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THE NEW ROLE OF SURGERY IN HYPERTENSION

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1. Introduction.

1. Scope.

This paper is to deal with the surgical treatment of hypertension. It is not our intent to dwell upon a full treatment of the syndrome, but rather to investigate surgical obligations in the disease and to concern ourselves with etiology in so far as treatment may be directed toward it.

Our interest has always been attracted by hypertension because of its prevalence and because of the apparent inadequacy of treatment. We are to concern ourselves with malignant hypertension and the so called "essential or benign" hypertension, hoping to prove the obsolescence of the latter as a diagnostic term. Of the hypertension associated with senile sclerotic vascular change and which is part and parcel of the generalized sclerosis and aging of the tissues, we will not take issue, hoping it will be acceptable to the reader to divorce this senile type from the large group of "essential hypertension." To do so we must establish that the underlying pathology differs: this we hope to do.

2. Divisions of Hypertension.

It is in accordance with most beliefs to state that increased blood pressure is due to a more or less uniform narrowing of the small arteries and arterioles (48). There are three ways
this constriction may occur.

1. Morphologic change in the vessels themselves.

This is the pathology of the increased arterial tension found in the older age groups where the vascular walls are participating in a sclerotic change of tissue which we ascribe to "old age." This sclerosis is a function of this tissue of the vessel wall, it is not dependent in its initiation upon neurogenic factors or upon stimulation by chemical agents in the circulating blood.

This is the type we were speaking of earlier and with which we will not be concerned further. The pathogenesis is apparent; the management is a medical problem, not surgical.

2. Decreased arterial caliber due to vasoconstriction of nervous origin.

This is a less clear cut group, because of the manifold methods in which this nervous stimulation may be initiated. This group will be discussed at length subsequently.

3. Stimulation of the vessel walls by chemical agents in the circulating blood.

This latter is a new and as yet incompletely established concept. It was born through animal experimentation and has grown with clinical support. To establish that it is the sole operating factor in the greater proportion of malignant and essential hypertensions and can be attacked directly by surgery is the purpose of this paper.
3. Hypotheses.

The thesis takes its life from three hypotheses.

1. Hypertension is a result of generalized arterial and arteriolar constriction, a response to renin, the chemical substance produced by an ischemic kidney, this constriction ultimately producing intimal change and sclerosis.

2. The treatment of malignant hypertension, and of the lesser degrees of increased blood pressure which are not a part of the primary vascular sclerosis of senility, is a surgical problem; this surgery is to be curative, and in no sense a palliative procedure.

3. A diagnosis of essential, idiopathic or benign hypertension is no longer tenable; it is an admission of an incomplete search for the underlying pathology.

II. History of the Surgical Treatment of Hypertension.

White has estimated that 70,000 persons die annually as a result of hypertensive heart disease, and an equal number succumb to the other consequences of hypertension (54). Of these 140,000 persons, 10% have the malignant form (51).

The first surgical attack for the relief of one of these unfortunates is generally credited to Pieri, who, following the suggestion of Pende successfully carried out resection of the left splanchnic nerves in a patient suffering from high blood pressure (16).

However, the idea of splanchnic resection as a treatment
for hypertension was conceived in 1923 by Danielopolu. In this same year Druning suggested the same surgical procedure (16). The following year, 1924, before the Congress of Internal Medicine at Milan, Pende proposed a surgical treatment for arterial hypertension which consisted of resecting the left splanchnic nerves; this Pieri executed.

In 1932, Louis Durante of Genoa resected the large and small left splanchnic nerves, and his cases were reported by Santucci whose post operative observations indicated a decreased blood pressure. Unfortunately he also admits that these were not true cases of hypertension, but clarifies himself no further (16). The Mayo Clinic did its first splanchnic resection in October of this same year.

In April, 1924, Rountree and Adson (51), denervated the left femoral artery in a patient suffering from sclerosis, terminal nephritis, and uremia. By this procedure, a Leriche-Handley operation, they hoped to;

1. Lower systemic blood pressure.
2. Provide an area of diminished resistance so that if a vascular accident occurred it would happen in a relatively harmless region and not in a retinal or cerebral area.

There was no effect from their operation, but they believed the case to be too far advanced. C. H. Mayo, (43), is apparently the first surgeon to have operated for relief of hypertension with evident result. His was an extraordinary case, that
of a retro-peritoneal malignant blastoma. Perhaps it will be re-called that this case was described by Dr. Reginald Fitz of Boston at the 1940 meeting of the Omaha Midwest Clinical Society. In this instance the pathogenesis of the hypertension was presumably due to pressure of the tumor upon the left suprarenal gland, aggravated further by mechanical irritation of the abdominal sympathetics.

So with this instance of a surgical reduction of blood pressure from 300 mm., systolic, during a paroxysm to 120 mm., we hopefully scrutinize present day claims.

III. Secondary Hypertension Due to Known Causes.

It is our intent to consider secondary hypertension, which contributes 15% to all hypertension, only to clarify our picture of primary or "essential" hypertension towards known causes, the most common of which are:

1. Coarctation of the aorta.
2. Hyperthyroidism.
3. Arteriosclerosis of the senile type defined previously.
5. Aortic heart disease.
7. Tumors of the suprarenal gland.

The diagnosis of the latter is perhaps the most difficult as it is often to be confused with a primary hypertension. V. L. Evans, (25), has described a syndrome characteristic of a supra-
renal tumor, differentiating it clinically from malignant hypertension. This is a brownish red discoloration of the skin over the face and arms, blueness and coldness of the feet with the attacks, and a heart rate that is markedly increased. Laboratory examination reveals an increased blood sugar, and a slight albuminuria. The condition is characterized by being paroxysmal in nature, as was Mayo's retroperitoneal tumor (43).

IV. Primary Hypertension.

Keith, as quoted by Wetherell, (58), divides primary hypertension into three categories;

1. Benign hypertension—characterized by a slow development, beginning at 40 to 55 years of age and continuing for 10 to 15 years.

2. Early malignant hypertension—characterized by a faster development, beginning at 18 to 50 years of age, and continuing for 3 to 4 years.

3. Malignant hypertension—onset at 18 to 50 years of age, with rapid termination within 18 months with systolic pressure ranging from 250 - 300 mm., and diastolics from 100 - 160 mm.

