Remarks

ON

POSTURAL OR SO-CALLED STATIC DEFORMITIES.

BY

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I.

STATIC deformities have been defined as those which are due in the main to the action of gravity. But, as everyone is subject to the action of gravity, it is clear that this is not enough in itself to account for deformity in an otherwise normal person. It would be more correct to say that static deformities are due to default of the normal mechanism for counteracting the influence of gravity and maintaining the body in the upright position. This mechanism is a nervous mechanism, for it has been shown by Sherrington¹ that the upright attitude or posture of the body is maintained by continuous reflex muscular activity (postural activity). For this reason I prefer to speak of these deformities as postural rather than static. The word static implies something almost wholly mechanical, whereas I believe that many of these deformities are really nervous in origin.

I must say, however, that by postural deformity is meant not merely a faulty posture as such, but also the deformity that may develop from it. This is important, because many writers have tried to draw a distinction between postural deformity and structural deformity, holding that a deformity is no longer postural when structural changes have taken place in it. Such a distinction is wholly artificial and misleading. A rachitic deformity is still a rachitic deformity after the active rickets has subsided. A paralytic deformity is a paralytic deformity even though the paralysis has recovered. Similarly, a postural deformity is one that begins as a faulty posture and passes through all degrees up to severe structural alteration.

GENERAL CONSIDERATIONS.

In the skeletal muscles we meet with two kinds of muscular activity. The one is phasic—quick and transitory and concerned with the execution of *movement*; the other is tonic—relatively slow and prolonged and concerned with the maintenance of *posture*. These two kinds of activity are quite distinct. Indeed, in certain invertebrates there are separate muscles, differing in structure and in appearance, for the execution of movement and for the maintenance of posture respectively. In vertebrates the same muscle is utilized for both purposes, but there is reason to believe that different parts of the muscle fibres are concerned in these two functions. It has been suggested by Bottazzi² and others that the sarcostyles constitute the contractile or movement mechanism, and the sarcoplasm the tonic or posture mechanism. Further, there is some evidence that these two mechanisms have independent nerve supplies, and that the sympathetic may be concerned in the tonic or postural contraction of voluntary muscle. Perronito³ and Boeke⁴ have described accessory (sympathetic) nerve endings in voluntary muscles. These consist of fine non-medullated nerve fibres connected with the perivascular sympathetic nerve plexus and terminating in the sarcoplasm independently of the motor end plate. De Boer⁵ was able to abolish the tonus or postural contraction in the hind limbs of the frog and cat by section of the rami communicantes of the sympathetic.

This question is not yet settled, but it is particularly interesting in view of the close similarity between postural contraction in skeletal muscle and contraction in the smooth unstriated muscle of the hollow viscera and blood vessels. Indeed, Sherrington maintains that postural activity is the mechanism whereby these hollow viscera (stomach, bladder, etc.) and blood vessels normally adapt themselves to variations in the volume of their contents.

In the skeletal system postural activity occurs only in the antigravity muscles—that is, those which constantly resist and counteract the influence of gravity. This does not mean that no other muscles under any circumstances are capable of acting posturally, but that the normal postural or standing reflex involves contraction of the antigravity muscles only, their antagonists being inhibited.

Sherrington pointed out that the failure of many of the earlier experimenters to demonstrate tonus in skeletal muscle was due to their failure to appreciate this fact. Many of the observations were made on the gastrocnemius of the frog, which is an extensor muscle. But the habitual posture of the frog is squatting, not standing, and in this attitude the hind limbs are folded in nearly full flexion. Accordingly, reflex postural activity is found in the flexor muscles of the hind limbs of the frog, and not in the extensors. In Brondgeest's classical experiment demonstrating reflex tonus in the hind limbs of the frog it was the flexor muscles which exhibited the tonus. On the other hand, in the cat, dog, and monkey (and man), it is the extensors which exhibit the tonus or postural activity.

Postural activity in man is essentially a continuous act of extension—extension of the head and neck, the spine, the hips, and the knees—with adduction and inversion of the feet.

The chief characteristics of postural activity as summarized by Sherrington are: (1) the low degree of tension it usually develops; (2) the long periods for which it can be maintained without fatigue; (3) the difficulty of obtaining by artificial—for example, electrical—stimulation reflex contraction at all closely resembling the postural contraction produced by natural stimuli; (4) the relative ease with which reflex inhibition interrupts the postural contraction; and (5) and (6) the "lengthening" and "shortening" reactions obtainable from muscles exhibiting postural contraction.

