at present, a matter of conjecture. But the polymorphonucleareytosis of epidemic poliomyelitis is caused not only by the lesions of the nervous system, but also by lesions of the lymphatic tissues and liver. This consideration will serve to explain certain discrepancies in the cell-findings in the cerebrospinal fluid, removed by lumbar puncture, and in the circulating blood.

Sixty-Sixth Street and Avenue A.

REPORT OF A CASE OF PAROXYSMAL TACHYCARDIA *

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Last year I published a paper on paroxysmal tachycardia¹ in which I reported one case which had been under observation for about three years. This patient has a mitral stenosis. She is still seen occasionally, has better general health than for several years, but is subject to these attacks at regular intervals. Another case came under my observation last May, and it presents certain interesting features which prompt me to report it.

History.—This patient, a married woman of 29, came to me complaining of a distressing irregularity of the heart. A few weeks previously she had had a miscarriage followed by a peritonitis. I did not see her during this time. At my first visit she complained of weakness, shortness of breath, and pain in the cardiac region in addition to the irregularity mentioned above. I found no murmur, the heart slightly enlarged to the left (nipple line), sounds at the apex diffuse, with some tenderness in the fifth intercostal space just outside the nipple line. There was no arrhythmia but an apparent unevenness in the force of the beats, as though the ventricular contraction were incomplete. In view of the later developments, this seemed to be an interference with nervous impulse rather than muscular insufficiency. The patient gave an interesting cardiac history. At the age of 13 she had jumped rope until her heart gave out. She was confined to bed at this time and was very sick. At intervals since she has had attacks of rapid heart, or "palpitation." These attacks always begin with a "thud" and she realizes instantly that an "attack" is on. It ceases as suddenly as it began, and is followed by a momentary faintness, after which the patient is conscious of no after-effects. The duration varies from a few minutes to eight or nine hours. She sometimes wakes out of a sound sleep at night to find her heart racing, while more often it follows some severe exertion. These attacks have now persisted for sixteen years with no essential change. She has a few times noticed that the palpitation would cease after some sudden movement, such as jumping abruptly out of bed. From this story I made a provisional diagnosis of paroxysmal tachycardia and asked the patient to call me when she had an attack. Bearing in mind that the pathology of this condition is a fibrosis of the primitive cardiac tissue, it is not difficult to believe in the possibility of such a traumatic origin as that mentioned.

Treatment and Result.—October 29 I was fortunate enough to see her in what she called a typical attack. Her pulse was 192, regular, embryocardial, no murmur; patient anxious, skin moist, breath short. She thought this attack was lasting too long; it had already persisted three hours. As soon as I was sure of the condition, I explained to her that no drugs were known to have any effect on the immediate attack and suggested trying the postural methods referred to in my previous paper. She was first placed with her head down and instructed to take deep breaths, to hold and to push. This only choked her. After several other positions were tried without success, I advised the patient to continue these efforts

at intervals and was putting on my overcoat to go, when she sat up in bed to try again. She folded her arms tightly across her chest, the right arm passing directly over her heart, leaned forward, took a deep breath, closed her glottis, and then compressing her chest with her arms, pushed as hard as possible. Then instantly she looked up and said, "It has stopped." I grasped her wrist and found that her pulse, which two minutes before had been 192, had now dropped to 96. In two minutes more she was walking about the room feeling as well as ever and discussing her attack as though it had been weeks before.

The interesting points in the case are:

1. The persistence of the attacks for sixteen years, in a person otherwise well and in a heart without valvular defect.
2. The unusual traumatic origin.
3. The interruption of the nodal rhythm and immediate relief by the deliberate and persistent attempts of the patient to increase her intrathoracic pressure under medical direction.

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PUBLIC AGITATION AND SOME UNNECESSARY ADENECTOMIES

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There is no abnormal condition pertaining to childhood that has received greater publicity during recent years than that of adenoids. The profession has known for many years the evils attending this particular lymphatic enlargement, and has done much to enlighten the public on its harm. The newspapers and magazines must be given credit for the wide-spread dissemination of information among the laity, while public lectures by physicians in the larger cities, notably in Chicago, have had good effects. This agitation has reached the stage at which city health departments, acting through salaried inspectors, are practically excluding from the public schools children thus afflicted until the defect has been remedied. It is doubtful if there is anywhere within the domain of medicine and surgery a class of cases that show such uniformly satisfactory results as these. It has been my privilege to witness results of the adenoid operation in thousands of cases and such an experience must necessarily convince one of its great usefulness in properly selected cases.

Without depreciating in the least the good work done in this important field, however, I would like to sound a note of warning. The diagnosis of adenoid enlargement is relatively easy and for this reason is undoubtedly arrived at in many cases by the "snap-shot" method. The clinical picture is well known—the open mouth, protruding upper teeth, high-arched palate, the staring expression, history of sleeping with open mouth, snoring, restlessness, night terrors, imperfect development of the nares, impairment of voice tones, and in many cases faulty mentality. In brief, there are evidences of interference with nasal breathing and proper oxygenation. These signs are so clearly apparent in many cases that an actual, painstaking examination of the child is not made.

The steady increase in the number of children who apply to the free clinic for treatment, with a ready-made diagnosis and a prearranged method of procedure, has

* Read before the Wayne County (Mich.) Medical Society, December, 1911.

