially in a *relatively acute process.* Despite the partial protection afforded by the tubercular membrane, this is a constant menace, and to secure a local cure it, or the contained bacilli, must be destroyed. Two different courses may be pursued by any tubercular focus which undergoes caseation. In one, the caseated material, whether first distinctly undergoing liquefaction or not, has its fluid portions gradually absorbed until nothing but a relatively small putty-like or friable, cheesy mass is left ; or small areas of the same are interspersed in an irregular mass of scar-tissue, all being isolated from the surrounding tissues by cicatricial tissue. This latter appearance results from the partial conversion of the specific granulation-tissue into cicatricial tissue, or a similar change in the remains of the uninfiltrated environing normal tissues, the bacilli either being destroyed or *inhibited.* This statement contains a pregnant truth—*i. e.*, the process may only be quiescent, ready to break out anew if anything, like a slight traumatism or deterioration of health, lowers the resistance of the tissues. Relapses may thus occur a score of years after apparent cure, often resulting in the formation of the "residual abscess" of Paget. In such cases, within a very few days, a large collection of pyoid fluid may occur, which is chiefly composed of liquefied caseated material which has lain dormant for years, the immediate cause of the change being usually a recent pyogenic infection. When these

"residual abscesses" are of more gradual formation, it is possible that the intense hyperemia following the traumatism inflicted, say by "brisement forcé," may supply sufficient fluids to cause liquefaction of the old solid caseated mass, but this is probably very rare, genuine pyogenic infection usually producing the change.

The contents of a typical cold abscess consist of a yellowish, whey-like fluid, containing, suspended, more deeply tinged particles or masses of "curdy material," which, when examined, are found to consist of numerous caseated cells entangled in what resembles altered coagulated fibrin. In such cases, not a single pus-cell can be detected, nor do pyogenic organisms develop after the most careful culture experiments. It is futile to say that these organisms did exist but that they died for want of pabulum, the tubercle bacilli proving the more resistant. This is a mere clinging to antiquated notions, the liquid contents of the so-called abscess resulting from degeneration of tubercle, and no pyogenic organisms are detectable because none ever were present. In one class of cases of cold abscesses, the caseated tubercle liquefies, more fluids accumulate, thus gradually enlarging the cavity, while at the same time the process extends peripherally, until the superjacent structures including the skin are converted into tubercle. These infiltrated tissues in turn caseate and break down, spontaneous evacuation taking place. In the remaining class, secondary pyogenic infection supervening, symptoms of acute suppuration are noticed, evacuation resulting from rapid pyogenic destruction of the infiltrated tissues.

Symptoms.—When accessible, from the first there is an area of induration detectable, which softens without any inflammatory phenomena, while at the same time enlarging. If not readily accessible, the first symptoms may be those of nerve-irritation from pressure, the pain being referred to the terminal distribution of one or many branches of a plexus. In many cases, no symp-

toms are noticed indicative of an abscess until a more or less distinctly fluctuating tumor is discovered in the groin, the pharynx, the lumbar or some other region, although the patient may have complained of pain or soreness in these localities, leading to a futile examination only a short time before. There are usually present evidences of some chronic osseous or articular lesion, or a history of the existence of some such in the past. The skin is not reddened nor otherwise altered, except when the accumulation becomes superficial, when the cutaneous veins become enlarged and the skin bluish or dusky. If pyogenic infection now supervenes, the ordinary appearances of an acute abscess are present in varying degrees. When secondary infection does not take place, the skin becomes purplish and gradually thinned until it gives way, the originally small opening rapidly enlarging to form an irregular, ragged hole. Of course, suppurating joints, diseased bone, or softening lung-tubercle, may coincide, and produce constitutional symptoms, but otherwise there are usually no constitutional symptoms in cold abscess until the accumulation has been opened and *infection has taken place or this accident has preceded the evacuation*, when rapid death may result from sapremia; later, hectic fever and lardaceous disease of the viscera may be caused by the prolonged suppuration. Sometimes a deterioration of the general health precedes the detection of large cold abscesses, no adequate cause being discernible until the softening tubercular focus is detected. If deeply seated, cold abscesses may be mistaken for cysts, or, earlier, for fatty tumors, differentiation often being impossible except by aspiration or the use of the exploring needle. The associated presence of old osseous or other possible sources of tubercular disease when a slowly forming mass is detected, even far distant from these lesions, although all suspicion of fluctuation is absent, should suggest cold abscess, especially if the general health be causelessly deteriorating. When no tumor is detectable,

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increasing or recurring neuralgic pains in the parts distant from any perceptible lesion should give rise to the suspicion of abscess, especially if the health is failing without pyrexia or any reasonable explanation. The detection of evidences of old lymphatic, chronic articular, or osseous lesions, even if apparently cured, will give still more color to this suspicion. The differential diagnosis between cold abscesses located in certain regions, as the groin, and other conditions, as aneurysm or hernia, with which they may be confounded cannot be discussed here but must be sought in treatises dealing with the practice of surgery demanded in each special region.

LECTURE XXV.

TUBERCULOSIS (CONTINUED) : METHODS OF INFEC-
TION ; DESTRUCTIVE PROCESSES ; TREATMENT.

Diagnosis.—This is involved in the description of the symptoms and course of tubercle as it is shown in various organs and parts ; in doubtful cases the injection of tuberculin may be tried.

No attempt will be made to minutely consider the endless manifestations of tubercular conditions, modified as they are by locality and anatomical structure ; but there are some peculiarities common to the disease, wherever located, which require mention. Wherever the tubercle-germ multiplies, there results a form of granulation-tissue, which, as has been said, is formed at the expense of the tissue-elements. This tissue is deficient in vascularity, and therefore predisposed to undergo degeneration, the inherent tendency to development into a permanent tissue—scar-tissue—which it possesses in common with other granulation-tissue being repressed, as it were, because of lack of pabulum ; still further, so long as the bacilli multiply, there is a steady increase in the amount of the toxic substances which are attacking and will eventually destroy the vitality of the feebly nourished cells : nevertheless, the tendency to repair, although partially thwarted, *is present*. Tubercular granulation-tissue always extends by *infiltrating the surrounding tissues*, destroying their individuality, and, therefore, not only their vital but also their physical functioning powers. Thus, in osseous tissue the lime salts disappear, and the once dense, resisting, compact bone, or the cancellous tissue so capable of

resisting a crushing force, alike disappear, producing many of the characteristic symptoms of tubercular bone- and joint-disease, because physically incapable of normal function. The head of the femur and the cup of the acetabulum, once smooth and mutually adapted, irregularly disappear; the firm bodies of the vertebræ, capable of sustaining the superincumbent weight, yield to this as they are gradually converted into granulation-tissue, producing the characteristic spinal deformities so often seen. Still further, owing to its tendency to spread along lymph-routes, tuberculosis may be found disseminated around the primary focus, with healthy tissue intervening, as is so frequently seen in the medulla of bone, a fact it is well to bear in mind when operating. This regional infection is due to the predilection shown by the tubercle bacillus for endothelium-covered tissues, the germs, travelling along the perivascular lymph-sheaths and lymph-vessels and locating at various points, become new centers of the disease. Again, bacilli reaching the blood indirectly, through the medium of the lymph-circulation, or directly, microscopic masses of the infected tissue or of germs alone being swept away by the blood as emboli, tubercle is liable to give rise to metastasis exactly as malignant neoplasms do. In this possibility lies much of the danger of local tuberculosis. So long as one focus is present, the viscera or other structures may become involved; in the removal of a focus, fragments of the infected tissue may be dislodged to become emboli, whereby generalization of tuberculosis is effected. The recognition of such a possibility is important because deciding the propriety of adopting one method of operation in preference to another. It must be here emphasized that a number of diseases whose pathology was formerly entirely misunderstood are now recognized to be due to the action of the tubercle bacillus. Thus all varieties of lupus are tubercular, as are many cutaneous lesions and those of mucous membranes, some of which were considered can-

cerous, others, syphilitic. "*Anatomical tubercle*" is tubercular, and the importance of its prompt eradication has thus become apparent. The proper method of cure for the various scrofulodermata and their sequences, "strumous cutaneous ulcers," is now clear. Many cases of tubercular ulcerations of the tongue, mouth, and nasopharynx are no longer aggravated by a mercurial cause or abandoned as hopeless because "cancerous." Most of these conditions are "primary"— *i. e.*, the infection occurs at or near the spot where the tubercular "gumma," as it is sometimes called, develops. Very exceptionally, tubercle bacilli which may gain access to the tissues at once enter the lymph-stream, and, becoming arrested in a lymph-node (gland), give rise to a primary lymphatic tuberculosis; but almost invariably tuberculosis of the lymphatics is secondary to an often non-infected lesion in the mouth, nose, respiratory, intestinal, or genito-urinary tract. Osseous tubercle is always secondary, never primary; that is to say, the germs can only reach the bone through the medium of the circulation.

While, as has just been said, it is theoretically true that bacilli may enter through a distant infection-atrium, the point of entrance escaping infection, clinically this remains to be proved. It must be plain that the majority of the bacilli which gain access to the tissues enter the lymph-spaces or migrate into them, hence nearly invariably they are first arrested by those "filters," the lymph-nodes (glands), where, setting up tuberculosis, this new center serves for a point of departure for germs. Again a similar course is pursued if infection of any mucous or cutaneous surface occurs, the only possible exception being as already stated, if the germs directly enter a blood-vessel or escape the filtering action of the lymph-nodes. A demonstration is often afforded of the secondary nature of bone tubercle by the shape of the sequestra often noted in tubercular disease of the epiphyseal extremities of long bones, these sequestra being wedge-shaped or conical,

this form evidently resulting from the occlusion of a vessel by a tubercular embolus, the cone-shaped infarct becoming eventually necrotic from anemia, or possibly infiltrated with tubercle and then perishing. Owing to the anatomical peculiarities of the circulation of the epiphyses of the long bones and the mechanical strains to which these regions are subjected, tubercle bacilli most frequently locate at these portions of the bones, although the medulla is often attacked with tuberculosis. While the area infiltrated with tubercle softens and is destroyed, outside the disease-process the hyperemia induces an excess of bone-formation productive of " osteosclerosis." This formation of bone at a little distance from the disease-focus in consequence of the hyperemia caused by the active changes taking place in the tubercular focus, explains the " travelling acetabu-lum " of hip-joint disease and the buttresses of bone which unite carious vertebræ. Bone-tubercle evacuates itself by the same process of conversion of the overlying tissues into tubercular tissue and their subsequent de-generation and liquefaction, as has been described for the soft parts.

Tuberculosis of synovial membranes is of special sur-gical interest because of the changes effected in joints and the sheaths of tendons. All the soft parts of the joint, ligaments as well as synovial membrane, are replaced by layers or masses of soft, gelatinous, fungous granulation-tissue, until, when the bone becomes seriously diseased with consequent partial or complete destruction of the encrusting cartilages, the articulation ceases to exist as such, the constituent bones being held together by little more than a soft mass of cells. Note carefully, however, that in many instances numerous disseminated areas of cicatrical tissue are to be found among the tubercular granulation-tissue, showing that a tendency exists toward the formation of permanent tis-sue, hence a partial victory has been won over the bacilli. Many of the more characteristic symptoms of tubercular

joint-disease are due to the massive formation of this granulation-tissue. Instead of the characteristic form assumed by each joint when its synovial cavity is distended with fluid, tubercular joints, for instance the knee, appear squared or rounded. Pseudo-fluctuation, not true fluctuation, is felt, due to the gelatinous condition of the granulations. Free mobility in abnormal directions shows that in advanced cases the ligaments are extensively disorganized, and the same condition accounts for the spontaneous partial or complete luxations so commonly observed. The tubercular process not being accompanied by acute hyperemia, the distended skin presents a peculiar white appearance, with perhaps a few enlarged veins; this absence of color over an advancing enlargement has given rise to the term "white swelling."

Fungous, gelatinous granulations are also common in tubercular disease of the tendon-sheaths, small masses of which tissue, separated from the main portions, constitute one of the varieties of "rice-bodies" so characteristic of many cases of chronic thecitis; it is not maintained, however, that all instances of chronic thecitis in which "rice-bodies" occur are tubercular, although the majority unquestionably are.

Although many of the numberless phases of tuberculosis as it occurs in different tissues and organs have not been touched upon, yet the essential types have been described, leaving for works treating of the surgery of the special tissues and organs all minute description of the symptoms and appearances presented by tuberculosis as it is modified by anatomical peculiarities of structure or surroundings.

Treatment.—It must be plain, from the preceding statements, that there are two general indications for the treatment of tubercle. The first, radical, complete ablation of the diseased structures, which is unquestionably the better plan if the process be localized and the operative measures capable of execution without inflict-

ing unnecessary crippling or destruction of the function of important organs or parts ; even this latter evil is often to be deliberately chosen if all tubercular disease can be thereby removed. Ablation is however at times impossible or inadvisable, either from the operative standpoint alone, because its absolute necessity is doubtful in the face of the dangers or destruction entailed, or most commonly, because only one focus of the disease can be reached, leaving other more dangerous foci which will of themselves entail death as advanced phthisis. This last objection should be given due weight, if the operation itself will put an additional strain upon the already lowered vital powers of the patient and thus favor the more rapid progress of the inaccessible lesion. Nevertheless, ablation should always be effected with incipient visceral tuberculosis—as that of the lung—if it can be done by an operation which will entail only a short confinement to the house and the absence of, or the minimum of, suppuration, as amputation of a limb for articular disease. Such an operation should always be chosen in these cases, rather than erasion or excision, either of which would perhaps be theoretically the proper operation *if no other focus of tubercle were present.* The second indication is to call into action the natural tendencies of the least infected portions of the granulation-tissue to develop into scar-tissue, to induce destruction and absorption of the hopelessly infected portions, and to neutralize the action of the toxic bacterial products as well as to remove or destroy the bacilli which are manufacturing them. The measures calculated to carry into effect this indication are also often put in practice after attempted ablation of the disease, where a complete operation has proved impossible or there are good grounds to suspect that this is the fact ; this is common when operating for many cold abscesses where it is impossible to be sure that all the tubercular lining membrane, which is often very adherent to the subjacent structures, has been completely removed. Clinical experi-

ence has long since demonstrated that certain general measures occasionally enable the tissues to resist the action or overcome the results of the implantation of the tubercle bacillus. Science now shows why sunlight, dry air, altitude, fatty food, etc., are useful. For instance, direct sunlight will kill tubercle bacilli. Albuminous food is not so readily assimilated if a proper proportion of fatty food be not present, and tubercular patients often both have a distaste for fat and also cannot assimilate it in its ordinary forms. What then are the indications for the constitutional treatment of tuberculosis? To improve the resistance of the tissues, to destroy or inhibit the multiplication of the bacilli, and to neutralize the effect of the toxic substances they generate, so that the least infected portions of the granulations may develop into a permanent tissue—*i. e.*, scar-tissue. Sunlight, abundance of germ-free air, stimulation of the cutaneous and general circulation by sea-bathing, when stimulation, not depression, follows its use, as is too frequently the case ; the administration of fatty substances, by inunction with cod-liver or other oils if they disagree when taken by the mouth ; regulation of all the secretions and excretions, with such drugs as will probably induce a leukocytosis, so that the bacilli may be removed or destroyed by phagocytic cells—are the general measures calculated to fulfill these indications. The now abandoned tuberculin certainly, by inducing congestion, brought more phagocytes to the tubercular foci, and nucleinic acid and the so-called nucleins produce an artificial leukocytosis. Iodin in the nascent form is certainly inimical to the multiplication of tubercle bacilli, either directly or by alteration of the soil, while it seems to chemically neutralize the bacillary poisons. The attempt to secure encapsulation by injecting the tissues around the tubercular area with irritating substances, such as solutions of zinc chlorid, has not met with much favor, whatever benefit accrued probably resulting from the antibacillary action of the substance employed. At present, two

drugs alone seem to have stood the test of prolonged use —viz., iodoform and balsam of Peru. The former to be efficacious must be maintained in contact with the tissues, *all air being excluded.* Thus, if a cold abscess is opened, after the most thorough removal possible of the tubercular lining membrane, iodoform emulsion must be introduced into the cavity and the soft-tissue wound tightly closed by accurate suturing, and sealing with collodion, even if it be certain that no permanent healing can be secured. Firm packing with iodoform gauze probably secures the same result in a minor degree, because the iodoform in contact with the tissues acts in the absence of much, if any, air. The best results are attained from injecting iodoform directly into the tubercular tissue or into a tubercular joint. The action of iodoform has already been partly explained. Besides the direct action on the germs and the neutralization of their products, the tubercular tissue becomes infiltrated by round cells (phagocytes), the diseased tissue undergoes fatty degeneration, liquefies, and is absorbed, and healthy, organizable granulation-tissue appears. Balsam of Peru stimulates to a remarkable degree the transformation of the pale, flabby, avascular granulations of tubercular tissue into the healthy, florid, fully vascularized non-specific granulations capable of definitive healing. As has been explained, normal granulations contain numerous phagocytic leukocytes, hence probably the beneficial action of the balsam is to be ascribed to the attraction of blood to the part which primarily brings the leukocytes necessary to restrain the action of the tubercle bacilli, and then granulations form, both capable of defending themselves against future attacks of the bacilli in virtue of the phagocytes they contain and also of cicatrizing. Ignipuncture, both for tubercle of the soft part and of the bones, is too much neglected. When employed to evacuate a tubercular focus, the track is sealed against infection, while the radiated and conducted heat destroys the infection for a

wide area outside the portions of tissue actually brought into contact with the cautery ; in this way numerous softened tubercular lymph-nodes can be successfully treated in the neck with the minimum of scarring, annoyance, and risk to the patient.

While much more time might be occupied in illustrating the application of the principles enunciated, nothing really essential could be added without trenching upon the technic of operations in each special region, which would be manifestly out of place.

LECTURE XXVI.

HEMORRHAGE: SYMPTOMS, GENERAL AND LOCAL;
NATURAL HEMOSTASIS; ARTIFICIAL ARREST OF
HEMORRHAGE.

Hemorrhage.—Hemorrhage may be arterial, venous,
or capillary, or it may arise from all three sources. The
blood issuing from a wound usually comes from all three
sets of vessels, but unless arteries of a certain magnitude
be opened, natural hemostasis soon checks the venous
hemorrhage, next the arterial ceases, while later only
the capillary oozing has to be contended with. A special
tendency to bleed from inadequate causes exists in cer-
tain individuals (hemophiliacs), but is also seen in pur-
pura, scurvy, and poisonings by drugs such as phospho-
rus—in this last condition fatty degeneration of the
vascular walls having been detected. When the blood
finds exit upon a free surface, as the exterior of the
body, one of the hollow viscera or a serous cavity, or
into the substances of organs or the general cellular tis-
sue, certain descriptive terms are employed, as ''exter-
nal''; ''internal'' when occupying the abdomen, pleura,
mediastinum, etc.; ''ecchymoses'' when the blood is
effused into the cellular tissue, in which locality, if it is
of large amount, it is sometimes called an ''extravasa-
tion.'' Localized collections of fluid blood are called
''hematomata,'' and sometimes even when occupying
the cellular tissue, ''hematoceles,'' as those of the neck
or spermatic cord; but this same term ''hematocele''
is employed to designate effusions of blood into some
of the serous sacs, as that of the tunica vaginalis testis.
Traumatic hemorrhages—*i. e.,* those produced by in-

jury, can best be considered under four heads—viz., Primary, Intermediary or Consecutive, Secondary, and Parenchymatous. Primary hemorrhage is that which immediately follows the injury, and in amount varies with the vessel wounded or with the kind of traumatism. An incised wound bleeds more freely than a contused or lacerated one, whole limbs being often avulsed with only an insignificant loss of blood. Intermediary or consecutive hemorrhage is that which comes on between the stage of shock and that marked by suppuration, but usually appears shortly after reaction is established. This variety of bleeding results most frequently from the increased vascular tension induced by reaction, from the same caused by the violent struggles of the patient or the straining of vomiting. Again, the too early removal of external pressure permits the loosening of the coagulum occluding the mouth of some vessel of considerable size which has ceased bleeding because of the feeble heart-action induced by the shock. Secondary hemorrhage is that which comes on after suppuration has been established, and is the result of infective inflammation causing disintegration of the hemostatic thrombus, ulceration, or sloughing of the vessel ; possibly the aseptic separation of a slough, if this be forcibly removed by some movement of the patient before the vessel has become firmly sealed, may explain a secondary hemorrhage. Parenchymatous hemorrhage is a general capillary oozing, due either to inflammatory dilatation of the capillaries, or to thrombosis of the principal veins.

A few words must be devoted to a consideration of the results of loss of blood.

Constitutional Signs of Hemorrhage.—The countenance, especially the ears, lips, and conjunctivæ, as well as the general integument, are of a pallid color and shrivelled, pinched appearance. The general surface is bathed in a clammy sweat, the countenance is vacant, the pupils are dilated. Because the brain is anemic,

humming, roaring, or ringing sounds are heard. . The patient complains of the passage before the eyes of a thick mist, or even darkness alternating, perhaps, with flashes of light. General sensibility is benumbed, and unconsciousness, with syncope or convulsions, follows if the loss of blood be severe. Intervals of consciousness occur during which the debility is great, evinced by the faint whispering voice, feeble sighing respiration, marked dyspnea, and small, frequent, fluttering, almost imperceptible pulse. The patient rallies for a time if the loss of blood be not fatal. With each renewal of the hemorrhage, the patient faints and is restored to consciousness with increasing difficulty. Dyspnea becomes more marked, and the pulse increasingly frequent and feeble, while the slightest elevation of the head, if the patient be recumbent, still more certainly the attempt to assume the upright posture, will insure fainting. The face is waxy pale, the lips and mucous surfaces almost bloodless, the flesh is soft, the movements languid, and any blood now effused is little more than bloody serum; anasarca and other forms of dropsy now appear, and the slightest loss of blood will prove fatal. When from the wound of a great vessel or the rupture of an aneurysm death results from sudden hemorrhage, the blood, instead of being forced onward by the elastic recoil of the arteries, runs backward from all parts of the body toward the opening in the vessel. The pressure in the veins being thus relieved, the blood no longer flows toward the heart, which ceases to act, and the face, as in asphyxia, becomes somewhat livid in hue, from the stagnation of venous blood. Before the fatal issue, the face suddenly becomes deathly pale, except a livid circle around the eyes, the lips are purplish, and the extremities cold. Syncope occurs, is recovered from and recurs, the voice becomes whispering, nausea sets in, the pulse becomes almost imperceptible. Marked and incessant tossing of the limbs begins, the head is at times suddenly raised or the patient struggles to rise, gasping for breath with an

agonized expression of face ; convulsive, sighing respiration, with loss of pulse at the wrist, comes on and the patient expires. The face has not the waxen, translucent pallor of one dead from slow loss of blood, but is of a clayey, leaden hue. In both varieties of fatal hemorrhage, the previous constitutional state, whether robust or the reverse, has much to do with the result from a given loss of blood, as has also the age of the patient, neither the young nor the old bearing the loss of blood well.

Because of the deficiency of the oxygen carriers—*i. e.,* the red cells—great frequency and irritability of the heart's action follows severe losses of blood, often resulting in the phenomena formerly called "hemorrhagic fever." There is a hurried, jerking, irregular pulse, slight flushings of the face, with brilliancy of the eyes, alternating with pallor and syncope, while if the hemorrhage prove fatal, delirium with convulsions and an extreme, indescribable restlessness, precede death. It is hardly requisite to state that there is no rise of temperature in uncomplicated cases, the phenomena resulting from deficiency of pabulum to the nerve centers and of oxygen to the whole economy. The convulsions mentioned are epileptiform in character, and are rare except after sudden, copious losses of blood. When treated properly—viz., as for bad syncope, they are generally not dangerous. In those predisposed to convulsions, especially former epileptics, a small loss of blood may cause eclampsia.

Treatment of Hemorrhage.—Needless repetition will be best avoided by a few preliminary remarks applicable to the treatment of hemorrhage in general. Its primary effects are to be combatted by all those means which will favor the retention of a functioning amount of blood in the brain, especially the respiratory centers *because of the risk of fatal syncope.* The head and shoulders should at once be lowered, and kept so by raising the foot of the bed twelve or more inches, not even a bolster being left

beneath the head. The limbs should, in addition, be raised nearly at right angles to the body, and so maintained. In the worst cases, Esmarch's elastic bandages can be applied to one or all of the limbs, thus utilizing most of the blood of the extremities. If elastic bandages are not available, ordinary muslin ones may be used, and finally, in default of either variety of bandage, digital or instrumental compression of the arteries of the limbs in the raised position may be tried. The most essential, if not all, of the above measures can always be put into practice at once, without attempting the removal of the patient to a perhaps distant home or hospital ; by neglecting an immediate resort to these expedients, many lives are lost. Next, stimulants by the mouth or rectum must be administered. Turpentine, $\frac{1}{2}$ to 1 fluidounce, best emulsified by beating up with a raw egg and water, although this is not essential, is an admirable stimulant to administer by the rectum. Subcutaneously, strychnin, whiskey, brandy, or, better still, ether when the patient has not been anesthetized, as frequently repeated as seems necessary to ward off syncope, are indicated, and at times atropin subcutaneously acts well as a respiratory and cardiac stimulant. External heat by hot-water bottles, etc., should be assiduously applied, and a sinapism over the heart is never amiss. Normal salt solution, at a temperature of 100° to 105° F. as it *emerges from the needle or cannula*, should be unhesitatingly injected into a vein, the subcutaneous tissue of the mammary or abdominal regions (hypodermatoclysis) in severe cases, or into the rectum. The first two methods require no apparatus beyond a large-sized hypodermic needle (a small cannula is preferable for introduction into a vein if such an instrument is procurable), a few feet of rubber tubing, and a funnel or a small vial with its bottom broken out. The amount to be injected varies from a few ounces to several pints, administered at intervals in such quantities as will secure the desired effect upon the circulation. Indeed, I have succeeded in throwing enough saline solution into

the cellular tissue to save life in cases of severe hemorrhage, by means of three of four hypodermic syringes rapidly used by as many assistants.

