Lumbar Sympathectomy for Peripheral Arteriosclerosis

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Lumbar sympathectomy as treatment for obliterative arteriosclerosis of the legs has developed in accordance with better understanding of the physiology of the collateral circulation. Its importance is demonstrated by the results observed in 44 extremities followed postoperatively up to 14 years. The indications for sympathectomy and method of case selection are discussed at length. As experience has broadened, more advanced cases have been operated on, and good results have been obtained.

FIFTEEN years have passed since the senior author began to use lumbar sympathetic ganglionectomy in the treatment of obliterative arteriosclerosis of the extremities. In 1934 it generally was conceded by vascular surgeons that patients with degenerative (arteriosclerotic) peripheral vascular disease were not satisfactory subjects for ganglionectomy. It was questioned how such a procedure could hope to influence the flow of blood through arteries permanently occluded by the arteriosclerotic process. Nevertheless, an increasing number of surgeons have employed this method of treatment with growing confidence.1-4, 6, 20, 22 It is our purpose herein to review our progress in this field, giving special attention to selection of cases for operation and to report our experience with a modest number of cases, many of which have been followed for extended periods of time.

Re-examining the early concepts of vascular pathologic physiology, one is aware of the confused thinking of those times. For example, vasoconstriction was improperly named "vasospasm." We now know that in most cases of obliterative disease, whatever vasoconstriction is measurable is the normal vasoconstrictor element, or what is left after "vasoconstrictor recession." This is the authors' term for progressive loss of vasomotor tone that is a feature of these diseases. Estimates of vasomotor tone were used to differentiate the "spastic" from the "occlusive" diseases, basing judgment entirely on how closely the normal was approximated after induced vasoconstrictor paralysis. We now recognize that this concept is invalid, that in the early stages of peripheral obliterative arterial disease there may be normal or only slightly reduced vasoconstrictor capacities; that as occlusive disease progresses this element of vasoconstriction gradually recedes, and eventually may reach a stage where it is no longer demonstrable. It is now known, too, that it is not necessary to open occluded major vessels to increase the blood flow to an extremity. Because the collateral circulation is under vasomotor control, sympathectomy can be depended on to increase the total circulation in extremities with vasoconstriction, whether or not the ability of the major vessels to transmit blood is thereby improved. This increase may occur even in parts where vasoconstrictor paralysis tests are inconclusive,2, 18 although more reliably in areas where such tests are definitely positive.

Although it is well recognized that the total circulation of an extremity can be increased by sympathectomy, there is some disagreement about the distribution of the increased blood flow. It is conceded that the circulation through the skin of the hand or foot is markedly increased. Whether or not this occurrence is detrimental to muscle blood flow in the forearm or leg is problematic. The persistence of intermittent claudication after sympathectomy is frequently observed. This has been accepted as an indication that muscle circulation is adversely affected. However, slow improvement in intermittent claudication also has been

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Contrary to a somewhat prevalent contention there are no satisfactory data that demonstrate a decrease of muscle circulation caused by sympathectomy; whereas it has been shown that muscle circulation may even be increased under conditions of stress. However, even if the operation did no more than improve skin circulation its worth would be definite. Most of the serious complications of peripheral degenerative arterial disease begin as traumatic or infectious lesions in the skin. The increase of skin circulation which follows sympathectomy is an important factor in the healing of those lesions, at times making the difference between preservation and amputation of the extremity. The theory that the deeper tissue may not share in the increased blood flow should not deter the surgeon from performing the operation.

In 1934 our ideas on the treatment of vascular disease began to change. Under an erroneous preoperative diagnosis of thromboangiitis obliterans we performed sympathectomy on an extremity with a pregangrenous toe which we later found due to degenerative arterial occlusion. We had thus committed the "surgical crime" of performing lumbar sympathectomy for peripheral arteriosclerosis. The result was excellent. We came to an early realization that, in the operation of sympathetic ganglionectomy, normal sympathetic nervous tissue was removed to produce a nonspecific functional effect. We could find no valid reason for denying operation in properly selected cases of arterial insufficiency due to peripheral arteriosclerosis. Accordingly, and encouraged by our previous success, we deliberately subjected our first case of peripheral arteriosclerosis to lumbar sympathetic ganglionectomy in 1934. From 1934 to 1948, 44 extremities with the same process were similarly treated. The authors present herewith their experiences with these cases.

Selection of Cases

In the earlier years of our experience patients were selected for operation according to the following criteria: (1) the response to conservative management was unsatisfactory; (2) vasomotor studies under controlled conditions showed satisfactory vasoconstriction potential.

The conservative management consisted of suction-pressure therapy (long since discarded), intermittent venous compression, Mecholyl by iontophoresis, vasodilating drugs, circulatory exercises, foot hygiene, and other measures. A response was judged favorable if symptoms were alleviated, or if there was improvement in the force of the pulses, the response to elevation and dependency, the character of the tissues, the temperature of the part under ordinary conditions of living, and the condition of any ulcers already present. Progression of the arterial insufficiency during conservative management as indicated by enhanced symptoms and unfavorable physical findings, including the onset of ulceration or enlargement of ulcers already present, was an added incentive to operation. Conservative management was not instituted in extremities with extensive ulceration, gangrene and infection in which major amputation was obviously unavoidable; nor was it used in cases of rapidly progressing thrombosis of main and collateral arteries when delay in performing a lesser amputation would compromise the chances of saving a functioning limb.

