

THE POST-GRADUATE MEDICAL JOURNAL

Vol. I.

SEPTEMBER, 1926.

No. 12.

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BREATHLESSNESS AND DYSPNŒA IN CHILDHOOD.*

BY

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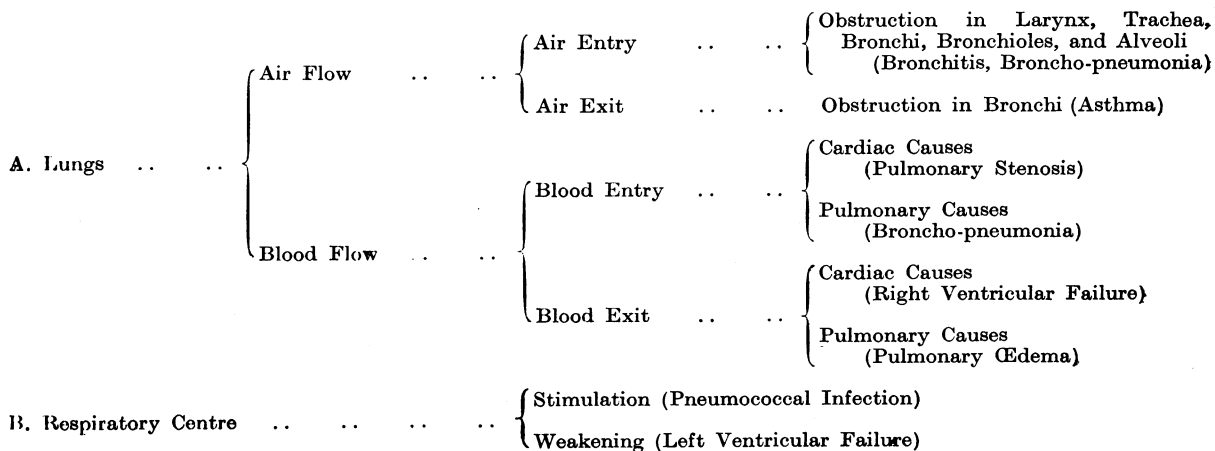
THERE are many causes of breathlessness in early life. In considering the mechanical causes which may be met with it is useful to have some such scheme as is here shown for purposes of classification. To ensure comfortable breathing there must be a constant air flow and a constant blood flow through the lungs, and air and blood must meet in the alveoli. Failure to accomplish this is the most common cause of breathlessness, and the failure usually depends on some disease of the lungs or of the heart. We shall consider some of

* A Clinical Lecture delivered at the Paddington Green Children's Hospital.

these diseases which are associated with breathlessness.

There is no more pronounced example of breathlessness and dyspnœa than that seen in broncho-pneumonia, and in no disease are there so many factors which disturb the respiratory balance at work at the same time. First of all we have obstruction in the respiratory passages due to swelling and exudation into the bronchi and bronchioli. Secondly, there are areas of active inflammation throughout the lungs mixed with patches of collapsed tissue, leading to the cutting off of much of the pulmonary tissue from contact with the air and to interference with the pulmonary circulation. As the result of this we have, thirdly, a strain thrown on the right side of the heart, and also dilatation and weakness of the right ventricle which fails to maintain an efficient circulation through the lungs. The fresh air cannot reach the blood and the blood cannot reach the alveoli. No wonder that broncho-pneumonia is a distressing

SCHEMA SHOWING MECHANICAL CAUSES OF BREATHLESSNESS AND DYSPNŒA.



illness to watch and a disappointing one to treat.

