**Inflammation regarded as a physiological reaction to an infecting lesion : the oration of the Hunterian Society, delivered at the London Institution, February 11th, 1885 / by F. Charlewood Turner.**

# INFLAMMATION

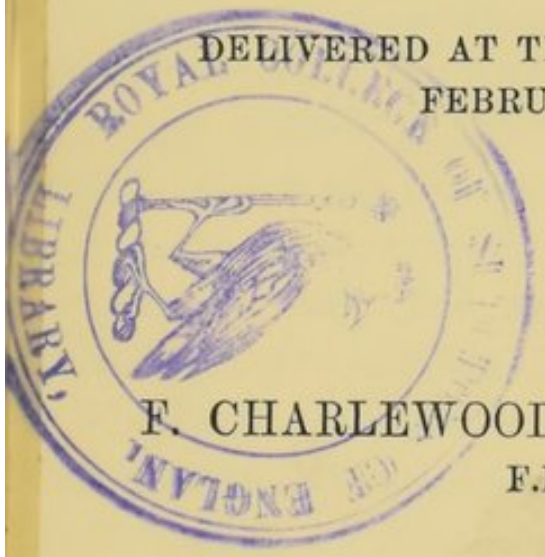
REGARDED AS

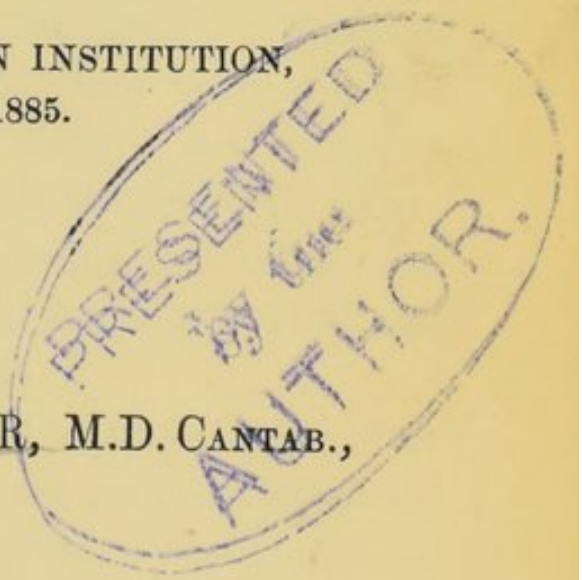
## A PHYSIOLOGICAL REACTION TO AN INFECTING LESION.

THE

## ORATION OF THE HUNTERIAN SOCIETY,

DELIVERED AT THE LONDON INSTITUTION,
FEBRUARY 11TH, 1885.




BY

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THE

# ORATION OF THE HUNTERIAN SOCIETY.

## 1885.

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Mr. President and Gentlemen,

In addressing you this evening I have, in the first place, to express my great regret, in which all who hear me will fully sympathise, that the distinguished member of our Society, whose place I take, has been obliged through failure of his sight, to resign his hospital appointments and to retire from active professional work, and is prevented from discharging the honorable and responsible duty for which he had been selected, and which he had undertaken to fulfil.

Mr. James Adams is well known to the members of this Society, who are indebted to him for services rendered in the office of Secretary during several years, and he has gained our esteem by his professional acumen and judgment, and by his singular skill as an operator, both in general surgical practice and in the special department of ophthalmic surgery. His personal character and geniality have won for him warmer feelings than those of respect and esteem alone in a wider circle of friends, all of whom will deplore the unfortunate circumstances by which a useful and distinguished professional career has been so prematurely interrupted.

I have also to express to you, sir, and to the members of the Council of the Society, my sense of the honour

conferred upon me by the request that I should undertake this important duty.

This responsibility I ventured to accept because there is a subject, and one most appropriate to this occasion, on which my thoughts have been much engaged, and about which I hoped to be able to make some observations deserving the consideration of the members of the Society, and such as might not unprofitably occupy the time allotted to me.

No subject could be more appropriate to an occasion like the present, than that of the pathology of inflammation, in which Hunter was so especially interested, about which he thought and wrote so much, and to the elucidation of which he so largely devoted his great powers of observation and his ingenuity as an experimenter. And I think that this subject, about which I am going to speak, is especially appropriate at the present time, because I am myself convinced that the confusion and obscurity in which the subject has become involved is due, not so much to obscurity or insufficiency in the facts themselves, which recent research has done so much to elucidate, as to their being regarded from an erroneous point of view, owing to the departure of some modern pathologists from the clinical usage of the term by Hunter, and from his conception of the nature of the inflammatory process; and because I am convinced that this obscurity of the subject in great part disappears when the facts are fairly viewed from Hunter's standpoint, in the light of the results of more modern investigation.

Considering the fundamental importance of this subject, of which Hunter speaks as "an operation of the body" requiring our greatest attention, and "one of the

most common and most extensive of any in the body;" and of which Travers speaks in the yet stronger terms quoted by Paget, stating that "a knowledge of the phenomena of inflammation, the laws by which it is governed in its course, and the relation which its several processes bear to each other, is the keystone to medical and surgical science;" there is surely no chapter in our text-books less satisfactory than that which treats of inflammation.

Notwithstanding the numerous and extended researches which have been made on the subject of the pathology of inflammation, and the great skill and scientific acumen which have been employed in these investigations, so great is the obscurity in which the subject is involved, and so inadequate are the proposed definitions of the condition felt to be, that in the most important text-book on pathology which has been recently published in England, in Macalister's translation of Ziegler's 'Text-book of Pathological Anatomy and Pathogenesis,' inflammation is described as a complex of many processes, partly vascular and partly textural, of which it is impossible to give a concise and exact definition.

That, in every condition of damage to living tissues there is, with this complex of abnormal vascular and textural phenomena, a combination also of remedial and reparative processes with the structural lesions produced by the injurious agent, is a result of the necessary provision made in the organisation of the body, for the maintenance of the nutrition of injured parts preparatory to, and during, the process of permanent repair.

Damaged tissue,—*i.e.* tissue in which there is structural discontinuity or derangement, with more or less extensive

vascular stasis, disturbing without arresting its nutritive function,—being reduced to a state approximating to that of a non-vascular structure, (but not, as has been stated, to the condition of ordinary matter while vitality remains,) is necessarily in a state of diminished vitality and of arrested function; and, at the same time, it is necessarily also in a condition in which there is a modification of the process of nutrition, adapted to the alteration in its structural arrangements. And we know that this necessary modification of the nutritive process in damaged tissue is, in fact, provided for (1) by the coagulation of the blood in the relaxed or wounded vessels, and of the plasma exuded from them—this constituting the first step in the process of repair of injured tissues, in operation from the moment of the infliction of the injury —the fibrinous coagulum closing up the divided vessels in a wounded part, and forming the temporary uniting medium between its surfaces, and in every condition of damage from injury or disease, affording support to the weakened structures, and sustaining their nutrition by forming a sponge-like structure permeable to the nutritive fluids; and (2) by an increased afflux of blood to the part, compensating for the effect of the impediment presented to its nutritive function, comparatively slight where the effects are those of mechanical injury alone, but more or less intense where an irritant substance is present.

