Vasomotor Responses in the Extremities of Subjects with Various Neurologic Lesions

I. Reflex Responses to Warming

By Walter Redisch, M.D., Francisco T. Tangco, M.D., Lothar Wertheimer, M.D., Arthur J. Lewis, M.D., and J. Murray Steele, M.D.

With the technical assistance of Dorothy Andrews, B.A.

Vascular responses to warming were studied in hemiplegic patients and after sympathectomy, using venous occlusion plethysmography of foot and leg. Comparisons were made with corresponding age groups. The pattern of response was essentially unchanged in hemiplegic patients, but was altered substantially where sympathetic pathways had been interrupted.

During the course of studies concerned with responses of various vascular beds to physiologic and pharmacologic stimuli, it became apparent that the responses evoked in human limbs that had been deprived of their sympathetic innervation were of considerable interest. For purposes of comparison, it seemed advisable to study also the behavior of the vascular response of paralyzed extremities in patients following cerebral vascular accidents and transection of the spinal cord. The present paper describes some of the abnormal vascular responses encountered in a study of these neurologic disorders.

Sympathectomy

A number of workers have studied the altered vasomotor responses after sympathectomy. Usually consistently elevated basal flow was described after sympathectomy.1-5 However, reports have varied as to the changes in response to vasodilator and vasoconstrictor stimuli. Goetz6 found that flow to the toe did not respond to either constrictor or dilator stimuli after sympathectomy and that in some cases blood flow was decreased in response to vasodilator stimuli and increased in response to vasoconstrictor stimuli. Mendlowitz and Touroff7 found the responses to vasodilator stimuli to remain directionally the same after sympathectomy. Prinmetal and Wilson,8 in 1936, tested 2 patients with Raynaud’s disease before and after sympathectomy. These patients showed an increase in blood flow to the forearm in response to the Gibson-Landis procedure before sympathectomy, but after operation a decrease in blood flow was observed. In earlier work, however, Grant and Holling9 had found that after sympathectomy a human forearm exhibited an increase in blood flow in response to body warming. Wilkins and Eichna,10 in 1941, studied vasomotor reactions to ice, pin prick or pinch, deep breath, immersing a hand or foot in very cold or very hot water, exercise, loud noise, and mental arithmetic. They found marked changes in flow in the nonsympathectomized extremity and occasionally also some changes in the sympathectomized one; the latter, however, correlated with changes in blood pressure. Duff and Swan,11 in 1951, and Barcroft and Swan12 measured blood flow to the calf and forearm in sympathectomized human extremities during infusion with epinephrine; they reported an initial rise to 2 to 3 times the resting level returned to the

From the Research Service (Third New York University Medical Division), Goldwater Memorial Hospital, and the Department of Medicine, New York University College of Medicine, New York, N. Y.

This work was aided by grant No. B-346 from the National Heart Institute, National Institutes of Health, the New York Heart Association and the Josiah Macy Foundation, and presented in part at the Annual Meeting of the Eastern Section, American Federation for Clinical Research, December 1954, Washington, D. C.

Drs. Tangco and Lewis are Fellows of the New York Heart Association; Dr. Wertheimer is a past Fellow of the Public Health Research Institute of the City of New York.

baseline during the infusion. In contrast, in nonsympathectomized limbs there was some decrease after this initial rise, immediately followed by another rise that was sustained throughout the infusion. These authors could not correlate the changes in blood flow with changes in blood pressure. Still more recently, Ahmad\(^3\) reported a case of hyperhidrosis with homolateral sympathectomy in whom local warming of the sympathectomized hand to 41°C caused vasoconstriction, while the normally innervated hand responded with vasodilation.

**Hemiplegia**

As long ago as 1888, Gowers\(^4\) encountered some interesting circulatory phenomena while studying cases of hemiplegia. The paralyzed extremities showed no change, an increase in skin temperature and coloring, mostly in the early stages, or a decrease in skin temperature associated with pallor, followed later by cyanosis. He found the same type of disturbance in traumatic lesions of the central gyrus that was "frequently accompanied by vasomotor disturbances in the contralateral extremity." He believed that destruction of subcortical vasomotor centers caused increase in blood flow, while "irritation" of these centers had the opposite effect. Uprus and co-workers\(^5\) (1935) found no consistent difference in temperature between the 2 sides of patients with hemiplegia; there was likewise no difference in reflex responses to vasomotor stimuli, except in the rate of cooling. In the same year, Sturup and associates\(^6\) reported that response to vasoconstrictor stimuli in patients with lesions of the cerebral hemispheres did not differ from the normal. On the other hand, Ellis and Weiss\(^7\) demonstrated in 1936 that the circulation of the hemiplegic limb was increased and might remain increased for as long as 13 years after a cerebrovascular accident.