As soon as possible, hot, concentrated meat-essences, to which abundance of sodium chlorid has been added, and milk, must be given, with any reasonable amount of water the patient can dispose of, the rectum being also utilizable for this last purpose, especially if the stomach be irritable, employing enemata of about 4 to 8 ounces of the saline solution every four hours : the main object being to secure the rapid absorption of a sufficient bulk of fluid to mechanically enable the circulation to be properly carried on. As the patient rallies, first the bandage on one limb (if such has been applied) may be partially removed, then entirely. If the pulse does not flag after an interval, the bandage from another limb should be removed, and finally from all the limbs, similar advice being applicable to those cases where instrumental or digital compression has been used. Last of all, the limbs may be lowered one by one, but the dependent position of the head and shoulders must be uninterruptedly maintained, sometimes even for days, and always for some hours, until the bulk or quality of the blood has improved to a degree compatible with a more ordinary position, which must also be gradually assumed by lowering the foot of the bed by degrees.

The above remarks chiefly apply to a first and copious hemorrhage, but are applicable according to their degree to recurrent attacks. As soon as seems advisable, the internal use of iron and albuminous articles of food must be resorted to : after a single copious hemorrhage, to obviate the secondary evil consequences of the blood loss ; in the recurrent form of hemorrhage, to keep up the supply of the vital fluid so that new losses may not prove fatal, and also to prevent or cure the dropsical condition often induced by repeated hemorrhages. Although the internal use of acetate of lead, oil of

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erigeron, dilute aromatic sulphuric acid, ergot, and opium have been relied upon in the past to control internal hemorrhages or prevent the return of external ones, it is a serious question in my mind whether, with the exception of the two last named, they are of any real value. The value of subcutaneous injections of sterilized solutions of gelatin in normal salt solution will be considered when speaking of the treatment of hemophilia. The local means for the *temporary arrest* of hemorrhage are in the order of their availability : local compression over the wound by means of fingers, compresses, bandage, tourniquet, etc. ; compression of the wounded vessel in the wound by similar means—it matters not whether the wounded vessel be artery or vein ; compression of the main trunk-vessel, either by the finger, improvised tourniquet (Fig. 13) or an ordinary
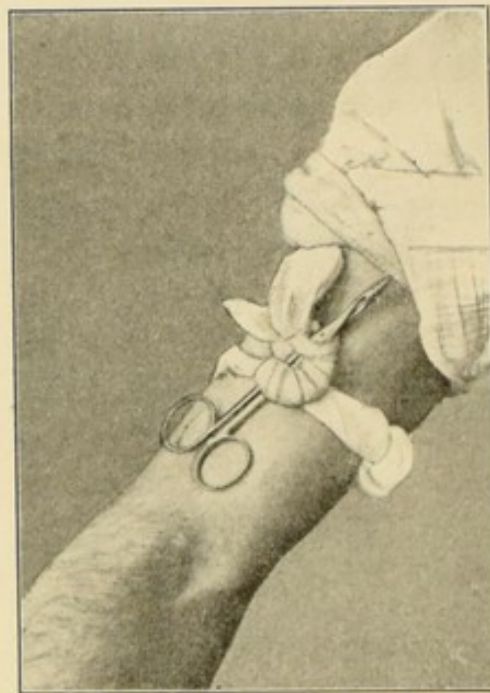
FIG. 13.—Showing so-called Spanish windlass, an improvised tourniquet, compressing the brachial artery.

tourniquet, including forced flexion and position. Forcible flexion has often proved useful in cases of injury of the arteries of the forearm and hand, or of the leg and foot. A roll of lint or some other soft material should

be placed in the flexure of the joint, and the limb then bent until the hemorrhage is arrested, and maintained in this position by a handkerchief or bandage. When the bleeding is from the vessels of the leg or foot, the effect of the flexion is materially increased by flexion of the thigh upon the abdomen, as well as of the leg upon the thigh. Some of these measures may also be relied upon for the permanent arrest of certain varieties of hemorrhage, and may be, in very exceptional cases, available for all. Baron Larrey reports a successful case of compression of the external carotid artery ; a solitary exception, not a rule. I have myself succeeded in permanently arresting the bleeding from a wounded vertebral artery by firm packing of the ball-wound track through the transverse process of the vertebra.

From the multitude of styptics such only "should be selected for use by the surgeon as imitate, hasten, or assist the natural processes of hemostasis." *Natural hemostasis* is effected first by the passive retraction of the artery within its sheath caused by the recoil of the elastic tissue it contains. Retraction is still further induced by the contraction of the longitudinal muscular fibers of the arterial wall, while the circular fibers diminish the size of the aperture. An external clot forms within the roughened sheath around the orifice of the severed vessel, the thrombus thence gradually extending into its lumen until, usually, it reaches the first collateral branch above, if completely divided, or above and below if wounded in continuity (Fig. 14). The internal and external clots, acting as "buffers" to the circulation, permit the undisturbed formation and organization of granulation-tissue formed by the vessel-walls where divided : this process has already been described in Lecture IV. when speaking of the repair of blood-vessels. Cold is the oldest and best known hemostatic. After removing all clots, mere exposure to the air frequently stops troublesome bleeding from wounds that ooze continuously when closed and covered up with

cumbersome dressings. Fanning or blowing upon the wound-surface increases the effects of atmospheric cold. Still more powerful in their effects are compresses, dipped in ice-water, squeezing the contents of a sponge dipped in ice-water over the wound, or syringing it out with the same. Finally, ice itself, either in substance or in bladders or India-rubber bags may be tried. Hot water at the temperature of about 125° F. to 130° F. — roughly estimated, the greatest heat bearable by the back of the hand—directed upon the wound in a stream from a sponge or syringe, but best of all applied by means of a towel wrung out in it and steadily pressed upon the wound, is far more efficacious than cold, is sterile, and not depressing but actually stimulant. Alcohol, either pure or diluted, acts admirably, both contracting the blood-vessels and forming coagula

Fig. 14.—Clot (*d*) extending to first collateral branches in both ends of cut vessel; also retraction of vessel within its sheath (*b*), and curling in of inner coat (*c*) of vessel. (Diagrammatic.)

around and in their orifices. The direct application of ice to arrest bleeding, or the use of ice-water, is objectionable, because asepsis is hardly possible. Rubber bags can of course be sterilized.

Iodin, either in the form of the pure tincture or variously diluted, acts well, both it and alcohol possessing the additional advantage of being sterile and probably actively germicidal. Turpentine, applied by means of

little masses of gauze laid upon the bleeding points, has proved useful in passive hemorrhages.

Perchlorid and subsulphate of iron are popular but unreliable styptics. While they unquestionably control capillary oozing, this can be usually done equally well by other less objectionable means. Their coagulating power is not instantaneous, hence where blood is flowing freely, an outside crust is formed beneath which fluid blood accumulates, while the gaping vessel itself is not acted upon, the only means by which the hemorrhage can be permanently arrested. Monsel's solution is less objectionable than the perchlorid, but a hard, insoluble coagulum is formed, difficult to detach and an absolute bar to rapid union. In leech-bites, however, a morsel of cotton or sponge moistened with Monsel's solution and pressed well down into the wound is a useful means to arrest obstinate bleeding. Transfixing the edges of the minute wound with a fine sewing needle and throwing around it a figure-of-eight ligature is, however, a more certain, an aseptic, and therefore less objectionable, measure. Liable to the same objections, but in less degree, are tannic and gallic acids and alum. Chlorid of zinc, a 5 per cent. solution of antipyrin, and a 4 to 5 per cent. aqueous solution of suprarenal gland are germicidal or can be made sterile. Alum is least objectionable among the chemical astringents, and may be applied in a tepid state by means of sponges, absorbent cotton, bits of lint, etc., impregnated with it, the warm saturated solution on cooling, depositing fine aseptic crystals of the salt around the vascular orifices. It is quite probable that much of the good effects attributed to the astringents is really due to the compression produced by the lint, etc., and the multiplication of the points of contact favoring rapid coagulation.

The objection to all chemical styptics is that they interfere with or absolutely prevent primary union, and render suppuration nearly certain with all its dangers. Dilute alcohol or tincture of iodin are not so obnoxious

to these charges, and antipyrin little if at all, while a hot 2 per cent. solution of gelatin in normal salt solution is both efficacious and unobjectionable so far as primary union is concerned ; still, where wounds are concerned, unless immediate union is either expected or desired, after careful ligation of all bleeding points has failed to arrest all hemorrhage, compression or antiseptic tamponade has taken the place of styptics in modern surgery, the packing being removed in a few days and secondary suturing being done. As hemorrhage may take place from various cavities and from wounds where sometimes nothing but a styptic is available, in the absence of other facilities, I have felt called upon to mention some of the more common ones. The actual cautery is the most certain and powerful of styptics, the best form in which to employ it being the Paquelin thermocautery. Producing a charred surface—which is antiseptic—and an eschar, which when it separates leaves a healthy granulating surface—*i. e.*, usually an efficient barrier against serious infection—it is our most efficient hemostatic in certain operations where union by the first intention is not to be expected. If we were deprived of the cautery to arrest the oozing from separated adhesions, many abdominal operations would prove far more difficult and dangerous procedures than they now do. No eschar separates after intra-abdominal cauterizations, the charred tissue being removed by aseptic absorption. Arteries of any considerable size should not be sealed by the cautery, except perhaps when removing the superior maxilla, where the vessels oftentimes cannot be isolated and ligatured, but it is an admirable remedy in appropriate cases to check venous or capillary oozing. The cautery should be applied just off the black—*i. e.*, at a dull-red heat, a higher temperature destroying the tissues too rapidly to secure hemostasis. Cautery irons can be improvised out of telegraph wire coiled into various shapes, the poker, or any conveniently shaped piece of metal (Fig. 15). The measures adapted for the *permanent arrest* of hemorrhage

are torsion, forcipressure, ligature, acupressure, occasionally antiseptic tamponade, and possibly the cautery.
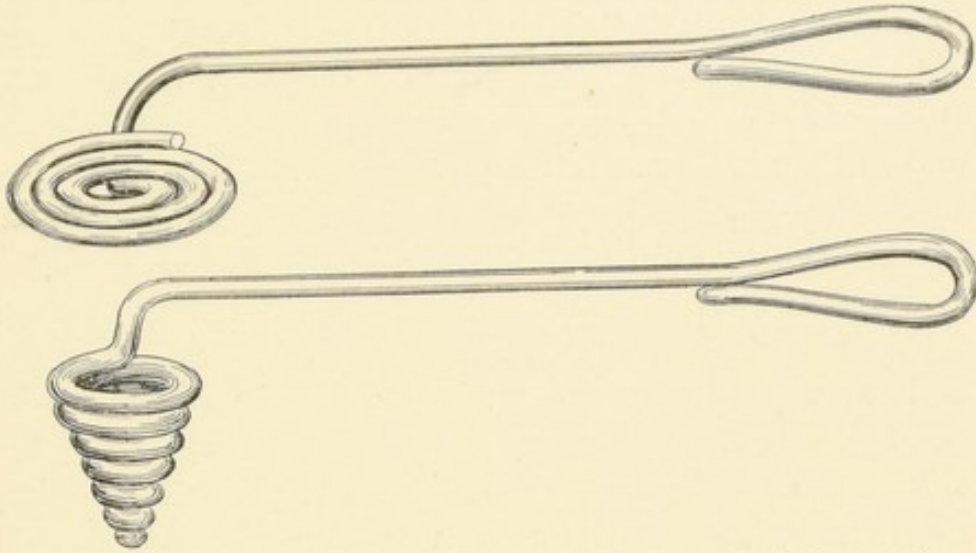
FIG. 15.—Two forms of cautery which may be improvised from ordinary heavy wire.

Torsion is only applicable to completely divided vessels. Although special instruments have been devised, the sim-
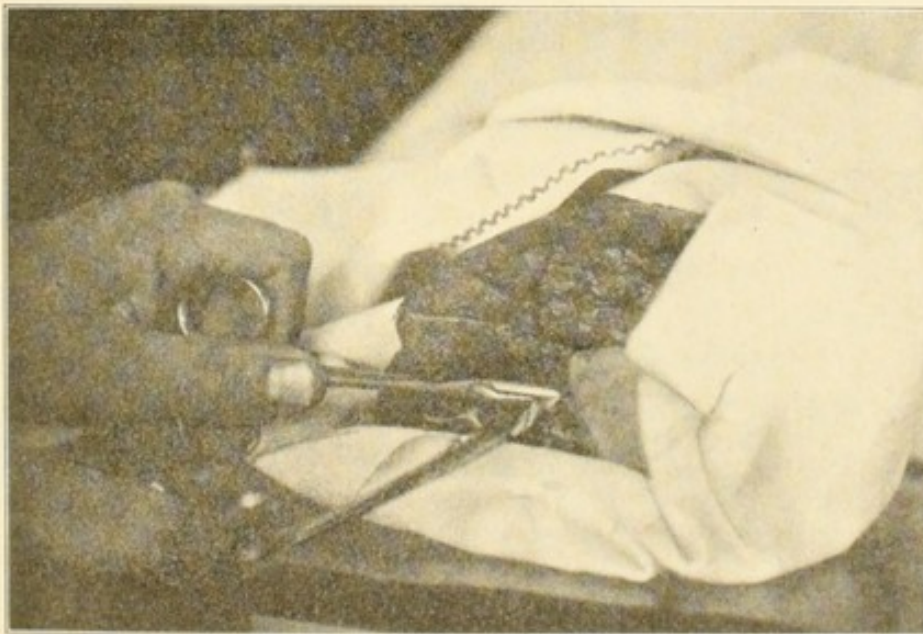
FIG. 16.—Proper method of employing torsion to accomplish hemostasis.

plest method is to seize with one pair of forceps the whole cut end of the vessel, drawing it out of its sheath for

about half an inch, when it must be grasped transversely by a second pair of forceps close to the tissues (Fig. 16); the forceps should be strong catch-ones with accurately fitting serratures. The end may be twisted off by about a dozen turns of the first pair of forceps, or better still, three or four sharp turns should be given until the inner and middle coats are felt to give way, when all resistance to further torsion apparently ceases. This method of torsion is only applicable to the larger vessels. Those of small size which cannot be isolated must be grasped with some of their surrounding tissues, which with the end of the vessel must be twisted off. Small vessels in structures normally dense, or rendered so by inflammation, cannot be treated by torsion, forcipressure proving more available. Torsion is a reliable hemostatic, but more troublesome than the ligature. Forcipressure is effected by seizing the bleeding point or mass of tissue
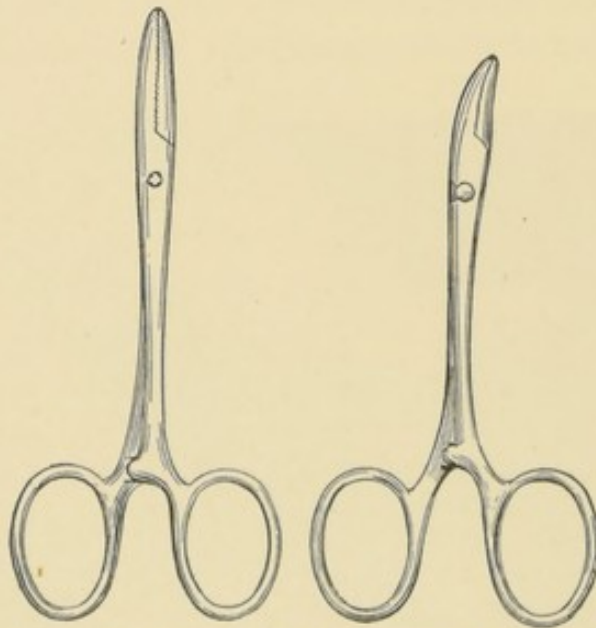
FIG. 17.—Varieties of hemostatic forceps.

and crushing it by means of catch-forceps, having strong, deeply serrated blades. In many cases their removal in a few minutes is followed by no bleeding, but they may even be left on for so long as forty-eight to seventy-two hours, as in vaginal hysterectomy.

LECTURE XXVII.

HEMOSTASIS (CONTINUED); LIGATURE; ACUPRESS-URE; HEMOPHILIA.

Ligatures for vessels are best made of properly pre-pared silk or catgut. When tying a divided vessel, as in an amputation, the mouth of the artery must be seized and drawn outward either by a tenaculum or by artery-forceps. The latter is the better instrument in most cases, since the tenaculum may accidentally punc-ture the vessel behind the part ligatured during unsuc-cessful efforts to take it up, this accident often resulting in ulceration of the arterial coats and secondary hemorrhage. The tenaculum, however, is often indispensable when the vessel is small and situated in dense tissues, since then the noose of the ligature must include not only the vessel but also some of the surrounding tissues. In this event the tenaculum should be slightly withdrawn as the first half of the knot is tightened in order to insure a firm hold on the included tissues. Sometimes a curved needle armed with a ligature can be more conveniently used than either forceps or tenaculum, by passing it under and around the bleeding point including some of the surrounding tissues, but as little as will enable the ligature to hold. When tightening the knot, the surgeon must be sure that the thread completely surrounds the cut end of the artery, and that the tip of his forefingers or thumbs are in close contact with the forceps or ten-aculum, so as to press the ligature somewhat down, thus preventing dragging off the instrument or pulling the vessel too much away from its sheath, which endangers its nutrition and so favors secondary hemorrhage. The

first part of the knot should be tightened only sufficiently
to completely close the lumen of the vessel. Formerly
when the silk ligature had to be loosened by ulcerating
through an artery tied in its continuity, or by a similar
process cutting off the included extremity of a divided
vessel, the thread was drawn so tightly as to insure the
complete division of the middle and inner coats, leaving
only the external to slough through. The reverse is now
aimed at. If an animal ligature be employed, firm appo-
sition of the inner coats maintained for a few days is all
that is essential, a more tightly drawn ligature endanger-
ing the vitality of the included segment of vessel and
thus favoring secondary hemorrhage. In like manner,
the modern silk ligature is meant to remain, hence must
not be drawn so tight as to destroy the vitality of the
vessel-walls and be cast off. The knot used should al-
ways be the square or "reef knot" (Fig. 18), never the
"granny" (Fig. 19). The surgeon's knot may be used,
but should be reinforced by a second tie (Fig. 20).

FIG. 18.—Method of tying square
or reef knot.
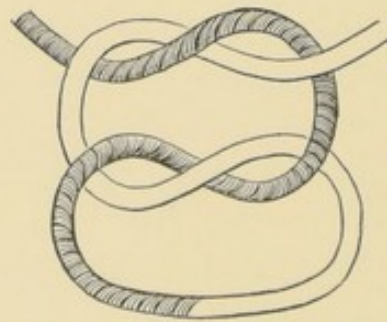
FIG. 19.—Method of tying granny
knot.

If an artery be wounded in its continuity, the surgeon
must reach the injured part by a careful dissection,
avoiding all unnecessary injury of neighboring parts.
Both the proximal and distal ends must be tied, or, if
not entirely divided, two ligatures must be passed
beneath the vessel by means of an aneurysm-needle or
eyed probe with the least possible disturbance of parts.
One ligature must be tied above and one below the

wound. If these do not stop the flow of blood, the sides and under surface of the part included between the ligatures must be carefully examined for some small branch returning blood by means of the collateral circu-
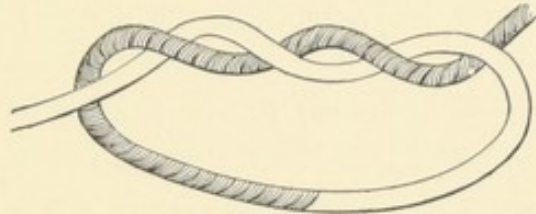
FIG. 20.—Method of tying surgeon's knot.

lation. Neglect of this imperative rule of tying both ends of a wounded artery at the point injured, and any branch opening into the included segment, rarely ends in anything but disaster ; tying the main vessel at a distance from the wound hardly ever proves successful, besides entailing other dangers.

Direct pressure is chiefly efficient for small arteries, and is usually only applicable when the vessel overlies a bone. Sometimes, however, the firm, uniform pressure of a bandage is effectively used to check oozing, or a sand- or shot-bag may be employed, or filling the wound with shot may prove successful. These measures are chiefly applicable for recurrent hemorrhages after ligature of arteries, where no room remains for a new ligature, as in the innominate ; or where the bleeding comes from the *distal end* of an artery, in which event methodic bandaging and direct compression with a graduated compress should be tried before the vessel is ligated higher up or amputation is performed. Whether a graduated compress or a pointed vial-cork be used, the artery should be controlled above by digital or other compression, the wound cleared of clots, and the compress accurately applied over the bleeding point. Less force is requisite than the profession seems to think, and even this had better be carefully relaxed after twenty-four to thirty-six hours, lest a slough and con-

sequently secondary bleeding occur, although the compress must be allowed to come away of itself. Obstinate bleeding from hollow cavities, such as the rectum, vagina, or nares, can only be checked by pressure—*i. e.*, packing with cotton, pledgets of lint, or pieces of sponge, impregnated with some styptic or not, as the surgeon prefers.

By *acupressure* is meant the occlusion of an artery by the pressure of a pin or needle so placed as to control the blood-current. Various methods have been devised
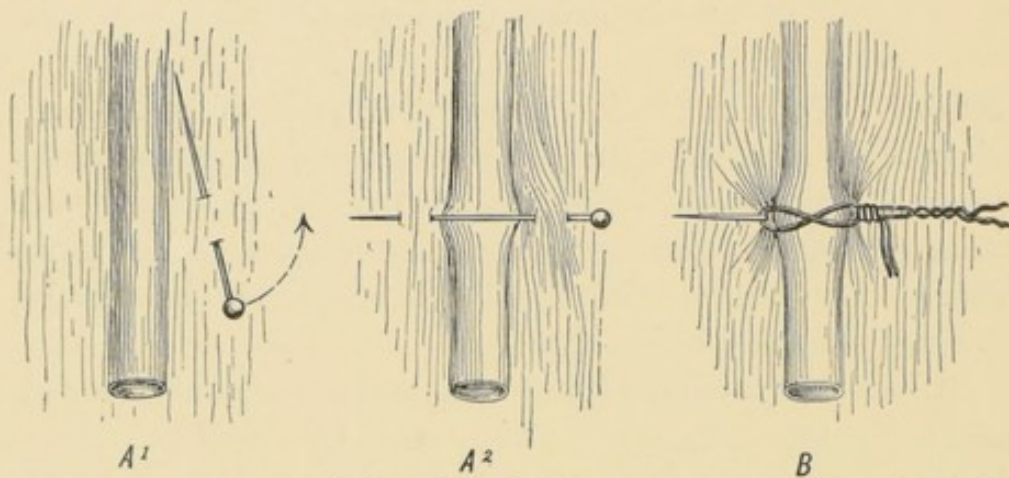
FIG. 21.—Two methods of employing acupressure: A^1 and A^2, by means of a pin passed in front of vessel; *B*, by means of a threaded needle passed behind and a figure-of-8 wire passed in front of vessel.

by Sir James Y. Simpson, its originator, but only two will be described and figured. A needle threaded with a piece of twisted wire is passed beneath the artery, entering and emerging from the contiguous tissues a few lines from the vessel (Fig. 21, *B*). A loop of wire is then thrown over the point of the needle, carried across the vessel with sufficient force to compress it, and secured by a half-twist around the eye-end of the needle. The needle is readily removed by traction on the twisted wire, when the wire loop can be withdrawn. According to the size of the vessel, the needle should be removed in from thirty to sixty hours. Since the introduction of absorbable ligatures, acupressure is chiefly valuable as a prompt and ready method of arresting hemorrhage in an emergency.

The chief **danger of arterial hemorrhage** is from the amount of blood lost, or its pressure upon important parts or organs, when not escaping externally. It matters not whether the blood is poured out externally, into the areolar tissue, or into a serous sac, the constitutional effects are the same for the same amount of blood lost during a given period. An oblique cut in an artery gapes more than a longitudinal one from the combined retraction of the circular and longitudinal coats, while from these causes the same vessel completely divided may permanently cease to bleed ; this method of hemostasis was formerly sometimes used after bleeding from the temporal artery. A small twig divided close to the parent trunk will bleed as furiously as a similar sized wound of the main vessel, and must usually be treated as if the trunk-vessel were wounded. Although a wounded artery usually throws out a jetting stream of scarlet blood, yet in a deep narrow wound—a stab, for instance—I have seen a wounded brachial with only a steady flow of venous-hued blood at the margins of the stream, while in the center a tiny thread of arterial blood could readily be detected. While a fine needle-puncture of an artery will heal, there is no proof that larger ones do without an obliteration of the vessel's lumen. If then the current of blood be not permanently arrested by ligature or some other means, the arterial wound remains open and repeated hemorrhages occur if the soft parts do not heal, while in the latter event a traumatic aneurysm will form.

The *treatment* has already been indicated—viz., ligature or acupressure above and below the wound for a vessel wounded in continuity; ligature or acupressure for a divided vessel, say in an amputation, and, under certain conditions pointed out before, forcipressure, direct pressure, the actual cautery, etc. Secondary hemorrhage is to be treated on the same lines as primary. The bleeding vessel must, when possible, be secured at the point where it bleeds. If the hemorrhage comes from the

distal extremity of an artery ligatured in its continuity, methodical bandaging of the limb with local compression in the wound should be tried ; next, the same with ligature of the main trunk higher up ; finally amputation of the extremity. This latter extreme measure would be indicated in almost every case of aneurysm of an extremity which has ruptured externally. Ligature, except under the circumstances just given, of the main trunk for secondary hemorrhage at a distance from the wound, is bad surgery ; but in the case of a stump which has become soundly healed, except for a few narrow sinuses, tying the vessel just above the stump is good practice.

