Digital Vascular Response to Angiotensin II in Normotensive and Hypertensive Subjects

EVIDENCE FOR A QUALITATIVELY ABNORMAL RESPONSE TO ANGIOTENSIN IN ESSENTIAL HYPERTENSION

By Peter Gaskell, M.D., Ph.D.

ABSTRACT

The critical opening pressure (COP) of digital vessels after digital nerve block and the change in COP caused by angiotensin at 4 μg/kg per min intravenously after the block were compared in 9 normotensive subjects, 8 patients with essential hypertension and 7 patients with various kinds of renal hypertension. The COP after nerve block was less than 20 mm Hg in the normotensives and in 6 renal hypertensives, but more than 20 mm Hg in 7 essential hypertensives. The angiotensin reduced the COP in 7 of the normotensives, increased it substantially in 7 essential hypertensives, but had little effect in 6 renal hypertensives. A significantly greater increase in systemic blood pressure occurred in the essential hypertensives than in the normotensive subjects. In normotensive subjects doses of intravenous angiotensin from 2 to 16 μg/kg per min decreased the COP whether preceded by digital nerve block or not. Angiotensin at 8 μg/kg per min increased total digital vascular resistance as estimated by venous occlusion plethysmography. The results indicated that angiotensin relaxed smooth muscle in digital vessels of normal subjects but increased vascular resistance. In patients with essential hypertension angiotensin caused contraction of the smooth muscle, a response apparently qualitatively different from normal. In renal hypertensives the smooth muscle had little response to the exogenous angiotensin.

ADDITIONAL KEY WORDS

vascular reactivity vascular critical opening pressure vascular resistance renal hypertension digital nerve block angiotensin infusion unanesthetized man

In studies of hypertensive patients and experimentally hypertensive animals, it has been frequently reported that there is an increase in the reactivity of their vessels to a number of vasoconstrictor agents. An increased vascular reactivity or response to one of these agents, angiotensin II, has been found in hypertensives by several investigators (1-10) but this has not been confirmed by others (11, 12). "Vascular reactivity" and closely similar terms are used rather loosely in the literature. They may sometimes refer to general cardiovascular reactivity but are often defined only in terms of the parameter measured, such as the increase in systemic blood pressure or the degree of change in rate of blood flow or resistance to blood flow in a part of the body. But, while defining reactivity in terms of the parameter measured, the results are often interpreted as indicating differences in activity of the vascular smooth muscle. Thus, one has the impression that most authors are interested in the reactivity of vascular smooth muscle itself to a particular substance but that because of the lack of a direct measure of the force exerted by the muscle, they have had to fall back on indirect indices of its activity. In this paper experiments will be described in which the critical...
opening pressure (COP) of digital vessels was measured as a more direct index of the constricting force and of the change of constricting force exerted by vascular smooth muscle in response to angiotensin II in normal subjects and in patients with hypertension.

Methods

The method of measuring the critical opening pressure of digital vessels has recently been described in detail (13).

Reactivity Experiments. In all the hypertensive patients, tests were carried out before any treatment with antihypertensive agents or after the drugs had been withdrawn for several days. The patients also received 5 g of NaCl per day as enteric coated tablets in addition to an ordinary ward diet for 4 days before the test. A digital nerve block was performed shortly before the measurements of COP in the finger so that central nervous system control of the small vessels via the nerves was abolished during the administration of angiotensin. The extra salt was given because the tests were carried out in conjunction with the angiotensin infusion test described by Kaplan and Silh (14, 15). Tests were also carried out on normotensive subjects, some of whom received the extra salt to serve as controls for tests on the hypertensive patients. Similarly, the measurements of COP in the normotensive subjects were sometimes preceded by digital nerve block and sometimes not, as will be noted. The digital nerve block was produced by injecting 2% xylocaine into each side of the base of the finger.

Angiotensin II (Hypertensin®) was administered to all subjects by intravenous infusion in 5% dextrose solution. Throughout the experiment, an intravenous infusion of 5% dextrose into a forearm vein was maintained by motor driven syringes. A syringe containing the angiotensin in a concentration to deliver a dose of 4 μg/kg/min in 2 ml of solution was substituted for the syringe containing plain dextrose solution at the appropriate time. The standard dose of angiotensin for the hypertensive patients was 4 μg/kg/min. This dose was also administered to the normotensive subjects in the earlier tests but in later experiments other doses were used.

