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NON-OPERATIVE VERSUS OPERATIVE MEASURES IN THE TREATMENT OF PULMONARY TUBERCULOSIS.

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The forces organized against tuberculosis today are pressing forward in three columns. The physicians are making a frontal attack, while the public health officials and surgeons are holding the right and left wings and pressing rapidly toward the center, where all forces, united, hope to cause a complete rout of the enemy.

At the outset of this discussion I wish to make plain that there is no antagonism between non-operative and operative measures in the treatment of tuberculosis. There is a vast difference in opinion as to when the one or the other should be employed, but such antagonisms as exist are between partisan advocates of the one or the other method. In this paper I wish to discuss the curability of tuberculosis and attempt to point out the manner in which healing is accomplished. I wish further to point out the factors which favor and those which prevent such an accomplishment, and to show how treatment must be suited to each case and further show that the correct treatment is conservative whether it be operative or non-operative.

I wish further to make plain that the particular measure which will be adopted will depend very much upon the opinion which the physician holds regarding the curability of tuberculosis, on the one hand, and its infectiousness both as far as forming metastases in the host and its danger of infecting others is concerned, on the other. He who has great confidence in its curability will be slower in interfering, while he who is imbued with the great danger of its spreading and forming new metastases, likewise he who is imbued with the great danger of infecting others, will interfere more quickly.

If patients could all be treated in an ideal environment by physicians who not only understand tuberculosis but who possess a mental attitude adapted to the treatment of chronic illness, and if the patients were so desirous of getting well that they would rigidly adhere to the necessary regimen, then the cure of early tuberculosis, like that of other infectious diseases, would be largely a matter of natural and specific defense, and only complicating conditions would require a deviation from the well-established hygienic, dietetic, open air, rest regimen.

But such is not the case. Patients with various personalities which determine the degree of coöperation or lack of coöperation which they will give, with various domestic, social and economic problems to face, must be treated in various environments by physicians who have different conceptions of tuberculosis and personalities which endow them with different degrees of fitness for carrying out the treatment of such a chronic disease. These variants influence the success to be attained as much as the character of the treatment which is instituted to cope with the disease *per se*. Therefore, we cannot discuss the treatment of tuberculosis from a purely scientific standpoint, but must include its psychologic, social, domestic and economic aspects.

Therefore, a favorable result obtained by operative measures does not necessarily mean that it could not have been attained by non-operative measures, nor does one attained by non-operative measures mean that it could not also have been attained by operative measures. The point at issue is, which method best conserves the interest of the patient? and this can be determined only after taking into consideration and carefully weighing the dangers that may come to the patient by not using some operative measure and comparing them with any injury that may follow the employment of operative procedure.

Development of the Modern Methods of Therapy. For centuries, physicians were called only to treat patients suffering from what we now look upon as far-advanced tuberculosis. There was no organized program of treatment based on a knowledge of the disease, nor was there any accurately recorded experience on which to base opinions. Tuberculosis was not recognized as coming under public health control. It was not even classed among the reportable diseases. It was simply looked upon as an unpreventable and hopeless malady.

At the beginning of this century the entire picture changed. There was a steadily growing belief that tuberculosis could be cured and, further, that it probably could be prevented. A group of leaders with an increasing army of followers began to act upon this belief. A campaign of education ensued which was prosecuted with such zeal as had never before been witnessed in connection with any disease. Medical societies gave tuberculosis an important

place on their program; public addresses were given; pamphlets were distributed; and laws were enacted to provide the measures necessary to prevent and the facilities necessary to cure the disease.

For the first time in history the medical profession began to take a real vital interest in tuberculosis. The result, the reduction of tuberculosis to the sixth rank as cause of death, and the reduction of infection to one-half of its former amount, is one of the truly great medical triumphs. In the space of time marked by less than one-half of a generation we have so changed our attitude toward the disease, and so successfully devised methods for its prevention and cure, that we now feel that it is well on the way to be conquered or at least to be reduced to a position of importance comparable to that of other serious diseases.