When, therefore, it is said that inflammation is "a change wrought in the natural act of nutrition then existing in the wounded part" (Bowman), or "a modification of the existing normal processes" (Goodsir), or when it is stated, on the other hand, that it is a condition of lessened vitality and arrested function, according

to the views of Lister and Burdon Sanderson, such statements appear axiomatically true, but in effect to amount to no more than saying that inflamed tissue is structurally injured, but not dead.*

But an objection must, I think, be made to Burdon Sanderson's more explicit statement that the *essential nature* of inflammation is *fully* expressed in the word damage; to the assertion, that is, that there is no essential difference between a condition of inflammation, as exhibited in its most typical form in traumatic inflammation, and any other condition of damage; in accordance with which the state of a freshly made wound is spoken of by Mr. Watson Cheyne, in a recent publication, as being in a state of intense inflammation.†

When Burdon Sanderson defines inflammation, in accordance with these views, as "the succession of changes which occur in a living tissue when it is injured, provided the injury is not of such a degree as at once to destroy its structure and vitality," it may be objected that this formula, though applicable to a wound in its successive stages, from its infliction to the completion of its repair by primary union, does not seem applicable, without some modification or straining of its terms, to the condition of a wound in which traumatic inflammation has supervened. For traumatic inflammation is not pre-

* Professor Burdon Sanderson takes exception to the statements of Bowman and Goodsir quoted above, observing that "we find, when we actually watch the effects of an injury on a living part, that the old order changes but does not give place to new—that we have before us not disorder of function but arrest."—('Brit. Med. Journ.,' 1882, i, p. 411). Disorder has not, however, led to arrest of *nutrition*, while vitality remains in the damaged structure.

† 'Brit. Med. Journ.,' 1884, ii, p. 647.

sented clinically as a necessary and direct result of an injury, but as an exceptional complication of its effect, from unfavorable conditions adversely affecting the nutrition of the injured part; a complication causing disturbance and subversion of those remedial and reparative processes, which constitute the succession of changes naturally and necessarily occurring in injured parts under ordinary circumstances. And to the statement that the essential nature of inflammation is fully expressed in the word damage, it must be objected, that the term, as primarily applied by the older authorities to the spreading local phlegmon, with systemic disturbance, of traumatic inflammation, was employed to designate a very definite pathological condition, presenting striking clinical features, and having a grave prognostic significance—very different from the fresh state of a wound subsequently healed by primary union, which involved only a greater or less liability to the supervention of traumatic inflammation, with the condition of danger attending it,—and that to apply this time-honoured term, so forcible and expressive of the danger involved, when used as the clinical designation of traumatic inflammation, to a condition so widely different, is to deprive it of all force and clinical significance. Used in a wide and indefinite sense, as a mere synonym of damage, the term is rendered redundant and valueless.

Such a conclusion is, I think, sufficient evidence of the erroneousness of the premises from which it is the logical deduction, *i. e.* of the acceptation of the traditional clinical definition of inflammation. For no objection can be made to Burdon Sanderson's position, when he says that, "if we confine the word inflammation within the

limits of the original definition, and accept as its phenomena those which have been assigned to it by the authority of tradition (which is the only authority which has any claim to be heard in such a question), its essential nature is fully expressed in the word damage;" * since modern research has shown that there is, in fact, no essential difference between the vascular and textural changes, to which the redness, heat and swelling of traumatic inflammation, or of other spreading phlegmon, are due, and those changes which are covered by the blush of congestion along the margins of a recent or healing wound.

It may be pointed out, however, that the voice of tradition here appealed to as the ultimate authority on the question, gives a twofold utterance in respect to it, and that very different conclusions are arrived at, according as the one or the other is listened to.

It seems to me quite clear, that a mistake has been made in following a defective clinical definition of inflammation, as the basis of our views on the subject, instead of holding to the traditional usage of the term as primarily applied to traumatic inflammation, restricting its application only, by excluding conditions improperly regarded as inflammatory, from a superficial resemblance to that condition.

Hunter, adopting the traditional definition, was led to regard as inflammatory the great congestion and tumefaction, which he observed to occur in the ear of a rabbit, and in the comb of a cock, when thawed after being completely frozen; and he would have so regarded the similar phenomena observed in Cohnheim's well-

* 'Brit. Med. Journ.,' 1882, i, p. 411.

known experiments, when the blood was allowed to flow again into the vessels of a rabbit's ear, from which it had been excluded for several (24—48) hours, or for a brief period only while the anæmiated tissue was put into hot water. But a reference to his works shows, that this definition was an inadequate expression of the clinical characters of inflammation, as he himself recognised them.

Taking a different, but I think a truer view of the nature of this condition, than those modern pathologists who regard it as essentially a condition of damage, Hunter, in his great work, 'On the Blood, Inflammation, and Gunshot Injuries,'* observes that "inflammation is not to be considered as a disease, but as a salutary operation consequent either to some violence or to some disease," "an action produced for the restoration" of injury, and "an effect intended to bring about a reinstatement of the part, nearly to its original function." But he also clearly notes a destructive and indefinitely spreading, or as we now say infecting, morbid process as affecting the tissues in inflammation, observing that, "in one point of view it may be considered as a disease in itself," and "may be looked upon as an increase of the mischief, when it is a consequence of some injury;" and further on he says,† that "inflammation, in most cases, appears to begin at a point; at the very first commencement all the local symptoms are within a very small compass, and they afterwards spread according to the violence of the cause;" and "this is the case with those inflammations which arise from accident, for all accidents are confined to fixed and determined limits, but the inflammation which follows them is not;" and Hunter

* Ed., 1828, p. 314, *et seq.* † Ibid., p. 350.

notes also the incubative period of the morbid process by which traumatic inflammation is brought about; observing that "after a wound has been received, inflammation does not begin for twelve, eighteen, or twenty-four hours."*

By these clinical features traumatic inflammation was shown to be indicative of the development of a fermentative process, of an injurious and destructive nature, in the wound, subsequently to its infliction. And by these features it was distinguishable from the more immediate and direct effects of all injuries of whatever kind. In adopting a definition of inflammation in which its objective clinical phenomena alone were taken into account, Hunter failed to sufficiently recognise the great difference in the significance of these phenomena, according as they are localised or spreading, and according as they appear as the direct effect of an injury, or a day or two after its infliction. And I think that an explanation of this is to be found in the fact that, in the great work to which I have alluded, he strongly opposes, what he speaks of as "an old opinion, and still the opinion of many," "that the living solids of an animal body are dissolved into pus," and "that the pus already formed has the power of continuing the dissolution,"† by what was at that time supposed to be a fermentative process.

