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THE PRODUCTION OF BRONCHIAL BREATHING IN CONSOLIDATION AND OTHER CONDITIONS OF THE LUNGS.

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THE PRODUCTION OF BRONCHIAL BREATHING IN CONSOLIDATION AND OTHER CONDITIONS OF THE LUNGS.[1]

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THE title of this paper is perhaps subject to misconception. It includes the discussion of the physical principles involved in the production of tubular breathing, and suggests that these same principles induce the broncho-vesicular and cavernous breathing, which differ from the first only in degree. There has been some little controversy upon this subject, limited, however, largely to those authors that have practical minds and a fancy for acoustics. Most of our modern text-books contain descriptions of the sound, and account for its production by quoting one of the few authorities that have written on the subject. The explanation offered by Laennec, and still held by some, was that the sound was produced by the vesicular murmur being cut off and the tubular or bronchial sound alone being audible. In the fourth edition of his work he expresses himself as follows: "The sound observed in the larynx, trachea, and larger bronchial trunks is due to the

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absence of that slight crepitation that accompanies the dilatation of the air-cells of the lungs, and the idea of a drier sound is suggested to us. It is strong in most parts of the neck. When the texture of the lungs becomes indurated or condensed from any cause, such as pleuritic effusion or the changes occasioned by a severe peripneumony or hymophysis, the vesicular respiration having then disappeared, or being much lessened, we can frequently perceive distinctly the bronchial respiration, not only in the large, but even in the small ramifications of the bronchi. The cause of this bronchial respiration appears to me very obvious; in fact, when the air is prevented from penetrating the cells this is the only kind of respiration that can exist, and is louder and more distinct in proportion as the lung is more condensed and thereby becomes a better conductor of sound."

Dance, a French authority of later date, adopts virtually the views of Laennec, and states in the *Dictionnaire de Médecine, ou Répertoire Générale des Sciences Médicales*, of 1833, that bronchial breathing is present when the air passes through the bronchial tubes without reaching the pulmonary cells. It produces in the ear a sensation of columns of air winding in the tubes of a certain caliber, without the sound of vesicular expansion; sometimes the sound is so intense as to make it seem that the patient's breath is passing through the stethoscope instead of through the bronchial passages.

Skoda thought that the tubular breathing was the transmitted laryngeal sound intensified or otherwise altered through what he called consonance; but he does not use this word in its ordinary acoustic

sense, but explains that he applies it with the compound meaning of consonance and resonance, applying the principles of the sounding-board to obtain the intensity that he suggests. He does not make his views clear, except so far as the principle is concerned. The following is a translation of what is written in his *Abhandlung über Perkussion und Auskultation:* Bronchial breathing can be higher, stronger, deeper, or weaker, or even just as high and as strong as laryngeal breathing, and is due to consonance in sound in the upper and lower portions of the air-passages; the difference in volume and pitch depends on the condition of the inner lining of the air-passages in admitting of the production of the consonant sounds.

The most elaborate treatment of this subject that has come to my notice is found in Flint on the *Respiratory Organs.* Here Austin Flint devotes considerable space to this subject, and concludes by regarding bronchial respiration as "consisting of transmitted sounds that are produced within the trachea and primary bronchi, and probably also within the subdivisions of the latter, and are conducted by the air in the tubes and the consolidated lung to the ear of the auscultator. In what proportion they are due to the trachea and the larger bronchi exterior to the lungs, and to what extent sounds generated within the pulmonary bronchial subdivisions may be combined, are points not easily determined."

Flint certainly had an analytic mind, and his description is the most satisfactory. More modern writers adopt this view without notable modification.

Guttman devotes fair consideration to the opinions of others, but expresses his views as follows: "The bronchial respiratory murmur heard in disease is nothing more than the laryngeal sound carried with scarcely diminished intensity through consolidated lung-tissue." The views of R. C. M. Page and Vierordt are virtually the same. So also are those of S. S. Burt, excepting that the latter speaks of the sound as being transmitted through the bronchial tubes as through a speaking-tube, directly to the surface and ear of the auscultator. It will be observed that there is no great variety of opinion, and, without quoting at greater length, authorities are not fully satisfied with their own explanations, for they quite universally acknowledge differences of pitch over different regions in the same individual, and even contrast the laryngeal and tubular pitch, pointing out differences that all will concede could not exist if the source of the sound were the same, for pitch cannot be altered by the medium of transmission, and, as Dr. Flint says, "there are circumstances which give rise to variations in pitch which we do not understand, and that the sounds generated within the trachea and bronchi as in conditions of health, will not suffice to explain the intensification of sound which sometimes characterizes bronchial respiration in disease, nor the disparity in pitch which is observed." He further concludes that "other physical influences are doubtless involved which are not as yet satisfactorily explained."