This makes a workable clinical classification of the disease, is important for prognosis, but also intimates that the pathogenesis differs. It is presented here as it is alluded to by the authors who support the theory of sympathetic preponderance as the cause of hypertension.

We will now approach the first of the two main theories
of etiology of hypertension with the demands upon surgery in each instance. Our discussion hereafter will be limited to primary hypertension in the three phases which have been described above.

1. Surgery Directed Towards the Relief of Sympathetic Preponderance.

a. Etiological concepts.

Adson (3) believes that "essential" hypertension is probably due to a fault in the neurogenic - endocrine vascular mechanism since no other apparent etiological factors can be demonstrated - that surgical measures will probably come to play an important role in the treatment of essential hypertension. The inadequacy of medical treatment has further justified surgical investigation and attempts at surgical treatment." In the same year, 1936, F.S. Wetherell, (58), admits that "surgery at least today can attack only the distal factors." So at least one of these workers admits that the surgery is of a palliative nature, and is not a direct attack at the etiology.

They all recognized that hypertension was due to a more or less uniform narrowing in the caliber of the arterioles and arteries throughout the body. This was thought to be neurogenic in type, but few writers concerned themselves with the mode of origin of the increased sympathetic tone.

Craig and Brown, (17), ascribed the pathogenesis of hypertension to an hyper-excitability of the medullary vasomotor center, supporting the contention by the fact that in early
hypertension there are no demonstrable pathologic abnormalities in

the endocrines, arterioles, or sympathetic nerves. In the preceding

year, (12), they called attention to an abnormal response to

the stimulus of cold before blood pressure was in an hypertensive

range; these patients were described as early potential hyperten-

sives. In early and advanced hypertension the responses of blood

pressure to stimulation was often three times the normal. They

believed this response was due to a hypersensitive vasomotor center.

This response has been noted by Heuer, (37), and Flothow, (26),

and accredited to a pre-hypertensive state, but Mulholland, (46),

states "it has been shown that a cold pressor test with a marked

response to cold is not peculiar to essential hypertension; it is

found in normal individuals."

G. W. Crile, (19), believes that the etiology lies in a

pathological physiology of the adrenal medulla-sympathetic complex,

proposing that hypertension is a universal Raynaud's disease. He

laid much importance on the adrenal factor as evidenced by his

surgical attack and his extensive inquiry into the ratio of adrenal
to body size in many African animals, (18). The strongest

supporter of adrenal dyscrasia is J. L. DeCourcy, (22), (20), who

postulates as the cause of hypertension a hyperadrenalinemia

resulting from hyperplasia of the medullary tissue of the adrenal

glands. He admits considerable evidence of a neurogenic factor

in the disease. Oisgard and Sharpe, (9), point out that this

medullary hyperplasia is usually not evident macroscopically or
microscopically. Criticism directed at DeCourcy because of his failure to demonstrate his "hyperepinephria" is met with the explanation that the increase in adrenalin is not detectable because of its great dilution.

b. Surgical Methods with Support.

The surgery of the supporters of the neurogenic endocrine theories are directed towards two objectives; in one to interrupt the flow of sympathetic impulses to the splanchnic area; in the other to partially remove the adrenal glands by subtotal adrenalectomy. The various surgical attacks will be considered not as to technique primarily, but as to claims made of their effectiveness.

1. Rhizotomy - this procedure consists of extensive laminectomy from the 5th Thoracic to the 1st Lumbar vertebrae with section of the corresponding anterior nerve roots.

The purpose of the operation is to remove the vasoconstrictors of the splanchnic area by interrupting the efferent fibers in the anterior roots, resulting in a splanchnic vasodilation and the formation of a blood pool to relieve tension. The results of this surgery vary markedly with different operators, the poor results being defended by the arguments that cases must be carefully selected. Flothow,(26), states that with removal of visceral nerves, results in all clinics show a 50% chance of alleviating the hypertension and a 50% chance of failure. Shook, (54), rather discouragingly remarks that "it is possible that the
temporary rest as occasioned by a surgical operation is the chief factor in producing the temporary fall in pressure."

The selection of cases for the various methods of sympathectomy is based upon results obtained in the following examinations, (26):

1. Careful heart examination.
2. Tests of kidney function.
3. Hourly P.P. determinations for 48 hours.
4. Effect of sodium amytal and nembutal on blood pressure when used in high doses.
5. Effect of sodium nitrite on blood pressure.
6. The cold pressor test.
7. Intravenous pentothal test.

If a candidate shows a lowering of blood pressure with sleep or with sedation and an adequately functioning kidney, he may be selected for operation. It is curious to note that kidney damage with reduction of renal function excludes a patient from surgery. Although this "kidney damage" is rather all inclusive term, we know that certain forms of renal damage have been reported in many instances to be the cause of hypertension. The very man who elaborated this method of case selection, Plothow, (26), concludes with the statement that "it is impossible to tell from a case what the outcome of the surgery will be."

The major splanchnic nerves arise from fibers in the anterior roots of the fifth to tenth thoracic nerves; the minor
splanchnics from the ninth to tenth or tenth to twelveth thoracic. These join the coeliac plexus and are distributed to the vessels of the stomach, liver, pancreas, intestine, and kidney. Bradford (11), has shown that vasoconstrictor fibers are present from the sixth thoracic to the second lumbar roots, but only the tenth thoracic to the third lumbar contained constrictors in any abundance. He also concluded that the splanchnics contained both constrictors and dilators.

Adson and Brown, (1), hoped by the section of these vasoconstrictors (67 to 21) to:

1. Prepare a vascular reservoir for emergency.
2. Avoid sudden rise in blood pressure.
3. Sympathectomize sufficient arterioles and arteries to modify arterial response.
4. Sympathectomize the adrenal glands.
5. Remove effects of intra abdominal tension, by paralyzing abdominal muscles.

Their report of a case treated in this manner showed a blood pressure drop to 140/80 from 200/10, present one year after operation with removal of all subjective complaints. Bountree and Adson, (51), sectioned the ganglia, rami, and branches from the second to fourth lumbar in a case of malignant hypertension, with immediate relief from symptoms, but with intermittent return of these. The blood pressure was lowered "about 40 points" for a time but returned to its former level of 200 mm., systolic.
However, this patient's response to sedation and nitrites was better since operation. Brown, et al, (14), in a series of six patients followed postoperatively for four years has shown a lowering of blood pressure, but, "the ultimate value of this operation on the course and progress of serious types (of hypertension) is unknown." The untoward effects of surgery were hyperesthesias of skin of thighs, urinary retention in one case, and abdominal weakness. Hountree and Adson, (51), report transient sciatica and erythema of the legs as complications.

