Conference on Neural Control of the Circulation in Hypertension

Led by Sibley W. Hoo blister, M.D.

D R. HOOBLER: I have a number of distinguished panelists here, but I am going to reserve the prerogative of offering Dr. Conway a little more than the time permitted earlier today so that he may present his work on differentiating vascular distensibility in human hypertension.

Dr. Conway: We have been interested for some time in the effect of aging of arteries in hypertension and have been trying to develop methods of testing for it. We have, as a start, developed a fairly simple test which depends upon the administration of amyl nitrite. Figure 1 shows the responses in 2 patients. The blood pressure was measured from the brachial artery while a whiff of amyl nitrite was administered and the arterial blood pressure fell. In the upper curve a fall in blood pressure was accompanied by a fall in the amplitude of the pulse pressure. We believe that this occurred because the patient has rigid arteries. For our purposes the trend of the change in pulse pressure, as the diastolic pressure falls, must of course be limited to the period preceding tachycardia because an increase in heart rate will itself reduce pulse pressure. Up until that time there are between 6 and 20 beats in which changes in pulse pressure are believed to reflect arterial elasticity. In another patient of similar age to the first (fig. 1, lower tracing), the diastolic pressure and the systolic pressure came down together. We believe that this patient has normally elastic arteries.

I do not want to elaborate on the test any further at present, and you may or may not think it very sound, but I should like to show you the results obtained by investigating patients with hypertension in this way. We have studied an unselected group of patients and separated them into 2 groups according to their responses to the test. Patients with arteriosclerotic hypertension were not included in this study and there were no clinical characteristics by which patients could be assigned to one group or the other. After separation into groups by the test, it was found that the average age of those with rigid arteries was higher than those who had normal arteries; the resting pulse pressure was also higher in those with rigid arteries. The greater pulse pressure occurred in spite of the fact that patients with rigid arteries had on an average a lower initial diastolic pressure.

Thinking about these patients in relation to what Pickering has said about the normal trend of blood pressure for age and sex, we have compared the level of blood pressure in the normal population with that found in our patients. The diastolic pressure of the patients with rigid vessels falls just at the upper limit of normal for their age. This would fit precisely with the concept that this type of hypertension is an exaggeration of the normal trend. The patients who we thought had normal arteries presented an entirely different picture. Except in a few cases the diastolic pressure was above normal limits for their age and sex, and in some of them above normal for any age. We believe, therefore, that there are probably 2 different processes at work in hypertension—one in which the blood pressure rises with age and another in which some process, perhaps a vasoconstrictor agent from the kidney, imposes hypertension on a normally elastic arterial system.

Having achieved a separation of patients
with hypertension into 2 groups according to the quality of their arteries, our next interest was to ascertain whether this would be of any practical importance in clinical practice. Preliminary investigations of the clinical status of these patients show 3 differences. A family history of arterial disease is found more commonly in those with rigid arteries than in those with normal arteries. This applies particularly to the siblings and not so much to the parents. The fall in blood pressure on bed rest is much greater in those with rigid arteries than in those with elastic arteries. Finally, the response to a test dose of one of the ganglion blockers, hexamethonium or pentolinium, gives a greater fall in pressure in the patients with rigid arteries. These clinical differences between the 2 groups also suggest that 2 different disease processes might be involved.

DR. HOOBLER: Given the same diastolic pressure, can you distinguish hypertensive patients by this test? Is this more useful than just the clinical recording of the diastolic and pulse pressure?

DR. CONWAY: I think this test can be used to assess the rigidity of arteries where it is not possible to do so on clinical grounds. Two patients of similar age and blood pressure may give different types of response to the test.

DR. HOOBLER: Now we will have to get to the business of the conference questions. The first question: "Is there any good evidence that experimental renal hypertension is initiated and maintained by a neural pathway?" I think this could be broken down wisely into 2 divisions, initiate and maintain, because there might be quite distinctly different views on these points. I will ask Dr. McCubbin if he would start the ball rolling.

DR. MCCUBBIN: There are the experiments done by Grimson in which he completely sympathectomized dogs except for the renal innervation. Then by cutting the buffer nerves, he increased sympathetic vasomotor
discharge to the kidney, resulting in hypertension. The hypertension was subsequently relieved by renal nerve section. That would seem evidence that neurogenic impulses can initiate hypertension.

Dr. Hoobler: That experiment of Grimson, I think, is a very crucial one. I personally think it would be an interesting one to see repeated. As far as I know it has never been tried again. Does someone here know of an attempt to repeat that specific type of experiment?