Dr. J. Bloodgood, of Chicago, published an article on this subject in 1860 which has not received the consideration that it deserves, and my

attention was only called to it within the past few days. In this article it is claimed that hepatization is not necessary for the production of tubular breathing and that the normal lung-tissue offers a good medium for conducting sound, as demonstrated by the clearness with which sibilant râles are heard when the parenchyma is normal; that when a bronchus becomes temporarily blocked by viscid mucus a clear tubular sound is at once detected, and this disappears when the secretion is coughed up; during the time it is heard the lungs are normal. Dr. Bloodgood argues from this that the air in its passage downward is diverted into many tubes the aggregate capacity of which is much greater than that of the trachea and bronchi; that it therefore rushes more and more slowly the more deeply it penetrates; that should a small bronchus become plugged at a little distance from its origin, or lead to cells filled with inflammatory product, it would furnish a cavity analogous to the Pandean pipe, and yield a tone determined by its size; that if it were located high up in the lungs, where the air has the greatest velocity, it would be intense, but if located in the lower lobes the sound would not be induced, or if present the intensity would be diminished. His argument is based upon false anatomic grounds, and he does not accord the transmitted laryngo-tracheal sound its place.

I began, about three years ago, a line of work and experimentation having the great charm called original work, which always acts as a wholesome spur, and after eighteen months of time it developed most successful results; but in that time I also made

the discovery that my undertaking was not new, though I had succeeded without the knowledge that the ground had already been staked out by others. The work referred to is the making of casts of the air passages, and for that purpose I used an alloy composed of—tin, 4 parts, lead 8, bismuth 15, and cadmium 3. This alloy softens at 60° Centigrade, and fuses at 65°.

I do not know how my castings may compare with those made by others whose opportunities may have been better than mine, and who may have purused their work for a longer period than I did, for I have never seen any of the kind; my object is only to illustrate the basis of the explanation here advanced as to the causes that contribute to the production of the sound that we all term tubular or bronchial breathing. The castings are made from healthy lungs. These should be procured as soon after death as possible, and the cast is formed by pouring the alloy of low fusion into the trachea and allowing it to penetrate as far as possible into the tubes and vesicles before it chills and consolidates. It hardens almost immediately into a solder-like form, and the lung is then macerated in caustic soda, and eaten off, leaving the casting, representing what must be the exact caliber and shape of the air-passages, as far as the metal has penetrated, without burning or destroying the pulmonary tissue. I need not remind you that success does not attend each attempt, that the degree of heat, the pressure and the presence of moisture will modify the result and act as factors in the frequent failures, and determine the degree of success. In most instances castings

of the larger bronchial tubes were obtained; in some, of the most minute tubular subdivisions; and, in a few, of the surrounding vesicles.

Whenever the vesicular castings were obtained the tubes were of course hidden from view. The castings were also not complete throughout the lung, and were often unavoidably broken off in the preparation, even by their own weight. Human lungs do not prove good specimens unless procured from subjects dying from hemorrhage and after a short illness, as the mucus always present debars the possibility of a perfect cast. The preparations were made from adult and infant human lungs, and the lungs of sheep, calves, dogs and rabbits. In many cases the alloy was poured into the trachea while the lungs were still *in situ*.

It is not within the province of this paper to discuss the anatomy of the lungs or to point out the errors that exist in some of our best descriptive Anatomies; but I particularly invite attention to the method of division or branching of the bronchial tubes, which quite uniformly takes place at about the same angle, and uniformly by bifurcation, and that the air only reaches the air-cells after passing through a long route and varying in its course at the different points of bifurcation. Furthermore, at these points the bronchial tube possesses a sharp edge, which acts as a prow to cut the column of air at the bifurcation. This cutting edge is described in Sappey's *Descriptive Anatomy*, and he says that on a level with each division one sees in the cavity of the bronchial tubes a spur or prow to cut the column of inspired air. If the bronchi are given off

at an acute angle this spur is very pronounced and very sharp. In collapsed lungs the bronchi are naturally much diminished in size, and this cutting edge is not very apparent, as it is not thrown out in relief; but in consolidated lungs, as in pneumonia, it is readily seen. I am confident that bronchial breathing is a mixed sound, composed of laryngo-tracheal and cavernous elements, the laryngo-tracheal sound being transmitted through the bronchial tubes by the air in their cavities, and thence communicated to the consolidated or compressed lung, to the chest-wall, and heard by the auscultator, diminished in intensity according to the lack of transmitting power of the different media through which it has to pass; that added to this sound in proportions differing under different circumstances, but often predominating in volume, is a *tubular blow* that is produced by the air rushing over the open and cavernous mouth of a patulous bronchus that leads to a lung not receiving its quantum of inrushing air.

