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The Nature and Frequency of Cavities: The formation of cavities in the course of tuberculosis is intimately associated with the particular degree of specific cellular reactivity which is present at the time. While both the primary lung nodule and the corresponding peribronchial and peritracheal lymph nodes of the primary complex caseate regularly, cavity-formation is not a prominent part of first infection. Neither is cavity-formation a factor of consequence in the early stage of preponderantly proliferative lesions, but it occurs commonly in the early stage of exudative lesions and regularly during the late course of both exudative and proliferative tuberculosis. It is associated with severe allergic reactions, which are apt to be much more severe in the early stages of specific defense than later.

Cavities must be recognized as undesirable complications of tuberculosis. They must also be looked upon, if not designedly, yet actually, as being at times conservative, the same as the spontaneous or surgical emptying of an abscess caused by pus-forming bacteria is conservative. As the tuberculous focus ruptures in forming a cavity it rids the lung of large numbers of tubercle bacilli and large quantities of tuberculoprotein. If the bacilli should remain in the tissues undestroyed, they might subject the patient to overwhelming metastatic infections; and, if destroyed, to dangerous allergic reactions from bacillary substances set free during their destruction.

Cavity and the Primary Complex: The primary nodule in the lung is caused by comparatively few bacilli settling in nonimmune tissues. While caseation takes place, the lesion usually remains a closed one, and open cavity rarely forms. The tissue cells have not yet been specifically trained to defend the host; so they have not been rendered hypersensitive to bacilli and bacillary protein, nor have they the property of reacting allergically. The bacilli of first infection call out only such cellular reaction as would be required to meet any minute foreign body. The inflammatory reaction which results is more or less temporary in character and of mild degree.

Many of the bacilli of first infection are engulfed by leucocytes and immediately carried to adjacent lymph nodes; others are destroyed, and still others remain in the tissues where they lodge. The bacilli, wherever they happen to be implanted, are imprisoned and walled in by epithelioid cells before the inflammatory phenomena which are so intimately connected with specific defense have made their appearance. While both the primary focus and the regional lymph node or nodes caseate they develop inflammatory characteristics slowly; and because of the absence of acute reaction do not readily form cavity. The caseous lymph nodes are also protected from the ready formation of cavity by their location and by their enveloping capsule.

Cavity and Proliferative Tuberculosis: It was stated above that cavity is an unimportant part of proliferative tuberculosis in its early stages, but that it may appear later in its course. An inquiry as to why it is absent early and why it appears later in the course of the disease might throw some light upon its cause.

Preponderantly proliferative tuberculosis seems to be free from acute and violent reaction during its early progress, the very time when the preponderantly exudative lesions are showing their most inflammatory reactions. The lesion which stimulates fibrosis most is the one accompanied by only mild irritation. It is accompanied by a minimum of exudation, and is not destructive in its action. The process, though mild in nature, is characterized by the same tendency to form metastases that is manifested by the preponderantly exudative type. The tissues are more thickly studded with infection than is the case with the preponderantly exudative lesions, in which the foci may be fewer but, as a rule, are larger and more inflammatory in nature; and where so much of the pathological process is due to the allergic reaction caused by the diffusion of toxins through the tissues (the collateral inflammation of Tendeloo) rather than to the actual foci themselves.

When proliferation predominates the infecting bacilli are few and of relatively low virulence, and the cells are not highly sensitized; therefore, the allergic phenomena are mild. One may speculate as to how much the tendency of mild proliferative lesions to spread may be due to the lack of inflammatory reaction because of the weakness of the allergic response. There is ample evidence that the allergic reaction, or any inflammation for that matter, has the property of being able to hold bacilli *in situ*, and prevent their ready dissemination.

Sooner or later, however, increased exudation with its more active inflammation may appear throughout the proliferative areas. While the course of the infection up to this time may have been afebrile and unattended by sputum, or accompanied by small amounts only, the patient may develop an elevation of temperature, accompanied by other symptoms of a toxic nature, and for the first time have a productive cough with sputum containing bacilli; or have an increase in cough and sputum if they have been previously present. The loss of tissue may be insignificant or it may reach the proportions of a clinically recognizable cavity; and the preponderantly proliferative lesion may now take on characteristics of a more acute exudative process.