The tension developed is usually quite mild, but it may be considerable. Usually it is just enough to maintain ordinary postures, and when greater tension than that is required it is obtained by the same kind of contraction as that employed for movements. But this latter kind of contraction cannot be long maintained owing to fatigue.

A remarkable feature of postural activity is its insusceptibility to fatigue. It is well known that phasic or voluntary muscular contraction cannot be maintained for long owing to fatigue. But postural activity can be maintained for long periods—hours or even days—without appreciable signs of fatigue. It has been shown that the energy-cost of a posturally acting muscle is extraordinarily low as compared with voluntary contraction. Indeed, the contrast is so striking as to suggest that these two forms of muscular activity are fundamentally different.

But perhaps the most characteristic feature of postural activity is what Sherrington⁶ has called the "lengthening" and the "shortening" reactions. By this is meant the property which a postural muscle has of adapting itself to different lengths without change of tension. The lengthening and shortening reactions are best seen in cases of decerebrate rigidity, in which postural activity is free from the control of the higher centres. For example, if the extended knee be passively flexed, the limb remains fixed in the position to which it was moved. By this movement the quadriceps muscle is, of course, lengthened. But there is no increase of tension on the muscle. For, if the tension is measured by the weight which the muscle will just counteract, it is found that the weight is exactly the same whether the muscle is in the longer or the shorter state. In other words, the quadriceps adapts itself to and maintains the increased length with exactly the same tension that it possessed before it was lengthened. Similarly, when the flexed knee is extended the quadriceps adapts itself to the shorter length without change of tension. And in whatever way changes of posture are brought about—whether passively or by reflex or by voluntary muscular actions—these lengthening and shortening reactions are appended to the phasic contractions, so that different postures are maintained with the minimum expenditure of energy.

The afferent impulses which give rise to the postural reflex originate in the posturally acting muscles themselves—that is, it is a proprioceptive reflex, and the principal nerve centres concerned are situated in the paracerebellar nuclei. The higher parts of the brain, though not essential to the crude postural reflex, yet normally

exert a reinforcing and modifying influence upon it. In this way posture is influenced by the labyrinths, the cerebellum and the cortex cerebri.

Postural activity is acquired in early life. A newborn child cannot hold up its head, still less can it support its body in the upright position. This is not because the muscles are too weak, but because the nervous mechanism for these actions is not yet developed. In due course, by constantly repeated voluntary effort, the child learns to hold up its head, then to sit, to stand, and finally to walk. The upright attitude thus acquired laboriously by voluntary effort, then becomes habitual and subconscious, and is maintained by reflex tonic muscular activity (postural activity).

When the child begins to stand, the feet are flattened and everted by the body weight; this, again, is not because the muscles are weak, but because postural activity is not yet fully developed. When the child learns to walk, postural activity becomes established, and the feet are adducted and inverted—that is, the tendency to passive abduction and eversion is from that time onwards constantly resisted by reflex tonic muscular activity (postural activity).

Postural activity, then, being the normal mechanism by which the upright position of the body (including the normal attitude of the feet) is maintained, it is to postural activity and its modifications that we should first turn for an explanation when we meet with abnormal departures from the upright position.

SCOLIOSIS.

One of the commonest "static" deformities is scoliosis or lateral curvature of the spine, and gravity is, no doubt, an important factor in its development. The influence of gravity on an initial curve of the spine is to tend to increase it and to bring about those rotatory changes in the vertebrae which are so characteristic a feature of the established deformity.

But gravity by itself is responsible for scoliosis to a limited extent only. In its purest form gravity scoliosis is seen in those cases which arise directly as the result of some occupation, such as carrying heavy weights on one shoulder. In such cases a moderate amount of scoliosis may develop very slowly in the course of years. It is a true adaptation deformity. It cannot be corrected, and it shows little tendency to increase spontaneously.

When the bones are softened by disease the force of gravity may produce severe deformity, as is seen in rachitic scoliosis, though it may be noted that in rickets loss of tone is as marked a feature as bone softening. Again, when the spinal muscles are paralysed, gravity produces deformity, but in this case for a long time the structural changes are slight compared with the extent of the curvature. It is true that severe fixed deformity often occurs in paralytic cases, but, if such a case is compared with a case of postural scoliosis of the same degree, it is remarkable how flexible the paralytic deformity remains, and to what an extent it can be corrected, at least for a very considerable time.

