Active Constriction of Small Pulmonary Arteries in Rabbit

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With technical assistance of Mrs. Dorothy Elston

This communication deals with the demonstration of active pulmonary vasomotion by injecting plastic material (vinyl acetate) which forms casts of the small pulmonary arteries simultaneously on a control side and on the test side submitted to action of vasoconstrictor drugs. Comparisons were made by direct microscopic observations and by weighing the casts after the lung tissue had been digested. Other indices such as relative weight of the two lungs, relative denseness of vascularity and estimates of pulmonary resistance supplied supplementary evidence of pulmonary vasomotion.

The subject of vasomotor control of small pulmonary vessels is still controversial. The approach, so far, has been to note changes in pulmonary vascular resistance, calculated from simultaneous measurements of driving pressure and flow, after nervous stimulation or administration of a vasoconstrictor drug. A change in resistance gives indirect information regarding the geometry of the pulmonary vessels at the time, since resistance depends on viscosity and the geometry of the vessels.

We have attempted to approach the problem by developing a plastic injection technic similar to that of Boquet et al.* In addition, some estimate of resistance to flow could be made from the initial rate of flow, at a known pressure of injection.

Method

A plastic material, 12 per cent vinyl acetate in acetone† was injected into the pulmonary artery of rabbits. A plastic cast of the arterial side only of the pulmonary vascular tree was thus obtained. Casts of the venous side of the vasculature could also be made by injection into the pulmonary vein.

Rabbits weighing 2.0 to 3.5 Kg. were anaesthetised with urethane intraperitoneally (2.5 Gm./Kg.), supplemented when necessary with more urethane and occasionally with ether. Thoracotomy was performed with the animal connected to a positive-pressure respirator.

An injection needle placed in the main pulmonary artery was fixed from outside by an artery forceps with a hole (the size of the needle) between its blades. The soft tissue surrounding the needle sealed the hole and effectively prevented backflow. After the injection, the plastic material was allowed to set. This took a few seconds in the case of small vessels and 2 to 5 min. for larger vessels. After an experiment the soft tissues were digested with concentrated HCl and a cast was obtained within 24 hours.

For injection purposes a 25 ml. burette, connected to a size 15 needle, and rinsed with acetone, leaving a little acetone in the needle, was used. The burette was then filled with the plastic material and a known pressure was applied to it from a bottle through air transmission. An outside stop was applied to the stop-cock of the burette, so that a fixed resistance to flow was offered each time the stop-cock was opened. Pressures around 100 mm. Hg were found to be suitable. Higher pressures (up to 270 mm. Hg) tend to break the fine pulmonary vessels and may passively open up constricted vessels, thus masking the effect of a vasoconstrictor drug. Injection pressures below 50 mm. Hg did not seem to fill the small vessels completely.

In order to evaluate critically any procedure affecting pulmonary vascular resistance the inflation pressure was measured by disconnecting the respiratory pump, and connecting the trachea directly to a large bottle with known pressure just prior to injection.

According to our observations, the shrinkage of the plastic material on setting is of little practical significance in casts of small blood vessels. Histologic sections of the lung filled with this material showed that the small vessels filled completely, but some of the larger vessels looked hollow in the center. In any case this factor would affect the test and control side equally.

The relative viscosity of the plastic material ($\eta_p$) was determined as follows. Plastic material was
allowed to flow through a hypodermic needle at a known pressure head \( (P) \), and the rate of flow \( (F) \) was measured. The fluid resistance of the needle to the plastic flow \( (NR_p) \) was calculated as: Pressure \( (\text{mm. Hg})/\text{Flow (ml./sec.}) \). The fluid resistance of the same needle to water \( (NR_w) \) was then determined. At a constant temperature, the 'relative viscosity' of the plastic material \( (NR_p/ATRy) \), varied from 1.4 to 2 for the plastic material from different bottles, with one exception (4.2).

**TECHNIC**

**Microscopic Examination and "Gnarly Count."** The fine plastic endings of the cast were examined under a low power microscope, using a blind technic for the test or control specimens. Usually a little piece of the vascular tree was chosen at random, the fine endings clipped, and placed flat on a slide for examination. Twenty vessels were examined from each vascular tree for their size, and then graded arbitrarily from 0 to 4+ for "gnarliness"* as shown in figure 1. The endings in figure 1A would be graded as zero, whereas those in B and C would be graded as 4+. The total gnarly count was expressed as the percentage of maximum possible. If 20 vessels were examined, the maximum possible count would be 80; if the actual count were 40, it could be expressed as 50 per cent of the maximum.