The conclusion seems warranted that the failure of cavity to form early, in proliferative lesions, is partly accounted for by the fact that the process is only mildly inflammatory, and further because the numbers of bacilli causing the infection are relatively small. The bacilli neither multiply rapidly nor do they produce large quantities of tuberculoprotein, yet the lesion progresses, involving more and more lung tissue as time goes on.

Whether the lack of allergy is due to the fact that the lesion is caused by relatively few and relatively avirulent bacilli which fail to produce sensitivity, or whether it is due to a state of desensitization which represents a high degree of immunity, is not clear. In this connection we must bear in mind the experiments of Swift on rabbits, in which he reports the production of immunity to streptococci without allergy by the intravenous injection of microörganisms. We must also bear in mind that desensitization takes place in clinical tuberculosis as the disease advances. We know that tuberculosis may be attended by a bacillaemia any time after first infection has taken place.

It may be that the lack of inflammatory response is sufficient to allow the mild infection to go on spreading and producing metastases regardless of their low numbers and low virulence. Before the preponderantly proliferative processes form cavities of any considerable size, the cells may become more highly sensitized, for the exudative phenomena of allergy appear in the areas which later cavitate.

One may speculate as to why this occurs. Is it due to the accident of infection by which in the natural course of the disease a larger number of bacilli suddenly become responsible for a focus, and so set free larger quantities of reaction-producing substances? Can it be that the normally low sensitization of cells in the proliferative lesion is actually one

of a lessened specific defense, and that it is suddenly opposed by an excessive reinoculation which results in destruction? Can it be due to a sudden loss of immunity with its relative desensitization to bacilli and bacillary protein, and a restoration of the cells to a state of previous hypersensitivity in which they take on the inflammatory, exudative reaction which belongs to a more marked allergic state? Can it be due to certain changes in the physicochemical properties of the tissues which permit a mildly nonvirulent bacillus suddenly to take on properties of greater virulence and to multiply more rapidly than previously, such as might follow the transmutation of the rough bacilli, as described by Petroff, into the smooth form; or, without transmutation of types, is it possible that the smooth forms of the bacillus might suddenly dominate the process which has formerly been dominated by the nonvirulent rough form? We have long noted that when the phenomena which accompany acute exacerbations appear there is a relative increase in the numbers of short bacilli as compared with the longer forms found in the sputum the same as we note in the predominantly exudative lesions. We have made hundreds of observations in our laboratory to confirm this point.

Cavity and Exudative Tuberculosis: Preponderantly exudative tuberculosis is tuberculosis which shows marked inflammatory phenomena. It is generally accepted that inoculations in this form of the disease are caused by many or by relatively virulent bacilli, or by large doses of bacillary protein, and that this effect takes place in tissues which are markedly sensitized and highly endowed with the property of allergic reaction. Whether the large numbers of bacilli and large quantities of bacillary substances are primarily due to peculiarities of the tissues of these particular individuals, which cause them to furnish a suitable soil for the rapid development and ready dissemination of bacilli and bacillary protein, is a question for the future to decide; but the indications point strongly that way.

No matter whether the cause exists on the part of the patient or on the part of the bacilli, when such reactions occur dissemination of bacilli takes place readily. The metastases in exudative tuberculosis take place largely by contiguity or by bronchogenic infection; so offer the greatest opportunity for large reinoculations. Early in the course of exudative disease, before the tissues have fully developed the property of opposing the multiplication and spread of bacilli, as they do later, the

bacilli multiply rapidly, spread, and produce large quantities of tuberculoprotein which diffuses readily through the tissues, producing exudative phenomena which are often accompanied by destructive effects. In case reinoculations do not take place too rapidly, the tissues of the host develop an increased resistance toward bacilli and an increased tolerance toward bacillary protein. As a result the danger of new infections taking place is rendered smaller, and at the same time the inflammatory response on the part of the tissues is lessened, and the tendency to cavity-formation is accordingly reduced.

A reinoculation of a large quantity of bacilli during this early stage of high sensitization of the cells is commonly met by a vigorous allergic response, which quickly leads to caseation and cavity. Tendeloo teaches that the location of the inoculation is a factor of no mean degree in determining its outcome. He states that cavity is prone to occur in those portions of the lung where the tissue juices move slowest. No doubt this same slowness of movement holds the toxins in prolonged contact with the tissues and is responsible for maintaining an exudative reaction when once established.