The commonest variety of scoliosis—namely, postural scoliosis—differs in origin, course and development from all these other varieties. It is essentially a progressive deformity, and its progress is often rapid and out of all proportion to the mechanical factors involved.

It has been customary to include in the etiology of this deformity all sorts of conditions which may cause more or less constant curves in the spine. Thus, scoliosis has been attributed to faulty attitudes assumed by children at school, to inequality in the length of the lower extremities, to weak muscles, etc. But nearly every child sits with a crooked back when writing at a desk or table, yet few develop scoliosis. Boys sit just as badly as girls—or worse, yet scoliosis is far less common in boys than in girls. Inequality in the length of the lower extremities is much more common without scoliosis than with it. We all know many cases of short leg from one cause or another, in which there is a constant curve in the spine on standing, but they have not developed scoliosis. Some such cases, it is true, develop a certain amount of curvature which hardly alters at all in the course of many years. It is an adaptation deformity and quite different from the ordinary postural scoliosis.

General debility and muscular weakness is so common apart from scoliosis that it cannot alone be considered as a

cause of the deformity. In cases of actual paralysis of the spinal muscles—which may be taken as the extreme of muscular weakness—the deformity is almost entirely mechanical, and it differs in many ways from postural scoliosis. There is no greater mistake than to suppose that scoliosis is a deformity of weedy girls who are not strong enough to hold themselves up. Most of the cases seen in practice are of quite average muscular development.

It has been said that the ultimate cause of scoliosis is "spinal insufficiency"—one of those clever-sounding phrases which seem to mean a great deal, but which really mean nothing at all. Those who use the expression say that spinal insufficiency is a want of proportion between the weight which the spine has to carry and the "resistance" or carrying power of the spine. They do not say what the resistance is, nor in what way it is "insufficient."

Now, we have seen that the upright position of the body is maintained by continuous reflex muscular activity; that this postural activity is of a special kind, capable of being sustained for long periods without fatigue; and that it is controlled by a special nervous mechanism of its own. A faulty posture therefore suggests, in the first place, a faulty nervous mechanism or an abnormal nervous influence brought to bear upon that mechanism.

It has been mentioned that the labyrinths normally influence postural activity. The natural stimulation of the labyrinths arises from their position in space, and it has been shown experimentally by Magnus and de Kleijn⁷ that postural activity is influenced reflexly by the position in which the head is held. When the head is bent forwards postural activity is inhibited, not only in the extensor muscles of the head and neck, but throughout the length of the spine and in the lower extremities. Conversely, when the head is extended, postural activity is increased. In lateral flexion there is a corresponding adjustment of postural activity on the two sides.

These observations forcibly recall certain attitudes frequently associated with deformities, as, for example, the bent head familiar in cases of adenoids, myopia, neurasthenia, paralysis agitans, old age, and other conditions which are often accompanied by kyphosis. When the head is held erect the spine is usually straight and the lumbar lordosis well developed. Lateral flexion of the head may, I think, find a place in the etiology of scoliosis, either as a contributory causative factor or as determining the localization of the principal curvature.

The cerebral cortex exerts a modifying influence on postural activity, either directly or through the medium of the cerebellum. The hypotonia of cerebellar deficiency and the characteristic posture assumed in some cases of cerebellar disease are familiar to neurologists. It is an everyday observation that, other things being equal, the upright figure and the bent figure reflect two very different types of *mind*. The difference between the attitude of a street loafer and that of a trained soldier is not merely physical—it is far more mental. In the "setting up" of a raw recruit discipline, drilling, and so-called physical exercises, produce a mental change. By their means a habit is formed, at first conscious and with effort, but finally subconscious and ingrained.

These are variations of posture within normal limits. But I would put forward the view that abnormal postures also are primarily nervous or mental in origin—that postural scoliosis, in fact, originates as a functional nervous disorder. Mental fatigue or inertia—in fact, neurasthenia—leads to deficient postural activity, even though the muscles are well developed and voluntary muscular power is unimpaired. When postural activity is deficient the erect posture is imperfectly maintained, and under the influence of gravity the spine or part of it becomes curved. At first the spine can be straightened by voluntary effort, but, as we have seen, this form of activity cannot be sustained on account of fatigue. Postural activity is essential for sustained muscular action.