As long as tuberculosis exists, however, it will be a disease of special importance, because of the fact that its prevalence is so much greater during the productive period of life than is that of most other serious diseases and, further, because of its chronicity and its slowness in healing.

Public health agencies are now spending their energies in the prevention of tuberculosis, and clinicians are devising methods for meeting the problems which arise in its treatment. Even the more serious types of the disease, those which were entirely beyond hope but a short time ago, are now being successfully treated. That we are able to relieve patients suffering from far-advanced lesions must not blind us to the most important fact that has been brought out by a quarter century of experience in the intensive treatment of tuberculosis, *viz.*, that early lesions are the most easily cured, and that they leave the patient in the most satisfactory state of health and most capable of carrying on his life's work. My experience leads me to state that it is practical rather than Utopian to believe that operative measures are only justifiable until such time as we are able to make early diagnoses and institute adequate treatment as soon as diagnoses are made.

In order to appreciate what each individual measure contributes to cure, it is necessary to have a fairly accurate understanding of the fact that tuberculosis is a bacteriologic disease which produces pathologic and immunologic responses which in turn alter anatomic structures and physiologic reactions. Some of these changes in structure and function are favorable while others are unfavorable. It is the duty of the physician, as far as he can, to recognize and promote the former and to prevent the latter.

Cure of Tuberculosis—An Immunity Reaction. The important factor in the cure of all infectious diseases is immunity. This is particularly evident in diseases of short duration, such as diphtheria and tetanus, in which the toxins are the lethal agents and the immunity is all but absolute; for these can be cured by the adminis-

tration of immune bodies artificially produced. It is also readily understood in such diseases as typhoid in which protective inoculation of killed bacteria will immunize, or smallpox, in which an artificial vaccination will produce a miniature disease and protect the host from future disease. In tuberculosis, on the other hand, we have a disease, chronic in nature, which produces an immunity, but one more difficult to understand. A miniature disease caused by a small number of bacilli will produce, at least for a time, a relative immunity against future inoculations, but is not able to grant a full immunity. Protection may also be granted by dead bacilli similar to that produced in typhoid vaccination, but again only relative and not so high as that given by a real infection caused by living bacilli.

Bacilli and the toxic substances produced by them are responsible both for the production of a relative increase in resistance to further inoculations and for injury and destruction of tissues. We have experimental evidence showing that inoculation of both living and dead bacilli will produce a relative degree of protection against new infection, and we have clinical observations which prove that the protective mechanism which is set going by natural inoculations of bacilli and bacillary products is effective in lessening the seriousness of later inoculations and in healing the disease.

In the natural healing of tuberculosis we rely on the patient's own body reactions to bring about healing, the same as in all other infectious diseases for which we do not have specific remedies. This protective mechanism differs with different individuals, both as to its efficiency and as to the time required for its development; but it is slow in all cases. This fact furnishes one of the chief causes of failure.

Tuberculous patients do not die because the disease is incurable, but because it is not cured. There is a time when nearly every patient suffering from tuberculosis has it within his power to attain a cure. That he does not do so is due to the fact that for one or another reason the disease is not brought under treatment at this favorable time, or that the kind of treatment is not suited to the condition at hand; or is not properly applied. We assume that healing is primarily a physiologic process, and that its accomplishment depends upon the establishment of a much higher degree of immunity than was necessary successfully to overcome the minor lesions of early infection.

We cannot describe categorically the factors which make for immunity, nor can we assign to each its particular part in bringing about healing, but we have learned that a good physiologic balance is a great asset. We have learned that rest is the most important single factor in maintaining such a balance during the stage of active disease. It produces its favorable effects in many ways. It lessens

the energy requirements of the body at a time when the demands of increased energy cannot be easily supplied. It calls for a minimum of circulatory activity in the body and so in the lungs, and calls for a minimum of respiratory effort, thus putting the diseased areas at relative rest. This reduces the danger of metastases occurring and minimizes the escape of toxins from the foci, thus relieving the patient of avoidable toxic symptoms. It reduces the body's food requirements to a minimum and permits of storing of any excess of energy for supplying future demands.