A study of the clinical phenomena and the X-ray films of early exudative tuberculosis seems to warrant the conclusion that large reinoculations with rapid multiplication of bacilli are constant causes of acute cavity-formation.

Further observation seems to warrant the conclusion that there is a marked reinforcement of the patient's immunity following these early severe allergic reactions. This seems to be followed by such a desensitization of the cells to bacillary substances that another similar acute inflammatory reaction rarely recurs immediately. Further metastases are not likely to occur until some time has elapsed. Severe exudative processes may remain in the tissues for a long time without other cavities forming. Now and then, however, multiple cavitation will take place, either early or late, even though immunity seems to be raised to a high plane. This type of pathological process, however, is, as a rule, found late in the course of the disease, when it may be interpreted as meaning that immunity is broken down and the patient's defense is waning. A rational interpretation of our observations seems to justify the belief that large numbers of bacilli implanted at any one place at any one time, or extraordinary amounts of concentrated tuberculoprotein brought into intimate contact with the tissues, may cause necrosis and cavity-formation.

Not infrequently we see cavity form in the midst of an area of acute or chronic exudative inflammation. We assume that the cause, commonly, is the detention and imprisonment of focal contents within a bronchus which causes a violent response on the part of the tissues with a resultant cavity.

Acute Cavity Favorable for Healing: The conception which one holds of the cause and nature of cavity-formation and the conditions necessary for its healing will determine what method of treatment he will institute.

That their formation is favored by excessive allergic response seems well established. Further it seems most reasonable to assume that those of considerable dimensions are usually caused by bronchogenic dissemination. Most of the acute cavities which are formed early in the course of the disease are not completely excavated at first. The focal contents have caseated, but they have been only partially expelled. The walls are not clean-cut as they would be were the contents fully emptied. There are masses of caseous material, which may be absorbed, and others which may be readily replaced by fibrous tissue if only conditions favorable to such a process are at once established.

Aside from the favorable conditions in the acute cavity itself, there are other important factors which operate to aid healing, such as the following: (1) The patient's powers of reaction have not been broken down and worn out by long illness, consequently his physiological mechanism is in a condition for responding; (2) as a result of the large reinoculation which was responsible for the cavity, the patient's specific resistance is temporarily raised; (3) the cavity as a rule takes place in tissues which prior to the time of the infection under consideration had not departed far from the normal; (4) the lung having the normal elasticity of its tissues and, as a rule, not being bound by pleural adhesions, compensates readily and closes the cavity by fibrosis aided by emphysema; and (5) there is no fibrous wall holding the cavity open.

Chronic Cavity Heals with Difficulty: The case of late or chronic cavity is different. It offers a different prognosis and requires different therapeutic measures. Late cavity forms as a response to an unusually large dose of bacilli or bacillary protein after the patient has been injured by the stresses of his disease; or results from an early-formed cavity which has failed to heal. In chronic cavity, as compared with acute cavity, the mechanical factors within the thorax are less favorable to healing.

The pleura is usually adherent at the apex, very often at the base, and not infrequently over other portions of the lung as well. Inflammation in the mesial tissues often has fixed the mediastinum. Multiple infiltrations as well as cavities may be found in both lungs and cause marked pathological changes which interfere with the compensation which is necessary for healing. This is more serious; but often one lung will heal first, and then the other may be dealt with satisfactorily.

If cavitation occurs following metastatic spread during the regular course of the disease, it may be found either in the midst of an extensive exudative or proliferative lesion, or standing out by itself in some distant portion of the lung. Under these circumstances the patient's resistance may be ample, and other conditions may be sufficiently favorable to produce healing, particularly before dense fibrous tissue surrounds it. After dense fibrous tissue has once formed, however, the prognosis is different and a different therapeutic attack is required. The cavities which form as terminal phenomena when specific resistance is low or in abeyance offer little hope for any therapeutic procedure.

The healing of early cavities, particularly those of the first and second degree, as described by Jaquerod, is primarily a question of the patient's ability to marshal an adequate defense, both specific and nonspecific, and secondarily a question of mechanics; the healing of late cavity, on the other hand, is probably primarily a mechanical problem. The very fact that the disease has become chronic shows that marked resistance must have been present; and the fact of the failure to heal must lie at least partly in other causes, of which mechanical factors are most evident.