Dyspnoea is a very marked feature. The respiratory rate is always increased and mounts up quickly to 40 or 60 per minute. Higher rates may be present, even up to or above 100 per minute. It is probable that these higher rates are associated with some disturbance of the respiratory centre rather than due directly to the pulmonary affection. The normal diaphragmatic breathing of infancy and childhood is reinforced by costal breathing, and gradually all the accessory muscles of respiration are brought into play. The child has a distressed look, and this distress is due to the urgent call for special respiratory efforts. One can see this muscular over-action in the dilatation (inspiratory) of the *alæ nasi*, the descent (inspiratory) of the larynx, the raising of the clavicles and sometimes of the shoulders, and the strongly acting sterno-mastoid and scalene muscles. The first effort at the onset of dyspnoea is to get the chest fully expanded, and the tendency is for this distension to persist as long as the respiratory muscles are overacting. If cyanosis develops the dyspnoea may become of such a character that the child, if old enough, must sit up in order to use his muscles more powerfully—the condition of orthopnoea. In some cases the dyspnoea may be as great as in laryngeal diphtheria, and, further, the signs of obstructed breathing may be very similar. There may be great sinking in of the soft parts below the ribs and above the clavicles, and this is due to the failure of the more or less consolidated lungs to expand sufficiently to meet the thoracic expansion, and to the bronchial obstruction. In some cases of broncho-pneumonia these signs of obstructed breathing have been so marked that tracheotomy has been suggested as a means of relief. Naturally such a measure would be useless, and if one is tempted to employ it the golden rule should be kept in mind—namely, never operate for laryngeal obstruction unless laryngeal stridor is present. The combination of the three forms of dyspnoea—from bronchial obstruction, from lobular pneumonia, and from cardiac failure—makes it very difficult to determine the exact part which each of these factors plays in the production of that breathlessness which is such an outstanding feature of the disease.

LOBAR PNEUMONIA.

Consider next a disease which is often confused with broncho-pneumonia and which is often described as being accompanied by breathlessness—namely, lobar pneumonia. During the early and acute stages one of the leading features of this disease is the absence of breathlessness. The breathing is usually rapid from the onset, and may quickly run up to 40, 50, or 60 respirations per minute. But look at the child. There is no sign of distress or anxiety; there is no overaction of the thoracic muscles or cyanosis; and there is no difficulty in lying down. The special type of respiration in lobar pneumonia is not dyspnoea but

tachypnoea—rapid breathing. As the pure pneumonic breathing of childhood presents certain characteristics which are of diagnostic value it may be described more fully.

In character the breathing is shallow or superficial, and from this fact the increased frequency may easily be overlooked. The bystander sees nothing in the face or in the chest movements to suggest tachypnoea; but if he places a hand on the abdomen, the rapid, shallow breathing will be at once detected. The breathing is chiefly abdominal, slightly lower costal, and scarcely at all upper costal. In other words, it is natural breathing accelerated. If upper costal movement is at all marked one may be quite sure that there is some other cause than a patch of pneumonic consolidation. The patient is not in the least distressed and is, in fact, unconscious of any breathing difficulty. The type of respiration is that which has been described as medullary, automatic, and unconscious, and the increased rate is probably due to stimulation of the centre for respiration in the medulla by the pneumococcal toxins.

In this disease a condition of dyspnoea is sometimes taken for granted because of a "panting" or "grunting" type of breathing which is not uncommon, and because of active movements of the *alæ nasi* which accompany this type. On closer examination it will be found that this is associated with an inverted type of respiratory rhythm. The normal respiratory cycle consists of inspiration—expiration—pause, the pause being the imperceptible termination of the expiration. Very frequently in pneumonia this cycle is inverted and becomes expiration—inspiration—pause. During expiration the air is driven forcibly out of the chest, a rapid inspiration follows, and during the pause the air is, as it were, held up in the chest, the vocal cords being closed. This form of breathing may occasionally be seen in children under other conditions, as, for example, after a fit of crying, but in pneumonia it may persist for hours or days. The "pant" or "grunt" occurs when the glottis opens at the end of the pause and the air is forcibly expelled by the expiratory muscles. During this panting breathing the appearance of breathlessness is further simulated by the movement of the *alæ nasi*. Reference has already been made to the inspiratory dilatation of the *alæ nasi* in the dyspnoea of broncho-pneumonia. It will be found that there is no such inspiratory dilatation of the nares in uncomplicated lobar pneumonia, because there is no such obstruction of the air passages as would induce it. Nevertheless there is often in pneumonia an exaggerated movement of the *alæ nasi*, but it consists of an expiratory dilatation. It occurs during or at the end of expiration, and it occurs only in association with the inverted type of respiration to which reference has been made. The method of its production is probably as follows. In the inverted type of breathing the expiration is forcible, and air is driven out with a certain explosive force, so that

the air rushing through the nostrils distends the lax *alæ nasi*.