Estimation of the degree of vasoconstriction was made in most cases by measuring the surface temperatures after vasoconstrictor paralysis had been induced by procaine block of the posterior tibial nerves under controlled temperature conditions. In a few cases subarachnoid block, paravertebral sympathetic block, or reflex dilation (hand warming of Landis-Gibbon) were used. After careful comparison posterior tibial nerve block was selected as the most accurate and reliable method of securing the desired information. The attendant superposable anesthesia gives indisputable evidence of a successful and complete block of the more readily affected autonomic fibers, the test is ambulatory, discomfort is minimal, and there is little or no risk. In over 450 such tests the method has proved its value in the study of a wide variety of vascular and neurautonomic disturbances of the extremities.

The tests were performed as follows: The legs were exposed to a constant room temperature of 16 to 20 C. for 30 to 60 minutes until the surface temperatures of the toes reached...
19 to 23 C. Skin temperatures of selected sites were then measured with a thermocouple. The posterior tibial nerves were infiltrated with procaine solution. When complete anesthesia, anhidrosis, and loss of superficial reflexes were obtained the block was considered successful. Thereafter skin temperatures were measured every 10 minutes until each site reached a maximum. The normal surface temperature after peripheral nerve block is at least 30.5 C. Vasoconstrictor recession was thought to be indicated by failure of the temperature to reach this level and the degree of recession is directly proportional to the temperature deficit. Extremities in which skin temperatures reached maxima of 28 C. or over were considered to be good subjects for ganglionection; between 26 and 28 less optimism was professed; if the rise failed to reach 26 C. operation was eschewed.

Cautiously at first, and with more courage as time advanced, extremities which showed what was considered to be a great degree of vasoconstrictor recession were treated by sympathectomy. Of 6 extremities so treated, the results were good in 5. Some extremities, the preoperative maximum digital surface temperatures of which after block were in the range of 19 to 23 C., and the resting early postoperative surface temperatures of which were in the same range, showed progressive rise of resting surface temperatures over a period of months, reaching normal and near normal values (30.5 C.). Others in the group that showed seemingly marked vasoconstrictor recession displayed healing of ulcers and/or moderate rises of postoperative resting temperatures. Other writers1-3 have decried the use of sympathectomy in extremities with little or no vasoconstrictor potential, claiming that extensive gangrene was precipitated by ganglionection. Our gratifying experiences with this group do not allow us to accept this concept. Furthermore, a review of the reports in which extensive gangrene was alleged to have been precipitated by ganglionection leaves considerable doubt as to the role of the denervation itself in the unhappy result. We have not performed ganglionection on extremities with marked vasoconstrictor recession which, in addition, had extensive chronic ulceration or gangrene, extremities which were obvious candidates for amputation, in which sympathectomy could not hope to effect satisfactory improvement.

The favorable postsympathectomy course of several of those cases that had little warming after nerve block indicated that surface temperature warming after vasoconstrictor block could not be relied on alone as a criterion for selection of cases for operation. This has been pointed out by other workers.11, 16, 18, 19, 21 Especially is this true in those with occluded main arteries, where the capacity of the smaller vessels for vasoconstriction may be difficult to demonstrate by this test because of the lack of availability of sufficient blood to produce significant warming. The presence of sympathetic nervous activity can be further demonstrated by increased sweating, pilomotor activity and reflex superficial venospasm, and by observing the effect of a cool environment on the surface temperatures. The latter test is performed by exposing the patient, trunk lightly clothed, in a cool (18 C.) constant temperature room for one hour. If significant vasoconstriction is present, the skin temperature of the foot and lower leg will approach that of the room. As vasoconstrictor recession advances, the cold room stimulus will cause progressively less reduction of skin temperature. The temperature of the tips of the toes is not so important as the response of the entire foot and ankle.16, 21 The cooling reaction is similar to the basal vascular tone evaluation described by Naide and Sayen11 and Rector.16 Extremities with advanced ischemia will remain cool throughout the exposure period, showing a slow fall of temperature. The demonstration of the presence of vasoconstrictor potential by skin temperature warming after nerve block, by fall in surface temperature in a cool environment, or by the presence of objective signs of vasoconstrictor activity indicates that the circulatory status of the extremity will be improved by sympathectomy. Even in those extremities in which one has not been able to demonstrate vasoconstrictor potential, in which vasoconstrictor recession has been complete, sympathectomy may be of considerable benefit. Thus, as our experience with sympathectomy for arterio-
sclerosis has increased, our indications for operation have gradually been liberalized. Extremities which in our early experience were refused operation because of lack of sufficient skin warming after nerve block, have been accepted for sympathectomy, with less and less evidence of vasoconstrictor potential. The liberalization of indications has progressed to the point that at present we now advise sympathectomy in every case of progressive arterial insufficiency due to degenerative arterial disease, whatever the stage, so long as hopelessly extensive gangrene or ulceration is not present and so long as the patient is able to stand the operative procedure with little risk.