Mechanics enters into the problem of healing more and more as scar-tissue becomes extensive and as the pulmonary surfaces become fixed to the thoracic walls and the mediastinum becomes immobile. The thoracic tissues are put on tension and the whole intrathoracic compensatory mechanism is disturbed. These facts indicate that our opinion of the ability of cavities to heal must vary with the condition met when each cavity presents.

Treatment of Acute Cavity: Patients who excavate an area of pulmonary tissue as one of the very early manifestations of active disease, as evidenced by the fact that they are experiencing at the time or have just passed through an acute immunity response consisting of a temperature reaction usually of several days' duration, with tiredness, loss of strength, diminished appetite and loss of weight, possibly night-sweats,

and a cough, usually accompanied by free expectoration,—the so called "cold;" and who show evidence of a freshly formed cavity on physical examination or on an X-ray film, according to my experience, can usually be treated successfully without compression measures.

Such patients should be put to bed at once and be given the benefit of the modern hygienic, dietetic regimen. A five-pound shotbag should be placed over that portion of the lung which contains the loss of tissue, so as to limit the motion. Bathroom privileges alone should be permitted.

Inasmuch as it is especially desirable to raise such a patient's tolerance to bacilli and bacillary protein as rapidly as possible, I always administer tuberculin in ascending doses to the point of focal response. The focal stimulation is desirable because of its action in promoting fibrosis in the periphery of the excavating mass.

In completely formed cavities the newly proliferating fibrils will penetrate the caseous tissue before it has been expelled and favor its transformation into scar.

Theoretically, the larger the doses of tuberculin which the patient can be given without marked focal reaction the more favorable it should be, because it means a greater desensitization and consequently a less tendency for the repetition of cavitation should further reinoculations of large quantities of bacilli or bacillary protein take place. Fortunately, the reaction responsible for early cavitation raises the patient's resistance to further bacillary onslaughts, and desensitizes him to further quantities of tuberculoprotein so that tuberculin may be given without the danger of reactions that might be expected.

Fortunately, again, the patient's physical condition at this time is usually the most favorable that it will ever be for opposing infection. While he may be in a temporarily lowered state of resistance because of such stresses as those due to previous illness, overwork, or methods of living, yet a rapid recuperation can usually be attained if the proper regimen is instituted. The most important time to treat cavity is before it has completely excavated, or, if not detected then, as soon as it is detected; at least, before it develops thick fibrous walls and a profusely secreting lining membrane, and while intrathoracic compensatory changes are easily made.

Conditions Which Militate against the Healing of Acute Cavity: There are certain conditions under which acute cavities may not heal or may heal with difficulty, even though they are detected when freshly formed,

or before their contents are completely emptied, and even though the patient's resistance seems favorable. Such conditions surround cavities located near the apex when it is covered with a pleural cap; and particularly if at the same time the apex in the contralateral lung is also infiltrated. If, however, the lesion in the contralateral lung is slight and the upper mediastinum is free, the situation still may be favorable; for the mediastinum may move toward the more severely affected side, and sufficient compensatory emphysema may take place to relieve the tension on the contracting tissues. When, on the other hand, the apices and upper mediastinum are adherent, compensatory changes are obliged to come from below and to be made by the diseased lung; and this adjustment may be difficult to make.

The ability of a cavity to heal is further modified by its nearness to the diaphragm or the hilum. Cavities in these locations do not yield so readily as those in the upper third of the lung partly because of the greater motion of the lung near the diaphragm which keeps up a repeated, maximum stretching of the cavities, and partly because of the lessened elasticity of the tissues about the hilum. However, it is common experience to see cavities in these areas heal without mechanical compression of any sort.

Given an incompletely formed cavity or one which has just expelled its contents, in a patient whose lungs have not been previously seriously injured, healing should be expected to take place naturally, with only a little greater difficulty than the healing of an infiltration. But should acute cavities fail to heal by the measures of noninterference, they may be treated successfully by one or another of the measures of compression. Nearly all of them may be successfully treated by some combination of the methods at one's disposal.

