A British Medical Association Lecture ON THE NATURE AND TREATMENT OF AURICULAR FIBRILLATION.*

BY

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THE choice of this subject has been largely determined by three considerations: (a) the disease is very common, (b) its nature is comparatively well understood, (c) much can be done by adequate treatment.

Intelligent treatment of the condition must depend upon an appreciation of the functional pathology of the disease, any understanding of which, in turn, connotes a familiarity with the present teachings of cardiac physiology.

Normal cardiac contraction is determined by the rhythmic generation of impulse material at the pacemaker—that mass of primitive tissue lying in the right auricle, near its junction with the superior cava, known as the sino-auricular node. Experimental proof of this does not come within the scope of this paper, but can be found elsewhere very clearly presented.¹

The speed of the pacemaker is controlled by the interaction of (a) the vagus, stimulation of which slows the pacemaker, and (b) the sympathetic, which in turn raises the speed. The contraction impulse, having been formed in the sino-auricular node, spreads throughout the myocardium in definite orderly sequence. There is much evidence to show, and it is now generally believed, that the impulse spreads across the sheet of auricular muscle by contiguity of muscle fibre, and in no other way. Each auricular fibre is believed to hand on its stimulus to its neighbour, by which means the whole sheet is put into contraction, commencing around the great veins, and ending at the auriculo-ventricular junction.

Conduction of the contraction impulse is carried in turn to the ventricle, by means of the auriculo-ventricular node and bundle, and again in no other way, for total destruction of the bundle, by disease or experiment, prevents the auricular impulse from reaching the ventricle, and the auricular contraction is then followed by no ventricular response. The auriculo-ventricular bundle, on reaching the ventricular area, divides into right and left branches to be distributed to each ventricle respectively, by means of complex branching and arborization on the endocardial surface of the muscle, and ends in the Purkinje tissue, by means of which the impulse is believed to be handed on to the ventricular musculature.

There is, then, a complex and elaborate conduction system which carries the contraction impulse from the auricle, across the tissue of the auriculo-ventricular junction, and distributes the impulse to the ventricular muscle—an arrangement in sharp contrast to that of the auricle, where the impulse spreads, from its point of generation at the sino-auricular node, depends upon contiguity of muscle fibre only, and no conduction system exists.

It has long been known that the heart will respond to direct electrical stimulation, and if this be carried out at the apex of the auricular appendix, the speed of the heart can be raised above the speed dictated by the pacemaker; but if the speed be raised to 250 beats a minute, or thereabouts, a certain number of impulses fail to reach the ventricle. In fact, the auriculo-ventricular junctional tissue shows phenomena of fatigue before the cardiac muscle mass.

It is further known that stimulation of the vagus, particularly on the left side, is capable of reducing the conduction capacity of the bundle, even when the heart is not beating at high speed, and so a certain number of auricular impulses are prevented from reaching the ventricle—in other words, the auriculo-ventricular conduction system may be partly or completely "blocked"

to auricular impulses by disease or other damage to the bundle, or by vagal stimulation. When complete block occurs, the ventricle initiates its own rhythm, which is regular, but very much slower than normal pacemaker rhythm, and is usually about 32 a minute, but may be either higher or lower.

From this it follows that, when the auricle is beating at high speed, a very slight depreciation of the capacity to conduct possessed by the bundle will suffice to arrest a certain number of auricular impulses; in this way the ventricle may be guarded against the fatigue and damage of a high auricular rate, and a sufficient depression of conduction would be easily brought about by vagal stimulation of a mild grade.

A great deal of experimental work has been done upon the results of high-speed direct stimulation of the heart's auricular appendix. It is found that when the stimulation speed rises to 300 to 500 a minute the ventricular responses are usually grossly irregular in force and rhythm. In some cases cessation of stimulation is not followed by return to normal rhythm, but the events in the heart continue exactly as if electrical stimulation were still going on.

It is believed that this phenomenon is due to partial fatigue of the auricular muscle, with the result that a contraction wave runs round a ring of the musculature, probably that muscular band which embraces the mouths of the great veins. The impulse is believed to run in one direction only, and to act as the stimulus for the whole auricular muscle. The condition is known as circus movement; it can only occur at high speeds, and no definite organized muscular contraction results; the capacity of the auricular cavity is not reduced by the muscle contractions, the fibres of the muscle being in a condition of continual quiver.