How long should one persist in conservative management before sympathectomy is advised? If the arterial insufficiency progresses under treatment, sympathectomy should be done in any stage of the disease unless the patient is an obvious candidate for amputation. If the symptoms and signs of arterial insufficiency remain unchanged over a three month period of conservative management, operation is indicated. Extremities with mild degrees of arterial insufficiency and with one foot pulse present may be treated conservatively over extended periods, so long as the insufficiency does not progress. The extremity deprived of part of its normal blood supply may hold its own under ordinary conditions; but comparatively mild insults, bacterial, chemical, mechanical or thermal, are prone to precipitate ulceration or gangrene and threaten the integrity of the limb. Sympathectomy should be advised when the threat is apparent and when the operation gives good promise of alleviating or removing it. The measure of available vasoconstriction may prove in the future to be less important for case selection, but more valuable as a prognostic guide. (See chart 1.)

Procaine block of the lumbar sympathetic nerve as a preoperative test for the effect of sympathectomy on rest pain or claudication has been most dependable in our hands. The psychologic aspects of the test and the proximity of the somatic nerves are some of the complicating factors which interfere with the interpretation. The transient increase in circulation effected by procaine sympathetic block is not comparable to the long continued vasodilation produced by sympathectomy, and is therefore no true guide to the effect of chronic vasodilatation in relieving pain.

In making the judgment for or against operation, the general condition of the patient must, of course, be taken into account. The operation of extraperitoneal lumbar sympathetic ganglionectomy as described by the senior author and in experienced hands, does not cause shock, has a surprisingly low mortality and morbidity incidence (see Technic), and is extremely well tolerated, even by patients in the older age groups. Consultation with an experienced internist should be routine in all patients over 60 years of age when operation is contemplated. The vital functions of the circulatory, renal, and central nervous systems should be carefully evaluated and pitted against the risk of operation, even though it is slight. Progressive renal failure with increasing nitrogen retention, and actual or imminent myocardial failure would contraindicate surgery; but a coronary occlusion from which the patient has recovered or severe diabetes would not of themselves rule out operation. In good risk patients both sides can be done at a single sitting; when desirable because of the general health, and in all patients over 65, the operation is performed on one side at a time. The second side may be done 5 to 10 days later.

Technic

All ganglionectomies here reported were performed by the senior author. Excepting the first 2 cases, the muscle-splitting extraperitoneal technic developed by him and reported in detail in 1937 was employed. It has proved eminently satisfactory, and has been adopted as the routine method in this clinic. One alteration in the operation originally described has been made to facilitate exposure of the L-2 ganglion; the internal oblique muscle is split just inferior to the costal margin, instead of lower in the flank.

Attention is called again to the frequent variations in the anatomy of the lumbar paravertebral ganglia. The latter can be identified with certainty only by their connections with the segmental nerves, not by their relations to the vertebrae. Those connections often cannot be some out accurately at operation.

The extent of ganglionectomy proposed, and usually achieved is excision of L-2 and L-3 ganglia,
the chain between them, and the attached rami. The upper ganglion usually lies just caudal to the medial lumbarcostal arch of the diaphragm. The L-3 ganglion often is thick, and stellate in shape, with a short but rather heavy posterior ramus. Although the rami excised have not been found to have ganglion cells by our pathologists, the existence of those cells in the rami, especially near the junctions with the spinal nerves or roots, has been described by Orloff,\textsuperscript{13} and by Alexander and co-workers.\textsuperscript{1} We attempt, therefore, to excise as much of the rami as feasible. We have been pleased by the absence of clinical evidence of regeneration following the lumbar ganglionection described above.

In the exposure and removal of the sympathetic chain there is a tendency for the assistant to allow the medial retractor (Royle) to press upon the abdominal aorta. The retraction is tiresome, and constant vigilance is required to prevent the sagging of the retractor. If the aorta contains calcified or fatty plaques, undue pressure may cause them to be fractured, setting fragments free in the blood stream to act as emboli distally. Furthermore, pressure on the aorta may diminish the blood supply to the extremities beyond the critical minimum, causing gangrene and thrombosis. In the infrequent cases of gangrene that have followed sympathectomy, which have been ascribed by some authors to the removal of the ganglions in itself,\textsuperscript{3, 7} the possibility of aortic compression has received little consideration. That must be kept in mind, as well as the fall of systemic and local blood pressure previously mentioned.

Our choice of anesthetic agents has shifted from spinal anesthesia to the combination of nitrous oxide oxygen with intravenous Pentothal sodium, with curare supplements if additional muscular relaxation is needed. With the latter anesthesia there is much less chance of hypotension with its dangerous central and peripheral sequelae. Ether has not been used because of its ill effects in some diabetic patients.

When indicated in a good risk patient, a one-stage bilateral lumbar ganglionection can be performed. In patients older than 64 years, we perform the operations 5 to 10 days apart.

**Results**

This report deals with 44 extremities treated for various degrees of arteriosclerotic vascular disease by lumbar sympathic ganglionection between 1934 and 1948. Each extremity has been considered a separate clinical unit for follow-up studies (see Case Histories).

The operations were performed 36 times on male patients and 8 times on female patients. The average age was 53.8 years. There were 24 right extremities, and 20 left. Fourteen of the 44 cases were diabetic. There were no operative or postoperative hospital deaths.

