pregnancy, abdominal binders, etc. Elevated venous pressure after heart failure seems, in most instances, to cause back pressure in the renal vein. A great deal of clinical study will be required, however, to define the effects of increased renal vein pressure as a potential cause of hypertension and to explain the adverse effects on renal function that may be secondary to increased renal pressure.

Summary

Animal experiments were designed to determine whether systemic hypertension and renal ischemia, known to develop secondary to decreased blood flow to the kidney, would result from increased renal venous pressure or decreased flow from the kidney. The experiments in which blood flow was artificially decreased have been described and the results given.

The effects of venous constriction were observed and the animals destroyed so that histologic examination of renal tissue could be made. Fibrotic development, tubular degeneration, ischemic glomeruli, and thickening of the walls of the arterioles attested to the development of chronic hypertension.

Acknowledgment

The authors would like to thank Dr. E. J. Poth for his help and suggestions.

References

1. Beckman Model 160 Physiological Gas Analyser Instruction Manual, Palo Alto, Beckman Instruments Inc., 1962.
2. Denison, A. B., Jr., M. P. Spencer and H. D. Green: A Square-wave Electromagnetic Flow Meter for Application to Intact Blood Vessels. Circulation Res., 3:1, 1955.
3. Dennard, E. J., Jr.: The Square-wave Electromagnetic Flow-Meter: Recent Advances and Use. Presented at Southeast District 4 and South Central District 13 Joint Meeting, American Institute of Electrical Engineers, New Orleans, April 5–7, 1961.
4. Derrick, J. R. and K. R. T. Tyson: The Association of Aberrant Renal Arteries and Systemic Hypertension. Surgery, 48:907, 1960.
5. Goldblatt, H.: Studies on Experimental Hypertension: the Pathogenesis of Experimental Hypertension Due to Renal Ischemia. Ann. Int. Med., 11:69, 1937.
6. Houssay, B. A. and E. Braun-Menendez: Role of Renin in Experimental Hypertension. Brit. Med. J., 2:179, 1942.
7. Page, I. H. and O. M. Helmer: A Crystalline Pressor Substance (Angiotensin) Resulting from the Reaction Between Renin and Renin-activator. J. Exper. Med., 71:29, 1940.
8. Spencer, M. P. and A. B. Denison, Jr.: Square Wave Electro-Magnetic Flow Meter for Surgical and Experimental Application. Methods in M. Res., 8:231, 1960.

Discussion

Dr. Francis D. Moore (Boston): When I saw this paper on the program I did not know just what shape it was going to take! I think it raises two extremely interesting points.

First, as pointed out in the studies of Ganong and others, the hypertension associated with renal arterial stenosis appears to be due to hypotension in the proximal arteriole of the glomerulus, permitting it to collapse down and literally squeeze out the renin from the juxtaglomerular cells. Therefore this work is a very interesting and conflicting experiment; hypertension has resulted from a situation in which intravascular pressure in the kidney is *increased* rather than decreased.

Secondly, it has been shown by many people, particularly by Dr. Harrison, and Dr. Crocker of our service, that in renal artery stenosis there is hypertrophy of the juxtaglomerular cells. Angiotensin not only affects blood pressure but also produces an immediate response in the adrenal cortex with an outpouring of aldosterone, affecting the excretion of sodium, and therefore the ECF volume.

Therefore, I would like to ask if venous occlusion also produces hypertrophy of the juxtaglomerular cells, since, if it does, we have to make a new theory!

Dr. Florindo A. Simeone (Cleveland): I want to comment on three observations which

need to be explained on the basis of the findings reported in this paper.

The first has been referred to by Dr. Moore; namely, it is difficult to accept the concept of the juxtaglomerular apparatus as another baroreceptor on the basis that venous occlusion produces hypertension.

The second is that many years ago Dr. Goldblatt, in the early days of studies of this problem, pointed out that one way of preventing the hypertension which follows partial occlusion of the renal artery in the dog was to ligate the renal vein; and he had various explanations for that observation.

And, finally, we have recently had a patient in whom we relieved portal hypertension by doing a portorenal anastomosis, ligating the left renal vein just beyond the ovarian, and anastomosing the remaining renal vein to the portal. This was done six months ago, and the patient did not develop hypertension, either acutely or chronically.

DR. C. BARBER MUELLER (Syracuse, New York): Just to make this matter more confusing, we have recently been trying to determine what influence the medulla of the kidney has had on the renal concentrating mechanism. We have developed a series of dogs in which we opened the kidney and scooped out the renal medulla of one kidney. In addition to making observations relative to urinary concentrating ability, we found that this series of dogs develop arterial hypertension. We have no explanation at the present time for this hypertension but it may be that it takes more than just the juxtaglomerular apparatus to produce high blood pressure.

DR. JOHN R. DERRICK (closing): I wish there were some of the questions that I could answer that have been given to us.

I think it is important to point out that we see patients who have rather severe constriction of the renal artery and have no hypertension.

We have worked closely with a group doing electron microscopy studies of the juxtaglomerular apparatus, and to add to the confusion, we found that in some experimental that did not develop hypertension the granules that have been considered the cause of liberating the pressure substances that may result in the changes that cause hypertension.

I would like to summarize by pointing out that mechanical and circulatory difficulties of the kidney in many instances cannot be separated from the adrenal itself, because of the intimate nature of the circulation—the artery and venous circulation—to the two organs. This adds greatly to the complexity of this very fascinating problem.

Another point which appears important is to prove whether or not renin is really the cause of the hypertension that one occasionally may observe in patients, or in experimental animals, that have increased renal venous pressure and increased intraparenchymal pressure. One would need renin studies from the renal venous system.