Regional Vasomotor Tone in Normotensive and Hypertensive Dogs

By Arnold H. Williams, M.D., and Henry A. Schroeder, M.D.

A new method for estimating vasomotor tone in a local circulation by means of the asystolic arterial pressure gradient was applied to the renal, femoral, brachial, and mesenteric vascular beds of normal dogs and of dogs made hypertensive by various technics. The splanchnic circulation apparently contributes the greatest proportion of the increase of resistance in anesthetized hypertensive animals. The correlation of vasomotor tone with diastolic pressure was good only in the mesenteric area. Some differences in these four areas were found between renal and neurogenic hypertension.

For many years it has been known that "essential" hypertension is due to an increase of total peripheral resistance.* Opinion diverges, however, about the location of the changes of vasomotor tone, some studies indicating that the increase is uniform throughout the body,¹ ² ³ ⁴ ⁵ ⁶ ⁷ ⁸ others suggesting that regional differences occur.⁹ ¹⁰ ¹¹ Possibly the regional differences depend upon the type of hypertension studied. Since in some ways the varieties of experimental hypertension are similar to those of "essential" hypertension,¹² patterns of regional vasomotor tone in the former should furnish some insight into the nature of the conditions. This study is concerned with establishing these patterns.

Because the usual approach to the estimation of regional vasomotor tone is both awkward and difficult, a new method was developed. The asystolic arterial pressure gradient, defined as the descending curve of intra-arterial pressure following occlusion of an artery supplying a local circulation, can be used to measure regional peripheral resistance. The measure is quantitative when collateral circulation is excluded and qualitative in the presence of collateral circulation.¹³ Although changes in the curves parallel changes of vasomotor tone, only one function of the gradient is directly applicable to the measurement of tone, namely, the level of intra-arterial pressure six seconds after arterial occlusion or EP₆.¹⁴ Because the value of EP₆ is relatively unaffected by systemic blood pressure, intra-arterial blood volume, or the rate of local flow, it provides an index of regional vasomotor tone. From such a value a crude approximation of the actual degree of constriction may be made in certain areas.¹⁵

The present report concerns measurements of regional vasomotor tone in four vascular

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areas of anesthetized dogs, both those with normal blood pressure and those with various types of hypertension. Evidence is presented that, of these areas, the mesenteric is most intimately concerned with the regulation of diastolic blood pressure. Although hypertension of the neurogenic variety has been ascribed to changes of cardiac output alone, vasoconstriction is present. The pattern of regional vasomotor tone in renal hypertension differs but mesenteric constriction is also marked.

Methods

Asystolic arterial pressure gradients were obtained in four major arterial beds of dogs anesthetized with pentobarbital, 0.02 Gm. per Kg. intravenously. Forty-three normal animals were used, determinations being made in the femoral region of 24 dogs, the brachial of 17, the renal of 22, and the superior mesenteric of 18 according to methods previously described. Usually measurements were taken in two areas, occasionally in four. Several determinations were made in each region and the order of procedure from one area to the next was varied intentionally. With few exceptions that of collateral flow were only noticed in the limbs. Collateral circulation was excluded from the brachial circulation in five instances by tight tourniquet; in five others the femoral was isolated similarly. Normally it was minimized by occluding these arteries above the origin of the deep or profundi branches and did not affect the first and second curves obtained.

The blood pressures of the "normal" dogs varied considerably. For purposes of comparison, those with diastolic levels above 110 mm. Hg were arbitrarily classified "spontaneous hypertensive," those with systolic pressures below 110 mm. as "shock." Hypertension was produced in three ways, a minimal rise of 30 mm. Hg mean pressure being considered significant. Partial constriction of one renal artery caused renal hypertension in 8 dogs, silk perinephritis in one, both methods being supplemented by contralateral nephrectomy. In some instances the affected kidney was explanted beneath the skin. In 4 dogs chronic neurogenic hypertension was engendered by section of the moderator nerves in two stages. Acute neurogenic hypertension was produced in 11 dogs by section of the vagi and ligation of the carotid arteries above and below the sinus. Progressive ligation of the arteries supplying the head induced chronic hypertension in one dog; in 8, normal saline solution was forcibly injected into the cisterna magna to cause the abrupt onset of hypertension.