Failure in the healing of acute cavity will usually be due to one or more of four causes: (1) an inability to raise the patient's resistance sufficiently to check the spread of the disease and desensitize him to the toxins of the disease; (2) the cavity is located in such a position or in such tissues that healing is prevented; (3) the cavity is too large to heal by the compensation that the tissues are able to make; and (4) the cavity is prevented from healing because of adhesions of the pleura, mediastinum or diaphragm, which increase the tension over the tissues of the lung and prevent the compensatory effects of emphysema from allowing the cavity walls to approximate, and at the same time cause a drag on the cavity walls during each inspiratory effort.

Conditions Which Militate against the Healing of Chronic Cavities: Chronic cavities with dense fibrous walls or walls lined with a secreting membrane, particularly if surrounded by extensive infiltrated tissues or scar, are not so apt to heal without some mechanical assistance. These cavities, too, are prevented from healing by many mechanical factors. Pleural adhesions, fixed mediastinum and diaphragm and rigid thorax, one or all may be present to interfere with nature's attempt at compensation. These conditions place difficulties in the way of healing which can only be removed by measures which help the lung to decrease the total intrathoracic space which it must occupy either temporarily or permanently, as the case may be. Some of these difficulties are met in many, in fact most chronic cavities. While chronic cavities, too, may heal, they will not heal so readily as the acute ones.

Clinicians with extensive experience have occasionally seen the healing of large cavities with dense fibrous walls. This has been particularly true of cavities situated in an upper lobe, particularly the left upper lobe. Such a result is aided by the presence of a free mediastinum and a large pericardium which permit the maximum of compensatory assistance from the contralateral lung, and the lower lobe of the affected side. They must also have observed the healing of large cavities follows the filling of the pleura with exudate which organized and compressed the cavity, furnishing a permanent splinting of the excavated portion of the lung.

One also frequently sees the disappearance of one or more cavities situated in the midst of extensive pulmonary infiltration which involve one or both lungs, provided the patient's resistance is kept on a high plane.

Patients with chronic cavity should be treated with the same hygienic regimen and the same amount of rest as was suggested for those suffering from acute cavity. They should likewise have the benefit of tuberculin therapeutically, and have the motion of the chest over the cavity restricted by shot-bags, or other methods of local compression.

Chronic cavities, however, offer problems that do not exist in connection with the acute ones; but problems which are not always insurmountable. With our present-day methods of treatment many chronic cavities can be successfully handled, provided we can raise the patient's immunity sufficiently high, and maintain it for a sufficiently long period of time; and provided, further, that we are able to overcome the mechanical conditions which interfere with healing.

Special Measures for Aiding the Healing of Chronic Cavities: When a cavity fails to respond to the simpler measures after a reasonable time, or, in case it appears from conditions present that spontaneous healing is improbable, then one has at his command four helpful measures: artificial pneumothorax, with or without intrapleural pneumolysis, phrenic interruption, apicolysis and thoracoplasty. These measures possess different degrees of desirability and are applicable to different conditions.

1: Artificial pneumothorax may be employed in either acutely active or quiescent lesions. It takes off the tension from the tissues by filling part of the intrathoracic space with air. It may be made to actively compress the pulmonary tissues and cause the walls of cavities to be pushed together if such force is necessary. On account of the fact that the degree of pressure decreases as the air becomes absorbed, the tissues are alternately compressed and expanded. This permits drainage of cavities to take place instead of permanently blocking the reservoirs of secretions, as may follow thoracoplasty. For this reason pneumothorax, if cautiously instituted, can be used successfully in the treatment of cavities when the lesion is acute and when areas of caseation are present, conditions which are not well suited to thoracoplasty. The treatment of choice in single or multiple acute cavities which fail to yield to ordinary therapeutic measures in patients with an extending disease, particularly when a competent immunity has not yet been established, is pneumothorax. With air in the pleural space the lung may be kept at rest, tension may be removed from the tissues and healing may keep pace with the patient's increase in strength and specific resistance.

When adhesions are present so as to interfere with full compression, sometimes they may be successfully divided by intrapleural pneumolysis. If they are too dense to be cut, or if there is danger of wounding the lung in cutting them, one must not forget that a cushion of air which occupies any great portion of the intrathoracic space may be made to remove tension and aid healing to a certain degree, even though complete compression is not attained. One must be sure that the compression does not exert traction instead of a compressing effect on the cavity. This actually may happen when the cavity is near the apex and adhesions are present both over the apex coming well down over the cavity and at the base. As air is put in, the line between the second rib above, for example, and the eighth to tenth below, is forced to bend

inward and cause traction on the points of attachment. Under these conditions greater good may be done at times by the use of small than by large amounts of air. One sometimes forgets this in the great desire to see the cavity fully compressed, or because of the fear that other adhesions will form and the pneumothorax be lost.