To test the consistency of this subjective judgment, duplicate blind examinations were conducted on several specimens. Also, where 100 endings had been counted, the gnarly count for the first 50 was compared to that of the second 50. The results indicated that the samples were satisfactorily representative of the population. The results of microscopic examination in the left and right side in 27 control experiments also were not very different, either in the distribution of the size of vessels filled, or the gnarly count. The difference between the gnarly count on the two sides was not statistically significant, \( (0.8 > p > 0.7 \) by statistics of paired data). Perhaps the best way to establish confidence in the reliability of this gnarly count is to correlate the counts for the left side and the right side of the lungs, in control experiments and those experiments where the drug or other procedure affected both sides alike (fig. 2). The correlation coefficient is 0.93, and the error of estimate of the gnarly count is ±6.4.

**Relative Denseness and Weight.** The lungs were cut off at the hilum where the main branch of the pulmonary artery entered the lung parenchyma, and weighed (correct to ±0.05 Gm.). The volume of each lung (correct to ±0.05 ml.) was determined separately by fluid displacement in an apparatus devised by one of us (fig. 3). The weight of the cast was obtained after the soft tissues had been digested (correct to ±0.05 mg.).

Denseness of the lung, indicating its vascularity, was calculated by dividing the weight of the cast in mg. by the volume of the lung in ml. The vascularity of the left lung \( (L) \) could then be compared with that of the right \( (R) \) and the result expressed as: **Relative Denseness** = \( L/R \times 100 \). A similar calculation was made for weights of the cast: **Relative Weight**

\[
\frac{\text{weight of left cast per gm. of lung} \times 100}{\text{weight of right cast per gm. of lung}}.
\]

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Fig. 3. Apparatus designed for measurement of the volume of an excised lung.

Table 1.—Values in 27 Control Experiments

<table>
<thead>
<tr>
<th>Index</th>
<th>Mean value</th>
<th>Standard deviation</th>
<th>Standard error of mean</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gnarly count</td>
<td>13</td>
<td>11</td>
<td>2.1 %</td>
<td>of Max.</td>
</tr>
<tr>
<td>Relative denseness of cast L/R</td>
<td>94</td>
<td>16.8</td>
<td>3.2 %</td>
<td>%</td>
</tr>
<tr>
<td>Relative weight L/R</td>
<td>98</td>
<td>22.8</td>
<td>4.4 %</td>
<td>%</td>
</tr>
<tr>
<td>T.P.R. for inflation pressures S to 15 cm. of water</td>
<td>23</td>
<td>15.3</td>
<td>2.2 P.R.U.</td>
<td></td>
</tr>
</tbody>
</table>

This also was used for comparing the vascularity of the left and right side of the lung.

Total Pulmonary Resistance (TPR). An approximate estimate of the total pulmonary resistance was obtained by measuring the initial flow rate of the plastic material from the burette under known pressure (R) and subtracting from this, the resistance at the ejection needle, determined as described.

The question next arose as to whether this TPR value should be corrected, since the relative viscosity of the plastic material from different bottles varied. It was recognized that the plastic material would displace blood in the small vessels and that the initial resistance to flow would be proportional to the viscosity of blood, rather than to that of the plastic material. However, when the time for 4 ml. of the plastic flow is taken, the plastic had already entered the small vessels, and the viscosity of the plastic material would exert considerable effect on the value of TPR obtained. Because of this consideration it was decided to correct all TPR values to a relative viscosity of 2. This correction factor was selected because it is similar to the value for blood flowing in a small blood vessel. The dependence of the effective viscosity of blood upon the size of the tube when the diameter is below 0.5 mm. (the Fahreus effect), is now known to be due to the size of the red cells being finite compared to the diameter of the vessel. Obviously, the question of anomalous viscosity of this type does not arise in the case of the plastic material, since there are no large particles in it.