**The Relief of Symptoms**

The most prominent symptoms were intermittent claudication, pain, and numbness. The shortest duration of symptoms prior to the first examination at this clinic was 10 days; the longest 8 years.

**Intermittent claudication** was present before operation in 35 extremities. In 12 the symptoms were completely relieved, and in 11 various degrees of improvement were noted after sympathectomy. In 9 there was no change, and in one the claudication became somewhat worse. (See table 1.)

The tabular expression of data, however, is not adequate to describe the effects of ganglionection on claudication. For example, of the 9 cases who continued to have claudication equal to the preoperative condition, one could use a power sewing machine for a full eight hour work day after operation, but could not do so before; and 4 others were limited in walking after the operations by painless fatigue instead of by cramping pain. One of the patients who obtained only slight relief of claudication was enabled, after sympathectomy, to work full time as an elevator operator.
The relief has been sufficient in 2 other extremities to permit the patient to swim as much as two miles, a feat not possible before sympathectomy. In this group, also, 2 extremities were completely relieved of pain if walking years. Recently, very slight claudication has occurred after walking four blocks.

Rest Pain. None of the 14 extremities that were free of pain before operation developed pain afterward. At least 16 of 24 extremities

was slow; and another claimed 55 per cent improvement. Of 2 patients markedly improved, one walked many miles before claudication interrupted him, and another (2 extremities) was completely relieved for five in which pain was prominent or were completely relieved, either immediately or within a few weeks after operation. Two cases had temporary exacerbations of pain many months postoperatively, possibly following local arterial

CHART 1. Correlation of results of vasomotor block with postoperative improvement.

CHART 2. Effect of sympathectomy on rest pain in 38 extremities. Data were inadequate in 6 extremities.
thrombotic episodes, which subsided spontaneously. It will be seen on chart 2 that those with the more severe pain gained the greater relief.

Numbness was present in various parts of 14 extremities (see chart 3) before operation.

Objective Changes after Sympathectomy

Pulse and Postural Color Changes. The changes in the pulses and the postural color responses are indicated in table 3. The satisfactory estimation of pulses and postural color changes depends on a long experience with vascular disease. With physicians so experienced, the grading is surprisingly consistent, but variations in individual interpretations are bound to occur.

The preoperative status of the pulses and color changes are seen in table 2. The high proportion of abnormal findings is demonstrated.

In scrutinizing the postoperative data on pedal pulses and postural color responses, one

![Chart 3](http://example.com/chart3.png)

**Chart 3.** Effect of sympathectomy on numbness in 31 extremities. Data were inadequate in 13 extremities.

**Table 2.** Preoperative Pulses and Postural Color Changes*

<table>
<thead>
<tr>
<th>Pulse</th>
<th>Dorsalis Pedis</th>
<th>Posterior Tibial</th>
<th>Popliteal</th>
<th>Femoral</th>
<th>Elevation Ischemia</th>
<th>Dependent Rubor</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>33</td>
<td>34</td>
<td>27</td>
<td>1</td>
<td>13</td>
<td>8</td>
</tr>
<tr>
<td>+</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>7</td>
<td>12</td>
</tr>
<tr>
<td>++</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>17</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>+++</td>
<td>3</td>
<td>4</td>
<td>11</td>
<td>23</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>Total diminished pulses</td>
<td>41</td>
<td>40</td>
<td>33</td>
<td>21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total abnormal postural color changes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Normal Pulse is +++
Normal Postural Response is 0

In 13 others the records of that symptom were inadequate. One patient with diabetic neuropathy who had no preoperative numbness complained of it slightly for several months after operation. At least 7 of the 9 extremities in which numbness was a major complaint were completely relieved of the symptom.

**Table 3.** Effects of Lumbar Sympathectomy on Pedal Pulses and Postural Color Changes

<table>
<thead>
<tr>
<th>Pulse</th>
<th>Dorsalis Pedis</th>
<th>Posterior Tibial</th>
<th>Elevation Ischemia</th>
<th>Dependent Rubor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unchanged</td>
<td>31</td>
<td>32</td>
<td>20</td>
<td>24</td>
</tr>
<tr>
<td>Improved</td>
<td>10</td>
<td>7</td>
<td>21</td>
<td>15</td>
</tr>
<tr>
<td>Absent before but present after</td>
<td>8</td>
<td>7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Improved to normal</td>
<td>4</td>
<td>3</td>
<td>15</td>
<td>3</td>
</tr>
<tr>
<td>Worse</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>No Data</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
is impressed with the great improvement in the latter. On the other hand, the effect of operation on pulses is not nearly so dramatic. It is of considerable interest, however, that the dorsalis pedis pulse could be felt postoperatively in 8 extremities in which it was impalpable before operation; the same was true of the posterior tibial pulse in 7 extremities. The return of pedal pulses is an event most uncommon after other methods of treatment of this disease. The improvement in postural color responses may occur without improvement in pulses, and is a more important evidence of improved skin circulation. This is in agreement with our present concept of the benefits to be derived from sympathectomy.

Surface Temperature Measurements. With few exceptions, all extremities in this collection had estimations of the degree of vasoconstriction before and after operation. The technic of study has been described above.