Rest of mind is as essential as rest of the body, so the development of a proper mental attitude is important. It can thus be seen that the control of the patient is necessary to guarantee to him a physical and mental rest. No matter what other measures are used in treatment, mental and physical rest should be maintained during the period when the disease is active.

If a carefully devised program, suited to the patient's particular requirements, is carried out sufficiently long, it will assure success in nearly all of those cases in which healing depends alone upon the resistance of the patient. In other words, a well-balanced physiologic state is sufficient to produce the cure in nearly every case at this favorable time.

I wish to emphasize this fact, because in our enthusiasm for operative methods we are prone to forget that if the disease were treated early, the conditions which require operation would be rarely met. Our slogan should be "early diagnosis and immediate, adequate treatment." In what instances are non-operative measures insufficient? This is the question to be considered.

The clinician may have his faith in the ability of the patient to cure his own disease greatly increased by patiently treating those who seem to offer little hope of cure unless the lung may be compressed by pneumothorax, or the tissues be relaxed by phrenic evulsion, and in whom attempts at the former have been unavailing and the institution of the latter has failed to cause elevation of the diaphragm, or by patiently treating certain extensive bilateral lesions which are not suitable for operation. In such cases it is not a rare thing to see the usual sanatorium regimen followed by healing.

I have formed the opinion from treating several thousand patients suffering from advanced and far-advanced tuberculosis that, if non-operative treatment is followed with sufficient detail and continued sufficiently long, most patients in whom we can establish and maintain a resistance sufficiently high to prevent extensive caseation with necrosis and serious new metastases from forming have an excellent chance of ultimately bringing their disease to a state of quiescence, and that, unless cavities which are too large or cavities possessed of dense walls, or mechanical conditions which prevent adequate compensation from taking place are present, they

may go on to an eventual healing. It is the patients in whom metastases continue to take place, in whom destructive lesions are uncontrolled, or in whom mechanical factors interfere, that cannot heal readily.

The Mechanical Factors Which Interfere With Healing. As the disease progresses, however, there are certain factors which enter into the picture that make healing difficult, sometimes even impossible, no matter how high resistance may be raised. These factors are largely of a mechanical nature.

The therapeutics of tuberculosis cannot be understood without taking these mechanical problems into consideration, for they really are the most important factors in separating tuberculosis into operative and non-operative. Too often, to the therapist, the cure of tuberculosis means some one particular thing—a hygienic regimen with rest and control as the chief factors, tuberculin, heliotherapy, pneumothorax, a phrenic evulsion or a thoracoplasty. Such an attitude toward cure is not rational and does not take into consideration the best conception of tuberculosis or the best interests of the patient.

The lungs are confined within the thoracic cage and are in close contact with rigid bony structures on all sides except at the base, where they are confined by the diaphragm. In tuberculosis a shrinking of the pulmonary volume takes place, whether as a result of destruction of tissue or by the replacement of elastic tissue by fibrosis, or by the reduction of the volume by exudation and atelectasis.

On the principle that Nature abhors a vacuum, the total volume of pulmonary tissue always must approach the amount necessary to fill the thoracic cage. When the volume of pulmonary tissue is reduced it can be compensated in only one of two ways: the structures which confine the lungs must contract and make the thoracic cage smaller, or the lungs themselves must compensatorily enlarge. These two processes go hand in hand as tuberculosis advances.

During the early stages of the disease the reduction in lung volume is of comparatively slight importance and usually is readily compensated by an emphysema of the uninvolved or less involved lung tissue. Later, too, it may be compensated, but with greater difficulty, and, finally, not at all. As soon as the stage is reached in which compensation is not readily made, healing is interfered with, and no matter how stable the patient's physiologic equilibrium or how competent his immunity, healing can go forward only with difficulty.

The mechanical conditions which particularly interfere with compensation are pleural adhesions, fixation of the mediastinum, adhesions and fixation of the diaphragm, widespread fibrosis, the emphysema which has developed prior to the time of the destructive lesion and rigid thorax.

