Thebesii. The wide mouths of these various vessels open and become filled thoroughly with blood and then, by a secondary capillary circulation through these vessels, the heart muscle is supplied with fresh blood. I do not doubt that there are many cases found at autopsy with advanced degeneration of the coronary arteries showing that digitalis given alone would be useless, perhaps injurious. Then, again, such an appearance might controvert the idea that digitalis is useless, for the benefit the patient has received may be due to the influence of the drug on the heart muscle carried there through the veins of Thebesii. I feel quite convinced that the blood so carried to the heart will readily part with its oxygen and nutritive material. I have always felt that the subject of the use of digitalis was an important one because of the grave condition of patient. I am convinced that many physicians do not have a sufficient regard for the pathology of the heart, i. e., the morbid physiology of the heart. I think this is one of the faults of the medical course of to-day; students are taught to listen to diseased hearts, but little is taught them about the proper function of the organ; they are told what happens in a normal heart in its normal cycle, but not informed as to what happens to the heart when it is diseased, or if the valves are destroyed. They are not taught enough about the employment of the different stimulants on such a heart. I am convinced that physicians who understand the use of chloroform and ether, who use chloroform in some cases and ether in others, nevertheless treat every case of valvular disease of the heart with digitalis as a routine method. When administering this drug one should first study the condition of the heart muscle and its valves and arteries.

THE ORIGIN OF THE VESICULAR RESPIRATORY SOUND.*

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The origin of vesicular breathing was attributed by Laennec to friction of the inspired air in the bronchioles and its entrance to the infundibula from the bronchioles. This inflammation was sanctioned by such masters as Skoda and Wintuch and remained unquestioned, so far as I am able to learn, until Baas attempted to explain on purely acoustic grounds the improbability of any audible sound originating in tubes the size of the bronchioles from the passage of a current of air with the slow velocity which must attend the entrance of inspired air in the vesicles of the lung. Baas' article published in the Deutsche Arch. für Klin. Medicin, vol. xix, 1876, is a profoundly thoughtful and logical discussion of the subject which was well calculated to carry conviction to the mind of the reader. I was greatly impressed with the acute reasoning of the author the first time I read the article several years ago, but in the light of recent clinical experience and the attempt to confirm some of Baas' observations, I am reminded of the voluminous monograph on the habits of the camel, the contents of which emanated from the inner consciousness of the author.

Penzoldt later offered experimental evidence to sustain the logic of Baas. The experiments of Penzoldt were very simple. They consisted in inflating a calf's lung and placing it over the trachea of a man who is instructed to breathe in the ordinary way. If a stethoscope be placed on the inflated lung the observer will perceive (according to Penzoldt) the tracheal breathing transformed to the vesicular type of respiration. This is accounted for by the refraction and partial reflection of sound waves which must occur during their transmission through such heterogeneous media as are presented in the inflated lung. If a more homogeneous medium be used, such as the calf's liver (according to Penzoldt) the tracheal breathing retains its so-called bronchial character. Sahli tried the experiment as described by Penzoldt but found the respiratory murmur transmitted through the inflated lung retained its bronchial character. The only modification, according to Sahli is a diminution in intensity of the respiratory sound.

My own observations confirm those made by Sahli. In repeating this experiment it seemed to me that only a vigorous use of the imagination could transform the audible sound into a vesicular murmur or anything approximating it. Vesicular breathing and bronchial breathing have several essential differences which will not admit of the transition of the former to the latter on the acoustic grounds cited by Baas. The terms, sound and murmur, are differentiating terms which are descriptive of two phenomena. Bronchial respiration gives a clearly definite, and simple sound which can be assigned a certain pitch. Vesicular respiration gives a confused mingling of sounds which suggest a multiplicity of points of origin. The pitch is much lower and the expiratory sound is much shorter in duration than in bronchial breathing. However remote and faint bronchial breathing may be, the duration of the expiratory sound remains the same, whereas, in vesicular breathing the expiratory sound is relatively short, whatever may be its intensity or proximity.