The acceptance of the amount of skin warming after vasomotor block as a prognostic guide has proved itself in general. This is most clear if preoperative test elicited a near normal response. The excellent results in nearly all of 14 extremities which had warm skin after vasomotor block are compared with the inferior results in the 28 extremities which were less warm (chart 1).

Although we have no charted data to illustrate it, our clinical impression is definite that the skin temperature responses immediately after operation closely paralleled those obtained from nerve blocks. That is, a like amount of warming, or lack of warming was noted. On the other hand, there has been no consistent relation between preoperative tests and skin tem-
perature measurements at long intervals after sympathectomy. (See Charts 4, 5, 6.)

The reasons for the lack of correlation have been explained in part earlier in this paper in the section on Case Selection. However, the explanation is incomplete. Several variable factors are difficult to control, especially in clinical studies of this type. Sympathectomy produces increases of skin temperature by preventing peaks of vasoconstriction of central nervous or reflex origin even in cool extremities, but does not abolish the changes in peripheral blood flow which may result from humoral, drug, and local mechanical or thermal influences. Would advance beyond rescue by peripheral collateral vessel efficiency.

There have been many cases in whom the postoperative skin temperatures have remained as high as or higher than the preoperative testing level for years (chart 4). Also, there are several cases in whom the skin temperatures have dropped as the years have passed (chart 5). Of greater interest are those several patients (cases 4, 10, 11, 33, 34) in whom repeated skin temperature measurements over the years have shown swings from coolness to warmth, or the reverse. In those cases the errors of measurement, including the temporal influences

Furthermore, sympathectomy is enlisted to promote the development of collateral circulation and to maintain the peripheral vascular bed as widely open as possible for indefinitely prolonged periods of time. We have repeated this assertion several times for emphasis. Although there have been testimonials that ganglionectomy may alter the chemical and histologic abnormalities of the arteriosclerosing process, until the evidence for that is indisputable, we do not propose that it alters the advance of the damaging changes in the large blood vessel walls. It is possible that, in some cases in whom sympathectomy provides great benefit, if survival of the patient was long enough, the occlusive disorder in the limbs of all sorts, may be explicative, or intermittent obstructive and recuperative processes may have occurred, which were not made obvious by the patient or the examiner.

Ulceration

Ulcers of varying extent were present in 12 extremities at the time of sympathectomies. In 10, healing followed sympathectomy, and ulcerations did not reappear during the period covered by this report.

Deep ulcers in one foot and ankle did not heal (see case history 33), and the extremity was amputated at the low thigh level eight months later.

Ulceration occurred in 4 extremities after
sympathectomy. In one instance (case history 13), an ulcer of the third toe which existed at the time of sympathectomy healed afterward, but recurred three months later. Healing followed local debridement. The ulcers that developed in the 3 other extremities during the years after sympathectomy healed when the causes were corrected (ill fitting shoes in 2 cases, and chronic paronychia in the third).

Amputations

Three amputations were performed on the 44 extremities treated by sympathectomy. A toe was amputated for deep ulceration 44 months postoperatively (extremity 13).

A low thigh amputation was performed on one patient after sympathectomy in the face of persistent ulceration and pain, and when conservative measures failed to halt the process. This patient was afflicted with chronic alcoholism, and failed to provide adequate foot hygiene (extremity 33).

Another thigh amputation was performed about one and one-half years after sympathectomy because of new ulcers on the foot. This amputation was done in another city.

In both cases that had major amputation, sympathectomy was performed in the face of very poor prognosis on the basis of preoperative studies: cold feet, presence of ulcers, atrophy of soft tissues, poor responses to nerve blocks.

Three patients who had had previous thigh amputations were treated for arteriosclerosis of the remaining extremity by sympathectomy. Continued good function and comfort has been reported for all 3 legs 13, 36, and 50 months postoperatively, respectively.

Case Histories

The following are brief descriptions of the 44 extremities that form the basis of this report. All skin temperature studies were performed in a controlled cold room as described in the body of this paper.


Extremity 2. Same patient as extremity 1. Moderate claudication in right leg one and one-half years. No pulse except for subnormal femoral. Severe postural color changes. Maximum toe temperature 31.7 C. Right lumbar ganglionectomy 1934. No claudication after 46 months, some improvement in postural color changes. Maximum toe temperature 34.4 C. at 43 months.

Extremity 3. J. L., a 55 year old male. Severe claudication for five years, recent severe rest pain. No pulses except for subnormal femoral. Moderate postural color changes and trophic changes of nails. Ulcer at tip of malleolus. Maximum toe temperature after nerve block 31.6 C. Right lumbar ganglionectomy 1935. Follow-up 153 months. Claudication not changed, but rest pain completely relieved. Postural color changes unimproved. Maximum toe temperature 30.6 C. after 12½ years. Patient had recurrence of small ulcers eight years postoperatively with rapid healing. Unsympathectomized left leg showed progressively disabling occlusive disease. Right leg maintained improved status through complete follow-up period.

Extremity 4. H. E., a 51 year old male. Diabetes in poor control for four years. Myocardial infarction in 1934. Severe intermittent claudication for two years with severe rest pain and numbness in more recent months. Normal popliteal and femoral pulses, moderate postural color changes. Maximum toe temperature after nerve block 32.8 C. Left lumbar sympathectomy, 1936. Follow-up 69 months, during which time claudication was unchanged, but rest pain and numbness much improved. Two years postoperatively pedal pulses reappeared, but disappeared at three years. Maximum temperature around 31.0 C. for three years, and then a slow drop off to 26.9 C. at 67 months. Died eight years postoperatively; myocardial infarction.

