Comparison of Adrenergic Mechanisms in an Elastic and a Muscular Artery of the Rabbit

By J. A. Bevan, R. D. Bevon, R. E. Purdy, C. P. Robinson, C. Su, and J. G. Waterson

ABSTRACT

Elastic and muscular arteries are known to differ in function and in the magnitude of their response to vasoconstrictor influences. With the isolated thoracic aorta and ear artery of the rabbit as representative arteries, the morphological, physiological, and pharmacological correlates of these differences have been sought among their presynaptic adrenergic mechanisms. The adrenergic nerve plexus in the ear artery is wider and the nodes are denser than they are in the aorta. There is some evidence consistent with the hypothesis that the thicker the plexus, the greater the reuptake of released transmitter, the smaller the transmitter overflow, and the lower the efficiency of uptake of exogenous norepinephrine. Measurements of fractional release of tritiated norepinephrine suggest that qualitative differences in the adrenergic transmitter storage and release mechanisms may exist between the two vessels. Thus the considerable functional difference between the two vessels is, at any rate, a consequence of adrenergic mechanisms which differ both quantitatively and qualitatively from each other.

KEY WORDS aorta adrenergic transmission node density ear artery norepinephrine release elastic artery norepinephrine uptake muscular artery adrenergic plexus

Differences in function and in responsiveness to vasoconstrictor influences between elastic and muscular arteries have been well defined. Elastic arteries are essentially conduits contributing little to vascular impedance and, because of the elastic properties of their wall, convert pulsatile to continuous flow, the so-called "Windkessel effect." In contrast, muscular vessels contribute significantly to vascular resistance and its regulation and, consequently, to the distribution of blood flow (1). These two vessel types not only show dimensional and structural differences appropriate to their function, but also in the rabbit some representatives, at any rate, exhibit variation in the nature of their effector response to sympathetic and other vasoconstrictor influences (2).

The studies described in this paper were undertaken to determine whether presynaptic neuronal mechanisms are the same in vessels of contrasting size and function. The two vessels selected for this comparative study of presynaptic adrenergic mechanisms were the aorta and the central ear artery of the rabbit—an elastic and a muscular artery, respectively. Our comparisons suggest quantitative differences in innervation density and the possibility of qualitative differences in fractional transmitter release. Differing innervation densities, because of a "node-crowding" effect, influence the efficiency of the neuronal uptake of exogenous and neurogenic l-norepinephrine.

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Methods

The thoracic aorta and both central ear arteries of rabbits were removed rapidly after animals of either sex, weighing 2.0–2.5 kg, had been stunned and bled. The aorta from just beyond the arch to the level of the diaphragm and the ear arteries from 1.5 cm distal to the base of the ear for 4–5 cm were used. Tissues were rapidly cleaned of extraneous loose fatty material and then subjected to the following techniques.

Noradrenaline Content

After they were blotted with filter paper, the isolated arteries were weighed and homogenized in 5% trichloroacetic acid with a Willens Polytron homogenizer. After centrifugation, the supernatant fluid was neutralized to pH 8.4 in the presence of alumina, and the latter was eluted with 0.1N acetic acid. Noradrenaline in the eluate was assayed spectrofluorometrically by the trihydroxyindole method (3).

Tritiated Noradrenaline Uptake

Total Uptake.—The method employed was essentially the same as that described before (4). Arterial specimens were equilibrated in Krebsbicarbonate solution at 38°C and bubbled with 95% O₂-5% CO₂ for 90 minutes. During the last 30 minutes of the equilibration period, some specimens were treated with either phenoxybenzamine (10⁻⁴M) or cocaine (10⁻⁴M). All tissues were then soaked in d[⁷⁻H]-noradrenaline hydrochloride (10⁻⁴M) for 60 minutes in the absence or presence of the phenoxybenzamine or cocaine. After a rapid rinse, tissues were blotted, weighed, and digested. Tissue radioactivity was measured by scintillation spectrometry, and uptake was expressed as milliliters of bath fluid cleared per gram of wet tissue.

