

develop in public was sufficient to excite them. This apparently capped the climax and rendered further work exceedingly difficult. As an exciting cause, auto-suggestion in the form of apprehension undoubtedly played a part, as I believe. He was in constant apprehension that when he was placed in certain positions the neurosis would strike him. As already explained in the previous cases, this is equivalent to an auto-suggestion, such as, "When I am acting under such circumstances and such a thing happens, I shall feel so and so." This thing does happen and the neurosis is exploded as an automatic process. When over-tired his condition was distinctly worse.

The treatment in this case was based upon the above analysis. The idea of neurasthenia was rejected, and the pathology as above outlined was explained at great length to the patient, who recognized its correctness and lent himself heartily to the new plan of treatment. Without this co-operation I believe this would have been a failure.

My idea was to artificially create in the patient's mind a fixed idea which should comport with the truth on the one hand, and which should be antagonistic to and therefore would inhibit the morbid idea and its consequences. To this end the following was written on a piece of paper:

"These symptoms are only physical processes which by habit have become associated together by a previous pathological mental state. This mental state having subsided, the association will subside and I have nothing to apprehend."

This was later changed to:

"I know I am the equal of the others. These symptoms are only physical processes which by habit have become associated together by a previous lack of confidence in my ability. Having now regained this confidence and knowing that I am the equal of others, this association will subside."

This he was told to learn by heart, and to repeat it constantly to himself on every possible occasion—when he was by himself, and as he walked the streets, the last thing before going to sleep, and especially before undertaking his professional work in public.

The result was decidedly favorable. The psychosis steadily grew less and in a month or two had substantially subsided. Nevertheless, there has been during the past year a tendency to relapse. But the course has been steadily towards improvement, until now his symptoms occur in so moderate a form that he no longer dwells upon them.

[Another year has passed since this was written. Although it is not easy to state with absolute accuracy the present condition of this patient, it is well within the truth to say, from my own frequent observations and the patient's own statement, that although not completely free from trouble, yet when we compare his condition with what it was, as shown by my earlier written notes, he is immensely improved. He is still troubled at times, but the attacks are mild, the secondary symptoms have disappeared, he does his professional work without particular discomfort, and his whole attitude of mind towards his trouble is different.]

Sometimes this neurosis does not occur in a pure form by itself, but forms a group of symptoms superimposed upon and complicating another disease of a different nature, perhaps true neurasthenia. For example, I have under observation now a young man who is a neurasthenic of probably a degenerate type,

and therefore in certain respects incurable. In him certain habits of mind and certain peculiarities of body can be recognized, which are undoubtedly due to degenerative processes. Besides symptoms of neurasthenia he exhibits in a very neat way this phobo-neurosis. When brought face to face with people, particularly women, though the same may happen with men, he has a peculiar set of sensations which develop in the following order: (1) a feeling of shyness or confusion; (2) the face flushes; (3) confusion of thought; (4) feeling of general weakness.

Some palpitation occurs during the attack, but its exact position in the order is uncertain. In extreme attacks, ataxia or clumsiness of gait is added to the above.

These symptoms are of such intensity as to be a matter of considerable suffering and to cause the patient to make many sacrifices for relief. They come on with a rush or suddenness which does not allow for thought, and all the circumstances under which this occurs allow of no doubt of the independent automatism of the neurosis.

AN UNUSUAL FORM OF BRONCHIECTASIS.

BY H. B. WHITNEY, M.D.,

Professor in Medicine in University of Denver; Visiting Physician to Arapahoe County Hospital, etc.

CERTAIN features of the following case will be seen to differ markedly from those commonly met with in this affection:

Wm. Mosler, age forty-four, laborer, was admitted to the County Hospital March 25, 1897. He gave the following history: At the age of twelve he had "pleurisy." At eighteen he began to suffer from asthma, which continued off and on in a mild form until twelve years ago, about which time he came to Colorado. It then ceased, and he has since been perfectly well, without cough or other evidence of his former trouble.

His present illness began three months ago as a pain in the region of the left nipple which lasted two hours and was very severe. This was followed immediately by a cough, which still continues. Expectoration was moderate at first, and for a day or two reddish in color; later it became purulent, increased in quantity up to a pint in twenty-four hours, its present amount, and almost at the outset, he thinks, became offensive in odor. He has not at any time been confined to bed. He speaks of chilly sensations at the first, but does not think he had either fever or dyspnea. He now weighs one hundred and twenty-five pounds and says his loss of flesh has been very moderate.