Dr. Wakerlin: I cannot say that we have repeated the experiment, but we have performed an experiment that might be of some value in this connection. We tested the effect of hog renin treatment in a number of buffer nerve hypertensive dogs to see if there might be a renal or renin factor in the maintenance of the hypertension. We found that with a course of hog renin therapy, the antirenin titer increased, there was a significant fall in the blood pressure of chronic buffer nerve hypertensive dogs. But even though the treatment was continued and the antirenin titer went higher, the blood pressure usually gradually increased over a period of 3 to 5 months to the original hypertensive level. This suggests that there is a renin factor involved but that it is not basic to the maintenance of the hypertension. We think that the increased vasomotor tone includes an effect on the renal circulation and produces some alteration in renal hemodynamics which sets off the renin angiotonin (hypertensin) system. Apparently we can block this temporarily with antirenin but other unknown factors compensate and the pressure goes back up.

Dr. Hoobler: That was a very helpful comment. Have you got something to say, Dr. Stamler?

Dr. Stamler: Kottke, Kubicek, and Laker showed that electric stimulation of the renal sympathetic nerves induced hypertension which persisted as long as the stimulus was maintained. Blood pressure returned to normotensive levels upon cessation of the stimulus. Grimson and his colleagues presented evidence that sustained renal hypertension could be produced by reflex renal vasoconstriction.

This problem of irreversible renal hypertension possibly resulting from prolonged or repeated periods of functional renal ischemia is an extremely important one for the theory of hypertension. The definitive demonstration of such a mechanism might serve to bridge the gap between the concept of a significant role for cerebral factors such as psychological stress and tension as opposed to renal factors in the genesis of hypertension. It would suggest a mechanism whereby disordered higher nervous activity might ultimately "trigger" renal hypertension. At the present stage of our knowledge, the foregoing must, of course, be recognized as speculative.

Dr. Langford: These experiments of Kubicek lasted to 3 months only, and were very difficult to do, but I think it is not correct to use this as a proof against the neurogenic origin of the hypertension. It takes longer than that to block off an artery, I believe.

Dr. Hoobler: Then perhaps Dr. Goldblatt would agree that one of the most important theories in the field of clinical hypertension might well be that some neurogenic stimulus does something to the kidney which thereafter perpetuates the hypertension. I think the critical experiment has not been done: maintaining a neurogenic tone to the kidney until the kidney itself can maintain the hypertension. One must perhaps devise better and more ingenious ways of creating a chronic neural stimulation of the kidney. I am sure that 3 months was the limit of anybody's endurance using the technic devised by Kottke and Kubicek.

Now I want some help concerning the role of the nervous system in chronic ren hypertensive. At least one experimental study in the literature confirms that if you pith a chronic ren hypertensive animal the blood pressure will come down to the same level as that of a normotensive animal. This occurs in spite of all the reports about sympathectomy and hexamethonium not lowering the blood pressure in renal hypertension.
might also say that in occasional cases in the hypertensive patient where there has been a transverse myelitis with paraplegia after surgery, malignant hypertension has reverted to a normal blood pressure for many years. These all involve an important question: What is there about pithing the cord that can cure chronic renal hypertension in animals?

Dr. Braun-Menéndez: If you hit a rabbit on the head and hit it hard you will get the same hypotensive effect. I think that pithing an animal is a very shocking experiment. When an animal is treated this way his cardiac output falls.

Dr. Hoobler: Dr. Dock actually measured and maintained atrial pressure in some of those experiments so that a fall in the cardiac output was probably not responsible. When he was in California, Dr. Bohr did some experiments of that sort and he found essentially the same thing. Would you care to discuss the objection of Dr. Braun-Menéndez to this experiment?

Dr. Bohr: We used Dr. Grollman's figure-8 hypertensive rats and pithing in any stage of hypertension caused a fall in pressure to the same level as that reached by a normal rat when pithed.8 We measured vena cava pressure by putting a polyethylene catheter into the jugular vein and with pithing the venous pressure did not fall. The fall in blood pressure, therefore, was not due to a decrease in cardiac output based on an impaired cardiac filling. The blood was there for the heart to pump out. The hypotensive level was caused by either a fall in peripheral resistance or an impaired pumping action of the heart. The shock state which concerns Dr. Braun-Menéndez is impossible to evaluate.