Distribution.—The distribution profiles of tritiated noradrenaline in the wall of the aorta and the ear artery were determined by an isotope frozen-section technique (5, 6). Aorta and ear artery sections were cut at 24 μ and 8 μ, respectively. Alternate sections were stained with hematoxylin and eosin for examination by light microscopy to establish the site of the adventitio-medial junction. The radioactivity of each section was determined and expressed as milliliters of bath fluid cleared per gram of tissue, and mean distribution profiles were constructed by superimposition of profiles from a number of tissues.

Tritiated Noradrenaline Release

Helically cut arterial strips were mounted, superfused, and transmurally stimulated as previously described (7) with the following changes: incubations were for 60 minutes under 1 g of tension with L[⁷⁻H]-noradrenaline (specific activity 2.18 c/mmole) 5 μc/ml (2.29 μl). Stimulation periods were 6 minutes in length with 14 minutes between them. Pulses were delivered at supramaximal voltage, frequency 10/sec, and duration 0.3 msec.

Fluorescence Histochemistry

Using the method of Falck et al. (8) as modified by Waterson and Small (9) for vascular tissue, catecholamine fluorescence was demonstrated in the adrenergic nerve plexus of the arteries. Except for mounting on short lengths of loosely fitting polyethylene tubing no effort was made to preserve precise in vivo dimensions.

From selected transverse sections showing a uniformly circular shape of the adventitial and intimal surfaces estimates were made of the width of the node-containing fluorescent band at the adventitio-medial junction. From each section four measurements were made. These measurements involved a somewhat subjective averaging of the band width for a small arc of the circumference. From longitudinal sections of 5 or 6 μ in thickness which passed tangentially through the node-containing fluorescent band (which was more than 12 μ thick) areas were selected in which nodes were readily identified. Node counts were made using a calibrated grid eyepiece and, by continually focusing through the section thickness, nodal structures in each area of the grid were identified and counted. Each count was made independently by two observers. These two sets of measurements allowed estimates of fluorescent band thickness and node density per unit volume of tissue, respectively.

Results

Noradrenaline Content

The catecholamine content was expressed in terms of noradrenaline since the fluorescence spectrums of the arterial extracts were characteristic of this substance, and no attempt was made to assay other amines. An earlier work (10) suggests that the contribution of epinephrine to the catecholamine content in rabbit ear artery is less than 10%. In Table 1, the noradrenaline content of the thoracic aorta and the pooled right and left ear arteries from 12 rabbits is shown. The mean content of noradrenaline was 0.78 and 3.04 μg/g wet weight, respectively, and the ratio of the means was 1 to 4. A similar difference was found between the pulmonary artery (another elastic artery) and the ear artery, and the ratio of the means was approximately 1 to 3. The
COMPARISON OF ADRENERGIC MECHANISMS

TABLE 1
Norepinephrine Content of Rabbit Arteries

<table>
<thead>
<tr>
<th>Rabbit no.</th>
<th>Norepinephrine concentration (nA/g wet wt)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TA</td>
</tr>
<tr>
<td>1</td>
<td>0.47</td>
</tr>
<tr>
<td>2</td>
<td>1.30</td>
</tr>
<tr>
<td>3</td>
<td>0.86</td>
</tr>
<tr>
<td>4</td>
<td>0.55</td>
</tr>
<tr>
<td>5</td>
<td>0.44</td>
</tr>
<tr>
<td>6</td>
<td>0.72</td>
</tr>
<tr>
<td>7</td>
<td>0.59</td>
</tr>
<tr>
<td>8</td>
<td>0.68</td>
</tr>
<tr>
<td>9</td>
<td>0.63</td>
</tr>
<tr>
<td>10</td>
<td>0.71</td>
</tr>
<tr>
<td>11</td>
<td>1.28</td>
</tr>
<tr>
<td>12</td>
<td>0.92</td>
</tr>
</tbody>
</table>

Mean 0.78 3.04 1.04 1/3.99
SD 0.28 0.44 0.37 1.20
SE 0.08 0.17 0.11 0.46
CV 0.36 0.15 0.36 0.30

TA = thoracic aorta; EA = ear artery; PA = pulmonary artery; CV = coefficient of variation.
*Ear arteries of rabbits 1-4, 5 and 6, and 7 and 8 were pooled.

relative variation between individual values was greater for the thoracic aorta than for the ear artery (coefficients of variation were 0.36 and 0.15, respectively). Since the total norepinephrine content of a specimen of thoracic aorta was greater than that of pooled ear arteries, the variation was not dependent on a lower norepinephrine content of the specimen tested. Although the coefficients of variation for the thoracic aorta and the pulmonary artery were equally large, there was a correlation between values from the same animal at the 2% level of significance (r = 0.615).