The resistance (TPR) as calculated above is not strictly a measure of the absolute amount of total pulmonary resistance and, among other things, depends upon the distensibility of the pulmonary artery, and its main branches, for part of the flow into the artery is needed to fill the arteries as they distend under the increased pressure during injection. This makes obvious the importance of using constant injection pressures when making comparisons. It is also important to use inflation pressures in comparable ranges. It was found that with increase in positive inflation pressures, a U-shaped curve was obtained for pulmonary vascular resistance, rather flat minimum value being reached between pressure of 8 to 15 cm. of water. Therefore, comparisons of resistance between test and control experiments were usually made within this range.

Results

Control Experiments. The lungs of 27 rabbits were studied as controls in which no test procedure was done. The casts on the two sides provided control values which could then be compared to those of other rabbits in which a test procedure was carried out, usually on the left side. Table 1 summarizes the findings for the various indices used in these control experiments.

The scatter about the mean values may well be because the inflation pressure is not equally transmitted to all alveoli of both lungs, for the effect of the inflation pressure on the capacity and resistance of the vascular bed may be considerable.

It was realized that all the effects shown after injection of a drug, though statistically significant, might be in response to the general stimulus of putting the injection needle in LPA. To rule this out, 5 per cent glucose in saline was injected in LPA in 1 experiment.
TABLE 2.—Effect of Norepinephrine in Rabbits

<table>
<thead>
<tr>
<th>Rabbit no.</th>
<th>Wt. (Kg.)</th>
<th>Dose and administration</th>
<th>Inflation pressure (cm. Hg)</th>
<th>Injection pressure mm. Hg</th>
<th>Relative denseness L/R X 100</th>
<th>Relative wt.</th>
<th>Gnarly count % of max.</th>
<th>Test</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 53</td>
<td>2.8</td>
<td>20 µg./ml. r. lung soaked for 5 min.</td>
<td>7.0</td>
<td>119</td>
<td>110</td>
<td>102</td>
<td>26</td>
<td>79</td>
<td>50</td>
</tr>
<tr>
<td>2. 51</td>
<td>3.2</td>
<td>20 µg./ml. 8.5 ml. in main PA rate 7.9 ml./min.</td>
<td>11.0</td>
<td>170</td>
<td>73</td>
<td>66</td>
<td>41</td>
<td>74</td>
<td></td>
</tr>
<tr>
<td>3. 64</td>
<td>2.5</td>
<td>1000 µg./ml. 3 ml. in LPA, rate 1.5 ml./min.</td>
<td>6.0</td>
<td>99</td>
<td>55</td>
<td>58</td>
<td>94</td>
<td>49</td>
<td>5</td>
</tr>
<tr>
<td>4. 62</td>
<td>2.9</td>
<td>1000 µg./ml. 2 ml. in LPA, rate 2 ml./min.</td>
<td>9.7</td>
<td>116</td>
<td>39</td>
<td>39</td>
<td>123</td>
<td>11</td>
<td>44</td>
</tr>
<tr>
<td>5. 4</td>
<td>3.4</td>
<td>40 µg./ml. 5 ml. in LPA</td>
<td>4.0</td>
<td>—</td>
<td>20</td>
<td>31</td>
<td>—</td>
<td>33</td>
<td>8</td>
</tr>
<tr>
<td>6. 5C</td>
<td>2.2</td>
<td>20 µg./ml. 5 ml. in LPA</td>
<td>12.0</td>
<td>255</td>
<td>48</td>
<td>41</td>
<td>47</td>
<td>37</td>
<td>18</td>
</tr>
<tr>
<td>7. 93</td>
<td>2.1</td>
<td>10 µg./ml. 5 ml. in LPA rate 1.3 ml./min.</td>
<td>8.0</td>
<td>97</td>
<td>114</td>
<td>103</td>
<td>49</td>
<td>28</td>
<td>26</td>
</tr>
<tr>
<td>8. 95</td>
<td>2.1</td>
<td>10 µg./ml. 5 ml. in LPA rate 1.3 ml./min.</td>
<td>9.0</td>
<td>95</td>
<td>86</td>
<td>100</td>
<td>27</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>9. 75</td>
<td>2.5</td>
<td>5 µg./ml. 5 ml. in LPA, rate 2.5 ml./min.</td>
<td>6.0</td>
<td>87</td>
<td>57</td>
<td>50</td>
<td>41</td>
<td>28</td>
<td>9</td>
</tr>
</tbody>
</table>

LPA, left pulmonary artery.

