

removal, not alone for abnormal pulsations of aberrant vessels, but also for the detection of a long, sharp styloid process or ossified stylohyoid ligament in the tonsil and pharynx.

As long ago as 1870, Lucke reported two cases, one in a woman of 30 and the other in a girl of 20, both of whom suffered from difficulty in swallowing because of long, slender styloid processes.

It is well to remember that a bony rod may extend from the styloid process to the hyoid bone. It may be slender or thick. In the former case it is, at least

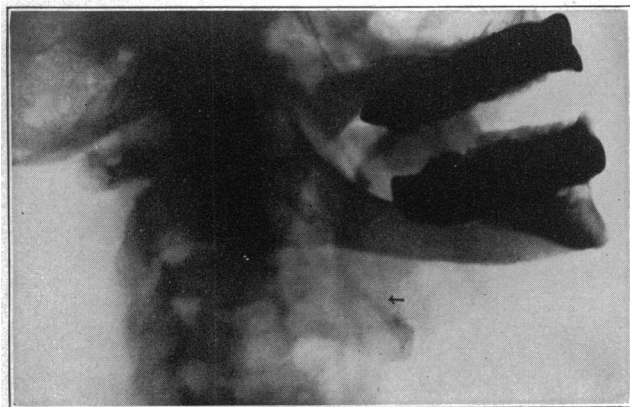


Fig. 2.—The arrow indicates the usual type of ossified stylohyoid ligament.

in early life, more or less elastic. In the latter it presents enlargements which at first, as a rule, mark the position of joints. This rod passes between the carotid arteries, and may interfere with the operation for tying them, and of those with the parotid gland which it indents on the inner side. Passing by the internal pterygoid muscle, to which it is attached by the fibrous envelop of that muscle, it lies against the outer side of the tonsil, which it may displace or indent. The displacement of the tonsil may be sufficient seriously to reduce the approach to the larynx, and may even encroach on the region back of the posterior nares so as to narrow the latter, as in the case reported by Lucke.

The length of the styloid process varies in individuals; and as long as the process is directed downward and parallel to the carotid artery, probably no other trouble than disease of the bones may be expected. Should the process be directed medially as the result of development or trauma, more or less irritation of the pharynx may occur.

In the cases reported by a number of laryngologists, the symptoms were those of difficulty and distress in swallowing, a dragging sensation in the throat, worse on swallowing, a sensation of cutting in the throat, etc. It is particularly to be remembered that in cases of painful or difficult deglutition, the styloid process may be the offending cause. Palpation and the roentgen ray readily determine the presence or absence of variations in the stylohyoid chain.

TREATMENT

Frequently mere fracture of the offending styloid process causes complete subsidence of all the clinical symptoms. At times it may be necessary to remove the distal portion of the styloid process by means of bone forceps. Only rarely is its complete surgical removal necessary.

1007 Spruce Street.

A CASE OF AURICULAR FLUTTER WITH PAROXYSMAL ATTACKS OF 1:1 CONDUCTION*

R. W. SCOTT, M.D.

CLEVELAND

The appearance of a 1:1 mechanism, i. e., the ventricle beating at the same rate as the auricle, is uncommon in patients with auricular flutter. Usually there is some degree of block at the junctional tissues which determines the rate of the ventricular response. However, under some circumstances not at present clearly understood, conduction through the auriculo-ventricular bundle is raised, and every auricular contraction spreads to the ventricle, causing it to beat at a rate seldom seen in any other condition in man. For example, in the case here recorded the rate during attacks was 272 a minute, as determined from electrocardiographic records. Similar elevations in ventricular rate have been recorded in children by Lewis¹ and by Koplik,² and in adults by Mackenzie,³ by White and Stevens,⁴ and by Blackford and Willis.⁵

REPORT OF CASE

V. R., a man, aged 25, a mechanical engineer, first came under observation in November, 1920. There was no history of rheumatic infection. He had always enjoyed good health, and was fairly active in college athletics. While in military service, in 1918, he contracted influenzal pneumonia, but recovered without complications, and was discharged in September, 1919, apparently well. He resumed his occupation in civil life, and worked steadily until August, 1920, when he developed attacks of "palpitation of the heart." At first the attacks appeared about once a week; a little later they were induced by any slight exercise, such as ascending stairs or even walking rapidly. During the periods of rapid heart