In each dog asystolic gradients were obtained in two or more of the above mentioned areas. Only those curves which showed no evidence of artefact, collateral circulation, or atypical contour were included. In all instances the intra-arterial pressure six seconds after occlusion (EPs) was measured to the nearest 2 mm. Hg, changes of ±7 mm. being considered significant.

Results

1. Regional Values of EPs (figs. 1–4)

(a) Renal. Normal dogs showed low values of EPs, their range varying from 4 to 24 mm. Hg (average 16); there was no correlation with levels of arterial pressure. Of 4 dogs in "shock,"

![Graph showing regional values of EPs](http://circ.ahajournals.org/)

**FIG. 1.** Regional levels of EPs in normotensive and hypertensive dogs. The ends of each open bar represent the extreme, and the central lines the average values of EPs (in mm. Hg) obtained in the labeled regional circulations of dogs serving as controls (C), in chronic neurogenic hypertensive (CN) and in chronic renal hypertensive animals (R). The dotted bars represent control levels before the production of acute neurogenic hypertension (AN); the change is indicated by the open bars above. The number at the tip of each bar shows the number of gradient determinations, no more than two in any one area being taken from a single dog.

2 exhibited very low and 2 very high values. In chronic neurogenic hypertension values varied widely (2 to 48 mm.). Acute neurogenic hypertension consistently caused a slight rise (average 5 mm.) (fig. 2). The value of EPs quadrupled in the single experiment in which intracranial pressure was increased.
Levels of $E_P$ were markedly increased in renal hypertension, being 2 to 4 times normal distal to the Goldblatt clamp. However, renal decapsulation reduced values of $E_P$ to high normal values in the 2 hypertensive dogs on which such experiments were made (table 1). In another animal the lumen of the renal artery was obliterated at the site of the clamp, the entire blood supply coming through collateral channels. On the other hand higher values were present in dogs which had had hypertension for less than two months than in those which had the disease for longer periods. $E_P$ was also high in the animal with perinephritis (table 1).

(b) Mesenteric. Values of $E_P$ of normal dogs were remarkably constant in this area (30 to 38 mm. Hg). The variation was greater (19 to 53 mm.), however, in those whose diastolic pressures were higher. Two dogs were in shock, a very low value being found in one and a moderately low value in the other.

Levels were uniformly high in renal and neurogenic hypertension but not in the single dog with cerebral ischemia. In all dogs there was a good correlation between levels of diastolic pressure and $E_P$ (fig. 4). Both acute neurogenic hypertension and increased intracranial pressure caused marked increases, averaging 27 mm. and 33 mm. respectively (fig. 2).

(c) Brachial. Relatively constant values of $E_P$ were found in the brachial circulation of normal dogs (31 to 46 mm. Hg).* They were moderately or markedly elevated in "shock." Slight to moderate increases were usually seen in renal and neurogenic hypertension, but in 4 Goldblatt dogs the rise was considerable. In acute neurogenic hypertension the average increase was 25 mm.

(d) Femoral. In general, the levels of $E_P$ in this region (range 31 to 50 mm. Hg) were similar to those found in the brachial circulation. However, high levels of $E_P$, comparable to hypertensive values, were found in dogs with elevated diastolic pressures whether or not collateral was excluded. As in the other areas the range of $E_P$ was wide in shock.

Of the two highest values obtained, one was in the dog with cerebral ischemia. They were approximately normal in chronic neurogenic but were normal or moderately elevated in

Fig. 2. Effect of acute neurogenic hypertension on regional vasomotor tone. Each diagonal solid line represents the change of tone produced by acute neurogenic hypertension in a single dog. Its lower end shows the control level and the upper end the value after the increase of pressure. Similarly the dotted lines show the changes produced by an acute increase of intracranial pressure. Note that in most instances the slopes of the lines are similar. Both scales are in mm. Hg.