The general procedure for the vascular reactivity experiments was as follows: The subjects were supine, comfortably warm with the room temperature controlled at 20°C. The intravenous infusion was begun, a digital nerve block was performed if this was desired, and the hand was prepared for the measurement of the COP of the small vessels in the finger. The room was darkened and, after a short period of quiet to allow the subject to relax, measurements of COP were begun. The brachial blood pressure was measured every 2 to 4 min by auscultation in the opposite arm. The COP was estimated at least six times before angiotensin was administered. An interval of 3 min was allowed between the end of one estimation and the beginning of the next. The mean of the last six measurements before the angiotensin infusion was taken as the control value for COP. The angiotensin infusion was begun and after 5 min, six more measurements of COP were made and the mean of these was used as the COP during angiotensin administration. Angiotensin was then stopped and many of the experiments on hypertensive patients were terminated when the systemic pressure had returned to the pre-angiotensin level. Other experiments were continued until six more measurements of COP were obtained beginning 5 min after the end of the angiotensin infusion. Sometimes more than one dose of angiotensin was administered during an experiment. The change in COP caused by the angiotensin after digital nerve block was taken as an index of the reactivity of arteriolar smooth muscle in digital vessels (16).

Blood Flow Experiments. The effect of intravenously infused angiotensin on rate of digital blood flow when the vessels of the finger could not be influenced reflexly via the vasomotor nerves was investigated in normotensive subjects only. A digital nerve block was produced at the beginning of the experiment. Rate of blood flow was measured by venous occlusion plethysmography. However, digital nerve block ordinarily causes such a great rate of blood flow in the finger that it is difficult to measure it accurately by venous occlusion plethysmography. To keep the rate of flow low enough to measure with some assurance of accuracy, the finger was kept cool by means of a plethysmograph made of phosphor bronze shim-stock covering about half the length of the finger; the remaining space in the plethysmograph was filled with cool water (22°C). The whole hand, up to the wrist and including the plethysmograph, was then immersed in stirred water which was 22°C. The measure from the plethysmograph was connected to a small airtight reservoir supported slightly above the level of the hand and partly filled with water. Air pressure changes in the reservoir during venous occlusion were monitored by a Statham pressure gauge (PM5 ± 0.15 — 350) and recorded rectilinearly by an Offner Type R Dynograph. The trace was analyzed in the standard manner (17).

1The Hypertensin was kindly supplied through the courtesy of Dr. C. Walter Murphy, of Ciba Company, Limited, Dorval, Quebec.
After beginning the intravenous infusion of 5% dextrose, which was maintained throughout the experiment except when angiotensin in 5% dextrose was substituted for plain dextrose solution, and after producing digital nerve block and preparing the finger and hand for measuring blood flow, the room was darkened and the subject was allowed to relax before measurements of flow rate were begun. Rate of flow was measured every 20 sec during periods of approximately 2 min separated by intervals long enough to measure brachial blood pressure by auscultation and digital systolic blood pressure plethysmographically (within 5 mm Hg). Angiotensin in a dose of 8 mg/kg per min was administered twice during each experiment for periods of about 15 min.

The mean of the estimations of rate of flow during the last 10 min of angiotensin infusion was compared with the mean of flow rates during the 10-min period immediately preceding the angiotensin and that beginning 5 min after the end of the angiotensin infusion (Fig. 3).

The intravenous angiotensin increased the digital as well as the brachial blood pressure. Therefore, an Index of Resistance was calculated for each block of flow-rate measurements by dividing the mean flow rate in the block into the digital systolic pressure measured immediately afterward. Again the mean of the estimations of the Index of Resistance during the last 10 min of angiotensin infusion was compared with the mean of the indices during the 10-min period immediately preceding the angiotensin and that beginning 5 min after the end of the angiotensin infusion (Fig. 3).

Subjects. The normotensive subjects were young women 18 to 21 years old, and one young man (T.D.) of 21 years. The hypertensive patients, who were kindly referred by colleagues, had extensive investigation of the etiology of their hypertension carried out under the supervision of their own attending physicians. Eight were believed to have essential hypertension and 7 to have renal of various kinds. Renal arteriography was performed in all but 1 (J.S.) of the patients. In 2 (D.V. and A.P.), separated renal function studies indicated ischemia of the right kidney. Further information about each patient will be found in Table 1.