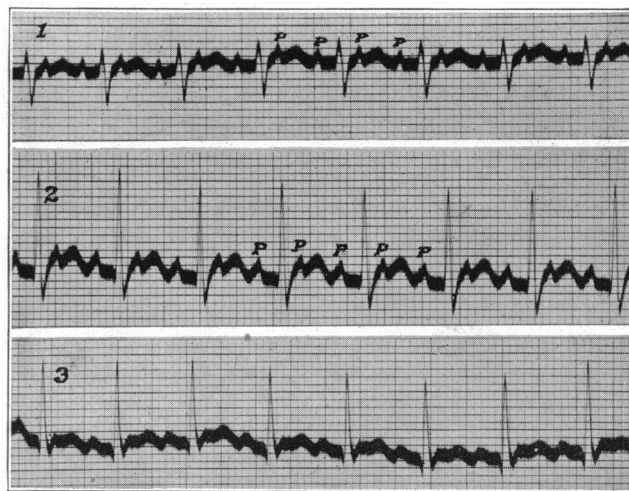


Fig. 1.—Three leads showing auricular flutter with 2:1 block; auricular rate, 272 a minute; ventricular rate, 136.

action, the patient experienced vertigo and other symptoms of cerebral anemia, but he never lost consciousness. The attacks began abruptly, accompanied by a sensation of "flut-

* From the Medical Clinic of Western Reserve University at City Hospital.

1. Lewis, Thomas: *The Mechanism and Graphic Registration of the Heart Beat*, New York, Paul B. Hoeber, 1920.

2. Koplik, H.: *Am. J. M. Sc.* **154**: 834 (Dec.) 1917.

3. Mackenzie: *Diseases of the Heart*, London, 1913.

4. White, P. D., and Stevens, H. W.: *Ventricular Response to Auricular Premature Beats and to Auricular Flutter*, *Arch. Int. Med.* **18**: 712 (Nov.) 1916.

5. Blackford, J. M., and Willis, F. A.: *Auricular Flutter*, *Arch. Int. Med.* **21**: 147 (Jan.) 1918.

tering in the chest." Lying prone for a few minutes relieved the cerebral symptoms and caused the rapid rate to subside gradually—that is, a certain transition period of several minutes was noted during which the heart beat irregularly.

When I saw the patient, he had what he believed to be his normal heart rate, and he was not aware of any circulatory disturbance. He appeared to be in good health, and the general physical examination was negative except for the circulatory disorder. The heart boundaries were normal to percussion, and no adventitious sounds were audible. The precordial activity appeared somewhat exaggerated, and although the patient had been at rest for several minutes,

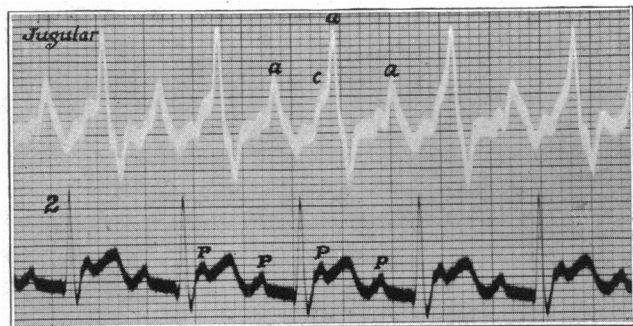


Fig. 2.—A simultaneous record of the electrocardiogram (Lead 2) and the jugular pulse registered optically: Alternate A waves of the jugular pulse are conspicuous, and correspond to those auricular beats which occur while the ventricle is in a state of contraction.

the rate was 136 a minute, but quite regular. It was not influenced in the least by forced breathing or change of position, although compression of either vagus caused a prompt slowing, accompanied by a definite irregularity. Inspection of the jugular bulb revealed an unusually forcible venous pulse, which appeared systolic in time and suggested the venous phenomenon seen in tricuspid insufficiency.