Extremity 5. B. S., a 66 year old male. Diabetes, angina pectoris, arrested pulmonary tuberculosis. Intermittent claudication 9 years. Weak popliteal, normal femoral pulse, slight color changes. Maximum toe temperature 30.4 C. after nerve block. Right lumbar sympathectomy 1937. Five months postoperatively pedal pulses were easily felt, although subnormal. Maximum toe temperature 30.2 C. This patient had some discomfort in early postoperative period, because of warmth in the foot.


Extremity 7. A. H., a 60 year old male. Mild
diabetes, severe intermittent claudication right leg four months, subnormal posterior tibial, popliteal, and femoral pulses, moderate color changes. Maximum toe temperature after nerve block 20.2 C. Right lumbar sympathectomy 1939. Eight and one-half year follow-up, during which time all symptoms were relieved. Postural color changes unaffected. Foot pulses absent after 104 months; but maximum toe temperature 30.9 C. Was able to perform eight hours of standing guard duty without symptoms. Mild cerebral vascular accident 1937.

Extremity 8. Same patient as extremity 7. Severe claudication in left leg for months. Near normal pulses in foot and leg. Moderate color changes. Maximum toe temperature after nerve block 30.3 C. Left lumbar sympathectomy 1939. Complete relief for 104 months. Pulses absent at end of follow-up, although toe temperature 30.6 C.

Extremity 9. R. M., a 49 year old male. Moderate claudication in left leg and mild numbness for 48 months. No pulses except for subnormal femoral. Marked pallor of foot accompanied by pain. Maximum toe temperature after nerve block 30.3 C. Left lumbar sympathectomy 1939. At end of one year had palpable foot pulses, unchanged claudication, no pain or numbness. Maximum toe temperature 30.2 C. Died six years postoperatively; myocardial infarction.


Extremity 12. G. M., a 63 year old female. Claudication, rest pain and numbness for 5 years. All severe at time of first examination. No pedal pulses, normal femoral and popliteal pulses. Some elevation pallor. Maximum toe temperature after nerve block 29.9 C. Acute popliteal arterial occlusion. Left lumbar sympathectomy 1940. Patient had severe femoral thrombophlebitis postoperatively, which prevented evaluation of claudication and rest pain. Postural color changes became worse, but dorsalis pedis pulse appeared and remained up to three and one-half years postoperatively. During second year, gangrenous area on fourth toe appeared and healed in eight weeks. Died of cerebrovascular accident four years postoperatively.

Extremity 13. G. H., a 51 year old male. Diabetes, hypertension, general arteriosclerosis. Extensive calluses on feet. No symptoms except for gangrenous ulcer on third toe. Maximum toe temperature after nerve block 28.1 C. Left lumbar sympathectomy, 1941. Postoperatively, ulcer healed progressively but recurred with osteomylitis. Healed again after debridement. Ulcer of great toe two and one-half years postoperatively after burn. Healed promptly. Successful extensive osteotomy of tarsus three years postoperatively. Amputation of second toe for recurrence of ulcer three and one-half years postoperatively. Worked full time as janitor six years postoperatively, with normal pedal pulses and maximum toe temperature, 28.8 C.

Extremity 14. Same patient as extremity 13. Right leg. Chief difficulty was recurrent ulcers of toes and heels under calluses. Maximum toe temperature after nerve block 22.7 C. Right lumbar sympathectomy 1941. Ulcers healed promptly and remained so during six year follow-up. No change in patchy analgesia of foot. Maximum toe temperature 31.2 C. six years postoperatively.

Extremity 15. H. P., a 56 year old male. Moderate claudication, mild rest pain, and numbness for one year. No pedal pulse, normal popliteal and femoral pulses, slight postural color changes. Maximum toe temperature after nerve block 30.1 C. Right lumbar sympathectomy 1942. Increased claudication during two and one-half year follow-up. Severe at end of follow-up period. However, patient successfully operates a power machine, using right leg for six hours daily, without pain. Pedal pulses appeared and remained postoperatively. Maximum toe temperature 30.5 C. postoperatively.

Extremity 16. A. M., a 42 year old male. Moderate claudication, moderate rest pain one year. No pulses distal to diminished femoral. Moderate postural color change. At time of operation, small ulcer on fourth toe. Maximum toe temperature after nerve block 30.4 C. This patient had been discharged from the army because of leg cramps. Right lumbar sympathectomy 1942. Ulcer healed promptly postoperatively. Worked full time as a street car operator throughout follow-up period. Maximum toe temperature 31.4 C.

Extremity 17. Same patient as extremity 16. Similar but milder symptoms for six months. Left lumbar sympathectomy 1942. Pedal pulses increased to near normal, with maximum toe temperature of 30.9 C. 45 months postoperatively. This patient
had only L-3 ganglion removed and had continued sweating of upper two-thirds of extremity.