**Spinal Cord Transection**

Guttmann and Whitteridge\(^8\) observed the reflex responses to distention of the bladder in patients with transection of the cord. While patients with low transection responded with vasoconstriction in the toes and "compensatory" vasodilation in the fingers, patients with high lesions responded with vasoconstriction in both the upper and lower extremities. Pollock and co-workers\(^9\) observed what they called "defects in regulation of heat production, sweat and vasoconstriction" in patients with spinal cord lesions. They believed these defects to be due to interruption of "impulses from suprasegmental levels." In 1953 Armin, Grant, and co-workers\(^10\) demonstrated increased reactivity to vasoconstrictor stimuli in the denervated rabbit's ear and referred to a similar phenomenon in the human finger after sympathectomy. They believed, on rather incomplete evidence, that this is due to interruption of fibers that normally act as dilators through release of acetylcholine.

In view of the conflicting evidence, it was considered worthwhile to restudy the respective roles of cerebrospinal and sympathetic innervation in regulating blood flow to the human extremities. The present report deals with the reflex response to warming the body.

**Method and Material**

All experiments were done at a constant temperature of 20°C (±0.5°) and 55 per cent humidity with an air current of less than 4½ feet per minute. Such environment provides a mild vasoconstrictor stimulus for testing the vasodilator response to the Gibbon-Landis procedure.\(^11\) The subject was in the basal state, clad in cotton pajamas with hands and feet exposed. Blood flow was measured plethysmographically.\(^12\) Surface temperature was recorded quasi-continuously on a 6-channel Leeds and Northrup Speedomax. The subject was considered "adapted" to the environment when the toe temperatures had remained constant for at least half an hour.

Twelve hemiplegic patients, 4 patients with "high transection" of the cord, and 8 patients with 11 sympathectomized limbs have been studied. Comparisons were made with studies in 10 young normal persons, 9 elderly subjects (50 to 75 years) without demonstrable vascular disease, and 8 patients with obliterator arteriosclerosis. Hemiplegic limbs were also directly compared with their nonparalyzed fellows in 5 patients, and the sympathectomized with the nonsympathectomized side in 4 patients. All experiments were repeated and the observations were relatively reproducible.