Page and Heuer, (48), treated a case with a flexible but high blood pressure, which could be dropped from 190 to 160, systolic, with rest and sodium thiocyanate. A laminectomy with section of each pair of nerves from 6T to 2L was performed with relief of symptoms and lowering of blood pressure to near normal limits. Since the upper limits of normal pressure is generally taken to be 150 mm., systolic, it is a bit hard to evaluate the success in this particular case when the authors are not more specific.

In a staff meeting at the Mayo Clinic in July, 1938, Allen and Aison, (4), attempted to answer some of the objections to extensive sympathectomy for essential hypertension. To the objection that sympathectomy does not modify hypertension produced by Goldblatt clamp in animals, (32), three answers were given. The first was that proof is lacking that essential hypertension in man is the counterpart of that in animals due to
renal ischemia. This paper hopes to refute this answer. The second is that occlusion in man is a vasospasm not a clamp, if we do agree that hypertension in man and animals is the same. This does not take into account that renal ischemia can be produced by circumstances other than vasospasm. The third is that sympathectomy does lower blood pressure in carefully selected cases; therefore it cannot be invalidated because it does not work in dogs.

To the objection that the reduction of blood pressure by sympathectomy is reported to be only transient, Adson and Allen agree, but state that it needs further study. They deplore the criticism that has arisen against this operation because its results are uncertain and palliative at best. "This is the best that can be offered in surgery of carcinoma of the breast or stomach;" Ascroft, (5), makes reply to this by stating that "we can't compare sympathectomy to the surgery of carcinoma of the breast or stomach because this is established and the surgery in high blood pressure is experimental, and it is wrong to sacrifice the majority for the benefit which might result."

2. Sub-diaphragmatic Splanchnic Nerve Resection.

This method of approach to the splanchnics differs from the supradiaphragmatic approach largely in the surgical technique involved, but has the advantage of offering direct inspection of the adrenal glands where the rare tumor causing hypertension might be discovered. The splanchnic resection is not
deemed sufficient for some men who also resect a portion of the adrenals for good measure.

The splanchnics supply vasoconstrictor fibers to the vessels of the abdominal viscera in the following manner; the greater splanchnics send fibers to the abdominal blood vessels; the lesser splanchnics supply the adrenals largely with the addition of a few fibers from the upper two lumbar; the third or lowest splanchnic supply the renal vessels, (56).

Section of these nerves, therefore, accomplished a three fold purpose, (9), in that it:--

1. Removes the vasoconstriction of the splanchnic bed which comprises more than 75% of the total vascular field of the body.
2. Denervates the suprarenals.
3. Improves renal function by establishing a more normal flow of blood through the kidneys and an exchange at a lower arteriolar tension.

In light of the experimental work done on animals it is quite probable that the benefits of splanchnic resection are largely derived by the improvement of renal circulation to an ischemic kidney from whatever cause, and not by its effects of suprarenal denervation and relief of splanchnic vasoconstriction in general. If the formation of a splanchnic "pool" and denervation of the adrenals were a factor, one should expect that this could be supported experimentally, which it cannot.
Goldblatt, et al, (29), found no lowering of blood pressure in his experimental animals after splanchnic resection. It is logical to assume, therefore, that the lowering did not occur because the circulation to the kidneys of his animals was still embarrassed by a mechanical clamp. The splanchnic vasodilatation and suprarenal denervation evidently occurred in his experiments.

That the relief obtained from this procedure of splanchnic section is though more than the production of a large area of vasodilatation is suggested by the evidence that the removal of the left splanchnic is more effective in lowering blood pressure than a similar procedure on the right, and equally effective as the removal of the splanchnics bilaterally (13). There is no evidence to show decussation of the splanchnics (11).

Unlike laminectomy and rhizotomy there are no post-operative symptoms relating to any of the abdominal viscera nor any untoward symptoms resulting from the operation (12).

Results reported again are variable, with no improvement on those obtained by rhizotomy. Smithwick, (56), summarizes as follows:

1. Marked lowering of blood pressure in 30% of cases.

2. Moderate lowering of blood pressure in 20% of cases.

3. No change in 50%.

He fails to state whether or not his cases with marked
lowering of blood pressure approached the limits of normal. Adson, et al, (2), state that "evidence --- also shows that a limited number of patients fail to respond to the same procedure, while others have shown a clinical improvement without lowering of blood pressure, some having a return of symptoms and pre-operative blood pressure levels." Brown, (13), has also noted that the objective improvement in his cases was not as good as the subjective. In Craig's, (16), patients the blood pressure has become stabilized rather than lowered.

Shipley and Aycock, (55), in their series, have found that interruption of the autonomic nerve supply via the splanchnics will cause a return of blood pressure approximately to normal levels in a small percentage of cases, will arrest the progress of the disease for longer or shorter periods in a somewhat larger group, will alleviate symptoms without any effect on blood pressure, but in a larger group it does not lengthen life or improve symptoms.

Though the results of this procedure of subdiaphragmatic section of the splanchnics are no better than those of laminectomy with rhizotomy, it appears more practical than the latter in that the patient is not subjected to an extensive and perilous laminectomy, nor is he as exposed to untoward complications. In either he has no better than a 50-50 chance of relief, and almost no chance of a cure.


This procedure differs from the subdiaphragmatic route
only in technique. Its aims and results are much the same. In it the greater, lesser, and minor splanchnics are resected, and in addition the four or five lower thoracic ganglia with their rami may be removed. This supplement adds much to the technical difficulties and may not add to the result, (38). Heuer,(37), has had some formidable complications with it in a streptococcic meningitis and a bilateral hemiplegia. He has, however, made a great contribution in his work on preoperative and postoperative observations, the lack of which, he declares, has made previous publications mean nothing. Preoperative patients were subjected to a 30 day observation period during which various tests on blood pressure response were made. Extensive renal studies were done, excepting excretory and retrograde pyelograms, a study we hope to establish to be most important.

Even with this careful selection of candidates for operation, those showing blood pressure reductions with sleep, sedation and evipal anesthesia, his results were no better than those previously described.