There is evidence to show that at speeds of about 300 a minute the path of the circus movement remains identical, as does the path of its offsets, but that the auriculo-ventricular bundle is unable to conduct all the impulses reaching it (300), with the result that every other or every third beat only is allowed to reach the ventricle, constituting 2:1 or 3:1 block, and yielding a ventricular rate of 150 or 100 respectively. This condition is known as auricular flutter.

When, however, the speed of circus movement in the auricular muscle rises to 450 a minute or thereabouts the path of the circuit is constantly changing, as is the path of the offset waves, the progress of both being constantly impeded by fibres still fatigued, and therefore refractory to stimulation. The result is an irregular circus movement, constantly changing its course, and irregular offset waves which may, or may not, succeed in traversing the whole of the auricular muscle sheet. From this it follows that the arrival of impulses at the auriculo-ventricular junctional tissue will be both high in speed and irregular in rhythm. The consequent ventricular responses will show a like result, and the ventricular stroke, therefore, becomes grossly irregular and of high speed. This condition constitutes auricular fibrillation.

Our knowledge of this condition as it at present stands is due almost entirely to electro-cardiography, and any consideration of the disease must include some reference to graphic records obtained by this means. The normal electro-cardiogram demonstrates a wave due to auricular activity, which is easily recognized and identified as separate from the deviations due to ventricular activity. In auricular fibrillation the auricular wave disappears and is replaced by undulations which are irregular in rhythm, and which appear and disappear in the length of the tracing. The ventricular complexes are grossly irregular in rhythm and in size, but their form remains quite unaltered. From this it was concluded (a) that no organized sequential auricular contraction takes place, and (b) that the stimulus to ventricular contraction reaches the muscle by the usual paths—that is, the auriculo-ventricular bundle and its branches. The irregular and constantly varying undulations are attributed to the ever-changing course of the circus movement within the auricular muscle.

Clinically auricular fibrillation is too familiar to need much description: the pulse is perpetually and grossly

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irregular; the speed is usually raised, in which case all the beats do not usually reach the radial artery; there is a definite "pulse difference" between the apical rate and the radial pulse. Signs of cardiac insufficiency are usually present, but may be declared by any phase between undue pulse rise and dyspnoea on effort, up to fully developed orthopnoea and anasarca.

The grade of the disease is usually estimated by the speed of the resting pulse—in most cases the higher the speed the more the discomfort; but this is not constantly so—there is a group in which the cardiac speed is never high, but the cardiac insufficiency is marked and obvious; this, however, is the exception rather than the rule.

The rise in the cardiac speed is probably the largest factor in the heart's breakdown. There is no auricular stroke to fill the ventricle, so that any shortening of diastole must leave the ventricles inadequately filled, the muscle inadequately stretched, the ventricular stroke of poor force, and the consequent circulatory effect indifferent.

If by any means the ventricular speed can be controlled and reduced a definite sequence of events will follow: the prolongation of diastole will permit of adequate ventricular filling; the ventricular muscle fibres will be thereby sufficiently lengthened to yield a contraction of good force; the ventricular muscle will receive sufficient rest for full recovery after each contraction; the general nutritional state of the heart muscle by way of the coronary arteries will be materially improved.

The treatment of auricular fibrillation may be considered under two heads: (1) the control of the high ventricular speed with consequent general improvement of the circulation, and (2) the re-establishment of a normal cardiac rhythm.

For the control of ventricular speed drugs of the digitalis group have enjoyed a very long reputation, but the large number of preparations at present offered to the profession would argue rather that the results obtained are by no means always satisfactory, and a review of the effect of the drugs under scientific observation may not be out of place. The criterion of action of the drug must be ventricular slowing, for that is the most usual result aimed at in administration.