While the observer will find various alterations in the type of breathing present in lobar pneumonia, he will not find any evidences of breathlessness or dyspnœa. If these develop he will be wise in looking for the cause outside the lung conditions. As the pneumococcus attacks the heart tissues it will be found that breathlessness arising in the later stages of the disease is dependent on cardiac weakness rather than pulmonary inflammation.

TWO CASES DESCRIBED.

We had recently two patients in adjoining beds, both suffering from cardiac dyspnœa and orthopnœa. One was three years old, and was very cyanosed as the result of congenital heart disease. The red cells numbered 6,660,000 and the hæmoglobin was 120 per cent. The dyspnœa in this patient was the result of the usual difficulty in the pulmonary circulation of securing a free interchange of gases. Unlike the dyspnœa of pulmonary disease, where the air cannot reach the blood in sufficient quantity, the difficulty here was that the blood did not reach the air in the alveoli in sufficient amount. Hence the increase in the number of red cells and hæmoglobin—a compensatory process. The pulmonary circulation was deficient owing to a congenital lesion of the right heart, which diminished the blood flow through the pulmonary artery. We can take this as a typical example of dyspnœa in that form of heart disease, associated with cyanosis, in which the pulmonary circulation is defective and a certain degree of oxygen-hunger results. This is met to some extent by forced respiratory movements in order to expand the lungs more fully, and this is often carried out more easily and efficiently by adopting a sitting position, as in this patient. This has been called the orthopnœa of choice, because the patient often can lie down or even sleep lying down, but prefers the sitting posture. After a week's rest the breathlessness in this patient passed off entirely while he was in bed, but was easily induced by any exertion.

The adjoining patient was also suffering from dyspnœa with orthopnœa. She was a girl of 12 years, and the cardiac condition was one of considerable dilatation, the result of active myocarditis and endocarditis. There was no cyanosis here; but, on the contrary, the face was pale and somewhat anæmic-looking. There was no difficulty in the entrance of air into the lungs, as all the respiratory passages were clear. There was no obstruction of the circulation through the lungs, as the right heart showed no sign of dilatation and there were no œdematous crackles in the lungs. Yet the patient was dyspnœic, intensely so, and during the following week the position of orthopnœa was maintained—in fact, she was unable to lie down at all. It is clear that some other factor than the respiratory exchange of gases in the lungs had come into play here. The condition of the left ventricle,

one of dilatation, the feeble rapid pulse and engorgement of the liver, all pointed to a weak arterial circulation outside the pulmonary area. Amongst the other parts of the system suffering from this was, in all probability, the respiratory centre in the medulla. A defective supply of pure arterial blood there would probably be signalled as a call of distress to the higher nervous centres. Now any call of distress in respiratory difficulties seems always to be met in one way—namely, the voluntary fixation and elevation of the upper chest, and then the bringing into strong play of all the thoracic muscles of inspiration and expiration. As in the case of this girl, the call seems to be a very imperious one when the arterial circulation is failing, and the result is intense dyspnœa and an orthopnœa of necessity.

It will be noted that in obstructive pulmonary difficulties of respiration the ordinary muscles of respiration are first called on and then the extraordinary ones. There is a second string to the bow when the first fails. But in the case of the heart in difficulty there is no other muscle to call in than the myocardium itself. Yet it would appear as if the signal of respiratory disturbance in cardiac disease was met by the same response as nature employs in pulmonary difficulties, and the result is a condition of orthopnœa—imperative and, at the same time, useless. It may be argued, and very reasonably, that if the dyspnœa is due to a defective supply of blood to the respiratory centre in the medulla, that supply would be maintained more easily with the patient lying down rather than sitting up. Gravitation would help in the former position. This probably applies up to a certain point, but the time comes when the supply in the lying position is so defective that the call of distress is sent out and nature has only one response. It is open to grave doubt as to whether the patient derives much benefit from this orthopnœic position—he is simply compelled to adopt it and cannot breathe otherwise. It is under the conditions described in this girl's case always to be viewed very seriously and as of grave prognostic significance. In chronic myocardial disease the condition may persist for months with little change, but in acute diseases of the heart muscle the end is not usually far off. The girl whose case has been described lived only six days in hospital, yet the marked distress in breathing had come on only two days before admission.

UNDERLYING CONDITIONS.