TRITIATED NOREPINEPHRINE UPTAKE

Total Uptake.—In Table 2, tritiated norepinephrine uptake, expressed as milliliters of bath fluid cleared per gram wet weight of blood vessel, is shown for control and for cocaine- and phenoxybenzamine-treated vessels. After 1 hour the ear arteries had cleared approximately 2.4 times the amount the aorta had.

The ratio of the cocaine-sensitive component of uptake (which represents neuronal uptake [11]) for the thoracic aorta to that for the ear artery was 1 to 2.4. When expressed as mean moles cleared per microgram of norepinephrine after 1 hour of soaking in tritiated norepinephrine the uptakes were equivalent to 7.8 X 10^-11 and 5.1 X 10^-11 for the thoracic aorta and the ear artery, respectively. The ratio of the phenoxybenzamine-sensitive component of tritiated norepinephrine uptake for the thoracic aorta to that for the ear artery was 1 to 2.9. The phenoxybenzamine-insensitive uptake, which probably reflects nonspecific binding and all extraneuronal binding, was similar in both tissues.

TABLE 2
Tritiated Norepinephrine Uptake in Rabbit Arteries

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Uptake* (ml/g)</th>
<th>TA/EA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TA</td>
<td>EA</td>
</tr>
<tr>
<td>Control</td>
<td>9.34</td>
<td>22.31</td>
</tr>
<tr>
<td>Phenoxybenzamine (10^-5M)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenoxybenzamine-insensitive</td>
<td>2.70</td>
<td>2.95</td>
</tr>
<tr>
<td>Phenoxybenzamine-sensitive</td>
<td>6.64</td>
<td>19.36</td>
</tr>
<tr>
<td>Control</td>
<td>9.13</td>
<td>19.07</td>
</tr>
<tr>
<td>Cocaine (10^-5M)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cocaine-insensitive</td>
<td>2.81</td>
<td>4.63</td>
</tr>
<tr>
<td>Cocaine-sensitive</td>
<td>6.32</td>
<td>15.34</td>
</tr>
</tbody>
</table>

Values are means ± SE. Number of arteries tested is given in parentheses.
*One ml is equivalent to 10^-14M tritiated norepinephrine.
†Obtained by subtracting phenoxybenzamine-insensitive uptake from the control.
‡Obtained by subtracting cocaine-insensitive uptake from the control.

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Mean distribution profiles of tritium uptake in the thickness of the walls of rabbit thoracic aortas and ear arteries after exposure to tritiated norepinephrine (0.5 μM) for 1 hour and subsequent washing for 10 minutes. Sections were superimposed on the basis of the site of their adventitiomedial junction (AMJ). Aortas were sectioned at 24 μm, ear arteries at 8 μm. Alternate ear artery sections were used for quality control and to establish the site of the adventitiomedial junction.

**Distribution.**—The distribution of tritium throughout the thickness of the thoracic aorta and the ear artery after soaking the tissues for 1 hour in tritiated norepinephrine (30 μM) and then washing them for 10 minutes in Krebs solution is shown in Figure 1. Although the general shapes of the curves for these two arteries were similar, peak radioactivity was higher in the ear artery compared with the thoracic aorta, reflecting the greater node density in the muscular vessel (see below). In the thoracic aorta, except adjacent to the adventitiomedial junction, medial uptake values were fairly constant throughout the media. In the ear artery mean values in the media were the same throughout. Medial uptakes in the ear artery were considerably higher than those in the aorta.

**Tritiated Norepinephrine Release**

Comparisons of the fractional release of tritium from both vessels were made after transmitter stores had been labeled with tritiated norepinephrine. The stimulation-induced overflow of tritium for the second minute of a 6-minute period of stimulation, when the overflow had reached a steady state, was divided by the tritium remaining in the tissue and the number of pulses delivered to the tissue during the period of collection. These measurements were made before and after a 10-minute treatment with phenoxybenzamine (10⁻⁶ g/ml). The data are summarized in Table 3.