In judging of the effects of the drug three factors must obviously be considered: (a) the dose given, (b) the preparation used, and (c) the potency of the preparation. At the outset, for purposes of careful clinical observation, it becomes necessary to limit investigation to one preparation of one drug, and the vast bulk of the work has been done with tincture of digitalis. The use of other preparations of other drugs will not give easily comparable results. Even so, the potency of various tinctures has been shown to vary sufficiently to make comparisons difficult, and preparations are now frequently labelled "standardized by physiological assay" in an attempt to overcome this difficulty. Without entering into the technicalities of physiological tests of this sort many methods have been tried, but it must be remembered that effects produced in the artificially perfused heart do not necessarily represent results obtainable under clinical conditions, and that injection methods allow the whole contents of the tincture to act, whereas gastrointestinal absorption may only be possible to certain compounds in the galenical preparation.

A recent method which we owe to the Pharmaceutical Society's laboratories is to hold a standard powdered leaf, and from this to make a tincture by a standard method, to compare the results obtained by experiment with this standard tincture to results obtained under identical conditions with the unknown tincture to be tested, and to express the result in percentage potency of the standard. Even so, it is tenable that the result attained must be corrected by observation under clinical conditions, as matters at present stand.

The dose of the drug, the preparation used, and the potency of the preparation all having been fixed, there yet remains another factor. It has been clearly recognized ever since Mackenzie's early work on the irregular pulse that the results obtained by digitalis administration in auricular fibrillation are totally different from the results obtainable by giving the drug to the patients with high

speed pulses from any other cause,² the one possible exception being auricular flutter.

In using the ordinary pharmacopoeial dose of the drug it is well known that immediate results are not to be looked for. The drug is said to be dangerous because its action is cumulative, and, under ordinary dosage, benefit can be expected after about a week. It would appear from this that the drug must be allowed to cumulate in the body until a certain degree of concentration or saturation has been attained. On the other hand, it would seem somewhat illogical to induce the saturation point slowly while the patient is suffering all the distress and discomfort of complete cardiac failure. Mackenzie has shown that the full effect of the drug can be attained by giving the tincture at the rate of a drachm a day,³ and that the time occupied is five to seven days.

The matter was carried considerably further by Eggleston,⁴ who worked with a tincture which had been standardized by the Hatcher and Brody cat unit method. The minimal lethal dose per kilo of body weight of cat had been carefully determined, and the tincture was used by Eggleston in the treatment of auricular fibrillation, the action of the drug being carefully watched both clinically and graphically. By this means it was found that one cat unit was usually represented by one cubic centimetre of a good-class active tincture, and that the full therapeutic effect could be attained clinically by the administration of a dose calculated on the basis of 0.1 c.cm. per lb. body weight of the patient. This dose was administered in three portions extending over a period of eight to twelve hours.

This is now known as Eggleston's method of massive digitalis administration. Toxic results do not follow, as the dose is carefully calculated to fall short of the toxic amount. The method has permitted of a much closer and more satisfactory study of digitalis effects. Robinson⁵ has shown that some therapeutic effect is recognizable within two to four hours after administration, and that the full therapeutic effect is attained usually at the end of six hours. Pardee⁶ has estimated that the duration of the good effect is from six to ten days, and that the rate of elimination is about 25 minims a day.

Given by this method of massive dose, therapeutic results are very rapidly attained, but it is imperative that the potency of the tincture used be fully known.

A modification of this method has been in use in my hospital service—the full calculated dose at the rate of 0.1 c.cm. per lb. body weight of patient has been given as a haustus made up to 3 iij with sodium bicarbonate, aromatic spirits of ammonia, and chloroform water. Electrocardiograms have been taken before and every two hours after administration, until five or six tracings have been made. With the particular tincture used, and at this rate of calculation, a full therapeutic effect was not frequently attained, and the tincture would therefore appear to be somewhat weaker than that used by Eggleston. The T-wave of the electrocardiogram in Lead II was not usually inverted even at the end of eight hours, but the slowing effect was well marked in nearly all cases within that time. Toxic effects did not occur except in one case; the nature of the changes in this case are not understood, but the pulse rose considerably in frequency and the electrocardiogram became profoundly altered, suggesting some severe toxic effect upon the heart, but nausea and vomiting did not manifest themselves.⁷

Altogether 54 cases have been treated by this method. The average dose of the tincture administered was 13 c.cm.; the average pulse speed before administration was 140, whereas eight hours later the average speed was 91. The average duration of satisfactory effect was six days, so that small routine doses of the tincture were usually necessary at the end of a week. Vomiting occurred in two cases, both of which had been given a dose calculated on a basis of 0.15 c.cm. per lb. body weight, a rate which was too high for the tincture used.

