CLINICAL PROGRESS

Cardiac Pain
Anatomic Pathways and Physiologic Mechanisms

By James C. White, M.D.

William Harvey demonstrated to Charles I that the heart was insensitive to pain. The subject of this observation was the son of Count Montgomery, a young man who had miraculously survived an injury to his ribs and costal cartilages that left the beating heart exposed in an open cavity. Harvey observed that neither pricking nor pinching the epicardium evoked any sensation of discomfort. Two and a half centuries later, after the advent of local anesthesia, Lennander concluded that the intestines were likewise insensitive, because his patients at operation did not complain of any discomfort on pricking, cutting, or burning of these structures. These misconceptions arose because of the paucity of sensory endings in the heart and other internal organs. Spatial summation is necessary to activate a sufficient number of nerve fibers in order to breach the sensory threshold. Therefore a massive stimulus to a large area of intestinal wall or myocardium is required. When pain is evoked, it is likely to be peculiarly disagreeable, but difficult to describe and to localize. The normal physiologic stimulus for intestinal pain is distention and for the heart ischemia of the myocardium.

François-Franck, professor of physiology in Paris, suggested in 1899 that sympathectomy be done for the relief of angina pectoris. For this purpose cervical sympathectomy was first carried out by Jonnesco in Bucharest in 1916. A decade later Fontaine and Cutler, after reviewing a large number of case reports, found that this operation had given satisfactory relief of anginal attacks in only 60 per cent of the published cases. After further study of the sympathetic innervation and the discovery of the thoracic cardiac nerves, it was established that the characteristic pain in the precordium and arms can be effectively relieved. Cardiac pain is not transmitted by the vagus. Direct stimulation of its trunk in the human patient suffering from angina pectoris (René Leriche, personal communication) has failed to evoke an attack, and bilateral vagotomy in the dog did not prevent pain induced by experimental coronary occlusion. In our consideration of cardiac pain it is therefore most important to describe the sensory fibers that accompany the sympathetic cardiac rami and that must all enter the upper thoracic segments of the spinal cord over its posterior roots.

A final point of interest that deserves mention here is the method of tracking pain fibers. This purpose unfortunately cannot be accomplished by the ordinary method of severing a spinal nerve distal to its posterior root ganglion and tracing the course of its degenerating axons by the Marchi method. The majority of axons that transmit painful impulses to viscera belong to the C fiber group of Gasser and Erlanger. These are not myelinated and therefore do not take the osmic acid stain for degenerating myelin. The cardiac nerves have been studied in great detail by modern anatomists and their content of sensory fibers has been verified unequivocally by physiologic testing in the dog and by many observations after various types of cardiac denervation in man.

Anatomy of Cardiac Pain Pathways
Sensory Nerve Endings and Terminal Fibers in the Heart

Mitchell has illustrated numerous undifferentiated and fine beaded nerve fibers in the walls of the heart. As he stated, "they are best
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seen in adventitia, but finer plexuses appear in the media and intima, or perhaps just deep to the intima or endocardium. Of course these networks, especially those in the adventitia, are composed of afferent and efferent fibres, but in the special receptor areas the proportion of thicker and presumed afferent fibres is unusually high." No encapsulated endings have ever been detected in primate hearts, but the fine beaded terminals are similar to those that are concerned with reception of painful stimuli in the skin and cornea. Many of the fine strands may be sympathetic efferent fibers concerned with cardiac acceleration and others belong to the vagus, but some are certainly terminal sensory axons and are capable of transmitting pain. Some of the afferent fibers in their proximal course along the coronary plexuses develop a certain degree of myelination. Nettleship\textsuperscript{12} was able to demonstrate degeneration of these fibers after resecting the dorsal root ganglia of the upper thoracic spinal nerves. On the contrary, few degenerated after vagal section. It is unfortunate that the majority of pain fibers are unmyelinated and therefore cannot be traced by the Marchi method.

