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RELATION OF TRAUMA TO PULMONARY DISEASE*

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Interpretation of the term, trauma, meaning originally injury caused by external violence, has been progressively broadened to include a great variety of injuries of structure and function caused by a great number of agents. The need for a clearer conception of the relationships has increased in proportion. The soundest basis for this is a broad understanding of the fundamental mechanisms which usually operate in the disease under consideration. Without this a reasonable estimate of the bearing of trauma is impossible. It is my purpose, therefore, mainly to discuss some of these mechanisms in pulmonary disease.

First, it is necessary to bear in mind the physiological mechanisms which guard the lungs against the invasion of foreign agents and dispose of those which get past the portal. The most important are the pharyngeal and laryngeal reflexes which, when effective, divert substances from the glottis, and together with the tracheal reflexes, initiate coughing for the expulsion of substances from the lower respiratory passages. Impalpable materials, such as dust and bacteria, are removed by the finer mechanisms of ciliary action of the bronchial mucous membranes and by phagocytosis.

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Another function which also has a protective value is a complicated one in which respiration and circulation operate to maintain a correct moisture of the pulmonary surfaces. These defences normally act as a harmonious whole. Their smooth and efficient play depends on intact nervous pathways, a healthy state of the tissues of the respiratory tract, normal tone of muscles of the chest and abdominal walls and diaphragm, and on good circulation, all of which, of course, means good general health. They are safeguards which do not yield readily even under the conditions of poor health.

In unusual situations, the failure of one protective mechanism may directly or remotely involve the failure of another, and it may be the combination which causes harm. Thus, the entrance of a foreign substance into the bronchi is not necessarily injurious unless it becomes lodged; and lodgment is due to ineffectiveness or failure of the coughing mechanism. Under other unusual conditions, perfectly intact defenses may fail, such as happens when a blow to the mouth dislodges a tooth and casts it through the larynx into a lower lobe bronchus,—unknown to the victim who thinks he swallowed the missing member. Protective reflexes may be impaired during states of unconsciousness, and this varies from slight dulling during sleep to almost complete abolition during deep coma from bodily injury, alcoholism or other cause. Material from the nasopharynx and mouth may then gain ready access to the smaller divisions of the bronchial tree and there find lodgment. The consequences depend on the physical character and toxic qualities of the substances aspirated and retained. Fluids which are not heavily contaminated with virulent bacteria may set up only temporary irritation or mild bronchopneumonia. If the fluid carries with it necrotic matter bearing anaerobic pyogenic bacteria from pyorrhea pockets or tonsillar crypts, acute suppurative pneumonia or lung abscess may follow. Inhalation and retention in the bronchi of vomitus during unconsciousness sometimes

causes rapid death of tissue or acute gangrene of the lung ending fatally in one to two weeks. Formed substances or foreign bodies lodging in the bronchi may at first be only mildly irritating, giving rise to the slight cough, wheezing, and slight difficulty in breathing which we associate with bronchial stenosis and catarrhal inflammation. Occasionally, incomplete or complete occlusion of a bronchus occurs abruptly with resulting obstructive emphysema or atelectasis. Regardless of the first effect, these occluding and irritating substances, if they remain, set up mild or severe infection beyond the point of lodgment, and this sooner or later leads to destructive, suppurating and organizing bronchopneumonia and bronchiectasis. The section of lung involved may be small or all inclusive, depending on the seat of the foreign body in a small branch or a main stem bronchus.

In many instances, the start of one or more of these pulmonary processes within ten days or two weeks after trauma may be reasonably attributed to the trauma on the basis of these mechanisms. Actual examples in our experience include bronchopneumonia following unconsciousness caused by automobile injuries, suppurative pneumonia ending fatally six months after a compound fracture of the jaw, bronchopneumonia from the aspiration of contaminated fluid during the careless irrigation of the paranasal sinuses, lung abscess developing in a man shortly after he fell into the East River near the mouth of a sewer, and others.