Dr. Goldblatt: Glenn and collaborators9, 10 pithed dogs and allowed the animals to recover, as much as an animal in that condition can, and then they constricted the main renal arteries. The blood pressure went up and stayed up so long as the animal was alive. That, in my opinion, is the crucial experiment.

Dr. Kezdi: May I say a word? If you depress the outflow by a very high sinus pressure, plus hexamethonium, there is still a very significantly different floor level in chronic renal hypertension as compared to normal dogs. This floor level cannot be due to sympathetic discharge.

Dr. Sapirostein: I think there is a hitherto overlooked fallacy in the Dock experiment. The experiment was designed to determine whether the difference between normotensive and hypertensive animals could be attributed to a humoral substance such as renin. From the fact that the blood pressure difference disappeared when the cord was destroyed, it was argued that there could be no humoral substance circulating and that the pressure difference must have been due to some activity transmitted through the cord or originating in the cord. But it has been shown,11, 12 that any circulatory catastrophe induces the release into the circulation of renin. Pithing represents such a catastrophe. Thus, the normal as well as the hypertensive animal is guaranteed to secrete renin into the circulation. Even if there were a difference with respect to renin before the pithing no such difference would be expected to exist after the pithing. Both the hypertensive and normal animals would be secreting renin maximally in the traumatic situation. The experimental procedure employed negates the whole purpose of the experiment.

Dr. Hoobler: I think we will have to call a halt here. I would judge the issue still a draw. I think it is well worth reconsidering this experiment despite what Dr. Braun-Menéndez says in objection. I would say that one of Dr. Dock's experiments included perfusion of renin in one series of animals before pithing. Then there was a difference of the blood pressure level in the 2 series of animals, as I recall.7

The next question is one for which I am responsible. The question is: 'What evidence exists in chronic essential hypertension or in human renal hypertension that neural factors are continuously active in maintaining the increased peripheral arterial resistance?' We have discussed the role of the nervous system in the maintenance of renal hypertension.
Now we come to human essential hypertension and I would like to make the point that the evidence from clinical investigation does not suggest that in the ordinary hypertensive human subject, in the recumbent position, there is any large element of continuously prevailing neurogenic arteriolar tone. Human essential hypertension, recumbent position, neurogenic arteriolar tone, are the important terms. I do not deny that neural mechanisms could raise the blood pressure or initiate a hypertensive reflex, or that changes in posture could produce neurogenic arteriolar constriction; I am simply asking whether, in the recumbent hypertensive human subject, the neurogenic tone to the arterioles is elevated. The arguments against it very briefly are that if you get a hypertensive patient into the recumbent state and do cardiac output studies on him, you find that when you give him hexamethonium or some other ganglionic blocking agent all of the decrease in pressure can be accounted for by a decrease in cardiac output. The total peripheral resistance stays constant. This has been shown by a number of investigators; most recently by Crump ton’s group at Wisconsin. Another bit of experimental evidence is that when you go from one major vascular bed to another you find that the reduction in pressure is more or less paralleled by a reduction in blood flow. The 2 cancel each other out so that peripheral arterial resistance in any vascular bed you measure before and after a ganglion block is the same.

Thus, I argued there was no sympathetic tone until Dr. Beck’s experiments showed that a blood pressure decrease per se should result in an increase in peripheral resistance if there were only passive changes in the arterioles in response to the blood pressure decrease. I think Dr. Beck made a good point that when we do lower the pressure, say 20 per cent, we should have some increase in total peripheral resistance if the arteriolar vascular bed is normally elastic. Would you say, Dr. Beck, that there is some inhibition of neurogenic arteriolar tone when a drop in pressure in these vascular beds is not accompanied by an increase in peripheral resistance? This is a fundamental point. Many of us have talked about peripheral resistance as being directly proportional to the ratio of pressure to flow. Dr. Beck brings up the point that even this ratio varies with perfusion pressure so that with everything else kept constant one must expect pressure reduction to result automatically in an increase in peripheral resistance.

**Dr. Beck:** Well first of all, I don’t want to take credit for finding that an artery can stretch. This is a very old observation. I wanted to reaffirm this in connection with the use of ganglionic blocking agents. Now, as far as the human being is concerned, obviously I cannot make any hard and fast statement. The only way I can answer the question is by analogy. I think I can show in the experimental animal essentially what you can show in the human subject. In the experimental animal I believe that if the total peripheral resistance does not rise with a fall in blood pressure, then there must have been a decrease in vasomotor tone, unless the vessel has lost its elasticity. To apply this to the human assumes that in the hypertensive individual the vessel has a certain degree of elasticity. Dr. Conway has just shown us some interesting results pertaining to elasticity so I would like to pass the buck right now over to him and see what comment he has on this point.