The observations on the case I am about to report offer absolute proof of vesicular breathing having an origin different from that of bronchial breathing:

I was called one evening to see a man 82 years old who, during the same day, had developed a violent hiccough. The patient had a temperature of 101 degrees. The base of the left lung posteriorly gave a high-pitched tympanitic percussion note with increased resistance. Tactile fremitus was not increased. During inspiration were an abundance of medium and fine moist râles. The respiratory sounds over the affected area was slightly higher in pitch, but unfortunately for the completeness of my observations, the sound had not acquired a bronchial character. The hiccough presented a very striking picture, and would recur about every half-minute during a period varying from one-half to two hours, and then disappear to return after an interval of three or four hours. So, the hiccough was present about four to six hours in every day. The patient laid 14 days in this condition until death came in the form of vagus exhaustion, so there was ample opportunity to study the details of the hiccough. The hiccough occurred frequently during sleep without waking the patient. Each attack commenced with a moderately loud laryngeal sound and was followed directly by from ten to twenty violent retractions of both hypochondria at the rate of about two to a second. The hypochondria fairly flopped, so that it strongly suggested the flopping of the wings of a barnyard fowl. The spasms of the diaphragm occurred in such rapid succession that a complete evolution of the diaphragm phenomena was not discernible, though the band of retraction over the pleural sinus was plainly visible. The contraction of the diaphragm lasted so short a time, the descent of the lung was so slight, it could not be demonstrable by percussion, though on occasions it was slightly perceptible to the eye. Auscultation during the attacks revealed a loud respiratory murmur everywhere over the lungs, over the apices, as well as the bases, which though short in duration plainly gave the characteristics of vesicular breathing. The duration of the sound during the relaxation of the diaphragm sustained the same relative duration to the sound during phrenic contraction which the expiratory sound sustains to the inspiratory sound during normal respiration. Over the infiltrated lung area râles could be distinctly heard during the contraction of the diaphragm, though not with the same intensity as with forced inspiration. If the stethos-

* Read at the Fifty-third Annual Meeting of the American Medical Association, in the Section on Practice of Medicine, and approved for publication by the Executive Committee: Drs. Frank A. Jones, George Dock and J. M. Anders.

cope was placed over the larynx or trachea during the spasms of the diaphragm there was no sound audible. It was perfectly clear that with the onset of each series of phrenic contractions the glottis was tightly closed. That there was movement of the air contained in the respiratory tract toward the infundibula is proven by the occurrence of râles over the pneumonic area, but this was accomplished by rarification of air in the trachea and larger bronchi.

The observation offers absolute proof of the existence of a true vesicular respiratory sound. Whether the sound is due to the disturbance of equilibrium of the lung tissue or to motion of the contained air, or both, is not determined, and I see no possible method of differentiating between these two factors as they are both essential premises for any clinical or experimental observations which can be made. In this case laryngeal and tracheal breathing were eliminated, there was an increase of the antero-posterior diameter of the thorax with each spasm of the diaphragm. All the muscles employed in inspiration were brought violentlv into action. The volume of the thorax was increased with each spasm of the inspiratory muscles by rarifying the air contained in the respiratory tract, and still the typical murmur of vesicular breathing was audible.

The nearest approach to these conditions I have found was in the case of fissured sternum, reported by Sahli. But in Sahli's case tracheal breathing was not eliminated. It would be impossible to reproduce the conditions of this clinical observation by any animal experiment. I can not conceive of such violent inspiratory efforts being produced by stimulation of the phrenic nerves alone. Since making the above clinical observation I have made the following experiment: On a healthy man, have the subject close the glottis and then make a violent inspiratory effort. At the same time auscult the lungs and you will hear a faint vesicular respiratory sound during the inspiratory effort and a faint expiratory vesicular murmur as the muscles are relaxed, though the glottis remain shut. The sound heard during relaxation of the inspiratory muscles is lowered in pitch and short in duration. The two sounds are identical in their character and their relations with the normal vesicular respiratory murmur. The sounds produced in this manner by a strong man do not, however, approach the intensity of the sounds produced by the old man with the pneumonia.