It would appear in connexion with cardiac dyspnœa that there may be different underlying conditions leading up to somewhat similar results. There is, first of all, the condition in which the pulmonary circulation is defective; the lungs tend to become engorged and cyanosis is present. This is the form in which the right side of the heart is more especially in difficulty, although the primary lesion, as in mitral stenosis, may be on the left side. There is, secondly, a condition of great

dyspnoea in which the circulation through the respiratory centre seems to be defective, and this is associated more especially with weakness of the left ventricle. There is no cyanosis, no evidence of any obstruction to air or blood in the lungs, and, in fact, no obvious reason why the patient should be intensely dyspnoeic. If we recall the two diseases of the lung already referred to—broncho-pneumonia and lobar pneumonia—it will be found that the disturbances of respiration in them were traced to two different sources, one being pulmonary and the other in the respiratory centre. In the case of broncho-pneumonia the dyspnoea was directly due to obstruction of the air to the lungs and of the blood through the lungs.

THE DIAGNOSIS OF URINARY DISEASES IN CHILDREN.*

BY

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I PROPOSE to deal with this subject from the point of view of an investigation of symptoms indicating disease of the urinary tract. One, therefore, will have to consider the following disturbances of micturition: Dysuria, difficulty, enuresis, frequency, hæmaturia, pyuria, and pain or swelling in one or both kidney regions, in association with, or independently of, any of the above.

Dysuria is most conveniently considered together with frequency. In their order of importance the causes of these may be *Bacillus coli* or a coccal infection, calculus, urinary tuberculosis, foreign body in the bladder or urethra.

BACILLUS COLI INFECTIONS.

These are recognised as of two types, the acute and the chronic. The former manifesting the usual constitutional signs of an acute infection combined with obvious features indicating the urinary tract as the seat of the trouble, such as renal tenderness, marked frequency with distress on voiding, often with terminal hæmaturia. The urine has generally an abundance of pus cells, and the causal organism is obtained in pure culture. Gastro-intestinal disturbance, with nausea and vomiting, constipation, or diarrhoea frequently precedes and accompanies the attack, which responds readily to castor oil and alkalies.

The chronic form is not so easily dealt with and must be investigated with the greatest care, as there is frequently some more important factor underlying

This, with the accompanying cyanosis, is comparable to what may be called the pulmonary form of cardiac dyspnoea. In lobar pneumonia, on the other hand, there was no evidence of any pulmonary obstruction—to air or blood flow—and the disturbance was traced to stimulation of the respiratory centre, producing rapid breathing but no dyspnoea. So in the dyspnoea which accompanies failure of the left ventricle we may reasonably place the source of disturbance in the respiratory centre. Apparently this centre is not stimulated by anoxæmia, but is weakened, and sends an appeal to the higher centres for help. The response, unfortunately, does not seem to be of any great value in the relief of the condition.

the constant presence of the coliform organism in the urine, so that the diagnosis must not be considered complete until all the established methods of inquiry into the condition of the urinary tract have been made use of and other possible causes excluded. This applies especially to calculus which if left long enough, will ultimately give rise to infection, which in 90 per cent. of cases will be coliform in nature.

CALCULUS.

Calculus is much more common in the bladder than in any other part of the urinary tract in children, the greatest incidence lying between the ages of 3 and 7 years. The forefinger well lubricated and gently introduced into the rectum will allow of a satisfactory examination of the bladder of a child even as young as 3 years without making the patient cry, and will enable the examiner to readily detect a stone not much larger than a pea if the examination is made bimanually. Before withdrawing the finger careful palpation of the posterior urethra should not be neglected, as a stone may lodge in this region (see illustration). In front of the triangular ligament a urethral stone, or a point of tenderness in the bulb indicating the presence of a stone, can be easily palpated.

Whether or not this method is fruitful of a positive diagnosis a skiagram covering the whole urinary tract is essential, for the reason that if a bladder stone has already been detected the possibility of urinary concretions at a higher level is stronger than under the opposite circumstances. Renal stone in children is met with more commonly than in former days when the necessity of radiographic examination of the whole urinary tract was not considered so important. General anaesthesia is a routine necessity in all young children for radiography. The continued absence of the evidence of calculus by this means of inquiry does not completely absolve the investigator from further search, for the reason that bladder stone in children may consist largely of ammonium urate or uric acid, and both these substances having but a poor opacity to X ray may enable a small stone to escape detection. Fortunately, this likelihood is less in

* A Lecture delivered at the Children's Clinic.