Hunter had no difficulty in proving that pus was not produced by a solution of the tissues. As he cogently remarks, "the whole penis could not afford matter enough to form the pus which is discharged in a common gonorrhœa."‡ And it is, I think, an interesting fact that Hunter, in accordance with what has been demonstrated in more recent times, regarded pus as an

* Ibid., p. 383. † Ibid., p. 523. ‡ Ibid., p. 528.

altered exudation from the blood-vessels, "by a new mode of action," as he expresses it, "consequent upon a changed structure or disposition," which he called glandular, pus being the "secretion."*

But in denying the corroding power of pus upon the tissues, we now know that he was wrong. He seems to have been misled in this matter, partly by the association in his mind of this question as to the qualities of pus, with the opinion as to its origin which he saw to be erroneous; partly by a misapprehension of certain clinical and experimental results; but, perhaps, chiefly by an unwarranted inference from physiological considerations, as when he says that "it must appear inconsistent, that the matter, which was probably intended for salutary purposes, should be a means of destroying the very parts which produced it, and which it was meant to heal."†

With the knowledge we now possess as to the nature of pus, we see how this apparent inconsistency is explicable. While recognising the formation of pus as an incident of a natural remedial process, as when a dead part has to be thrown off, we recognise also both the fact and the cause of its destructive quality, this being due to the fact of its abounding in actively multiplying micrococci. To Hunter, having no knowledge of micrococci, and no conception of an actively destructive morbific process occurring in the tissues of a living animal by the invasion of them by such foreign organisms, inflammation appears to have been presented as an effect of injury, or of defective or ill-regulated remedial processes, and its processes he appears to have

* Ibid., p. 523. † Loc. cit., p. 523.

regarded as essentially salutary in their nature though inefficient and injurious in their inefficiency. And such a supposition would have been just, had they been the effects of injury alone. We now know, however, that when the local appearances and systemic disturbance indicate that a wound has become inflamed, there is a collection of serum between the wounded surfaces, which has become impregnated to a degree injurious to the damaged tissues, by a poisonous product of micrococci actively developing in it, or of a fermentative process set up in the organic fluid by them. And from the extension of the local symptoms, we know that there is a progressive invasion and destruction of the tissues by the morbid process. And on this spreading or infecting quality of the morbid lesion depends that "character of danger" (Virchow), which is distinctive of inflammation as here typified, and gives it its grave clinical significance.

Had Hunter appreciated the fact that the appearance of a spreading lesion about a wound, at an interval after its infliction, is indicative of the development of a destructive fermentative process referable to the multiplication of foreign organisms in it, he would not, I think, have been led by a superficial resemblance, to regard any direct and immediate effect of injury as coming under the same denomination with traumatic inflammation. Still less do I think he would have continued to follow the traditional definition of inflammation, in the face of the logical conclusion of modern pathologists, that the term so defined is applicable to conditions so different as those of a freshly-inflicted wound, and of one in which traumatic inflammation has supervened.

For a definition of inflammation according with the

clinical facts, and clearly distinguishing this grave complication from other conditions of damage, may be constructed by a combination of some of Hunter's statements quoted above, and nearly in his own words. Inflammation may thus be defined as "a salutary operation consequent either to some violence or to some disease," where "the local symptoms have afterwards spread beyond the limits of the injury," with "an increase of the mischief;" "an effect intended to bring about a reinstatement of the parts nearly to their original function." Or the same thing may be expressed in other words, as the recuperative and remedial action of the organism directed to arrest the advance of an infecting (fermentative) morbid process of destructive tendency, developed in the tissue consecutively to damage from injury or other cause; or more precisely, and I think equally well, as the state of a part, in which a condition of damage has become complicated by the development in the damaged tissue of an infecting morbid process of destructive and irritant character, and which is the seat of a recuperative affluxus of blood determined thereby.

For, as I shall endeavour to explain in more detail, this afflux of blood is the efficient agent by which, in all degrees of the injurious and destructive effect of the morbid process, and in the different stages or forms of the inflammatory action determined by it, the infective process (or its infecting power) is arrested; and by which the way is thus opened for the advance of the processes of repair, the initial steps of which its occurrence had disturbed.

The justness of Hunter's conception of inflammation, as primarily and essentially a salutary and remedial action

of the organism, and the distinction to be drawn between the condition of damage and destruction caused by the morbid process, and this condition of reaction, in which the destructive and recuperative processes are in conflict, are clinically conspicuous in the progressive destruction of the tissues, unchecked by any reaction in the part, which is observed to occur after slight injuries in conditions of debility and exhaustion, and especially where innervation of the part is impaired, either peripherally or through the nerve-centres.

A definition of inflammation such as that above suggested, appears to me to accord with, and to adequately express the clinical facts, and to truly indicate the essential nature of the condition. On the other hand, those direct effects of severe and painful injuries, which present the same objective signs and textural changes as infecting inflammatory lesions, and which have been designated by Burdon Sanderson as *normal inflammation* (in which the exudation has no infective quality, and which does not tend to spread beyond the limits of the injury of which it is the effect), are, I think, more properly described as conditions of uncomplicated (non-infecting) *damage with irritation*.

Cohnheim's experiments, thus viewed, can only be regarded as exhibiting, in a very striking manner, the structural changes resulting from severe injury with irritation to healthy tissues, and as clearly demonstrating that the vascular relaxation, and the surcharging of the interstices of the tissue with fluid and corpuscular exudation, there seen, are phenomena in no way characteristic of inflammation, but are necessarily incident to that condition from its occurrence as a complication of the

effects of injury to a part, attended with a high degree of irritation to it.

A condition of inflammation, as above defined, being one of damage with irritation, necessarily presents the same objective features as uncomplicated lesions of that nature. And the significance of the combination of phenomena is the same in both cases, viz. that the vaso-motor organisation of the part is *sensitive* to the action of the noxious agency, and that a recuperative affluxus of blood to the part has been brought about through its mechanism.

Only by the renovating effect of an afflux of blood, increased to a degree proportional to the injurious effect of the poisonous product of the morbid process, can that efficient state of nutrition in the infected tissues be maintained, by which, as we know, the continuance of the morbid process in them is prevented. Only thus, indeed, could the condition admit of remedy, since no living organism could maintain its existence unless able, when in a healthy state, to resist the infective morbid processes by which its tissues are liable to be invaded.

In the slightest grades of the infecting lesion, where a condition of damage only has been produced, the recuperative action of the inflammatory affluxus may alone suffice to arrest its advance, and cause it to come to an end by restoring the nutritive state of the whole of the tissue affected.

Where the injurious effect has been too great to admit of restoration by resolution of the vascular and textural phenomena of damage, through the renovating action of the affluxus alone—where the tissue has become in part necrotic—it is still the same remedial agency by which

relief is given to the part in the secondary, or second and third, methods provided for in the organism, by which the phlogogenic process may be ultimately arrested and the way opened for the processes of repair.