The steady-state overflow after treatment with phenoxybenzamine, which presumably blocked neuronal and most extraneuronal uptake of norepinephrine, may be considered to be a measurement of total release of transmitter. Total fractional release in the thoracic aorta and the ear artery, respectively, was 2.4 × 10⁻⁵ and 5.4 × 10⁻⁵. These values are significantly different (P < 0.001). At a stimulation frequency of 10/sec, the ratio of fractional release from the thoracic aorta to that from the ear artery was 1 to 1.23. After phenoxybenzamine, the ratio was increased to 1 to 2.25. The change in the ratio was the result of a disproportionate increase in release after phenoxybenzamine in the ear artery compared to that in the thoracic aorta. These figures suggest that the percent of reuptake of transmitter for the thoracic aorta and the ear artery was 29% and 61%, respectively. From the values of fractional release (Table 3), norepinephrine content (Table 1), Avogadro's number, and node density (Table 4), it can be calculated that at this frequency the magnitude of norepinephrine release from the thoracic aorta and the ear artery expressed as molecules of norepinephrine per node per pulse was approximately 1600 and 1100 molecules, respectively, before and 2200 and

---

*Figure 1*

Mean distribution profiles of tritium uptake in the thickness of the walls of rabbit thoracic aortas and ear arteries after exposure to tritiated norepinephrine (0.5 μM) for 1 hour and subsequent washing for 10 minutes. Sections were superimposed on the basis of the site of their adventitiomedial junction (AMJ). Aortas were sectioned at 24 μm, ear arteries at 8 μm. Alternate ear artery sections were used for quality control and to establish the site of the adventitiomedial junction.
COMPARISON OF ADRENERGIC MECHANISMS

TABLE 3
Release of Norepinephrine from Rabbit Arteries during Transmural Stimulation

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Treatment</th>
<th>Fractional (X 10^-6)</th>
<th>Release (pg/g tissue/pulse)^*</th>
<th>Percent reuptaken%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ear artery</td>
<td>Control</td>
<td>2.1 ± 0.24</td>
<td>63.7</td>
<td>61</td>
</tr>
<tr>
<td></td>
<td>Phenoxybenzamine</td>
<td>5.4 ± 0.13§</td>
<td>163.9</td>
<td>29</td>
</tr>
<tr>
<td>Thoracic aorta</td>
<td>Control</td>
<td>1.7 ± 0.22</td>
<td>13.2</td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>Phenoxybenzamine</td>
<td>2.4 ± 0.54</td>
<td>18.6</td>
<td>29</td>
</tr>
</tbody>
</table>

Values are means ± s.e. Five experiments were carried out for each treatment.
*Calculated from fractional release and norepinephrine content (Table 1).
†Calculated from picograms released per gram tissue per pulse and node density (Table 4).
§Significantly different from ear artery control (P < 0.001) and from thoracic aorta phenoxybenzamine (P<0.001).

2900 molecules, respectively, after phenoxybenzamine.

FLUORESCENCE MICROSCOPY

In Table 4, a summary of the measurements made from specimens of the thoracic aorta and the ear artery of the rabbit prepared for catecholamine fluorescence microscopy are presented. These measurements were designed to quantify certain features of the node-containing fluorescent band of the two vessels. The width of the fluorescent band at the adventitiomedial junction was estimated from transverse sections. Node density counts were made from sections which were cut in the long axis of the vessel and passed tangentially through the fluorescent band. The product of fluorescent band width and node density per unit band thickness represents the total number of nodes per unit surface area of band or adventitiomedial junction.

Experimental errors included distortion resulting from freeze drying and histological processing, passive elastic contraction after removal from the animal, and computational inadequacies. Consequently absolute values were unreliable. If it is assumed that these errors were somewhat comparable in both vessels, relative estimates would be of value and relevant to this study.