**Extremity 18.** J. T., a 53 year old male. Moderate claudication for eight years. No pedal pulses, diminished femoral and popliteal pulses, moderate postural color changes, diabetes. Maximum toe temperature after nerve block 29.7 C. Right lumbar sympathectomy 1943. Claudication somewhat improved. Patient died five months postoperatively; coronary thrombosis.

**Extremity 19.** Same patient as extremity 18. Same symptoms and findings as right. Maximum toe temperature after nerve block 30.4 C. Left lumbar sympathectomy 1943. Similar results.

**Extremity 20.** T. A., a 52 year old male. Moderate hypertension and heart disease. Moderate intermittent claudication for one year with recurrent severe rest pain. No pulses distal to femoral. Slight postural color changes. Maximum toe temperature after nerve block 20.5 C. Right lumbar sympathectomy 1943. Thirty-three months postoperatively he had no pain or claudication. Pedal pulses reappeared although not normal. Maximum toe temperature 28.6 C.

**Extremity 21.** 57 year old male. Sudden onset of symptoms of severe claudication, numbness and mild rest pain three months before treatment, suggestive of popliteal artery thrombosis. No pulse distal to femoral. Advanced postural color changes. Maximum toe temperature after nerve block 28.2 C. Left lumbar sympathectomy 1934. Claudication improved, pain relieved for two and one-half years postoperatively, during which time posterior tibial pulse returned to normal. Patient died 35 months postoperatively; myocardial infarction.

**Extremity 22.** Same patient as extremity 21. Moderate claudication and moderate numbness one year. Weak dorsalis pedis pulse; marked elevation pallor. Maximum toe temperature after nerve block 31.6 C. Right lumbar sympathectomy 1934. Five months follow-up.

**Extremity 23.** S. B., a 54 year old female. Diabetes observed for six years before operation. Progressive diminution of pedal pulses noted, until posterior tibial absent and dorsalis pedis weak. Postural color changes moderate. Claudication moderate, but numbness severe. Patient had femoral thrombophlebitis four years before; subsequent increased sweating of foot and epidermophyosis. Maximum toe temperature after nerve block 31.6 C. Left lumbar sympathectomy 1944. No claudication or numbness noted during four year follow-up. Pedal pulses returned to normal, although postural color changes unaffected. Maximum toe temperature 31.3 C. at 45 months.

**Extremity 24.** C. K., a 56 year old female. Severe claudication for three months. No pulses distal to femoral, although five months before all pulses found to have been normal by another examiner. Probable femoral artery occlusion. Postural color changes marked. Maximum toe temperature 32 C. Right lumbar sympathectomy 1944. Claudication gradually improved; absent from one and one-half years on. Dorsalis pedis pulse present from two years on. Maximum toe temperature 33.5 C. at 45 months.

**Extremity 25.** Same patient as extremity 24. Moderate claudication for three months. This patient has rheumatic valvulitis and hypertension. No pulses distal to femoral. Maximum toe temperature after nerve block 32.4 C. Left lumbar sympathectomy 1944. Similar improvement during 45 months follow-up. Maximum toe temperature 33 C.

**Extremity 26.** J. M., a 60 year old male. Diabetes. Left thigh amputation three years before. Edema of right foot two years. Weak dorsalis pedis pulse, no postural color changes. Maximum toe temperature after nerve block 30.2 C. Right lumbar sympathectomy 1944. Edema of foot for three months, then lessened. Dorsalis pedis pulse returned to normal. During 41 months of follow-up, patient had no claudication, pain or numbness. Maximum toe temperature 32.4 C. at 41 months.

**Extremity 27.** H. M., a male 64 years old. Moderate claudication, and severe rest pain six months. No pulses distal to femoral. Moderate postural color changes. Maximum toe temperature after nerve block 27.8 C. Right lumbar sympathectomy 1945. Claudication improved, and rest pain completely relieved during four years of follow-up. Patient continued employment as hoisting engineer. Maximum toe temperature 30.4 C. at 32 months.


**Extremity 29.** J. J., a 64 year old male. Previous left thigh amputation for gangrene. Patient had no symptoms on right side, with normal pulses. Maximum toe temperature after nerve block 30.5 C. Right lumbar sympathectomy 1945. Maximum toe temperature 30.5 C. at 13 months. No symptoms during one year follow-up.

**Extremity 30.** V. P., a 57 year old male. Rapidly developing pain, claudication and coldness two weeks before operation. Suggestive of popliteal artery thrombosis or embolus from higher atheroma. No pulses below femoral, slight postural color changes. Left lumbar sympathectomy 1945. Continued slow improvement during 33 months postoperatively with relief of pain and numbness and 65 per cent improvement in claudication; this despite no change in pulses or postural color changes. Maximum toe temperature 26.8 C. at 33 months.

**Extremity 31.** J. R., a male 52 years old. Abrupt onset of symptoms two weeks preoperatively. Femoral artery thrombosis in midthigh. Claudication moderate. No pulses below femoral, moderate–severe postural color changes. Right lumbar sympa-
Sympathectomy 1945. Improvement during nine months follow-up. Foot pulses appeared postoperatively, postural color changes almost cleared.