Cardiac Plexuses

The cardiac plexus is situated above the base of the heart between the aortic arch and the bifurcation of the trachea (fig. 1). The superficial portion lies in the concavity of the arch and the deeper part of the plexus behind it. According to Mitchell\textsuperscript{11} the superior cardiac nerve and vagal filaments from the left side enter the former, while all others enter the deep
plexus. This disposition is not a constant one and, as both parts are so interconnected, it is best to consider the cardiac plexus as a single unit. In it the sympathetic and vagal fibers intermingle and lose their identity, but there is a tendency toward a subdivision into right and left halves from which fibers are distributed to the heart along the 2 coronary arteries. Surgical interruption of the superficial portion of the plexus has been advocated for the relief of angina pectoris by Arnulf and pericoronary neurectomy by Fauteux. Both methods have proved capable of relieving pain.

**Sympathetic Cardiac Nerves**

The anatomic arrangement of the sympathetic cardiac nerves and their central pathways in the spinal cord and brain stem are illustrated in diagrammatic fashion in figure 2.

The paravertebral chains of ganglia run on the anterolateral surfaces of the vertebrae on each side of the spinal column from the first cervical down to the lower end of the sacrum. In the thoracic spine there is a ganglion for each vertebra, but the first thoracic is usually attached by an isthmus to the larger inferior cervical ganglion. This dumbbell-shaped structure, which lies in contact with the costovertebral articulation of the first rib, is known as the stellate ganglion. There are only 3 ganglia in the neck. The inferior or upper half of the stellate is connected with the middle cervical ganglion by a number of delicate fascicles that surround the subclavian artery (annulus of Vieuxens). The sympathetic chain from this point upward consists of a single well-defined trunk situated behind the carotid sheath on the fascia over the longus colli and
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Fig. 3. Sensory denervation of the heart in dogs by White, Garrey, and Atkins.9 These kymographic tracings are reproduced from article by White and Bland,22 with the kind permission of the publisher, Williams and Wilkins, Baltimore. After recovery from the preliminary neurosurgical procedure the effectiveness of denervation was tested by Sutton and Lueth’s4 method of interruption of blood flow in the descending branch of the left coronary artery. Changes in respiration in upper tracing denote persistence of cardiac pain. The signals in lower tracings represent periods of stimulation.

capitis muscles. Anterior to the second and third cervical vertebrae it broadens into a long fusiform superior cervical ganglion.

The white communicant rami, that carry cardiac accelerator and other autonomic as well as sensory fibers, join each of the upper 3 or 4 thoracic spinal nerves with the corresponding paravertebral ganglia. They are not found in the cervical portion of the sympathetic trunks. The gray rami, which carry postganglionic sympathetic fibers from the ganglia back to the spinal nerves, are present in the cervical as well as lower portions of the ganglionated chains. These distribute efferent sympathetic impulses to the blood vessels, glands, and smooth muscle of the upper extremity, trunk, and head. In the past it has been taught that all sensory fibers run in the white rami. This is not always true, as White and Sweet15 have evoked pain on stimulation of gray as well as white rami in conscious patients on the operating table. Nevertheless, as will be made clear below, there is no reason to assume that there are any sensory pathways of clinical significance between the heart and spinal cord above the first thoracic spinal nerve.

In addition to the communicant rami between the sympathetic chains and the spinal nerves, the cervical and upper thoracic ganglia give off visceral rami to the heart. The superior, middle, and inferior cardiac nerves, which arise from the corresponding ganglia, have long been known and were clearly illustrated in a superb plate by Lancisius published in 1728. With the possible exception of the superior, all contain sensory as well as sympathetic motor axons.
The thoracic cardiac nerves, accessory connections of variable importance, were first described in 1830 by Swan, but then entirely forgotten. They were rediscovered by the anatomists less than 30 years ago. These delicate rami are given off in a variable fashion from the second down to the third or fourth thoracic ganglia. Their demonstration by careful anatomic dissection did not necessarily imply that they were important accessory pathways in the transmission of cardiac pain. This, however, seemed to be a likely possibility in view of the frequent failure of cervical sympathectomy to relieve angina pectoris.

The transmission of pain over these structures was tested in dogs (fig. 3) by White, Garrey, and Atkins. The stimulus used to induce pain was Sutton and Lueth’s method of temporary occlusion of the posterior descending branch of the left coronary artery. This procedure invariably evoked evidence of definite discomfort in control dogs. In others, in which various types of denervation had been previously carried out, it was clearly demonstrated that: (1) bilateral vagotomy does not reduce pain after coronary occlusion; (2) bilateral resection of the stellate ganglia may reduce, but certainly does not eliminate pain after coronary occlusion; (3) after resection of the stellate and upper 4 thoracic ganglia no pain can be evoked; (4) after bilateral section of the upper 4 thoracic posterior spinal roots pain cannot be evoked.