Considering, in the first place, (1) the engorgement of the interstices of the damaged and irritated tissue by the exudation from the congested vessels of the part—impeded in its escape, by the dilatation of the lymphatic channels, and by their obstruction by coagulation of the fibrin of the exuded plasma in them—and the *strain* on the framework of the tissue, by the transmitted mechanical force of the increased blood-pressure of the irritative inflammatory affluxus, thus brought to bear upon it—the immediate and direct effect of which is exhibited in the separation of the epidermis, and the formation of a tense blister, by the effect of the considerable irritation attendant upon the trivial injury caused by a slight burn :—and considering, in the second place, (2) the *softening* of the fibrinous coagulum of the exuded plasma, and of the framework of the tissue at the borders of the necrotic area, by the effect of the absorbing, quasi-digestive, action of the living elements on dead structure with which they are in contact, and especially by the absorbing action of embryonic granulation tissue, developed from the connective tissue of the living structures contiguous to the dead—the formation, and the eroding action of which are probably necessary for the separation of the connections of a mass of dead tissue in an inflamed part, as under other conditions (as where a superficial slough or eschar is thrown off, or where a dead portion of a limb is separated by a line of demarcation) ; and the formation of a delimiting wall of which

tissue probably constitutes the essential feature in the process of "maturation" of an abscess:—considering thus the softening of the borders of the necrotic area, and the strain upon the tissue from the vascular congestion, there seems little difficulty in understanding both the formation of an abscess, by the free disruption of its weakened framework and by the disgorgement into the comparatively free space thus afforded of the contents of the interstices of the tissue around, and the subsequent advance of the abscess in the direction of least resistance,* and thus ultimately to the surface of the body (external or internal), under the strain of the same force, by continued exudation into its cavity from the congested vessels in its walls, when a temporary relief only has been afforded by its formation.

We can understand, however, that in the improved nutritive state of the tissue, obtained by the discharge, (with such great relief to the patient), of the purulent contents of its surcharged interstices impregnated with the poisonous products of the mycotic growth, the depurating and renovating action of the inflammatory affluxus to it, though previously ineffectual, might then suffice to restore its efficient nutritive state, and thus arrest the advance of the morbid process, and cause it to come to an end;

* From protrusion of less supported parts of the abscess wall, and destruction of adjacent structures through interference with their nutrition by the bulging mass; probably with invasion of them by micrococci and necrosis, as is seen when the skin or mucous membrane (or serous membrane, if no adhesions have been formed) is protruded, or compressed against some resistant structure, by the increasing fluid mass, and the blood supply to the tissue over its projecting or compressing point cut off: the advance of suppuration being slow through fibrous fasciæ, (?) impermeable by micrococci, and nourished by the tissues they protect.

as we know may happen under favorable circumstances. And such a result must be greatly aided by the formation of a wall of granulation tissue round the cavity; a wall of highly vascular tissue of simple structure, and, probably on both accounts, more resistant to the infective process than the more highly developed structure of the part.

The fact that inflammation may cease after the formation of an abscess, and that repair may be brought about with complete absorption of its contents, or with the encapsulation of a caseous or cretaceous residuum, prove that the recuperative effect of the inflammatory affluxus, aided by the formation of a wall of granulation tissue round the cavity, may suffice under favorable circumstances, and it probably does most commonly suffice, to definitely arrest the extension of the infective process, and cause it to come to an end by thus effectually circumscribing it.

When this does not happen, relief is obtained by advance of the abscess to the surface; the further progress of the infecting lesion being arrested, when unimpeded exudation from the walls of the cavity is permitted, by opening a passage for the free discharge of its contents.

Such seems to me to be the significance of the occurrence of suppuration and abscess formation; such the means, mechanical and vital, by which the formation of an abscess is brought about; and such also the manner in which relief is afforded to the inflamed part by its occurrence and subsequent progress.

And in these facts, thus regarded, three things appear to me to be especially noteworthy.

1. That in these phenomena we may trace through the different stages of the inflammatory process, the remedial action of the same recuperative agency, brought into operation through the nutritive vascular mechanism, —*i.e.* the inflammatory or irritative affluxus—aided by the conservative vital processes of the part, and, by their co-operation, rendered effective for the relief of the affected part by methods adapted to the degree of injury sustained.

2. That, recognising the softening and the disruption of the tissue of an inflamed part, as effects of physiological processes and agencies, similar to those produced by them under other conditions, the pnenomena of abscess formation,—being then recognised as essentially those of conditions of damage with irritation, complicated with destruction of the tissue,—cannot be looked upon as in their nature distinctive of inflammation, any more than its initial phenomena of damage and irritation. That the formation of an abscess, though clinically distinctive of inflammation, by its spreading character, and owing to the fact that such a combination of destruction and intense irritation of the deeper structures is observed to occur in conditions of phlogogenic infection alone, is, pathologically considered, an incident only of the inflammatory process,—an alteration in its course, (*i.e.* in the character of the remedial processes brought into action), in adaptation to circumstances,—but no indication of the introduction of any new morbific agent, or of any essential change in the nature of the condition.

3. That in the phenomena of abscess formation we see the initial step of the secondary process of repair, by the formation of a covering of granulation tissue to the surface of the living parts, carried out in inflamed parts,

concurrently with, and in a manner to be subservient to, the recuperative reaction of the organism directed against the destructive process affecting it, the arrest of which is a necessary preliminary to the advance of repair.

Quite distinct from the consideration of the physiological action of the inflammatory affluxus, is that of the mechanism by which it is brought about. Those who with Hunter regard inflammation as essentially a remedial and salutary process, will be disposed to give especial weight to those facts which indicate the reflex character of this vascular congestion. I think, however, that there are objections to that view of this phenomenon, in which it is regarded as a local action of the vessels, the condition *sine quâ non* for the production of which is not innervation, but simply muscular excitability and contractility of them.

Independently of the difficulty of conceiving how such a co-ordinated vascular action could be brought about without the agency of vasomotor nerve-centres, central or peripheral, it seems highly improbable that a complex mechanism, by which an afflux of blood to a part is observed to be brought about in the presence of a trivial disturbance of it, should not be made available in bringing about a similar affluxus, directed to sustain the nutrition of the part in the presence of serious danger, urgently demanding its recuperative support.

It appears to me also that the clinical and experimental facts, adduced by Burdon Sanderson in support of this view, are inconclusive on this account; that the division of the nerves of a part, or the division of all its structures but the principal vessels, cannot be said to cut off its

vasomotor connections, until it is shown that there are no centripetal fibres connected with vasomotor centres, mingled in the coats of the vessels with the motor fibres, by which the contraction of their muscular walls is regulated.

Passing now from the consideration of the definition of inflammation, to that of the etiology of the condition as here understood, the most essential question regarding this latter point, is that as to the nature of the infecting agent, or agents, concerned in its occurrence.

Nor do the results of clinical observation and experiment seem to leave any doubt, that the practical answer to be given to this question, is that embodied in the third proposition discussed by Burdon Sanderson in his second Lumleian Lecture, that "whenever an inflammation becomes infective, it owes that property to chemical change in the exudation liquid, of which the presence of microzymes is a necessary condition."

That is to say, that the infective agents in ordinary (non-specific) inflammation are micrococci, present as such, or in the form of germs or spores, in the atmospheric dust and in fluids contaminated by it; since it is organisms of this type only that have been found constantly associated with inflammatory lesions, and since such generally diffused organisms can alone have any causative relation to ordinary inflammation, to which all wounds are more or less liable.