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* In this region, as in the femoral bed, the values of $E_P$ represent the sum of the effects of vasomotor tone and collateral circulation. The progressive reduction of collateral circulation by the application of tourniquets reduces levels of $E_P$. However, when collateral was excluded (with the exception of that passing through bone), values were similar (19 to 32 mm. Hg) to those obtained without exclusion of collateral. It was obvious that the effects of collateral might vary in different animals but it was assumed that these differences were unimportant when series of dogs were compared.
renal hypertension. Acute neurogenic hypertension caused a considerable increase of EP₆, averaging 30 mm. Hg.

2. Regional Patterns of EP₆ (Table 2)

(a) Normotension. In anesthetized normotensive dogs levels of EP₆ tend to be low in the kidney and mesentery, higher in the limbs (fig. 1).

(b) Shock or Hypotension. Although the number of dogs is small, values of EP₆ varied widely, probably depending upon the state of shock. They ranged from extremely low (1 to 8 mm.) to very high levels (43 mm.) in the kidneys, in the brachial area from normal (30 mm.) to high (52 mm.), in the femoral from low (18 mm.) to high (58 mm.), while those in the mesenteric bed were normal to low (2 to 38 mm.). Many of these values are shown in figures 3 and 4.

(c) Hypertension. In chronic neurogenic hypertensive dogs the principal increase of EP₆ was in the mesenteric bed (average 23 mm. Hg), with but slight elevations in the femoral (+5), brachial (+5) and renal (+7) areas (fig. 1). The principal change in chronic renal hypertensive dogs was also in the mesenteric region (average +20 mm. Hg) but levels were higher in other regions (renal, +30 mm. Hg; femoral, +12; brachial, +17). The femoral area was the only one involved in "spontaneous hypertension" (average +18 mm.). A similar situation was seen in the single dog with chronic cerebral ischemia, levels in the femoral bed being +35, but those in the mesenteric area only +5. Gradients were not determined in other areas. Femoral curves were obtained by digital pressure in 3 unanesthetized chronic hypertensive dogs. Value of EP₆ in a renal hypertensive dog was 60 mm., 77 in a neurogenic dog (a very high level), and 37 in a dog with both types of hypertension. Under Pentothal anesthesia the values changed to 46, 85 and 76 mm. respectively. Although the
shapes of the curves were excellent, the method, used in this way, has its obvious limitations.

Comparable increases of EP6 were found in the femoral (+30 mm.), brachial (+30), and mesenteric (+25) areas in acute neurogenic hypertension. However, only slight changes occurred in the kidney (+5) (figs. 1 and 2). Because of the small number of experiments the changes resulting from an acute increase of intracranial pressure are not clear. Although mesenteric values rose markedly in one instance (+56 mm.) the rise was smaller (+22) in another animal whose blood pressure changed less. In one dog renal values rose from 2 to 16 mm. Hg.

There was a large drop of arterial pressure across the Goldblatt clamp, averaging 92 mm. Hg systolic and 48 diastolic in four dogs (table 1). Pulse pressure distal to the clamp was very small, ranging from 6 to 20 mm. Hg.

3. Relations of Blood Pressure to Values of EP6 in Different Regions

Although there was no obvious relationship between levels of systolic pressure and regional values of EP6, the correlation between levels of diastolic pressure of all dogs and EP6 was good in the mesenteric area, fairly good in the limbs, but absent in the kidney (figs. 3 and 4).

* The correlation was with diastolic pressure in the smaller radicles of the mesenteric artery, an area where levels are lower than but presumably proportional to those in the aorta.

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**Fig. 4.** Relationship of mesenteric and femoral vasomotor tone to levels of diastolic pressure. Left. Mesenteric. This figure shows the determinations of EP6 in the mesenteric vascular beds of normotensive dogs, of animals with chronic neurogenic and chronic renal hypertension, and of the single dog with chronic hypertension due to cerebral ischemia. Note the close correlation of diastolic pressure with EP6. Right. Femoral. EP6 values in normotensive dogs, chronic neurogenic and chronic renal hypertensive dogs show a fairly good correlation with diastolic pressure at the lower levels.