This experiment proved, at least for the dog, that there are sensory axons transmitting painful impulses caudal to the cervical cardiac nerves and that all central connections must pass through the upper 3 or 4 thoracic sympathetic ganglia and the posterior roots of the corresponding spinal nerves. Fortunately clinical experience in man has shown that the dog is a reliable experimental animal in this respect, which is far from the case with transmission of pain in the spinal cord. It is now thoroughly established that pain often reaches the spinal cord over the thoracic, as well as the 2 lower cervical cardiac nerves; in these individuals removal of the stellate ganglia or even the entire length of the cervical chains will not relieve angina pectoris as long as the upper 3 thoracic ganglia with their rami are left intact. On the other hand, since painful impulses that reach these structures over the cervical cardiac nerves must descend caudally at least as far as the first thoracic ganglion, removal of the sympathetic chain down to the third or fourth thoracic will relieve pain consistently. Removal of the upper 3 thoracic ganglia is usually sufficient (table 1), but in view of possible more caudal connections (posterior fixation) it is safer to remove the fourth ganglion in addition. When this is done, the chances of subsequent regeneration are lessened as well. Destruction of the sympathetic ganglia in the upper thorax can be carried out either by surgical resection or by paravertebral injection of ethyl alcohol. This latter method is reserved today for only the poorest risk patients with coronary disease. In such individuals, who often cannot tolerate general anesthesia and a major neurosurgical operation, chemical destruction of these focal ganglia is a most valuable substitute (table 2).

Alternative methods of interrupting the peripheral autonomic fibers to the heart have been proposed by Fauteux and Arnulf. These consist of resecting short portions of the pericoronary nerves or the pre-aortic plexus through an anterior thoracotomy. These procedures interrupt vagal and sympathetic motor as well as their accompanying sensory fibers. Both seem illogical. Opening the pleura and operating on the exposed heart must involve more trauma than either extrapleural sympathectomy or section of posterior spinal roots. It is always more difficult to be sure of removing all the finer sympathetic rami in their

| Table 1.—Results of Thoracic Ganglionectomy in Intractable Angina Pectoris |
|---------------------------------|-----|-----|
| Total personal cases            | 17  |     |
| Complete relief of precordial and arm pain on denervated side | 13  | 77  |
| Residual slight pain in neck and jaw | 2   |     |
| Late partial recurrence         | 3   |     |
| Early failure (incomplete denervation) | 1   | 6   |
| Hospital deaths                  | 3   | 18  |

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peripheral plexuses than the well-defined paravertebral chains. A final consideration is the notorious proclivity of sympathetic axons to regenerate over short gaps. Patients so far reported have not been followed over sufficiently long periods to exclude this possible cause for recurrent anginal attacks.

**Posterior Spinal Roots**

When in-coming sensory axons from either the cervical or thoracic cardiac nerves have passed through the upper thoracic sympathetic ganglia they continue over the rami communicantes to reach the corresponding spinal nerves. After a short course within the intervertebral foramina they enter the posterior roots with other sensory axons. These come from the deeper tissues and skin over the medial surface of the arms and chest from the clavicles down to the level of the nipples. The cells of these sensory axons are situated in the posterior root ganglia, and their peripheral fibers differ from the efferent sympathetic preganglionic and postganglionic fibers in running nonstop without synapses through the paravertebral ganglia to the heart. Their central processes terminate in the posterior horn of gray matter, where they establish synapses with nerve cells of the secondary sensory neurons that run in the spinothalamic tracts. There is evidence, cited by Ruch, that there are insufficient secondary sensory fibers to supply all the in-coming primary pain fibers in the posterior roots. Therefore it is probable that visceral and somatic impulses often share a single pain fiber in the spinothalamic tract. In view of this arrangement it is easy to see why cardiac pain is referred by the sensorium to the precordial region and inner surface of the arms.