**Dr. Conway:** Well, last things first. I was really entirely concerned in the clinical work with rigidity of the very large arteries so we have no evidence on the arterioles. But I am very disturbed by Dr. Beck’s work because it does run contrary to what I have thought; not that there is any harm in that. Evidence from Sweden suggests that if you lower the blood pressure in a limb, for instance, the peripheral resistance goes down, not up. This also applies to human arteries, normotensive ones at any rate. Reactive hyperemia is produced partly by an accumulation of metabolites and partly by a reduction in transmural pressure across the arterial wall.

**Dr. Beck:** I think there is no question about the reactive hyperemia. I will say briefly that I tried to get around it as best I could by employing a shunt; that is, in the interval...
when I was not making flow-pressure determinations, I utilized the shunt to minimize reactive hyperemia.

**Dr. McCubbin:** Dr. Dustan pointed out earlier today that we need more and better tools and I think this is probably another instance. Dr. Freis has recently done some experiments that I think are very helpful in explaining this lack of fall in peripheral resistance in dogs and man after giving a ganglionic blocking agent. He substituted a pump for the heart in dogs. By maintaining a constant cardiac output, he showed that hexamethonium then did lower peripheral resistance. It is becoming more and more apparent that venomotor pathways are much more sensitive to the ganglionic blocking agents than are pathways to arterioles. With this sort of selective blockade there is a decrease in venous return, lowering of cardiac output and fall in pressure. But since sympathetic efferent pathways to arterioles are not blocked completely, the fall in pressure causes the buffer reflexes to increase sympathetic discharge and thus raise peripheral resistance. This simply means that the blocking agents are not good tools for determining whether there is or is not neurogenic vasoconstriction in hypertension.

**Dr. Kezdi:** If we maintained intracarotid pressure by a pump independently of the systemic circulation, as I described in my paper, both cardiac output as measured by the Fick principle and total peripheral resistance decreased following tetraethylammonium chloride (TEAC) injection in 3 dogs. The regulatory mechanism is apparently still effective in increasing peripheral resistance because of the proportionally great decrease in cardiac output following ganglionic blockade. This is not the case when the carotid sinus is excluded from the systemic circulation and the receptors do not perceive the decrease of the blood pressure during TEAC block.

**Dr. Stamler:** One could go on endlessly with this particular discussion. Dr. Hoobler and I have had a stimulating exchange of correspondence along these lines. I would like to agree with Dr. McCubbin that the tools currently being utilized are inadequate to yield a definite answer. Every clinician knows that if a hypertensive or normotensive patient is given a large enough dose of blocking agent, his blood pressure will fall to shock levels. What does that prove about the role of neurogenic factors in determining peripheral resistance in hypertension? As to the pitfalls in animal research on this type of problem, an old set of experiments in our laboratory illustrates them very well. Many years ago we showed that renal hypertensive dogs respond to the tissue injury represented by abscess formation with a sustained period of normotension. In studies on the mechanism of this response, Taylor and Page showed it was unrelated to the associated leukocytosis and fever. Subsequently it was demonstrated that this "pyrogenic" and "antipressor" response was associated with a sustained increase in renal blood flow, although it could not be definitely concluded that this was the mechanism of the restoration of normotension.

Following these studies, experiments were carried out on the hemodynamics of these dogs. It was found that the unanesthetized dog exhibited a normal resting cardiac output and blood volume, along with renal hyperemia, during the period of abscess-induced restoration of normotension. The animal anesthetized with pentobarbital manifested an increased cardiac output and marked renal ischemia. This led us to repeat Homer Smith's warning concerning the fallacies of renal function studies in anesthetized animals, a caution worth reiterating with regard to Dr. Beck's experimental efforts. The further decrease in total peripheral resistance under pentobarbital anesthesia indicated that the abscess operated to lower blood pressure in these hypertensive dogs without completely paralyzing autonomic vasomotor activity. Finally, further experiments with renin, angiotensin, epinephrine, pitressin, and tetraethylammonium chloride indicated that vascular reactivity was not seriously impaired in unanesthetized renal hypertensive dogs during the period of abscess-induced normotension. The mechanism of this response, therefore, remains an unresolved problem challenging in-
vestigators of experimental hypertension. May I re-emphasize, on the basis of these experiences, the conviction that pharmacologic agents and the responses to them may be inadequate tools to elucidate the status of the peripheral resistance and its neurohumoral regulation in hypertension, especially in the anesthetized animal.

