DAMAGED LUNGS AND BRONCHIECTASIS.

BY

CLIVE RIVIERE, M.D.LOND., F.R.C.P.,

PHYSICIAN TO CITY OF LONDON HOSPITAL FOR DISEASES OF THE CHEST
AND TO EAST LONDON HOSPITAL FOR CHILDREN, SHADWELL.

VERY commonly does it happen that some amount of permanent damage remains as the sequel to an inflammation, of whatever causation, in the pulmonary area; and the more severe and prolonged the attack the greater, as a rule, the disability. Lung tissue, air tubes, and pleura alike may be implicated in different degrees, the end-results showing themselves in fibroid lung, or simple bronchiectasis, or more commonly some combination of both these conditions. Such gross lesions as follow the textbook account under these headings are readily recognized and need no recapitulation. What I would here emphasize is the common occurrence of slighter degrees of damage, readily overlooked, but often the explanation of anomalous signs and symptoms at some later period. Mistaken as a rule for pulmonary tubercle, they present a ready pitfall, unless recognized, in the realms of diagnosis, prognosis, and also of therapy, for they are very amenable to well directed treatment. "Damaged lung" is the somewhat loose term that best covers, to my mind, the varied mixture of mild fibrosis and dilated bronchial tubes with which I am here dealing, but it is, of course, the bronchiectasis that counts—or rather "bronchiolectasis," for it is the small tubes which are generally implicated in these cases. It will be most convenient to consider the subject under separate headings in children and in adults, but it is in the early years of life that the majority of all cases originate.

In Childhood.

Many years ago I collected some 40 cases of pulmonary fibrosis and bronchiectasis in childhood for purposes of study and analysis. These, which included slighter cases of similar etiology, showed clearly the common origin of lung damage from the inflammatory lung diseases of childhood, particularly in association with the infective catarrhs of measles and whooping-cough. It is interesting in this connexion that American investigators have recently associated a definite thickening of x ray lung shadows after these two diseases. But bronchopneumonia stands out in childhood as the prime causation of lung damage, and this is found clinically at the bases of both lower lobes.

Bronchopneumonia—	Cases.
Simple	6
With whooping-cough... ..	5
With measles	3
	—14
Bronchitis—	
Simple	6
With whooping-cough... ..	3
With diphtheria	2
With measles and whooping-cough	2
	—13
Lobar pneumonia	4
Congenital atelectasis	2
	—33

To these I should add, with further experience, two other causes in childhood—influenzal pneumonia and tuberculosis. Influenzal pneumonia leads very readily, as in adults, to fibrosis and bronchiectasis, and I have seen several cases which well illustrate this fact. The lung damage left after tuberculosis in early childhood will be an object of scepticism to many who still hold desperate views as to the mortality of the disease at this age period. The x rays have long convinced me of the recoverability of pulmonary tuberculosis even in infancy, and in such cases some signs of lung damage generally remain behind.

Let us now consider some common types of "damaged lung" in childhood.

First, there is the case in which the condition may be seen to develop after bronchopneumonia. The signs at the bases of the lungs, percussion impairment, and crepitations do not completely clear up after the child appears otherwise well and vigorous. A not uncommon sequel is a further attack, which may appear to be bronchopneumonia, but is somewhat milder than the original attack or than the usual course (very varied, by the way) of this affection. Sometimes repeated attacks supervene, and it is always wise when a case of bronchopneumonia is seen for the first time to inquire for former similar illness. A history of this, the mild course of

the illness in some cases, occasionally a slight clubbing of the nails also, may help us to suspect that an attack is occurring on the top of lungs already "damaged." These further attacks may amount to a mere acute catarrh of the dilated bronchioles, with some "congestion" of the surrounding lung, but no true pneumonic consolidation. In other cases a fresh bronchopneumonia occurs; the physical signs are very similar in both cases.

The second commonest simulation of "damaged lungs" in childhood is of pulmonary tuberculosis. Most of the cases are basal, and then the mistake is less likely to arise; but when a coughing child presents an area of impairment and crepitations at an apex, commonly the right, it is no wonder if he gets treatment for pulmonary tuberculosis sometimes for years on end. Only recently two such cases, which had been under prolonged treatment, were sent to me by a tuberculosis officer in the London district for comment and discussion. Such cases originate, I think, in apical lobar pneumonia, the right apex being the second commonest situation of the lesion in this disease in childhood (the left base being the commonest). The children are much less ill (often not ill at all) than they would be with tuberculous lesions of similar extent, and the history and course, if other points fail, should suffice to make clear their nature. The physical signs of a "damaged lung" in childhood necessarily vary with the case. Where there is much fibrosis this is indicated by marked dullness, feeble breath sounds, often with diminution of vocal vibration, and by contraction of the diseased area. Where bronchiectasis involves the larger bronchi, no added sounds may be audible or a few coarse râles are heard. It is where the smaller bronchial tubes, and these near the surface, are affected that the more typical signs—breazy consonating crepitations, sounding very superficial—are heard. In many cases these sounds may simulate pleural friction. Typically, as occurring after a protracted bronchopneumonia, they closely simulate, both in nature and in distribution, the physical signs found in this disease—the symptoms of illness disappear but the signs remain! When a lung from such a case can be viewed on the *post-mortem* table it shows a reticulated appearance from the development of numerous bronchiolar sacs.