At the present time his chief complaint is cough, abundant fetid expectoration, soreness in the region of the left nipple, and some general debility.

The *Physical Examination* shows a man of medium size, rather poorly nourished, facies pale and somewhat phthisical in appearance. Cough almost constant, with frequent raising of a purulent and very offensive sputum, necessitating isolation. Patient is up and about; his pulse ranges from 95 to 100, respirations 22 to 28, and temperature normal to 101°, there being daily a rise of at least one degree.

The chest presents nothing abnormal on inspection or palpation. Percussion discloses a peculiar condi-

tion: Just outside the precordia, occupying the angle between its left border and the lower margin of the lung, is an area the size of an orange, which is flat in the erect posture and changes to slight tympany when the patient is on his back. Over this area there is friction. Respiration here is somewhat bronchial, and accompanied by scattered moist râles, especially for a short distance above and to the outside of the dull area. The voice sounds also bronchial, and there is distinct whispered pectoriloquy. The lungs are otherwise negative and the heart is normal. On examination the abdomen gives also negative results.

These local signs in the left lung were so plainly indicative of a cavity containing both fluid and air that the diagnosis seemed at once restricted to a very few conditions. Phthisis seemed presumptively excluded by the location, and still more by the concentration, so to speak, — the non-diffusion — of the signs; and, indeed, a later examination of the sputum for bacilli was negative. For gangrene the pain had not been sufficiently intense nor had the course of the affection been sufficiently acute and grave. The same might be said of a pulmonary abscess, although here the ground did not appear to be quite so safe. Bronchiectasis seemed hard to reconcile with the sudden origin, the absence of general bronchitis, and the limitation of the process to one side and apparently to a single cavity. The affected area lay close to the interlobar septum; and I was therefore, by exclusion, led to think that an encapsulated interlobar empyema had barely reached the surface when it had ruptured into a bronchus, become infected, and thus furnished the source of the foul pus which had since been so constantly discharged through the mouth.

On this hypothesis I made an exploratory puncture in the fifth space about one inch outside the main line, and obtained a small amount of very offensive pus. Operation was at once advised, and on the following day, in my absence, the man was anesthetized, and another exploratory puncture made as a preliminary to operation. No pus could be found, even on repeated trials, and the case was referred back to me. On the following day I again made several punctures with negative results, and the man then absolutely refused any further operative procedure.

Strange to say, he shortly afterward began to improve. He steadily gained flesh, and on October 4th his expectoration had diminished to ten ounces in twenty-four hours. Careful examination at this time failed to show any sign of the previous trouble except a small area of bronchial respiration, the size of a dollar, attached like a wart to the outer and upper segment of the precordia. In the "angle" there was no dulness, and the respiration was clear vesicular.

I did not see the patient again until January 1, 1898, three months later, and ten months after he had first come under my care. He had in the meantime continued to improve and had become a hospital "helper." Two days ago, however, while coughing, pain had suddenly appeared in the lower *right front*; and with this the amount of expectoration had suddenly increased from four to sixteen ounces. Physical examination of the chest now disclosed the following condition: *Left Front*:—No remains of the original trouble except slightly broncho-vesicular respiration at site of former pericardial excrescence. *Left Back*:—Very few moist râles in mid-scapular region. *Right Lung*:—Numerous moist râles through lower third,

front and back. Friction in region below right nipple.

One month later the physical signs had become concentrated about the lower right front and axilla, and bore a marked resemblance to those originally found on the left. There was a zone of flatness about two inches wide, continuous with that of the liver and bounded above by a slightly curved line extending from near the nipple to the line of the angle of the scapula. Over this zone respiration was distinctly bronchial — almost amphoric — and there was distant whispered pectoriloquy. Vocal fremitus was diminished but not absent. There was no tympany, and hence, of course, none of the Wintrich signs of cavity. For a short distance above the flat area there was slight dulness and a few moist râles. At least the upper half of the lung was absolutely negative.