In planning a neurosurgical procedure for relief of cardiac pain, posterior rhizotomy is the best if the patient’s coronary circulation is adequate to permit an operation of this extent. In 32 patients, summarized in table 3, it has been successful in all but a single case. Failure in this instance, one of my own patients, can be ascribed to a technical error, as I failed to sever the important first posterior root. It is of vital importance to localize the first thoracic vertebra by an x-ray taken on the operating table and to remove all but the uppermost portion of its lamina. The roots from this first thoracic segment leave the cord at the lower border of the seventh cervical vertebra and pass through the dura at so high a level that they will not be seen unless a considerable portion of the uppermost thoracic lamina has been removed. After division of the posterior sensory roots regeneration is impossible and postoperative pain, which may be an annoying complication after resection of the sympathetic ganglia, as well as after paravertebral injection, is never a problem. In addition, bilateral denervation can be carried out at a single stage.

**Central Conduction of Cardiac Pain in the Spinal Cord**

The secondary sensory axons, whose neuron cells lie in the posterior horn, soon decussate and cross in the anterior commissure to the spinothalamic tract in the opposite anterior quadrant. Figure 2 illustrates their rostral...
course through the brain stem to the posteroventral nucleus of the thalamus. While no observations on the effect of high cervical cordotomy have been reported in cases of angina pectoris, it is probable that this operation would interrupt pain provided analgesia were complete to the lower level of the brachial plexus. In other varieties of visceral disease unilateral transection of the spinal pain pathway has interrupted pain from the biliary tracts, kidney, and intestine on the contralateral analgesic side. When the pain has been felt on both sides, as in intestinal obstruction, it has not been reduced on the ipsilateral side in which sensibility has remained intact. Cordotomy, however, is never necessary in cases of cardiac pain, as sympathetic denervation or posterior rhizotomy are more simple and innocuous procedures.

Perception of Cardiac Pain in the Thalamus and Cerebral Cortex

Pain-conducting fibers in the spinothalamic tracts terminate in the postrolateral and ventral nuclei of the thalamus (fig. 2). While more refined modalities of sensation from the surface of the body are carried upward to the postcentral cortex, it is doubtful if this occurs to any notable extent in perception of pain. The sensory cortex doubtless is the means whereby cutaneous pain is localized, but this ability is not developed for the viscera. In a number of total removals of a hemisphere that I have recently studied, the disagreeable quality of pain on the opposite side of the body has not been diminished, although the ability to localize accurately the point pricked, especially in the extremities, has been markedly reduced. On the other hand, when the thalamus has been destroyed by an abscess or tumor, all contralateral perception of pain has been abolished. While cardiac pain cannot be experimentally produced in man, I hope soon to test the extent to which pain on distention of the contralateral renal pelvis is altered after hemispherectomy. I doubt that it will be altered in any significant way.

In terminating this section on anatomy of the sensory innervation of the heart it seems appropriate to emphasize its therapeutic value. The effectiveness of interrupting the pathway either in the spinal roots or in the paravertebral sympathetic ganglia has been summarized in tables 1, 2, and 3.

It will be seen from a study of these data that failure to relieve precordial pain and arm radiation is extremely rare when it is certain that the known afferent pathways to the heart have been interrupted. After excision of the central end of the second rib complete resection of the first and third ganglia with their rami is often difficult. Similarly, when the anterior supraclavicular approach is used, exposure of the third ganglion is not always easy. At times pain-conducting fibers must pass through the fourth thoracic ganglion below as well. In performing a rhizotomy the surgeon, to be certain of cutting the important first thoracic posterior root, must remove nearly the entire lamina of the corresponding vertebra, as I have found to my chagrin in investigating a case that failed. Failures are of course more frequent after attempts to interrupt pain conduction through the sympathetic ganglia and their rami by chemical block with ethyl alcohol. In this group I have seen no failures provided the injection resulted in a Horner's sign and hot, dry arm indicative of effective interruption of the regional sympathetic vasoconstrictor and sudomotor outflow.

In selecting the method of denervation for a given patient it is best to be guided by the degree of cardiac reserve. Patients who have had recent coronary thrombosis, the sufferers from angina decubitus, or those who have aortic regurgitation and are threatened with cardiac failure are not proper risks for any major surgical procedure. With experience in paravertebral block it is possible to treat these patients by injecting alcohol with a reasonable chance of success and fair degree of safety. Section of the posterior sensory roots is undoubtedly the surest method of securing permanent relief, because regeneration of sensory as well as sympathetic fibers is occasionally seen after ganglionectomy, as well as after chemical blocking.