**Extremity 32.** E. M., a 62 year old male. Frost bite of right foot at 17, followed by cyanosis, and excessive calluses. Left thigh amputation for gangrene two years before. Severe claudication, moderate rest pain and numbness for two months. No pulses below femoral. Severe postural color changes. Small ulcer on fifth toe and extensive plantar calluses. Maximum toe temperature after nerve block 26.4 C. Right lumbar sympathectomy 1945. All symptoms relieved during four year follow-up, although postural color changes only moderately improved. Ulcer healed promptly. Maximum toe temperature rose to 34.6 C. at three years, but fell to 27.2 C. at four years.

**Extremity 33.** G. S., a 65 year old male. Severe claudication and severe rest pain for two years, during which time patient had ulcer at ankle. Patient was a chronic alcoholic. No pulses below weak femoral, advanced postural color changes. Maximum toe temperature after nerve block 22 C. Sympathectomy offered as last hope after all symptoms could not be controlled. Right lumbar sympathectomy 1945. Although ulcer healed, it broke down within six months. Patient was not relieved of pain. Low thigh amputation eight months postoperatively. Complete obliteration of femoral and popliteal arteries found.

**Extremity 34.** J. R., a 63 year old male. Pernicious anemia one year. Severe claudication and numbness during three years before operation. No pulses below diminished femoral; moderate elevation pallor. Maximum toe temperature after nerve block 29.7 C. Left lumbar sympathectomy 1946. Mild claudication during ensuing one and one-half years. Maximum toe temperature 22.4 C. at that time.

**Extremity 35.** Same patient as extremity 34. Parallel symptoms and findings. Maximum toe temperature after nerve block 27.9 C. Gangrenous ulcer of first toe present at time of operation. Right lumbar sympathectomy 1946. Ulcer healed promptly postoperatively with almost complete relief of claudication one and one-half years postoperatively. Maximum toe temperature only 21 C.


**Extremity 37.** Same patient as extremity 36. Moderate claudication and severe rest pain for two years. Similar findings on right leg. Left lumbar sympathectomy 1946. Relief of pain, increase of claudication similar to right leg. Maximum toe temperature 30.7 C. at 20 months.

**Extremity 38.** H. W., a 50 year old male. Moderate claudication and occasional rest pain for five years. No pulses, advanced postural color changes. Ulcer on second and fifth toes during 10 days before operation. Maximum toe temperature after nerve block 26.2 C. Right lumbar sympathectomy 1946. Patient followed for only three months, during which time rest pain relieved, claudication unchanged. Ulcer healed. Low thigh amputation done elsewhere one and one-half years postoperatively for recurrent ulcer.

**Extremity 39.** Same patient as extremity 38. Parallel symptoms and findings, duration two years. Maximum toe temperature after nerve block 38.5 C. Left lumbar sympathectomy 1946. Relief of pain and unchanged claudication during three month follow-up period.

**Extremity 40.** G. S., a 43 year old male. Frost-bitten fingers and toes in childhood. Cold sensitivity. Sudden onset of leg symptoms eight months before operation with moderate claudication and rest pain and numbness. No pulses distal to femoral, advanced postural color changes. Maximum toe temperature after nerve block 30.4 C. Right lumbar sympathectomy 1946. Relief of rest pain and numbness, no change of claudication during three month follow-up. Immoderate use of alcohol and tobacco.

**Extremity 41.** M. A., a 58 year old female. Diabetes 15 years. Occasional mild rest pain for 10 months. Patient had severe heavy calluses with recurrent ulcers for three years. Pulses diminished, postural color changes slight. Maximum toe temperature after nerve block 24.5 C. Left lumbar sympathectomy 1947. Appearance and comfort of foot near normal since operation. However, still has recurrent ulcers and calluses. Foot warm. Maximum toe temperature 30.8 C.

**Extremity 42.** Same patient as extremity 41. Same findings as in left leg. Maximum toe temperature after nerve block 26 C. Right lumbar sympathectomy 1947. No symptoms since operation, with no reappearance of calluses or ulcers. This foot has less patchy numbness than left.

**Extremity 43.** M. S., a 71 year old female. Severe pain and numbness in foot appeared three months before operation, with ulcer on heel appearing about two months later. No pedal pulses. Moderate postural color changes. Maximum toe temperature after nerve block 23 C. Right lumbar sympathectomy 1947. Ulcer healing and all pain relieved at end of follow-up period of three months.

**Extremity 44.** Same patient as extremity 43. No symptoms but atrophy and coldness apparent. Weak dorsalis pedis pulse. Maximum toe temperature 29.3 C. No changes during postoperative observation of three months.
SUMMARY

Lumbar sympathectomy has been employed in the treatment of arteriosclerotic vascular insufficiency of 44 lower extremities, and its effects have been assessed during the 14 years of this study. There has been significant improvement in the symptoms and the signs of the disease, far exceeding the original expectations. The changes in the criteria for selection of suitable cases for sympathectomy that have come about during the last decade are discussed. The rationale for the procedure is based on sound physiologic principles.

CONCLUSIONS

Lumbar sympathectomy is recommended as a therapy of proved worth for many cases of arteriosclerosis of the legs. It is suggested by the data contained in this paper that in time it may become the method of choice in most cases.

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Lumbar Sympathectomy for Peripheral Arteriosclerosis

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Circulation. 1951;4:402-415
doi: 10.1161/01.CIR.4.3.402

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