The dissociation of phasic and tonic or postural muscular activity is seen in an aggravated form in certain nervous diseases, as, for example, in myasthenia gravis. In this condition the patient is able to perform a voluntary movement, but the tonic or postural activity is absent from the affected muscles, so that the attitude assumed by the voluntary contraction cannot be sustained. Thus, the patient can raise the eyelids voluntarily, but cannot keep

them raised. The muscles voluntarily contracted rapidly tire, and the movement can only be repeated after an interval for their recovery. Normally, a postural contraction would be appended to the voluntary contraction, and the patient would be able to keep the eyes open without effort or fatigue.

The same phenomenon can be demonstrated by electrical (faradic) stimulation of the muscles, and gives rise to a characteristic reaction—the myasthenic reaction. If the muscle is stimulated repeatedly at short intervals with the faradic current, it gives at first a brisk response, but the succeeding contractions become weaker and weaker until at last no response is obtained with the strongest current. Or, if the faradic current is applied continuously, the muscle at first gives a brisk contraction which gradually fades away and disappears, although the current is still passing. After a short interval the muscle recovers its excitability, which, however, again disappears on repeated or continuous faradic stimulation. The galvanic reactions of the muscles are not affected.

I have found the myasthenic reaction to be present in cases of postural scoliosis. Naturally, one would not expect it to be so well marked as it is in myasthenia gravis, though in that condition it is, as a matter of fact, variable. But it has been demonstrable in many cases which I have examined for it, and I think it clearly indicates that deficient postural activity, and not muscular weakness, is the immediate underlying cause of postural scoliosis.

The frequent association of scoliosis with neurasthenia, and with a neuropathic family history, was noted twenty years ago by Oppenheim,⁸ and I have frequently noted the same among my own patients. Again and again I have seen scoliosis coming on apparently without cause in girls who were perfectly well developed, who went to good schools, and who lived apparently in the best conditions. Sometimes in such cases one would obtain a history of overwork—particularly, working for an examination. Sometimes a neurotic tendency or peculiar temperament would be noted in the patient herself. But most common of all, I think, is a history of neurosis of some sort or other in the parents or their immediate relatives. I have convinced myself that the mental condition is of the greatest significance in these patients—often far more so than the physical aspect of the deformity.

But while the functional origin of scoliosis may thus be accounted for, the development of the deformity requires further consideration. Gravity, no doubt, plays a part, but, as I have already said, the progress and fixation of postural scoliosis is often rapid and out of all proportion to the mechanical factors involved. The early fixation of a postural curvature is due to contracture of the soft parts in relation to the vertebrae, and I would suggest that this, in the first place, is of the nature of a functional contracture.

Some light may be thrown on the question of functional contracture by considering the occurrence of contracture in paralysed muscles and in their non-paralysed antagonists respectively. Structural fixation, as has been said, occurs slowly in paralytic as compared with postural scoliosis, and this is in accord with our experience of paralytic and non-paralytic contractures generally. In a case of drop-foot, when all the muscles of the leg are paralysed, shortening of the calf muscles occurs slowly, and for some time after it has begun it can easily be corrected by simply stretching the contracted muscles. But, in a case of drop-foot in which only the dorsiflexor muscles are paralysed, contracture occurs with great rapidity if the foot is not supported, and it firmly resists correction by mechanical means.

The two processes are quite distinct. Contracture in a paralysed muscle is due to loss of elasticity consequent on fibrosis in the degenerating muscle. In the case of non-paralysed muscle, physiologists are generally agreed that lengthening is as much an active process as shortening or contraction. When a muscle has contracted in response to a nervous impulse it requires another nervous impulse to initiate the process of lengthening. The natural impulse for lengthening a muscle is the direct outcome of the impulse for causing contraction of its antagonist. Therefore a break in the arc of reciprocal innervation has a twofold effect. Not only is the paralysed muscle unable to receive impulses to make it contract, but its non-paralysed antagonist is unable to receive the impulses necessary to make it lengthen. The latter, therefore,

having once shortened, remains shortened, and very soon its power of elongation is permanently lost.

In postural scoliosis we have a condition in which postural activity is deficient. The spine is curved to one side, and the law of reciprocal innervation holds good for the erector spinae muscles on either side of the curvature. In order to straighten the spine, the muscles on the convex side must be made to shorten, while at the same time those on the concave side must be made to lengthen. In early cases this can be done to some extent by voluntary action, but, as we have seen, this is quite different from postural activity. The nervous impulses necessary to bring about postural readjustment are not forthcoming. The muscles on the concave side, therefore, remain shortened, and, just as in the case of the non-paralysed antagonists of paralysed muscles, their power of elongation is soon permanently lost. The analogy holds good except for the fact that in the paralytic case the faulty innervation is due to an organic lesion, whereas in postural scoliosis it is functional—that is to say, the brain is unable to initiate the process of postural readjustment.