The absence of a respiratory cardiac arrhythmia even with forced breathing, and the prompt but transient irregularity induced by vagal pressure, suggested the possibility of auricular flutter. This was demonstrated later by the electrocardiogram, the three leads of which are shown in Figure 1. The auricular rate is 272 a minute, and a 2:1 block is seen; i. e., each alternate auricular beat spreads to the ventricle, causing it to beat at 136 a minute.

In order to analyze further the pulsations noted over the jugular bulb, a simultaneous record was made of the electrocardiogram (Lead 2) and the right jugular pulse registered optically by Wigger's modification of the Frank capsule. This record is shown in Figure 2. Comparing these two curves, one notes that the alternate and more conspicuous A waves correspond to those P deflections of auricular systole which occur while the ventricle is still in a state of contraction. The auriculoventricular orifice is closed so that the force of the auricular beat is spent on the column of blood in the veins, producing the conspicuous venous pulse.

After the foregoing observations were made, the patient suggested that he might induce an attack of rapid rate by walking briskly about the laboratory for a few minutes. This was done, and presently the extreme tachycardia appeared. The pulse in the accessible arteries was barely palpable, while the heart beat was uncountable with the stethoscope. A record was made immediately, and is reproduced in Figure 3. This shows that the ventricle had now assumed the auricular rate of the previous record (Fig. 1); that is, a 1:1 mechanism was established. After the paroxysm had continued for several minutes, compression of the right vagus trunk restored the original 2:1 block, and the ventricular rate returned to 136. The transition from the 1:1 to the 2:1 block, however, did not occur abruptly, but required approximately a minute during which time both mechanisms were seen.

The well known influence of the vagus on auriculoventricular conduction was well illustrated in this case. While flutter with 2:1 conduction maintained, slight pressure over either vagus trunks caused the prompt appearance of varying

grades of block (Fig. 4 a). An attempt was made to determine on which side the mechanism was more sensitive, but no appreciable difference was found. As correctly as one could judge, blocks of similar grade appeared just as promptly when like pressures were applied to either nerve trunk. After observing the facility with which his attacks were stopped by vagal pressure, the patient, on his own accord, resorted to this procedure to obtain relief on several occasions before finally coming to the hospital for further study.

After admission the patient was carefully studied in an attempt to find a satisfactory explanation for the cardiac disorder, but nothing definite was obtained. So long as he remained in bed, flutter with 2:1 block prevailed; but any slight exercise, such as ascending stairs, appeared to facilitate conduction, and the 1:1 mechanism with the rapid ventricular rate promptly appeared. This was accompanied by a fall in systolic blood pressure from 120 to 80 mm. of mercury, with the subjective symptoms attributable to cerebral anemia.

A course of digitalis was begun, and after receiving 18 c.c. of the tincture over a period of four days, the patient developed a slight diarrhea, and the drug was discontinued. Records taken at this time (Nov. 11, 1920) showed auricular flutter with 3:1 and 4:1 block (Fig. 4 b). The following day (November 12) the heart rate was found to be slower and more irregular than usual, while the electrocardiogram demonstrated auricular fibrillation (Fig. 4 c). Some time during the night a normal rhythm was established, so that it is not known just how long the auricles fibrillated; but a record taken the morning of November 13 showed a normal mechanism with the usual digitalis effect on the T wave (Fig. 5). The exercise which formerly induced attacks now had no more than the physiologic effect on the heart rate.

Having no information as to why auricular flutter should have appeared originally in an otherwise normal young adult, it was decided simply to observe the patient for the possible return of the abnormal mechanism. This occurred sixteen days later (November 29) with the usual attacks of rapid ventricular rate from slight exertion. Records showed that the auricular rate was exactly the same as before (272 a