That the effect of digitalis in auricular fibrillation stands by itself, and has no counterpart when the drug is administered to hearts at normal rhythm, is clearly shown in the following table, in which it will be noticed

that no slowing of the pulse takes place as the result of exhibition of the calculated dose.

	Age.	Dose.	Before.	After.
<i>Normal rhythm.</i>				
Wy.; F.	48	11.3 c.cm.	132	132
Bs.; M.	41	15.9 c.cm.	108	108
Sd.; M.	35	13.7 c.cm.	84	84
<i>Fibrillation.</i>				
Tn.; M.	56	11.2 c.cm.	144	90
Lp.; M.	41	15.6 c.cm.	132	108
Ht.; F.	57	13.0 c.cm.	144	84
<i>Fibrillation on a larger dose.</i>				
Cd.; F.	36	36.5 c.cm.	180	96 (vomited)
Wy.; F.	39	20.0 c.cm.	144	96 (vomited)

The clinical indications for this method of administration of the drug would appear to be clear—a large dose is only indicated to save time and suffering, and therefore only in the presence of cardiac breakdown of the major type, with dyspnoea to the point of distress, or marked anasarca.

Under such conditions the pulse speed, or rather the apical cardiac rate, is materially raised in most cases, and it is in these that the most brilliant demonstrable results are attained. At the same time the method has been used where cardiac failure with fibrillation is present in spite of an apical rate of under 100 a minute, and under these conditions the results are just as satisfactory and no untoward effects have occurred.

In a considerable proportion of cases the patient has already been given digitalis, or members of that therapeutic group in some form or other, and toxic effects are certain to result if a large dose be administered when the patient is already to some extent under the influence of the drug. In view of the calculated excretory rate of 25 minims a day, and the duration of effect as seven days,⁸ some estimate of the therapeutic position is best arrived at by calculating the excess of tincture received in the previous week over (175 m or) 3 iij, and deducting this from the therapeutic dose calculated at 0.1 c.cm. per lb. body weight.

A very real difficulty, however, remains with regard to the potency of the tincture which is actually administered to the patient; provided the preparation is in constant use and under careful clinical criticism, the difficulty is small, but this is only likely to be so in hospital service. At the same time, the calculation at the rate given above is sufficiently far removed from the toxic dose to permit of 25 per cent. increase in potency of any tincture over the normal, without producing any severe toxic signs.⁴ And moreover, since vomiting is an early toxic effect, it will probably mechanically protect the patient from further absorption, and thus from further damage. It may be remarked in passing that preparations of digitalis which cannot produce vomiting are likely to be inert in all other respects, and that the vomiting itself is a valuable indication. Want of response to digitalis therapy in auricular fibrillation is not a tenable statement, unless and until vomiting has been caused by the drug. It has been remarked, however, that the vomiting caused by a massive dose of digitalis differs materially from that which arises in the course of prolonged and continuous administration of the drug. Of the two cases in this series in which vomiting occurred one vomited twice within three hours, and the other four times within five hours; in both cases no further gastric disturbance was observed. This differs widely from the results of long-continued digitalis administration, in which, when vomiting starts, it is most likely to continue for four days, with much gastric disturbance.

As already stated, the administration of a massive dose of digitalis is only advocated as a measure of urgency, to overcome the suffering and discomfort always associated

with complete cardiac breakdown. This having been accomplished, there yet remains the future condition of the patient to be considered and treated. The beneficial effects of a massive dose usually begin to disappear at the end of six or seven days, the indication being a distinct rise in the pulse frequency. All the cases in this series have remained at rest in bed for at least a week after the initial large dose; usually at the end of that time the pulse has returned to 100 a minute or thereabouts. The patient is then allowed up for a short time each day, and is started on tincture of digitalis, 5 minims three times daily. It is usually necessary to increase this amount up to 10 minims thrice daily, in order to maintain the pulse at 90 a minute after half an hour's rest in the recumbent position immediately after the mid-day meal. Lewis has pointed out that in auricular fibrillation the pulse speed may be set to a nicety by means of digitalis;⁹ the speed and conditions quoted above are the ones which are aimed at.