**Results**

**Hemiplegia**

Basal flows in limbs of hemiplegic patients were within the range of those in elderly sub-
TABLE 1.—Influence of Gibbon-Landis Procedure on the Blood Flow of the Lower Extremities, (ml./100 cc. tissue/min.)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Basal flow</th>
<th>Max. response</th>
<th>Patient</th>
<th>Basal flow</th>
<th>Max. response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects without demonstrable vascular disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–40 years</td>
<td>50–50 years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I.W.</td>
<td>13.6</td>
<td>17.7</td>
<td>F.K.</td>
<td>3.6</td>
<td>5.9</td>
</tr>
<tr>
<td>S.D.</td>
<td>9.1</td>
<td>18.0</td>
<td>W.F.</td>
<td>3.4</td>
<td>6.5</td>
</tr>
<tr>
<td>S.L.</td>
<td>3.2</td>
<td>7.0</td>
<td>H.T.</td>
<td>10.5</td>
<td>12.1</td>
</tr>
<tr>
<td>M.H.</td>
<td>24.0</td>
<td>25.4</td>
<td>J.J.</td>
<td>8.4</td>
<td>14.2</td>
</tr>
<tr>
<td>D.S.</td>
<td>20.9</td>
<td>30.5</td>
<td>J.B.</td>
<td>8.6</td>
<td>10.1</td>
</tr>
<tr>
<td>E.C.</td>
<td>10.5</td>
<td>14.8</td>
<td>J.H.</td>
<td>15.9</td>
<td>22.7</td>
</tr>
<tr>
<td>A.O.</td>
<td>16.0</td>
<td>21.6</td>
<td>M.S.</td>
<td>10.5</td>
<td>14.8</td>
</tr>
<tr>
<td>S.P.</td>
<td>16.7</td>
<td>18.9</td>
<td>J.F.</td>
<td>5.7</td>
<td>9.9</td>
</tr>
<tr>
<td>A.S.</td>
<td>14.5</td>
<td>24.4</td>
<td>E.V.</td>
<td>8.3</td>
<td>12.2</td>
</tr>
<tr>
<td>E.G.</td>
<td>9.4</td>
<td>20.8</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>13.8</td>
<td>19.9</td>
<td>Average</td>
<td>8.3</td>
<td>12.0</td>
</tr>
<tr>
<td>Elderly patients with obliterative vascular disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R.K.</td>
<td>2.8</td>
<td>6.9</td>
<td>M.N.</td>
<td>6.7</td>
<td>8.5</td>
</tr>
<tr>
<td>J.C.</td>
<td>2.0</td>
<td>3.9</td>
<td>M.S.</td>
<td>4.3</td>
<td>7.2</td>
</tr>
<tr>
<td>J.K.</td>
<td>2.3</td>
<td>3.2</td>
<td>L.J.</td>
<td>8.4</td>
<td>12.3</td>
</tr>
<tr>
<td>M.E.</td>
<td>3.2</td>
<td>9.7</td>
<td>M.G.</td>
<td>6.7</td>
<td>10.0</td>
</tr>
<tr>
<td>M.G.</td>
<td>4.4</td>
<td>6.2</td>
<td>E.P.</td>
<td>16.6</td>
<td>35.5</td>
</tr>
<tr>
<td>M.N.</td>
<td>2.3</td>
<td>4.9</td>
<td>M.E.</td>
<td>3.9</td>
<td>4.3</td>
</tr>
<tr>
<td>C.B.</td>
<td>2.8</td>
<td>5.1</td>
<td>E.H.</td>
<td>10.7</td>
<td>15.5</td>
</tr>
<tr>
<td>A.D.</td>
<td>6.0</td>
<td>9.4</td>
<td>A.R.</td>
<td>6.4</td>
<td>10.0</td>
</tr>
<tr>
<td>Average</td>
<td>3.2</td>
<td>6.2</td>
<td>C.J.</td>
<td>5.5</td>
<td>7.1</td>
</tr>
<tr>
<td>Hemiplegic patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R.K.</td>
<td>2.8</td>
<td>6.9</td>
<td>M.N.</td>
<td>6.7</td>
<td>8.5</td>
</tr>
<tr>
<td>J.C.</td>
<td>2.0</td>
<td>3.9</td>
<td>M.S.</td>
<td>4.3</td>
<td>7.2</td>
</tr>
<tr>
<td>J.K.</td>
<td>2.3</td>
<td>3.2</td>
<td>L.J.</td>
<td>8.4</td>
<td>12.3</td>
</tr>
<tr>
<td>M.E.</td>
<td>3.2</td>
<td>9.7</td>
<td>M.G.</td>
<td>6.7</td>
<td>10.0</td>
</tr>
<tr>
<td>M.G.</td>
<td>4.4</td>
<td>6.2</td>
<td>E.P.</td>
<td>16.6</td>
<td>35.5</td>
</tr>
<tr>
<td>M.N.</td>
<td>2.3</td>
<td>4.9</td>
<td>M.E.</td>
<td>3.9</td>
<td>4.3</td>
</tr>
<tr>
<td>C.B.</td>
<td>2.8</td>
<td>5.1</td>
<td>E.H.</td>
<td>10.7</td>
<td>15.5</td>
</tr>
<tr>
<td>A.D.</td>
<td>6.0</td>
<td>9.4</td>
<td>A.R.</td>
<td>6.4</td>
<td>10.0</td>
</tr>
<tr>
<td>Average</td>
<td>3.2</td>
<td>6.2</td>
<td>C.J.</td>
<td>5.5</td>
<td>7.1</td>
</tr>
<tr>
<td>Average</td>
<td>8.1</td>
<td>13.0</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Subjects of comparable age without demonstrable vascular disease, while young adults had a much higher average flow (table 1). In response to the Gibbon-Landis procedure, hemiplegic limbs showed an increase in blood flow comparable to that observed in a control group of similar age (table 1). In 5 hemiplegic patients the paralyzed limbs were compared with the nonparalyzed fellows. Here the basal blood flow appeared to be generally lower in the nonparalyzed than in the paralyzed limb (table 2). One example is shown in figure 1.