Experimental values suggested that the relative number of nodes per unit area of adventitiomedial junction, or the ratio of node density for the thoracic aorta to that for the ear artery, was 1 to 4.2. Based on wall thickness measurements from the freeze-dried preparations, the number of nodes per gram

TABLE 4
Catecholamine Fluorescence Microscopy of Rabbit Arteries

<table>
<thead>
<tr>
<th></th>
<th>TA</th>
<th>EA</th>
<th>TA/EA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thickness fluorescent band (μ) *</td>
<td>4.8 ± 0.4 (16)</td>
<td>12.3 ± 0.6 (22)</td>
<td>1/2.6</td>
</tr>
<tr>
<td>Nodes/mm² surface area at AMJ†</td>
<td>8,260 ± 310 (39)</td>
<td>34,700 ± 1,590 (25)</td>
<td>1/4.2</td>
</tr>
<tr>
<td>Arterial wall thickness (μ)</td>
<td>273 ± 11.6 (12)</td>
<td>165 ± 3.1 (11)</td>
<td>1/0.60</td>
</tr>
<tr>
<td>Mean no. nodes/g artery‡</td>
<td>3.0 X 10⁵</td>
<td>21.0 X 10³</td>
<td>1/7</td>
</tr>
</tbody>
</table>

Values are means ± s.e. Number of arteries tested is given in parentheses. TA = thoracic aorta; EA = ear artery; AMJ = adventitiomedial junction.
*Obtained from transverse sections.
†Obtained from tangential longitudinal sections.
‡(Nodes/mm² surface area) X (arterial wall thickness)/(specific gravity of arteries). The specific gravity was assumed to be 1.02.

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of thoracic aorta and ear artery was $3 \times 10^7$ and $21 \times 10^7$, respectively. It is of interest that the number of nodes per gram of pulmonary artery obtained by an entirely different technique was previously found to be $9.3 \times 10^7$ (12).

**Discussion**

Some features of the adrenergic neuronal mechanisms in blood vessels from the same species but with divergent functions were compared and evidence of both qualitative and quantitative differences obtained. The norepinephrine content was selected as the standard or basis of comparison, since this was probably the most accurate determination made and there is evidence that norepinephrine content is related to the magnitude of the sympathetic neurogenic response (13).

Estimates of norepinephrine content showed considerable variation, which reflected individual variation since there was a positive correlation between the norepinephrine contents of the aorta and the pulmonary artery from different animals (correlation coefficient $\gamma = 0.61$). Such variation has been observed by others (13).

The ratio of mean norepinephrine content ($\mu g/g$ tissue) for the thoracic aorta to that for the ear artery was 1 to 3.99. We cannot explain, except possibly on the basis of difference in animal strains and methods, why our measurements of norepinephrine content of the ear arteries were much higher than those reported by de la Lande and Head (10). If sympathetic function differs only quantitatively between the thoracic aorta and the ear artery, the ratio of other measurements, when expressed on the same basis, should be similar to that for norepinephrine content, namely 1 to 4.

The values of total, phenoxybenzamine-sensitive, and cocaine-sensitive uptake of tritiated norepinephrine were greater for the ear artery than for the thoracic aorta, although the ratios were less than 1 to 2.4–2.9, which was less than the ratios for norepinephrine content, namely, 1 to 4. Thus the uptake of tritiated norepinephrine per unit content of norepinephrine was less for the ear artery than for the thoracic aorta. In contrast the values of phenoxybenzamine-insensitive uptake, a component of uptake which is independent of tissue type (6), were the same for both vessels.

The fluorescent band in the ear artery was between 2–3 times thicker than in the thoracic aorta (Table 4). In Figure 2, these bands are represented in an idealized semiquantitative manner based on the data presented in this paper. For the following reasons, it is to be expected that plexus thickness should influence the uptake of exogenous tritiated norepinephrine.

It is well known that neuronal uptake is related to the local concentration of norepinephrine (11, 14). This concentration for the unilayered plexus in the thoracic aorta would be similar to the bath concentration. With the ear artery on the other hand, although the concentration at the superficial nodes would be similar to that in the bath, the concentration within the plexus would be lower, since only some of the tritiated norepinephrine entering the plexus would escape uptake by the outer nodal layers. The quantity of tritiated norepinephrine taken up per node or per unit norepinephrine content would be less in a vessel with a thick plexus compared to one...
with a thin plexus. Thus one of the consequences of the crowding together of nodes in a thick plexus is a diminished efficiency in the uptake of exogenous norepinephrine.