Now the failure of bronchopneumonic signs to clear is no uncommon occurrence; many such cases of bronchiolectasis leave our children's hospitals, and it may well be asked what becomes of them. Some may, perhaps, be still recoverable; some, we have seen, return with recrudescences of catarrh; but there are others whose health does not appear to be prejudiced but who yet retain some damage to their lung bases and carry this presumably into adult life—where we shall meet them again farther on in this paper.

As to the symptoms of "damaged lungs" in children, these will depend on the amount and nature of the bronchiectasis. Some will have cough and nothing to show for it; others will bring up sputum, and occasionally blood, in varying quantities. But few appear to be really ill or toxic in their early years, apart from the severe febrile lung attacks to which a proportion of them are prone. The sallowness, with wasting and foul sputum characteristic of bad cases of adult bronchiectasis, are much less common in childhood.

In Adult Life.

"Damaged lungs" in adult life are very commonly the inheritance of inflammatory diseases in childhood. Not to speak of marked and typical cases, we may meet with the conditions to which I would draw attention under very different circumstances.

First, there are cases where a "basal bronchitis," strictly confined to the broncho-pneumonic area, is found—generally as a chronic condition with exacerbations. My belief that these are cases of bronchiolectasis derived from a childhood bronchopneumonia is a mere pious opinion. I have no real proof to offer. May not they represent a proportion of those cases of unresolved bronchopneumonias which retained their damage with but little immediate ill health? The history of certain of them suggests that this is so. More likely still is this the case in many cases of so-called bronchopneumonia, apart from influenza, in adults. Influenzal bronchopneumonia must of course be given its due place, but I am myself doubtful of most of the so-called bronchopneumonias of other causation in adult life. In many such cases the signs and symptoms are much more suggestive of a severe catarrh in an old basal bronchiectasis, and in such cases the signs will not completely clear up after the attack. They may, however, be slight and easily overlooked in the quiescent state,

where the dilated tubes are not close to the surface (this is illustrated by experience of less debatable cases in childhood). If a careful history of such a case is taken, with the assistance of an elder relative, a clear record of bronchopneumonia, sometimes repeated, in early life is not infrequently obtained.

It has already been mentioned in dealing with bronchiectasis in childhood that blood is not infrequently coughed up, and that, occasionally, in notable amount. This may occur in adult life also, and I have known it to occur in a patient who had lost all recollection of former chest disease, but who presented a well marked basal bronchiectasis, of which his female relatives were able to supply a perfectly clear account from his early childhood. The case was, of course, under dire suspicion of pulmonary tubercle.

In addition to damage from childhood infections, the lungs of adults are also very apt to suffer scarring from the results of recovered influenzal pneumonia and of tuberculosis. Basal bronchiectasis or bronchiolectasis, not infrequently double, is not uncommonly to be seen since the recent epidemics. Sometimes it gets forgotten until some acute exacerbation brings it once more to the notice of the medical attendant. Occasionally it has been so far overlooked that some fresh disease, generally tuberculosis, comes under suspicion when new symptoms arise.

The lung damage caused by the healing of tuberculous lesions is so large and varied a subject that I shall not attempt to touch more than one aspect of it here. I would only urge that some amount of bronchiectasis is the usual accompaniment of chronic tuberculous lung disease. The point I wish to call attention to is the presence of crepitations over the diseased areas in cases of arrested or healed pulmonary tuberculosis. Such an occurrence is not uncommon, and yet one finds but little reference to it in the literature. I have many cases under observation which have been long arrested, but where crepitations remain, and might cause anxiety in the mind of an uninitiated observer. In some of these cases there are but the dry crackles of expanding lung, but in others the crepitations are somewhat moist and are due, I feel sure, to dilated bronchioles over a contracting fibroid area—generally in the region of a deep contracting cavity. The diagnosis of "arrest" may be difficult where these are present—it is greatly helped, in my experience, by the absence, in these cases, of the "reflex bands of percussion impairment" which I have described elsewhere on more than one occasion.

In conclusion, I should like to insist on the point, which perhaps I have hitherto only implied, that fibrosis and bronchiectasis, and "damaged lung" in general, vary according to the severity of the inflammation which produced the condition. It is attention to the slighter grades for which I here plead—the textbook account of bronchiectasis applies to the few marked and uncommon cases, whereas the slighter forms are not rare and are seen at all ages. They do not provide us with "pints of fetid sputum which separates into three layers," but they provide us with a varying symptomatology, and with problems in diagnosis, prognosis, and treatment, which may be of vital importance to our patients, and not without moment to our own *amour propre*, and, incidentally, our reputation.