**Results**

**Reactivity Experiments**

Twenty-one normotensive subjects were given intravenous infusions of angiotensin II in 37 experiments. Figure 1 indicates the effect of the angiotensin on COP and brachial blood pressure in 9 subjects who were prepared for the test in the same manner as the hypertensive patients. Results in these experiments may be compared with those obtained in the hypertensive patients (Fig. 1). All 9 normotensive subjects were given a 4 mg/kg per min dose of angiotensin as were the hypertensives, but in addition some also received double that dose during the same experiment. In 2 of the 9 subjects the smaller dose of angiotensin produced a rise in COP but in 7 it caused a fall. In the group of 5 receiving the 8 mg/kg per min dose, the fall was more definite, but the systemic blood pressure was increased and, as will be seen, the rate of blood

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**Table 1**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>BP</th>
<th>Etiology of hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.S.</td>
<td>28</td>
<td>M</td>
<td>160/105</td>
<td>Essential</td>
</tr>
<tr>
<td>D.H.</td>
<td>28</td>
<td>M</td>
<td>190/120</td>
<td>Essential</td>
</tr>
<tr>
<td>N.C.</td>
<td>40</td>
<td>F</td>
<td>155/105</td>
<td>Essential</td>
</tr>
<tr>
<td>C.S.</td>
<td>50</td>
<td>M</td>
<td>240/120</td>
<td>Essential</td>
</tr>
<tr>
<td>M.B.</td>
<td>52</td>
<td>M</td>
<td>220/140</td>
<td>Essential</td>
</tr>
<tr>
<td>J.S.</td>
<td>58</td>
<td>M</td>
<td>180/115</td>
<td>Essential</td>
</tr>
<tr>
<td>S.S.</td>
<td>59</td>
<td>M</td>
<td>225/120</td>
<td>Essential</td>
</tr>
<tr>
<td>N.S.</td>
<td>59</td>
<td>F</td>
<td>220/120</td>
<td>Essential</td>
</tr>
<tr>
<td>D.V.</td>
<td>18</td>
<td>F</td>
<td>190/120</td>
<td>Renal artery stenosis</td>
</tr>
<tr>
<td>A.P.</td>
<td>62</td>
<td>F</td>
<td>210/115</td>
<td>Renal artery occlusion</td>
</tr>
<tr>
<td>S.F.</td>
<td>28</td>
<td>F</td>
<td>175/105</td>
<td>Diffuse renal lesion, on biopsy resem-</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>bles diffuse and exudative diabetic</td>
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<td></td>
<td></td>
<td></td>
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<td>glomerulosclerosis</td>
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</tbody>
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flow in the fingers was reduced by angiotensin after digital nerve block.

The effect of angiotensin on COP in hypertensive patients appears to depend upon the kind of hypertension present (Fig. 1). All received 4 μg/kilo per min of angiotensin. In 7 of the 8 believed to have essential hypertension, angiotensin caused a substantial increase in COP ranging from 9 to 18 mm Hg. In the eighth patient there was no change in COP.

Results in the 7 other patients, who were believed to have various types of renal hypertension, indicate (except in one case) that angiotensin produces little change in the COP of their digital vessels. This was true whether the hypertension was thought to be the result of renal artery stenosis or the result of intrarenal or parenchymal disease.

Among the hypertensive patients there was a difference also in the level of COP after digital nerve block, but before angiotensin administration. In all but 1 of those with essential hypertension, the COP was greater than 20 mm Hg. The COP of digital vessels in normal subjects after digital nerve block is 20 mm Hg or less, with a mean of about 10 mm Hg (13). All the normotensive subjects in this study had COP's of less than 20 mm Hg after nerve block. All but 1 (J.P.) of those patients who were thought to have renal hypertension of various types also had COP's in...
the normal range after digital nerve block. The difference between the COP after digital nerve block in patients with nonmalignant renal hypertension and those with essential hypertension was briefly reported in 1961 (18) and results obtained in a larger group of hypertensive patients will be published.

The results of the experiments illustrated in Figures 1 and 2 indicate that in the normotensive subjects intravenous angiotensin at 8 μg/kilo per min reduces the COP of digital vessels whether or not the subject is prepared by ingestion of extra salt before the test and whether or not the central nervous system control of the digital vessels is abolished by previous digital nerve block.

Doses of 1, 2, 4, 8, and 16 μg/kilo per min of angiotensin were administered to normal subjects after digital nerve block (Fig. 2). The smallest dose did not appear to change the COP. Two μg/kilo per min seldom affected it; when the dose was increased to 4 μg/kilo per min, the COP was reduced. This suggests that the threshold dose of intravenous angiotensin for producing a change in COP is about 2 to 4 μg/kilo per min. All the doses so far tried reduced the COP in the normotensive subjects, or had no effect on it at all.