A large vein may be ruptured *subcutaneously* from a blow or strain, or in reducing a dislocation, producing at times a dangerous loss of blood. From direct injury or strain the saphenous vein or its branches are those most commonly ruptured. There are especial dangers inherent in open wounds of certain veins dependent upon their size and position. For instance, hemorrhage from the internal jugular is not dangerous so much from its amount, but because the brain is directly depleted, resulting in rapid and fatal syncope. Wounds of veins at the root of the neck or in the axilla are dangerous, in addition to the amount of blood lost, from the risk of the entrance of air into the circulation. In general terms this accident results from permanent canalization[1] of the vein by inflammatory thickening, or a similar temporary condition from tension of tissues. A third danger is septic or spreading inflammation. That a hemorrhage is venous can be determined by observing that the blood is poured out from one or more points in a steady stream and of a venous hue ; that contrasted with capillary hemorrhage, the flow is greater, and can be arrested by pressure, on the distal side of the wound alone unless one of the largest venous trunks be wounded ; and finally, anatomy will indicate in many instances that a vein and not an

[1] See page 147.

artery is implicated. Occasionally, the blood from the distal extremity of an artery of the lower extremity, ligatured in its continuity, is of so much darker a hue than ordinary arterial blood that it may readily be mistaken for venous, especially as it flows in a steady stream and is arrested by pressure *below* the wound. Bearing this fact in mind, when bleeding occurs after tying a main artery, will prevent any mistake.

Capillary hemorrhage can never be of serious import, except in the case of "bleeders," or in those excessively rare cases of purpura hemorrhagica, in which, as I have once seen, fatal effusion occurs into the brain. When the wounded surfaces can be seen, the blood is observed to come either from a series of minute points or as a general ooze from the whole surface. If the wound be a deeper one, it gradually and steadily fills up, pressure at no one point more than at another being effective for its arrest. The color of the blood is intermediate between arterial and venous. Position, exposure to the air, hot water, alcohol, some other of the styptics mentioned, or best of all, antiseptic tamponade, will suffice to check this form of bleeding. Prolonged bleeding after the extraction of a tooth is best arrested by placing a sterilized vial-cork, cut to fit, in the socket, and binding the jaws together. In the *parenchymatous* form, due to inflammatory dilatation of the capillaries, hot water, the actual cautery, or Monsel's salt may be necessary. When this variety of hemorrhage is the result of thrombosis of the chief veins, ligature of the main artery or amputation higher up is indicated, if the bleeding is serious and uncontrollable.

Hemophilia.—This consists in a tendency to excessive and continuous hemorrhage which occurs spontaneously or after traumatism, the latter being usually trivial. It is rare to see any manifestations of this tendency before the second year, and the first attack of the inherited disease is equally uncommon after puberty; indeed after this period, the tendency usually diminishes.

The oozing, continuing for hours, days, or even weeks, is always capillary. Epistaxis is the most common form of spontaneous hemorrhage : slight scratches of the skin, bites of the tongue, leech-bites, blisters, extraction of teeth, and circumcision are some of the traumatic causes ; the last three seem especially dangerous. A hematoma extending from the knee to the trochanter has resulted from the fall of a rubber ball on the thigh. Petechiæ, chiefly occurring beneath the skin of the extremities, but sometimes seen in serous and mucous membranes, may be accompanied by swelling and pain in the joints. Arthritic complications are very common even in the absence of petechiæ. They vary from simple pain to suddenly developed redness and intense inflammation accompanied by fever, thus closely simulating acute rheumatism : the knee-, elbow-, ankle-, and shoulder-joints are those most usually attacked. Repetition of the attacks is apt to result in marked deformity and disability.

Pathology.—The blood shows nothing abnormal, except ordinary anemia just after the bleeding, and wounds heal as in other anemic patients. Histological examination of the vessel-walls has only once afforded confirmation of the statement of Blagden, so often since repeated by others on his authority, that the walls are unusually thin, Kidd finding degeneration of the muscular tissue of the middle coat of the arteries and proliferation of the endothelium of the arterioles, veins, and capillaries. Although some peculiar frangibility of the vessels and absence of a proper amount of fibrin-ferment has been inferred, there are no reliable histological or chemical facts in support of this view.

Diagnosis.—In a bleeder family this is easy. If occurring in a member of a family hitherto free, and not the result of slight, prolonged sepsis, reliance must be placed upon the following points : the traumatic hemorrhages are initiated by injuries well known not usually to give rise to prolonged bleeding, the spontaneous hem-

orrhages are multiple, joint complications are frequent, the tendency to bleed persists for years or for life. Such an individual would constitute a "congenital bleeder," not a hereditary hemophiliac, and if a male, he would be likely to found a "bleeder family" if he should have offspring, because, although females may transmit, they do not originate and very rarely, some say never, suffer from this ailment. The diagnosis from hemorrhagic or infective purpura, peliosis rheumatica, etc., must be learnt by the study of works on the practice of medicine.

Prognosis.—Few die from the first bleeding. The older the patient the better the chances. Females "of bleeder families" are not especially prone to post-partum hemorrhages, although they often menstruate early and profusely, yet any accidental or operative bleeding is usually easily controllable. In the course of prolonged and pronounced sepsis and when severe jaundice of hepatic origin has lasted for long periods, a decided tendency often exists to bleed persistently and dangerously : this may be termed "acquired hemophilia."

Treatment.—Prophylaxis is imperative. In a known or suspected hemophiliac, avoid all operative injuries, extraction of teeth, in fact anything but life-saving operations. When bleeding has started, cleanse the wound and apply moderate pressure, impregnating the compresses with a solution of antipyrin, or a 2 per cent. one of gelatin in normal salt-solution. As a 4 to 5 per cent. solution of powdered suprarenal gland has proved an efficient styptic in operations upon the nose, its use is suggested for hemorrhages in bleeders. Repeated injections of cocain into the gum has checked the oozing after the extraction of a tooth. Injections into the cellular tissue of a sterilized solution of gelatin, of the strength suggested has in my own experience apparently proved successful. Ergot by the stomach, purgation effected by sodium sulphate—except when enterorrhagia is present—and large doses of the astringent salts of iron

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seem to have done good. Chalybeates, cod-liver oil, tonics, etc., are useful in the intervals, and residence in a warm climate has been thought beneficial in warding off attacks. The joint troubles must be regarded as grave. A first attack often leaves an articulation normal if properly treated by absolute rest and moderate compression, but when untreated or repeated bleedings occur, an arthritis results, closely simulating some forms of tuberculosis. In such cases, puncture of the joint may be tried so as to irrigate with antiseptic fluids, but the procedure is a most dangerous one despite the success attending it in the hands of König. Pilcher and Bertrand believe that the dangers of necessary operative interference is overrated, but in this I cannot agree with them. Undoubtedly, in the hands of an expert, and by the employment of special devices, operations may sometimes be conducted to a successful issue in *certain* hemophiliacs. As the larger vessels, although perhaps more numerous, bleed no more than in ordinary patients, quilting of the whole wound-surface with catgut ligatures, the application of compresses freely dusted with iodoform (which is styptic), with or without the application of the actual cautery, will often succeed in arresting the parenchymatous oozing. Once, failing by all the measures described, I succeeded by packing the wound with lint soaked in Monsel's solution, and firm bandaging. This practice should not be repeated, unless beyond all question necessary, because it is impossible to maintain asepsis of the wound, a matter of special importance in hemophiliacs, "since," as Pilcher says, "secondary subcutaneous or intermuscular hemorrhages make the danger of suppuration greater, while infection in turn renders hemorrhage almost certain." I recently succeeded in arresting all oozing after amputation of an arm by the use of hot gelatin-solution, the cautery, and bandaging the flaps firmly after interposing several layers of iodoform-gauze wet with the same solution ; this man was a moderate "bleeder."

LECTURE XXVIII.

TREATMENT OF WOUNDS; ANTISEPSIS; ASEPSIS.

IN practice all pus and interference with normal wound-processes result from the presence of pathogenic organisms, for all germs are not harmful. To secure their exclusion or destruction it is requisite to understand the sources whence derived and the conditions favoring their development. The air, water, dust, the soil and pathogenic wound-discharges contain germs. If currents of air can be prevented, germs will gravitate, leaving the air completely or relatively free ; moreover, the germs found in the air of ordinary dwellings are usually non-pathogenic moulds and fungi. Draughts must be strictly avoided when operating or dressing wounds, lest quiescent germs be wafted into them. For a similar reason nurses should not wear long skirts, and all sweeping, dusting, or moving hangings, carpets, or furniture shortly before an operation or dressing must be forbidden. Although air, except in mid-ocean, above the snow-line, etc., is never actually germ-free, in a properly kept operating-room, ward or dwelling house, if draughts are avoided, the number of germs under ordinary circumstances are too few to be considered. All water contains germs except sea-water far from land. The soil- and house-dust, especially the latter, from apartments where suppurating cases have been treated, teems with organisms : hence the advantages of such methods of hospital construction and material as will least favor the lodgement of dust, which entangles and precipitates the germs. Exposed instruments and dressings may afford lodgement for germs, although the

surgeon's, attendants', and patient's integument, or, in the case of the latter, certain cavities or canals lined with mucous membrane, are the chief sources whence pyogenic organisms gain access to wounds. Although some germs cannot grow in the presence of oxygen and others require the presence of air, all varieties must have a proper temperature, moisture, and pabulum. Theoretically, pyogenic organisms can then be destroyed by the withdrawal of one of these essentials, but practically, moisture is the only requisite that we can nearly absolutely control, although the amount of pabulum can be lessened. While it is true that if a wound is germ-free, nature's reparative processes will pursue an uninterrupted course, it is also true that germs may be present and no harm result. Germs in numbers sufficient to overwhelm the resistance of the tissues, or a lowering of vitality of the tissues is necessary to render infection operative. Vascular tissues, as those of the face, seem to possess the highest immunity in virtue of the germ-destructive powers of healthy blood-serum and phagocytic leukocytes. Grawitz's experiments demonstrate the effect of absence or scarcity of pabulum. In these, injections of sterilized water into the peritoneal cavity causing an outpouring of serum—*i. e.*, pabulum —determined a suppurative peritonitis after inoculation with pyogenic organisms, because the peritoneum could not remove rapidly enough that which the germs fed upon and multiplied in, so that they overwhelmed the tissues which could readily have disposed of the original number of micro-organisms. In these experiments the slight traumatisms produced by the needle-punctures— *i. e.*, areas of lessened resistance—were the starting-points of the peritonitis.

These facts teach a two-fold lesson : (1) that wound-fluids serve as germ-food, hence must not be allowed to accumulate, and (2) that all unnecessary damage to tissue must be avoided, because it diminishes their germ-inhibitory and destructive power. The effects of lowered

tissue-resistance have been so often dwelt upon in previous lectures that nothing further need be said under this head. The general belief is untenable that atmospheric influences as heat, cold, moisture, etc., can of themselves interfere with wound-healing. There is no such thing as an individual predisposition to suppuration. Neither cancer, tuberculosis, nor syphilis can of themselves produce failure of primary union. Local or general depression of vitality by cold, etc., and congestions induced by such conditions, may render *efficient* the implantation of a *few germs* which, under other circumstances, would be disposed of by the tissues. If germs are absolutely excluded from the tissues, primarily and secondarily, wounds will pursue a similar course, whatever the atmospheric conditions may be, whether the patient has tuberculosis, has had syphilis, or is free from any taint. Of course it is not contended that the *time* occupied in repair does not vary with different patients, but it is asserted that *no interruption* of the processes will occur if germs be excluded. When operating or treating wounds, the surgeon may pursue either one of two courses. He may remove, inhibit the growth of, or destroy all germs upon his own hands, those of his assistants, his instruments, sponges, the parts to be operated upon, and in the dressings, nothing but that which is aseptic—*germ-free*—coming into contact with wounded surfaces. With proper precautions, this germ-free condition persists, healing occurring with the minimum of disturbance. The ideal outcome of Lister's work is this "*aseptic method.*" Owing to the impossibility of certainly excluding all germs, or germs in harmful numbers, in many operations within the mouth, rectum, and in most accidental wounds, measures must be adopted to destroy—rarely possible—or at least inhibit the growth of all micro-organisms which may have gained access to the wound ; still further, subsequent multiplication of germs in the dressings, whence secondary infection might result, must be prevented. Wound-treatment

founded upon such principles is that originally advocated by Lister, and is correctly termed "*antiseptic.*" As many of the procedures employed in antiseptic surgery are necessary preliminaries by which alone aseptic operating is rendered possible, they must be first considered.

Beware of being misled by laboratory experiments and those upon the lower animals. Lacking knowledge thus gained, our present successes would be impossible or rest upon an insecure basis ; yet the essential differences (1) that in the laboratory, chemical and thermic agents are afforded a freedom of access to the germs impossible clinically, and (2) that susceptibility to the action of pathogenic organisms is different in man and in the lower animals—indeed, varies among them—must be noted. Chemical germicides are often clinically either too much diluted by wound-fluids to be effective, or are precipitated or form inert combinations, or are mechanically prevented from gaining access to the germs: therefore laboratory results warrant experiments upon man, but are not conclusive.

Disinfection depends upon sterilization—*i. e.*, destruction or inhibition of the growth of all germs. Disinfectants or sterilizing agents should be germ-destroyers, but as often employed they are really only *inhibitors* of germ-growth. Although heat and heat alone can always be relied upon to be germicidal if employed of sufficient intensity and for a proper time, yet in practice certain inherent difficulties confront us. Thus it requires actual contact with boiling water for from one to five seconds to destroy the adult forms of most pathogenic organisms, the spores of anthrax require two minutes' exposure, while ten to fifteen minutes is required for their destruction by live steam. Steam by being superheated because generated under pressure will, of course, act in a shorter time. In this connection it must be remembered that spores are much more resistant than adult organisms, and that fortunately all other varieties of pathogenic

organisms, notably the pyogenic, succumb to temperatures innocuous to the anthrax bacillus, much more its spores. It is hardly necessary to point out that heat therefore cannot be employed to sterilize either the hands or the field of operation. A common mistake is made in forgetting that these statements only are true when the temperatures mentioned are applied to every adult germ and spore for the prescribed period, hence that even boiling water requires a longer time to be germicidal than in laboratory experiments, when employed to sterilize bulky, tightly-folded or wrapped dressings, or when, as is often true, germs are included in masses of coagulated pus, blood, or mechanical filth. These statements are still more true of the effects of steam, which must reach all parts of the dressings to be efficient. For this reason all dressings, instruments, etc., must be mechanically cleansed and so arranged that the steam readily gains access to all parts, the interior as well as the exterior. This is facilitated by the rapid expulsion of all air, the vent for this being below, that for the access of steam being above : from one to two hours had better be employed for sterilizing dressings, twenty minutes should suffice for instruments.

Placing cold, folded towels, tightly rolled packages of gauze, or large bundles of cotton in a steam-sterilizer for the minimum time employed in the laboratory to destroy pyogenic organisms, is sheer folly if aseptic results are expected. Hot air is the least efficient method of employing heat, because of the higher temperature and longer period of exposure requisite and its feeble power of penetration. Anthrax spores, none of which survive after two minutes' contact with boiling water, require three hours dry heat at 140° C. to produce the same effect, and much longer if the germs are enveloped in dressings, folded clothing, etc. ; of course pyogenic organisms are not so resistant, but proportionately the same law holds good.

The essential differences between the actual destruc-

tive effects of chemical germicides and their power of merely inhibiting germ-growth, with the dilution and chemical changes produced by the wound-secretions, account for the apparent conflict between laboratory theory and clinical practice. Even after the most elaborate disinfection of the hands or field of operation, it is rare to fail to detect germs by culture methods, *after the chemical conversion, for instance, of the mercuric chlorid into an inert salt.* Before the chemical change was effected in the bichlorid, these germs could not develop ; but they were not *dead.* Although capable, when used in comparatively dilute form, of destroying germs in the laboratory, in the strength safe to employ in a wound, our so-called germicides inhibit only, they do not kill the microbes, hence the importance of the mechanical removal and exclusion of all micro-organisms and the conservation of tissue-resistance, because under certain circumstances chemical inhibition may fail. The most powerful and at the same time available germicides and germ-inhibitors are mercuric chlorid, oil of mustard, iodin, carbolic acid, borax, boric acid, quinin and dilute alcohol : there are a host of others, but the mention of these must suffice. Oily materials preventing contact of the germicide with the micro-organisms, or albumin forming inert albuminates are the chief foes of chemical germicides, but by slightly acidulating the solution of mercuric chlorid for instance, albuminates cease to be formed, while soap, alcohol or ether will remove all grease. By far the most certain method of sterilization of both the surgeon's hands and the field of operation is the mechanical removal of *extraneous dirt, accumulated epithelium and germs,* and the *superficial epidermic layers* in which at least one pyogenic organism (staphylococcus epidermidis albus) has its normal habitat ; by the same means, much of the oily matter abounding in the skin is removed. If extra precautions are requisite, other agents especially adapted for the removal of fatty matters must be employed.

Mechanical Sterilization.—That of the surgeons', assistants', and nurses' hands will first be described. Sterilized water, as hot as can be borne, should be employed, and if this requires rapid cooling, refrigeration must be secured by the addition of cold *sterilized* water, some of which should always be on hand. In hospital practice, both hot and cold water are drawn directly from the receptacles in which they were sterilized, but in private practice, after filtration when necessary, all water should be thoroughly boiled, transferred to sterilized vessels, and be protected from dust by a sterilized towel. The cup or dipper used to transfer the water from the vessel to the receptacles must always be carefully replaced in the boiling water to maintain its asepsis. Nail-brushes, best made of vegetable fiber, should always be sterilized by boiling for five minutes or exposure to live steam for fifteen minutes, be carefully rinsed free from all soap after use, be re-sterilized by heat, and kept in carbolic, not bichlorid, solution, because soap will be decomposed by the latter. Although it may be true that all soaps made by heat are sterile and that potash soap is an active germ inhibitor in the proportion of 1 : 5000, yet it will be more prudent to combine with the soft-soap 5 per cent. of hydronaphthol or thymol, to insure that the soap itself is sterile. After thoroughly rubbing into the hands and arms an abundance of soap, the nail-brush and hot water must be vigorously used, especially beneath and around the nails for from two to five minutes. Carefully remove all soap with sterilized water, clean the finger-nails and around them with a nail-cleaner, and immerse the hands in alcohol or alcohol containing 5 per cent. of dilute acetic acid, or wash off with ether to remove all remaining oily matters. Rinse off again with sterilized water, to remove the last traces of soap, and finally immerse—not merely dip —the hands in a 1 : 2000 solution of mercuric chlorid for not less than three, preferably five, minutes. Instead of corrosive sublimate, ordinary mustard-flour mixed in

the hands into a thin paste with sterilized water, used with gentle friction for from two to five minutes, and then removed with abundance of sterilized water, will prove a most successful germicide. Scrubbing the hands with a sterilized brush while in the mercuric bath adds to the efficacy of the germicidal solution, but it must be distinctly understood that the chemical is merely supplementary to the really important part of the sterilization, serving to inhibit the growth of those microbes which have escaped mechanical removal, or those which are normally resident in the epidermis, hence cannot be all removed mechanically. It would seem superfluous to warn against wiping the hands on an ordinary towel after this final cleansing, or to avoid touching anything but the disinfected instruments, towels or field of operation ; yet this fatal blunder is too common for me to omit a caution on this point. Should circumstances arise necessitating the use of a towel, a recently sterilized one or one wrung out of an antiseptic solution must be employed. Accidental contact with unsterilized objects, as clothing, the hair, or beard of an assistant demands, if slight, washing in a germicidal solution and rinsing this off with sterilized water. During operations about the head and face the hair must be covered with an aseptic or antiseptic towel carefully secured in place by pinning. Fouling of the fingers with buccal or nasal mucus, feces, or pus necessitates a repetition of one or all of the original procedures. Mustard-flour is especially to be recommended, promptly sterilizing, and removing the odor of feces.

LECTURE XXIX.

Sterilization of the Field of Operation.—The
same principles just enunciated are applicable and are
to be carried out in practice by almost identical meas-
ures. When possible, a general warm bath should
be taken, after which recently laundered clothing
should be donned. Carefully shave *all* surfaces, ap-
parently hairy or not, and after washing off the loose
hairs, follow with prolonged, gentle scrubbing with
sterilized brush, soap and hot water, especial attention
being paid to such parts as the axilla, pubes, umbilicus,
and scalp. Alcohol and acetic acid, or ether, must be
used to remove all grease, freely bathing or gently
scrubbing the parts. Finally, carefully scrub with a
brush and plenty of a 1 : 2000 mercuric chlorid solution—
possibly with a cream of mustard-flour—and, after free
flushing with sterilized water, apply a wet bichlorid
dressing or one containing 2.5 per cent. of carbolic acid ;
this latter is especially applicable to regions where much
oily matter is to be expected, as the scalp or axilla.
This dressing should only be removed after the induction
of anesthesia, when the parts should again be cleansed
with a germicidal solution, removing this by free ablu-
tions with sterilized water. Additional precautions
must be taken prior to brain operations, those about the
feet, hands, or those involving the opening of non-sup-
purating joints. The oily, sebaceous secretions of the
scalp are liable to render the germicides inert or nearly
so, while the accumulations of thickened epidermis on

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the hands and feet secure the germs against either mechanical removal or chemical destruction. The simplest adjuvant after shaving, alcohol, etc., is to apply, for a few hours, a thin layer of soft-soap on absorbent cotton as a poultice, which must be removed by free irrigation with sterilized water, although a weak carbolized solution is preferable, after which the permanent antiseptic dressing can be applied until the time for operation has arrived.

Certain modifications become requisite when dealing with such cavities as the mouth, vagina, rectum, or where operative interference may possibly open into them. All of these, with the exception of the bladder, teem with organisms, and in none of them can strong chemical germicides be employed.

Mouth.—Careful attention to the condition of the teeth, removal of all salivary calculus, extraction or filling of carious teeth, mouth-washes and sprays containing thymol, boric or salicylic acids—one of the best being listerine—with such treatment of any nasopharyngeal catarrh as circumstances will admit of, is the most desirable preparation for operations for cleft-palate, temporary or permanent resection of the jaws, ablation of the tongue, nasopharyngeal tumors, etc. The after use of iodoform-packing is also efficacious.

Vagina.—Mechanical cleansing is here our mainstay. Abundance of soft-soap on a vaginal mop made of sterilized cotton or gauze, or a long soft jewellers' brush, should be employed to scrub the vagina, free irrigation with sterilized water being employed while doing this.

Follow this by douching with a 2.5 per cent. solution of carbolic acid or a 1 : 2000, or even 1 : 1000, mercuric solution, provided all the chemical be removed by free flushing with sterilized water and care be taken to empty the vagina by firm pressure on the posterior commissure ; permanganate of potassium or Thiersch's solution may be substituted; when vaginal hysterectomy is contemplated, these procedures had best be adopted

some hours before operation, and repeated on the operating table. Dilatation of the cervical canal, curettage, and the application of the thermocautery should also precede in most instances such operations. Curetting followed by milder but efficient disinfection must be employed before trachelorrhaphy.

Intestines and Rectum.—Thorough purgation and liquid diet must be employed, the former being sometimes properly secured in tight rectal strictures by a previous inguinal colotomy. Free and repeated lavage of the colon with sterilized water in the "knee-elbow" position is indicated shortly before operation.

After the patient has been anesthetized, when dealing with the rectum, a good-sized sponge secured by a strong thread should be passed well up the rectum, and the field of operation be freely swabbed and flushed with Thiersch's solution. In certain cases of ulcerating neoplasms, the surfaces may even be cautiously curetted and touched by the thermocautery. The internal exhibition of salol or thymol may possibly aid in securing asepsis. Subsequent packing with iodoform gauze will aid in maintaining the asepsis. Temporary proximal and distal ligation or clamping of the intestines, walling off of the healthy peritoneum by iodoform-gauze packing, stripping away by the fingers the intestinal contents before applying the clamps, mopping up rapidly with pledgets of aseptic gauze all remaining fluid after incising the gut, usually secure asepsis during intestinal operations. When the loop or loops of gut involved in the procedure can be placed entirely outside the belly, as is often the case, complete isolation from the general peritoneal cavity can be effectually secured by gauze-packing, and removal of the bowel contents by flushing with sterilized salt-solution. During operations for appendicitis or pustubes, when healthy peritoneum is incised—*i. e.*, when the opening is outside the adhesions—careful packing with sterilized gauze should precede opening the pus-focus, all infected material should be removed as com-

pletely as possible, and, the soiled gauze having been cautiously removed, a clean packing should be introduced to remain until adhesions have shut off the general serous cavity. The Trendelenburg posture must be carefully avoided when dealing with pus-foci, lest the remaining healthy portions of the peritoneal cavity become flooded with infectious material.

The object in view when preparing the field of operation thus far has been the prevention of contamination of the deeper parts by germs derived from without, or resident upon or in the integument or mucous membrane. While not, strictly considered, preparation of the field of operation, the special measures adapted to prevent infection, by pus or secretions, of the peritoneum, cerebral membranes, pleura, pericardium, and healthy bladder, can best be considered in a general way here.

Cerebral Membranes.—Except when operating for a known or suspected abscess no precautions other than those already mentioned are demanded. Before the abscess is opened, the site of the purulent collection must be surrounded with (aseptic) iodoform gauze to avoid a generalized leptomeningitis, after which every particle of pus should be removed by careful irrigation with sterilized salt-solution. If the pus be especially virulent, such slight risk as attends the employment of a weak bichlorid solution may be safely encountered. If desired, instead of the sterilized water, Thiersch's solution may be employed with impunity.

Stomach.—When fecal vomiting exists from any cause, or preceding the operations of gastrostomy, gastrectomy, gastro-enterostomy, gastrotomy, etc., gastric lavage should be done with Thiersch's solution or the normal salt-solution, the latter being usually abundantly sufficient. By suture of the stomach to the parietal peritoneum during the operation of gastrostomy before incising the viscus, or after bringing the organ as far as possible into the parietal wound by careful walling off by packing with

sterilized or iodoform gauze, peritoneal infection must be further guarded against.

In gastrectomy and gastro-enterostomy, clamps or temporary ligatures must be applied to the stomach (if possible, in gastrectomy) and to a segment of intestine, carefully stripping this of all feces, drawing the loop outside the abdomen when feasible, packing gauze about it, and then, after opening, thoroughly but rapidly removing what little contents remain by pads of dry (sterilized) gauze ; or, when outside the abdomen, by flushing with sterilized salt-solution.

Liver, Gall-bladder, and Ducts.—Similar procedures are indicated when dealing with these structures.