The manner in which pleural adhesions interfere with healing

may be studied in cases of bilateral apical tuberculosis, in which an apical, pleural cap is found on both sides. The tissues are rigidly fixed in an area as extensive as the adhesions. Contraction of the pulmonary tissues which accompanies the disease makes traction on the unyielding pleural adhesions. Since they cannot give way, whatever compensating change occurs must take place from below, and, since the diaphragm is a muscle with strong contracting power, the compensation really must occur largely by the enlargement of the air cells, causing an emphysematous condition in that portion of the pulmonary tissue which is not involved in the tuberculosis. The seriousness of the interference which is caused by adhesions in double apical tuberculosis must be evident to any one who will note the extent of the shifting of the upper mediastinum which is at times necessary in order to compensate for the contraction which follows healing of unilateral lesions which involve the upper portion of the lung. The tension to which these tissues would be subjected were the mediastinum fixed is evident.

Again, the interference with healing which is caused by pleural adhesions is shown when an extensive one-sided adhesive pleuritis exists over a lung which is the seat of a severe lesion of either the exudative or the proliferative type. If a fixed mediastinum is also present, healing may take place, but is rendered very difficult without some operative measure which will aid in adjusting the intrathoracic space to the lung volume. If the adhesions do not involve the entire pleura, a partial pneumothorax may be of value, or a phrenicectomy may afford a considerable degree of aid. If a cavity of any considerable size is present, and particularly if it is situated in the midst of fibrous tissue and possessed of a thick wall, healing is rendered very doubtful, except as a result of operative assistance. Under these circumstances thoracoplasty may become practically our only hope.

The Characteristics of Proliferative and Exudative Lesions Which Modify Treatment. While the curability of tuberculosis depends upon the extent, age and activity of the lesion, it again depends upon whether it is predominantly proliferative or predominantly exudative. That there is a difference in exudative and proliferative tuberculosis has long been known, and recently we have begun to understand certain of the fundamental causes of this difference. The fact of this difference must be considered in applying therapeutic measures.

Predominantly proliferative tuberculosis has long been recognized as a comparatively benign process, one which primarily makes for fibrosis with chronicity rather than for acuteness with destruction. It has long been assumed that it is caused by relatively few and mildly virulent bacilli, and that the predominant reaction of the tissues is that of cellular proliferation rather than exudation and destruction.

Now that pure fractions of the bacillus have been given to us

with which to work, it has been found experimentally that bacillary lipoids produce fixed-cell proliferation and tubercle formation, with little toxemia or exudative reaction, while bacillary protein causes exudation and destruction, and the polysaccharid seems to be the chief toxic element. Sabin, using a phospholipin and injecting it into the peritoneal cavity of animals, has been able to produce huge proliferative masses of tissue, so we now explain proliferative tuberculosis as being predominantly a reaction of the body to the lipoids of the bacillus, to the bacillus as a foreign body, in contradistinction to its reaction to the bacillary proteins and polysaccharids or the bacillus as a living immunizing substance.

It is also characteristic of predominantly proliferative tuberculosis that it extends in spite of its mildness. It seems to fail in calling out the immunizing mechanism which raises the patient's specific resistance, prevents the ready passage of bacilli through the tissues and increases the body's power to destroy them. Sooner or later, however, exudative phenomena appear, destruction of tissue takes place, symptoms of intoxication supervene and the process becomes a combination of proliferative, exudative and often destructive characteristics with increased acuteness, and, at the same time, with evidence of a heightened immunity. Unfortunately the immunity makes its appearance, as a rule, after extensive fibrosis has taken place, and the tissues are harboring widespread infection.

Predominantly proliferative tuberculosis, because of its mild nature, bears a reputation for ready healing which it does not deserve. In my experience a proliferative lesion produces milder reactions, extends more slowly, but often is far more difficult to heal than a preponderantly exudative lesion of similar extent. And when it does heal it leaves the lung tissue replaced by extensive scar, while the exudative lesion heals largely by resolution, leaving only a minimum amount of scar.