Experience goes to show that one large factor in the causation of cardiac breakdown in auricular fibrillation is the cessation of digitalis dosage. This has been repeatedly emphasized in the past,⁹ and it is probably true that to enter upon digitalis life is much the same as to enter upon catheter life—continuance of the usage is imperative. The bugbear of "cumulative effect" still appears to sway the mind of the profession, but, as has already been pointed out, the drug is probably inactive until a certain degree of cumulation has taken place, and it is only the maintenance of this cumulative effect that will save the patient from disaster.

The precise method of action of digitalis yet remains unproven, but for purposes of therapeutic judgement it is wise to regard the drug as affecting the vagus.^{10 11} Sufficient drug must be used to stimulate the vagus up to a point where the degree of auriculo-ventricular block is just sufficient to allow impulses to pass at about normal speed from auricle to ventricle; when the block exceeds this in degree, and particularly when coupled beats, giving bigeminy, arise, the vagus is being overstimulated and vomiting is near.

An estimate of the daily excretory loss of the drug can be formed, as Pardee has pointed out, by determining the average daily dose necessary to maintain a certain clinical effect over a period of time. In the out-patient clinic the usual dose necessary for patients up and about and following such light occupations as auricular fibrillation will generally permit is 30 minims daily, and it is given in three daily doses. Over a large series of cases it is found that smaller doses require periodic increase, while larger doses require periodic decrease.

The general supervision and treatment of these patients calls for careful and frequent examination, and constant small adjustments of their lives, requiring experience and worldly wisdom, but the results attained are very satisfactory. No effect whatever must be looked for upon the irregularity of the pulse under digitalis treatment; the rhythm will remain as chaotically irregular as ever under correct dosage, but the pulse difference (the difference in speed between apical and radial counts) will be materially reduced, and in many cases will disappear altogether.

Any statistical study of the results of auricular fibrillation is rendered difficult by the usually long duration of the condition, and also by the fact that it may in some cases be "spasmodic," and occur in attacks of sudden onset, variable duration, and spontaneous disappearance.

Of 133 cases studied recently the sex incidence was equal, but a history of rheumatic origin was obtained in 50 per cent. of females, and in only 30 per cent. of males. The additional frequency of mitral stenosis in women, and, still further, the frequency of fibrillation as the third stage of stenosis, might have been expected to raise the female percentage much higher. The frequency of auricular fibrillation in chronic interstitial nephritis, bronchitis, and as a part of general senile changes could not be directly estimated. The group caused by exophthalmic goitre accounted for only 11 cases, 5 male and 6 female, but they all appear to have responded indifferently to digitalis. Parturition appeared to have been the immediate cause in 3 cases, 1 of which died and 2 responded very badly to

treatment. The importance of this group can hardly be overestimated, more particularly in view of a recent paper by Robinson,¹² in which 13 deaths occurred as the result of parturition in the 16 cases studied. Hemiplegia occurred in 5 cases, and death took place while under treatment in 14 cases, but this cannot represent the real mortality, for most of the patients attended the outpatient clinic, and such are frequently lost sight of, and cannot be traced. Poor results of treatment were obtained in 19 cases, presumably on account of extensive myocardial damage, although this was by no means always demonstrable by any diagnostic method—for example, in the exophthalmic goitre group. No digitalis was given to 14 patients, there being little or no sign of cardiac insufficiency.

Cases of typical auricular fibrillation are frequently reported in which the pulse speed is at, or about, 80 a minute, and, although grossly irregular, yet the pulse rise on effort is not great and the capacity of the patient for work is sufficiently high to allow him to continue fairly laborious occupations. It was in this group that no digitalis was used.

In a small number of cases there is a real difficulty in the administration of any digitalis, because the passive congestion of cardiac failure has itself already caused vomiting. It was thought that if the active principle of the drug were a glucoside, and glucose could be readily absorbed by the rectum, it might be possible to administer a massive dose by that route. The calculated massive dose of the tincture was so administered in two cases, but, although the enema was well retained, no effect upon the pulse speed was observed. This result corresponds well with results obtained by the administration of glucose by the rectum, in which no rise in blood sugar could be found, and it is probable that no absorption takes place.¹³

The treatment of auricular fibrillation by digitalis or its allies does not aim at the correction of the abnormal rhythm, nor at the cure of the causative condition, and no such result is attained. The drug is merely a means, and a very valuable one, for defending the ventricle adequately against the onslaught of auricular stimuli of high frequency, by which means the ventricle is given adequate diastole for reasonable filling and reasonable rest; apart from this, complete cardiac breakdown is both proximate and inevitable.