The injection pressure was 250 mm. Hg, and inflation pressure was 12.5 cm. of water. The results were: gnarly count 5 per cent, relative denseness 77 per cent, relative weight 86 per cent, total pulmonary resistance 10 P.R.U. None of these are statistically different from the control values of table 1.

Norepinephrine (NE) Experiments. The results of 9 experiments are summarised in table 2. The dosages varied from 2 to 8.5 ml. solution of NE in 5 per cent glucose saline. The strength of NE (base) varied from 1,000 µg./ml. to 5 µ/ml. In experiments 3 to 9 the NE was administered in left pulmonary artery (LPA) at a rate varying from 1.3 to 2.5 ml./min. In experiment 2, it was given in main pulmonary artery (main PA). The needle administering NE was always removed from the pulmonary artery before the plastic injection, so that it would not obstruct the plastic flow.

Figure 4 shows the striking difference in the microscopic appearance of the fine endings of the cast after NE. The test casts (C to G) show greatly increased gnarliness over the control casts (A and B), where the small vessels are relatively smooth and straight.

Table 3 summarizes the results of the norepinephrine experiments with the statistical evaluation of the differences in the various indices from the control values given in table 1. For each index the difference is significant.

In experiments 3 to 9, in which NE was injected in the left pulmonary artery, it probably reached, by recirculation, the other side in less concentration. This could account for the higher gnarly count even on the control side, compared to experiments (table 1) where no NE was injected. However, except in experiment no. 4, the gnarly count was always higher on the injected side, and the statistics of paired data shows it was significantly higher (p < .01). No valid reason was found for the deviation of results in experiment no. 4.

In one experiment, NE was applied locally to the right lung (no. 1 in table 2). An increase in relative denseness and relative weight was noted. Since in this particular case the right lung is the test lung, this increase indicates that the vascularity of the test side decreased. An increased TPR, and gnarly count were also noted. This showed that NE produced vasocostriction with local application also.

In two experiments the pulmonary venous side was injected through the left atrium after clamping the aorta. One experiment served as a control and NE was administered...
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FIG. 4. Untouched photographs of the plastic cast, using combinations of transmitted and reflected light. A and B, controls; C to G after NE.

TABLE 3.—Values of Indices in Norepinephrine Experiments

<table>
<thead>
<tr>
<th>Index</th>
<th>Mean value (%)</th>
<th>Standard deviation</th>
<th>Standard error</th>
<th>p (table 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gnarly count</td>
<td>35</td>
<td>20.0</td>
<td>7.1</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Relative density</td>
<td>50.5</td>
<td>31.2</td>
<td>12.5</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Relative weight</td>
<td>60.2</td>
<td>29.4</td>
<td>11.8</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Total pulmonary Res. P.R.U.</td>
<td>51.4</td>
<td>37.0</td>
<td>13.0</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

All the indices are significantly altered after NE.

The mean gnarly count is useful as an index in the tables, but the most appropriate evaluation is by the chi-square test on the actual numbers of vessels characterized as having the different grades of gnarliness. Figure 5A shows the distribution of grades given by the observer in the case of controls (994 vessels), control side of NE casts (160 vessels) and test side of NE controls (200 vessels). The shift in distribution from controls to NE controls, evidently representing the effects of a low concentration of NE, and from this to NE test is of very great statistical significance.

through the main PA in the other. The gnarly counts for the venous tree were 30 per cent of maximum in the test experiment, and 6 per cent in the control, suggesting that NE produces a constriction of small pulmonary veins also.
The data also show that the effect of the drug in producing gnarliness is more marked on the smaller vessels than on the larger arterial vessels. In figure 5B and C the data of 5A are separated into two categories according to whether the vessel observed was of diameter less or greater than 25 μ. There is evidently no tendency or bias of the observer to notice gnarliness more in smaller vessels, for the chi-square test shows that in the control casts the distribution of grades is not significantly different (p = 0.2). Yet for the NE controls and for the NE test casts the smaller vessels are significantly characterised more often as gnarly than are the large vessels (p < .001 and p < .05). The distributions for the large vessels (Figure 5C) are not significantly altered as between controls and NE controls (p = 0.7) i.e. by low concentrations of NE, but are significantly altered (p = .001) from the control sides to the test sides of the NE casts. This means that, for small concentrations of NE, the vessels smaller than 25 μ are much more constricted, but with greater concentrations the larger vessels are also affected.