Accidental Operative Wounds of the Pleura, Pericardium, and Peritoneum.—If the conditions are favorable, immediate suture is preferable, but if subsequent manipulations might again open the wound, temporary gauze-packing must be done. At the close of the operation this may be removed and the wound sutured, or a clean packing (the so-called Mikulicz drain is the best way to employ packing in such cases) allowed to remain to induce adhesions, aid in disinfection, and serve as a drain if the serous membrane has certainly or probably been infected during the operation.

Bladder.—Although the introduction of a few pathogenic germs into a healthy organ may prove harmless, owing to their prompt removal with the urine, they may produce the most disastrous results. The surgeon usually has to deal with an already diseased viscus containing a stone or tumor, and here the introduction of streptococci or staphylococci—the usual causes of cystitis when infection follows the use of instruments—is certain to give rise to trouble. As, in nearly every instance, operations involving the bladder demand the introduction of instruments *per urethram*, an antecedent aseptic condition of this canal must be secured. Normally, the urethra is alleged to harbor many germs which, if carried into the bladder, can originate a cystitis. When

possible, most careful lavage of the urethra must be performed by a retroacting deep urethral catheter, using sterilized salt, borosalicylic, or bichlorid solution according to the condition present.

Any introduction of instruments is to be deprecated if urethritis be present, and, when unavoidable, only after a most careful employment of the measures indicated. All operations upon the bladder, except where impassable stricture exists, must be preceded by urethral first, then vesical, lavage. As soon as any such impediment is overcome, most thorough washing out of the bladder and urethra should follow or be used during the operation—first with an antiseptic solution, then with an abundance of sterilized salt-solution. For a few days previous to operation, the internal use of salol, quinin, or boric acid often markedly changes the character of unhealthy urine, and is imperative when the upper urinary passages or kidneys are involved in the infective process. Salol must not be exhibited for a lengthened period, owing to its injurious action upon the kidneys.

Antiseptic Surgery.—This aims to *remove, destroy, or neutralize* the noxious effects of germs which have gained lodgement in the tissues. Heat when applicable in the form of the cautery, is most efficacious, directly destroying the germs and the tissues in which they reside, converting these into an aseptic eschar, which must separate by processes which commonly leave a layer of healthy granulations, usually a competent bar to the further ingress of germs. *Chancroids, lupus, tubercular disease*, and such spreading processes as *hospital gangrene*, are amenable to this treatment. Except when employed as potential cauteries, chemical substances cannot be used in sufficient concentration to destroy all germ-life in an infective process. It is true that hospital gangrene and some few analogous conditions have been successfully combated with pure bromin, carbolic, and chromic and fuming nitric acid, and strong solutions of chlorid of zinc; but these must destroy, as the hot iron

does, all the infected tissue, otherwise, after a period of quiescence, the disease will break out anew. Many of these and other substances employed, as *corrosive-sublimate paste*, are poisonous if applied to large areas. Most usually, disinfection by lotions is limited to the superficial portions of the infected area. *Removal of all infected tissues* by excision—when limited by curetting or dissection with knife or scissors—is most efficacious, as in some carbuncles or in anthrax. Complete mechanical removal being impossible, partial excision may be supplemented by the actual cautery, as is often done in anthrax. *Incisions by relieving tension*, giving exit to discharges and sloughs, mechanically remove many germs and toxins, besides rendering possible the access of germ-inhibitory substances. *Irrigation* with a powerful stream is an important mechanical adjuvant to incision, but distention of any cavity must never be permitted, two tubes being employed or a counter incision made. Rough handling must also be avoided, as calculated to rupture granulations or the tissues, and thus open up new avenues for infection. An exception to this rule is often presented by certain ischiorectal abscesses, where the cavity should be made a simple one by breaking down the irregular partitions.

Hydrogen peroxid is useful to disinfect irregular cavities when not too large. In all cases where it is not certain that all infection has been removed, tubes are preferable to packing, because the solid portions of the pus cannot be removed by capillary action ; but packing to secure the prolonged contact of iodoform with sloughs or infected tissues, because of its germ-inhibitory and toxin-destroying property and to prevent re-infection, is often useful and may be combined with tube-drainage. *Drainage can be dispensed with when the discharge becomes serous* and small in amount, but gradual shortening of the tubes and lessening of the quantity of packing at each dressing until the cavity becomes nearly effaced must precede the total suspension of drainage.

At each dressing, any sloughs remaining must be removed, but frequent irrigation is only necessary for anfractuous cavities, or where efficient drainage is anatomically impossible. Sometimes continuous irrigation or the continuous bath is indicated, as in extensive cellulitis of an extremity, when only non-toxic chemicals, as boric or salicylic acid or aluminum acetate, should be employed, lest systemic poisoning occur. To favor drainage and relieve congestion, the powerful aid of *position* must be invoked. When packing a wound, each recess should have its own piece of gauze, one end being left protruding, and careful count be kept of the number of pieces. Replace each piece with a clean one as the soiled one is withdrawn, when re-dressing, although this rule often cannot be followed. Moist antiseptic dressings are indicated until only serum is secreted, because drying of discharges on the dressings would interfere with drainage ; moreover, the wound-discharges, being septic, would tend to infect the wound if the germs were permitted to multiply unchecked. A dry aseptic dressing may be applied when the wound becomes aseptic.

Drainage.—Many aseptic operations require no drainage. Increasing experience and improved technic lead each surgeon gradually to discard it. In some form it becomes necessary—(1) *when much bloody serum will be poured out;* (2) *where cavities must be left;* (3) *where perfect asepsis or its maintenance is doubtful;* (4) *where infection has occurred.*

Drainage may be direct—*i. e.,* where discharges are removed by tubes, etc. ; or indirect—*i. e.,* by leaving a part or a whole of a wound open ; packing or employing secondary suture ; by buried sutures, compresses, bandages, etc., so disposed as to leave no cavities in which fluids can collect.

Direct drainage is *tubular* or *capillary.* Well-annealed glass tubes with lateral openings are best when of the proper length, because non-collapsible and readily sterilized by boiling. Rubber tubes are more com-

monly employed because capable of being used of any length.

The tubing selected should be sufficiently rigid to prevent kinking. Boiling for five minutes in soda-solution or placing them in the steam-sterilizer for twenty minutes will sterilize them. They should be cut in lengths and stored in a 5 per cent. carbolic solution. Tubes should reach well into the cavity, but not impinge upon its base, and with this end in view must be cut flush with the skin, being secured *in situ* by a sterilized safety-pin thrust through the tube, or by a special stitch through one margin of the wound including the tube. No tube should rest upon or against a nerve or blood-vessel ; if of glass, its end must not rest against an intestine.

Capillary drainage is only adapted for the removal of blood or serum and must never be used for pus. A *strip of gauze* protruding from an angle of a wound is sometimes employed. *Sterilized horsehair* or *fine catgut* are the usual materials. Certain precautions must be observed.

Secure the middle of a bundle of from twenty to forty strands of hair or gut to the deepest portion of the wound
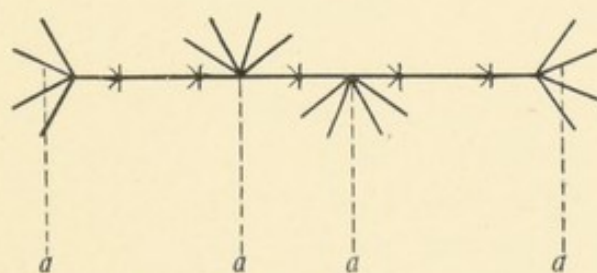
FIG. 22.—Strands of catgut or hair (*a*) improperly arranged for capillary drainage—*i. e.,* diverging, and not covered with a protective to prevent desiccation.

by a stitch of catgut. If buried sutures are employed, bring four strands between each two stitches. The same disposition is to be made between each pair of skin-sutures. The threads must be carefully smoothed out, so as to be parallel and in contact, their ends cut

off squarely, and a piece of protective laid over all (Fig.
23).   This must extend some distance beyond the ends
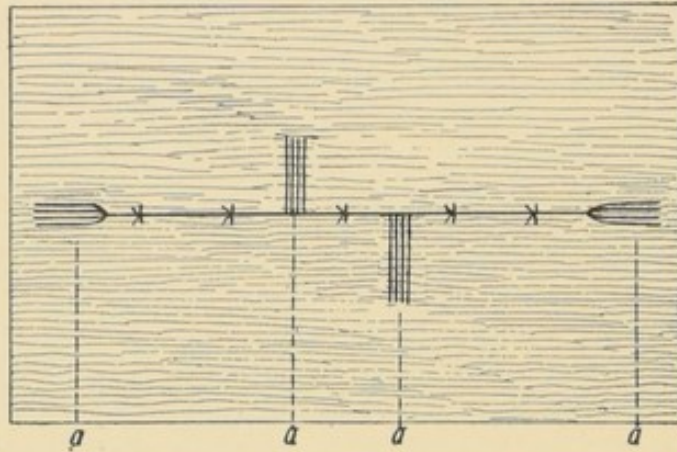
FIG. 23.—Strands of catgut or hair properly arranged for capillary drainage—
i. e., parallel, and covered with rubber protective to keep them moist.

of every drain, otherwise they become agglutinated by
desiccation and cease to exercise capillary attraction.
Neglect of these precautions invariably leads to failure.
Catgut is absorbed, but horsehair must be withdrawn at
the end of forty-eight to seventy-two hours, either en-
tirely or a few hairs at each dressing.

LECTURE XXX.

STERILIZATION OF INSTRUMENTS, LIGATURES, SPONGES, AND DRESSINGS.

Instruments.—All instruments should be entirely metallic, with smooth plane or simply curved surfaces. If complex, they must be readily separable into their component parts to permit mechanical cleansing. Wooden or ivory handles are damaged by heat, and from the inequalities of their surfaces are hard to cleanse mechanically ; still they can be sterilized by the exercise of care. Aluminum being attacked by alkaline fluids is therefore undesirable. Too much stress has been laid upon the receptacles in which instruments are stored. It is demanded that they be constructed of enamelled iron and glass, tightly closing to exclude dust. Even the pocket-case must be metallic or made of canvas, so that it can be frequently sterilized. Unquestionably, if instruments are sterilized for operation at the surgeon's office, an aseptic case is requisite ; but few adopt this plan. While desirable, these are unnecessary refinements, deluding to those not thoroughly versed in aseptic principles, because the asepsis is only relative, and ignored by the expert because he never trusts to such inadequate precautions, but specially sterilizes his instruments for each operation. I have more than once taken a polished steel instrument off a velvet-covered shelf in a general hospital, and been unable to detect germs by culture-methods, showing the *possibility* of maintaining instruments aseptic for some time after their sterilization, although the propriety of attempting this is questionable.

The importance of mechanical cleansing by soap, hot water, and friction is demonstrated by recent experiments where smooth, metallic, or even gum instruments have been made germ-free by brisk rubbing first with a wet (sterilized) cloth, then for a few minutes with a dry sterilized towel. All instruments after use should be mechanically cleansed and kept polished.

Chemical disinfection of instruments has long been abandoned in favor of heat, except to meet special indications, because of its unreliability and the injurious effects exerted upon instruments, destroying the cutting edge and polish, and interfering with the smoothness of working of complicated ones. Dry heat being tedious in its application, injurious to temper unless skilfully used, and requiring cumbersome apparatus, is rarely employed. Some prefer in hospital work "live steam," the water from which it is generated being charged with one per cent. of washing soda to prevent rusting; this
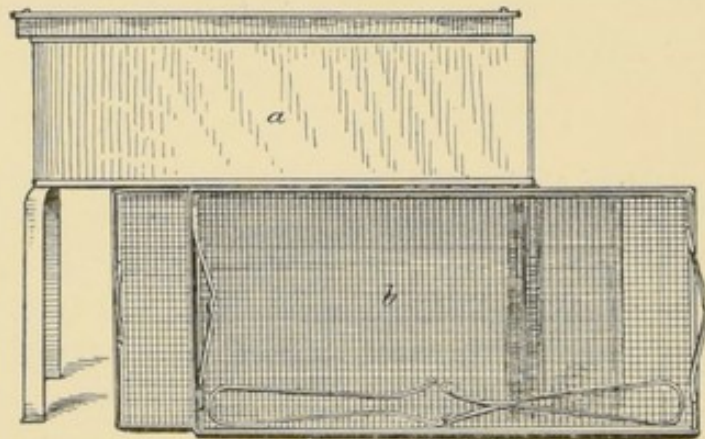
Fig. 24.—Schimmelbusch instrument-sterilizer: *a*, copper tank; *b*, copper gauze trays.

also adds to the germicidal power. As twelve minutes will destroy anthrax spores, from five to ten minutes exposure will kill all pyogenic organisms. Of course, it is better not to rely on the minimum time of exposure. In private practice, instruments can be more easily sterilized by boiling in water containing one per cent. of soda, any vessel large enough to contain the instru-

ments serving to boil them in. Exposure of pyogenic organisms for three seconds to this boiling soda-solution will destroy them, and even anthrax spores perish in two minutes, hence five minutes amply suffices for clean instruments. In hospital work, Schimmelbusch's apparatus (Fig. 24), with its separate wire trays, is the best. Any instrument accidentally infected during an operation can be sterilized by dipping into the boiling soda-solution for a few seconds. While the sterilization of metallic bougies and catheters by heat or boiling presents no difficulties, it is far otherwise with the elastic (English), the soft (French), and the pure rubber (Nélaton), instruments. If oily substances be used as lubricants, the instruments should be soaked for a short time in warm (not hot) solution of washing soda previous to sterilization. If glycerin be employed, simple sterilized water will suffice. The soft-rubber catheters may be sterilized by immersion in soda-solution for fifteen minutes, the water being kept just off the boil ; a too frequent repetition of this will render the instruments dangerously brittle. The statement so generally made that soft-rubber instruments will not withstand frequent boilings, for the purpose of sterilization, has, in our experience, proved erroneous. In a number of instances, soft catheters have been subjected to prolonged boiling in distilled water thrice daily for two months, and they have been found to become neither brittle nor rough. The asepticity, in whatever way secured, must be maintained by suspending the catheters in a tall jar containing a 5 per cent. carbolic acid solution or a 1 : 2000 bichlorid one. They must be rinsed in sterilized water before using, and carefully cleansed before replacing them in the jar, using a soda-solution if grease instead of glycerin is employed as a lubricant. A short immersion in a 20 per cent. carbolic acid or 1 : 500 mercuric chlorid solution, removing all traces of these by sterilized water before using, will probably render aseptic the other varieties of catheters ; storage in anti-

septic solutions seriously damages these instruments. Exposure in proper metallic receptacles to the vapor of formaldehyde (moistened) is claimed to be efficient for all varieties of catheters.

All inhalers, mouth-gags, tongue-forceps, throat-mops or sponges, hypodermic syringes, and hypodermic solutions must be most carefully sterilized. Extemporaneous inhalers should be made out of sterilized or recently laundered towels, while the usual types of inhalers employed for the administration of either chloroform or ether can readily be sterilized by boiling or steam. One of the chief grounds for not using gags and inhalers without sterilization is because of the danger of conveying syphilis or pathogenic germs, as those of diphtheria, etc. Fatal sepsis, tetanus and malignant edema, and serious abscesses have so often followed the use of non-sterilized hypodermatic injections, that asepsis is essential. Boiling the hypodermic pellet and needle in a spoon over a lamp, or by means of a wax match, can always be done, and will insure asepsis. When possible, hypodermic syringes should be so constructed as not to be damaged by boiling; but only an occasional renewal of the packing will become requisite if the instrument is occasionally filled with warm water and then carefully boiled for a short time. Repeated rinsing out with hot water has been declared to be efficient against pyogenic organisms, but an occasional boiling must not be omitted. Brisk friction of the skin with alcohol, followed by washing with a germicidal solution, must precede the introduction of the needle. Because vulcanite syringes can only be sterilized by repeated filling with and emptying of hot water, gravity should always be employed by preference instead of such instruments, using fountain syringes or irrigators, the receptacles, tubes and points of which can all be readily disinfected. In hospital practice a glass point should be employed for each patient, carefully disinfecting this after using. Syringes for enemata, either rectal or vaginal, with

their reservoirs should be similarly cared for, as neglect of these precautions has sometimes resulted disastrously. Rubber bags and tubing can be immersed in strong germicidal solutions and then rinsed off in sterilized water.

Sterilization of Dressings.—Cheese-cloth, butter-cloth, cotton, jute, moss, pine-sawdust, peat, ashes, asbestos-wool, sand, and innumerable absorbent substances have been employed, but cheese-cloth, butter-cloth, cotton, sawdust, and moss, are those most available. Moisture being essential to germ-life, an ideal dressing should *desiccate the wound—i. e.*, promptly abstract the wound-secretions, absorb them, and also permit rapid evaporation of the fluid portions ; they must be aseptic, and capable of maintaining this condition by preventing the multiplication of germs. All oily material should be removed from the cheese-cloth or other textile fabric, the cotton, etc., by means of boiling for fifteen or twenty minutes in a solution containing 5 per cent. of washing soda, rinsing in cold water, and drying—the dressings will thus be made perfectly absorbent. Sawdust, oakum, moss or gauze must be sterilized by dry or moist heat or by immersion in a germicidal solution, if moist dressings are to be employed. Dressings can be sterilized and maintained aseptic by placing them in metal boxes, as those of Schimmelbusch, (Fig. 25) or in sterilized fruit-preserving jars, screwing on the cap immediately upon removal from the sterilizer. Dressings can be efficiently sterilized by exposing them, *loosely arranged*, to steam heat for thirty minutes in any steam-sterilizer (such as Arnold's or Schimmelbusch's) which fulfils the indication of previous warming of the dressings and generation of steam under some pressure, *and in which the steam enters from above;* a longer exposure is desirable if time suffices, and nothing like tight rolling or close packing is permissible, otherwise hours exposure will not *destroy germs located in the interior of the dressings.* Maintenance of the aseptic

condition of the dressings after an aseptic operation is
secured by so arranging them as to favor rapid drying of
wound-secretions.   By pinning up the loose dressings in
the towels upon which they lie in the sterilizer, any
necessity for metal boxes or jars is done away with.   If
no sterilizer is available in private practice, boiling of
aprons, dressings, and towels for fifteen minutes in soda-
solution (or plain water) will thoroughly sterilize them,
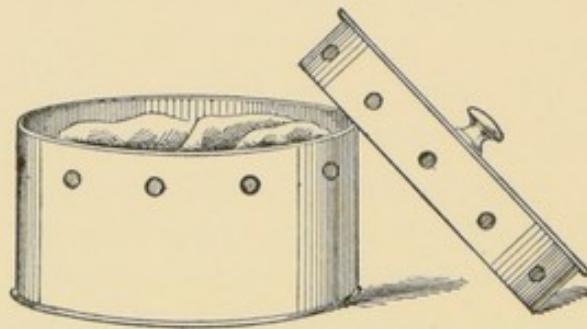
Fig. 25.—Schimmelbusch dressing-sterilizer, with fenestra in box and cover
to permit entrance of steam; the cover being partially rotated, closes the open-
ings after removal from the sterilizer.

and if dry dressings are absolutely required, draining
off the water in the vessel, covering this with another
sterilized one, and placing all in the oven or over the fire
until dried will secure the desired result.   No reliance
should be placed upon commercial antiseptic gauzes or
ligature materials when it is possible to prepare them
yourselves.   Chemical sterilization is best effected by cor-
rosive sublimate—when not contraindicated—employed
in the strength of 1 : 2000.   The gauze (if possible,
previously sterilized by heat), cut and folded, should be
steeped, not be merely dipped in the solution, and when
applied, wrung as dry as possible.   *Sponges* may be
marine, or better, made of knitting-wool, absorbent
cotton, or wood-wool, loosely gathered up and secured
within a double layer of absorbent gauze ; or pads of
sterilized gauze may be substituted, so folded that no
ravellings can be left in the wound ; any of these are
cheaper than marine sponges and easily sterilized
by heat, the best way being to boil them for fifteen

minutes in the 1 per cent. soda-solution, although exposure to live steam for thirty minutes will do. The preparation and sterilization of marine sponges is so troublesome, and the gauze sponges are so superior, that I hardly think it worth while to spend time describing methods you will be unlikely to make use of.

Sterilization of Ligature and Suture Materials.— Heat is superior to chemicals and can be employed in a number of ways : usually both methods are combined. Metallic wire, horsehair, silk-worm gut, silk or flax thread, after having been loosely rolled upon glass spools or rods should be boiled for thirty minutes in ordinary water and stored in a 5 per cent. carbolic acid solution, or in a 1 : 3000 corrosive sublimate solution, unless they are to be immediately used, when they can be temporarily kept in the water in which they were boiled. If the theoretical objection to the presence of any chemical in the ligatures, because diminishing the resistance of the tissues, be considered important, store the ligatures in recently boiled alcohol.

Animal Ligatures.—Of the many methods employed I shall describe only three. 1. Roll loosely on glass spools or rods, or tie in skein-form ; place in a large jar of absolute alcohol provided with a screw cap (as a preserve jar), only closing the cover moderately tight to prevent unnecessary waste of alcohol ; place in a waterbath and subject to the boiling temperature for two hours, screw the cover firmly down, and remove the gut as wanted, with sterilized forceps. 2. A still better method is to tie the gut into skeins ; wrap each in a small piece of gauze; dry in a hot-air sterilizer at 80° C. for three hours; place in a vessel containing cumol and boil in a sand-bath (boiling-point 165–170° C.) for one hour ; remove and drive off cumol with heat in dry-air oven ; remove with sterilized forceps the gauze and drop the skeins into test-tubes sterilized by heat, or into glass tubes one end of which has been sealed by fusion ; if employing a test-tube, cork with a cork sterilized by

heat and seal with hot wax ; if a glass tube, close the open end by the blow-pipe flame. This cumol gut is absolutely reliable and is more easily prepared than is believed, the only special care requisite being to suspend the gut in the cumol so as to prevent its touching the sides of the vessel in which it is boiled, otherwise it will be burnt. A file-mark around the fused tube will secure its ready fracture when needed for use, if the hands are guarded by a sterilized towel. 3. Place receptacle and glass spools in a steam-sterilizer for forty-five minutes, or boil in soda-solution for ten minutes ; roll the gut on the spools and soak in ether for forty-eight hours to remove grease ; pour off the ether, and substitute a solution of corrosive sublimate parts 10, absolute alcohol parts 800, distilled water 200 parts ; replace this in twenty-four hours, because it will become turbid ; allow the gut to remain for seventy-two hours ; store in the same solution, or, if it is desired to render the gut moderately stiff, place in a mixture of alcohol 80 parts and glycerin 20 parts, both having been previously boiled.

By treating the gut with chromic acid, the ligature does not disappear so rapidly. Macewen places the commercial gut in an aqueous solution of chromic acid (20 per cent.) 1 part and glycerin 20 parts for a period of two months ; it is then washed and stored in a 20 per cent. solution of carbolic acid in glycerin : instead of starting with the commercial gut it is far safer to employ the cumol gut.

ASEPTIC SOLUTIONS; CHEMICAL GERMICIDES; ANTISEPTIC AND ASEPTIC OINTMENTS.

Sterilized Salt-solution.—This is a 6 per mille solution of sodium chlorid prepared by boiling for fifteen minutes.

Corrosive Sublimate.—As most waters contain lime, which decomposes this drug, acetic, tartaric, citric, or some mineral acid, as hydrochloric, or table-salt, must

be added. The vegetable acids and table-salt may be used in the same quantities as the mercurial salt, the hydrochloric, so as to render the solution faintly acid to litmus paper. Moreover, these acids prevent the formation of an inert albuminate when used in wounds. The strength of mercuric solutions is for irrigation 1 : 15,000 to 1 : 5000 ; and 1 : 2000 to 1 : 1000 for infected wounds, disinfecting hands, and the field of operation ; for bone-cavities, possibly as strong as 1 : 500. Where strong solutions are employed, or even weak ones, it is good practice to secure the removal of all traces of mercury from the wound by flushing with sterilized water. Patients must be closely watched lest salivation or gastro-intestinal irritation result, shown by abdominal pain and frequent mucous or bloody stools. Locally, dermatitis and vesication may result. Carbolic acid is employed in 1, 2, and 5 per cent. solutions, the weaker for irrigation and instruments, the stronger for septic wounds. It is readily absorbed, requiring watching, especially in children. An olive-green tint of the urine is usually an early symptom of poisoning, which soon may be followed by cerebral and circulatory symptoms, as headache, vertigo, coma, eclampsia, vomiting, feeble heart-action, suppression of urine and entorrhagia, in the worst cases terminating fatally. Locally it is irritant, often causing eczema, while the long application of strong solutions has often determined dry gangrene of the fingers and toes.

Salicylic acid is best used as Thiersch's solution— viz., acid. sal., parts 2, acid boric., parts 12, aqua, 1000 parts. This is a weak, non-poisonous germicidal combination. It may also be used in ointment form.

Acetate of aluminum in 1 per cent. solution is effective, safe, and especially adapted for irritable skins.

Potassium permanganate, 1 : 500 to 1 : 3000, weak but safe, is useful in the mouth, urethra, and bladder.

Hydrogen peroxid, a powerful disinfectant, but slightly if at all germicidal, is especially useful for cleansing

foul, sloughing wounds, by the generation of gases mechanically forcing pus and sloughs out from the deepest recesses of wounds. It may be used in full strength or variously diluted : free exit for the gases must be provided lest infective materials be forced into the surrounding healthy tissues.

Zinc chlorid, 5 to 20 grains to the ounce, is most efficient for foul wounds and bone-cavities.

Iodoform is invaluable, acting by virtue of the iodin set free in the presence of infected living tissues *in the absence of air*, neutralizing the ptomains, etc., and somewhat inhibiting germ-growth. It is poisonous, especially to the old and anemic, and often produces dermatitis. As iodoform-gauze it is the chief reliance in the oral, rectal, vaginal, and vesical cavities, or, when secondary suture is to be employed, as a protective dam in various conditions, and as packing to arrest oozing after certain abdominal operations. Slight poisoning is shown by headache, mental depression, and anorexia, or nausea and vomiting ; more severe cases exhibit insomnia, have a rapid pulse, high temperature, delirium (sometimes maniacal), coma, and convulsions ; iodin can usually be detected in the urine. These symptoms may develop early or late, may disappear upon the removal of all dressings containing iodoform, or may persist ; they most often follow the use of large amounts of iodoform, but the reverse has been observed (see page 175).