These signs remained unaltered until the man's death, from a pulmonary hemorrhage, on May 30, 1898, fourteen months after he entered the hospital. The only local change which occurred was a very slight increase of the flatness upward and toward the spine. The heart had continued practically normal; and there was never any enlargement or downward displacement of the liver. The general condition had shown a steady decline from the time, in January, when the signs in the right lung had first been discovered. There were no chills, but an irregular though steady hectic, together with the constant drain from the lungs, had produced a very considerable degree of weakness and emaciation when the final catastrophe came.

On two different occasions several exploratory punctures were made over the flat area with negative results.

Before stating the results of autopsy, it is to be remarked that the difficulties of diagnosis in this case were very great. It was, of course, quite evident with the right lung, as earlier with the left, that we had to deal with some sort of cavity or cavities communicating with the bronchi. But what sort of a hypothesis was consistent with an apparent cure of the condition which had first appeared on the left? The original thought of interlobar empyema, or possibly pulmonary abscess, seemed inadequate to explain a secondary and similar involvement of the right lung; experience tells us that such does not occur. Hydatid was considered, but the condition is extremely rare; there was but little pain and even the slightest positive evidence was wanting. Bronchiectasis, while not dismissed from consideration, was very difficult to reconcile with the history of acute onset, the relative recovery of the part first affected, and the subsequent invasion of the right lung.

Autopsy disclosed the following pulmonary conditions: In the lower part of the interlobar fissure of the left lung was a small quantity of cicatricial tissue, evidently the result of a former pleurisy. Following this up led to a cavity, situated close to the fissure in the lower part of the upper lobe and about as large as a small egg. It was collapsed, wholly empty, and communicated directly with a somewhat dilated bronchus. Its walls appeared to be extremely thin and were uninfamed. With the exception, perhaps, of a very slight injection of the bronchus leading to this cavity, the left lung was normal.

In the right lung the changes were more recent and profuse. The upper half was negative except a small isolated cavity, the size of a walnut, lined with a thick

membrane and containing pus. The lower half, on the contrary, was riddled with cavities varying in size from a chestnut to a small egg. The shape of these cavities was in the main saccular, and many of them had a wide communication with a bronchus. In a general way they resembled the cavity found upon the left, except that the walls were more inflamed, and they contained usually a considerable quantity of offensive, muco-purulent secretion. The surrounding lung tissue was comparatively uninvaded; the absence of tuberculous or cheesy masses was especially noted, although there was, perhaps, around some of the cavities a slight pneumonic infiltration. In a general way the cavities presented the peculiar appearance of being scooped out of comparatively healthy lung. They were most numerous and largest at the extreme base in front — in the middle lobe — corresponding to the situation *intra vitam* of the most prominent physical signs. Throughout the affected portion there was a considerable degree of acute bronchitis without marked chronic changes in the thickness of the bronchial walls. The heart and other organs were relatively normal.

Let us attempt a brief analysis of this case, which is exceedingly instructive with reference to the clinical history of bronchiectasis. It certainly discloses features of this disease which differ decidedly from those generally accepted, and it is here to be especially emphasized, as being of the greatest importance in the interpretation of the case, that the history of onset given above is to be accepted as reliable. The patient was a man of more than ordinary intelligence, and the history was taken first by the interne and later, with great care, by myself. A persistent cross-questioning failed to alter in any way the statements originally made.

We find, then, an acute onset of pain, cough, and abundant fetid expectoration in a man previously well, and — what is particularly to be noted — absolutely free from any cough whatsoever. Signs of a cavity soon develop in a circumscribed region of the left chest, persist for two or three months, and then gradually disappear; at the same time there is a marked general tendency toward recovery, and a great diminution in cough and expectoration. Several months later the right side suddenly becomes affected in a similar way, the old symptoms return, expectoration again becomes exceedingly fetid and abundant, and after three or four months of sepsis with steadily increasing exhaustion, the case terminates fatally by a pulmonary hemorrhage. Corresponding to this clinical course the post-mortem shows a large quiescent bronchiectasis on the left, and on the right numerous bronchial dilatations in a state of active catarrhal inflammation.