DR. BECK: I would like to say in response to Dr. Stamler’s comment on anesthesia that I feel our results are still preliminary and that much more information is required before we can accept the evidence, let alone make an extension to the human.

DR. HOOBLER: There is time for only one more comment. I think Dr. Kirkendall has it.

DR. KIRKENDALL: I believe there is evidence to support the belief that there is a decrease in arteriolar resistance and perhaps tone in hypertensive patients treated acutely with ganglionic blocking agents. To deny this would be to deny the role of neural impulses in the maintenance of any portion of the arterial pressure in hypertension, and I do not believe the literature supports this.

Most of the studies of the cerebral circulation after the acute injection of ganglionic blocking agents demonstrate a decrease in peripheral resistance which one would expect if neurogenic arteriolar tone were initially increased. Finnerty and Freis have demonstrated a great increase in skin blood flow after ganglionic blocking agents, and Freis and his group have demonstrated a small increase in muscle blood flow and decrease in hepatic vascular resistance after the injection of hexamethonium. Ford, Moyer, and Spurr have shown with chronic administration of hexamethonium in hypertensive patients that renal blood flow increased and renal vascular resistance decreased. The acute administration of hexamethonium has been shown occasionally to be followed by an increase in renal blood flow by Ullman and Diengott and by our group at Iowa.

Although changes in arteriolar tone in most of the vascular circuits measured have not been great, a decrease in tone must have occurred for normal or decreased vascular resistance to have been recorded in these areas.

My explanation for this decrease in tone after the administration of ganglionic blocking agents is that neural impulses important in its maintenance have been interrupted. This belief is supported by the early abrupt change in arteriolar tone after ganglionic blocking agents. Another explanation for this phenomenon is that autonomous local regulatory mechanisms may cause small arterial vessels to dilate when pressure or flow fall. Finally the drug used may have a direct effect on blood vessels to cause the decrease in tone. With this evidence for an effect on the arterial circuit of hypertensive man by ganglionic agents, I cannot believe that the entire effect of these agents on the blood pressure is due to relaxation in venous channels and decrease in cardiac output.

DR. HOOBLER: My reply to your comments must be brief. First, I would exclude arterioles to skin and to hands and feet which are under continuous and marked neurogenic arteriolar stimulation, but do not represent a major part of the total resistance and are so specialized a circulation as to prevent generalizations concerning other areas. These unquestionably dilate after ganglionic blockade. Those in which peripheral resistance falls do so only to a very minor extent and after quite some delay, which suggests an autonomous readjustment rather than a reduction in neural tone, as you have suggested. Finally, many reports, some previously mentioned in this conference, are in contradiction to the studies you quote in that they describe no change in hepatic, renal, or total peripheral resistance. I suspect that if there is neurogenic arteriolar tone to these areas in the recumbent human, the previously mentioned autonomous and carotid reflex readjustments obscure the conclusions.
to be drawn from measurements of the human circulation before and after ganglionic blockade.

Now our time is short and we have one more question to deal with: ‘‘Can a lasting hypertension hemodynamically comparable to that seen in the human be produced by a neural mechanism? If so, is peripheral resistance increased and continuously maintained by neural stimuli?’’ I can think immediately of 2 people here in the room who have experience to contribute. Dr. McCubbin?

**Dr. McCubbin:** Drs. Taylor and Page attempted to tie off the entire circulation to the head of dogs sometimes in combination with diathermic stimulation of the vasomotor centers and chronic hypertension occasionally did develop. But whether this was hemodynamically comparable to essential hypertension was not known.