Now, bearing in mind the cavities represented by these bronchial tubes, either of the first, second or third magnitude, the air-cutting edge referred to by Sappey, at the point of bifurcation, I believe we have the explanation needed for variations in pitch, as well as one of the contributing elements in inducing tubular breathing, and, perchance, the same tubular blow will be induced, whether the air is passing in or out of the lungs.

This point can be demonstrated by five figures of a purely conventional character, to illustrate how tubular breathing can be produced in the higher

grades of pneumonia with consolidation; in pleurisy, with effusion, when the lung is in contact with the chest-wall; in necrosis, with a cavity; and how, when produced, it could not be heard in cases of effusion, if fluid intervened between the lung and the chest-wall. In all of these cases it is apparent that over areas in which tubular breathing is heard there is diminished or suppressed vesicular expansion. Under these circumstances it can hardly be questioned that, in order to reach other portions of the lungs, the air rushes by and over the bronchus leading to the diseased portion, to fill the air-vesicles that do expand, and that at the point of departure the cutting edge described by Sappey must produce a reverberation, engendering a sound analogous to that which would be produced by blowing over a test-tube; and its pitch will depend upon the length and caliber of this bronchus; this is the additional sound that, for lack of a better term, I have called the *bronchial blow*. If the rush of air to other parts of the lungs is rapid, the intensity of this sound will be augmented. If the size of the tube be small and shallow, its pitch will be raised; if large and deep, the pitch relatively lowered; but in any case, higher than a laryngo-tracheal sound, except in conditions of disease of the larynx, in which case its pitch might be raised much above the normal. When this sound is intense, as we find it in the rapid breathing of acute pneumonia, or extensive effusion, with the lung pressed or bound to the thoracic wall, it contributes largely to the sounds heard, thus raising the pitch and augmenting the intensity. Should rapid

breathing subside in the cases named, or should secretion obstruct the mouth of the tube, or fill its cavity, then the intensity of this element would diminish, or the sound entirely disappear. The pitch of this blowing sound is determined, as has just been stated, by acoustic laws and depends upon the length of the bronchus and its caliber at its point of departure. The principle is susceptible of ready demonstration, with apparatus to be found in any of our offices. Blowing over a small test-tube will yield a sound of a certain pitch; on filling the tube partially with water, the pitch will be raised; a long, narrow tube will yield a low-pitched sound; a flask may be treated in the same way to illustrate the reverberation in a cavity. This experiment may serve to explain how, after a short lapse of time, both pitch and intensity of sound may be altered in conditions of consolidation by the presence of secretion in the tubes. I would further offer as an explanation of the exaggeration of the tubular element heard over cavities, particularly when they are near the surface, that the intensity is very much augmented by the proximity of the sound to the ear of the auscultator, and this rule applies equally to all physical sounds, and the more remote from the point of its production, the less audible it becomes; it is for this reason that sounds are sometimes limited to very small areas, particularly when the medium of transmission is poor. Again, sounds produced externally to the larynx cannot be heard with distinctness, and those which are most distinctly heard are produced at the periphery of the air-passages, or near the ear of the

auscultator; labial sounds, such as chirping and whistling, are only heard as transmitted by the external air; they are never intense, whereas any artificial means that produce a rush of air in the respiratory tract nearer the ear are markedly exaggerated, as, for instance, the expiratory "puffs," produced in this way; whisperings are intensified in like manner; the intensity of sound at different periods of respiration depends upon the same law. I do not think it would be possible to state what proportion of the sound is attributable to the different elements that go to make it up, for I am convinced that the proportion varies notably with different degrees of consolidation and under varying conditions, the discussion of which would be foreign to my purpose here. It seems to me that the reason why this explanation has not been offered before is because the size of the tubes has been very much underrated by those who are in the habit of making physical examinations; commonly when examined the lungs are collapsed and the bronchi are diminished in size, but the castings show that there are fully sixty tubes in the human lung capable of inducing a bronchial, blowing sound, while several bronchial casts in distended lungs have a diameter of about half an inch in the clear; these latter would certainly yield a large volume of sound.

Before closing I would not be understood as saying that the larynx does not play a rôle, and a very important one, in determining the pitch and quality of bronchial breathing, for I have experimented upon some of the lower animals, inducing pulmonary consolidation, and with an opening into

the trachea I have been able to cut off the laryngeal sound at will; from this experiment, although it was not complete, for I found it difficult to produce pneumonia in a dog at will, I was convinced that the larynx, as well as the trachea and the *bronchial blow*, are the three elements that, in the aggregate, induce the sound that has been the subject of this paper.