Effect of Privine. Privine (-2-(1-Naphthylnethyl)methyl)imidazoline hydrochloride), a long acting vasoconstrictor was injected into the left pulmonary artery in 2 experiments. The effect is shown in figures 6 and 7. Compared to its own control side, the test side in figure 7
Fig. 7. Photograph of lung cast. Left, control lung; Right, after Privine.

resembles a winter tree, whereas the control looks like a summer tree in full leaf.

**Discussion**

It is presumed that the plastic cast reflects roughly the state of vessels just prior to death, for (a) the injection of plastic is made within 1 min. of the animal's death, (b) the contraction and relaxation of vascular smooth muscle is very slow, and (c) after a vasoconstrictor drug, such as Privine, it is possible to get a markedly constricted vascular tree on the test side, when its own control side resembles those in the control set of experiments (fig. 6). Even if the microscopic appearance of the cast does not accurately reflect the physiologic state, the differences in gnarliness and other indices between test and control specimens in the same animal must have a basis in real physiologic differences.

The appearance of the fine plastic endings under the microscope was noteworthy. It can be seen from figures 1 and 4, that vessels around 100 μ in size look like straight, relatively smooth tubes in control specimens; when they constrict in response to the pressor drugs used they are gnarled. This suggests that perhaps a band of smooth muscle in the form of an open helix might be responsible for active constriction. It is conceivable that such a helical band may escape histologic detection, either in a transverse or a longitudinal section. Larsell has clearly shown that in rabbits, the muscular coat is not continuous in small pulmonary arteries less than 100 μ in size. He also found nerve endings in the neighborhood of these muscle cells.

As to other indices, relative denseness and relative weight, compare the arterial capacity on the left side (usually the test side) with the right. All the intrapulmonary arteries would contribute to this capacity, since all lungs were separated from the rest of the tissues at the point where the pulmonary artery entered the lung parenchyma. A decrease in relative denseness and relative weight was noted after NE administration (p < .01 in all comparisons). Strictly speaking, this would indicate either a decrease in arterial capacity on the left or an increase in arterial capacity on the right side. This would not necessarily be accompanied by a corresponding change in resistance.

T.P.R. could be approximately estimated and was found to be increased significantly after NE (p < .01). The absolute values thus obtained are not valid, but comparisons between test and control experiments, at comparable injection pressures of the plastic material and comparable inflation pressures in the lungs are considered justifiable.
Thus, it can be said that even though each index separately may not be completely reliable, the evidence obtained from all these indices, together with evidence from examination of the cast by microscope and by the naked eye, makes a strong case for active vasoconstriction in rabbit lungs in response to NE or Privine. A similar effect of NE on pulmonary circulation has been reported by others.8-11

Rodbard12 introduced the concept that bronchomotor tone could influence pulmonary vascular resistance by increasing the pressure in alveoli where air was trapped. We do not think that this could significantly affect the results in this series for the following reasons:

1. NE has a slight dilator effect on the bronchioles, therefore there is little chance of air being trapped due to inequalities in alveolar pressure, which will equal the pressure in the bottle to which the lungs were connected.

2. While 'passive' effects could alter the other three indices used, it is difficult to see how gnarliness could result from any type of outside force acting on the small vessels, rather than specifically from contraction of a muscle element in the wall having that particular helical configuration.

The aim of this communication is to present the evidence of the existence of vasomotor control in the small arteries of the rabbit lung. It will require much more research to establish the physiologic function of this control. However, in the course of the work, there were several indications of this. In 6 experiments where the pulmonary veins were clamped to produce venous congestion, and released just before the plastic injection (except in 2 cases) the casts gave statistically significant evidence of vasoconstriction by gnarly count, on the congested side, (.02 > p > .01) suggesting that there was a vena-vasomotor reflex in the lungs. In 2 rabbits that died for an unexplained reason just after intraperitoneal injection of urethane, the casts showed violent spasm of the small vessels (fig. 1 is taken from one of these). One rabbit had a cyst in one lung, and the vessels were markedly constricted. It may be difficult to see clearly the role of intrinsic vasomotor control of the lungs in normal physiology, but it is easy to speculate as to its role in abnormal physiology or disease.