I would suggest that progressive functional contracture on the concave side of the curvature is the cause of the rapid fixation which occurs in so many cases of postural scoliosis. One is tempted further to speculate whether the structural changes both in the soft parts and in the bones are in any way connected with the sympathetic innervation of these parts. During recent years we have become so familiar with physical effects of functional nervous disorders—for example, cold and blue limbs, congestion, sweating, oedema, atrophy, contracture, peri-articular fibrosis, etc.—effects which are nutritional and therefore under control of the sympathetic, that it is no longer justifiable to assume, as once was done, that because a change is structural, therefore it cannot be functional in origin. I shall, however, return to this question in the second part of this article.

TREATMENT OF SCOLIOSIS.

If the foregoing inferences are correct it is obvious that they must have an important bearing on the treatment of scoliosis. How often does one see scoliosis appraised and treated almost entirely from behind the patient's back! If you want to know whether a patient has got scoliosis, look at her back. If you want to know why she has got it, and how to treat it, look at her face. The face is said to be the mirror of the mind, and you will find there and in her neurological history information of at least equal importance to any that can be obtained from examination of the back.

The principal cause of deficient postural activity is mental fatigue or inertia. Temperament, environment, occupation, and the general state of health are important factors. The child who sits badly at school and develops scoliosis is the child not with weak muscles, but with the overtaxed brain. It may be remarked that this depends more on the mental capacity of the individual than on the actual amount of work done. A clever child may be tempted to compete with older children, while a dull child may be overworked in merely trying to reach an average or a low standard. In such cases it is useless merely to introduce a course of exercises into the ordinary school routine. Either the routine should be radically altered or the child should be taken away from school.

Patients with scoliosis can be divided roughly into three classes—(1) an active type, apparently active in mind and body, and generally well developed; (2) a listless apathetic type, disinclined for exertion, poorly developed or flabby, and lacking interest in her surroundings; and (3) an obstinate type—lazy, careless of appearances, and difficult to influence. Each of these requires different treatment. The active type requires rest or at least curtailment of excessive activity—usually mental; the apathetic type requires stimulation, persuasion and re-education; the obstinate type requires vigorous stimulation, discipline and work.

It need hardly be said that the patient's general condition should be treated. As in neurasthenia generally, change of environment and occupation is often the ideal, but is not often attainable. Anaemia, constipation, adenoids, etc., should be treated if necessary. Strychnine augments the postural reflex, and is the basis of most "tonics," so it may often be given with benefit.

With regard to so-called physical exercises, postural scoliosis is due, not to muscular weakness, but to deficiency of the habitual subconscious state of reflex activity which normally maintains the upright attitude. The object of treatment, therefore, is not to strengthen or develop particular muscles or groups of muscles, but to redevelop a habit—the postural reflex. This may be done now in the same way as originally in early childhood—namely, by voluntary muscular effort constantly repeated until it becomes involuntary, subconscious and habitual—in other words, by exercises.

Exercises are only one of the means to this end. They should be designed to correct faulty postures as such, and not with the idea of developing particular muscles. Intermittent exercises are of little use in themselves. The patient must be got to strive for a constant effect. Her co-operation is essential, and to this end the personality of her instructor is of far more importance than the particular exercises done.

Restoration of the postural reflex is the basis of all curative treatment of scoliosis. If a curvature is flexible, so that it can be straightened by active or passive movement, it will be kept straight when the postural reflex is re-established, and the patient will be cured.

A curvature that is partly fixed by structural changes can be corrected only so far as it can be made flexible. The flexibility of a structural curve can often be materially increased by active and passive corrective movements, but rarely, if ever, can a structural curve be made completely flexible and therefore curable by these means. A moderate amount of fixed residual curvature may be held in check indefinitely, if the postural reflex is re-established. Such a curve, however, tends slowly to increase mechanically under the influence of gravity, just as an occupational or adaptation curvature does, and many such cases eventually show a poor return for the time, labour, and money that has been expended on their active treatment. Therefore, if a curvature is not held in check by a reasonable amount of active treatment, it should be given mechanical support.