The subjects with obliterative arteriosclerosis reacted directionally in a similar fashion to the other 3 groups, but the initial flows as well as the maximal flows were lower. This fact was not expressed in the measurement of surface temperature, which eventually reached essentially the same values as in the other control groups, although increase was delayed in starting and proceeded at a much slower rate (fig. 2). There is then no qualitative difference in the reflex vasodilator response in these 4 groups (table 1) with intact sympathetic innervation.

Spinal Cord Transection

In the patients with "high transection" of the cord, the average basal blood flow in the paraplegic limbs was higher than in the arteriosclerotic patients but lower than in the other 2 control groups. Reflex vasodilator responses varied (table 3). In response to the Gibbon-Landis procedure the blood flow in the first of these 4 subjects decreased markedly, in 1 it increased markedly, while in the other 2 it
TABLE 2.—Influence of Gibbon-Landis Procedure on the Blood Flow of the Lower Extremities in Hemi-
plegic Patients, (ml./100 cc. tissue/min.)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Duration of hemiplegia (at time of study)</th>
<th>Paralyzed side</th>
<th>Nonparalyzed side</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control reading</td>
<td>Maximum response</td>
<td>Control reading</td>
</tr>
<tr>
<td>M.N.</td>
<td>8 months</td>
<td>6.7</td>
<td>8.5</td>
</tr>
<tr>
<td>M.S.</td>
<td>2½ months</td>
<td>4.3</td>
<td>7.2</td>
</tr>
<tr>
<td>L.J.</td>
<td>3 weeks</td>
<td>8.4</td>
<td>12.3</td>
</tr>
<tr>
<td>M.G.</td>
<td>3 months</td>
<td>6.7</td>
<td>10.0</td>
</tr>
<tr>
<td>E.F.*</td>
<td>11 years</td>
<td>16.6</td>
<td>35.5</td>
</tr>
<tr>
<td>Average</td>
<td></td>
<td>8.5</td>
<td>14.7</td>
</tr>
</tbody>
</table>

* This patient is hypertensive.

TABLE 3.—Influence of Gibbon-Landis Procedure on the Blood Flow of the Lower Extremities in Sympa-
thetomized and Paraplegic Patients, (ml./100 cc. tissue/min.)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Basal flow</th>
<th>Maximum response</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sympathetomized patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.B.</td>
<td>12.6</td>
<td>8.6</td>
</tr>
<tr>
<td>F.B.</td>
<td>5.25</td>
<td>6.0</td>
</tr>
<tr>
<td>L.S. (R)</td>
<td>4.45</td>
<td>1.6</td>
</tr>
<tr>
<td>L.S. (L)</td>
<td>4.4</td>
<td>3.5</td>
</tr>
<tr>
<td>A.F. (R)</td>
<td>9.4</td>
<td>6.5</td>
</tr>
<tr>
<td>A.F. (L)</td>
<td>12.9</td>
<td>9.2</td>
</tr>
<tr>
<td>S.Q.</td>
<td>10.3</td>
<td>10.0</td>
</tr>
<tr>
<td>H.M. (R)</td>
<td>15.7</td>
<td>14.8</td>
</tr>
<tr>
<td>H.M. (L)</td>
<td>17.2</td>
<td>6.3</td>
</tr>
<tr>
<td>Average</td>
<td>10.2</td>
<td>7.4</td>
</tr>
<tr>
<td>F.K.*</td>
<td>14.7</td>
<td>10.7</td>
</tr>
<tr>
<td>A.M.†</td>
<td>18.1</td>
<td>10.7</td>
</tr>
<tr>
<td>Average</td>
<td>16.4</td>
<td>10.7</td>
</tr>
<tr>
<td>Paraplegic patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A.R.</td>
<td>7.1</td>
<td>1.4</td>
</tr>
<tr>
<td>D.J.</td>
<td>7.0</td>
<td>6.8</td>
</tr>
<tr>
<td>J.R.</td>
<td>5.6</td>
<td>17.1</td>
</tr>
<tr>
<td>S.P.</td>
<td>6.1</td>
<td>7.5</td>
</tr>
<tr>
<td>Average</td>
<td>6.5</td>
<td></td>
</tr>
</tbody>
</table>

* No peripheral arterial disease. Sympathectomized for "cold feet."
† No peripheral arterial disease. Sympathectomized for hypertension.

showed little change. The diversity of response in these patients may well be due to differences in location and extent of the transection. The first patient was the only one in whom the transection was radiologically proved to be above Ts.