Bismuth subnitrate is non-irritant, is desiccant, but poisonous when used in large quantities ; it may be employed as powder, emulsion, or ointment.

Zinc oxid is a weak, non-poisonous, desiccating substance, and may be employed in the same way as bismuth.

Acetanilid is far more powerful than the preceding two, is safe and efficient ; the powder is simply dusted over wounds. Numerous others might be mentioned, but those recommended are both those most available and useful.

LECTURE XXXI.

TREATMENT OF WOUNDS; VARIETIES OF WOUNDS.

Treatment of Wounds.—A wound is a solution of continuity suddenly effected by anything which cuts or tears. When the skin remains intact, the injury is a *subcutaneous wound*, and little if any constitutional disturbance results, the lesion being repaired by those processes erroneously termed "simple adhesive inflammation" or "aseptic inflammation." Wounds are called *incised* when caused by a sharp-edged object; *contused*, when produced by a more diffused force dividing the tissues, leaving the wound-surfaces bruised; *lacerated*, when irregularly torn; *punctured*, when the depth much exceeds the superficial area.

Incised Wounds.—The pain is apt to be less than in the other varieties, because the tissues are cleanly divided, the vulnerating object not dragging upon or injuring contiguous sensitive parts. Bleeding is apt to be more free than in lacerated or contused wounds, varying, however, with the vascularity and structure of the tissues. Thus, facial wounds bleed freely, even if no considerable vessel be wounded; scalp-wounds bleed profusely, not only because of the free blood-supply, but because the vessels cannot readily contract and retract in the dense tissues. Retraction of the edges of incised wounds always occurs, varying with the subjacent structures and the line pursued. Proper planning of incisions therefore may lessen the number of sutures requisite. Skin and facial wounds passing across the course of underlying muscular fibers gape widely. If made parallel to their course, their edges will remain nearly in contact

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and require but few sutures. Skin and muscle retract most freely when the former is divided across the "line of cleavage"—*i. e.*, the direction in which the skin splits most readily when subjected to strain[1]—the latter, at right angles to its fibers : inflammatory tension of the subjacent parts increases the gaping. If asepsis is maintained and proper coaptation is secured, union occurs by the normal processes detailed in Lecture III. When the epithelium is thin, the wound-edges may present a faint blush for from two to three days, but practically nothing more will be observed. Do not forget that although at the end of seventy-two hours union appears to be firm, this is due to a mechanical, not a permanent, vital bond of union, which requires some days longer to form, as described in Lecture III. If true inflammation occurs, the faintly congested wound-edges become decidedly reddened, swollen, and tense ; throbbing pain is felt, union fails, and pus appears. The possible chill, and the nervous and circulatory constitutional symptoms of septic traumatic fever appear, to continue until effective drainage or the formation of granulations prevents further absorption, union now necessarily occurring by granulation, unless two aseptic granulation-covered surfaces can be induced to unite by fusion—*i. e*, secondary adhesion takes place.

Treatment.—Perfect asepsis being presupposed, all bleeding must be permanently arrested by torsion or ligature, hot water, etc. If for any of the reasons given in previous lectures an amount of oozing continues such as will not be arrested by the compression of the flaps and dressings, tamponade with iodoform gauze may be resorted to, which later must be removed and secondary suturing employed, when the wound will heal quite as rapidly as if it had been put up at once. The packing can be safely removed in forty-eight hours if the oozing comes from capillaries and small veins, but if employed

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[1] See Langer's Observations as quoted by Kocher, *Op. Surgery, Am. Trans.*, Wm. Wood & Co., 1894, pp. 25–28.

to check bleeding from a wounded cerebral sinus or a large vein, either of which is often hard to secure by a ligature or forcipressure, at least a week had better elapse before disturbing the tampon, unless evidences of retention of infective material rendered this delay dangerous. Remove foreign bodies with forceps, and gently cleanse the surfaces with a stream of sterilized salt-solution, or sterilized water, avoiding antiseptic solutions unless imperatively demanded. If it is deemed advisable to employ the "dry method," use bits of dry sterilized gauze, or cotton, to cleanse the parts, applying only gentle pressure, not "scrubbing" the parts, as is too often done. Never distend any wound-recess with the irrigating fluid. With a deep wound or one where the tissues retract so as to leave spaces, either buried sutures must be employed to efface all "dead spaces," or drainage be employed, supplemented by compresses, bandaging, etc.; this is because, although the coagulable portions of the wound-fluids contain enough nucleinic acid to be germicidal, serum usually does not. Moreover, the accumulation of blood and serum will mechanically break down the mechanical bond of union formed by the coagulated exudate, and thus delay union. Drainage for an aseptic wound is only requisite for twenty-four to forty-eight hours. If infection has taken place or the success of disinfection be doubted, drainage had better be maintained until the dangers of sepsis are passed. Splints, position, compresses, and gentle bandaging by maintaining coaptation of the deeper parts and rest of all, will promote prompt union and do away with much of the necessity otherwise arising for drainage. Incised wounds are best closed by sutures, which may be interrupted or continuous. Union of cut muscle with muscle, fascia with fascia, and skin with skin is the ideal plan, the skin stitches being preferably passed only through the dense corium, avoiding the epithelium with its possible germs and certain scarring of the needle-punctures. Although seldom requisite, relaxation-sutures may be employed if

strain is likely to come on the coaptating sutures. Absorbable sutures should always be used when possible, especially when buried, but are too uncertain for use with the skin if there be any strain on the line of suturing. Although any pliable substance may be employed for suturing the skin, as silk, linen thread, silkworm-gut, etc., for the subepithelial stitch nothing equals silver wire, but this of course requires removal at the end of eight, ten, or fourteen days. Coaptation-sutures must only be tight enough to bring the surfaces gently into contact, less constriction impair the vitality of the tissues and favor suppuration. Strain can sometimes be advantageously removed from the coaptation-sutures, as was just mentioned, by relaxation-sutures. Needles should be only just large enough to carry the thread, and be curved or straight, depending upon whether the wound is on a free surface, a concave, or a convex one. Round sewing needles are best for suturing the bowel, bladder, or peritoneum. Curved surgeon's needles, with their sharp lateral edges ground off on a hone, are best for suturing the dura mater. Hagedorn needles serve well for skin and not especially vascular parts. Strips of gauze, fixed first on one side of the wound by collodion and then on the other after it has been approximated, are sometimes excellent substitutes or succedanea to sutures. This expedient can be used to excellent purpose after removal of sutures, to support the line of union. Never use adhesive plaster to directly approximate a wound, but it may be employed to take strain off stitches or to support a recent scar, if sufficient aseptic gauze be interposed. Aseptic compresses and bandages, exercising pressure through superimposed elastic materials, such as sterilized cotton, oakum, etc., serve as adjuvants to suturing by promoting drainage, securing quiet of the deeper parts, and thus relieving strain upon the stitches. In small uncomplicated operation-wounds, where no drainage is requisite, catgut sutures may be used and the whole line of union sealed by a strip of gauze saturated

with collodion ; sterilized silver-foil may be used instead of the ordinary protective. Aseptic or antiseptic gauze, in amount proportioned to the expected oozing, must next be laid in place. When germicides are employed, some form of protective should cover the wound, as silver-foil, thin rubber tissue or oil-silk protective. Outside of all, abundance of sterilized absorbent cotton or oakum should be used, being secured in place by a firmly applied bandage, carefully avoiding all constriction. If a limb is involved, proper splints should secure quiet ; if the thorax be concerned, the arm of the corresponding side should be secured ; if the neck, a stiffened collar or dressing is advisable.

Indications for change of dressings. Dressings should never be changed except for good cause. If penetrated by discharge at some spot or spots, and prompt drying at the margins of the stained area tends to occur, a pad of antiseptic gauze had better be secured over the stained spot rather than undress the wound ; but if the dressings are thoroughly soaked, the superficial portions must be changed, leaving those immediately related to the wound unchanged, if possible. When drainage-tubes require removal, dressings must be changed, usually about the fourth day. If the tube is filled with firm clot, the wound is aseptic and the tube should not be replaced ; when doubt exists as to the asepticity, drainage had better be continued until this question is settled. A sustained temperature, unexplained by complications external to the wound, demands inspection, since drainage may be defective or infection have occurred.

Constitutional effects of wounds. It is a common mistake to expect no constitutional symptoms after wounds or operations, and when they arise to ascribe them invariably to infection. Aseptic fever, its causes and phenomena have been fully treated of in Lecture VIII., and should be remembered in this connection. True wound-fever from infection commences later than aseptic fever, on the second or third day, is often preceded by a chill or

rigor, and the local pain increases, and the pulse and nervous symptoms clearly indicate a systemic intoxication.

Lacerated and Contused Wounds.—*Symptoms.*—Some contusion being usually combined with laceration, these two classes of wounds will be considered together. Pain is greater than in incised wounds, but hemorrhage is not so marked. Where contusion preponderates, much blood is extravasated in the tissues, interfering with their blood-supply ; hence, sloughing is usually proportionate to the contusion, as is also the risk of secondary hemorrhage when the dead parts separate. Despite all efforts at antisepsis, sloughing and free suppuration will occur in certain cases. Septic cellulitis and gangrene may occur, the former often resulting in extensive sloughing and serious scarring ; any form of infection or sepsis may occur.

Treatment of Lacerated and Contused Wounds.— Although it is difficult to render these wounds thoroughly aseptic, no reasonable effort should be spared. Temporary sterilized or antiseptic dressings must be used until efficient disinfection can be done. The surrounding parts should be thoroughly scrubbed with a nail-brush and soap and water, which measures should even be extended to the wound itself, if this be fouled with machine grease, street-dirt, etc.

Free irrigation with a germicidal solution should be employed, under anesthesia if necessary, exposing and disinfecting every recess. Light iodoform-gauze packing will prove serviceable in many such cases. When seen after infection is well advanced, incisions to liberate pus, sloughs, and the contained poisons, and to relieve tension, free irrigation and drainage by tube or packing, or by both, become requisite. If the form and location of the wound insures free escape of wound-fluids, no drainage is requisite, but if drainage is needed, tubes must be employed until the discharge becomes aseptic and small in amount. Although slight trimming

of the margins of a face-wound is permissible in exceptional cases to secure primary union and a smaller scar, unaided nature is often equal to the task, any serious deformity being better remediable later by operation. The same advice applies to hopelessly damaged structures in slightly vascular parts, but for the scalp, oral cavity, or face, the surgeon should usually rely solely on antisepsis. Sutures are only applicable to the face, where good results often follow their use. Rest, secured by voluminous aseptic dressings, splints, and position, with possibly the external application of dry cold, is often useful. If serious contusion exists, cold should be employed tentatively, watching lest the sloughing be increased. The continuous warm bath is usually better than a closed dressing when spreading cellulitis and free suppuration occur, proper incisions having first been made.

Punctured Wounds.—In these the depth much exceeds the width. They are caused by pointed objects, as knives, swords, nails, stakes, etc. Dangerous hemorrhage, primary or secondary, is not uncommon from injury of the deep vessels. Important nerves are often damaged, cavities are penetrated, and if infection occurs, deep, widespread inflammation results. If the wound be inflicted by a smooth, uninfected instrument, as a trocar, no trouble will result, but when a rough object, as a board-nail or stake, is the vulnerating object, both almost necessarily harboring germs, the results differ, because fragments of tissue, pieces of clothing, the softened inner sole of a shoe, may be, and they often are, torn off and deposited in the depths of the tissues, the tissue-fragments usually being infected, as the fragments of the foreign bodies always are ; while the change of relation of the wounds in the various planes of tissues interferes with escape of exudates, and this favors spread of an infective inflammation, of itself it cannot initiate such a process. This mechanical fact alone does not explain the gravity of punctured wounds, but its combination with infection does.

Symptoms.—These must vary so with the tissues and parts involved—nerves, vessels, or cavities—that no general description can be given.

Treatment.—Hemorrhage must be arrested, if serious, after enlargement of the wound. Any divided nerve must be sutured. If infection has occurred, thorough disinfection, including removal of any foreign body, must be effected by incisions, irrigations, etc., employing anesthesia if requisite. Drainage-tubes reaching to the bottom of the wound, possibly a counter-opening, and absolute rest by splint and position, are demanded.[1] When septic inflammation follows, proper incisions, counter-openings, and the treatment suggested for infected, contused wounds must be employed. If the brain-case, spinal column, thorax, or abdomen be penetrated, effective disinfection and drainage are indicated, demanding an exploratory operation in most cases, certainly if infection is known or strongly suspected to have occurred.

Constitutional Treatment of Wounds.—This is seldom of importance, certainly immaterial if infection does not occur. Simple, easily digested liquid or soft food is advisable for the first two or three days, after which, if the bowels act naturally and fever is absent, ordinary full diet may be given when the patient desires it. Forced alimentation and rectal feeding may be requisite to relieve the effects of severe hemorrhage or the drain of pus. Alcohol, ammonia, strychnin, digitalis, quinin, etc., will often prove useful. Proper emptying of the bladder must be insured. The renal and alvine secretions must be normally maintained or even stimulated, especially where sepsis is present, to eliminate toxic substances and germs. Sleep in proper amount must be secured, especially for the young and the old, even by drugs when necessary.

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[1] To provide for possible infection.

LECTURE XXXII.

SHOCK; COLLAPSE; TREATMENT OF SHOCK AND COL-LAPSE; SALINE TRANSFUSION.

Shock.—Although the risks of sepsis no longer confront the patient as in the past, shock still claims too many victims.

Pathology.—The dangers of shock can only be made less formidable by a correct appreciation of its pathology. The following statements describe the essential pathology of shock as correctly as is at present possible. Shock is a sudden check or modification of the circulation, effected through the medium of the nervous system, which may be productive of almost immediate death or a prolonged depression of the vital powers, succeeded either by reaction or by a deepening of the prostration, terminating in death. The changes in the circulation which produce this generalized depression of all the vital powers may be the resultant of a powerful impression upon the nerve-centers or upon the peripheral nerves, or upon both; this impression is strongly reinforced by loss of blood. Post mortem, nothing is discoverable beyond great fulness of the intra-abdominal veins and possibly the whole venous system, including the right heart, with cerebral anemia: but when shock occurs in a patient who has lost much blood, the heart-cavities and the arteries are empty, while the tissues are blanched, contrasting with the often intensely engorged abdominal veins. The sudden deaths from uncomplicated shock result from excitation of the inhibitory action of the pneumogastrics on the heart, bringing it to a stand-still; the slower deaths are caused by a prolonged diver-

sion of such a bulk of the blood from the general circulation into the abdominal vessels, (resulting from paralysis of the abdominal sympathetic), and also into the systemic veins (from vasomotor paralysis), that the nutrition of centers and tissues essential to life becomes impossible. A rapid, actual diminution in the amount of the circulating fluid should materially aid the lethal powers of either of the factors of shock just mentioned, and, accordingly, it is well known clinically how hemorrhage determines or intensifies shock.

Symptoms.—Careful scrutiny of cases of shock show that there are apparently two classes : first, those where the general circulatory changes do not materially affect the blood-supply of the cerebrum, or at least that of its cortex. The dilatation of the peripheral vessels, especially those of the abdomen from vasomotor paralysis, even without any direct depression of the cardiac powers, renders the heart's action weak and frequent ; and the consequent feebleness of propulsion of the blood tends to still further favor the venous engorgement, which again in turn renders it increasingly difficult for the heart to overcome the peripheral resistance and maintain the tissues' nutrition. It hardly needs demonstration that this condition is identical with the results caused by severe loss of blood, for it matters not whether the blood be lost externally or be merely withdrawn from the circulation by accumulating in the abdominal veins, capable of containing, as Goltz has shown, such a large proportion of the total bulk of the blood. The patient is usually conscious until just before death, possibly toward the end becoming slightly delirious or eclamptic. He is restless, tosses about ; the respiration is sighing and irregular ; he gasps for air ; the pupils are apt to be dilated ; he complains of thirst, yet is nauseated ; the skin is pale, cool and covered with a clammy sweat ; the temperature is subnormal ; the features are shrunken ; the pulse is feeble and frequent. If recovery ensues, the pulse gains in force and lessens in frequency ;

the respiration improves; the skin dries and becomes warm; the jactitation ceases; the temperature becomes normal, and full recovery follows: this is all a matter of a few hours at most. This has been sometimes called the "erethistic type" of shock, and is difficult to distinguish from the effects of hemorrhage;[1] indeed, as has been shown, it is due to a similar cause—*i. e.*, a withdrawal of a large amount of blood from the circulation. Far more commonly, shock is due to a partial overwhelming of the cerebrospinal as well as the sympathetic system, or, more accurately stated, the nutrition of the cerebrospinal axis suffers from a disturbance of its circulation, resulting from paralysis of its own vasomotor system. The patient feels no pain even when a mangled limb is roughly handled; the pupils are dilated and sluggish to light; dyspnea is not complained of, because the higher centers no longer realize afferent impressions; the patient lies quiet; marked loss of power of the voluntary muscles exists; nausea is absent, but the shallow, and occasionally sighing respiration, the rapid, feeble pulse, the pale, cold, sweating skin and shrunken features are all present, with relaxation of the sphincters. The patient may be slightly delirious, still more rarely is convulsed, but usually is dull, apathetic, and hard to arouse. Ill-advised efforts in the last direction may, however, render the patient actively delirious, the "delirium of shock" being then said to be present. Shock can only be confounded with concealed hemorrhage and fat-embolism, the characteristic points of difference from the latter condition having been fully described in Lecture XII., page 145.

Predisposing Causes.—Certain precedent conditions render shock more probable or more pronounced. Great fear, or a firm conviction as to the fatal outcome of an operation is greatly to be dreaded. To incur, in such cases, the mental shock of a major operation by employ-

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[1] It has been suggested that an estimate of the hemoglobin would settle the question between hemorrhage and shock, by its diminution in the former.

ing local instead of general anesthesia is folly. The
poor nutrition or actual poisoning of the tissue-cells by
the constant presence of excreta in the blood supplied
them, as in chronic renal affections, lithemia, diabetes,
the opium habit, mental or physical strain such as is in-
duced by business worry or the hardships of a campaign,
as well as extreme youth or old age, render them (the
cells) unable to sustain the further deprivation of pab-
ulum induced by the circulatory changes present in
shock. Injury to organs exceptionally well supplied
with nerves, as the testicle ; to many peripheral nerves,
as those of large surfaces of the skin in burns, etc. ;
or to the organs within the chest or abdomen upon
whose perfect functioning the maintenance of respira-
tion, circulation, and the tone of the abdominal ves-
sels—preventing their extreme dilatation—depends, will,
in the absence of all hemorrhage of any moment, pro-
duce profound shock. Prolonged general exposure to
cold after an accident intensifies or determines shock ;
or loss of bodily heat from exposure or wetting during a
prolonged operation, especially where large areas of skin
are reflected—for instance, from the chest—or the abdom-
inal viscera are exposed, are unquestioned causes of
shock. Certain cases of syncope and shock have been
ascribed in the past to the sudden removal of supporting
pressure to the intra-abdominal vessels by rapidly evac-
uating large quantities of ascitic fluid in the upright
posture ; and this is what might be expected theoretic-
ally to happen, owing to the long absence of any extra-
ordinary call upon the vasomotor regulating apparatus
of the abdominal veins, which have been subjected to
constant extravascular support : some of the shock
often seen after the removal of large abdominal tumors
is doubtless in part due to the same cause.

Exciting Causes.—These are practically reducible to
three : loss of blood ; loss of heat, produced by prolong-
ation of the operation and the greater amount of the
anesthetic required, this being largely gotten rid of by

vaporization—*i. e.*, by consumption of vital heat ; and unnecessary depth of the anesthesia, which in addition to the increased loss of heat unquestionably adds to the severity of the shock.

Treatment.— Prevention is peculiarly important, because when once shock becomes pronounced, we are too often unable successfully to cope with a condition which might readily have been *prevented*. Having now ascertained what favors shock, means calculated to improve the tone of the peripheral vasomotor system and the force and endurance of the heart should be adopted, if time suffices before operation. Strychnin in fair doses should be exhibited for some days before, or given hypodermatically just before operation in an emergency. Do not empty the vascular system or the tissues of fluids shortly before operation by free purgation, or if this be imperative, see that the bulk of the circulating fluid is restored by large rectal injections of normal salt-solution. Give as large hot, normal salt-solution rectal enemata as the bowel will contain just before, and during operation, if shock begins to manifest itself. While it is proper to have the stomach empty, do not deprive the heart of its most powerful and lasting stimulant, nitrogenous food, but give this in the form of clear, strong soup some hours before operating. In special cases, calm fear and excitement by a moderate dose of morphia and atropia administered hypodermatically a half hour before operation ; this will also serve the additional purpose of lessening the amount of anesthetic requisite. It need hardly be said that attention should be paid to the condition of the bowels, intestines, and liver, in order to secure proper elimination of effete materials, thus leaving these organs in the best possible condition to excrete the excessive amounts of tissue-waste resulting from operation or injury. Especial attention should be paid to the possibility of the supervention of intestinal sepsis, intestinal antiseptics (as calomel) being administered,

and laxatives, with careful regulation of the diet, when time permits before operation, or as soon thereafter as the conditions allow. Operate as rapidly as circumstances will permit; avoid exposure of tissues by covering them with warm sterilized towels; envelop the limbs in cotton held in position by bandages, or use flannel leggings; apply hot bottles or water-bags, or employ an artificially warmed operating-table; get rid of all wet towels as soon as possible; avoid all draughts; employ as little of the anesthetic as possible; place the patient in a warmed bed, maintaining the head low—possibly elevating the foot of the bed; apply external heat, carefully guarding against burning, by never allowing the heated vessel to be in direct contact with the skin; and warm the atmosphere of the room in which the patient lies, in order to conserve the body-heat by supplying external warmth to vaporize the anesthetic and thereby lessen the amount of this which must be excreted by the kidneys. All these measures are intended as *preventives* of shock; but can nothing be done when this condition has developed? If the operation be not completed, hasten or suspend all further intervention if this be possible. Apply external heat, and place a sinapism over the heart. At once adopt autotransfusion by lowering the head of the table, elevating the limbs to a right angle with the body, and then bandaging them so as to expel all blood with elastic or muslin bandages; employ saline transfusion; add coffee or oil of turpentine to the rectal enemata of hot water; push the strychnin hypodermatically; later, give nourishment by the rectum, and introduce fluid by the same route to assuage thirst, lest nausea be produced when water, etc., is introduced into the stomach, which would add to the shock; wash out a stomach containing fecal matter after intestinal obstruction; finally, be careful not to overdo your therapeutics, exhausting a weak heart by over-stimulation. Measures to combat shock will not be needed beyond a few hours, and when the pulse has dropped below 120,

the more active treatment should be gradually, but quickly intermitted. A word of caution is requisite as to stimulants by the mouth or rectum. During profound shock absorption is slowly effected, hence giving alcohol repeatedly at short intervals may lead to intoxication, when, shock being passed, the whole amount is suddenly absorbed ; moreover, alcohol—indeed, anything by the mouth—is apt to induce vomiting. Hot black coffee is better than alcohol, when procurable, the heat stimulating the heart and abdominal sympathetic. Atropia is sometimes indicated when respiratory failure seems imminent, while digitalis hypodermatically is often employed, but probably is seldom beneficial until the shock has commenced to diminish, when it may be of service. In the erethistic variety opium is decidedly indicated.

A few words as to *saline transfusion.* First, the operation must be done strictly aseptically ; next, saline, not *plain*, water must be used, because plain water promptly kills by destroying the red cells. A heaping teaspoonful of sodium chlorid to the quart (liter) of water is sufficiently accurate for emergency-use : this must be thoroughly boiled for some minutes. The temperature of the solution must never be lower than 100° F. *when it emerges from the needle or cannula*, and had better be higher, say 105° F., although Dawbarn advocates 120° F., or as hot as can be borne by the back of the hand. The heat will stimulate both the heart-muscle and the muscular tissue of the coats of the dilated veins. To maintain the temperature, the reservoir must be wrapped in hot towels or a heated blanket (both sterilized). Gravity is the best method of introducing the fluid. The amount requisite for an adult varies from one to three quarts (liters). Never let it flow in faster than ten minutes to the quart (liter), suspending the injection instantly if embarrassment of the heart results from over-stimulation. The repetition of the dose will often be needed, but reopening of the vein may often be

avoided by employing the rectal route or hypodermat-oclysis : in extreme cases, however, venous transfusion must be repeated again and again. Rectal enemata can be administered by elevating the foot of the bed and using a funnel attached to a piece of tubing or a catheter. When venous transfusion is employed the median basilic vein is usually the one employed, although a vein in the wound can sometimes be utilized. The needle, when hypodermatoclysis is done, should be introduced into the loose cellular tissue of the infraclavicular regions, or into that of the lateral abdominal walls. A resumption of even a level position when the head of the bed has been lowered must be cautiously made, lest syncope result or the heart's action become enfeebled by lack of fluid to work on. Similarly, removal of bandages from the limbs, employed for autotransfusion, and lowering of the limbs if they have been raised for the same purpose, must be gradually effected.

When shock is the result of a crushed limb, it often becomes a nice point to decide whether the mangled part is not maintaining the shock. If the patient's circulation and respiration improve under the use of ether as an anesthetic, a rapid amputation done under primary ether anesthesia will usually be borne : if res-piration and pulse rapidly increase, stop all further interference at once, or death will follow under the knife or shortly after the completion of the operation, because the impression of the ether upon the peripheral branches of the pneumogastrics in the lungs seems to overwhelm these nerves, resulting in a sudden increase in the rate of the pulse and respiration, soon followed by failure of both, and death.

LECTURE XXXIII.

TRAUMATIC DELIRIUM; DELIRIUM TREMENS; TRAU-
MATIC HYSTERIA; TRAUMATIC INSANITY.

Traumatic Delirium.—This, occasionally following accidents and operations, results from more than one cause. Thus it is oftenest a symptom of *septic traumatic fever*, when it usually becomes pronounced during the height of this—viz., from the second to the fifth day—subsiding with the fever producing it. It is most marked at night, and is usually in proportion to the pulse and temperature.

