Reflex Vascular Capacity Reduction in the Dog

CARL F. ROTHE, PH.D.

SUMMARY The maximum degree and time course of active reflex venoconstriction in chloralose-anesthetized dogs was studied. The mean circulatory filling pressure (Pmc) was measured by fibrillating the heart and rapidly pumping blood from the aorta to the vena cava until the systemic arterial pressure equaled the central venous pressure. Ventricular fibrillation was continued for 1 minute and was assumed to induce maximal sympathetic discharge to the capacity vessels. Blood was removed to maintain the Pmc constant at the level determined at 8 seconds after the start of fibrillation. It was necessary to remove 13.8 ml/kg by the end of 1 minute. After autonomic nervous system blockade by hexamethonium only 4.8 ml/kg were removed to hold the Pmc constant. Thus, in conclusion, the maximal degree of active venoconstriction induced by circulatory arrest was 9.0 ml/kg during the 1st minute of cardiac fibrillation. A basal capacity vessel tone was present, equivalent to 10 ml/kg under the experimental conditions used.

THE MAXIMUM degree and time course of active, reflex venoconstriction to transfer blood from the capacity vessels to the heart in response to hemorrhage, low cardiac output, or blood pooling still is uncertain. In a previous study it was concluded that "sympathetic activity can alter the total vascular capacity of the body by about 13 ml/kg." In that study, the total vascular pressure-volume relationship was measured 0.5, 2, and 5 minutes after hemorrhage or transfusion by estimating the mean circulatory filling pressure (Pmc) at a given total blood volume. The Pmc was obtained by fibrillating the heart and rapidly transferring blood from the aorta to the inferior vena cava until the pressures equilibrated. In addition, reflexes were blocked with hexamethonium, or the vasculature was stimulated with norepinephrine. The difference between the hexamethonium block and the norepinephrine stimulation was considered to be the range of smooth muscle activity of the capacity vessels. However, the technique required 30 seconds from the start of rapid hemorrhage or transfusion before the Pmc could be measured. Significant reflex compensation would likely have already been made. Furthermore, the stimulus may not have been maximal, although the large hemorrhage reduced the systemic arterial pressures to less than 40 mm Hg, the Pmc to less than 2 mm Hg, and a large dose of norepinephrine, in addition to the hemorrhage, did not significantly change the response.

In this study the goal was to evaluate the capacity vessel response to maximal sympathetic stimulation during the 1st minute of response. The heart was fibrillated for 1 minute and blood was removed from the vasculature to keep the Pmc constant at the value recorded soon after (8 seconds) the start of fibrillation. Within 8 seconds after the start of fibrillation and the Pmc maneuver, I assumed that the passive elastic recoil of the arteries and the passive elastic distention of the large veins had reached equilibrium, but that the smooth muscle surrounding the capacity vessels had not, as yet, started to contract. The measured volume removed during the 1 minute of fibrillation provided an estimate of reflex venoconstriction from a basal level in the anesthetized dog. A near zero arterial pressure and zero flow through the brain and chemoreceptors gave, I assumed, a near maximal stimulus for active venoconstriction. After administering a ganglionic blocking agent, hexamethonium, a blood transfusion was given to restore the Pmc to the control values. The difference in response before and after ganglionic blockade was considered to be the active reflex venoconstriction; the residual response was attributed to passive, viscoelastic responses of the vasculature related to either the vascular wall properties or blood flow from the periphery to the thorax.

Methods

Fourteen mongrel dogs (12 female) weighing 14.4 ± 2.6 (SD) kg, were anesthetized with α-chloralose (Nutritional Biochemicals). In some dogs methohexital (Brevital, Lilly), 12.5 mg/kg, iv, was used to induce the anesthesia, and with others 12.5% urethane (ethyl carbamate, Fisher) was used with chloralose (40 mg/kg as 2% solution) to reduce the volume of fluid infused. Heparin (Nutritional Biochemicals), 500 U/kg, was used as the anticoagulant. The dogs were intubated to ensure a free airway and to permit occasional positive-pressure respiration. They were placed on their left sides on a table through which warm water was circulated to maintain a body temperature of about 39°C.