The apparent higher rate of mortality in my 17 ganglionectomies (18 per cent), as compared to the figure of 10 per cent after 30 rhizotomies,
is not of significance in this small series. It amounted to only 7.6 per cent in Olivecrona's larger series reported by Lindgren. If anginal attacks are purely unilateral, it should be somewhat safer to do a sympathetic denervation than a laminectomy. On the other hand, when pain radiates to both arms, rhizotomy, which can be carried out bilaterally, is undoubtedly a safer procedure than a 2-stage gangliectomy. Rhizotomy carries the lowest risk of disagreeable postoperative complications. After injection of alcohol pneumothorax is an occasional but not serious complication. Inter-costal neuralgia is more frequently troublesome, but this has lasted for prolonged periods in only 10 per cent of our cases and never for longer than 3 months. Postoperative discomfort has been equally troublesome in 1 of the patients after resection of the ganglia.

In following the surgically treated patients, particularly those who are having such frequent attacks of angina decubitus that they are in fear of imminent death and unable to rest, the results have been particularly gratifying. A number of my patients, rescued from the intolerable pain of frequent severe nocturnal attacks, are reported in detail in published accounts with Bland, with Smithwick and Simeone and with Sweet. Many recovered to regain a useful degree of activity and continued to live in comfort for long periods. One is alive, free of angina, and leading a moderately active life at 85, 19 years after her paravertebral injection. It would appear that sensory denervation of the heart, when medical methods fail, may prolong life by reducing pressor responses that accompany severe pain and prevent relaxation by day and sleep at night. Giving the patient a reprieve from suffering is often an effective method for promoting the spontaneous recovery of a more adequate coronary circulation.

Physiologic Mechanisms

Pain experienced in an attack of angina pectoris is felt characteristically beneath the sternum and down the inner surfaces of one or both arms, and at times in the little and ring fingers as well. Occasionally it may be referred to the neck or jaws. The typical area of reference is supplied by the first to fourth thoracic spinal nerves. As mentioned above, it has long been known that there are no white rami communicantes connecting the paravertebral ganglia with the spinal cord in the cervical region and that the thoracolumbar sympathetic motor outflow begins at the first thoracic spinal segment. It would appear that this must apply to afferent sensory, as well as sympathetic motor fibers, as cardiac pain is invariably relieved by cutting the upper 4 thoracic sensory spinal roots. When pain is referred to both arms, sensory denervation, either by dividing these posterior roots or by destroying the corresponding paravertebral sympathetic ganglia, must be carried out on both sides. On the other hand, when the arm on only one side is involved, pain is eliminated by a unilateral denervation. While attacks of unilateral angina usually radiate to the left arm, 7 of my 96 patients had purely right-sided involvement, and good results followed a right-sided operation in these cases.

The mechanism of pain occasionally felt in the neck and jaws is still not understood. A complete resection of the cervical sympathetic chain with removal of its superior, middle, and inferior ganglia had no effect whatever in relieving 1 of my patients with residual angina in the left side of the neck and lower jaw, after all pain in the precordium and arm had been controlled by removal of the upper thoracic sympathetic ganglia. Lindgren and Olivecrona subsequently reported that pain referred to the jaws can be interrupted by alcohol block of the mandibular or maxillary nerves.

James Mackenzie, who wrote so extensively about angina pectoris over a period of 30 years, summarized his ideas in his final monograph in 1924. He emphasized 2 points about cardiac pain, both of which have been proved incorrect in recent years. In the first place, he claimed that surgical relief of cardiac pain would endanger the patient's life because angina was an important warning signal of overexertion. This is fortunately not the case, because even after bilateral sensory denervation the patient continues to experience an adequate warning signal, even though he is completely spared from his former agonizing attacks. Painless equivalents persist as a sense of constriction in
the suprasternal notch, often accompanied by dyspnea and palpitation.