Assuredly these bronchiectases could not have originated in a few days, or even a few weeks. They must have been of long standing and slow development, already at least partially formed at the time of the original acute attack. This latter I conceive to have been an acute inflammatory process, involving the pre-existing bronchiectatic cavity, and particularly the surrounding pulmonary tissue. Hence the pain and reddish sputum at the outset, the pleuritic friction, and the suddenly increased activity of bronchial secretion. An explanation, also, of the abrupt development of cavity signs is not far to seek. A collapsed cavity in normal lung tissue may give little evidence of its presence; the same cavity dilated by abundant

secretion, surrounded by a consolidated lung, and perhaps thus provided with smooth and rigid walls, will present conditions favoring the production and transmission of abnormal sounds.

This inflammatory process then gradually subsided. The cough and expectoration diminished, the local signs disappeared, and the *status quo ante* was again restored. Unfortunately, however, the same process was repeated in larger measure in pre-formed bronchial dilatations of the opposite side, and an accidental hemorrhage produced the fatal result which otherwise would probably have occurred later from sepsis alone.

The chief lessons to be drawn from this case are, first, that bronchiectasis may be wholly latent, and second, that the symptoms which it often produces — chiefly cough and abundant fetid expectoration — are susceptible of spontaneous cure.

In the first place as regards latency: The conception of the bronchiectatic process as latent, and hence essentially primary, is certainly not a commonly accepted one. We usually think of it as either obstructive, where there is some local mechanical obstruction of one or more large bronchi; or tractional, as in a fibroid lung; or inflammatory, as where, from a long-continued bronchitis, the bronchial walls become gradually relaxed and eventually distended. A primary, slow, relatively benign, and wholly latent development of saccular dilatations of the bronchi in the midst of healthy lung tissue strikes one at first as a strange idea.

It is, however, not new. In Vol. VI of the "Twentieth Century Practice of Medicine" may be found an admirable description of primary bronchiectasis, by Stewart and Gibson, together with a complete historical review of the subject. It is there shown that Briquet, Dittrich and Barlow had observed apparently primary bronchial dilatations, of saccular character, previous to a more complete study of this form by T. Grainger Stewart in 1867. Stewart then maintained and still holds the opinion that these cases of bronchiectasis are often the result of a *primary atrophy* of the bronchial walls. Leroy, also writing in 1879, and apparently unacquainted with the article by Stewart, advanced the same view. The cause of the atrophy itself is admittedly obscure, and is referred to in a general way as a constitutional peculiarity.

A few quotations from Stewart and Gibson concerning the clinical history of these primary cases will be of interest: "In many, if not all, of the cases the affection comes on insidiously, no symptoms appearing to attract the attention of the patient or his friends perhaps for long after the dilatations have been formed. But gradually or suddenly inflammation of the bronchial mucous membrane is lighted up." After decomposition of the secretion, "The patient's history is now one of intermittent cough, with fetid sputum and breath. He becomes liable to severe febrile disturbances due to a septicemia from absorption of the products of decomposition, which attacks may be recovered from under treatment, or may prove fatal, or the decomposition of the secretion may be followed by inflammation and consolidation of the lung — a septic pneumonia; sometimes also by ulceration, abscess, or gangrene." "Sometimes death results from exhaustion induced by the constant discharge of sputum." "Barth met with two cases in which hemoptysis proved fatal, and we have met with similar cases."

In the case reported by the writer it is of course possible that the atrophic dilatations of the bronchi had some relation to the preceding history of asthma. It is, however, to be considered, that the patient had suffered neither with asthma nor bronchitis for twelve years previous to the acute onset of his present disease.

As regards the second point of interest — the spontaneous recovery from the first attack — I can conceive no other explanation of the marked improvement in general condition and the great diminution of cough and expectoration which took place, together with the recession of local signs, and particularly the post-mortem appearance of the bronchiectatic cavity. The latter was so collapsed, and the surrounding pulmonary tissue was in so normal a condition, that, much to my chagrin, the lung, after numerous sections, was pronounced negative. Confident however, that there must be something to show for the pronounced physical signs which I had heard, I followed up the septum and finally came upon the collapsed cavity described in the report.