All scales are in mm. Hg. No more than two values are entered for any one dog.
Table 1.—The Drop of Blood Pressure across the Goldblatt Clamp and Levels of Renal EP_{1} in Chronic Renal Hypertensive Dogs

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Duration of Hypertension (days)</th>
<th>Blood Pressure (mm. Hg)</th>
<th>Blood Pressure Difference (mm. Hg)</th>
<th>% Constricted (%)</th>
<th>Remarks</th>
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<tbody>
<tr>
<td>50*</td>
<td>130</td>
<td>310†</td>
<td>215</td>
<td>271</td>
<td>210</td>
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<tr>
<td>56</td>
<td>15</td>
<td>231</td>
<td>183</td>
<td>138</td>
<td>130</td>
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<tr>
<td>78</td>
<td>260</td>
<td>205†</td>
<td>145</td>
<td>147</td>
<td>127</td>
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<tr>
<td>112</td>
<td>90</td>
<td>218</td>
<td>176</td>
<td>156</td>
<td>135</td>
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<tr>
<td>113†</td>
<td>360</td>
<td>266</td>
<td>177</td>
<td>202</td>
<td>163</td>
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<tr>
<td>114</td>
<td>220</td>
<td>256</td>
<td>172</td>
<td>78</td>
<td>72</td>
</tr>
</tbody>
</table>

Averages ......................................... 92.4 48.6 40.1 87.2

* Hypertension produced by silk perinephritis.
† Goldblatt clamp on one branch of renal artery and pressures obtained in the other branch.
‡ Femoral arterial pressure.
§ Not included in averages.
 See Discussion for method.16

Table 2.—Typical Patterns of Vasomotor Tone in Various Vascular Beds

<table>
<thead>
<tr>
<th>Dog No.</th>
<th>Type of Hypertension*</th>
<th>Systemic B.P. Syst./Dias. mm. Hg</th>
<th>Mesenteric EP_{1} mm. Hg</th>
<th>% Constricted (%)</th>
<th>Renal EP_{1} mm. Hg</th>
<th>% Constricted (%)</th>
<th>Femoral EP_{1} mm. Hg</th>
<th>% Constricted (%)</th>
<th>Brachial EP_{1} mm. Hg</th>
<th>% Constricted (%)</th>
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<tr>
<td>65</td>
<td>Normal</td>
<td>135/104</td>
<td>25</td>
<td>—</td>
<td>14</td>
<td>68.5</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td>63</td>
<td>Normal</td>
<td>125/105</td>
<td>11</td>
<td>63.7</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td>14</td>
<td>Spontaneous Hypertension</td>
<td>149/124</td>
<td>21</td>
<td>75.9</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>89</td>
<td>Chronic Neurogenic Hypertension</td>
<td>180/134</td>
<td>35</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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</tr>
<tr>
<td>37</td>
<td>Chronic Cerebral Ischemia</td>
<td>178/137</td>
<td>65</td>
<td>54.2</td>
<td>42</td>
<td>74.4</td>
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<td>2</td>
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<td>100</td>
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<td>48</td>
<td>98.4</td>
<td>55</td>
<td>80.1</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>34</td>
<td>Chronic Renal Hypertension</td>
<td>197/148</td>
<td>60</td>
<td>52</td>
<td>44</td>
<td>89.2</td>
<td>48</td>
<td>77.5</td>
<td>54</td>
<td>80.0</td>
</tr>
<tr>
<td>78</td>
<td>Shock</td>
<td>201/162</td>
<td>50</td>
<td>—</td>
<td>44</td>
<td>89.2</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>50</td>
<td>Shock</td>
<td>310/215</td>
<td>52</td>
<td>—</td>
<td>35</td>
<td>85.5</td>
<td>38</td>
<td>71.7</td>
<td>50</td>
<td>78.7</td>
</tr>
<tr>
<td>4</td>
<td>Shock</td>
<td>90/52</td>
<td>2</td>
<td>7.5</td>
<td>57.8</td>
<td>38</td>
<td>40</td>
<td>73.5</td>
<td>41</td>
<td>73.5</td>
</tr>
<tr>
<td>9</td>
<td>Shock</td>
<td>86/78</td>
<td>43</td>
<td>88.8</td>
<td>41</td>
<td>73.8</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>Shock</td>
<td>86/65</td>
<td>43</td>
<td>88.8</td>
<td>41</td>
<td>73.8</td>
<td>—</td>
<td>—</td>
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</tr>
</tbody>
</table>

* See text.
† See Discussion for method.16
tal to the clamp but showed no relation to the systemic blood pressure. Values of EP, showed no correlation with pulse pressure in other regions.