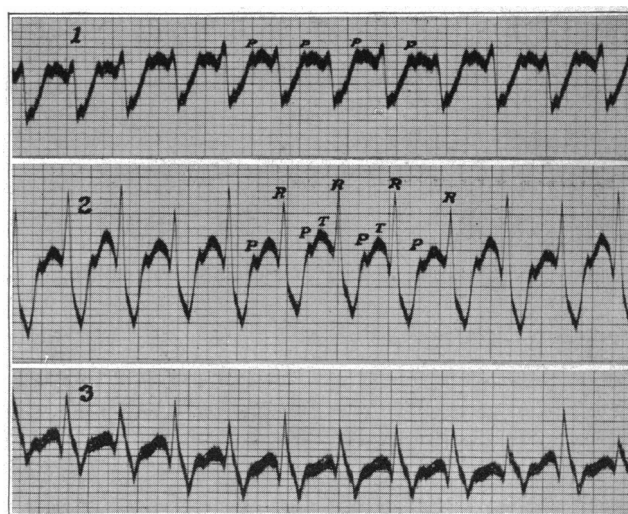


Fig. 3.—Three leads showing the mechanism during an attack: Each auricular contraction is followed by a ventricular response; rate, 272 a minute.

minute), but between attacks alternate 2:1 and 3:1 block was present. Another course of digitalis was given, and again attempts were made to record the anticipated transformation through fibrillation to normal, but without success. In a thirty minute period between records during the day, fibrillation occurred, and some time during the following night the normal mechanism was restored.

The patient was then discharged and remained at home for a month. The first two weeks he was given digitalis, 1 dram (4 c.c.) of the tincture on alternate days, and then every third day. The dose was gradually diminished, and

at the end of three months the drug was discontinued altogether. The patient resumed his regular occupation in January, 1921, and has continued uninterruptedly to the present time. Records taken at intervals show a normal cardiac mechanism, and the patient reports that he plays tennis and golf without any untoward effects.

COMMENT

The foregoing case presents several interesting features which merit consideration. Among these may be mentioned the etiologic factors underlying the establishment of auricular flutter, in a heart showing no other signs of disease. The disappearance of the disorder under digitalis, and the persistence of the normal mechanism for more than a year, suggest that the underlying cause, admittedly obscure, is eradicated. It appears, then, that flutter may supervene in an otherwise normal heart, continue for a period, and then disappear. Had our patient not been subject to attacks of 1:1 ventricular response, it is probable that the disorder would have gone unnoticed.

The regularity with which the auricular rhythm was maintained during the periods of flutter was remarkable. Records taken during the second period of flutter showed that the auricles were beating at the same rate as in the first attack. Comparator measurements were made with the assistance of Dr. Feil, from the ventricular deflection in the curves of Figures 1 and 3, and it was found that the maximum variation in length of interauricular cycles was 0.0026 second in Figure 1, and 0.0042 in the curves of Figure 3.

It is noted that the ventricular rate is 136 (in Figure 1), or exactly half of that of the auricle. Owing to

of 2:1 ventricular response. There is obviously no question as to the ability of the ventricle to respond, for if the stimulus is conducted, it may follow the rate of the auricle, as illustrated in the record of Figure 3. The block therefore occurs at the auriculoventricular node, and is attributed by Lewis⁶ to the duration of the refractory state of the node. If we accept this explanation, it appears that this state of nodal refrac-



Fig. 5.—Three leads showing the normal mechanism after digitalis.

toriness may be profoundly influenced by slight bodily exertion. For example, simply walking briskly about the laboratory for a few minutes sufficed to convert the 2:1 into a 1:1 mechanism.

A similar phenomenon was recently observed in another patient, aged 45, who was suffering from cardiac failure and hypertension without any clinical signs of kidney insufficiency. On admission the cardiac mechanism was normal, and the rate was 110 a minute. After he recovered sufficiently to walk about with comfort, it was noted that while up the heart rate was 171 and quite regular; when he lay down for a few minutes, an irregularity appeared with the heart slowing gradually until it became regular at 114 a minute. This rate then continued so long as the patient remained in bed, but was promptly elevated to 171 when he took any slight amount of exercise. Records taken while the patient was quiet showed that he had auricular flutter with the auricles beating at the rate of 342 a minute; every third impulse spread to the ventricle, causing it to beat at 114. With the electrodes still applied, the patient simply got up and walked about in a circle for two or three minutes, when 2:1 conduction appeared with the ventricle beating at 171 a minute. When he lay down again this gradually reverted to a 3:1 mechanism, and the ventricle dropped back to 114. Compression of either vagus had the usual effect of increasing the existing block. It seems probable that the refractory state of the junctional tissue may be altered through some mechanism set into play by exercise, the result being to speed up the rate of recovery in the node. This renders the node more excitable, and impulses formerly falling during its refractory state are now transmitted, and excite the ventricle to contraction. Acting in the opposite direction, i. e., to delay the rate of recovery, are the effects of digitalis and vagal stimulation (Fig. 4 *a* and *b*).