REFERENCE.

¹ St. Bartholomew's Hospital Reports, 1905.

AN UNUSUAL CASE OF TYPHOID SPINE WITH SYMPTOMS OF SPINAL CORD AFFECTION.

BY

PROFESSOR H. TURNER,

ORTHOPAEDIC CLINIC OF THE MILITARY ACADEMY OF MEDICINE,
PETROGRAD.

NUMEROUS clinical observations and pathological research of past years have brought convincing evidence of the fact that the infective agent of so-called acute internal disease may settle on different parts of the skeleton, bringing injuries to bone, cartilage, or component parts of the joints (Virchow, Rokitsky, Ponick). It is well known that even such exclusively internal diseases as influenza may involve the osseous tissue. The "amphibious" qualities of the pneumococcus are proved by every day's observation. Its nefarious rôle in the production of osteomyelitis and arthritis has been very often the cause of diagnostic errors. The ranks of specific "surgical"

microbes, as primarily defined by Ogston, W. Cheyne, Rosenbach, and others, have been gradually enlarged by the enlistment of many other pathogenic elements, which have come forward from the domain of acute infectious diseases such as those belonging to the multifarious group of typhus—that is, typhoid, recurrent, and typhus (spotted) fever. The widespread epidemic of these latter diseases observed in Russia during recent years has given birth to vast material concerning this subject, and Russian literature, notwithstanding external difficulties, has been able to bring abundant illustration of the surgical complications of typhus fever in general. Typhoid or enteric fever, with its stubborn bacteria, capable of retaining vitality in the organism for many years after the conclusion of morbid symptoms, stood foremost in this relation, although recurrent and spotted fever did not remain much behind in showing the same quality. Clinical observations on a large scale gave us the opportunity of confirming the findings of Ebermeyer, Fränkel, Quincke, and many others, who discovered typhoid bacilli in the vertebrae, ribs, sternum, and other bones of the dead body.

The special tendency of the spine to become the seat of infection, as explained by Fränkel's demonstrations of living typhoid bacilli in the cancellous tissue of the vertebral body, we have seen represented in the numerous cases of typhoid spine which came under our observation in the orthopaedic hospital of the Medical Academy. Experience has taught us to mark off these forms of spondylitis amidst the greater number of tuberculous cases, and generally there was not much difficulty in avoiding errors of diagnosis.

The involvement of the spine in the typhoid process takes place, with great probability, during the flourishing period of the disease, and must be of not very rare occurrence. The symptoms of this complication may pass unremarked on the background of the grave picture of the illness. A new wave of temperature, accompanied by severe pains in the back, may draw attention to the intrusion of the virus into the spine. The further progress of the complication may be very slow, not taking a malignant course. It seems as if bone tissue, especially the cancellous part of it, is not a suitable pasture for the typhoid microbe, the feeble pyogenic and destructive character of which has been proved by many communications. The symptoms of the infection may in some cases subside without having been clearly understood, natural curative forces having extinguished its continuance. But as in other parts of the body, and especially in the intestinal channel, the virus of typhoid fever, secluded in the vertebrae, may retain its vitality for months and years, only to regain force under convenient circumstances, trauma in any form being the auxiliary agent.

It may be stated that the pathological anatomy of typhoid spine in the different stages of its development is not founded on such abundant data as in the case of Pott's disease. The benign course of typhoid spondylitis allows the patients to evade further clinical observation, and the final changes in the vertebrae have to be studied on occasional findings in the anatomical theatre or on the shelves of museums. The examination of these specimens, and the interpretation of the pathological changes presented by them, are hampered by the want of clinical notes. It is not easy, therefore, to define clearly the type of typhoid or other infectious spondylitis among the multifarious pathological processes to which the spine is so conspicuously liable. Roentgen rays have done very much to help our advance in this difficult field. The findings seen on the plate, elucidating the origin of clinical symptoms, present themselves as useful guides in the examination of dry pathological specimens. Taking this course of study, I have been able to discern with a great degree of positiveness the doubtful cases of infectious spondylitis compiled by me in the surgical museum attached to my clinic. The general impression received from the examination tends to confirm the supposition that the process of typhoid spondylitis is actually a form of subacute inflammation involving the periosteum, ligaments, and vertebral articulations. Gibney's term, "perispondylitis," remains the most suitable for a brief determination of the pathological changes. The posterior lateral parts of the laminae are principally involved, their mobility probably influencing the exacerbation of the process.

This point of view may help to explain the frequent localization of the illness in the lower dorsal and upper lumbar regions. Ossification of the tissues adjacent to the bone brings firm consolidation of the laminae and locking of the joints. The massive layers of newly formed bone on the