Prognosis.—This is favorable if the cause can be removed, as is usually the case.

Treatment.—Disinfection and drainage of the wound, with cold to the head, laxatives, and possibly diaphoretics or diuretics to aid in the excretion of ptomains, toxins, and germs, are all that is requisite.

Accidental, or operative traumatisms inflicted upon those previously subjected to prolonged brain-strain, especially if of a susceptible nervous temperament, occasionally give rise to a delirium sometimes called **nervous traumatic delirium,** probably an obscure auto-intoxication from accumulation of the products of excessive metabolism. This occurs without fever and in those not addicted to the use of alcohol. While usually low and muttering, the delirium may be violent, even maniacal.

Prognosis of Traumatic Nervous Delirium.—This is usually favorable, especially if sufficient sleep can be secured.

Treatment.—Carefully selected, nourishing food, with

stimulants if requisite, will improve the nutrition of the nervous system, which has suffered from the previous overstrain, provided sufficient elimination of retained excreta can be secured to permit the nerve-cells to profit by the pabulum. While this is being effected, the cerebral centers must be kept at rest to prevent exhaustion, by the judicious use of opium, bromids, chloral, or a combination of the last two. Quiet of mind and body must be maintained.

Delirium tremens, or the delirium occurring in alcoholics, is characterized in its uncomplicated form by the absence of fever, but it must not be forgotten that it may occur after a serious injury, which in itself is a cause of fever. The delirium is usually of the low muttering, busy, vigilant form, with delusions, the patient seeing snakes, insects of all kinds, animals, or devils, constantly conversing with himself or imaginary persons, and, while capable of answering rationally on other subjects than his delusions, when left to himself promptly becoming again incoherent. At times maniacal outbreaks occur, with suicidal and murderous impulses, this violent form usually resulting from injury received during alcoholic excesses : this is true *mania a potu.* Apparently suicidal acts, such as jumping from windows, are often only efforts to escape from imaginary pursuing demons or persons threatening bodily harm ; indeed, genuine attempts at suicide are dictated by the desire to escape from imaginary evils worse than death. Apparently insensible to pain the patient will attempt to walk with a broken limb, unless physically restrained in his efforts to escape from his imaginary foes. The skin is moist ; the hands shaking ; the muscular weakness pronounced ; the tongue when protruded is tremulous, coated with creamy fur at first, later, in bad cases, dry, brown, and covered with sordes ; the pulse is full, soft, and quick ; and the temperature—in the absence of complications—is about normal. The facies and whole manner indicate suspicion and fear. Constipation

is the rule ; the urine is scanty or, possibly, in the worst cases, suppressed ; complete anorexia is present, although fluids may be greedily swallowed, and sleep is impossible.

Prognosis.—With a young patient who has sound kidneys the prognosis is good, but when the reverse obtains death by exhaustion is not uncommon.

Treatment.—Many cases of hard drinkers can be safely tided over a threatened attack by attention to the emunctories (bowels, kidneys, etc.), anodynes to produce sleep, proper food, and lessening (but not stopping) stimulants. The experienced surgeon always expects and guards against an outbreak of delirium tremens in those tremulous, sleepless individuals so often brought in injured into the large city hospitals. Injuries to or operations upon those who are "moderate drinkers" often suffice to precipitate an attack of delirium tremens which otherwise would never have occurred, hence the importance of a correct history as to the patient's previous habits. Sudden withdrawal of the accustomed stimulant not uncommonly precipitates an attack. During an attack three objects must be kept steadily in view—viz., to induce sleep, thus preventing fatal exhaustion of the nerve-centers, to secure elimination to enable normal cell-nutrition to go on, and to supply the nerve-cells with proper pabulum. Calomel followed by a laxative or enema should be given, and, as liquids are usually acceptable, the patient can be induced to take beef-tea or meat-broths, which, in the case especially of old drunkards, will be rendered more palatable by the free addition of cayenne pepper, this condiment acting as a stimulant both of digestion and of the circulation. Digitalis is useful both to sustain the heart's action and, more important still, to excite free diuresis, thus aiding in the elimination of the injurious metabolic products productive of much of the trouble. Occasionally forced feeding with the stomach-tube may become requisite. In young, sound patients, the hypodermatic use of

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morphin may be tried, but in older individuals whose kidneys are probably unsound opium is not advisable. Strychnin in doses large enough to sustain the circulation must be exhibited ; indeed, some think that it exerts other beneficial effects beyond its stimulant one upon the heart. Chloral, in from 15 to 20 grain doses, combined with potassium bromid, every two hours until sleep is induced, and carefully watched, is usually safer and more prompt in action than opium. Stimulants can rarely with safety be entirely withheld, and then only from the young and robust ; the old and feeble should never be subjected to this experiment, alcohol being gradually reduced, not suddenly withheld. That stimulant to which the patient is accustomed should be given, but in smaller quantities than usual, and it should be gradually withdrawn. After securing prolonged sleep, strychnin, tonics, good food, and the gradual stoppage of stimulants will usually effect a cure. Although it may be possible to secure to the bed a patient with delirium tremens who has a broken limb, it is exceedingly difficult to avoid further injury to this, a closed fracture being often converted into an open one. Plaster of Paris would seem ideal were it not that a patient will break this up by pounding the limb about. When well-padded splints will not suffice, owing to the violence of the patient, the parts should be sterilized, enveloped in a voluminous antiseptic dressing, and then firmly bandaged in a large feather pillow. If now the injury be rendered an open one, no special harm will result, and this form of dressing will not be required for more than forty-eight to seventy-two hours—probably for a much shorter time. As many patients will get out of bed despite broken limbs and other serious injuries, some means of restraint will often be requisite whereby the individual shall be secured to the bed.

Traumatic Hysteria; Traumatic Insanity.—Severe physical shock, especially when combined with a profound mental impression, often gives rise to a set of

nervous symptoms termed "traumatic hysteria." Such combinations being more frequent in railway accidents, certain more pronounced sets of symptoms have been specially studied under the head of "railway spine" and "concussion of the spine," but similar conditions result from other forms of accident. Traumatic hysteria must not be confounded with actual physical injuries to the spine or brain, or the resulting inflammatory or degenerative changes following such accidents. Traumatisms also arouse dormant pathological conditions, and injuries of peripheral nerves give rise to reflex effects, which, with ascending neuritis originating central trouble, may all simulate traumatic hysteria, and must carefully be distinguished from it. The following symptoms of exhaustion of the nervous system, which often follow railway accidents and not uncommonly ordinary shock from other forms of accident, must be differentiated from "traumatic hysteria:" they might be appropriately called "traumatic neurasthenia."

Symptoms of Traumatic Neurasthenia.—According to Thorburn, these are "general debility, confusion of thought, loss of memory, mental irritability, disturbed sleep, dreaming, headache (usually posterior), interference with visual accommodation, photophobia, palpitation, frequency of pulse, dyspeptic troubles (furred tongue, foul breath, constipation, and nausea, or epigastric pain), sweating, a concentrated condition of the urine, etc." While neurasthenia is often associated with hysteria, and is much more frequent than the latter, it is probably a distinct condition, hysteria being due to "suggestion" or "autosuggestion" and neurasthenia to mere exhaustion. Age predisposes to traumatic hysteria and neurasthenia, these affections belonging chiefly to middle life. Hysteria is more likely to occur in females if injured, but as more males are exposed to the causes of traumatic hysteria, more men actually suffer from it. A neurotic temperament and chronic alcoholism both favor the development

of traumatic hysteria, as they do the ordinary form, but do not have so much influence as in the latter variety, especially the neurotic temperament. The exciting cause is some accident, but mental impressions have a large share in the causation of traumatic hysteria ; thus Thorburn insists that a previous period of terror, horrible surroundings, and the instantaneous occurrence of the accident have more effect than the physiological injury. Direct injury to a nerve-trunk is a "potent cause" of hysteria. No special determining influence can be ascribed to the severity of the injury, but the locality has, "complete hemianesthesia or double monoplegia on the same side as that of the lesion often occurring after head injuries." The traumatism of surgical operations often gives rise to various functional neuroses.

Symptoms of Traumatic Hysteria.—The ordinary screaming, laughter, crying, or possibly, convulsions so often observed after accidents, are evanescent and of no moment, producing at most temporary nervous exhaustion; but there are certain peculiarites at times observable, especially after railroad accidents. Thus patients will maintain that they have been unconscious for a longer or shorter period, yet investigation shows by the absence of vomiting, relaxation of the sphincters, etc., that this condition could not have been due to cerebral concussion. Such patients walk, travel long distances, do many voluntary acts almost automatically, sometimes regaining their consciousness, as they describe it, after a considerable interval of time and distance of space. They present a peculiar, dazed appearance—in fact, resemble in many ways those in the somnambulistic state. Exaggerated or purely imaginary statements are made pertaining to the accident. These illusions are probably the results of "autosuggestion," during a state closely approximating that of the "minor degrees of hypnotism." These preceding conditions have been called "acute hysteria," while those following belong to

"chronic hysteria." The chief manifestations of chronic hysteria may be "(1) psychical, including epileptiform attacks and hysterical insanity ; (2) motor, including paralysis and contractures of the limbs, and special effects upon the larynx and the bladder ; (3) sensory symptoms—anesthesia, hyperesthesia, and paresthesia of the general or sensory nerves ; and (4) vasomotor, secretory, and trophic troubles." In most cases, symptoms suggestive of combined neurasthenia are present.

Prognosis.—When pecuniary compensation is not sought or is promptly granted, with favorable surroundings, proper treatment usually leads to a cure. Marked changes in the severity of the symptoms, transference from one side to the other, and temporary disappearances afford grounds for a favorable prognosis.

Treatment.—Isolate from friends and relatives ; encourage the use of any paretic part ; discountenance any idea that true paralysis exists ; employ faradism and massage ; give tonics ; possibly try overfeeding, avoid the bromids, employing the ammonium salt if demanded by marked cerebral excitement ; and secure a prompt settlement of any legal questions.

Traumatic Mania.—This is a rare sequel of traumatisms, usually seen in females after operations on the breast or genital organs. Although sometimes assuming the violent type, it more commonly is melancholic. Viewing it as a form of auto-intoxication, as some do, purgatives, the diaphoresis induced by steam baths, and a systematic attempt to insure increased elimination, will often result in recovery. If maniacal and liable to become exhausted by loss of sleep, chloral, opium, etc., must unhesitatingly be resorted to as a temporary life-saving expedient : the possible relation of drug-poisoning to post-operative delirium and mania must not be forgotten, and Lecture XVI. should be consulted upon this point.

LECTURE XXXIV.

ANESTHESIA AND ANESTHETICS; CAUSES OF DEATH;
PREPARATION OF PATIENT; ACCIDENTS DURING
ANESTHESIA; LATE ACCIDENTS.

Anesthesia and Anesthetics.—The most important
point to emphasize at the outset and never to lose sight
of is, that general anesthesia should never be employed
unless really needed. Any agent which brings a patient
into a condition where the depressing effects on the
higher nerve-centers may at any moment extend to and
overwhelm the vital centers in the medulla oblongata,
should not be recklessly employed. Discrimination must
be exercised in the choice of the anesthetic, chloroform
being better for one class of patients, ether for a second,
while, in a third, local anesthesia not only will suffice
but is really preferable to general anesthesia, as in many
eye operations. When inducing general anesthesia, the
rapidity with which the patient is brought under the
influence of the agent is of minor importance as com-
pared with the absence of mental shock, physical
exhaustion, and the partial asphyxia so often resulting
from the fright and violent struggles induced by a too
rapid, rough method of overcrowding the inhalation. In
a course of lectures such as these have to be, anything
like a history of anesthesia and anything beyond that
which is strictly essential for a safe use of this class of
drugs is necessarily precluded. An important point too
often ignored is, that while the patient may recover from
the anesthetic—indeed, may never have been in any
danger of life *during the administration*—the result of
an operation may be jeopardized, convalescence be

delayed, or even a lethal result be brought about by a too liberal use of the drugs, producing severe vomiting, pulmonary edema, bronchitis, or anuria. The fact that individuals with unsound hearts, lungs, and kidneys survive prolonged anesthesia induced by ether, chloroform, etc., and that the administration of one of these is often an imperative necessity in many cases where disease is known to exist, does not relieve the anesthetizer from the responsibility of ascertaining the condition of these organs, because the knowledge thus gained may enable him to avoid an otherwise certain disaster by sparing a fibroid heart, the already congested lungs, or kidneys only barely equal to elimination of the minimum of excreta.

It might seem superfluous to warn students that before inducing general anesthesia all artificial teeth, a quid of tobacco, or a mass of chewing gum should be removed from the mouth, were it not that such omissions are sometimes observed in our own clinic. Still further, teeth so loose as to be likely to be dislodged by the mouth-gag, tongue-forceps, or throat-mop, must be remembered and watched, lest a sudden death result from their entrance into the larynx. It is also well, and my invariable rule, to ascertain whether the patient has ever been anesthetized before, and whether any idiosyncrasy exists. Some patients cannot take ether who act well under chloroform, while in others the reverse is true ; indeed, I have seen one patient nearly die twice from ether, take chloroform twice with no difficulty, yet this man, upon a third occasion gave rise to the liveliest apprehension when inhaling chloroform. Never anesthetize a patient without his or her free consent, or that of a friend, unless it be a matter of life and death, and never anesthetize a *female*, unless a third person be present, preferably also a female, because you lay yourselves open to a charge of rape. Either because of the fear that such a thing should be attempted, or because the sexual feelings are excited under anesthesia, women often

exhibit evidences of a venereal orgasm when coming from under the influence of the drug, indeed openly charge the physician with rape in the presence of their husbands or friends. This has occurred so often in my own experience, and that of others, that the imperative necessity of the presence of a reliable witness has been impressed upon me. There is no time to discuss the grounds for the belief that ether is unquestionably safer so far as danger while under its influence is concerned. Although chloroform, if carefully administered, is a relatively safe agent and often far more convenient than ether, still, as the latter can be given with practical safety by an inexpert layman, while chloroform cannot, it is important to recognize the relatively greater safety of one over the other. Although only two general anesthetics have thus far been mentioned, others will be spoken of later, yet but briefly, because the advantages and accessibility of ether and chloroform render them practically the only ones employed, except by a few isolated individuals. Statistics are proverbially unreliable, yet the immense preponderance of fatalities of chloroform over ether in all published records show that whatever may be the exact risk for each, the relatively more dangerous one is chloroform.

As you will find elsewhere better accounts of the methods of preparation of ether, chloroform, etc., and tests for their purity than space will permit here, all such information will be omitted.

Preparation of the Patient.—In addition to the use of strychnin for a few days previous to operation to improve the condition of the heart, and, possibly, the administration of a salt-solution enema per rectum just before operation (mentioned when speaking of shock), in order to render both cardiac and respiratory failure less likely, an empty stomach must be secured by the absolute interdiction of all solid food, or indeed of any food not capable of prompt removal by absorption with little previous digestion, for a number of hours previous

to the administration of the anesthetic. This precaution secures against the interruption of the anesthesia by vomiting, and, more important still, inspiration of the stomach-contents into the lungs during emesis under anesthesia is prevented, an unsuspected but frequent cause of the so-called "pneumonias" following general anesthesia. The heart, however, must not be deprived for too long a period of its most powerful and permanent stimulant—*i. e.*, food, so that a half pint of strong *clear* soup should be administered about three hours before any important operation. Should a patient have eaten a large meal of solid food or have partaken freely of alcohol shortly before anesthesia is demanded for an emergency operation, as a crushed limb, a strangulated hernia, etc., it is better to empty the stomach by inducing emesis with mustard, or even the hypodermatic use of apomorphia, unless the patient's circulation forbids the employment of the latter agent. When it is desirable to calm a nervous patient's abnormal dread of an operation and also to save strain upon damaged kidneys, in an adult the administration of $\frac{1}{6}$ of a grain of morphia hypodermatically, twenty minutes before the anesthetic is to be given, will often render it easy to induce general anesthesia with a surprisingly small amount of ether. If previous experience with the patient has shown that ether excites the secretion of a large amount of mucus productive of embarrassed respiration and cyanosis, the addition of a proper amount of atropia to the morphia will often obviate this.

Effects of the Administration of Ether.—Owing to its irritant properties even when much diluted with air, when first inhaled, ether often excites a sense of oppression, "suffocation" the patient terms it, which by inducing shallow respiration retards the induction of anesthesia. This sensation soon passes off, if the concentration of the vapor is gradually increased, or if the patient can be persuaded to breathe a few times deeply ; sometimes the removal of the inhaler for an instant will

enable the patient to take a deep inhalation, which, if the cone be promptly reapplied, will fill the lungs with so much of the anesthetic that the sensibility of the respiratory mucous membrane will be numbed. A little coaxing to breathe more deeply, with an occasional removal of the cone will soon enable the patient to inspire deeply. With many forms of inhalers the concentration of the vapor can be so controlled and so gradually increased that neither coughing nor struggling will be caused. Enough air must always be admitted to prevent cyanosis, and this expedient is a potent means of preventing coughing from undue irritation of the bronchial mucous membrane. It must be remembered that not saturation of the air in the lungs, but the entrance of enough ether into the circulating blood to produce and maintain the phenomena of anesthesia is required. Cyanosis should not be permitted to occur at any stage of anesthesia. Only upon rare occasions and in a perfectly healthy individual, when it is necessary to secure the effects of the so-called '' primary ether-anesthesia,'' is it proper to saturate the ether cone at the outset and to forcibly hold this over the mouth and nostrils in order to insure the entrance of saturated ether-vapor into the lungs and prompt overwhelming of the general sensibility. With a weak heart or atheromatous vessels, much more with both conditions present, this is a hazardous experiment, because the fright, struggling, and partial asphyxia are apt to produce dangerous intravascular tension ; in any event, a resort to this method of inducing anesthesia is rarely necessary. The early arrest of respiration results from irritation of the trifacial filaments in the nose, etc., producing by reflex action a spasm of the glottis. Irritation of the pulmonary branches of the vagus seems also to partially account for the temporary arrest or shallowness of respiration, and the inhibitory action upon the heart is diminished, thus accounting in part for the accelerated movements of this organ. At intervals during the earlier stages of

ether-anesthesia, a few inspirations are taken, followed by fixation of the thorax for a few seconds, then one or more deep inspirations succeed, especially if the thorax be compressed, when rapid full inspirations either initiate partial anesthesia, lasting for about a minute, or the stage of excitement commences, the patient shouting, screaming, swearing, fighting, etc. This stage, lasting usually only for a few minutes, is succeeded by gradual relaxation, slower and deeper respiration, and unconsciousness—*i. e.*, anesthesia is present, even the cornea being insensitive to touch. Both respiration and heart action are more frequent than normal, although the latter may become slower and stronger when the patient has been much frightened, been in much pain, or has not thoroughly recovered from the shock of any injury before the administration of the anesthetic. The skin is dry and warm and usually flushed, especially that of the face and neck. If the anesthesia be pushed beyond this point, from paralysis of the soft palate the respirations become stertorous, they are slower and more shallow than normal, and the face becomes either pale or cyanotic; this is a symptom of overdosing. Even when not carried so far, or earlier in the case, from the accumulation of mucus in the throat, the breathing may be noisy. If carried to the danger-point, the pale, then deeply cyanotic face ; the cold, clammy skin ; the weak, rapid pulse ; the increasingly shallow, then interrupted, finally arrested, respiration, are all characteristic, the heart continuing to beat feebly for some moments after breathing has ceased. As Hare says, "ether first depresses the perceptive and intellectual cerebral centers, next the sensory side of the spinal cord, next the motor side of the cord, then the sensory and motor portions of the medulla oblongata ; and with this depression death ensues." What advantages and disadvantages inhere to ether as a general anesthetic ? It is safer than chloroform, does not depress the heart, is reasonably rapid in its effects if skilfully administered, and does not require much of the drug to

maintain anesthesia when once this is secured ; but, on the other hand, much more ether is usually required than chloroform to render the patient unconscious; ether is decidedly irritant to the respiratory tract, the consequent accumulation of mucus causing frequent interruptions to the administration of the anesthetic ; the anesthesia is hard to maintain during operations about the face requiring frequent removals of the cone ; and it is more apt to be followed by severe vomiting, late pulmonary edema, and renal accidents than chloroform. Certain accidents, although possible during chloroform anesthesia, are more common when ether is being used. Thus, respiration may be seriously embarrassed by a collection of mucus in the throat ; accumulation of mucus in the bronchial tract often causes a distinct cyanosis which persists until the lungs are cleared of the mucus, the locality of which is often demonstrable by auscultation. Edema of the lungs, productive of a free outpouring of thin mucus, is an accident commoner than is usually believed. During profound anesthesia from any agent, the jaw with the tongue tends to fall back, with the patient in the supine position, when the lingual dorsum forces down the epiglottis like a lid over the glottis, into which air would find it hard to enter, even with the epiglottis out of the way, because the pharynx is blocked by the base of the tongue. All these conditions produce cyanosis or pallor in varying degrees, with weakened pulse. When mucus in the throat produces cyanosis, etc., the bubbling, rattling sounds suggest the cause, and the repeated removal of the mucus with a mop relieves the condition. When resulting from bronchial mucus, clearing of the throat, pulling forward of the tongue, etc., produces no change, while the hand on the thoracic wall often recognizes the location of the obstructing material, vomiting induced by the throat-mop or forcible compression of the thorax, causing the ejection of large amounts of foamy mucus. Removal of the mucus, however effected, is followed by an improvement in the color.

When lung congestion and edema is the cause of the cyanosis, clearing out of the mucus effects but little change or but a temporary one, because the mucus is being continually poured out, and the blood fails to be properly oxygenated. Mere falling back of the tongue and jaw, causing embarrassed or arrested respiration, will be at once relieved by removing this condition by measures presently to be described. Either from abnormal collapsibility of the alæ nasi, or from paralysis of the dilatores narium, in some patients during inspiration the alæ act as valves, being drawn in during every act of respiration, preventing all access of air ; sometimes a deviated septum or hypertrophy of the tissues over the turbinates interferes with the access of air, genuine asphyxia resulting from both conditions, puzzling to the anesthetizer because he cannot recognize any cause. An aural speculum in each nostril, or maintaining the teeth and lips slightly open by means of a gag will relieve this form of cyanosis, which is seen not to be due to the tongue falling back, because its tip rests against the front teeth ; not to the root of the tongue obstructing the pharynx, because elevation of the jaw produces no effect upon the cyanosis ; and not to mucus, because the noise produced by its presence is absent and none can be found with the throat-mop, *but every time the mouth is kept open for any purpose the character of the respiration improves and the cyanosis diminishes.* The special treatment of these causes of cyanosis will be specifically described later, but in passing, emphasis should be laid upon the necessity of employing artificial respiration if, when the cause of the cyanosis is removed the lividity still persists, in order to secure a larger supply of oxygen ready for absorption so soon as the air can gain access to the lung alveoli, *and to empty the lungs of* the ether-saturated vapor. Pallor due to a combination of respiratory and cardiac failure will be indicated by the persistence of a weakening pulse and respiration *after* the removal of all

causes of interference with the access of oxygen to the blood.

The late accidents from ether-anesthesia are bronchitis, pulmonary congestion, and edema, and possibly, catarrhal pneumonia. Renal disturbances are rare in individuals with previously healthy kidneys, although this is possible after surgical anesthesia, but none can doubt that in the presence of actual renal disease serious consequences may follow, ether producing at least a passing congestion, while at the worst complete anuria may result, a condition rarely recovered from. Partial suppression of urine is not uncommon after severe operations followed by marked shock, but the diminution in the secretion of urine cannot then be solely chargeable to the anesthetic, being often largely due to the intense congestion of the kidneys, which become engorged with blood in common with the other abdominal organs : the general loss of vascular tone from vasomotor paralysis in addition so lowers the blood-pressure as to favor anuria. Ether seems to be especially dangerous for diabetics, acetonuria and coma being quite common after its use. The marked loss of body-temperature occurring both during and after operation must be taken account of and guarded against ; this may amount to as much as 4° F., being chiefly caused by the abstraction of heat during the elimination of the drug by vaporization. Vomiting is rarely troublesome unless the anesthetic has been administered when the stomach is full, been unduly pushed, or when the anesthesia has been maintained for a long time : its treatment will be indicated later.

LECTURE XXXV.

ANESTHESIA EFFECTED BY ETHER, CHLOROFORM,
AND NITROUS OXID GAS; TREATMENT OF ACCI-
DENTS DURING ANESTHESIA; TREATMENT OF
SEQUELÆ.

Chloroform.—In all essential respects chloroform
produces its anesthetic effects in the same manner as
ether does. While chloroform-death may result from
respiratory failure, this is exceedingly rare when the
heart is sound. Even with a healthy heart, violent
struggling, fright, and partial asphyxia—*i. e.*, cardiac
engorgement from disordered respiration—will render
death by cardiac failure that to be guarded against.
From its depressant effect upon the vasomotor system
and heart, chloroform unquestionably kills by inducing
cardiac failure if there be disease of this organ, and
often when it is sound, from "idiosyncrasy," as we are
pleased to term it. The risk of sudden death is much
increased if any peripheral reflex is excited by commenc-
ing to operate when anesthesia is not complete—even
an aspirator needle-puncture has sufficed to kill : this
cannot be said of ether-anesthesia. Again, the first few
inhalations of a concentrated chloroform-vapor may
either by reflex excitation of the depresser action of the
pneumogastrics through impressions on the branches of
the fifth nerve in the nose, etc., produce cardiac arrest ;
or, irritation of the pulmonary branches of the pneumo-
gastrics may result in the same way. Finally, chloro-
form may be administered with the happiest effects two
or more times, only to excite the liveliest apprehensions,
or death results when exhibited upon a subsequent occa-

sion ; ether rarely varies in its effects upon the same patient, always giving rise to the same difficulties upon every occasion where it is used. In health, the first few inhalations of chloroform sometimes produce slowing of the pulse and slight rise of the arterial pressure, the latter in part from cerebral excitement possibly, but the slowing of the heart is induced by irritation of the pulmonary branches of the pneumogastrics.