**Dr. Hoobler:** Dr. Wakerlin, would you comment very briefly on this question?

**Dr. Wakerlin:** We proceeded from the general premise that when he narrowed the renal artery Dr. Goldblatt was able to produce hypertension in dogs. The general consensus now is that there is some alteration in renal hemodynamics, the nature of which is not settled even after this conference. Since there is evidence that the brain may be involved in the pathogenesis of hypertension we asked ourselves: Why not try similarly to alter cerebral hemodynamics? All such efforts up to this time had involved ligation of arteries in the neck area. This we felt was comparable to some of the old experiments in the pre-Goldblatt days when investigators tied off the renal pedicles and during the period of survival observed mild increases in blood pressure. Instead of tying off vessels of the neck and getting low-grade hypertension, only moderately persistent at best, in a minority of dogs, we decided to constrict the arteries to the brain in the dog, which might do the same thing to the cerebral circulation that Dr. Goldblatt’s renal artery clamp did to the renal circulation. We devised a plastic clamp (fig. 2) which reduced the volume pulse of the sinus and of the internal and external carotid arteries to one-third normal size. This we have found to be optimum for the production of hypertension in the case of constriction of the renal artery. When we so constricted the internal and external carotid (and occipital) arteries bilaterally, we got a high level hypertension in a high percentage of dogs, some of which have had hypertension now for more than four years (fig. 3). So far as we can tell from limited studies, this hypertension, except for the clamps on the carotid sinus area, resembles essential hypertension. We do not think the carotid sinus mechanism is primarily involved in the pathogenesis for we have applied constricting Goldblatt monkey clamps on the internal and external carotid (and occipital) arteries bilaterally above the sinus, with the carotid sinus nerve intact, and still have obtained the hypertension. So far as the carotid sinus nerve mech-

---

**Fig. 3.** Chronic neural hypertension from bilateral constriction of the carotid sinus area in the dog using a clamp of the type shown in figure 2. (Reprinted from Circulation Research 5: 685, 1957.)
anism is concerned, it ought to have operated against the hypertension in the latter experiment. We are in the process of studying the pathogenesis of this hypertension, particularly since superficially it appears to resemble essential hypertension. The hypertension has been confirmed in 2 other laboratories. We would like to interest some of you in trying to produce this hypertension. We would be pleased to furnish clamps and advice.

Dr. Hoobler: I think this report is a very fitting close to an interesting 2 days. In the first day of this conference, we learned of the isolation and synthesis of the humoral substance released by clamping the renal artery to produce hypertension, a technic first reported to the Cleveland Academy of Medicine 25 years ago by Dr. Goldblatt. In this very last report hypertension has been produced by his method, applied to the carotid artery to produce this new form of "neurogenic" hypertension. On behalf of my colleagues at the University of Michigan I wish to express thanks to all of you for your participation in making this conference a success.

Summary

Dr. Conway reviewed his test for differentiating hypertensive patients into those with and without altered vascular elasticity. The discussion then turned to the role of the nervous system in hypertension. Experiments concerning the role of neural influences in initiating permanent hypertension were reviewed by Drs. McCubbin, Wakerlin, Langford, and Stamler. The Dock experiment of pithing hypertensive animals was then considered. His results seemed to indicate the importance of neural influences in maintaining experimental renal hypertension, but it was agreed that it was not justifiable to conclude from this work that renal hypertension was maintained by elevated neural tone (Drs. Braun-Menéndez, Bohr, Goldblatt, Kezdi, and Sapirstein). The conferences then examined the question of increased neurogenic arteriolar tone in human essential hypertension. The lack of evidence for increased neural tone, based on the failure of greater dilation following ganglionic blockade in the hypertensive subject was supported by Dr. Hoobler and opposed by Dr. Kirkendall. Most agreed that inferences from changes in peripheral resistance after blockade were fraught with error, due to the intrinsic elasticity of the arterioles (Dr. Beck), the predominance of venomotor blockade (Dr. McCubbin), the "break through" of buffer reflexes (Drs. McCubbin, Kezdi, Stamler), and the effects of varying types of anesthesia in animal experiments (Dr. Stamler). Finally, in reply to a question concerning ways to produce chronic, sustained, experimental, neurogenic hypertension, Dr. McCubbin mentioned the experiments of Taylor and Page, and Dr. Wakerlin concluded with a description of a technic for producing in dogs chronic neurogenic hypertension which had persisted in excess of 4 years. This consisted in applying a constricting plastic clamp to the carotid sinus area bilaterally, so as to reduce the volume pulse of the arterial branches of the sinus by two-thirds.

REFERENCES


34. Little, J. M.: Personal communication, 1957.

35. Goldblatt, H.: Experimental renal ischemia. Presented before the Experimental Section Cleveland Academy of Medicine, Cleveland, Ohio, November 11, 1932.
Conference on Neural Control of the Circulation in Hypertension
SIBLEY W. HOOBLER

Circulation. 1958;17:815-824
doi: 10.1161/01.CIR.17.4.815
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1958 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/17/4/815

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/