**BLOOD PRESSURE RESPONSE**

The administration of angiotensin at 4 μg/kilo per min intravenously increased the brachial blood pressure in both normotensive and hypertensive subjects. The increases observed in the 9 normotensive subjects of Figure 1 were compared with those measured in the 8 patients with essential hypertension. The increase in diastolic blood pressure in any subject was taken as the difference between the mean of the pressures measured during approximately 10 min just before angiotensin and of those measured during 10 min begin-
VASCULAR RESPONSE TO ANGIOTENSIN II

The mean of the increases in diastolic pressure in the normotensive subjects was 10.9 ± 1.0 (SE) mm Hg and in the patients was 16.1 ± 1.8 (SE) mm Hg. The increase was significantly greater in the hypertensives (P < 0.02). Similarly, the mean of the increases in systolic pressure was 5.2 ± 1.2 (SE) mm Hg in normotensives and 21.1 ± 3.7 (SE) mm Hg in the hypertensives. The increase was significantly greater in the hypertensives (P < 0.01).

In the group of patients with renal hypertension, the mean increase in diastolic pressure was 10.5 ± 2.6 (SE) mm Hg. This was not significantly different from the mean increase in patients with essential hypertension (0.1 > P > 0.05) or from the mean increase in the normotensive subjects of Figure 1 (P > 0.8).

DIGITAL BLOOD FLOW STUDIES

It has already been shown that intravenous administration of angiotensin II will decrease the blood flow in digital vessels (5-7, 12, 19). However, in view of the reduction in COP caused by intravenous angiotensin, which suggests relaxation of arteriolar muscle, it was considered worthwhile to ascertain again the effect of angiotensin on rate of digital blood flow and resistance to blood flow. Figure 3 indicates the results in all five of the blood flow experiments in which a total of 10 infusions of angiotensin were administered. The resistance to blood flow in the digits was increased during the angiotensin infusion. In Figure 2 will be found results of reactivity experiments in 3 subjects in which intravenous angiotensin at 8 µg/kilo per min reduced the COP of digital vessels after digital nerve block and cooling of the fingers as in the blood flow experiments.

Discussion

Many investigators have reported that hypertensive patients have an increased vascular reactivity to various vasoconstrictor stimuli including angiotensin. The investigation of vascular reactivity in vivo has been of interest to me for some time and as a measure of it I have used the increase in COP of digital vessels in response to a vasoconstrictor stimulus such as norepinephrine (16). The opportunity to carry out studies with angiotensin II has, however, led to discovery, in patients with essential hypertension, of what appears to be a reaction to angiotensin qualitatively different from normal and not merely an increased vascular reactivity of the same kind as that seen in normal subjects. Furthermore, in normal subjects the response to angiotensin brought to light what appears to be a paradox. The intravenous infusion of angiotensin II reduced the COP of digital vessels and yet the resistance to blood flow in the finger was increased.

The critical opening pressure and the critical closing pressure (CCP) of vessels are believed to be measures of the constricting force exerted by vascular smooth muscle (20, 21). The COP and CCP are of the same magnitude in millimeters of mercury (13, 21, 22, 23) and are regarded as indicating the activity of arteriolar muscle (13). But metarterioles and precapillary sphincters, as well as arterioles, have been shown to close critically (24). One would expect a decrease in COP of digital vessels to be accompanied by an increase in digital blood flow and a reduction in digital vascular resistance unless concomitant changes in transmural pressures dictated otherwise. This expectation is realized in other circumstances. Local cold, if not extreme
enough to cause cold vasodilatation, is known to reduce rate of blood flow and increase vascular resistance in the finger and also to increase the COP of digital vessels (25). Noradrenaline increases digital vascular resistance and also the COP of digital vessels (16). Local warming and indirect heating both reduce digital vascular resistance and also the COP and CCP of digital vessels (13, 21, 26, 27). But in normal subjects, changes in vascular resistance and COP of digital vessels, in response to intravenous angiotensin, do not correspond in the usual way. Further work will be required to elucidate these observations but possible explanations might be suggested.