With regard to forcible correction and the application of plaster jackets in scoliosis, it is known that, apart from its effect on the distortion of the ribs, this method, like others, depends for its success upon flexibility of the spine. Most orthopaedic surgeons will agree with the finding of the Scoliosis Committee of the American Orthopaedic Association (1916) that no case of fixed scoliosis has yet been shown to have been corrected in the anatomical sense by any method. At best, forcible correction is but a preliminary measure, and if it is not followed by restoration of the postural reflex or by efficient mechanical support, it is both useless and harmful.

The day has passed when it was customary to hang a so-called spinal support on every crooked back. Nearly everyone now treats scoliosis by exercises, and I think that exercises are indicated in the majority of cases. But I would plead for discrimination in their use, as in that of any other therapeutic measure. Some people have made almost a fetish of exercises. One sees cases so treated, literally for years, without any appreciable benefit, and a spinal support is often ordered only as a last resort. Some cases are quite unsuitable for treatment by exercises from the beginning. Exercises in many cases are given without appreciation of their real purpose. They are only one of the means to an end—namely, restoration of the postural reflex, and to attain this end it is the patient, and not merely her spinal column, that requires treatment.

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(To be continued.)

THE Department of Social Hygiene in the League of Red Cross Societies, whose headquarters are at Geneva, will in future be known as the Department for Combating Venereal Diseases. Mr. Walter Clarke has been appointed Chief of this Department and Lieut.-Colonel Ritchie, Assistant Chief. The League has published in English, French, German, Italian, and Spanish two pamphlets concerning the fight against venereal diseases, entitled respectively "The venereal diseases survey" and "Venereal diseases—a challenge to the Red Cross."

Oliber-Sharpey Lectures

ON
THE NATURE OF FLUTTER AND
FIBRILLATION OF THE
AURICLE.

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LECTURE II.—AURICULAR FIBRILLATION.*

IN the first of these lectures I have endeavoured to put before you the chief conclusions which we are able to form respecting that rather rare condition auricular flutter, and have stated that flutter as it occurs in man is due to a single wave circulating continuously. In this lecture I propose to deal with the much commoner and therefore more important disorder, auricular fibrillation. Now these two conditions are very closely related, as has been recognized for some years; they are known to be related, not only because of several resemblances, but because the one not infrequently passes into the other. Thus flutter of the auricle, as I pointed out some years ago, may usually be converted into fibrillation by the administration of heavy doses of digitalis; a conversion which is made use of in the present-day therapy of flutter. We shall see presently how the two states are related, and that circus movement underlies both. To explain the interrelation it is necessary for me to expand what I have already touched upon—namely, certain reactions of the auricular muscle to increased rate of beating. We must study in more detail the change which occurs in the length of the refractory period and in the rate at which the waves travel, as the rate of beating rises.

INFLUENCE OF HEART RATE ON THE REFRACTORY
PHASE, ETC.

The changes are somewhat complex, and may be illustrated most clearly by means of the accompanying diagram (Fig. 1). To the left of the diagram is a double-headed arrow, which represents the length of the auricular cycle when the heart is beating at a normal rate. This cycle is divisible into two parts, as indicated by the two double-headed arrows which stand just to the right of the first; it is divisible into the *refractory period*, during which the auricular muscle is in the excited or contracted state, and into the *responsive period*, during which the muscle is inactive or resting. I represent the refractory period in black and the responsive period in white, and the remainder of the diagram shows, on the basis of direct observation, how these two periods are proportioned to each other as the heart rate is raised. As the heart rate rises by equal increments—say from 100 to 150 to 200 to 250, etc.—so the length of the cycle shortens. If the lengths of the cycles, as these shorten, are charted vertically upon a horizontal base line (as in the figure), their ends join up to form a curve, the steepness of which gradually decreases. The curve of the end of the cycle approaches the base line, at first rapidly, but gradually less rapidly. The two lines will meet at infinity. The end of the refractory period also forms a curve which moves towards the base line (it is represented by the top edge of the black portion of the figure). Briefly, the refractory period of the muscle shortens as does the length of the contraction when the heart rate is raised. But the refractory period does not shorten to the same extent as does the length of the cycle; consequently the *responsive period* dwindles when the rate is advanced. It dwindles until, eventually and at a very high rate of beating, it disappears altogether. When this happens the muscle will not respond to every impulse reaching it; it breaks into what has been termed 2:1 (or half) response—that is to say, it responds only to alternate impulses entering it. That is but natural, seeing that only alternate impulses will fall during the responsive period of preceding cycles.

Now the precise manner in which this alternating response is foreshadowed in the mammalian auricle is

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