The Pmc technique was similar to that used before. Catheters were tied in both femoral arteries and in both femoral veins for rapid blood transfer, and were placed in one carotid artery and one jugular vein to monitor aortic and central venous (right atrial) pressures. In five experiments a plastic Y-connector with an inside diameter (i.d.) of 4 mm was placed in the other jugular vein for insertion of a 5-Fr. Swan-Ganz (Edwards) catheter to monitor pulmonary artery pressure. The arterial and venous catheter tips for blood transfer were at the level of the diaphragm, while the catheter tips for arterial and central venous pressure (Pcv) measurement and servo control were at the arch of the aorta and in the right atrium, respectively. Fibrillation was induced with a 3- to 10-V, 60-Hz stimulus between a needle in the skin over the sternum and three strands of 0.13-mm stainless steel wire, 5 cm long, located in the right atrium.
and right ventricle. The electrode system was inserted coaxially within the jugular vein catheter. Because the stimulus occasionally caused an increase in the Pmc, it was applied for only 2 seconds in most experiments. If the heart started to beat spontaneously the run was aborted. Defibrillation was obtained by a 75- to 150-joule, DC countershock (model D84M, Electrodyne).

A roller pump (Cooley, Med-Science Electronics) was used to transfer blood at about 1,800 ml/min, initially, from the aorta to the inferior vena cava via the polyethylene catheters (2.7-mm i.d.) in each femoral artery and vein. An analog computer (TR20, Electronic Associates) was used to provide a servo control for the electronically modified pump to bring and then maintain the aortic pressure equal to the Pcv. A combination of integral and proportional control was used. The resultant error was less than 1 mm Hg. The pump was electronically and hydraulically switched 20 seconds after the last heart beat to pump blood from both the vena cava and aorta to a reservoir (a 500-ml polypropylene graduated cylinder) to clamp the Pcv at the reference Pmc value. The volume was measured continuously with an electromagnetic flowmeter and integrator. The volume in the reservoir was read periodically directly from the graduated scale of the reservoir.

The analog computer also was patched to hold the value of Pmc found at 8 seconds after the last heart beat to provide the Pmc servo reference pressure. This 8-second value was an insignificant 0.03 ± 0.66 (RMSE) mm Hg less than that estimated from the recording of the data (Beckman, type R Dynograph), and averaged a small and insignificant \( P = 0.09 \) 0.12 ± 0.51 (RMSE) mm Hg higher than that estimated at 5 seconds for all runs reported in Table 1. [RMSE is the square root of the error mean square by one-way analysis of variance of four sets of observations (Table 1).] I thus considered the reference value used to be a reasonable estimate of the Pmc. The Pmc and Pcv were measured in reference to the pressure of the open, half-filled right atrium measured at autopsy with the dog remaining on its left side.

After two or three 10-second periods of fibrillation to obtain control values of Pmc with minimum stress, the fibrillation was continued for 60 seconds. Starting 20 seconds after the start of fibrillation, the Pcv [with arterial pressure \( (Pa) = Pcv \)] was clamped at the Pmc, estimated at 8 seconds, by pumping blood into the reservoir to provide a measure of total body venoconstriction.

After two or three determinations of the volume that had to be removed to keep the Pmc constant during 60 seconds of fibrillation (60-second volume), hexamethonium chloride, 10 mg/kg (Nutritional Biochemicals, as a 20 mg/ml solution in saline) was given intravenously. The Pmc was checked within 10 minutes and then 10 ml/kg of dog blood from a previous experiment was infused to restore the Pmc to a level similar to that found before ganglionic blockade. This was done to minimize possible changes in capacity vessel responsiveness from operating at a different point on the vascular pressure-volume curve. The blood for transfusion was checked the day before for compatibility by microscopic examination for agglutination or for hemolysis when mixing donor cells with recipient plasma and recipient cells with donor plasma. Less than 10% showed incompatibility; in those cases I chose a different experimental dog.

After ganglionic blockade and a 60-second fibrillation, further reflex blockade was attempted by infusing an additional 10 mg/kg of hexamethonium (nine dogs), 0.2 mg/kg of propranolol (Inferal, Ayerst) (seven dogs), 1.5 mg/kg of phenolamine mesylate (Registine, CIBA) (three dogs), or 1 mg/kg pentolinium (Ansolsyen, Wyeth) (three dogs), or some combination. All data are presented as mean ± SD.