If one considers the method of measuring the COP and CCP in relation to the concept of CCP as introduced and developed by Burton (20), it will be realized that the COP or CCP as measured will be that of the vascular channels (among groups of parallel channels) which have the lowest COP or CCP. But in a particular channel the measured COP or CCP pertains to that segment of the "consecutive segments" of the channel which has the highest COP or CCP. Ordinarily this would be considered to be the arteriole. Suppose the COP is an index of constricting force exerted by the arterioles in the parallel channels with lowest COP. Would a variation in response of different channels account for the fall in COP and rise in resistance to blood flow if the estimation of COP indicated activity of the precapillary sphincter rather than of arteriolar muscle. Response of venular muscle to angiotensin must also be considered. Folkow et al. (28) have concluded that there are few or no receptors for angiotensin II in venular muscle and Haddy et al. reported angiotensin II increased the resistance mainly on the arterial side of the capillary (29). It is also unlikely that venular constriction in response to angiotensin would offset arteriolar dilatation enough to increase overall resistance to flow during angiotensin infusion. If this were to occur, rapid formation of oedema would result but it has not been observed. Larger arteries than arterioles may constrict while arteriolar muscle relaxes in response to angiotensin, increasing overall resistance. Many in vitro experiments involving larger vessels have shown that angiotensin can cause vascular smooth muscle to contract but usually the doses employed are large and probably not physiological.

There is at least one other possible explanation. The overall vascular resistance might be increased while smooth muscle in small vessels is relaxed if angiotensin infusion causes
swelling of the endothelium of all or part of the small vessels. Angiotensin may cause these cells to imbibe water or salt and water and to swell and decrease the lumen of capillaries, venules and arterioles, unless relaxation of arteriolar muscle allows enough distension of the arteriole to compensate for swelling of the intima in that particular segment. It is of interest that Dollery, Hill and Hodge studied the effect of angiotensin administration on retinal vessels of various sizes, both arterial and venous (30). They found that the lumen of all vessels was uniformly narrowed very slightly but “expressed as a proportion of the initial caliber the effect is greater in the smaller vessels than in the larger ones.” They point out that only the larger arterioles near the disc have a continuous coat of smooth muscle and they did not know “what contractile elements were concerned in the contraction of the smaller vessels.” Swelling of the endothelium could produce this picture.

In the present study, the reactivity of vessels in the finger was estimated by measurement of changes in critical opening pressure of digital vessels in response to the angiotensin. Theoretically (20), the critical closing pressure and the COP measure the force exerted by the smooth muscle at one particular diameter of the vessel—the “unstretched radius.” They do not depend on a measure of change in resistance to flow. Thus, they are more direct measures of force exerted by smooth muscle at a definite degree of stretch in particular vessels than are measures, such as rate of blood flow or resistance to flow, which pertain to all the vessels in the part under study. These factors confer an advantage on the measurement of COP over those measures mentioned above as a tool for comparing reactivities of certain vessels in different individuals or in the same individual at different times. There are, as with all the other measures, disadvantages. In measuring the COP or CCP, a short period (order of 30 to 90 sec) of reduced or absent blood flow in the part is produced and changes in force exerted by smooth muscle occurring during this period will lead to an error in estimating the COP obtaining at the start of the measurement. However, the error is not great and is likely to be nearly the same for most individuals and circumstances as far as vessels in the fingers are concerned (16). Another disadvantage of the measurement when comparing responses in different individuals, such as normotensive and hypertensive subjects, is that the estimated force exerted by vascular smooth muscle is not expressed in terms of unit mass of smooth muscle. If hypertrophy of media has occurred in the vessels of the hypertensive subject, the increase in COP on application of a constricting stimulus may be greater on this account and may be attributed to a greater reactivity when no difference in reactivity is present. However, this difficulty is inherent in all the techniques presently in use for comparing reactivities in vivo.

Investigators who have reported an increased vascular reactivity to angiotensin in essential hypertension have considered it to be a greater response of the same kind as that occurring in normal subjects. The experiments reported here indicate that there is a qualitatively different response of the muscle of at least some small vessels to this agent in the patients with essential hypertension. In the interpretation of this result no difficulties arise from possible hypertrophy of media in the vessels of the hypertensives nor from the ischemia involved in the measurement of COP. However, it is still possible that the response observed is quantitatively, rather than qualitatively, abnormal. Perhaps at higher concentrations of angiotensin in the bloodstream the arteriolar muscle will contract in normal subjects. But it did not do so with doses of 16 μg/kg per min of angiotensin. This dose is four times that given to the patients and no doubt smaller doses than the 4 μg/kg per min given to them would still increase the COP of their digital vessels. It seems reasonable to consider that the digital vessels of essential hypertensives have a qualitatively abnormal response to angiotensin concentrations in the blood within the physiological range.