**Summary**

In this research, a plastic material (vinyl acetate) was injected through a needle tied in the pulmonary artery of anesthetized rabbits. Both pressure of injection (about 100 mm. Hg) and pressure of inflation of the lungs (from 8 to 15 cm. water) were controlled at the time of injection. The material hardened in a few seconds in the small vessels. Subsequent digestion of the tissue by strong acid gave casts of the arterial vascular tree, with endings down to 15 μ diameter.

Microscopic examination of the fine endings of the casts allowed grading, by a blind technic, of the degree of constriction (gnarly count). Other indices used were the weights of the casts, their denseness, i.e., weight of cast divided by volume of undigested lung tissue, and the total pulmonary resistance calculated from the pressure and rate of injection. These indices were obtained in control experiments, where no test procedure was used, and in experiments where, just before injection, norepinephrine in doses from 10 μg. up, or Privine (concentration 1 in 2,000) in some experiments were perfused through both sides, in others through one side only of the lungs. Relative indices, of test side over control side of the lungs, were used in the latter cases.

In norepinephrine experiments the casts of the small vessels, which were relatively smooth and straight in the control experiments and on the control side of test experiments, were markedly grooved and contorted (gnarly). The gnarly count was increased from 13 to 35 (out of a highest possible gnarly count of 100); the weight of the cast on the test side was only 60 per cent of that on the control side. The total pulmonary resistance was increased from 13 to 51 P.R.U. All these differences were significant statistically (p < .01). Taken together, the results give strong evidence that the small pulmonary vessels can exercise considerable vasomotor control on the blood flow. The microscopic examination suggests that the smooth muscle bands may be in the form of open helices, and so might escape histologic detection in either transverse or longitudinal sections.
ACKNOWLEDGMENTS

We are indebted to Mr. C. E. Jarvis and Mr. J. E. Walker of the Department of Microscopic Anatomy for photography of the casts and for histologic sections, and to Mr. W. Austin of the Photographic Department of the Medical School for special photography of the casts.

SUMMAIO IN INTERLINGUA

In le hic-reportate investigationes, un plastico (vinyl acetate) esseva injicite via un agulia fixate in le arteria pulmonar de conilios anesthesiate. Regulation del pression injectori (a circa 100 mm Hg) e del pression inflational del pulmones (a inter 8 e 15 cm aqua) esseva mantenite al tempore del injection. Le plastico se solidificava in alicun secondas intra le parve vasos. Le subsequente digestion del histos per un forte acido resultava in modulos del vasculatura arterial con terminationes de diametros usque al minimo de 15 μ.

Le examine microscopic del terminaciones del modulos permitteva le differentiation de grados de constriction. Altere indices usate esseva le peso del modulos, lor densitate (i.e. le peso del modulo dividite per le volumine del histo pulmonar ante su digestion), e le resistentia pulmonar total (que esseva calculate ab le pression e le rapiditate del injection). Iste indices esseva determinate in experimentos de controlo (i.e. in le absentia de ulla manovra experimentatoriori additional) e in experimentos in que le injection del plastico esseva precedite immediatemente per un perfusion de un o de ambe lateres del pulmones con norepinephrina in doses de 10 μg o plus o, in un certo numero de experimentos, con Privina in un concentration de 1 a 2.000. In le caso de perfusion unilateral, indices relative esseva establite per dividir le valores del latere experimental per le valores del latere de controlo.

In experimentos con norepinephrina, le modulos del parve vasos—que esseva relativamente lisie e rectilinee in le experimentos de controlo e etiam al latere de controlo in casos de injection unilateral de norepinephrina—esseva marcatemente rugate e contorquite. Post norepinephrina, le indice de rugation (basate super 100 como maximum possibile) cresceva ab le valor de controlo de 13 al nivello de 35. Le peso del modulo al latere perfusionate esseva solmente 60 pro cento del correspondente peso al latere de controlo. Le resistentia pulmonar total cresceva ab 13 in le experimentos de controlo a 51 unitates in le experimentos post-perfusional. Omne iste differentias esseva statisticamente significative (p < 0,01). In lor totalitate le resultatos supporta fortemente le conception que le parve vasos pulmonar es capace a exercer un considerable influencia vasomotori super le fluxo de sanguine. Le examine microscopic suggere que le bandas de musculo lisie ha possibilemente le forma de helices aperte. Isto explica que illos escappa al detection histologic in sectiones transverse e longitudinal.

REFERENCES

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