## Results

Venoconstriction, early in the experiment before ganglionic blockade, required that 13.8 ± 4.7 ml/kg of blood be removed within 1 minute to maintain the Pmc at the 11.0 ± 1.7 found at 8 seconds after the start of ventricular fibrillation (Fig. 1). During the interval from 5 to 8 seconds, the Pmc had increased an insignificant 0.39 ± 0.74 mm Hg, but from 5 to 20 seconds, the Pmc had increased a significant 3.6 ± 2.0 mm Hg (Fig. 1). With two or three subsequent control runs, the volume removed to keep Pcv = Pa = Pmc decreased to 11.0 ± 3.9 ml/kg and the 5- to 20-second Pmc increase declined to 2.0 ± 1.4 mm Hg (Table 1). After hexamethonium block and the 10 ml/kg blood transfusion to restore the Pmc, the volume removed by 60 seconds was 5.0 ± 2.2 ml/kg, a significant decrease, but also significantly greater than zero. Further runs with additional

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>Before blockade</th>
<th>After blockade</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Early</td>
<td>Late</td>
</tr>
<tr>
<td>Time from anesthesia (min)</td>
<td>104 ± 19</td>
<td>132 ± 20</td>
</tr>
<tr>
<td>Volume recovered, Pmc constant (ml/kg)</td>
<td>13.8 ± 4.7</td>
<td>11.0 ± 3.9</td>
</tr>
<tr>
<td>5-20 sec Pmc rise (mm Hg)</td>
<td>3.6 ± 2.0</td>
<td>2.0 ± 1.4</td>
</tr>
<tr>
<td>Mean circulatory pressure (mm Hg)</td>
<td>11.0 ± 1.7</td>
<td>10.9 ± 1.2</td>
</tr>
<tr>
<td>Systemic arterial pressure (mm Hg)</td>
<td>141 ± 11</td>
<td>136 ± 12</td>
</tr>
<tr>
<td>Central venous pressure (mm Hg)</td>
<td>-0.6 ± 0.8</td>
<td>-0.6 ± 1.2</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>144 ± 29</td>
<td>169 ± 34</td>
</tr>
</tbody>
</table>

Results are expressed as mean ± SD; \( n \) (number of dogs) = 14.

Before blockade, the "Early" column data were from the first determination of volume removed to hold the mean circulatory filling pressure (Pmc) constant during 1 minute of cardiac fibrillation and zero cardiac output; the "Late" set were from the determination just before administration of hexamethonium. After blockade, the "Early" data were from the determination about 10 minutes after the 10 ml/kg blood transfusion. The final "Late" column data were from the determination after administering additional blocking agents.

* Probabilities of differences by chance between the "Before blockade" and "After blockade" data.
REFLEX CONTROL OF VASCULAR CAPACITY

Pcv suggests a decrease in cardiac contractility or peripheral vascular resistance, or both, as a result of sympathetic blockade.

The systemic arterial pressure rapidly increased soon after the heart was defibrillated, especially in those preparations which had only 10 seconds of fibrillation. Before blockade, the peak pressure attained was 179 ± 26 mm Hg, an overshoot of 38 ± 21 mm Hg beyond the arterial pressure recorded before fibrillation. Within 7 ± 2 minutes after hexamethonium was given, the overshoot following 10 seconds of fibrillation was eliminated (−3 ± 14 mm Hg). Since the right heart, especially, is distended during fibrillation, the overshoot is not entirely attributable to reflex activity.

PRESSURE EQUILIBRIUM DURING THE Pmc MANEUVER

To monitor the pressure in blood vessels occluded by the femoral artery, carotid artery, or jugular vein catheters, a secondary set of catheters was inserted into these vessels (in nine experiments) in the direction opposite to that of the primary catheter. The assumption of completely equal pressures and so zero flow within the system 5 seconds after fibrillation was not supportable from data gathered in this series of experiments. The pressure in one of the femoral arteries downstream beyond the occlusion decreased from 94 mm Hg (Table 2) to a value 20 ± 4 mm Hg greater than the Pmc at 5 seconds and 19 ± 8 mm Hg at 20 seconds after the start of fibrillation. The distal femoral arterial pressure then increased progressively to 72 ± 41 mm Hg above the Pmc at 51 ± 15 seconds and so occasionally was greater than the prefibrillation arterial pressure. With ganglionic blockade, the 5- and 20-second values were 16 ± 5 and 12 ± 7 mm Hg above the Pmc; a significant reduction, but certainly greater than zero. After blockade, the distal femoral arterial pressure did not increase significantly after reaching the minimum. The carotid arterial pressure on the brain side of the cannulation was also significantly greater than the Pmc at 5 and 20 seconds (7.9 ± 4.1 and 2.8 ± 1.7 mm Hg, respectively), but attained equilibrium by 1 minute (0.4 ± 1.1 mm Hg). After ganglionic blockade, the differences were about half. The jugular venous pressure on the brain side of the cannulation was significantly less (−1.9 ±