Only the digital vessel response to angio-
tensin was tested, but if the abnormal response obtained in patients with essential hypertension was widespread among the tissues and organs of the body, it could be responsible for an increased blood pressure, when the concentration of angiotensin in the blood is lower than that which would produce hypertension in normal subjects. The fact that a 4 mg/kg per min dose of angiotensin produces a greater increase in blood pressure in the patients with essential hypertension than it does in the normal subjects is consistent with this suggestion. There are, however, other differences between the two groups of subjects that could possibly account for the greater increase in blood pressure in the patients. All but 1 of the normal subjects were young women while the patients were older and of both sexes. The presence of the hypertension in the patients may also enhance the pressor response to a vasoconstrictor agent because of the initially smaller caliber of resistance vessels. But it seems possible that the hypertension in patients with essential hypertension results from an abnormal contractor response of arteriolar muscle in the presence of a blood concentration of angiotensin not considered abnormally high. Further, the widespread presence of an abnormal contraction of arteriolar muscle to angiotensin would be expected to result in a severe, probably malignant, hypertension if a renal lesion with excessive release of renin and greatly increased production of angiotensin were superimposed.

Consider the implication of the responses demonstrated in digital vessels for the renal vessels, particularly those considered to harbor "stretch receptors" in their walls that act as baroreceptors to influence renin secretion (31-34). A fall in blood pressure in the region of the afferent arteriole is believed to stimulate increased secretion of renin by the juxtaglomerular apparatus. Presumably, decreased stretch of receptors results in increased secretion of renin. But angiotensin II is generally considered to be a vasoconstrictor in the kidney, causing vascular smooth muscle to contract with more force. If this is true and vessels containing the stretch receptors are contracted by angiotensin, it would constitute a positive feedback mechanism and the control of renin secretion would tend to be unstable unless other forces came into play. However, if in normal subjects the smooth muscle of the juxtaglomerular apparatus was relaxed as was demonstrated to occur in the normal finger vessels, a negative feedback mechanism would prevail and the control of renin secretion by stretch receptors would be more stable. In patients with essential hypertension, an abnormal contraction of vessels of the juxtaglomerular apparatus to angiotensin would, as noted, tend to cause an unstable situation and tend to produce a vicious circle—secretion of renin, production of angiotensin, contraction of vessels of juxtaglomerular apparatus leading to a further increase in renin secretion—unless offset by or checked by some other influence. Perhaps in the essential hypertensives, compensation for such an unstable mechanism was adequate in their youth, but eventually became incomplete, their abnormality resulting in hypertension.

Patients with chronic, benign hypertension believed caused by renal abnormalities appear to respond to angiotensin in a manner different from patients with essential hypertension. In fact, except for 1 patient, their COP after digital nerve block was changed hardly at all by the 4 mg/kg per min dose of angiotensin. Their response in terms of digital blood flow was not tested. In the 1 patient who was given a 8-mg/kg per min dose of angiotensin, the response to the greater dose was no different from that to the smaller. The type of renal anormality seemed to make little difference to the response, except that patient J.P. with hydronephrosis had an abnormally high COP after digital nerve block and a response to angiotensin similar to that seen in patients with essential hypertension. However, he may have had an underlying essential hypertension as well as hydronephrosis. In the rest of the patients with a renal hypertension, the lack of a definite increase or decrease in COP of digital vessels may be the result of saturation of recep-
tors by endogenous angiotensin. Kaplan and Silah (14) offered this as an explanation for the pressor response to their angiotensin infusion test being smaller in patients with renovascular than in those with essential hypertension. In 1 patient who had renovascular hypertension (D.V.), the vascular obstruction was corrected surgically. The angiotensin studies were carried out both before and after surgery (Fig. 1). Before surgery the COP after nerve block was low and was not changed by infusion of angiotensin. About 6 weeks after surgery when the subject was normotensive, the COP after nerve block was three times as high as that before surgery but still well within the normal range and it was decreased substantially by angiotensin infusion as in normal subjects.

The number of patients involved in this study is yet small. However, the results give promise of being able to distinguish between essential and other kinds of hypertension including renal, by a positive identification of essential hypertension rather than by the exclusion of other etiologies. Demonstration of an abnormally high COP after digital nerve block and a substantial increase in COP of digital vessels in response to administered angiotensin will likely identify it, although the responses in kinds of hypertension other than those listed in Table 1 will have to be investigated before a definite conclusion can be drawn.

Acknowledgments

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References


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