This atmospheric germ theory of the origin of inflammation is that obviously indicated, (1) by the dependence of the liability of injured parts to become inflamed upon contact of the damaged tissues with the atmospheric

dust; (2) by the presence of these organisms in the discharge from wounds which have become inflamed; and (3) by the success of Lister's antiseptic method, which has been recognised as the most brilliant achievement of modern surgery.

And this view is fully confirmed by the very convincing clinical and experimental evidence, (1) of the phlogogenic properties of these organisms when in active growth, and (2) of the accessibility of the deeper tissues to them, and of the constant association of their presence with suppuration.

On the former point sufficient evidence is afforded, (1) by the results of inoculation experiments on animals; such especially as those of Professor Ogston, in which abscesses were produced by the subcutaneous injection of small quantities of pus from acute abscesses, and of albumen from eggs, in which a pure cultivation of micrococci from a similar source had been obtained by the ingenious method devised by him; (2) by the results of Chauveau's experiments on animals subjected to the operation of bistournage of the testes, an operation employed in France to cause atrophy of these organs through occlusion of the spermatic artery by torsion of the cord, and effected without wounding the skin. In these experiments he found that the intravenous injection, shortly before operating, of a sufficient quantity of the fluid part of diluted pus from an acute abscess, in which there were no solid particles to obstruct the vessels, but abundant micrococci, caused suppuration in the scrotum; while no effect was observed to follow the injection of an equal quantity of the same fluid after the organisms had been removed by filtration through porcelain; results

which proved that the occurrence of suppuration in the former case depended upon the presence of the micro-organisms in the fluid injected, and was not due merely to septic matters held in solution by it. The same thing is shown (3) by the results of Wegner's experiments, in which he showed that the injection of distilled water into the peritoneal cavity of an animal, if in such amount as to remain in part unabsorbed after a certain time, induced rapidly fatal peritonitis by the development in the fluid of micrococci, derived from atmospheric contamination of the water, or absorbed from the intestinal tract. That the development of these organisms was the immediate cause of the peritonitis, is proved by the results of his previous experiments, which showed that the peritoneum is not appreciably injured by more prolonged contact with water, if sufficiently renewed.

That the same phlogogenic organisms have access to tissues unexposed to the air, either indirectly through the blood or by channels which elude observation, and that they are constantly associated with suppurative inflammation, in these as in exposed tissues, is proved by the results of Professor Ogston's extended investigations, in which he showed that micrococci are invariably to be found in the pus obtained from abscesses when first opened, in their acuter stages. Although from the partly negative results of other observers, there might have appeared to be some uncertainty as to the result of Ogston's careful and elaborate observations, it seemed most probable that this discrepancy was due in part to less reliable methods of investigation, and in part to the quiescent character of some of the abscesses examined by the less successful observers. And this has been

pointed out in a recent publication by Mr. Watson Cheyne, whose later observations appear to have fully confirmed Professor Ogston's results.*

They are confirmed also by the results of the subcutaneous injection in animals of irritating fluids, destructive to micro-organisms, or sterilised and employed with antiseptic precautions, which was found, in experiments both of Burdon Sanderson† and Watson Cheyne,‡ to cause suppuration with abundant development of micrococci.

In view of these results of Ogston's observation, the question as to whether these atmospheric organisms, or their inert germs or spores, are or are not present in the tissues of healthy animals, appears to be of secondary importance from a practical point of view. If it were proved that they are absent from the tissues in health,

* 'Brit. Med. Journal,' 1884, ii, p. 553.

† 'Medico-Chirurg. Soc. Trans.,' vol. lvi, p. 366.

‡ Mr. Watson Cheyne found abundant micrococci in the pus infiltrating the dorsal muscles of a mouse produced by injecting half a minim of croton oil twenty-six hours previously. The efficiency of the antiseptic precautions and the sterility of the croton oil being tested by a control experiment, there would appear to have been here no possibility of contamination of the tissues by micrococci directly introduced from the air. Mr. Watson Cheyne, however, observes, in reference to this and some other experiments, that they are open to great objection, and that "it may be rightly said that the micro-organisms crept in along the track of the needle, and, on reaching the abscess, developed in the pus;" but considering that the skin of the mouse was carefully purified beforehand, and that the puncture was dried with a carbolised rag and sealed with collodion, and considering also that these organisms, though carried in the fluids of the body, do not appear to have much power of spontaneous locomotion, it seems scarcely conceivable that they should have made their way along the needle track—discontinuous in the subcutaneous connective tissue—into the dorsal muscles.—('Brit. Med. Journ.,' 1884, ii, p. 645.)

the conclusion would be that where an abscess has formed, there must either have been direct inoculation of the tissues by some undiscovered channel, or a disturbed condition of the general nutrition, having the effect of facilitating the absorption of the micrococci, or more probably, perhaps, of enabling them to survive after absorption, long enough to reach the seat of damage, before perishing in the unfavorable medium presented to them in the blood. For it seems difficult to imagine that such minute bodies, or possibly far more minute germs or spores, introduced with floating particles of dust into the respiratory and digestive passages, could fail to be taken up into the lymphatic vessel, through which comparatively coarse particles are carried to the glands.

Only by such an effect of a disturbance of the general nutrition from the effect of a local injury, or by the presence of these organisms in the tissues, can the exceptional occurrence of spreading suppuration about the seat of simple fractures in previously robust and healthy men be accounted for : and within twelve months it has occurred to me to make autopsies on two such cases.

Such occurrences, and the presence of micrococci in great abundance twenty-six hours after the injection of croton oil under efficient and "controlled" antiseptic precautions by Watson Cheyne, appear to me to afford a strong presumption in favour of the view that these organisms are constantly present in the blood, though necessarily in a state of very feeble vitality, and doubtless quickly perishing, like the filaria shown to us a year or two ago by my friend Dr. Stephen Mackenzie, in the blood of one of his patients.

On the other hand, the negative results of Watson Cheyne's experiments by incubating portions of the organs of recently killed animals in an infusion of cucumber, which perhaps, more than any other experimental results, have appeared to afford decisive evidence of the freedom of these tissues from mycotic contamination, seem to me inconclusive; because a want of delicacy in this test of the presence of micrococci in pus from an abscess, appears to have accounted for the disagreement of Watson Cheyne's earlier observations on that subject with those of Ogston; and also because in the inference drawn from these experiments, no account is taken of the state of greatly enfeebled vitality, in which any organisms in the blood must be, or of the powerfully antimycotic quality of the tissues and their juices.

In view of such facts as those above referred to it is, I think, impossible to look upon the presence of these micrococci in abscesses as an incident without pathological significance, as has been done by some pathologists.