### Table 2 Cardiovascular Variables After Hexamethonium and 10 ml/kg Blood Transfusion

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>( n )</th>
<th>Control</th>
<th>After hexamethonium</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central venous</td>
<td>9</td>
<td>−1.0 ± 0.6</td>
<td>−0.7 ± 1.0</td>
<td>NS</td>
</tr>
<tr>
<td>Cephalad jugular vein*</td>
<td>8</td>
<td>8.0 ± 2.5</td>
<td>7.4 ± 2.7</td>
<td>NS</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>3</td>
<td>14.6 ± 2.2</td>
<td>14.2 ± 1.0</td>
<td>NS</td>
</tr>
<tr>
<td>Aorta</td>
<td>9</td>
<td>139 ± 10</td>
<td>105 ± 22</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cephalad carotid artery†</td>
<td>9</td>
<td>108 ± 12</td>
<td>76 ± 15</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peripheral femoral artery†</td>
<td>9</td>
<td>94 ± 19</td>
<td>63 ± 17</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Results are expressed as mean ± SD; \( n \) = number of dogs; NS = not significant. Control data were obtained 32 ± 10 minutes before experimental.

* Pressure distal (upstream) to occlusion of vessel by cannulation.
† Pressure distal (downstream) to occlusion.
1.5 and -1.0 ± 0.8 mm Hg, respectively) than the Pmc at 5 and 20 seconds. In the five experiments in which the pulmonary arterial pressure was monitored, equilibrium was attained by 20 seconds, but the pressure was higher (2.1 ± 3.1 mm Hg) than the Pmc at 5 seconds. The pressures in those vessels distal to the cannulations are not representative of the remainder of the body, however, because pressure equilibrium required retrograde flow through collateral vessels.

These collateral blood vessels generally were large enough to maintain a reasonable perfusion pressure under normal conditions. The pressure in the carotid artery distal to the tie was 77.8 ± 8.7% of the systemic arterial pressure before fibrillation and before block. It was 67.4 ± 12.9% for the femoral (Table 2). After blockade, the ratio decreased significantly (paired comparison) to 74.0 ± 10.0% for the carotid and 60.6 ± 10.1% for the femoral. Following cardiac fibrillation, the massive sympathetic stimulation probably caused many blood vessels to constrict to such a degree as to be completely occluded. The increase in distal femoral arterial pressure to values sometimes higher than those observed before fibrillation can only be explained by large vessel occlusion by catheter, plus arteriolar constriction causing closure to prevent significant capillary flow, plus collateral vessel closure, plus increasing vascular smooth muscle contraction about the remaining contained blood. Although the blood pressure was not the same throughout the entire vascular system at 8 seconds, the vascular beds with occluded vessels (both hindlegs and the head) represent a minor fraction of the total vasculature volume and so do not necessarily invalidate the technique.

To bring the central arterial pressure equal to the Pcv, 4.2 ± 1.1 (RMSE) ml/kg of blood was transferred in 2 seconds by arterial to venous pumping. Pumping was continued so that by 10 seconds a total of 10.1 ± 2.6 (RMSE) ml/kg was transferred. During the next 10 seconds, only 4.1 ± 1.6 (RMSE) ml/kg was pumped. Ganglionic blockade tended to increase the volume that had to be pumped from arteries to veins to maintain Pa = Pcv during the first 20 seconds. The volume is attributable to retrograde flow from all of the peripheral arteries, but especially from those occluded by catheters—the hindlegs. Some may have come from the pulmonary bed, since the mean pulmonary filling pressure is 3.5 mm Hg higher than that in the systemic circulation.

A small amount of the 60-second volume is attributable to transcapillary fluid shifts into the vasculature, as the capillary pressure decreased with the cessation of blood flow. However, because the venular pressure was maintained at near normal levels in these studies, the capillary pressure changes and fluid shifts were probably much less than would be expected following massive hemorrhage.