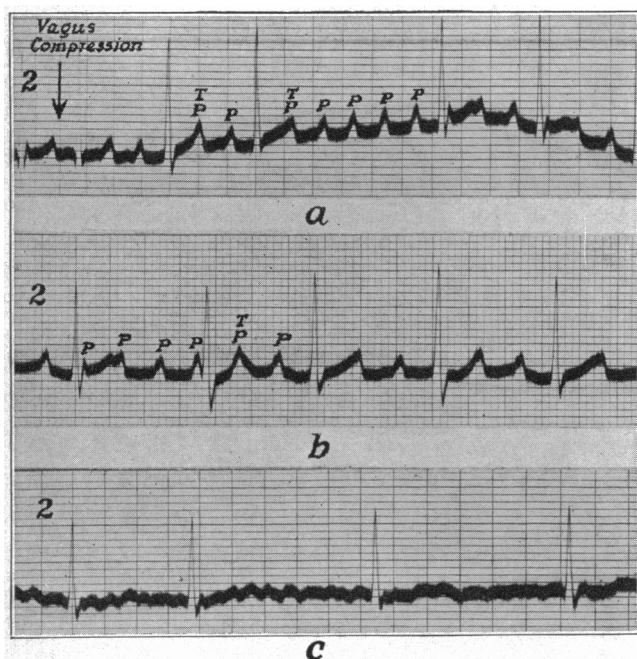


Fig. 4.—*a*, effect of vagal pressure applied at the point indicated; the existing 2:1 block was increased to 3:1, and in one place to 5:1; *b*, influence of digitalis in increasing the block at the junctional tissues; to be compared with Curve *a*; *c*, fibrillation which developed after the administration of digitalis.

the block at the auriculoventricular node, only alternate impulses were conducted to the ventricle. This conclusion is based on facts recently elicited by Lewis and his co-workers, who found that as the auricular rate approaches 270 a minute, there is a tendency for block to appear at the nodal tissues, causing the phenomenon

6. Lewis, Thomas: *Quart. J. Med.* 14: 339 (July) 1921.

SUMMARY

In a case of auricular flutter with paroxysmal attacks of 1:1 conduction, any slight exertion induced attacks during which the ventricles beat in response to each auricular contraction at the rate of 272 a minute. Digitalis converted the flutter through fibrillation to normal, which continued for sixteen days, when flutter reappeared. The sinus rhythm was again restored by digitalis, which was administered in decreasing doses for three months and then discontinued. The heart's mechanism has remained normal for one and a half years.

NEUROPATHIC ARTHRITIS

JOHN H. DUNCAN, M.A., M.B. (TOR.)

SAULT STE. MARIE, ONT.

For the performance of normal function, it is essential that every organ in the body not only receive an adequate supply of blood but also maintain constant communication with the central nervous system. This it does by means of afferent and efferent nerves, the former conveying information from the organ to the center and the latter carrying commands from headquarters to direct the activities of the peripheral structure. Through the afferent nerves the organism receives all impressions of the surrounding environment, and with the help of the efferent nerves suitable response is made by muscle, gland or other tissue.

The simplest reflex might be conceived of as involving the sensory end-organ, its fiber and cell in the spinal ganglion with a central branch arborizing around an anterior horn cell from which arises the motor nerve conveying an efferent impulse to distant muscle. However, the vast majority of reflexes are much more complicated, one relay after another of nerve cell and fibers being employed, some peripheral, some spinal and some cerebral. No nervous structure can be

sensitive nerve supply is often questioned by the sufferer from toothache, and grateful feelings extended to the conservative dentist who saves the tooth, by devitalizing it and replacing the sensitive pulp with unfeeling gold or silver. For a time all goes well, but the blessing is not unmixed. These insensitive teeth