REPEATED SMALL HEMORRHAGES AS A CAUSE OF SEVERE ANEMIA.*

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That recovery may rapidly follow a severe acute hemorrhage is well known. Even repeated severe hemorrhages may cause no permanent deviation from health, if the intervals between hemorrhages be sufficiently long to permit regeneration of blood to take place. The body may also stand repeated small losses of blood at shorter, even daily, intervals, with little or no perceptible change in the blood or in the performance of function on the part of any organ. Repeated hemorrhages from the nose, from hemorrhoids, from uterine fibroids may thus be endured for months or for years, the tolerance of the individual to the loss of blood and his power of blood regeneration being sufficient to withstand and make up for the daily drain. But in other instances, the daily loss of small amounts of blood produces the most severe and even fatal forms of anemia. It is my purpose in this brief paper to call attention to the importance of recognizing this form of anemia; and I treat the question from the practical and clinical, rather than from the hematologic point of view. My attention has been called to this subject by having seen five patients with bleeding piles in whom there developed a most grave secondary anemia. And I am led to write on this topic because in all of these cases the cause was for a long time overlooked and because in three of them even when the nature of the anemia had been pointed out, there was a disinclination on the part of some to regard so common and so insignificant a condition as bleeding piles as the cause of an anemia so profound and so clearly threatening life.

CASE 1.—The first case I saw while acting as assistant to the late Charles Warrington Earle. After trying in vain by iron, arsenic, tonics, rest, etc., to restore color, strength and health to a previously robust man, Dr. Earle decided to operate on the bleeding hemorrhoids. The result fully justified his suspicions as to the nature of the anemia, for the man made an uninterrupted recovery within a few weeks.

CASE 2.—The second case was the wife of a physician. She presented the typical appearance, symptoms and blood findings of a severe chlorosis. For weeks she had been losing blood with every bowel movement, often several ounces at a time. Iron and other remedies were without perceptible effect. The immediate operation that I advised was deferred because of the weak condition of the patient, a slight temperature and a loudness of the hemic murmur which made the surgeon feel that there was an organic heart lesion that might account for the anemia and that contra-indicated an anesthetic. Several months later when the patient could scarcely walk across the floor without fainting, and when my second examination confirmed the previous findings of a grave secondary anemia, most urgent advice was again given as to operation. The hemorrhoids were attended to, the anemia disappeared and the patient has ever since been in perfect health.

CASE 3.—A third patient, an old man in the sixties, had an appearance suggestive of malignant growth. Yet there could be found for his profound anemia of the chlorotic type, no tumor, tuberculosis, nephritis or other cause than some hemorrhoids that had bled daily for nearly three years. He refused an operation and was lost sight of. His blood on May 18, 1901, the date of the first examination, showed 2,450,000 red corpuscles, 6000 whites (74 per cent. polymorphonuclear), no nucleated reds, hemoglobin 29 per cent. The cells were smaller than normal, volume index .72, the time of coagulation was four minutes.

CASE 4.—The fourth case which I report more in detail concerns A. K., a Bohemian of 39. His family history was negative and he himself had pursued his occupation as a butcher, annoyed by no illness up to two years before the time I first saw him, the fall of 1901. In 1899 he had articular rheumatism and was kept from work for several weeks. From this he recovered. In September, 1900, he noticed that he was passing blood at stool. The amount was often several ounces, frequently a half teacupful being passed. He grew pale, weak, dyspneic, dizzy and by May, 1901, could barely keep at work. His feet and ankles by this time were edematous. Medicines seemed to do him no good and in August, 1901, he took to his bed, which he had left only rarely since. The bowels moved once a day, a varying amount of blood being lost with nearly every movement. He was under my care for a time in the

*Read at the meeting of the Illinois State Medical Society at Quincy, May 22, 1902.