Discussion

Active venoconstriction caused a net blood volume transfer of 9 ml/kg in 1 minute (Fig. 1). Although there seems to be a delay of 5–10 seconds before the smooth muscle starts contracting, this mechanism provides a rapid and effective mechanism to compensate for blood pooling in dependent extremities or to aid cardiac filling. After the 1st minute of vigorous stimulation, the rate of transfer slows. In an earlier study, the interpolated hemorrhage to attain an estimated Pmc of 4 mm Hg was 17.4, 23.8, and 26.5 ml/kg at 0.5, 2, and 5 minutes, respectively, after the start of a rapid (20-second) change in blood volume of 8.5, 17 or 25.5 ml/kg. Most of the 17.4 ml/kg of hemorrhage at 30 seconds was from elastic recoil resulting from the hemorrhage and drop in Pmc. The 6.4 ml/kg of hemorrhage between 0.5 and 2 minutes includes both continuing active venoconstriction and viscoelastic recoil. In another study, the added hemorrhage between 0.5 and 2 minutes was 7.7 ml/kg. In that study, dogs were rapidly bled to an arterial pressure of 40 mm Hg, the volumes removed at 0.5, 2, and 5 minutes were 29.0, 36.7, and 38.5 ml/kg, and the Pmc values were 2.2, 0.7, and 0.6 mm Hg, respectively. These data suggest that the rate of active venoconstriction between 1 and 2 minutes would be appreciably less than the initial 9 ml/kg per minute, and that it is virtually complete by 5 minutes, at which time transcapillary fluid shifts become important.

The pressure-volume-time relationship of the total vasculature is shown in Figure 2. There still is no information about the instantaneous vascular compliance, nor is there reliable information below a Pmc of about 4 mm Hg. From a previous study, it is clear that the relationship becomes nonlinear at very low distending pressures. Venoconstriction, expressed as ml/kg of volume transfer at a constant and normal Pmc, with maximal stimulation appears to be similar in magnitude to that seen at a Pmc of 4 mm Hg following a rapid massive hemorrhage, suggesting that...
venoconstriction causes a change in unstressed volume more than a change in compliance.

OTHER APPROACHES

The studies reported here are a more direct measure of vascular capacity vessel activity than experiments in which the circulation is maintained and Pcv is measured following hemorrhage or transfusion.4 With a functioning heart, the Pcv is not only dependent on the Pmc (assumed to be similar to the pressure in the major capacity vessels—the small veins and venules), but also depends on the balance between venous return and right ventricular output. Even if cardiac output is held constant,4, 8 changes in vascular resistance between the site of major blood storage (the venules and small veins) and the site of pressure measurement (right atrium) may confound the results. In addition, changes in distribution of flow can cause changes passively in contained blood volume in various segments of the body7 without any changes in capacity vessel smooth muscle activity. The method of Shoukas and Sagawa8 admittedly measures compliance (ΔV/ΔP) only, not changes in unstressed volume or vascular capacity. If the venous return is temporarily blocked and the venous pressure rise is measured while assuming a continuing and constant cardiac output,* it is difficult to justify the assumptions of equality of venous pressures throughout the system and constancy of continuing inflow to the various organ systems. Flow redistribution may occur. With flow continuing at the control cardiac output, there will be a pressure gradient throughout the venous system, also. Somehow, the pressure must be estimated at the site where most of the volume is located; the Pmc technique used in this study approaches this goal. Furthermore, a technique is needed to estimate vеноconstriction without a change in transmural pressure or compliance, i.e., a change in unstressed volume. Clamping the Pmc, as in this study, approaches this goal also.

Techniques for estimating vascular compliance using changes in blood volume1, 4, 8 may be erroneous because of the time required for the blood to enter or leave highly compliant regions. Caldini et al.3 and Green and Jackman12 (using the Caldini et al. data) have presented evidence for a vascular compartment with a large compliance and resistance for venous return with a resistance-capacitance (RC) time constant of the order of 25 seconds. This situation will lead to erroneous values for compliance if the segment is relatively large and if the Pcv must be assumed to be equal to the pressure in the capacity vessels (Pmc) within less than about a minute. This relationship, in addition to the difficulty of pumping a large volume into the vasculature within a few seconds and attaining equilibrium, precludes use of changing volume to study the early (10-30 seconds) reflex response of the vasculature. On the other hand, in this study the recorded Pmc is assumed to be closely similar to the pressure within the venules and small veins—the site of most of the blood. Thus, this pressure is not appreciably changed and so little or no volume needs to move from this region during the first 20 seconds of fibrillation. Assuming that the volume that would have had to be re-moved to hold Pcv at the Pmc during the 5- to 20-second interval is that shown by the dotted line of Figure 1, then at 20 seconds this volume would have been about 1.9 ml/kg. However, the volume was not removed and so the central venous pressure increased to 3.6 mm Hg. Note that the 1.9 ml/kg is not necessarily an actual distending volume, but is the volume that would have to be removed to restore the distending pressure to the control value.