These conditions often are related to the stasis of fluid secretions in the bronchi and lungs. Normally, the fluid interchange here is great, and excessive amounts are disposed of through absorption into the circulation, expulsion by coughing, and by evaporation. A serious failure of any of these mechanisms and the resulting accumulation of fluid may create a favorable medium for infection. A familiar example is hypostatic pneumonia in the patient with failing circulation. Likewise, failure of the extrinsic respiratory muscles and particularly of the diaphragm to function properly not only may cause impairment of ventilation of

the lung but also may interfere with disposal of fluid from it. The accumulation may then favor infection with organisms carried in with inspired air or in material aspirated from the pharynx. Trauma of the chest wall or upper abdomen may be the cause of inaction of this important respiratory mechanism. Usually the effect is only partial, temporary, and harmless, but, when the injury is severe and associated with shock, factors may combine to set up a train of disturbances leading to pneumonia.

Blood entering the bronchi and lungs behaves like any other fluid except for its power to coagulate. A clot may act like other foreign substances and, by occluding the bronchus, lead to obstructive emphysema or partial deflation of the lung. Retention of large clots, however, does not often occur. Fluid blood, retained in the alveoli, is irritating and so favors congestion and inflammation which usually is mild and fleeting. I have observed bronchopneumonia of a few days duration in an epileptic who, during a convulsion, bit his tongue and aspirated the issuing blood.

That bleeding from preexisting lesions in the lungs may lead to aggravation of the disease is well recognized. Rupture of a varicose vessel in a bronchiectasis may lead to hemoptysis. Except for the temporary break in the vessel, the incident may be quite harmless. If some of the blood accumulates and is retained in the alveoli, a mild and passing inflammation results; but if the blood washes out of the bronchiectatic sac some anaerobic bacteria and these are also retained, then the resulting pneumonia may be suppurative, leading eventually to more extensive bronchiectasis. Much the same is true of hemoptysis in pulmonary tuberculosis. If the bleeding arises from the vicinity of a healed and fibrotic section which is not casting off tubercle bacilli at the same time, the results are not likely to be very harmful. But this is relatively seldom. Hemorrhage in these cases usually arises from the rupture of a blood vessel in the wall of a cavity which is in direct connection with the bronchi and which is also discharging pus laden with tubercle bacilli. Exsanguination is infrequent. The grave danger is aspiration and retention of

blood in healthy parts of the lungs. Here is a very fluid medium becoming well charged with tubercle bacilli as it escapes. The flow stimulates stronger respiration and coughing, effective protection in some cases, but in others inadequate. A rapid stream overwhelms the physiological defenses and with each inspiration ebbs into the bronchi of the dependent parts. That which is retained in the alveoli initiates not only the usual posthemorrhagic aspiration bronchopneumonia but also an associated spread of tuberculosis which then runs the course of any fresh lesion of that nature.

I shall dwell further on the subject of tuberculosis because it is alleged to be aggravated by trauma more often than any other pulmonary disease. Knowing, as we do, that the course of tuberculosis is subject to many deviations, it often becomes an exacting task to distinguish the developments which are due to trauma from those which merely follow it. In some instances, the question is a matter of judgment which varies with the experience and conceptions of the clinician, but in a majority of cases it is possible to obtain objective evidence which is of definite and decisive value. The minimum requirements in examination are a thorough and accurate history, complete physical examination, a technically satisfactory roentgenogram of the chest, and careful examination of the sputum by one of the more refined and reliable methods for the detection of tubercle bacilli. It may be necessary to observe the patient under known clinical conditions for a time to secure complete data. If previous medical records, and, especially, previous roentgenograms of the chest are available for comparison the problem is greatly simplified. With the information from these studies, one must then decide whether the mechanisms in progress were influenced by the trauma. For illustration we may return to the problem of pulmonary hemorrhage in a tuberculous case. Most hemorrhages come during the natural course of the disease and start while the patient is quietly at rest. Nevertheless it is fair to conclude that some may be initiated by trauma, such as those which immediately follow a heavy blow on

the chest or a violent physical strain. An example is the case of a tuberculous workman who, in the course of his occupation, lifted a heavy box, felt a tearing sensation in his chest and immediately coughed up blood. After the cessation of bleeding in such cases, the question is to what extent the preexisting tuberculosis was aggravated. The mere break of a vessel usually heals itself within several weeks after the thrombus forms, and the mere loss of blood, therefore, does not indicate a serious aggravation. A short run of fever after the hemorrhage may represent only a simple bronchopneumonia which likewise resolves satisfactorily. Serious aggravation should show itself in a few days after the hemoptysis—usually by fever, the detection of rales at the base of one or both lungs and the finding by X-ray of the shadows of fresh bronchopneumonia which after a week or two fails to resolve or actually progresses; at the same time tubercle bacilli will usually be found in the sputum. It must be remembered that the usual cause of spread of tuberculosis in the lung is the aspiration into healthy parts of purulent exudate from cavities. A spread demonstrated for the first time two or three months after a hemorrhage is not likely, therefore, to be due to the hemorrhage. The necessity of giving due consideration to time relationships is most important.