Adhesions furnish the greatest obstacle to successful compression. A certain proportion of these may be severed by intrapleural pneumolysis. When this is impossible an extrapleural operation may prove successful.

2: Phrenic interruption may reduce the amount of intrathoracic space in the hemithorax by about 25 per cent, but fails to exert a positive force on the pathological tissues, such as may be accomplished by pneumothorax. The good that comes from it is exerted by the removal of tension from the tissues and the reduction of motion in the diseased parts. It must be understood, however, that a patient in whom the phrenic has been interrupted may be subjected to paradoxical breathing, and, instead of the diaphragm descending on inspiration, it may ascend and still keep up movement. This is evident from the fact that the motion of the lower portion of the chest on the side of a phrenic operation may be as great as that on the other side.

Operations on the phrenic nerve, like pneumothorax, may be used in the treatment of cases of quiescent cavity or where cavity is a part of an actively extending disease. It may be of value no matter in what part of the lung the cavity is situated. It is not a desirable procedure, however, unless the contralateral lung is in fairly good condition. In all instances in which immunity is not well established the danger of the disease spreading is always imminent, and any operation which permanently throws an increased amount of work on the contralateral lung, unless it be free from active infection, may defeat the purpose for which it was instituted.

3: Apicolysis, if it could be performed successfully, would be the ideal method of treating cavities in the apices which fail to heal after the usual regimen has been followed, although the patient is otherwise in good physical condition. The permanent success of the operation will be the greater the more thoroughly the patient's immunity is established. This is evident for two reasons: (1) the more immune the patient, the less the danger of the disease becoming active again, and (2) the more immune the patient the more completely may we expect the cavity and the diseased area about it to heal.

Apicolysis has to recommend it the one important fact that it destroys a minimum of lung tissue in order to accomplish the result, but it offers difficulties of accomplishment which, as yet, interfere with its wide use.

4: Thoracoplasty, as a means of treating patients with unhealed cavities, is particularly adapted to those cases of chronic unilateral tuberculosis in which no spreading of the disease is taking place at the time, and in which proliferation of tissue predominates. The patient should have an immunity sufficiently efficient to make recurrence of the disease improbable, for after the operation he will still have virulent bacilli embedded in his tissues which may cause future exacerbations; and he may also be reinfected from without. He is going to be required, permanently, to depend upon one lung, and this he can do only if it remains free from activity.

In this connection we talk of the contralateral lung being free from disease. It is rare to find a chronic tuberculosis which has gone so far that thoracoplasty is required for its treatment in which the contralateral lung has not been the seat of some infiltration. Therefore, it is risky to operate until immunity is well established. The contralateral lung from the time of the operation is required to take on all of the extra movement required for carrying on respiration and the oxygenation of the blood during the life of the patient.

Failure to appreciate the importance of attaining a highly immune state prior to operation has been responsible for much failure in the past. Patients may go on to a satisfactory state of immunization after operation, but it is safer to have it well established beforehand.

Large cavities with excessive, purulent secretion do not offer chances for best results. There is too much danger of the retention of secretion before the walls can collapse and check its formation. Such a condition can only increase suppuration unless the bronchus happens to be so situated as to furnish adequate drainage for the cavity.

On the other hand, chronic cavities with moderate quantities of secretion rarely give trouble. As the lung collapses the cavity becomes compressed with it, and healing is facilitated.

No matter what method is used in the treatment of cavities, there is one cardinal fact which must be borne in mind, namely, that it takes a long time to bring about a permanent healing. Those cavities which are subjected to surgical collapse still must heal like other tissues which are infected. They probably heal no more quickly than those which

heal spontaneously, as a result of the patient's own compensatory lung changes. The only difference is that one is held collapsed until it is healed.

A pessimism prevails regarding the healing of cavities which, according to my experience, is not warranted. Cavities are more serious than simple infiltrations, but they will often heal spontaneously, while the ordinary dietetic-hygienic regimen is being carried out, and many of those that do not heal may be treated successfully by mechanical aid.