The numerous clinical and experimental facts which show that these organisms, as they exist in the atmosphere and in ordinary liquids exposed to it, may be brought into contact with the healthy and uninjured tissues without injurious effect, afford no support to such a view. For the uninterrupted healing up of "unprotected" wounds, proves that these organisms are innocuous to *damaged* tissues, under conditions favorable to repair; and the result of Ogston's experiments, showing that a sufficiently small dose of virulently infective pus may be injected into animals without injury, proves that these organisms are not of themselves injurious to the tissues, even when in a most active state of pathogenic

growth in a virulently infective fluid. This being so, the result of Wegner's experiments, showing that no harm is done by the inflation of the subcutaneous connective tissue or of the peritoneal cavity of a rabbit with unfiltered air, or by the injection of a limited quantity of water or of a saline solution into the same cavity, or by the irrigation of it by a stream of water during several hours, and the no less remarkable result of an experiment of Hunter's, who produced a pneumothorax in a dog by an incision between the ribs and, on the death of the animal eleven days afterwards, found the pleural cavity filled with air but free from inflammation, prove only that the tissues of an animal may be subjected to what seems rather severe treatment, without suffering any appreciable injury. But such facts have no especial significance in relation to the potential phlogogenic properties of atmospheric micrococci. So long as no damage was done to the tissues no inflammation could occur.

Wegner's further experiments, by the injection of a larger quantity of distilled water into the peritoneal cavity of a rabbit, not only afford conclusive evidence of the virulent phlogogenic properties of these organisms, but in them we may also clearly see the conditions under which their growth became infecting, *i.e.* the cause of the very different effects of the injection of the smaller and of the larger quantities of water, and the point at which the balance turned. From the contact of the water injected into the peritoneal cavity with that membrane, there will at once have resulted (1) a more or less rapid absorption of the water into the denser fluid occupying the interstices of the membrane, and into the blood traversing its vessels, and (2) a slower passage

outwards into the peritoneal cavity of the fluids of the tissue, probably with some leucocytes and detached endothelial cells. By the continuance of the latter process conditions more and more favorable to the active development of the septic organism would be presented, and the toxic product of their growth would be generated in the fluid in a correspondingly increased amount, and would continually accumulate in it. Up to a certain point of concentration, there would be no important injurious effect produced upon the vitality of the serous membrane; and so long as there were no structural changes in it impeding the circulation through its vessels, absorption would continue to preponderate over exudation; and if the amount of fluid injected was not more than could be absorbed before such structural changes were brought about, no harm would result to the serous membrane. If, however, some portion of the infective fluid should remain, when the nutritive state and structure of the membrane has become so far disturbed as to impede absorption, and cause the preponderance of exudation from its surface, conditions would be presented most favorable to the luxuriant growth of the micrococci, and to the concentration of the poisonous matters generated by them. The fluid stagnating in the serous sac would thus quickly acquire a virulently septic and infecting character, according with the local and systemic effects observed to occur.

These facts exhibit the capability of the tissues of healthy animals, to prevent the development of the phlogogenic micrococci invading them, and to resist the injurious action of the poisonous product of their growth. Not until the activity of the mycotic growth, and the

concentration of the septic poison in the fluid stagnating in the peritoneal cavity, had reached a degree of intensity such as to be quickly fatal to the animal, was its injurious action sufficient to cause damage to the membrane. Only when conditions especially favorable to them are presented, can the atmospheric micrococci develop with phlogogenic activity in wounds.*

It does not appear to me, however, that any such wide distinction can be drawn, on this account, between these atmospheric micrococci, with potential phlogogenic properties, and other "specific" pathogenic organisms, which may, from time to time, be present in the air, or in fluids with which we are brought into contact, as to warrant Burdon Sanderson's statement that "these organisms as they exist in the atmosphere cannot be regarded as possessing phlogogenic properties," and that "the hypothesis that the atmosphere is charged with phlogogenic particles must be unhesitatingly rejected."† For it may

* The resistance of the tissues to the growth of these phlogogenic micrococci, and the dependence of their development, in tissues inoculated with them in a virulent form, upon the degree and extent of the interference with the nutrition of the part, were, I think, strikingly illustrated in a case of tropical dysentery, on which I made an autopsy some time ago, and in which there were two localised abscesses of older date in the liver, with numerous recent embolic abscesses studding the surface and substance of its right lobe only; the immunity of the left lobe being apparently accounted for by the fact that a large abscess in the situation of the quadrate lobe, projecting downwards and forwards, had drawn forward the point of attachment of the cystic artery with the right branch of the hepatic artery, in such a way that this vessel was bent at a right angle at that point, impeding the supply of arterial blood to the right lobe of the liver, and affecting most prejudicially the nutrition of parts in which portal venules had become plugged.

† Holmes' 'System of Surgery,' 1883, vol. i, p. 92.

be doubted whether any pathogenic organisms exist to which resistance is not presented by the tissues in greater or less degree—whether any contagia are capable of causing pathological lesions, unless a defective nutritive state of some part affords them the exceptional conditions necessary for their multiplication to a degree injurious to the organism. Only on such a supposition would it be possible that any unprotected individual should pass through an epidemic of an infectious disease without being attacked by it.

The latent period of incubation in the primary local lesions of certain infectious diseases after inoculation, as in the case of traumatic inflammation, shows that the specific germs of such diseases, like the micrococci, are innocuous as at first introduced, and become injurious only by active development inside the body; their infectiousness depending upon the comparative feebleness of the resistance of the tissues to their growth.

I think, therefore, that if any specific organism of infectious diseases in the air, or in water or other fluids, may be spoken of as pathogenic, these atmospheric micrococci (or germs) may be spoken of as infective phlogogenic organisms with equal propriety.

The comparatively great resistance of the tissues to these generally diffused micro-organisms, is a provision manifestly necessary to the existence of individuals continually subject to invasion by them.

It appears to me then, that, while the clinical facts present inflammation, as, on the one hand, a condition of *indefinitely progressive* damage caused by an *infecting* morbific process of destructive nature, and on the other, as a *remedial process* directed to arrest the advance of the

morbid lesion, by an irritative affluxus of blood determined to the part, through the sensitiveness of the vaso-motor organisation to the injurious effect of the infective agents, the results of experimental and clinical research clearly establish the practical conclusion, that these infective agents are phlogogenic micrococci in the dust of the atmosphere.

The facts do not prove that no inflammatory lesions originate independently of micrococci, or other pathogenic organisms. It may be observed, however, that such a conclusion can alone be regarded as according with the conservative principle of physiological laws; since the supposition that such an infecting lesion might originate in the tissues independently of foreign organisms, involves the assumption that a fermentative process *directly* destructive to the tissues of the body, might result from a perversion of the physiological functions of the elements of the tissue itself.*

* Mr. Watson Cheyne, in his investigation on "Micrococci in relation to Wounds, Abscesses, and Septic Processes" ('Brit. Med. Journ.,' 1884, ii, p. 645), describes two experiments in which he produced suppuration in the lumbar muscles of rabbits by rupturing subcutaneously glass tubes containing one minim of croton oil diluted with one minim of olive oil, introduced several weeks previously with antiseptic precautions; and he adduces the negative results of his microscopical examination for micro-organisms in the cheesy pus obtained from these abscesses, in one case twenty-seven, and in the other twelve days afterwards, as evidence of the occurrence of suppuration independently of their presence, under such circumstances.