The distribution patterns of tritiated norepinephrine uptake reflect the greater density and width of the sympathetic plexus in the ear artery. The adventitial peak does not correspond to the distribution of the nodes alone since there is some evidence that tritiated norepinephrine uptake under the circumstances of these experiments is not limited to the terminal node-containing ground plexus (6). Except for the outer 15-20% of the media where sympathetic terminals are known to be, medial uptake in the thoracic aorta is independent of depth. In the ear artery, uptake at the inner and outer media is not significantly different. The mean level of uptake in the media of the ear artery is greater than that in the aorta, about 6 ml/g compared to 1.0 ml/g. It seems unlikely that such a difference could be explained on the basis of muscle cell density in the two vessels alone.

Because of the small amounts of transmitter released from these small preparations, release can be measured only by labeling the transmitter stores with tritiated norepinephrine and using a combined superfusion and transmural stimulation technique (7). Values for fractional release of labeled transmitter obtained in these experiments (thoracic aorta $2.4 \times 10^{-5}$, ear artery $5.4 \times 10^{-5}$) are close to those reported previously for other tissues with the exception of the reported values of $20 \times 10^{-5}$ (15) and $40 \times 10^{-5}$ (16) for the cat spleen and $21.6 \times 10^{-5}$ (17) for the rat cerebral cortex. Calculation of total fractional release from isotope-loading and washout experiments has been previously employed (18). On a tissue weight basis, the ear artery contains seven times as many nodes as the thoracic aorta and, after phenoxybenzamine release, almost nine times as much transmitter per impulse is present (Table 3). If, as is generally believed, phenoxybenzamine blocks the neuronal reuptake of released transmitter, these data should approximate the total transmitter released from each artery. Thus if we are to take all the measurements including those from fluorescent microscopy on their face value (for comments see Results and below) approximately equal quantities of transmitter are released from each node in both tissues by each pulse: 2200 molecules per node per impulse for the thoracic aorta and 2900 molecules for the ear artery. In this sense, the release mechanisms are similar in the two vessels. It may be noted that a similar number of molecules of endogenous norepinephrine has been estimated by biological assay to be released from the isolated rabbit pulmonary artery, i.e., 2000 molecules per node per pulse (12). The fractional release of tritium after phenoxybenzamine for the thoracic aorta and the ear artery was $2.4 \times 10^{-5}$ and $5.4 \times 10^{-5}$, respectively. The significant difference of these values suggests that the adrenergic transmitter storage and release mechanism in the two terminal systems is not the same. Confirmation and elucidation await further experimentation. Zimmerman and Whitmore (19) found evidence suggestive of the nonuniformity of the adrenergic system between different vessels, using a perfused system.

The data presented in Table 3 related to the difference in release before and after phenoxybenzamine led to the conclusion that reuptake of released norepinephrine was more complete in the ear artery than in the thoracic aorta. Thus the overflow from the plexus was less from the ear artery than from the thoracic aorta than would be expected on the basis of node counts or norepinephrine content.

Rather than conclude that nodal reuptake mechanisms are more efficient in the ear artery than in the thoracic aorta, which is not supported by the data, it is more likely that the higher proportion of transmitter taken up by the ear artery is the result of node crowding as it is in the case of exogenous norepinephrine (see above and Fig. 2). Transmitter released from nodes in the interior of the multilayered plexus of the ear artery would be liable to uptake not only by the nodes of origin but also by those at the exterior of the plexus. Thus the thicker the
plexus, all other matters being equal, the smaller the percent of released transmitter that would escape from the neural apparatus because of the increased extent of neuronal reuptake. Thus increase in plexus width as a mechanism of increasing the concentration of transmitter overflow would be effective to only a limited extent.

Although the fluorescence technique of measuring node density is rather crude at present, some errors are undoubtedly common to both vessels, substantiating the value of comparative rather than absolute counts. In this connection however, it is of interest that node-count estimates made from material prepared for fluorescent microscopy were very similar to those made previously from methylene-blue studies (12). Data shown in Table 4 confirm what is abundantly evident on a cursory visual examination of the fluorescence in these vessels—the thickness of the neural plexus and the density of nodes is greater in the ear artery than it is in the thoracic aorta.

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It is a pleasure to thank J. V. Osher and K. Singh for their technical assistance.

References

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