Usually the pulse is from the outset more rapid and less powerful, with decrease of the arterial tension. Partial, transitory arrest of the respiration may occur, but is a markedly less prominent symptom than during ether-anesthesia. Primary dilatation of the pupil is followed by a permanently contracted pupil under full anesthesia, but sudden dilatation *during* anesthesia is a most alarming symptom, indicative of that sudden, general relaxation which means death. Violent struggling, inducing partial asphyxia and overdistention of the heart, is most dangerous when the drug is being freely inhaled, and the chloroform must never be pushed under these circumstances. According to one authority, athletes and drunkards are more apt than other individuals to die suddenly during the struggles of chloroform-anesthesia ; yet a stage of excitement is rare in most persons as compared with that seen during the administration of ether. Post-anesthetic sequelæ are rare after chloroform-anesthesia. Remember that, "quantity for quantity, ether is of course less irritant than chloroform," but as less of the latter is requisite, it (chloroform) is not so objectionable in the presence of renal disease ; but neither should be given unless absolutely necessary, be repeated, or the anesthesia be maintained a moment beyond the absolute requirements of the case in the presence of chronic renal complications, or when diabetes exists. Pulmonary complications rarely occur, but the effects of the depression of temperature must be guarded against as a predisposing cause of lung trouble. Although vomiting is much less frequent, it does occur,

and requires the same management as when the result of ether-anesthesia.

Neither the A.C.E. mixture nor those of Schleich can be recommended for general use, because much more dangerous than ether or chloroform *in the hands of tyros.* *Ethyl bromid* is so evanescent in its effects; requires to be absolutely pure; so readily decomposes when exposed to light and air, when it becomes actually poisonous; sometimes produces such persistent tonic muscular spasms; and is so much more dangerous than ether or chloroform, that it had better not be employed.

Nitrous Oxid Gas.—This is the most rapid and least dangerous of any general anesthetic. It had better not be administered to patients with fatty hearts and atheromatous vessels; yet in the latter condition it has been frequently given with impunity. It is a common statement in the books that anesthesia by nitrous oxid gas cannot be maintained safely for more than about one minute. By the proper admixture of oxygen or even atmospheric air, this is no longer true. The best results can be secured by using oxygen in the proportion of 2 per cent. at the commencement, increasing it up to 10 per cent. if untoward symptoms present themselves as the anesthesia is prolonged. If air be employed instead of oxygen, from 14 to 18 per cent. acts best for men, and 18 to 22 per cent. for women and children, but no one proportion of either oxygen or air will successfully anesthetize every patient. The asphyxial symptoms of nitrous-oxid anesthesia, such as cyanosis, stertorous respiration, and convulsive movements, may all be eliminated by the use of the mixtures suggested, without impairing the anesthetic effects of the gas. Operations requiring twenty minutes and upward can be safely performed with gas, but its administration had better be confided to an expert, and the mixtures recommended should be employed. Few, if any medical men, will have the necessary apparatus for the storing and administration of gas, hence no elaboration of the details of

this method of anesthesia will be attempted. The venous oozing under this form of anesthetic is freer than with ether or chloroform, and the color of the blood might alarm the inexperienced, but there is no real cause for concern because of the venous hue of the blood, which is what should be expected.

Treatment of Accidents Occurring During Anesthesia.—When explaining the causes of the accidents incident to anesthesia, the treatment of some was mentioned incidentally, such as clearing the pharynx of mucus by the mop; pulling forward of the tongue with the forceps ; securing free ingress of air when the nostrils were abnormally collapsible, marked deviation of the septum narium or hypertrophy of the tissues over the turbinates existed, by the insertion of nasal specula or keeping the lips and jaws slightly separated by a gag ; the expression of mucus from the chest by forcible compression of this during expiration, or by the induction of vomiting ; but the means to be employed when the respiration or circulation is failing, or when these have ceased, now require some special consideration. As a preliminary to any attempt to excite artificial respiration, any physical obstruction preventing free access of air to the glottis must of course be removed first. If the mouth be opened so that the lower teeth will clear the upper by depressing the chin by the fingers of both hands, while at the same time pressure of the thumbs behind the angles of the jaw forces this forward so that the lower teeth are slightly in front of the upper teeth, at once the stertorous breathing and evidences of obstruction to the entrance of air into the chest disappear in most instances ; this is because, from its attachments to the jaw, the base of the tongue is lifted—in the supine position occupied by the patient—until the pharynx is cleared sufficiently to permit air to readily enter the larynx. Sometimes during this maneuver the tip of the tongue will be seen to have fallen back, when protrusion of the jaw will fail to clear the pharynx. Seizing the tongue with forceps

and pulling it forward is often the quickest method of getting the tongue out of the way. Care must be exercised lest the respiratory movements of the larynx be interfered with by holding the tongue too firmly out of the mouth ; this would retard the re-establishment of normal respiration. Both the plans suggested also serve not only to clear away obstacles to the ingress of air, but also usually cause a resumption of the suspended respiratory movements. Grasping the tip of the tongue with the forceps, or by a towel between the thumb and fingers, will enable the anesthetizer to make rhythmical traction upon the tongue at the rate of 12 to 14 times per minute, which is one of the best methods of starting suspended respiration *if it has just ceased*. What shall be done in a case of ankylosis of the jaws when falling back of the tongue occurs, with or without accumulation of mucus or vomitus in the pharynx causing embarrassment of the respiration? Although a gag will force open a spasmodically clenched jaw, this instrument is of no avail in a case of close ankylosis. Fenger has shown that traction upon the hyoid bone, directed upward,[1] will open up the pharynx and clear the orifice of the larynx better than any other measure, and he has successfully employed this expedient by passing a loop of stout silk by means of a curved needle beneath and around the hyoid bone. Thrusting a tenaculum beneath this bone is a quick way of effecting elevation of the base of the tongue. After elevation of the tongue, clear the throat of all mucus and vomitus by the mop and by throwing the patient over so that the head projects beyond the edge of the bed face downward ; then roll the patient upon the back, and, maintaining the tongue well forward, institute artificial respiration, if voluntary efforts have not been excited by the preceding measures, traction, etc. If the respiration be shallow, or if from mucus in the bronchial tubes, or from pulmonary edema oxygenation of the blood is imperfect, artificial

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[1] The patient lying supine.

respiration should be at once commenced and maintained. Sylvester's method is both efficient and that most readily carried out. Failure of respiration without any obstruction to the ingress of air may occur from poisoning of the respiratory center in the medulla oblongata, or from a weakened heart failing to supply this with sufficient blood to enable it to carry on its function properly. Strychnin in proper doses, repeated *pro re nata*, administered hypodermatically, seems to be the most efficient remedy for either condition, although lowering of the head is also indicated. At times, $\frac{1}{100}$ of a grain of atropin sulphate hypodermatically will prove serviceable as a respiratory, cardiac and vasomotor[1] stimulant. Electricity employed to excite directly the action of the diaphragm by applying one pole over the base of the chest and the other over the course of one phrenic nerve is declared by Hare to be both useless and dangerous, but used so as to *irritate the sensory nerves of the skin* it may prove beneficial by exciting (reflexly) respiratory movements : it is rarely, however, available in an emergency.

Treatment of Sequelae.—Serious after-vomiting is best prevented by adopting the precautions already advocated, but in its presence, rectal feeding when the administration of food or fluid becomes imperative, and the administration of repeated doses of as hot water as can be borne by the mouth, is all that is usually requisite. Sometimes washing out of the stomach with hot normal saline solution will serve a good purpose, and this is especially true if fecal vomiting has preceded the anesthetic. One grain doses of acetanilid have been advocated by one author, repeated until 6–8 grains have been taken, but with the depression present with severe vomiting, this drug would seem dangerous.[2] A moderate dose of morphin hypodermatically will often succeed

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[1] Its effects must of course stop short of inducing capillary paresis, shown by reddening of the skin.

[2] Caffein and ice-bags to the epigastrium and nuchal region have been vaunted.

when all else fails, but the stomach must be kept empty. When mouth-feeding is commenced, this must be done by giving only a teaspoonful of liquid nourishment at stated intervals, gradually increasing the amounts. Partial or complete suppression of urine must be met by rectal, hypodermatic, or intravenous saline injections; by cold enemata when largely due to shock ; by digitalis, and dry cupping over the kidneys. Pneumonia, bronchitis, and pulmonary edema must be treated as when otherwise caused, employing in addition the ordinary remedies requisite for any existing shock, anuria, etc.

Choice of an Anesthetic.—When it is considered that the deaths from chloroform-anesthesia approximate 1 in 2500, the lowest figures being 1 in 6000, and that a number have been in patients under fifteen years of age, while ether is charged—at the worst—with 1 death in 14,000, it would seem clear that ether is preferable to chloroform as a general anesthetic. Although statistics are proverbially unreliable, when all available cases of both ether- and chloroform-anesthesia are considered, the number of cases is so large as to reduce the chances of error. Thus considered, ether will be seen to give about 1 death in 16,857 administrations, while chloroform shows a mortality of 1 in every 3142 anesthesias. These figures, of course, only give the risks during and immediately after ceasing to administer the anesthetic, not the later effects of either. It can also be said of ether that which is certainly not true of chloroform, that the former can be administered by an inexpert person, or at least the anesthesia can be safely maintained by such a one after its induction by an expert. From sudden reflex inhibition of the heart from irritation of the trifacial nerve-filaments in the nose and pharynx, death has resulted after the inhalation of thirty drops of chloroform.[1] Despite all of these apparent advantages

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[1] Preliminary cocainization of the nose and pharynx has been employed to obviate this, but so many instances of cocain-toxemia have occurred that this method cannot be recommended for general adoption.

of ether as an anesthetic, there are certain counter-balancing disadvantages which sometimes actually forbid its use ; indeed, if some of the late deaths resulting from the effects of ether were credited to their proper cause, the disparity between the dangers from ether- and chloroform-anesthesia would be less apparent.

Ether should not be administered to patients with acute bronchial and pulmonary complaints, and when the heart is sound, chronic ailments of the same parts render chloroform the safer agent to employ. Advanced renal disease should forbid the employment of ether as an anesthetic, but when any kidney trouble is slight and the condition of the heart renders the use of chloroform especially dangerous, an injection of a moderate dose of morphin and atropin hypodermically twenty minutes before commencing the administration of the anesthetic, will render the cautious induction of anesthesia by means of ether reasonably safe, because but little of the drug will be required, hence little irritation of the kidneys during its elimination will occur. The induction of anesthesia by ether in hot climates is difficult on account of its volatility, and it cannot be employed in army practice on account of its bulk and the time consumed in its administration. Ether is more dangerous for patients with aneurysms who require a general anesthetic than chloroform, because the former increases the blood-pressure. From its inflammability ether must not be used near a naked flame, although no harm can result if the flame is *kept high*, because ether-vapor is *heavy* and *accumulates near the floor*. From these statements it must be clear that chloroform is especially adapted for use in hot climates and in army practice. Chloroform is safer than ether in advanced renal disease, acute and chronic bronchial and pulmonary diseases in children, and during labor, when aneurysm is present, and in operations upon the nose and throat, requiring frequent removals of the inhaler. When ether causes a free secretion of mucus or such pulmonary congestion as to

interfere with the proper oxygenation of the blood, chloroform should at once be substituted. The necessity for the use of the actual cautery during anesthesia has long been considered a contraindication for the use of ether, but the removal of the ether cone for a moment with fanning of the ether-laden air away from the neighborhood of the patient, will render the cautery heat harmless. In many operations where there is any serious objection to the employment of chloroform for a long time, and where the cautery will be frequently needed, anesthesia can be produced by ether, all the soft-part incisions made, and hemorrhage checked from these, and chloroform substituted during the time the cautery is being used. Chloroform has its dangers at night as well as ether, but from a different cause. Its vapor becomes decomposed in the presence of a naked flame for instance, chlorin gas[1] being set free, which is injurious alike to patient and operator ; a free current of air will obviate this risk, which should never be forgotten, especially in cold weather or when administering chloroform in a small, ill-ventilated room.

Nitrous Oxid Gas.—This agent is especially adapted for short operations ; for comparatively short, bloodless operations, by the use of the mixtures suggested ; and when complete muscular relaxation is not requisite ; but it must be given by a trained anesthetizer, and requires special apparatus.

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[1] A chlorin compound is alleged by one chemist to be the injurious agent, not chlorin gas itself.

LECTURE XXXVI.

LOCAL ANESTHESIA; ADMINISTRATION OF GENERAL ANESTHETICS; POINTS REQUIRING ESPECIAL ATTENTION DURING THE INDUCTION OF ANESTHESIA WITH (1) ETHER AND (2) CHLOROFORM.

Local anesthesia is of conspicuous value when the operation is short and especially where the agent can be maintained for some time in contact with the tissues by means of elastic constriction applied above the part to be attacked, as during the avulsion of an ingrowing nail or a circumcision, the removal of small tumors, minor amputations, and the slitting-up of sinuses. Even major amputations have been done, when the risk of "mental shock" was not to be feared, but it must not be forgotten that cocain in large amounts sometimes kills as surely as chloroform. The special province of cocain- and eucain-anesthesia is operations dealing with mucous membranes, notably the conjunctival. Local anesthesia induced by cocain or eucain has sometimes served an admirable purpose when the operation was not a prolonged one, and the patient was too exhausted for the safe employment of general anesthesia. Thus, gastrostomy or a laparotomy for intestinal obstruction by bands, etc., has been done on a number of occasions. If any such operations are attempted, the surgeon must be quick, certain, well assisted, and the patient must be one who can be relied upon to obey implicitly. Local anesthesia by freezing is best suited to cases where necrotic infected tissues are to be curetted away, as those of a carbuncle, because they are rendered more friable. Opening abscesses, tapping the abdomen or pleura,

and the excision of small tumors can all be effected under the anesthesia produced by cold, but cocain or eucain are preferable. Freezing a part is followed by a free oozing which requires the maintenance of compression for its arrest, and the reaction is often very painful ; repair also seems occasionally unfavorably influenced by the freezing.

Administration of General Anesthetics.—After the adoption of all the preparatory treatment advised on pages 344, 345, the patient must be placed and maintained in a recumbent position during the whole process ; this is especially important during chloroform-anesthesia. The neck of the shirt must be unbuttoned, corsets removed, skirts loosened around the waist, and the patient be properly protected against loss of body-heat. During many prolonged and exposing operations, if an artificially warmed operating-table be not available, the limbs must be securely wrapped separately in blankets, or be enveloped in abundance of cotton-wool held in place by bandages, or blanket-bags can be placed upon the limbs ; hot-water bottles or bags should also be prepared for immediate use. The anesthetizer must have a mouth-gag to force open the jaws, often tightly clenched during partial anesthesia, a pair of tongue-forceps, a stout curved needle threaded with silk, or a tenaculum, if there be ankylosis of the jaws, and a throat-mop or sponge, both carefully secured so as not to be accidentally left in the throat; a hypodermic syringe should be ready filled with a solution containing a proper amount of strychnin. All these instruments should be carefully sterilized, lest from use in a previous case syphilis, tuberculosis, or diphtheria be accidentally conveyed. Ether is best administered by means of the Allis inhaler (Fig. 26), because it can be readily sterilized, admits sufficient air, and while apparently wasteful is in reality economical. A dropping bottle is advisable, since it enables the anesthetizer to maintain a more even saturation with ether of the inspired air. An efficient cone can be manufactured ex-

temporaneously out of a recently laundered (approximately sterile) towel and a newspaper. Fold a large towel longitudinally, usually so as to make three folds, but if it is narrow fold only twice, so as to give a width of about seven inches; fold a newspaper five or six
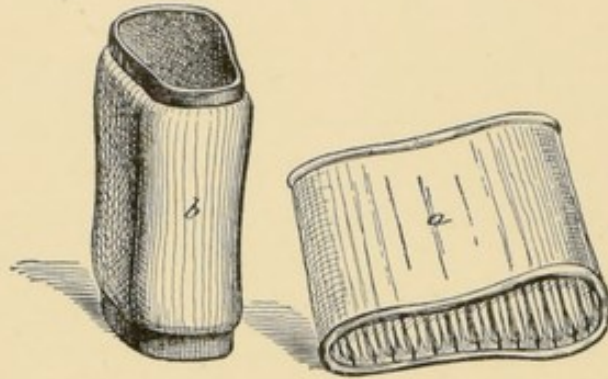
FIG. 26.—Allis ether-inhaler: *a*, aluminum or copper framework with gauze woven in; *b*, rubber cover for frame.

times of the same width and half the length of the folded towel; place the paper beneath the outermost fold of the towel, reaching only to its middle longitudinally; lay back the half of the towel free from the paper over the interleaved part; seize the two upper-
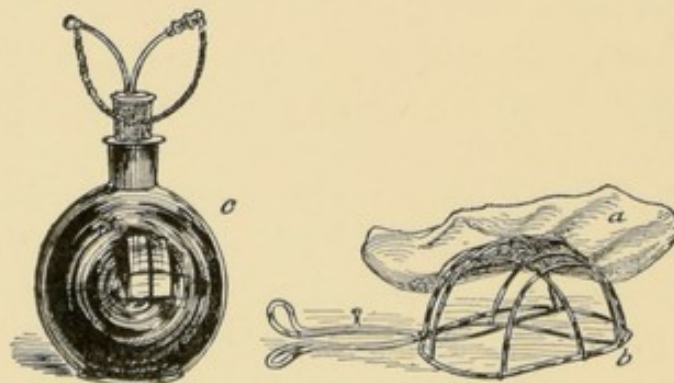
FIG. 27.—Esmarch chloroform-inhaler: *a*, stockinet cover, to be held tense by *b*, wire frame; *c*, dropping-bottle.

most corners of the folded towel, when by a single movement of pronation of *both* hands, passing one over the other, a cone will be formed requiring only one safety-pin to secure it: by keeping the surface upward where the extra layers of towels are, the cone will be

lined by three or five thicknesses of towel, affording
abundant material for the reception of, and surface for
the evaporation of, the anesthetic. Such a cone can
neither collapse nor be torn apart during the struggles
of the patient.

When chloroform is employed, the so-called Esmarch
inhaler with dropping-bottle (Fig. 27) is cheap, clean,
and efficient. If this is not at hand, a doubly or trebly
folded piece of lint about four inches square, or a folded
handkerchief must be substituted. The possibilities of
inducing anesthesia with nitrous oxid gas before com-
mencing the inhalation of either chloroform or ether
need only be mentioned, because, although excellent,
this plan is rarely feasible outside of a hospital.

**Points Requiring Especial Attention during Ether
Anesthesia.**—While the patient is struggling, a nearly
saturated vapor of ether with only about 5 per cent. of
air acts best, more air being gradually admitted as the
anesthesia progresses. It must never be forgotten that
partial recovery from the anesthesia during an operation
means only partial elimination of ether from the blood,
because a much smaller increment of ether than that
which was requisite at the outset will suffice to supply
the quantity lost and render the anesthesia complete ; a
forgetfulness of this fact while administering ether as
well as chloroform accounts for many of the accidents,
for as much of either drug is often poured into the
inhaler during the later stages of anesthesia as was
originally necessary at the outset of the adminis-
tration, thus producing a sudden over-saturation of the
inspired air and the blood. When commencing to ad-
minister ether, pour about half an ounce upon the cone
and place it before the patient's face at such a dis-
tance as will prevent the first few inspirations being so
charged with ether vapor as to excite coughing or chok-
ing. The patient should be urged to "blow out," as in
this way deep inspirations are taken, and this direction
is easier for the patient to comply with than one to

"breathe in deeply," when each such effort tends to induce choking or coughing. The inhaler should be gradually approximated until the nose and mouth are closely covered, and air fully charged with ether is inhaled. The respirations must be carefully watched to insure that they are regular and efficient, and if failure in either respect is even suspected, the cone must be at once removed. The throat will need to be cleared of mucus from time to time; the pulse must be watched, and the presence of commencing cyanosis, so often due to nasal obstruction, guarded against. Approaching anesthesia will be indicated by gradual relaxation of the muscles. If there has been any struggling or rigidity, a deepening respiration with commencing stertor, and finally, complete muscular relaxation with insensitive cornea and slightly stertorous respiration indicate complete unconsciousness; from this point on the insensibility should be maintained by the administration of the minimum of the drug, an ample supply of air being provided. In a few instances, where an abscess is to be opened, or a quick operation to be done—as an amputation because a crushed limb is maintaining shock—if the patient's confidence can be secured, anesthesia can be induced quickly and safely by crowding the ether from the start, insisting upon rapid and deep inspirations. The patient must be directed to hold the arm extended upright in the air at full length; in a short time this (the arm) will be seen to waver. Loudly order the patient to "hold up the arm" and push it upright again, urging him from time to time to "keep up your arm." Suddenly the limb will drop limp by the side, when it will be found that for from sixty to ninety seconds perfect anesthesia will exist; if the patient is allowed to recover consciousness, the ordinary cycle attending ether-anesthesia must be completed: this is what has been termed by some "primary ether-anesthesia."

Points Requiring Especial Attention during Chlo-

roform Anesthesia.—The same advice as to "blowing through" the inhaler should be given. Fifteen to twenty minims of chloroform should be dropped upon the inhaler or folded lint, and held at such a distance from the face as not to cause coughing, etc. As the sensibility of the respiratory mucous membrane becomes benumbed, the distance may be lessened, but the napkin or inhaler must never be nearer than one inch from the nose, because this interval insures that the vapor shall not be in a greater amount than 5 per cent., if the later increments of chloroform are added carefully drop by drop : this proportion (5 per cent.) should never be exceeded if possible, and 20 per cent. promptly kills. Careful attention should be paid to the respiration, which should be regular, if enough air is mingled with the chloroform-vapor ; the admission of more air by the immediate removal of the cone will usually at once restore the respiratory rhythm. The anesthetizer's fingers should frequently, it might almost be said, should be constantly on the pulse, to notice any changes in its regularity, frequency, or volume. Full anesthesia is announced by slight stertor during respiration, complete muscular relaxation, usually contracted pupils, and insensitive cornea. Sometimes the cornea retains slight sensibility *with stertor and contracted pupils :* do not push the anesthetic under such circumstances, but rather *lessen* the amount inhaled or remove the cone entirely, and in *a few moments the cornea will become insensitive.* Patients who keep the eyes completely or partially open during the anesthesia, opening them whenever the administrator attempts to close them, are apt to have syncope, or asphyxia during the anesthesia, or continuous vomiting after recovering from the narcosis. If pallor of the face, with or without sudden dilatation of the pupils occur, at once suspend further administration of the drug, invert the patient, draw forward the tongue, administer strychnin hypodermatically, and reinforce the failing or suspended respiration by artificial (best forced)

respiration. Even when respiration has not actually ceased, increasing its freedom will serve to empty the upper air-passages of chloroform-vapor and aid in the elimination of the same from the blood. Always watch both respiration and pulse, temporarily suspending the inhalation when full anesthesia has been secured, maintaining this by a carefully regulated administration of the minimum of chloroform. The anesthetizer is derelict in his duty if he pays attention to anything but the condition of his patient, his duties being usually more important, so far as danger to life goes, than those of the operator.

Local Anesthesia.—This can be secured by the application of a mixture of pounded ice and sodium chlorid, tied up in a piece of gauze or thin muslin, kept in contact with the parts until they are frozen ; placing a piece of metal, as a knife-blade, between the freezing mixture and the skin to be dealt with will hasten the congelation by conducting away the caloric in the tissues more rapidly. The advantages of this method of anesthesia are obvious, no apparatus being necessary, while the materials are nearly always at hand. The disadvantages are the impossibility of securing asepsis and the dangers of sloughing, as, for instance, when used on the scrotum. By employing an ordinary spray-apparatus, parts can be readily frozen by means of rhigolene or even ether; both of these substances are practically germ-free. The convenient glass tubes placed upon the market containing ethyl or methyl chlorid, by means of which a fine stream of these substances can be directed upon any desired part, are both convenient and efficient : sloughing is also possible after their use. Where freezing will be too evanescent in its effects, the dermatic and hypodermatic use of various solutions of cocain often render possible the performance of operations of considerable magnitude, when the trouble is located in or beneath cutaneous covered surfaces. When possible an elastic band should be employed to so interrupt the return circulation that the anesthetic is retained in the tissues, which much

facilitates its action. Where this cannot be dóne, and especially for incisions through the skin, the infiltration-method introduced by Schleich serves an excellent purpose. With one of the solutions, the formulas of which will presently be given, a small area of skin is to be infiltrated by a drop or two of the solution introduced into the substance of the skin itself by means of a hypodermic needle, careful asepsis being maintained throughout. Waiting for a few seconds, the hypodermic needle is next inserted inside the whitened anesthetic area, the point carried on in the line of the proposed incision, and a new area of skin infiltrated ; by continuing this procedure, a growth may be circumscribed by a series of these punctures, and then the base of the tumor and the surrounding tissues can be painlessly infiltrated. In comparatively prolonged operations infiltration of the tissues will have to be repeated more than once, as the operation progresses, because the anesthetic escapes as the tissues are incised. It will be seen from the strength of the formulas employed that it is hardly possible that a dangerous amount of cocain can be absorbed.

Formulæ.	No. i.	No. ii.	No. iii.
Cocain hydrochlor.,	grs. iij ;	grs. iss ;	grs. $\frac{1}{6}$;
Morph. sulph.,	grs. $\frac{1}{3}$;	grs. $\frac{1}{3}$;	grs. $\frac{1}{12}$;
Sod. chlorid,	grs. iij ;	grs. iij ;	grs. iij ;
Aqua,	℥j ;	℥j ;	℥j.

The formula employed for years at the University of Michigan Hospital is as follows :

Cocain hydrochlor.,	gr. j ;
Morph. sulph.,	gr. $\frac{1}{9}$;
Acid carbol.,	gr j ;
Sod. chlorid,	gr. j ;
Aqua,	℥j.