Mackenzie also thought that there were no spinal pathways for cardiac or other forms of visceral pain. He popularized the theory that all visceral pain was referred to the surface of the body in characteristic areas by means of a viscerocutaneous reflex. As illustrated in figure 4, he believed that painful discharges from a diseased organ travel centrally only as far as the posterior horn of gray matter in the spinal cord. He then assumed that there were no specific secondary neurons to conduct pain upward in the spinthalamic tract. According to Mackenzie's theory, cardiac pain, on reaching the posterior horn, sets up an irritable focus, which reduces synaptic resistance to such an extent that sensory stimuli from corresponding cutaneous dermatomes, ordinarily of subthreshold intensity, continue upward in the spinthalamic tract to reach the level of consciousness. This theory of cutaneous reference of visceral pain was adopted by Mackenzie from the previous writings of James Ross and Henry Head. Both these men, however, believed that there was also direct conduction of splanchnic pain, poorly localized in the midline. It is a pity that Mackenzie abandoned this concept and, through his prestige and frequent writings, confused this issue for nearly 50 years.

The theory of referred pain, although still a possible mechanism, must play a very minor role. Sir Thomas Lewis, in his monograph on Pain, stated that infiltration of procaine over the entire precordial area "failed to alter, in the least, anginal pain in an eminently suitable patient in whom the referred pain was focused..."
over the sternum and could be provoked, with regularity and by a constant amount of effort, both before and after thoroughly anaesthetising the affected part of the body wall.” Coher has reported the pertinent observation that 2 of his patients with high amputations of the arm developed angina pectoris with reference of pain to the phantom extremity. In other varieties of visceral pain a number of clear-cut observations prove that the sensation may persist with little or no alteration when all afferent impulses from the surface areas to which the pain is referred have been blocked.

It is therefore evident that pain from the heart and other viscera cannot be interrupted by any form of cutaneous denervation distal to the point of inflow of the sympathetic rami communicantes, over which visceral afferent impulses enter the posterior horn of the spinal cord.

There are good reasons why cardiac and other forms of visceral pain are localized so poorly. In the first place, all deep structures, not only the internal organs, but also skeletal muscle, fascia, and ligaments have very few painful nerve endings. The ability to localize pain, so accurately possessed by the skin and oral mucosa, is due to their rich supply of sensory endings. Of the deeper structures only periosteum has a sufficient supply to localize the source of painful stimuli with fair accuracy. The viscera differ in this respect in no way from skeletal muscle and ligaments, as Lewis showed in his experiments on himself and other workers in his laboratory. Injection of hypertonic salt solution in the upper thoracic interspinous ligaments was indistinguishable from cardiac pain. On injection of the fascia overlying the rectus abdominis muscle the discomfort evoked resembled an attack of abdominal colic. Furthermore, as Ruch has pointed out, the number of pain fibers in the spinothalamic tract is far less than the number entering the posterior sensory roots. This means that many primary visceral and cutaneous afferent neurons must share the same secondary spinothalamic fibers in order to reach the thalamus and sensorium. As a result, the individual will refer painful impulses from the viscera to the area of more commonly experienced cutaneous sensation. It is therefore not surprising that visceral pain is referred to certain characteristic superficial areas that are innervated from corresponding levels of the spinal cord.

In the case of the heart the dermatomal distribution of the first and second thoracic nerves includes the inner surface of the arm and forearm together with the hypothenar eminence and a variable portion of the little and ring fingers; the anterior divisions of the second, third, and fourth intercostal nerves innervate the precordial region above the nipple line. On stimulation of the stellate and second thoracic sympathetic ganglia in the course of operations under local anesthesia by my colleague, Dr. William H. Sweet, and myself, a number of patients have complained of pain difficult to localize in the upper thorax and occasionally radiating down the arm. The sensation evoked, however, has not resembled an attack of angina pectoris.

Why pain in angina pectoris is referred only to the anterior chest wall and skips the lateral and posterior divisions of the intercostal nerves is not known. This is true of many forms of pain from the abdominal viscera as well. Only pancreatic and renal pain are commonly felt in the back as well as in the abdominal wall. There is also no adequate explanation for the sense of constriction in the suprasternal notch that persists after complete interruption of the sympathetic cardiac pain fibers. It is possible that this sensation is transmitted by vagal fibers and also that some form of vago-trigeminal reflex mechanism may account for the pain that is occasionally referred to the jaws; yet no pain of any sort was experienced by a patient in whom I observed Leriche stimulate the exposed vagus in the course of a resection of the stellate ganglion under local anesthesia.