That severe injury, with intense irritation of the tissues, should result in the formation of a cavity with purulent contents, quite independently of micro-organisms, seems possible; but no proof of this is afforded by the negative results of an examination of the pus after the subsidence of all irritation, which would of itself be proof of the absence of micrococci, not only from tissue around the cavity (*i.e.* inflammation), but also from

The conclusion here arrived at, that the atmospheric micrococci are the infective agents concerned in the occurrence of inflammation, is in accordance with the views enunciated by Heuter. But all unprotected wounds being inoculated with atmospheric micrococci, and all the tissues being liable to invasion by them, one cannot say with him, that the *immigration* of viable organisms of this kind into the tissues is the one true cause of inflammation. The presence of these organisms in the tissues can only be spoken of as the cause of inflammation, in the especial sense in which gravity may be said to be the cause of the fall of a body whose equilibrium has been disturbed. But just as the proper determining cause of such an accident is the agency by which the state of equilibrium is disturbed, so those local influences, by which the nutritive equilibrium of the tissue is disturbed, and by which a condition of the damaged part affording the requisite nidus for the phlogogenic growth of the micrococci invading it, is ultimately or mainly brought about, are the proper determining causes of the inflammation resulting from the mycotic growth.

In wounds these conditions, in general, are afforded in the collection of exuded serum between the surfaces. And amongst the causes of traumatic inflammation may

its contents, where their presence would still cause septic irritation. This was demonstrated by Ogston in his very complete research, in which he showed that the number of micrococci in the pus in abscesses progressively decreases after the acme of the process, and that they are absent in conditions of quiescence.

These observations of Watson Cheyne's, therefore, afford no proof of the occurrence of suppuration in the absence of micro-organisms, and no evidence whatever of the occurrence of an infecting morbid lesion in the tissues (*i.e.* of a phlogogenic lesion as here understood) independently of them.

thus be mentioned, ill-adaptation of the surfaces of the wound, or separation of them by movements of the parts or by muscular contraction in them, or by too great strain upon the temporary uniting medium from congestion, active or passive, whether from an irritant in the wound or from pressure upon the veins.

But the occurrence of inflammation being the result of the combined action of two factors of causation—the potential power of the immediate and efficient agent, ever at hand, being rendered effective by special accidental circumstances affecting the tissue—there will, with a variation in the former factor, be a corresponding variation, in an inverse sense, of the degree of the latter factor requisite to bring about the result.

But when it is considered that the infective particles of atmospheric dust are dried fragments of partially decomposed organic matter, containing, therefore, clusters of micrococci (or of their germs) or portions of masses of them in zoogloœa, and impregnated with the poisonous product of the fermentative action and growth of these or of other micro-organisms, it will be seen that in all wounds, and in all tissues contaminated by phlogogenic atmospheric particles, (1) the damage sustained is in excess of the direct effect of the injury inflicted, and that (2) the micrococci are present in a form very favorable to their active growth, wherever the resistance of the tissues and fluids to them is weakened. It will be seen, on the other hand, that, the blood being necessarily the medium most unfavorable to the life of all pathogenic organisms, the micrococci can only reach damaged tissues through this channel, in a condition of enfeebled or suspended vitality—perhaps in the form of isolated

germs only—and unattended by their septic products, which when absorbed will be forthwith diffused through the blood.

And considering the great resistance of the tissues to direct atmospheric contamination, and to more virulent forms of inoculation, it might be anticipated that micrococci reaching the tissues indirectly through the blood, should be able to develop with phlogogenic activity only under most exceptional conditions of extensive damage and destruction of the tissue from severe and repeated injury. And it is in this way, I think, rather than in a supposed freedom of the tissues from the presence of these organisms, that the remarkable immunity of simple fractures and of injuries to the subcutaneous tissues from inflammatory complications, is to be explained. And the explanation of the exceptional occurrence of suppuration about simple fractures in robust and healthy subjects, is, I think, to be found in the especially favorable conditions for the development of the micrococci presented in such cases, rather than in an effect of the local lesion on the general nutritive state permitting their entrance into the body.

That the occasional occurrence of suppuration about simple fractures is due to the severity and repetition of the injury inflicted, and not to any mycotic infection of the damaged part of a degree at all comparable with that of compound fractures by particles of dust, was shown in a remarkable case on which I made an autopsy about twelve months ago. The case was that of a robust and healthy man, aged twenty-five, who was admitted into the London Hospital in an unconscious state from concussion of the brain, received with other

severe injuries in falling from the bulwark of a ship into a lighter alongside of her. The patient remained unconscious up to within a short time of his death, which occurred on the sixth day after the accident; but he was very restless all the time, continually rolling about in his bed, and at first he was noisy and violent. His temperature rose to 102° in the evening of the day after his admission, and continued high, with morning remissions, up to the day of his death, when it rose to 105°. At the autopsy enclosed collections of pus, over which the pleura was smooth and uninjured, were found about fractures of the eighth, ninth, and tenth ribs on the left side near the spine. About these fractures the periosteum had been separated for a considerable distance, exposing the rough and jagged ends of the fragments. The eleventh rib had also been fractured and had caused perforation of the pleura. There was some serous exudation in the pleural cavity and much lymph over the lower part of the lung. There was also a comminuted fracture of the left ala ilii, a fracture of the left pubic bone near the symphysis, and separation of the left sacro-iliac synchondrosis, about none of which injuries was there suppuration, or any sign of inflammation present. There was also extensive bruising over the left hip.

The absence of suppuration about the severe pelvic fractures in this case, which must have been also subjected to some movement—though little in comparison with the effect of the patient's movements on the fractured ribs, the pelvis being protected by the mass of soft tissues surrounding it, and the fractured parts, where subject to displacement, not being brought back into place

by rigid connections with the framework of the skeleton —shows that there was there no condition of the blood, produced by the effect of the injury on the general nutrition, capable of accounting for the occurrence of suppuration in simple fractures, even of severe character, and even though the systemic effect of such great injuries was supplemented by the supervention of inflammatory fever. And the suppuration about the simple fractures of the ribs must be recognised as the result of the severity of the injury inflicted on the soft parts, by the repeated contusion and laceration of them by the displacements of the fractured bones, caused by the rolling of the restless patient.

Comparable with the effects of repeated disturbance of injured parts shown in this case, are the results of Chauveau's experiment on an animal subjected to the operation of bistournage of the testes, in which he produced suppuration in the scrotum by daily manipulation of the parts.*

In the case I have referred to the *infecting* nature of the lesion, which could not perhaps be certainly inferred from the formation of a collection of puriform fluid under such circumstances, was proved by the extension of the mischief over the surface of the pleura, and by the occurrence of pyrexia of a hectic type.