Sympathectomy

The reflex responses to warming in sympathectomized limbs were of considerable interest. Eight patients were studied and in 3 of these measurements of blood flow were carried out in both sympathectomized limbs. Six of these 8 patients were sympathectomized for occlusive arterial disease, 1 for hypertension and 1 simply for "cold feet.” Thus 11 sympathectomized limbs were studied. Seven of these 11 limbs (table 3) responded to the vasodilator stimulus with a significant decrease in blood flow. In the sympathectomized limb initial basal flow and surface temperature are, of course, higher than in the nonsympathectomized fellow. Average basal flows were higher in limbs sympathectomized for vascular disease than in a comparable nonsympathectomized group (compare tables 1 and 3). Two patients who had been sympathectomized in the absence of occlusive vascular disease likewise had higher basal flows than their young control group (compare tables 1 and 3). In response to the Gibbon-Landis procedure blood flow increased as usual on the nonsympathectomized side, but decreased on the sympathectomized side (fig. 3). Surface temperature rose in the normally innervated limb, reflecting the increase in blood flow, but did not fall with the decrease in blood flow on the sympathectomized side.

DISCUSSION

Changes in blood flow through a part of the body or an organ of the body are generally con-
sidered to be expressions of vasomotor impulses. The results presented here then seem to indicate that cerebral lesions causing hemiplegia do not essentially alter the reflex vasodilator response (increase in blood flow) to warming, in either the hemiplegic or the unaffected extremity.

There was no correlation between basal flow and the degree of discoloration. A cursory study of possible changes in the minute circulation by capillary microscopy at the fingernail bed did not reveal any conclusive difference between the 2 sides except in those cases that showed hemi-edema: in the presence of hemi-edema there was blurring of the outline of the capillary loops.

The reflex vasomotor responses to a dilator stimulus in the 4 paraplegic subjects with alleged high transection of the cord varied from patient to patient. It is noteworthy that the only patient with radiologically proved traumatic transection at a level apparently just above T₆ was the only one in whom a decrease in blood flow was observed in response to the Gibbon-Landis procedure. In the other 3 patients the evidence either for the exact level of lesion or for the completeness of transection of the cord is insufficient. It seems probable that the degree of alteration of vasomotor responses depends upon the level as well as the completeness of transection. The variability of response in these 3 patients suggests that as these studies proceed, the type of response might prove to be an indication of the location and extent of the lesion. It has generally been taught that sympathetic pathways to the lower extremities leave the spinal cord not higher than T₆-₇.²³ It is also usually assumed that abolition of sweating is evidence for complete sympathetic denervation.²⁴ All 4 of the patients reported here showed complete absence of perspiration of the lower extremities tested by the Guttmann procedure,²⁴ but their vasomotor responses varied. Further investigation may bear out previously expressed doubt as to the acceptability of loss of perspiration as proof of complete sympathetic denervation.²⁵, ²⁶

The results, however, of studies on surgically sympathectomized patients are quite clearcut. In none of the limbs studied after sympathectomy could an increase in blood flow be produced reflexly by warming; in the majority of instances the opposite response, a decrease in blood flow, was observed. The regularity with which these carefully sympathectomized limbs fail to respond to a vasodilator stimulus suggests that this procedure might be useful as a test for completeness of sympathectomy.

The physiologic events that lead to alterations in vasomotor responses in limbs that have been deprived of most of their sympathetic innervation are still obscure. Systemic arterial pressure bears no demonstrable relationship to the changes in blood flow produced by the Gibbon-Landis procedure. In a person who has undergone unilateral sympathectomy, there is, in response to the Gibbon-Landis procedure, an increase in blood flow on the non-sympathectomized side and simultaneously a decrease on the sympathectomized side; the increase on the nonsympathectomized side is not in excess of that observed in controls. In bilaterally sympathectomized subjects, a decrease in blood flow usually occurs on both sides in response to warming. It appears unlikely, therefore, that a "borrowing-lending mechanism"²⁷ could explain the vasomotor responses.