The principle underlying the quinidine treatment of auricular fibrillation is, of course, entirely different. Here a definite attempt is made to restore the normal cardiac rhythm. This line of treatment is not in the least applicable to patients showing any serious grade of cardiac insufficiency. In the majority of cases a preliminary course of digitalis is necessary to re-establish the circulatory balance, and it is usually believed that the high speed pulse is a definite contraindication to quinidine.

The question of potency in the preparation does not arise, since it is a crystalline substance of high purity, and is readily soluble in acid solution in the same way and much to the same degree as quinine.

The success of the drug turns upon the fact that the continuance of circus movement within the auricular muscle depends upon three factors—the length of the circuit, the impulse conduction time, and the refractory period of the muscle.¹⁴ Any considerable change in one or other of these three factors would terminate the circus movement, and quinidine has been shown to prolong the refractory period of the muscle to an extent sufficient to bring the circus movement to an end. Immediately this occurs the cardiac pacemaker comes into action, normal rhythm is re-established, and the auricular muscle performs a definite systole, thus once more filling the ventricle normally. The fact remains, however, that the primary cause of the state of fibrillation still continues, and that, therefore, relapse is almost inevitable ultimately.

It is generally believed that almost all cases of fibrillation can be forced into normal rhythm, provided quinidine be given over a sufficient length of time and in sufficient concentration, but my own experience is that when large doses are required normal rhythm does not remain long.

The present routine in my service is, in the first place, to reduce the pulse frequency to between 80 and 100 a

minute while at rest in bed. Quinidine sulphate is then given in 6-grain doses every two hours for five doses—that is, a maximum of 30 grains. Electro-cardiograms are taken before every dose, and examined, so that, should normal rhythm become re-established, no further quinidine is given that day. The next day the patient is given a mixture containing 1-grain doses of quinidine. This is taken three times a day, and is continued for six to twelve weeks. In my opinion quinidine administration is difficult, and probably should not be attempted, in the absence of electro-cardiographic control. Signs of toxic effect upon the heart are fairly frequent, even with the strictly limited dose detailed above, and although no untoward event has occurred, yet the rise in speed can be very alarming, even when adequate tracings are made every two hours.

In successful cases—those in which normal pacemaker rhythm becomes re-established—the patients themselves very generally recognize the difference, for the continuous palpitation of fibrillation completely disappears. Those that have been regularized more than once can frequently tell the precise moment at which the change from one rhythm to another has occurred.

In spite of the enormous theoretical benefit that should accrue when a fibrillating auricle again resumes a correctly timed and regular contraction, yet a considerable number of regularized auricular fibrillation patients consider themselves no better than they were when under adequate digitalis control. On the other hand, another group of patients will return immediately for further regularization whenever fibrillation recurs, because they recognize very clearly that their capacity for effort is very much higher under pacemaker rhythm than it can ever be during fibrillation.

However brilliant the results of quinidine administration may be in a small proportion of patients, it yet remains obvious that the routine treatment of auricular fibrillation must be carried out with digitalis.

This paper will have attained its object if it helps in any way towards an appreciation of the underlying processes of fibrillation, the objective of digitalis treatment, and, above all, an adequacy of dosage.

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Remarks

ON

THE CAUSATION OF ANGINA PECTORIS:

WITH A REPORT OF THE RESULTS OF SOME EXPERIMENTAL AORTIC LESIONS.

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THE failure of medicine to supply a consistently satisfactory method of treatment for angina pectoris renders necessary the careful investigation of any new procedure for which a claim of cure, or even amelioration of the symptoms, has been advanced. Such is presented by the introduction of surgery into this field, as instanced by the successful operations of Jonnesco, or of Coffey and Brown. On the correctness of the theoretical view of the causal lesion depends the justification for surgical intervention; if it be in the heart or aorta it is beyond the present scope of such procedure, but if the anginal syndrome is attributable to a nerve lesion or depends on the integrity of a nerve path, this line of treatment merits more serious consideration.