From a general view of the subject of inflammation, regarded from Hunter's standpoint as indicated in his works, and in the light of some of the results of more recent

* And comparable with these facts also is the origination of an infective lesion of the endocardium along the lines of impact of the valvular curtains in certain conditions of defect in the systemic nutrition.

pathological research—such a view as I have here endeavoured to sketch out—the justness of his conception of inflammation, as essentially a salutary process directed to the restoration of a part affected by a spreading or infecting morbid lesion, will, I am convinced, become more and more apparent.

By distinguishing between the morbific and remedial processes in conflict in the inflamed part, and by tracing the different physiological agencies engaged, and the effects of their combined operation, we obtain a clearer conception of the significance of the phenomena of inflammation, and of the relations which its several processes bear to each other. And by recognising this condition as a state of reaction to an infecting morbid process, due to living organisms invading and destroying the tissues, we obtain a clear conception of the position of inflammation in reference to other conditions of injury and irritation—the effects of purely physical agencies of whatever sort, with the remedial and reparatory physiological processes by which those effects are mitigated and made good.

These relations may, I think, be clearly exhibited in a tabular arrangement,* which will at the same time present a summary of the view of the facts which I have endeavoured to explain.

Such a view of the facts appears to me to afford clearer and truer ideas on this important subject, than are obtainable by accepting the traditional definition of inflammation and the conclusions deducible from it.

In conclusion I would say, that whatever is defective

* See pp. 40, 41. The arrangement has been modified and the details amplified since the delivery of the Oration.

in my discourse, whatever is erroneous or ill expressed, this I shall claim for it, that in one respect at least it is not inappropriate to this occasion, being the result of an endeavour to follow in the path of our illustrious countryman, whose name and whose great work we commemorate to-day, in searching after truth.

## DISTURBANCE OF NUTRITION. DEGREES OF INJURY.

### I. "Irritation."

Nutrition disturbed.
No derangement of the nutritive function.
*a*. No structural injury.
*b*. Superficial injury.
*(Vesication from separation of cuticle by force of affluxus. Abrasions.)*

### II. "Damage."

Derangement of nutritive function (*vascular stasis*) without necrosis.
*a*. No structural lesion.
*b*. Structural lesion.
A. Without irritation.
B. With irritation.

### III. Necrosis.

A. Without irritation.
B. With irritation.

## ASSOCIATED REMEDIAL PROCESSES AND AGENCIES.

1. *Recuperative.*
   i. *Sustaining nutrition* (renovating, depurative).
   ii. *Supporting damaged tissues.*
   iii. *Relieving injured parts* (by removal dead tissues).
2. *Reparative.*
   Union, primary or secondary. Adhesion. Encapsulation.

1 (i). Irritative affluxus of blood ($R_1$).
Antimycotic quality of the blood (P) in *infecting* morbid lesions.
2.—*b*. *Adhesions* (protective).

1 (i). Affluxus { A. Vaso-paralytic* (?) ($R_1$). B. Irritative ($R_1$).
Aided by { (1). Fibrinous coagulum ($R_2$). (2). Granulation tissue ($R_3$).
(ii). Coagulation of fibrin ($R_2$).
Granulation tissue ($R_3$).
2.—*a*. Resolution of fibrin.
*b*. Primary union.
Fibrous adhesions and thickening.

1 (iii).—*a*. *Demarcation of dead part* by wall of granulation tissue ($R_3$).
Aided by affluxus { A. Vaso-paralytic ($R_1$). B. Irritative ($R_1$).
*β*. *Removal of dead parts or tissue elements.*
A. Absorption or encystment of granulation tissue ($R_3$).
Detachment through erosion by granulation tissue (superficial slough, eschar).
B. Detachment by disruption of weakened connections of dead tissue by force of affluxus transmitted as exudation ($R_1$).
Discharge at a free surface from deep parts by continued action of the same force ($R_1$).
2. Cicatrisation, encapsulation.
*Healing by granulation.*

* Affluxus to simply damaged tissue.

## CLINICAL PHENOMENA.—REMEDIAL AGENCIES.

| NON-INFECTING LESIONS. | INFECTING LESIONS. |
|---|---|
| IFIC NTS { *Physical, unorganised.* Mechanical, thermal, chemical. | *Organisms* multiplying in the tissues and spreading through them. |
| DIAL CIES } $R_1$ $R_1$ $R_2$ $R_3$ as above. | The same with the *antimycotic quality of the blood* (**P**). |
| BID ION } *No advance* beyond limits of effects of injury. | *Indefinitely progressive* } Until arrest of *infecting* lesion in the tissues. *No advance* } |
| CESS EPAIR } *Progressive*, unless repeated or continued injury, or irritation. | |

### I. Conditions of "Irritation."—No Vascular Stasis.

| | |
|---|---|
| Swelling and congestion ($R_1$) from serous and corpuscular exudation. *Erythema with papules, vesicles, abrasions, excoriations. Catarrh.* | 1. The same with infecting character ($R_1$ **P**). *Erythematous erysipelas, exanthemata* (?). *Infective catarrh.* |
| Irritative growth of granulation tissues. Adhesions. Thickening. | 2. The same. |
| [Irritative outgrowth of tissues, hyperplastic or neoplastic]. | Irritative outgrowths of *infective* character (?) ] |

### II. Conditions of "Damage" without Death of Tissues.

| | |
|---|---|
| A. Without "Irritation." | |
| Congestion and swelling (*slight*) ($R_1$). Condensation of damaged tissue ($R_2$—$R_3$). Resolution. Primary union. | |
| B. With "Irritation." | B. Inflammation without Suppuration. |
| Great congestion and swelling ($R_1$). Central consolidation ($R_2$—$R_3$). *Thickening. Callus.* | 1. The same with progressive tendency. { i. ($R_1$ **P**). ii. ($R_2$ $R_3$). Inflammatory congestion, consolidation, and œdema. Inflammatory exudation into cavities or spaces, fibrinous or sero-fibrinous. |
| above, with permanent callus or induration. | 2. Inflammatory adhesions and thickening. |

### III. Conditions of "Damage" with Death of Tissues.

| | |
|---|---|
| A. Without "Irritation." | |
| 1.—α. Delimitation of infarct, eschar or slough ($R_3$). β. Absorption or encystment of necrosed tissue. Detachment of eschar or slough. Healing by granulation. | Progressive ulceration or sloughing from advance of mycotic growth, unchecked by recuperative affluxus. (Vaso-motor mechanism inoperative). |
| B. With "Irritation." | B. Inflammation with Suppuration. |
| | 1 (iii).—α. *Purulent infiltration, exudation.* Disruption of fibrin and of framework of tissue by force of affluxus ($R_1$). *Abscess formation.* Demarcation and separation of dead tissue ($R_1$ and $R_3$). β. *Arrest of suppuration.* Arrest of infective process (mycotic growth) in the pus ($R_1$ **P** $R_3$). *Discharge of abscess at surface.* Extension of abscess by pressure of exudation ($R_1$) |
| | 2. Absorption of pus and dead tissue, cicatrisation, adhesion. Encapsulation of a residuum. Healing by granulation. |

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