Preliminary observations suggest the possibility of influencing toward normal reflex vasomotor responses in sympathectomized limbs by adrenergic blockade. This effect could be interpreted as indicating an active role of catechol amines.

Epinephrine and norepinephrine are released from the adrenal gland in response to hypothalamic stimulation.²⁹ Such stimulation may be effected by warming of the blood, as in the Gibbon-Landis procedure. "Sensitization" of sympathectomized organs to the action of circulating catechol amines has been demonstrated in the basic work of Cannon and his associates²⁹-³² and later workers³³ in animals. This work was applied to man as early as 1934.³⁴ Some of the evidence presented later³⁵, ³⁶ has not been entirely convincing.

Evidence that norepinephrine is liberated locally in the arterial wall has been reported.³⁷ Burn and co-workers³⁸ demonstrated the destruction of norepinephrine by amineoxidase
and showed that the rate of destruction can be slowed considerably by inhibiting the enzyme. It might be speculated then that the sympathetic supply influences the liberation of the catechol amines or its enzymatic destruction. If this is the case, it is still necessary to explain why such action appears to operate only when vasomotor stimulus is applied.

Increase in blood flow is generally followed by a rise in skin temperature but decrease in blood flow in response to the Gibbon-Landis procedure after sympathectomy is not necessarily accompanied by a fall in surface temperature. This poor correlation between skin temperature and blood flow confirms the previous report of Hoobler and co-workers and helps define the limits of usefulness of measurements of skin temperature as an index of blood flow to the extremity.

**SUMMARY**

The vasomotor responses to the Gibbon-Landis procedure (reflex response to warming) were studied in hemiplegic patients, subjects with "high transection" of the cord, and in sympathectomized patients. The response in hemiplegic patients was vasodilator in nature just as in the 3 control groups (young normal subjects, elderly subjects without demonstrable vascular disease, and patients with arteriosclerosis). One patient with documented transection of the cord above T5 behaved like subjects after surgical sympathectomy. The differences in response in 3 other paraplegic patients may be due to differences in location and extent of their cord lesions. Basal blood flow was higher in sympathectomized limbs than in comparable controls. Of 11 sympathectomized limbs tested for vasodilatation in response to the Gibbon-Landis procedure, 4 showed no response, while 7 responded with decrease in blood flow (vasoconstriction).

**SUMMARIO IN INTERLINGUA**

Le responsas vasomotori evocate per la manipulation de Gibbon-Landis (responsa reflexa a calefazione) eseva studiate in patientes hemiplegic, in subjectos con "alte transection" del medulla spinal, e in patientes sympathectomisate. In patientes hemiplegic, le responsa eseva de natura vasodilatatori, exactemente como in le individuos del tres gruppos de controlo (juvenile subjectos normal, plus vetule subjectos sin demonstrabile morbo vascular, e patientes con arteriosclerosis). Un paciente con documentate transection del medulla spinal supra T5 se comportava como le subjectos con sympathectomia chirurgic. Le differentias del responsas in tres altere patientes paraplegic es possibilemente causate per differentias del sito e del grado de lor lesions spinal. Le basal fluxo de sanguine eseva plus alte in extremitates sympathectomisate que in comparable casos de controlo. In un serie de 11 extremitates sympathectomisate, tests del vasodilatation in responsa al manipulation de Gibbon-Landis eseva negative in quatro casos, durante que septe casos respondeva per un reduction del fluxo de sanguine (vasoconstriction).

**REFERENCES**

10. Wilkins, R. W., and Eincha, L. W.: Blood flow to the forearm and calf. I. Vasomotor reactions


20 Armin, J., Grant, R. T., Thompson, R. H. S., and Tickner, A.: An explanation for the heightened vascular reactivity of the denervated rabbit’s ear. J. Physiol. 121: 603, 1953.


Vasomotor Responses in the Extremities of Subjects with Various Neurologic Lesions: I. Reflex Responses to Warming

WALTER REDISCH, FRANCISCO T. TANGCO, LOTHAR WERTHEIMER, ARTHUR J. LEWIS, J. MURRAY STEELE and Dorothy Andrews

Circulation. 1957;15:518-524
doi: 10.1161/01.CIR.15.4.518

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1957 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/15/4/518

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/