Perhaps the strongest advocate of this procedure is J. L. DeCourcy, (20), who believed that the cause of hypertension lies in an hyperplasia of the medullary tissue of the adrenal glands from a constant sympathetic stimulation, resulting in secretion of excessive amounts of adrenalin in the blood stream.
The overloading of the circulation with this hormone brings about the changes in the walls of the arterial blood vessels with a resultant rise in pressure to overcome these changes.

The surgical attack indicated, if this is truly the pathogenesis of hypertension, is partial adrenalectomy accompanied by denervation of the adrenals. DeCourey employs a two stage operation, (20), in which a subtotal adrenalectomy is performed, removing both cortex and medulla, and leaving a third to a quarter of the glandular tissue. With this procedure he claims to accomplish a lowering of blood pressure from 70-90 mm., systolic, and from 40-50 mm., diastolic. Patients with generalized arteriosclerosis or with arteriosclerotic eye ground changes are not accepted as candidates for the procedure. DeCourey also tends to exclude cases showing renal damage.

Crile is another advocate of the theory that adrenal hormone dyscrasia is the factor in the production of hypertension. He has done extensive studies on the gland, especially in studying the ratio of adrenal weight to total body weight in a large group of African animals, (18). He refers to the medulla of the adrenal as the local brain of the sympathetic system, the function of which is disturbed in hypertension; there results a pathological physiology of the adrenal medulla - sympathetic complex.
The generalized vasoconstriction in hypertension is compared to a universal Raynaud's disease, a sympathetic response in its entirety.

In a review of 822 operations which he performed on the adrenal sympathetic system, (19), he summarizes as follows;

1. In early cases the blood pressure falls to a maintained lower level or to normal.
2. In cases in which the hypertension has been associated with other diseases which are due to pathological physiology of the adrenal-sympathetic system good results are secured; e.g., hyperthyroidism and neurocirculatory asthenia.
3. There results an improvement in the eye grounds and renal function, as well as in the well being of the patient.
4. During the operation the blood pressure in cases of malignant hypertension is reduced to normal,(18).

He declares that the failure to lower the blood pressure in some cases is defensible in that the thoracic sympathetics are still intact.

C. Criticism.

It is not the position of a neophyte to criticize the aforementioned procedures, but it is our desire to air some of the objections made to them by prominent investigators.

No objection is made to the proposition that these pro-
cedures are palliative in nature, but opposition arises when they claimed to be a direct attack at the source of the disease, as DeCourcy and Crile maintain. E.O. Atchley, (16), probably speaks the truth when he writes of suprarenal denervation, "I am not proposing to write or advocate that the procedure I am about to describe is a final and complete solution to the great problem of malignant hypertension."

It is rather difficult to evaluate the results obtained in these operations directed toward the suprarenal glands and splanchnic area. Crile, for instance, does not define normal pressure standard, and has figures too complex to evaluate. Page and Foeuer, (37), (47), and (48), have had fair results with rhizotomy but have the complications of a relaxed anterior abdominal wall and submit the patient to the hazards of an extensive laminectomy. Max Peet, as stated by Ascroft, (5), has a 3.8% mortality but reports a 15% recovery and a reduction of blood pressure in 50% of 315 cases. Davis and Barber, also quoted by Ascroft, state that in all of their carefully controlled cases the high blood pressure returned. Adson, and associates, (1), (2), and (3), claim a functional cure in 83% of their cases, and are the loudest in the defense of their procedure which is the only one that is at all encouraging in results obtainable.

It is quite feasible that all the positive results were in a manner quite unrecognized by the operator. In all of the procedures the vasoconstrictor supply to the kidneys was
partially or totally interrupted with the production of a vasodilatation and increased blood supply to a possibly ischemic organ. The importance of renal ischemia in the genesis of hypertension will be dealt with at length in the following section.

a. Impetus.

Although the experimental work on renal hypertension is now being carried on by many researchers, the impetus to this study was given by the work and publications of Harry Goldblatt and his associates of Cleveland. His methods and results are common knowledge among the members of the medical profession today, but it seems fitting to this paper to more closely inspect his work, to correlate, if possible, the surgical methods already discussed, and to find a basis for future clinical study.

His initial publication in 1934, (28), was a study of the production of persistent elevation of blood pressure by means of renal ischemia which he produced in experimental animals by applying a special clamp to the renal artery; with this clamp it was possible to regulate the severity of the constriction. His working hypothesis was that ischemia limited to the kidneys may be the initial condition in the pathogenesis of hypertension that is associated with nephrosclerosis. He used an unselected group of dogs for the following experiments, the results of which will be given in some detail.

Experiment 1. - almost complete constriction of the
renal arteries, done singly, but with a short interval between operations.

1. All of the animals died within four days from uremia.

2. The blood pressure doubled in one animal.

3. The urea clearance fell to zero.

4. There was a rapid ascent of NPN and blood pressure.

Experiment 2. - severe constriction of both renal arteries with a short interval between operations.

1. The urea clearance dropped to 50%.

2. Blood creatinine and NPN remained at normal levels.

3. Blood pressure was raised 60 mm. of Hg.

Experiment 3. - moderate constriction of both renal arteries which was subsequently increased.

1. The blood pressure was raised above the highest levels of the initial control period.

2. There was no significant change in urea clearance.

3. Blood urea, creatinine, and NPN remained within the control limits.

Experiment 4. - bilateral moderate renal ischemia which was later increased -- still later nephrectomy.

1. After nephrectomy there occurred a rise in blood pressure which was not sustained.
2. Again the blood creatinine, NPN and urea remained within the limits of the control period.

3. PSP excretion was not affected.

Experiment 5. - effect on blood pressure of severe ischemia in other organs, followed by renal ischemia.

1. Clamps on the lienal and femoral arteries produced no increase in pressure.

2. Clamps then placed on the renal arteries resulted in an increased blood pressure, again without deviation of the blood urea, NPN, and creatinine from normal standards.

Experiment 6. - renal ischemia following excision of the right adrenal, section of the left splanchnics, and denervation of the left adrenal with destruction of its medulla.

1. Blood pressure after the above procedures remained normal.

2. With a following renal artery constriction the blood pressure raised with a tendency to lower, but not to the control levels.

3. The blood NPN, urea, and creatinine varied within the normal control limits.

His final experiment of this series was a compression of the carotid loop with which blood pressure readings were made to exclude the carotid reflex as a factor in the production of this
experimental hypertension. Such compression produced no blood pressure rise.