**Discussion**

1. **Validity of the Methods**

There is little reason to believe that values of EP do not measure the vasomotor tone of a given region in normotensive and hypertensive states. The asystolic arterial pressure gradient of a local circulation represents the descending curve of intra-arterial pressure and is a function of outflow through the arterioles. Although the initial or a portion of the curve is influenced by a) the volume of blood in the artery at the time of occlusion, b) intra-arterial pressure and the elastic recoil of the arterial wall, c) viscosity of the blood, d) the number and size of arterioles in the vascular bed, and perhaps, but infrequently, by e) venous pressure.16* EP, is but little affected by levels of systemic pressure at a constant degree of tone (±7 mm. Hg).16† With the minimal degree of blood loss characteristic of the present experiments, it is probable that blood viscosity did not vary appreciably; there is no proof that this factor is related etiologically to any type of experimental hypertension.15

Unfortunately, because the level of EP is affected by the ratio of intra-arterial volume to the number and size of arterioles in a given region (a factor which differs in each area), one cannot compare the conductance and/or tone of different types of vascular beds by this method, except possibly of the femoral and brachial.16‡ For the method to be valid the ratios of EP to resistance must be constant in all regions. With such data as are available these specifications could not be met. Consequently it must be concluded that levels of EP in any region measure vasomotor tone in only that area.

A method was therefore devised whereby the actual degree of constriction might be made comparable in different areas.16 Renal and brachial pressure-flow curves were plotted against values of EP, and arbitrary scales constructed. The brachial scale presumably may be applied to the femoral area.§ However, at best, the method is approximate. The scales are based on extremes of 100 per cent dilatation and 100 per cent constriction, both impossible values in a functioning circulation, and no correction is entered for the effects of blood viscosity12, 14 or for collateral circulation. The method, although subject to error, provides a useful means for comparing the relative degree of constriction in one local circulation with another.

2. **The Effects of Regional Circulations on Total Peripheral Resistance**

As levels of systolic pressure are primarily regulated by cardiac output, and diastolic pressure, by peripheral resistance,25 the observed disparity of levels of systolic pressure and EP was to be expected. The correlation with diastolic pressure furnishes additional evidence for the validity of the EP method as a measure of vasomotor tone in some regions (higher values of pressure being associated with increased regional tone). This relationship was surprisingly good in the mesentery, better in the hindlimb than the forelimb, and absent in the kidney, indicating a descending order of importance of these structures in the primary regulation of diastolic pressure both in the anesthetized normal and hypertensive state. Theoretically, changes of vasomotor tone in an

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* Ordinary variations of venous pressure are of insufficient magnitude to affect levels of EP, except when very low values are obtained. In hypertension venous pressure is normal.13

† It is known that arterial pressure-volume factors, specifically intra-arterial volume and elastic recoil, are altered in hypertension. Intra-aortic volume may increase 100 per cent.26 Although changed intentionally during pressure-flow determinations, they influenced primarily the initial or a sections of the gradients of the kidney and forelimb; presumably the gradient of other vascular areas was similarly affected.16

‡ While variations in the ratio occur in a given

§ It is unfortunate that no mesenteric pressure-flow curves were obtained; they are difficult to make valid.
area of high conductance (low resistance and a high rate of blood flow) have greater effects upon total peripheral resistance (TPR) than do variations in beds with the opposite characteristics.2 However, the effects must be examined on a functional basis. Although both the kidney and mesentery fit into the former category, only the latter can be considered to be of primary importance in the regulation of blood pressure. At rest the fore and hindlimbs are areas of relatively low conductance2 and, although they assist in the regulatory function, they are probably relatively unimportant in the anesthetized state. Similar conclusions were drawn when maximal vasoconstriction was simulated in the limbs of man.27