**Summary**

It may be concluded that the pathways of cardiac pain have been thoroughly established, with the exception of the conduction of anginal attacks occasionally referred to the neck and jaws. Afferent impulses traverse axons that travel in the cervical and thoracic sympathetic cardiac nerves. In the case of the cervical pathway, all impulses on entering the paravertebral
ganglionated chain in the neck must descend to the upper thoracic level before they can gain access to the spinal cord. Other impulses reach the 3 superior thoracic ganglia via the more direct thoracic cardiac nerves. Both the cervical and thoracic fibers join the spinal nerves over the communicant sympathetic rami. After passing through the intervertebral foramina, they enter the posterior roots and terminate in the lateral horn of the spinal cord. Here they establish synapses with secondary afferent neurons of the spinothalamic tract, decussate to the opposite anterior column, and are carried rostrally to the nucleus ventralis posterolateralis of the thalamus. This is the principal locus in the brain for the perception of visceral pain. In contrast to well-defined cutaneous sensibility, there is no cortical area for exact visceral localization in the postcentral region of the cerebral cortex.

Another factor in the poor localization of cardiac pain is the paucity of sensory endings in the heart. A third appears to be the limited number of secondary sensory fibers in the spinothalamic tracts. These central axons must be shared with other impulses from the surface of the body. As a result, pain from the heart is in large part referred to the cutaneous distribution of the upper 4 thoracic spinal segments. Accounting for the superficial reference of visceral pain by Mackenzie’s theory of a viscerocutaneous reflex is no longer justifiable. Even after cutaneous afferent fibers are interrupted by procaine or amputation of the arm, pain from the heart can still be felt over its previous distribution.

Neither stimulation of the vagi nor interruption of transmission in these nerves has been found to have any beneficial effect in patients suffering from angina pectoris. Sensory deervation of the heart must therefore be carried out by destruction of the upper ganglia in the thoracic sympathetic trunks or by severing the corresponding posterior spinal roots.

**Summario in Interlingua**

Il es permissibile asserir que le vias de transmission de dolores cardiac ha essite precise mente establite, con le exception del conduction de attaccos de angina que se refere a vices al collo e al maxillas. Impulsos afferente transversa axones que viajan in le cervical e thoracic nervos cardiac sympathetic. In le caso del via cervical, omne impulsos que ha entrare in le catena ganglionate paraverteboral in le collo debe descender al nivello supero-thoracic ante que illos pote obtenir accesso el medulla spinal. Altre impulsos attinge le 3 gangliones supero-thoracic plus directemente via le nervos cardiac thoracic. Le fibras cervical e thoracic se junge ambes con le nervos spinal supra le communicante ramos sympathetic. Post passar per le foramines intervertebral, illos entra in le radices posterior e se termina in le corno lateral del medulla spinal. Hic illos estabili synapses con afferente neurones secundari del tracto spinothalamie, decussa al opposite columna anterior e es portate rostramente al nucleo ventral posterolateral del thalamo. Isto es le loco principal del cerebro pro le perception de dolores visceral. In contrasto con le ben-definite sensibilitate cutanee, il non existe un area cortical pro le exacte localisation visceral in le region postcentral del cortice cerebral.

Un altere factor in le pauco precise localisation de dolores cardiac es le paucitate de terminations sensori in le corde. Un tertie factor es apparentemente le restrainite numero de secundari fibras sensori in le tractos spinalie. Iste axones central debe etiam servir altre impulsos ab le superficie del corpore. Le consequentia es que dolores in le corde es in grande parte referite al distribution cutanee del 4 superior segmentos spinal thoracic. Explicar le referentia superficial de dolores visceral per medio del theoria de Mackenzie de un reflexo viscerocutaneae ha perdite omne justification. Mesmo post que fibras afferente cutanee es interrumpite per procaina o per amputation del bracio, dolor ab le corde pote ancora sentir se in su previe distribution.

Ni le stimulation del nervos vage ni le interruption del transmission in iste nervos ha potite exercer un effecto benefic in pacientes qui suffre de angina de pectore. Disnervation sensori del corde debe per consequente esser ecutate per medio del destruction del gangliones superior in le trunco sympathetic thoracique o per dissecar le correspondent radices spinal posterior.
REFERENCES

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