ANESTHESIA AND ADEQUACY OF GANGLIONIC BLOCKADE

α-Chloralose was used to minimize both the sympathetic stimulation effect of anesthesia and possible attenuation of cardiovascular reflex activity. If the smooth muscles of the capacity vessels were stimulated reflexly by the anesthesia, with respect to the unanesthetized basal level, then the added amount of reflex activity that could be induced by temporary circulatory arrest would be reduced, since the control (baseline) venomotor tone would have been elevated by the anesthetic. For ease of induction and to minimize the volume of fluid used, methohexital and urethane, respectively, also were used. The mean arterial blood pressure and heart rate (Table 1) were higher than expected. Cox,13 for example, reported resting unanesthetized values for dogs of 101 mm Hg and 73 beats/min, respectively, and no significant changes (110 mm Hg and 82 beats/min) 15 minutes after induction of anesthesia with 100 mg/kg of α-chloralose. Because of the high heart rate and arterial blood pressure found under control conditions, in comparison to the resting, unanesthetized dog, various combinations of these agents were tried, including chloralose alone (100 mg/kg, as a 0.83% solution), but no statistically significant effect of the methohexital or urethane could be found. An excessive dose or deterioration of the crystalline α-chloralose are other possibilities for consideration.

Because the arterial pressure was reduced to only 102 ± 10 mm Hg immediately after blockade, and averaged 108 mm Hg after the blood transfusion, ganglionic blockade was possibly incomplete. Sagawa and Eisner14 recorded an arterial blood pressure of 40-60 mm Hg after 30 mg/kg of hexamethonium, but even so, they saw an increase in vascular resistance with a drop in perfusion rate. Lokhandwala et al.15 found a blood pressure of 70 mm Hg in conscious dogs treated with 10 mg/kg of hexamethonium and a heart rate of 160 beats/min, a value they considered to be the intrinsic (complete neurogenic blockade) heart rate of the dog. In pentobarbital-anesthetized dogs, they reported an intrinsic rate of only 120 ± 4 beats/min (before hexamethonium the heart rate was 136 ± 3 beats/min and after, 118 ± 3). The heart rate of their hexamethonium-treated conscious dogs was closely similar to that of our chloralose-anesthetized dogs, but the blood pressure was lower. Although hexamethonium chloride is considered a general ganglionic blocking agent, some studies suggest that there are some pathways resistant to blockade and that some smooth muscle is stimulated.14 Phenolamine, used in three experiments, did not provide a significant increase in the degree of apparent blockade in our experiments, however.
Although the 60-second volume removed after blockade is probably a result of viscoelastic recoil and continuing backflow, without direct measurement of venous smooth muscle activity or a more certain blockade, such as from a high spinal anesthesia, it is not possible to be certain that the autonomic nervous system was totally blocked.

Since a 10 ml/kg transfusion of blood restored the Pmc to the value found just before blockade with 10 mg/kg hexamethonium, I conclude that there is a basal capacity vessel tone equivalent to about 10 ml/kg in these dogs. The concomitant reduction in cardiac output and vascular resistance following blockade with hexamethonium could have led to a redistribution of blood within the cardiovascular system and changed the arterial and central venous pressures, but the change in cardiac output should not have directly influenced the Pmc as it would the Pcv when the heart was beating. The possibility must be considered that the basal capacity vessel tone is the result of abnormal sympathetic nervous system activity, just as the arterial pressure and heart rate were higher than normal for a resting, unanesthetized dog. Since the 10 ml/kg transfusion caused a change of only 1.89 ± 1.62 mm Hg in pressure, the apparent compliance of the vasculature was 5.3 ml/kg per mm Hg—an unusually high value. However, a change in unstressed volume could account for some of the volume infused. Incomplete blockade at the time of the first postblockade Pmc determination (7 ± 2 minutes after hexamethonium) may also have led to a substantially higher Pmc than would have been recorded later without the blood transfusion.

In conclusion, the maximum degree of active venoconstriction in these dogs amounted to about 9 ml/kg during the first minute of cardiac fibrillation. In addition, there appeared to be a basal tone equivalent to about 10 ml/kg of blood, giving a total range of reflex controlled capacity vessel volume change of 15–20 ml/kg.

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