In the first three experiments, Goldblatt, produced a picture similar to the clinical forms of mild, early malignant, and malignant hypertension as classified by Keith. The pathology varied with the degree of ischemia, from a moderate parenchymal degeneration and thickening of Bowman's capsule and membrane to a severe diffuse medullary parenchymal degeneration with renal atrophy and capsular thickening. The only laboratory test of renal function which showed a change was that of urea clearance which dropped 50% with severe bilateral constriction.

With Gross and Hanzal, Goldblatt next published in 1937 (29), with a study of the effect of section of the splanchnic nerves on renal hypertension. The sympathectomy subsequently referred to includes the splanchnics and the four lower thoracic sympathetic rami before constriction of the renal arteries.

Experiment 1. - sympathectomy before constriction of the renal arteries.

1. After the sympathectomy, clamping of the renal arteries produced a blood pressure rise in the usual manner.

2. There was no disturbance of renal function with minimal constriction.

Experiment 2. - sympathectomy at variable periods after clamping of the renal arteries, comparable to
the curative methods in humans.

1. Pressure may fluctuate after section to below previous level, but will return to the hypertensive state.

2. There was no significant permanent lowering in any of these studies.

The statement has been previously made that it is possible that the results the sympathectomists have obtained in humans are due to an unrecognized relief of renal ischemia by the production of renal vasodilitation. It would be illogical to criticize their attack because of the negative results obtained in this experiment, for if sympathectomy were to aid the hypertension by an increase in the arterial and arteriolar caliber of the renal circulation, we could not expect much in the dogs with the clamp still in place. However, the criticism that their surgical attack is not directed at the cause still remains. The first conclusion in Experiment 1 of this series rather definitely divorces hypertension from any possible neurogenic element.

Wood and Cash, quoted by Goldblatt,(30), have shown that constriction of the renal arteries in dogs causes also a rise in the diastolic pressure, a finding he has confirmed. In an effort to use an animal more closely related to man, Goldblatt,(30) reproduced his findings in Macaque Monkeys where normals of 128/86 were elevated to 200-300/130-196 mm. of Hg. With Keyes, (38), he found that retinopathy in his animals similitated that in hyperten-
sive humans.

In a further study, Goldblatt, (31), tried to approximate the conditions present in clinical arteriolar sclerosis. His work received strong support from Dominique, quoted in (31), who investigated the effect of the injection of uranium salts in the production of increased blood pressure. Only one animal showed an increased arterial tension, and this animal had an increased arteriolar sclerosis in the kidneys.

From this same study Goldblatt was able to conclude that ischemia of the kidney is important as the pathologic change and that the kidney is the primary site or origin of this experimental type of hypertension. These conclusions are acceptable for if one kidney is constricted and later removed, the blood pressure returns to normal.

An extensive postmortem tissue study was carried out in the animals after the production of the malignant phase of hypertension, (33). The clinical manifestations closely approximated those seen in humans suffering from a similar type of disease. Uremia and anuria were the terminal features with blindness due to intraocular hemorrhage, and rectal bleeding.

Gross examination of tissue showed petechial hemorrhages in the gastro-intestinal tract, pancreas, and bladder. No macroscopic evidence of hemorrhage was ever found in the kidneys, muscles, skin, lungs, liver or thyroid. Microscopic examination revealed arteriolar degenerative disease of varying severity,
present in the pancreas, gall bladder, urinary bladder, gastrointestinal tract, and spleen. Skeletal muscle changes were not common, but when present consisted of a hyalinization of the intima and a thickening of the media of the arteriolar walls. The pulmonary artery and renal arterioles did not show evidence of necrosis.

These arteriolar degenerative lesions consisted of hyalin deposits immediately beneath the endothelial lining of the intima, internal to the elastic lamina. In the smallest arterioles where endothelium composed the entire intima, the hyalin was then a sub-intimal deposit. This deposit was eccentric, or concentric with partial or complete occlusion of the lumen. All stages of hyalinization and necrosis or normalcy were present in the same arterioles, as in human disease. These necrotizing lesions of the arterioles require an azotemia with increase in pressure, for his essential hypertensive dogs without renal impairment did not show arteriolar degenerative change.

This pathological picture differs from man only in that he shows kidney arteriolar necrosis. This, however, gives us a lead as it means that a human's constriction is farther down the renal arteriolar tree. In the dog, of course, the constriction is extra-renal so he should show no renal arteriolar disease.

Goldblatt's later studies are directed toward other sources which might be held as the point of origin of his experimental hypertension. He described the work of Page and
Sweet, (31), on the effects of extirpation of the pituitary gland on arterial blood pressure of dogs with experimental hypertension. There is much contradiction of the work at present, and more experimental work should be done on various portions of the pituitary body. That the adrenal medulla is not a factor has been proven previously, (28), but some further interesting information was gained, (31). In the absence of both adrenals and with substitution therapy in the form of sodium bicarbonate, sodium citrate, or Eschatin, the dogs developed a significant but slight rise in blood pressure with the production of renal ischemia; without substitution therapy, the hypertension was not maintained. Apparently the adrenal cortical hormone is necessary for the production of the renal ischemia type of hypertension. The hormone is not a vasopressor in itself and only a small portion of the cortex need remain to aid in the production of hypertension; therefore, partial adrenalectomy does not seem to be a rational method of treatment. To remove enough cortex to combat the hypertension would be to produce an adrenal cortical insufficiency and a disease for more serious and discomforting than the original hypertension.

With Kahn and Hanzal, Goldblatt, (34), as his ninth study on experimental hypertension, investigated the effect of constriction of the abdominal aorta above and below the site of origin of the renal arteries; the blood pressure rose in about the same interval as after bilateral constriction of the arteries. There was an initial drop in the femorals with a delayed rise to levels
above the normal, although the aorta is constricted. With varying degrees of aortic constriction, either the benign or malignant phases of hypertensive disease could be produced. Release of the clamp permitted the blood pressure to return to normal.

When the constriction was done below the site of origin of the arteries, no rise in blood pressure occurred, so that the increased pressure was not on the basis of simple mechanical obstruction of the aorta.

It might be permissible to make an interesting digression at this point. Following this work an aortic constriction, Goldblatt, (34), made the suggestion that human eclampsia may be due to pressure of the gravid uterus on the renal arteries or aorta, sufficient enough to produce renal ischemia, since the disease occurs in the last trimester of pregnancy at a time when the uterus is greatly enlarged.

b. The Humoral Theory of Hypertension.