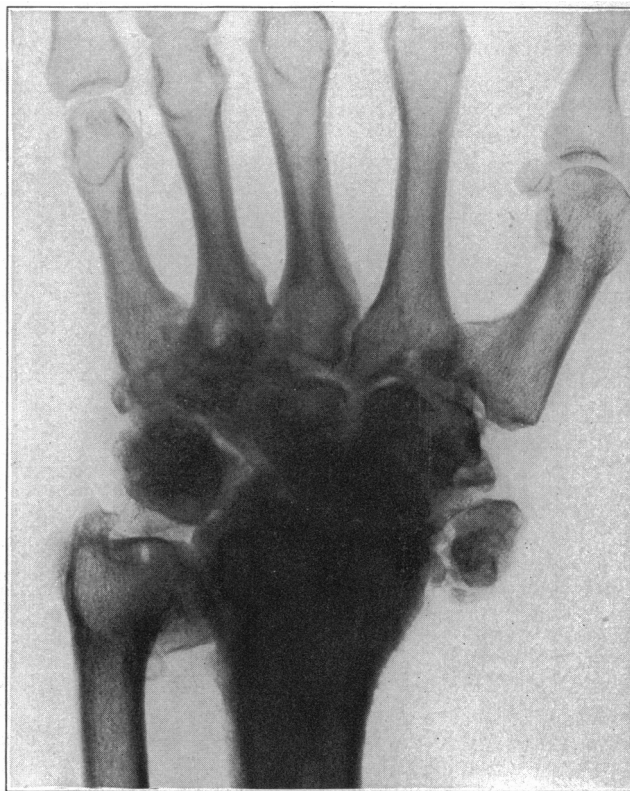


Fig. 2.—Appearance of right wrist of patient.

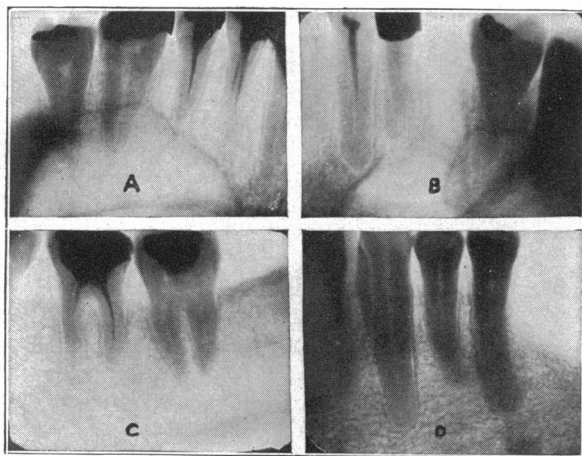


Fig. 1.—A, absorption of roots of devitalized teeth, especially the bicuspid; B, hypertrophy of root of devitalized cuspid; C, clubbing of roots of devitalized lower molar; D, hypertrophic process affecting the roots of teeth of patient in whom the gasserian ganglion has been removed.

injured without its happening that some reflex arcs are disturbed, and the evil effects are found in the organ supplied. The source of the injury may be trauma, infection or toxin. The result may be temporary, as in some cases of peripheral neuritis, or permanent, as frequently happens after trauma. The necessity for

are in the same position as any other part of the body deprived of nerve supply; and, as we shall see later in connection with other nerve lesions, one of the most important changes that occur is a perversion of metabolic processes. Sometimes there is increased anabolism and sometimes increased catabolism. This is illustrated in Figure 1 A, B and C. The only blood supply left to these teeth is what comes from the dental periosteum.

To show that this vascular deficiency is not altogether responsible for the changes noted, I will call attention to what has happened to the teeth of a patient who underwent a removal of the right gasserian ganglion for tic douloureux. The periosteal bone production is as marked as is ever seen in any devitalized tooth (Fig. 1 D).

In the roentgenographic study of the teeth, much has been made of apical abscesses and clubbed roots, as representing microbic infection. The results of extraction in many cases seem to verify this assumption. In the cases characterized by neuralgic symptoms of the face, it would seem well to keep in mind the possibility of the dental condition being secondary to a nerve lesion instead of the other more usual conclusion. According to Stewart¹ there is no unequivocal proof for the presence of specific trophic nerves, but all efferent nerves exert an influence on the nutrition of

1. Stewart, G. N.: Manual of Physiology, 1910, p. 699.