1. THE JOURNAL A. M. A., June 4, 1910, p. 1861.

forcibly called my attention to the careless tendency to omit proper diagnosis. It is by no means unusual for parents to volunteer the positive information that their children have adenoids and request immediate operation. They know something of the signs and symptoms and have made their own diagnosis, perhaps with more or less assistance from the neighbors. To convince some of them that they are wrong is not always easy, and to refuse to operate in certain cases causes evident disappointment and often a parting threat to have the desired operation performed elsewhere. Then there are the cases that have been passed on by the school inspectors. To their credit it must be said that they are usually correct in their instructions to parents, but they have many children to examine within limited hours and it is no doubt lack of time rather than lack of caution that is to blame for a small percentage of diagnoses that may be disputed. Still other cases of this sort are brought in by visiting nurses, who themselves have discovered what seem unmistakable signs of adenoids. A common source of fault is the presumption that, because in a given case there is enlargement of the faucial tonsils there must necessarily be troublesome enlargement of adenoids. A careful examination of the patients coming from various sources to the dispensary for adenectomy shows a few to be suffering from some obstruction to breathing other than adenoids. This paper is written in the interest of these occasional cases in which the adenoid operation is not clearly required.

Adenoids, also referred to as the pharyngeal tonsil and Luschka's tonsil, occupy a position in the vault of the pharynx, or nasopharynx, and are found variously distributed over the posterior wall. The adenoid mass, the two faucial tonsils and the lingual tonsil at the base of the tongue make up the lymphoid ring of Waldeyer, so called because of forming a more or less complete circle at the entrance to the pharynx. That there is a definite relationship between these various groups of lymphoid tissue cannot be disputed. Hypertrophy of one is usually accompanied by enlargement of the others to a variable degree, but this is no reliable criterion. For instance, many cases are observed in which the faucial tonsils are considerably enlarged, but without a noticeable increase in size of the pharyngeal tonsil. Again, we frequently find cases which demand attention because of a large adenoid mass, but in which there is no history of tonsillitis or sufficient change in the size or structure of the faucial tonsils to warrant their removal at the same time. Another exception to the alleged constant relationship between these lymphoid masses is that hypertrophy of the lingual tonsil is rare in childhood, during that period of greatest activity on the part of the other lymphoid structures. On the contrary, the lingual tonsil is prone to hypertrophy in adult life, at a time when its neighbors are quiescent. Therefore, reasoning from these well-known facts, it is my belief that we should take nothing for granted, but make our examination of these cases thorough before submitting a positive diagnosis and urging operation.

It is true that adenoid hypertrophy can often be diagnosed at a glance. The adenoid face or expression is pictured in every text-book on rhinology; but it is our ability to make a correct offhand diagnosis in most of these cases that is likely to lead us into occasional error.

Broadly speaking, any other obstruction of the nose may produce the same symptoms, and, in course of time, the same outward signs as those attributed to adenoids. Hypertrophic rhinitis is quite as prevalent in childhood as at any other time of life, and every rhinologist has

observed many children who were unable to breathe through the nose with any satisfaction because of enlargement of the turbinate bodies. Deflections of the nasal septum and spurs are common to childhood and cause nasal stenosis. Foreign bodies are also obstructive and may cause total occlusion of the nares. This calls to mind a clinical case in which a little girl was brought for operation on the advice of a physician who had pronounced it adenoids. The patient was unable to breathe through the nose and there was a constant profuse mucopurulent discharge. A better example of the "mouth-breather" would be hard to find. The mother said that the same condition had then persisted for a year. Examination of the nose showed a foreign body firmly imbedded in the right nostril well posteriorly. It proved to be a shoe-button which, as a source of irritation and infection, had effectually closed both nostrils. Normal breathing was restored in a few weeks without an adenoid operation.

Another factor that may lead to error is the high arched palate and irregular protruding front teeth. This deformity is invariably associated in the literature with adenoid enlargement, but such association may as often be fanciful as real. That the two are frequently coexistent is acknowledged, but to make a diagnosis of adenoid hypertrophy from a casual inspection of the hard palate is the height of presumption.

Another cause of difficult nasal breathing is some congenital or acquired deformity in that region. For example, a slight anterior deviation of the spine so as to encroach on the nasopharyngeal space has been known to produce the symptom-complex of adenoids. Furthermore, a considerable hypertrophy of faucial tonsils, even in the absence of any appreciable adenoid enlargement, may easily confuse one without a careful examination.

Every child has normally a small mass of lymphoid tissue in the nasopharynx. This may vary in size from a few nodules no larger than peas to a growth the size of a hickory-nut, or even larger. As to its location, the bulk of the mass may be located in the median line with smaller collections to either side, the bulk may be laterally placed, or, in a certain class of cases, there may not be much enlargement, but such tissue as there is has located most disadvantageously over one or both Eustachian orifices. Occasionally the adenoid tissue may be found clinging to the choanae or may extend downward sufficiently to be seen directly behind the uvula.

The symptomatology of adenoid enlargement covers a wide range, depending largely on the relation between the size of the mass and the dimensions of the nasopharyngeal cavity that contains it. For example, little or no disturbance of nasal breathing may arise from a fair-sized adenoid growth within a spacious nasopharynx. On the other hand, a small nasopharynx cannot accommodate much adenoid tissue without blocking the nares and causing marked ill effects.

It is not intended here, however, to attempt a detailed consideration of the various symptoms, the pathology or treatment. The purpose of this paper is merely to urge that we be not carried away by agitation or by enthusiasm, or by the desires or opinions of others, but that every suspected case of adenoids be placed on its own individual merits, that it be subjected to a careful examination to determine whether or not that particular child must undergo even so slight an operation. Indiscriminate operating in this class of cases is all the more objectionable in the light of our limited knowledge of the physiology of these tonsillar structures.

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