From his rather exhaustive experiments on hypertension, Goldblatt, (31), postulated a modus operandi for his experimental hypertension. He had to consider two possibilities: a nervous element was shown to be of no consequence, for a thorough sympathetic sectioning did not prevent the production of increased blood pressure, (29). Page and Heuer, (47), had hoped to show that hypertension may lie in nervous impulses from the kidneys, even those which appear normal at the onset. They performed a bilateral renal denervation on a patient with essential hyper-
tension uncomplicated by renal involvement. The patient showed no improvement, and they were forced to conclude that "nervous impulses from the kidneys do not share in the genesis of hypertension."

The logical conclusion then was that a humoral mechanism was at play through an "effective substance" liberated from an ischemic kidney. This conclusion is furthered by the finding that there is no increase in blood pressure when both the renal artery and vein is occluded, by a clamp, (31). Goldblatt considered three ways in which his "effective substance" might act.

1. The effective substance might act synergistically with a known pressor hormone from an endocrine organ, such as the hypophysis or adrenal.

2. It may sensitize the contractile elements of the arterioles to the action of a pressor substance.

3. The effective substance might also produce its effect by neutralizing or reducing the amount of hypothetical depressor substance circulating in the blood.

The fact that there is this pressor substance extant in the kidneys was revealed by Prinzmetal and Freedman, but it has also been extracted from other organs, (31). P.B. Ascroft, (5), has published a rather exhaustive survey of the experimental work on renal pressor substance.

Forty years ago Tigerstedt and Bergmann prepared a
pressor substance from normal kidneys which they called renin. Recently Pickering and Landis have extracted it from normal kidneys in considerable quantities and have demonstrated its extraordinarily prolonged and powerful pressor action. Landis makes the important point that renin, unlike adrenalin, pituitrin, and other substances that raise blood pressure, does not decrease the peripheral blood flow. This is a most significant observation for neither is the peripheral blood flow decreased in human hypertension. Prinzmetal and Freedman claim to have demonstrated abnormal amounts of renin in the ischemic kidneys of hypertensive dogs.

Houssay and his co-workers confirm the impression that the hypertension of renal ischemia is due to a pressor substance formed in the affected kidney. This they do by having grafted the ischemic kidneys from hypertensive dogs onto the main neck vessels of normal dogs with the production of a constant and prolonged rise in blood pressure. This investigator suggests that this renal pressor substance is eliminated or destroyed by the normal kidney. He gives as support the fact that:

1. When the artery of one kidney is constricted, the blood pressure rises, then falls to normal.
2. If the healthy kidney is then removed, there is a rapid rise in blood pressure.
3. A grafted, ischemic kidney has a greater effect in the production of hypertension on nephrectomized
dogs than on those with normal kidneys.

A recent editorial in the Annals of Internal Medicine, (59), brings the investigation of the humoral factors in hypertension to date. The nature of renin, the pressor substance, in normal renal tissue has been extensively studied by Page who has now forsaken the sympaticotonic theory of hypertension, the corrective surgery of which he has written much about. With Corcoran he has found that slow infusion of renin into experimental animals causes a constriction of the renal vessels, particularly marked in the efferent glomerular capillaries, resembling the condition found in human hypertension. If this infusion is performed with purified renin in some form of isotonic salt solution, no vasoconstriction occurs. Renin evidently, therefore, requires a compliment or activator present in normal serum; this has been termed "renin - activator." The potent product of the interaction of renin and "renin - activator" has been termed angiotonin and has been isolated in relatively pure crystalline form. A single injection of this substance causes an immediate rise in blood pressure, more prolonged than that produced by adrenalin; repeated injections of renin produces a diminution of response, until after about six injections, the animal becomes refractory. This fact along with other evidence suggests that renin injections stimulate the production of some neutralizing antipressor substance. Peculiarly enough the kidney may also be the source of this substance for bilateral nephrectomy greatly
increases the response to renin or angiotensin.

Grollman, et al, (59), report the extraction of such an inhibitor substance from renal tissue; this extract does not lower normal blood pressures in animals, but does so in hypertensive rats and dogs and in a few human cases. It reduces the response in normal animals to the injection of renin.

Much more study is needed before the final answer is obtained. It is possible that renin and its inhibitor are produced committantly by a healthy kidney in balance or with an excess of one or the other to meet the demands of the moment. Renal ischemia from whatever cause apparently either favors renin production or suppresses the liberation of its antagonist. The remaining kidney evidently meets the demands by excretion of renin or secretion of additional inhibitor substance to a certain threshold above which renin predominates and hypertension occurs.

Mulholland, (46), gives a simple and concise summary of the methods that have been used to produce arterial tension, all by decreasing the blood flow through the kidney.

1. Excision of varying amounts of renal tissue.
2. Constriction of the renal veins - this is not complete obliteration.
3. Production of interstitial fibrosis of the kidney by exposure to x-ray.
4. Legation of one or both ureters.
5. Partial ligation of the renal arteries.
6. Production of cellophane perinephritis.

7. Compression of the renal arteries by adjustable clamps.

It has been shown that hypertension produced by this last method is not relieved by denervation of the kidney pedicle, excision of the splanchnic nerves, complete sympatheticotomy, nor by complete destruction of the spinal cord.

c. Clinical Corollary and Reports of Case Studies.

We desire now to examine the various types of human pathology which might lead to the interference with renal circulation and which would, if the humoral theory of hypertension is to be substantiated, produce clinical hypertension. We hope to convince the reader that diligent and careful inquiry into a case of "essential" hypertension will often reveal an obscure renal lesion which is the cause of the increased blood pressure. If this is possible, the direction of a surgical attack is evident -- toward the correction of the lesion, and not toward palliation.

Again Mulholland, (46), has given us a classification. He regards the causes of circulatory interference as being either intrinsic or extrinsic. The intrinsic causes result from renal infection arising within the kidney with the impingement upon the finer circulation by fibrosis and contraction. Extrinsic causes interfere with the blood flow in the renal artery. His classification is as follows:

1. Intrinsic causes - from infection within the kidney.
sideration of problems involved, nor must we misinterpret anatomic changes which are the result and not the cause of hypertension.