3. Patterns of Regional Vasomotor Tone in Hypertension

Chronic neurogenic hypertension has been ascribed primarily to an increase of cardiac output18 although slight changes of peripheral resistance have been noted in a few instances.17, 28 In the present experiments tachycardia was one criterion by which the effectiveness of vagal section was evaluated. According to recent work,28 tachycardia should be associated with a normal degree of resistance. In our experiments, however, marked increases of EP₆ were found in the mesenteric area; in the others they were less. These findings do not agree with the theory that chronic neurogenic hypertension is solely dependent upon an increase of cardiac output. The onset of acute neurogenic hypertension caused marked rises of EP₆ in all areas studied except the kidney, and the changes were approximately proportional to the increase of pressure. Obviously, readjustments must occur in the limbs when the condition becomes chronic.

As comparable levels of pressure occur in neurogenic and renal hypertension, it would be expected that vasoconstriction would be more severe in the latter condition, cardiac output being normal.19 Indeed, such was the case, mesenteric levels of EP₆ being similar to the neurogenic conditions, but greater levels were found in the limbs. Although the kidney does not play a primary role in the regulation of pressure (see above), apparently this area may contribute to the increase of total peripheral resistance in renal hypertension. As the total resistance of a local circulation is equal to the sum of its component resistances29, 30 it is obvious that the Goldblatt clamp is implicated; in addition, intrarenal resistance may be increased. It is obvious that collateral circulation accounted for some of the high values of EP₆, but in general the levels were inversely related to the duration of the hypertension (a few days to 12 months). High levels of EP₆ were found in the single dog with silk peri-nephritis and at autopsy this kidney was enclosed in a thick fibrous avascular capsule.

If intrarenal resistance is increased in chronic renal hypertension, it is explained most logically by the theory that partial arterial constriction leads to renal ischemia, and, in turn, humoral vasoconstrictor substances are released which constrict the kidney.* Another explanation involves recurrent repeated stimulation of the renal sympathetic vasoconstrictor nerves by the mechanical action of the clamp. It is difficult to believe that this process could be continuous as the nerves probably would be damaged. A third explanation concerns the stimulation of these nerves, centrally or at their endings, by humoral constrictor substances, and resembles the first. Whatever the mechanism, it is possible that a certain element of reversible renal vasoconstriction may accompany renal ischemic hypertension. The previously observed drop of blood pressure across a Goldblatt clamp,28 which occurs even in the presence of collateral circulation, is confirmed.

The findings in anesthetized animals must be interpreted with caution, as they cannot be applied directly to the unanesthetized state. However, the general patterns found were surprising. In chronic renal hypertension the values of vasomotor tone in the limbs lie between the extremes found in acute and chronic neurogenic hypertension. Consequently if renal hypertension is caused by humoral vasoconstrictor substances, as is believed, their

* Acute experiments have shown that intrarenal constriction does not necessarily follow partial constriction of the renal artery.31
effects upon different vascular beds are probably not uniform.

4. Relative Degrees of Constriction in Different Vascular Beds

Rough as the calculations of the degree of constriction may be, the results obtained in the femoral, brachial, and renal vascular beds are illuminating (fig. 5). They show that the brachial and femoral areas are constricted relatively more than the renal in normotension and acute neurogenic hypertension. In the latter condition, however, the change of the degree more constricted than the brachial may be a manifestation of a large muscle mass.\(^\text{16}\)

**Summary and Conclusions**

1. Determinations of asystolic arterial pressure gradients were made in four major local circulations of anesthetized normal and hypertensive dogs. In all animals the relationship of vasomotor tone to levels of diastolic pressure was good in the mesenteric area, fair in the limbs, and poor in the kidney, indicating a descending order of importance of these beds in the primary regulation of diastolic pressure.

2. Acute neurogenic hypertension was accompanied by vasoconstriction in all these areas with, however, the renal changing least. On the other hand chronic neurogenic hypertension was primarily characterized by an increase of resistance in the mesenteric area, the other beds being but slightly constricted. Presumably readjustments occur when the condition becomes chronic. Chronic renal hypertension was associated with marked mesenteric vasoconstriction, with the limbs also participating. It is possible that constriction was present in the renal vascular beds.

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