The rarity of any spontaneous cure of bronchiectasis must be very great. Guitrac (quoted by Stewart and Gibson) speaks of two ways in which cure has been known to take place: one by cretification of contents, the other by discharge through the thoracic wall. Stewart and Gibson, however, have never met with any such cases, and regard recovery as extremely infrequent. My own case would seem to indicate that nature does work in the direction of at least relative recovery, and this must be regarded as very encouraging toward therapeutic efforts. The chief special medication in the case reported was large doses of the hyposulphite of soda, of which two drachms to half an ounce was taken daily, considerably diluted in syrup and water. There was certainly an almost immediate effect in diminishing the odor of the sputum; indeed during the first attack it entirely disappeared. Whether or not this treatment had anything to do with the temporary recovery is questionable.

TUBERCULAR CYSTITIS IN CHILDREN.¹

BY CHARLES GREENE CUMSTON, B.M.S., M.D.,

Assistant Professor of Surgical Pathology, Tufts College Medical School; Corresponding Member of the Association of Genito-Urinary Surgeons of France, of the Obstetrical and Gynecological Society of Paris, etc.

ALTHOUGH tubercular cystitis is more frequently met with in young adults, a number of cases have been reported in children during the past few years. The genito-urinary organs in children being in a rudimentary state of development are invaded with difficulty by the infectious agent of tuberculosis, but this is not the only reason for the infrequency of reported cases. The symptomatology of tubercular cystitis in children is often so masked that the attention of the physician is not directed to the condition of the bladder, and I am of the opinion that if microscopical and bacteriological examination of the urine of children were more frequently resorted to urinary tuberculosis would be more often discovered in little patients.

All the causes attributed for the development of vesical tuberculosis in the adult may be applied to the child, such as acute infectious cystitis, the extension of an infection of the genital organs to the urinary

tract, and explain why tuberculosis of the bladder is infrequent in children.

In young subjects, as is the case with adults, tubercular cystitis is either primary or secondary. When secondary it is more particularly due to tubercular lesions in the lung, but it may develop from a coxitis or adenitis. The primary form is seen in children born of apparently healthy parents, and its appearance is in the form of urinary disturbances that are often very misleading.

Our personal experience with tubercular infection of the bladder in children is limited to a single case which is here briefly reported.

Miss M. G., age eleven, was seen in October, 1896. She was the only child, born of healthy parents, both being alive, and had always enjoyed the best of hygienic surroundings. As a little child the patient had always been well excepting for measles at the age of four, and scarlet fever at seven. In neither of these affections had there been any renal or vesical complications, and the catheter had never been used. For the four months previous to our first visit the patient complained of a frequent desire to micturate, and this increased to such an extent that at our first visit she was going to the bath-room about every fifteen minutes. During the last six weeks the desire to pass water was complicated by a burning sensation at the time of micturition and following the act, and so intense that the child would cry out with pain. Strange to say, sleep was fairly good; every two or three hours there was a demand to empty the bladder. At the same time the appetite diminished and a yellow leucorrhœa appeared. The bowels were regular. Exercise seemed to increase the pain, but riding appeared to have little effect. The tongue was clean, the pulse was 80, and at no time did the thermometer register above 37° C.

Examination of the thoracic and abdominal viscera was negative; the child did not cough; there were no night sweats, and there had never been a rhinitis nor discharge from the ears. No enlarged lymphatic glands could be found, and a careful inspection of the throat showed that the tonsils and pharynx were in normal condition.

On account of the severe bladder symptoms we decided to examine the bladder and kidneys. Ether was given and after complete narcosis the abdomen was carefully explored, but no enlarged spleen or kidneys could be felt. The bladder could be palpated low down behind the pubis and its walls appeared rather thickened. A small Kelly's bladder speculum was then introduced. The trigonum was very hyperemic, the ureteral orifices were normal. Two small and superficial ulcerations, each about the size of a split pea, were seen; their borders were rather irregular, and they had a dirty yellow surface. The fundus of the bladder was somewhat hyperemic, although not very much so, and along the course of some of the vessels a few small pearly-colored points were present, having all the characters of tubercles. The urine was cloudy and slightly acid in reaction when voided, and muco-purulent deposit settled at the bottom of the tube after standing. This deposit was stained for tubercle bacilli and a few were found without much difficulty.

Four different samples of urine removed by a sterilized glass catheter into sterile tubes were inoculated into guinea-pigs; all four animals presented a well-

¹ Read by invitation before the Danbury, Conn., Medical Society, November 2, 1898.