Pulmonary tuberculosis may be stimulated detrimentally by a variety of nonspecific agents such as the injection of arsenical drugs or of vaccines, exposure to the X-ray, excessive sunburn, or severe intercurrent respiratory infections. Latent and fibrous lesions are not easily influenced, but fresh inflammatory lesions which are progressive or on the verge of progression may occasionally be aggravated.

Many occurrences, like those cited, are simple to explain on the basis of the objective findings. A more difficult situation is that, for instance, in which, following an injury, a person reports pain in the chest, fatigue and loss of weight which he ascribes to his tuberculosis. The point at issue is whether this obviously old but newly discovered disease was aggravated by the trauma. Objectively, there may be no evidence that can lead reasonably to the presumption of renewed activity or progression of the lesions.

The mental anguish of learning the diagnosis may be more of a factor in the genesis of the symptoms than anything else. It recalls Claude Bernard's definition of a healthy man as one who is unaware of his disease. We cannot escape the duty of recognizing and fairly estimating psychogenic factors in physical symptoms, though the opportunity of abusing the duty is open here. In clinical medicine it is not possible always to invoke the explanation of clear cut principles to explain a series of events, but, the goal should always be to search out and identify underlying mechanisms if possible.

This is equally true of such accidents as rupture of the lung with the production of pneumothorax. It is doubtful if a perfectly normal lung ever ruptures, except by a puncture wound from without. Aside from such wounds, caused by stabbing, the fracture of a rib, and the like, the common causes of rupture of the pleura are the ulceration of a subpleural tubercle, the breaking of a subpleural emphysematous bleb or the tearing of a pleural adhesion. If trauma, such as a sudden violent strain, precipitates the break of the pleura at a point where it is damaged by one of these lesions, it may in unusual cases be responsible for sudden pneumothorax, collapse and death. Immediate fatalities are not frequent, however. A simple mechanical tear usually heals itself rather rapidly, and the disability is not lengthy. An accompanying hemorrhage into the pleural space is more serious, and the opening of a tuberculous cavity into the pleura with the inevitable complication of empyema is most serious. "Spontaneous" pneumothorax is much more frequent than indirect traumatic pneumothorax. Evidence should be clear, therefore, to indicate the close time relationship between the trauma and the onset of symptoms.

Tuberculous pleurisy, induced by trauma which is severe enough to fracture ribs also, is obviously due to the activation of a preexisting focus in the pleura or lung. Such cases are very uncommon and, when they occur, run the usual course of pleurisy with or without effusion.

The late effects of empyema or hemothorax, which may have been related to severe trauma, such as a gunshot

wound, occasionally show themselves as fibrous and calcific organization of the pleura, shrinkage of the affected hemithorax and lung, and a degree of fibrosis and emphysema of the lung.

Silicosis is a subject in itself and it must suffice here to mention that it represents the results of long and often repeated injury of the pulmonary tissues by the physical and chemical action of inhaled dusts; and that this injury frequently opens the way for the development and progression of tuberculosis. On the other hand, there is no reason to believe that pulmonary tuberculosis is often influenced by the inhalation of irritating gases or fumes. The experience of the World War provided abundant evidence on this point and spoke eloquently for the efficiency of the physiological defenses and reparative forces of our respiratory systems. The usual effect of the inhalation of such gas is inflammatory edema of the bronchi and lungs which soon clears away. More serious or permanent effects usually depend on more intense initial inflammation and the entrance of infection causing bronchopneumonia. If prolonged, this may suppurate or undergo organization with the gradual development of disabling fibrosis and emphysema.

It is not possible or desirable to enumerate all the imaginable varieties of trauma which occasionally incite or aggravate pulmonary disease. It is more profitable to study fundamental mechanisms of morbid processes in order that the relationships of trauma may be fairly estimated. Consider also that under ordinary conditions of life the lungs are not very vulnerable.