To render the conjunctiva and cornea absolutely insensible it is only necessary to instil a few drops of a 2–4 per cent. solution of cocain into the eye, two to four

times during about twenty minutes preceding any operative interference. To secure absence of pain when dealing with mucous membranes covered by a thick epithelium, as those of the rectum and vagina, 10 per cent. solutions are recommended and it is alleged that few accidents have followed the use of these stronger solutions. This cannot be affirmed of such strong applications to the urethral and nasal mucous membranes, where dangerous or fatal accidents have frequently occurred, so that a repetition of applications of a weak solution, or the maintenance for a time in contact with the parts to be dealt with of one containing an amount of cocain not dangerous if all should be absorbed, is the safer plan. Beta-eucain is believed to be less poisonous than cocain ; can be repeatedly boiled for the purpose of sterilizing it ; does not alter when kept in solution ; and does not irritate the tissues. Braun recommends the following formula which he contends will maintain the anesthesia for from ten minutes to one hour :

Beta-eucain,	gr j ;
Sodii chloridii,	gr. viij ;
Aq. destil.,	℥ ij.

The symptoms of cocain-poisoning may evince themselves by syncope, delirium, or tetanic fixation of the respiratory muscles. In mild cases, nothing beyond pallor, tremor, restlessness, and feeble heart-action will be noted : for these cases, rest in the horizontal posture and stimulants are all that is necessary. In more severe cases, death is threatened from paralysis of the respiratory center and by tetanic fixation of the respiratory muscles. Respiratory failure must be combatted with strychnin administered hypodermatically, but where generalized eclampsia, or fixation of the respiratory muscles occurs, chloral has been found to act well, administered by the rectum. Inhalations of amyl nitrite or hypodermatic injections of nitroglycerin have been also employed for some cases of cocain-poisoning attended by

generalized convulsions. If violent delirium supervene, hyoscyamin in proper doses, administered either by the mouth or skin may be tried. Chloral would be indicated were it not that the heart is so frequently feeble, but when this organ seems strong, chloral can be given. Physical restraint may become necessary in some delirious cases ; the bladder will require emptying ; and diuretics may be necessary. Sleep must be secured by the proper remedies when wakefulness is a pronounced symptom. Much more space might be occupied in describing other methods of securing anesthesia, but enough has been said to indicate the directions in which efforts should be made to secure local anesthesia.

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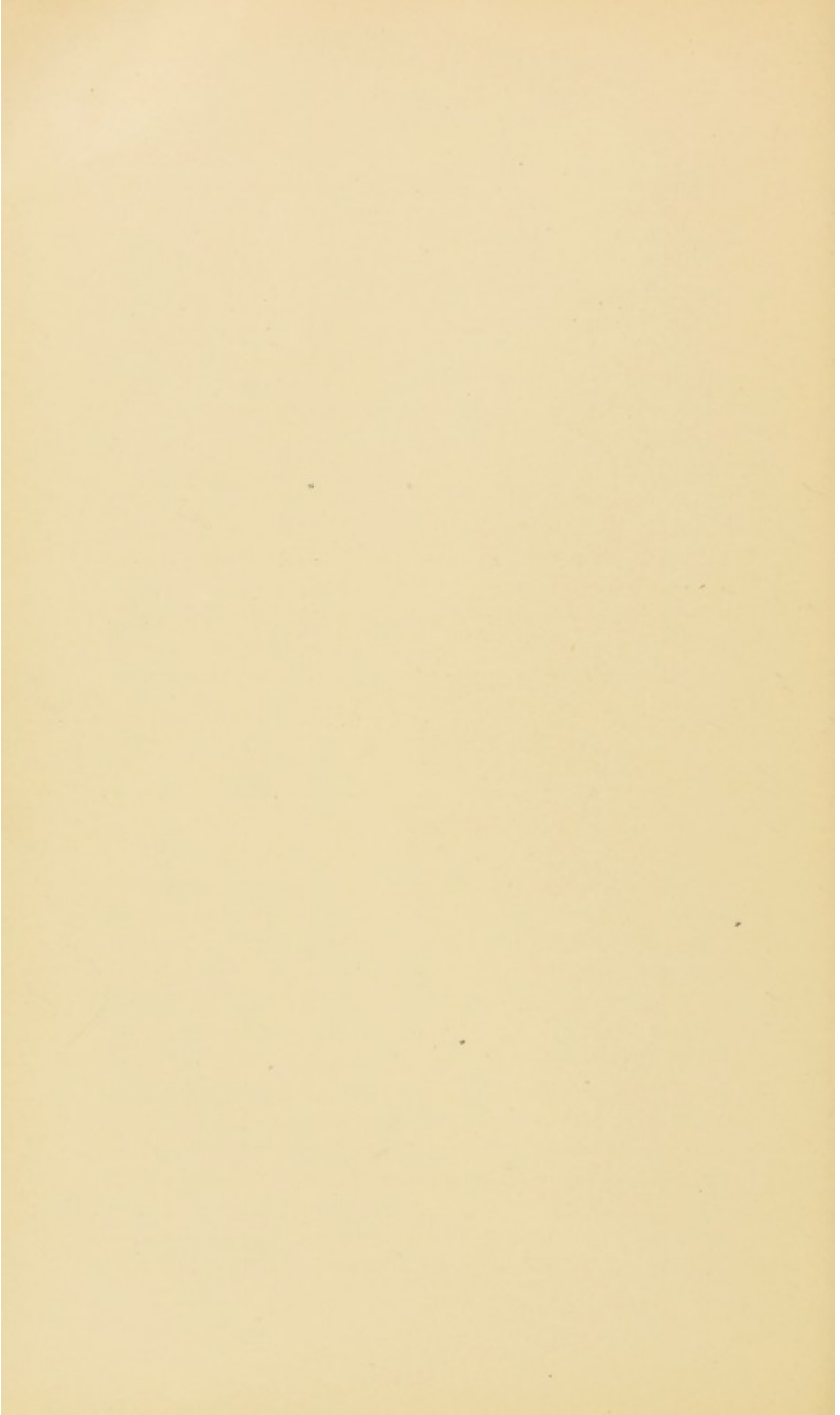
APPENDIX.

CONTAINING A RÉSUMÉ OF THE PRINCIPAL VIEWS HELD CONCERNING INFLAMMATION.

By W. A. Spitzley, A. B., M. D.

To the mind of the average student who reads modern text-books of pathology, surgery, or surgical pathology, the descriptions and discussions of the processes which we call inflammation present a very confused and unsatisfactory picture ; and the student, instead of having something definite to grasp, something which gives him positive information, finds himself surrounded by a great mass of facts, true enough in themselves, but often withal so inconsistently arranged or so illogically stated, that they fail of their purpose to teach him. His mind is not yet trained to select the useful and discard the useless, nor can it digest and assimilate all that is presented to it ; it consequently gains very little for itself.

Consistent with his belief that a teacher ought to present his own individual views on a subject, not surfeit the student with everything that has been written or said about it, the author has in the foregoing lectures, as you have seen, taken a position on the question of Inflammation somewhat more decided than most other clinical teachers, and has set forth his belief logically and consistently, that the only processes which are truly inflammatory are those in which are concerned micro-organisms, their products, or both. Since, however, this subject in Pathology has given rise to more discussion than almost any other, it was deemed advisable and proper to present some of the more important theories

advanced by scientists from time to time, concerning inflammation. I shall endeavor to do so briefly in this appendix.

The clinical significance of inflammation we all know; there is no discord in the opinion of pathologists from that standpoint; it is only when an accurate definition of the process is attempted that differences arise. The chief subject of contention is the determination of the dividing line between processes of repair and inflammation, and the part which hyperemia plays in each. Are both conservative? If not, when and where does the reconstructive process stop and the destructive begin?

The earlier workers, those before the days of the microscope and before bacteriology became a recognized science, were interested in a purely clinical way: the finer microscopical observations were entirely unknown to them.

Celsus, the learned Roman writer on many subjects, including medicine, gave us perhaps the first description approaching accuracy of the inflammatory process in connection with wounds and disease. His views, which were received and approved by Galen, have come down to the present almost unchanged. He mentions four of the five now classical signs and symptoms—Dolor, Calor, Tumor and Rubor, to which has been added by later writers, Functio Laesa. He recognized, also, some of the constitutional symptoms which always accompany a severe inflammatory process—diminished secretions, dry and foul tongue, and especially fever. The fever he regards as an agent conservative in nature and tending to dispel the pathological conditions; he says that "if the inflammation be slight, or if there be present pain without prominent evidences of inflammation, without redness and swelling, the fever will of itself drive this away." The redness he properly ascribes to the increased amount of blood in the part, the heat to the blood and to the violent tissue-destruction going on. Of swelling he writes: "Excessive tumefaction in

a wound is dangerous ; the absence of tumefaction is dangerous to the last degree : the former is a proof of inflammation to a high degree ; the latter, of a system dying from exhaustion.'' Readily do we see in the first of these statements his recognition of the possible harm of large collections of infectious material, even though circumscribed ; and the second gives us a picture, too often seen even now, of the condition in which no limitation of the infectious process has taken place ; but, instead, the poisons are being disseminated by the blood and lymph throughout the body, and the patient is dying in truth from exhaustion—the exhaustion of septicemia. On fever, Celsus makes this observation : ''One may know at an early period that a wound will heal, provided the senses are retained and no fever is present ; nor ought we to be alarmed even though fever should continue throughout the inflammatory stage accompanying a large wound. That fever, however, is dangerous which either supervenes upon a slight wound, or outlasts the inflammation, or when it does not put an end to a tetanus or to convulsions which have supervened on a wound.'' It is interesting to note a few of the therapeutic principles suggested by Celsus in the treatment of inflammation. Some of them are decidedly unique ; others can, with advantage, be followed to-day. He writes concerning inflammations, especially those involving the joints of extremities : '' The position of the limb also requires some method, for if agglutination be an object, it should be elevated : when in the inflammatory stage, it should not be inclined either way ; when the pus has begun to flow, it should be in a dependent position. Rest is also an excellent remedy, for motion and walking are only proper for people in health. Bathing before the wound is pure is one of the worst things that can be done ; for it makes it humid and foul and then gangrene is usually the result.'' He advises free purgation in severe inflammations, refuses wine in most cases, restricts patient's food only slightly, and

pays intelligent attention to the local conditions. "The wound having been treated for two days, on the third day it requires to be opened, and the sanies having been washed away with cold water, the same dressings are to be introduced. On the fifth day the inflammation is at its height. Then it is that, uncovering the wound, it becomes one's duty to consider its color ; if livid, or pale, or varicolored, or black, one may know that it is a bad wound, and whenever such an appearance is noticed there is just ground for alarm. It is most favorable that a wound be white or ruddy. A hard, thick, painful skin denotes danger ; but when it is free from pain, thin and soft, it is a good sign. But if the wound be agglutinated or if it be slightly swollen, the same applications are to be continued as were first employed : if the inflammation be severe, and there be no hope of agglutination, suppuratives are to be applied. And now also hot water becomes useful, that it may dissipate the matter, mollify the hardness, and excite the pus." The suppuratives, so-called, were fomentations of barley, or linseed, or flour.

Galen, following Celsus, concurred in most of his views regarding inflammation and accepted them almost unchanged ; he offered, however, a definite classification of the different forms as follows : 1, erysipelatous ; 2, pneumatous ; 3, phlegmatous ; 4, phlegmonous and 5, scirrhous. He also added a few new features in the treatment, principally in the form of medicinal agents supposedly possessing specific action against the various inflammatory processes.

Boerhaave, at the beginning of the Eighteenth Century, realizing that the phenomena of inflammation were being constantly confused with those of other pathologic and morbid processes, that the term was being applied to conditions utterly different from those described by Celsus and Galen, and that, therefore, the four cardinal signs were losing their significance, attempted to work out the real nature of inflammation, and to establish the

presence of some constant change which would allow the positive recognition of the inflammatory process in distinction from all other similar ones. He concluded that it was due to an obstruction of the minute vessels of the affected area, and that all the phenomena were dependent upon this arrest of the circulation. But as Hunter says, "this was certainly too confined an idea of all the causes, and reduced all inflammations to one species. The only distinctions must have arisen from the nature of the obstruction; but this could never account for the action of many specific poisons." So, while Boerhaave's leading doctrine was taken up by such men as Henle, Magendie, and others, and caused the presentation of many theories along its line of teaching, it soon was dropped as unsatisfactory and unscientific.

John Hunter, in his work on the *Fundamental Principles of Inflammation*, published at the end of the Eighteenth Century, writes at great length upon the subject. He regards inflammation as an effort on the part of the organism, after injury to any of its tissues, even to the slightest extent, to restore these tissues to integrity. In other words, inflammation is not a pathological process but a conservative one calculated to aid in the process of healing after injury or disease. He finds, even in those cases in which suppuration occurs, that inflammation is beneficial in bringing about a cure, only it does so in a way different from that in which primary union of a wound is secured. The latter part of this statement is certainly correct, the way is different; but how suppuration can be regarded in any other light than as a process detrimental to tissue-repair, inhibiting and oftentimes preventing healing, I believe no one can satisfactorily demonstrate. Hunter then states that inflammation must follow every injury to tissue, and for its occurrence ascribes three causes: first, mechanical violence which causes a wound or bruise that cannot heal except by inflammation; second, an irritation which affects the functionating power of a part, pressure,

heat, cold, friction, and fevers of all kinds; third, a particular disposition of the parts—*i. e.*, localized destruction in the form of boils, abscesses, etc. He recognizes three effects of the process, and therefore designates three kinds of inflammation, adhesive, suppurative, and ulcerative. The first is the most useful, not only bringing about primary union in wounds and obliteration of cavities, but also helping to limit the destructive tendency of the suppurative and ulcerative kinds, whose "salutary effects are in many instances not so evident." One form of inflammation he grants is not entirely propitious for healing—"where it cannot accomplish the salutary purpose of altering the diseased mode of action, as in cancer, scrofula, and venereal disease, it does mischief." With this single exception, however, he considers it as only a disturbed state of parts, not as a disease, but as a salutary operation consequent either to some violence or disease. He calls attention to healthy and unhealthy inflammations, the differentiation being made not so much because of variations in the symptoms of each but because of the nature of the individual affected. The healthy kind always attends a healthy constitution or part ; is more powerfully restorative in action, and appears to result more from a stimulus than from an irritation ; the unhealthy although ultimately the same in effect, is found in unhealthy constitutions. Hunter is by no means alone in stating this universal occurrence of inflammation ; nearly all writers, before these days of aseptic surgery, believed it to be a necessary accompaniment to the healing process. And perhaps it was—very few wounds went on to healing by means of the simple, uninterrupted, economical processes of repair ; infection was present in nearly every case : not suppuration, mind you, not by any means, for wound-contamination does not even in the majority of cases, mean suppuration, any more than the mere presence of a few germs means infection. But the restitutio ad integram was interrupted by invading organisms,

though this interruption may very speedily have been overcome by the disease-resisting powers of the tissues and the body.

Dr. Samuel D. Gross, in his work published in 1866, says that "all accidents whatever their nature or degree, are necessarily followed, if the patient survive their immediate effects, by inflammation. The little wound made in venesection, the incision made in cupping, and the bite inflicted in leeching, would never heal without the aid of this process ; the parts would remain open and be the seat of incessant bleeding, or they would become festering and putrid sores." The position of such men is readily understood : they failed to appreciate the fine points of difference between the minute changes in the processes of repair—processes which are absolutely conservative from the beginning of the preliminary acceleration of the blood-stream until the last fibroblast has become matured—and those going on in inflammation, in which so much energy is diverted from rebuilding tissue to working against active interfering influences in the form of germs or their products.

Rokitansky finds the characteristic phenomena dependent upon a condition of the blood itself, the changes in the vessel walls—*i. e.*, thinning, laceration, increased permeability, etc., being entirely secondary and resulting from the blood-state. He regards dilatation of the vessels, slowing of the blood-stream and stasis, as necessary accompaniments of every inflammatory process. The dilatation is not due to relaxation resulting from the exhaustion due to the initial contraction ; but, together with Henle, he believes the retardation of the current and the crowding together of the blood-cells to be a primary phenomenon and due, according to Henle, either to an augmented affinity between the parenchyma and the blood and especially to an abnormal attraction of the blood, and the blood-corpuscles in particular, by the affected parenchyma—the attraction-theory—or to paralysis of the vessel-nerves, caused by increased

excitement of the sensory-nerves—the neuropathological theory. Stilling, likewise, believes in a perversion of the normal nerve-functions, but considers the paralysis as the result of the direct effect of the inflammatory irritant. Rokitansky writes, "For our own part, we hold stasis to be dependent on the following momenta : (*a*) The cohering, crowding and impaction of the blood-disks within the capillaries, the blood-plasma being partly withdrawn into the veins. (*b*) The thickening of the plasma and its saturation with fibrin and albumin, owing to the transuding of blood-serum through the distended and thinned blood-vessel walls. (*c*) The accumulation of the colorless globules—that is, nucleus and cell-formations—along with blood-corpuscles, then conglutination and the delicate, transparent, fibrinous coagula collaterally developed. This is perhaps the most important stage in the inflammatory process, as at once illustrating the stasis itself and embracing plastic processes in which the blood engages when arrived at this point." The exudation of fluid from the capillaries, he sought to explain not so much by increased intravascular pressure but by "the thinning and increased permeability of the walls of the blood-vessels, produced by their distention ; and additionally, perhaps, by an effort at equalization between the condensed plasma in the vessels and any thinner serum before exuded." In accounting for stasis by means of the attraction-theory, Henle has had some followers, Paget, Vogel, and others, who likewise ascribed the phenomena of dilatation and stasis to an abnormal attraction of the tissues for the blood.

In 1854, Virchow set forth the cellular theory of inflammation. He believed that it was a slightly perverted, nutritional disturbance, depending upon an interchange of materials between the tissues and their nutritive fluids, taking place in the avascular as well as in the vascular structures. He lays little stress upon the Celsian signs, Rubor and Calor, because they are an

evidence of simple hyperemia which, he says, may exist almost indefinitely in a part without inducing nutritional changes : Dolor, because it is inconstantly present ; Tumor, because it is found in many conditions not at all partaking of the nature of inflammation ; Functio Laesa, because perverted function necessarily implies change in cell-integrity. The beginning of an inflammation is always irritation, from either external or internal causes ; this induces unusual activity of the cells, which are made abnormally inclined to take up materials from the blood or other fluids. The cells, therefore, become laden with these materials, which may not in themselves be harmful, may not even be transformed into harmful substances ; still, we are led to believe by Virchow that these inflammatory changes are more or less dangerous to the part of the organism affected. Unlike Rokitansky, who regarded the changes in the blood as primary, Virchow considers the perivascular phenomena occasioned by the inflammatory irritant as the important ones, the circulatory disturbances being, according to his conception, merely logical results of the former. Exudation is a common but not essential phenomenon ; it is explained partly on mechanical grounds—increased intravascular pressure—and partly on the ground of increased attraction of the tissues for blood-constituents. In the cells collecting in the affected area, he recognizes none as coming from the blood or lymph : all originate from a proliferation of the tissue-cells themselves. In a word, then, Virchow sought to establish that inflammation was nothing but a disturbance or perversion of cell-nutrition.

Cohnheim may practically be considered the leader of a new school devoted to the study of these processes ; and his observations are deserving of special consideration, since he was the first man to successfully investigate inflammation in the living animal under the microscope. Working with a frog's tongue so arranged that the circulation in the smaller vessels could be observed, he irritated a certain point with croton oil, and noted a

number of the changes which we now so well know. The stage of contraction of the vessels passed quickly by ; then came the acceleration of the current and the vessel-dilatation ; then the retardation of the blood-flow, and, finally, the peripheral arrangement of the white cells and the accumulation of the corpuscles within the distended capillaries and veins, together with exudation into the perivascular tissue of both fluid and corpuscular elements. The outpouring of the fluid Cohnheim referred not to increased intravascular pressure but thought it explained by a molecular rearrangement in the cells of the vessel-walls. In fact, this is essentially Cohnheim's view of inflammation, that the vessel-wall changes are the determining factors of the phenomena observed. He called attention to the fact of the extensive emigration of leukocytes and their presence in the exudate, thus disproving Virchow's statement that all the cells collected in the inflamed area resulted from proliferation of the tissue-cells there. At first, together with Binz and others, he regarded this emigration as an active process ; and this is undoubtedly true, since Appert and Kerner have demonstrated beyond cavil that, if the leukocytic mobility is diminished with quinin or iodoform, emigration is markedly restricted. Later he agreed with Schklarewsky that this phenomenon was wholly passive, the result of pressure from within. Cohnheim, unlike many of the earlier men, finds nothing conservative nor reparative in inflammation: it is simply a molecular change, resulting from injury of the vessel-wall.

Arnold's theory of the enlargement of the minute spaces between the endothelial cells, the stomata, is not without reason. While, of course, it does not begin to explain all the salient features of the inflammatory process, it deserves consideration. Not only, according to Thoma and Engelmann as well as Arnold, do the stomata, for purely physical reasons, enlarge when capillary distention occurs, but the intercellular ce-

ment-substance changes, softens, and therefore becomes more permeable to fluids and blood-cells, red as well as white.

Von Recklinghausen takes a stand intermediate between Cohnheim and Virchow, holding that the inflammatory process results both from the action of some irritant on the tissues and from changes in the vessel-walls.

Stricker considered mainly the suppurative forms of inflammation, and so it is difficult to correlate his work with that of other men. In general, it may be said that he emphasized the clinical sign of redness, counted stasis of little importance, and believed the essential factor to be a lesion involving the tissue or at any rate involving the nerves supplying the tissue. Active hyperemia begins the process ; next comes exudation, and, finally, in many cases occurs the formation of pus. In close accord with Stricker's are the views of Heintzmann, who likewise found the initial changes in the connective tissues, which he believed were wont to undergo a reversion to the embryonal condition. He, however, looks upon the circulatory disturbances as being of reparative and nutritive value.

Weigert inclines to the clinical side, and calls attention to the necessity of paying proper regard to the classical signs and symptoms of Celsus, especially swelling. This is always the result of exudation, and for him locates the primary disturbances in the vessel-walls. Microscopically he notes the usual characteristic changes, dilatation of the vessels, retardation of the current, white blood-cells appearing in the marginal stream, and diapedesis, which latter process he has shown to be an active one. With Cohnheim he acknowledges certain changes in the vessel-walls, but is not satisfied with his neuropathological theory of dilatation. The changes in the cellular tissue, proliferations, etc., he regards not as the result of the inflammatory process, but as the effect of the primary tissue-lesion. In other words, all tissues

are damaged by an irritant, and, consequently, nutritive phenomena may follow ; but it is only when the vessels are directly or indirectly injured that inflammation takes place.

Metschnikoff's theory is so well known and has been made so prominent by discussion, that it is necessary to do but little else than mention it. Laying great stress upon the fact that there exists the power of attraction and repulsion between cells, or between cells and inert matter—positive and negative chemotaxis, so-called— he constructed the phagocytic theory of inflammation : "The essential and primordial element of a typical in-flammation is a reaction of the phagocytes against the harmful agent." This carries us back to Hunter and others of the earlier men, who, likewise, regarded the inflammatory process as a salutary one. To Metschni-koff, inflammation means phagocytosis, "an adaptation of an organism to its environment, which has been de-veloped in its never ceasing struggles against deleterious agents." The phagocytic cells are not cells of a par-ticular class : they are in the vessels as well as in the tissues, both fixed and wandering, even endothelial cells having, to a certain extent, this power of taking into themselves foreign matters. By some, this phenomenon of phagocytosis is regarded as an act of nutrition ; by others as one of excretion ; by Metschnikoff as a strug-gle between the defending cell and the offending noxious agent. The substances taken up are disposed of in various ways—digested and assimilated, or excreted, or retained within the cell.

Grawitz, whose ideas correspond fairly well with those of Stricker, deserves a word of mention because of his highly interesting and unique theory concerning the cells found in cellular exudate and in pus. He says they are only to a slight extent accumulated and per-verted leukocytes, but are in reality what he calls slumber-cells. These slumber-cells lie normally in great quantities among the tissue-cells, and become

recognizable to us, by means of stains, only when an inflammatory process is determined.

Landerer, in 1885, put forth an explanation of the phenomena of inflammation which he called the mechanical theory. He assumed an equilibrium existing between extra- and intravascular pressure, dependent not altogether upon the vasomotor regulating apparatus, the vasoconstrictors, dilators, and perivascular ganglia, but largely upon the elasticity of the surrounding intercellular fluid media and enveloping tissues. These elastic tissues then give a certain amount of support to the vessels as a whole. Now, when some injury to the tissues occurs, their elasticity becomes impaired, their functions damaged, and circulatory changes must of necessity follow. Hence, are occasioned vessel-dilatation, retardation of current, and all the other phenomena of inflammatory hyperemia. He does not regard this as the only change in the vessel-walls, but, with Cohnheim, agrees that there may also be some specific chemical action affecting them as well as the extravascular tissues.

As expressive of the views of modern pathologists on this engrossing subject, I will conclude with recording a few of the observations of Dr. Ernst Ziegler. "Inflammation," he says, "is essentially a local tissue-degeneration combined with pathological exudations from the blood-vessels, caused by some injurious agency, with which are associated sometimes earlier, sometimes later, tissue-proliferations leading to regeneration .... The circulatory disturbance characteristic of inflammation is shown only when the slowing of the blood-current and the pathological exudation from the vessel sets in."

The retardation and exudation, he believes, with Cohnheim, Samuel, and Arnold, are due to a modification in structure, an alteration, of the vascular walls. The congestive hyperemia which precedes the inflammatory process he regards as no essential part of it but as the result of irritation or paralysis of the vasomotor

nervous system, or to a direct action on the walls of the vessels. The lasting dilatation of the capillaries he ascribes to the relaxation of the connective tissue surrounding them. The emigration of leukocytes is an active process. The outpouring of the fluid exudate he refers to "the alteration of the vessel-wall, in consequence of which its secretory function suffers a disturbance." The extravascular tissue-changes, such as regeneration and proliferation, he regards, as seen in his definition of inflammation, as intimately associated with the inflammatory process.

I have endeavored in all the above to give a concise statement of the observations of many of the more prominent workers in this particular field of pathology. It is necessarily abridged : a full exposition would require a volume. I have touched upon only the determining features of each individual view ; have purposely omitted unimportant minutiæ, and have abstained almost entirely from any critical remarks ; it is, with intent, an historical composition.

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