The first recognition clinically, of a possible interrelation between kidney disease and hypertension was in 1920, and resulted from some experimental work carried out by Ritter and Baehr, (50). They made a postmortem study of arterial blood supply in congenital polycystic kidney disease in which they injected the arteries with a mixture of gelatin and barium sulfate and examined the kidney by x-ray. Their studies revealed that the interlobar and interlobular arteries come to lie in the cyst walls; in almost every one of the larger cysts there are numerous arteries of various sizes which lie directly beneath the lining epithelium, certainly becoming vulnerable to slight pressures. All of the cases which they studied were adults who died in azotemia after a period of arterial hypertension. The authors describe the clinical course, "except for attacks of pain and hematuria (rupture of a vessel into a cyst) the entire sequence of events is indistinguishable from that which is observed in patients suffering from malignant hypertension."

The antithesis of this acumen is in a publication as late as 1938, four years after Goldblatt published his original work. Guild, et al, (35), presented two cases as arteriosclerosis in childhood. The first had a full blown pyelonephritis with abscess and a blood pressure in the two hundreds; they describe the pyelo-
lonephritis as being secondary to the hypertension. The second case did not go to autopsy, but showed pus and colon bacilli in the urine; this also was probably blamed on the hypertension. In their accompanying review of twenty cases of arteriosclerosis in childhood they gave full autopsy reports in fifty percent which fortunately gave a description of the kidneys. In all, the blood pressure was above 165, systolic, the greatest being 240, systolic, and 180 diastolic, and in all there was definite evidence of gross renal pathology. This varied from the markedly contracted kidney of pyelonephritis and hydro-ureter to congenital hypoplasia. Never were these renal lesions considered as a possible cause for the hypertension or arteriosclerosis.

Schwarz, (53), presented two very similar cases to those above in even younger children. In the single case that went to autopsy, a congenital hypoplasia of the right kidney was found. This author did not concern himself either, in whether the renal pathology was primary or secondary to the arteriosclerotic changes.

Goetz, (27), gives support to Goldblatt's work both with a parallel to his method of production and with argumentation in support of the humoral theory of vasoconstriction. The disease, periarteritis nodosa, produces an endothelial proliferation in the arterial intima; this disease is accompanied by hypertension only if the intimal lesions narrow the vessels of the kidneys, and further, if this be present so is an angiospastic retinitis. A rise in blood pressure occurs in amyloid disease if deposits in
the renal arteries lead to decreased blood flow through the kidneys.

"To answer the question as to whether this general vaso-spasm is of humoral or nervous origin, Goetz points to the fact that retinal vessels are spastic, which he contends could occur only with a humoral mechanism.

A good introduction to a study of urological lesions masked as hypertension is afforded by Ritch, (49), who published a report of such a study. His interest was aroused when he inadvertently discovered an urologic lesion in a case which had been treated five years for "essential" hypertension. This finding stimulated a search for urinary tract lesions in other cases of so-called, "essential hypertension." He urges that all hypertensive cases, where a definite cause for the condition is not assignable, be studied by pyelography. Any hesitance to this procedure because of expense or annoyance should be obviated by the realization of our helplessness in treating them medically.

His first case carried a pressure of 240/192 for five years. An ureteral stricture was discovered and dilated regularly with a drop of the systolic pressure to 170 or 180 mm. If the interval between dilations was too long, the pressure again rose to the 200's. The second case had a pressure of 170 for several years; the repair of a nephroptosis and ureteral kink dropped the blood pressure to 140. Another case was treated medically for twelve years for a heightened blood pressure. Suddenly he became
acutely ill with an infected hydro-nephrosis and a systolic pressure of 268 mm. A nephrectomy was performed and the pressure dropped to 156 even before the patient was dismissed from the hospital.

Much has recently been published about the role of chronic pyelonephritis in the production of hypertension. Longcope and Winhenwerder, (40), studied the means of recognition of this disease which often goes undiscovered. The examination of the urine may show only moderate amounts of albumin with leucocytes and no casts. This finding is often either neglected entirely or regarded as a secondary finding due to the hypertension which is usually present. The anatomical abnormality of kidney contraction in chronic pyelonephritis is usually discovered by pyelography. A careful history will often suggest the disease; the usual story is that of poor health over many years with repeated febrile illnesses ascribed to different causes, or there might have been indefinite attacks of lumbar pain, during which there may have been fever and cloudy urine. The report of an objective examination during childhood which has shown albumin or pus is highly significant.

Hypertension not infrequently is associated with pyelonephritis before there is any appreciable diminution in renal function. Butler, (15), had fifteen patients who had chronic pyelonephritis and hypertension over a period of years before there was appreciable reduction of renal function. This is reasonable since we are told that 30% of our total nephrons will support
Inadequate elimination. In another group of fifteen children between 3 and 11 years, shown at necropsy to have pyelonephritis, Butler found pressures from 140/110 -- 250/110 in eight; the pressure in the remaining seven had not been taken.

Longcope, (4), studied bilateral pyelonephritis as regards its origin and association with hypertension. He repeated Butler's findings that symptoms of renal insufficiency may not appear for ten or fifteen years after the infection is initiated; in fact the progress of the disease itself is for many years symptomless. In nine of his cases which came to autopsy, the hypertension was not associated with pronounced or extensive arteriosclerosis; five showed evidence of arteriosclerosis, and in two of these it was minimal in degree and extent. He found the lesion, chronic bilateral nephritis, to be most common in young women between the ages of fifteen and thirty.

A typical case has been reported by D.W. McIntyre, (44), and it will be repeated here in brief. Interestingly enough, Goldblatt did the pathological examination of the extirpated kidney.

Case Report.

A white male, age, 34, was admitted on December 29, 1936, for examination because of a rejection for employment due to an elevated blood pressure. The family history and past history were negative. Physical examination was negative.
Laboratory examination revealed a negative serology with a trace of albumin in the morning urine, but none at any other time. One specimen showed 3 RBC/HPF.

A diagnosis was made of early malignant arterial disease and the patient dismissed.

He returned May, 1938, complaining of pain in the left flank, fever, and vomiting of a week's duration. There is a history of three such attacks since his last hospital admission.

The physical examination, with ophthalmoscopic, were negative except for a blood pressure of 168/94.

Urine examination showed 15 pus cells HPF with an occasional renal epithelial cell. With ureteral catheterization material was obtained from the left kidney which produced staphylococcus albus from culture.