Predominantly exudative tuberculosis is a more acute process. We assume that it is caused by larger quantities of more virulent bacilli than those which are responsible for the preponderantly proliferative lesions, and that through their growth, rapid multiplication and destruction, large quantities of bacillary lipoids, protein and polysaccharids are set free in the tissues. The exudative response is called out as the body's reaction to the protein fraction, which is the same fraction that stimulates the patient's immunity mechanism. The toxicity, however, is probably largely due to the polysaccharid. New tissue formation takes place as a healing measure, but it is limited in extent and secondary to resolution of the exudate. Extensions do not take place gradually, as in the predominantly proliferative type of disease, but more precipitately and result in more acute reactions on the part of the patient, a greater toxicity and a more efficient immunity.

The symptoms of acuteness result from the rapid destruction of

bacilli and the liberation of bacillary products, particularly the polysaccharids and proteins.

Pulmonary Lesions Which May Heal By the Hygienic Rest Regimen Alone and Those Which Require Operative Assistance. From the purely scientific standpoint in which the curability of tuberculosis alone is considered, the following types will heal fairly regularly without operative assistance:

1. Early limited lesions of either the proliferative or exudative type.
2. Proliferative lesions more extensive than those mentioned under Group 1, involving one or both lungs, provided they have not taken on extensive metastases and destructive processes with multiple cavitation.

(*a*) Small cavities may usually be expected to heal, but the healing of large ones, especially if multiple, is more doubtful without operative aid.

(*b*) Whether or not such lesions will heal depends much upon the extent of injury which has been done to the lung tissue and the ease with which the necessary compensatory changes between lung volume and intrathoracic space may be made.

3. Exudative lesions more extensive than those mentioned in Group 1, with or without cavity, provided the non-infected lung tissue can take on the required emphysematous changes and the mediastinum is free to shift in case it is required by the compensation which must be made, and provided further that other limiting structures are able to accommodate themselves to the reduced lung volume.

Early cavity in exudative tuberculosis is not a contraindication to healing unless it is held open by pleural adhesions and a fixed mediastinum, or so located that it cannot close.

4. Exudative lesions which are accompanied by extensive atelectasis will usually heal even though they may be accompanied by high temperature which requires several months to reach normal.

From the purely scientific standpoint, this leaves practically no early cases that regularly require operative assistance, but as the disease advances there are several types of lesion which cannot be depended upon to heal without operative aid, some of which are the following:

5. Comparatively small lesions with a cavity which is held open by pleural adhesions, and is prevented from closing because the unaffected lung tissue is not able to make the necessary compensatory changes, such as apical or subapical cavities covered by a pleural cap, and especially when the upper mediastinum is fixed.
6. Any active lesion which continues to form metastases unduly long in spite of carefully followed non-operative treatment.
7. Lesions in which a destructive process is seriously threatening cavity formation; in fact, should cavitation threaten during the course of chronic tuberculosis, it should probably always be prevented by collapse if possible.
8. Any lesion which is prevented from healing by mechanical hindrances. This will include:

(*a*) Small cavities situated so that the walls cannot collapse as in the apex covered by a pleural cap; small cavities in dense scar tissue situated in any part of the lung; and those near the hilum or diaphragm.

(*b*) Extensive infiltration, with or without cavity, in which the tissues are put on marked tension, and in which compensation necessary to healing cannot be readily made.

(*c*) Large cavities with thick fibrous walls.
(*d*) Cavities in a much contracted lung, with displaced mediastinum, in which further compensation cannot be made.

In my experience this grouping roughly separates the cases which may be expected to heal by non-operative measures from those which require operative assistance. It does not, however, represent the manner in which tuberculous patients are generally treated, because operative measures are so frequently found necessary to meet the exigencies under which treatment is carried out. Not only are the Groups 5, 6, 7 and 8 recognized as requiring operative aid, but some of Group 1, many of Groups 2 and 3 and practically all of Group 4 are usually treated by pneumothorax or phrenicectomy or both.