A left retrograde pyelogram revealed a duplication of the pelvis with fusion of the ureters with marked hydronephrosis.

Nephrectomy was performed, and at operation it was found that there was a single blood supply to both kidneys from a left renal artery.

A pathological examination by Goldblatt reported microscopic evidence of chronic pyelonephritis, but no evidence of arteriolar disease. The renal artery showed arteriosclerotic changes with narrowing of the lumen.
The patient made an uneventful recovery; his blood pressure on dismissal was 132/84 and a year later was 134/78.

Barney and Juby, (8), reviewed all the cases of renal disease at the Massachusetts General Hospital since 1866. They selected all cases with a diagnosis of pyonephrosis, pyelonephritis, hydronephrosis, or pyelitis, totaling 305, of which 171 were females, and 134 males. Seventy six cases, 25%, had pressures above 140 mm. Of the 305 cases, 224 had undergone nephrectomy, and 67% of these had an average drop in blood pressure of 33 mm. systolic.

One may call attention to the fact that of the 305 cases of this series only 25% showed evidence of hypertension; this, however, in no way nullifies the possibility of an association between renal disease and hypertension, for we know that in pyelonephritis alone the blood pressure rise may not appear for ten or fifteen years after the inception of the renal lesion. Furthermore, one cannot say that all renal disease produces hypertension, for the latter is dependent on the degree of renal ischemia, and it is conceivable that lesions may be present without affecting renal blood flow.

Schroeder and Steele, (52), examined seventy two patients with hypertension for abnormalities of the urinary tract. In fifty of these, some abnormality of the kidneys or ureters was demonstrable. The study was made by intravenous injections of Diodrast, followed at five and fifteen minute intervals by a flat plate of the
abdomen. They summarize as follows:

1. Lesions suggesting obstruction to renal blood flow - 36 patients.
   1. Bilateral hydronephrosis - 11
   2. Unilateral hydronephrosis - 20
      1. Moderate in 11.
   3. Associated disease found in 4 of above.
      1. Contraction and distortion of the renal pelvis.
      3. Total absence of function.
      4. Replacement of kidney by calcified cysts.

2. Renal calculi - seen in pelves or calyces of 5 patients.
   1. In one patient an associated hydronephrosis.
   2. In one patient a total absence of function in the other kidney.

   1. In 4 patients no excretion of Diodrast in one kidney on repeated injections. Suggestive of some disturbance of function.
   2. In 2 there was ptosis of one kidney.
   3. In one there were double ureters and double kidneys.
4. In one there was a marked distortion of the calcyces, probably a result of old inflammation.

5. One patient showed a persistent sharp angulation of the ureter.

The average age for this series was 29 years, while that of Laher, (42), the next to be presented, was about 61 years. However, his pathology occurs in the older age groups; since it does "one might argue that they (high blood pressure and urologic lesions) are concomitant but unrelated vicissitudes of senility, but their appearance in younger age groups disproves this," (42).

Laher and Wosika, (42), examined 101 patients who had "no special factors of occupation, habits, or nationalities of importance." Their diagnostic criteria for hypertensive vascular disease, often obscure or undefined in other articles, are those of the American Heart Association as follows; "Persistent hypertension associated with enlargement of the heart."

<table>
<thead>
<tr>
<th>Urologic Pathology</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prostatic obstruction with upper G.U. complications.</td>
<td>31</td>
</tr>
<tr>
<td>Chronic pyelonephritis with complications.</td>
<td>27</td>
</tr>
<tr>
<td>Renal stone and complications.</td>
<td>18</td>
</tr>
<tr>
<td>Hydroureteritis with complications.</td>
<td>7</td>
</tr>
<tr>
<td>Diverticulum of the bladder with complications.</td>
<td>5</td>
</tr>
</tbody>
</table>
Urethral stricture in the male.  4
Ca. of the bladder with ureteral obstruction.  2
Renal tumors.  2
Congenital cystic kidneys.  2
Bladder stone.  1
Congenital absence of kidney.  1
Vesico-vaginal fistula.  1

101

In these two series alone, we have listed some fifteen or more urinary tract lesions associated with hypertension. True, the rationale of the part of some in the pathogenesis of hypertension is obscure and debatable, but there remains enough association to entitle a hypertensive patient to complete urological investigation and surgical remedy of such a lesion when found.

S. S. Blackman, (10), has investigated one interesting extrarenal cause for hypertension, arteriosclerosis and partial obstruction of the main renal arteries. In 50 cases exhibiting hypertension and in an equal number of control cases, a detailed study was made of the condition and caliber of the renal arteries. In 28 cases arterial and arteriolar sclerosis was found, of which 24 died in uremia. In 22 cases of the hypertensive group, clinical and microscopic evidence of nephritis was lacking; in fact none of the generally recognized causes of hypertension were
found at autopsy. Some of the cases showed a sclerosis without hypertension, but the possibility of supernumerary vessels to the kidney must be considered here as the hypertension is not a measure of the arterial sclerosis of the kidney out of its relative ischemia. His investigations revealed that —

1. In a majority of cases there was partial occlusion of the lumen of one or both renal arteries.
2. Occlusion is caused by the ordinary yellowish white arteriosclerotic plaques.
3. The plaques were largest and most numerous in a short segment of the renal artery near the aorta.
4. The sclerosis of the main renal arteries was part of a generalized arteriosclerosis in every case.
5. 86% of cases showed a moderate to severe sclerosis of the renal arteries.

A surgical attack against hypertension due to renal arterial and arteriolar sclerosis has been suggested, (46),(5); this is a nephro-omentumopexy in an effort to establish additional collateral circulation to the affected kidney. The procedure affords relief to laboratory animals with experimental hypertension, (5), but hasn't been investigated fully in man.

A very unusual type of hypertensive renal disease has been described by Leiter, (39); it is acute hypertension due to thrombo-arteritis obliterans of the small renal arteries proven at autopsy. The clinical diagnosis before death was acute non-
nephritic hypertension in view of the normal renal findings. This case is extraordinary to be sure, but well illustrates the manifold pathologies which may produce renal ischemia.

V. Conclusion.

We have attempted in this paper to stress the importance of surgery in the treatment of hypertension, not as a palliative procedure to relieve symptoms and possibly prolong life, but as a method of cure. It is realized that all hypertensives do not fall into the renal group, but the publications reviewed herein clearly show that many so called "essential" hypertensives do.

Page's recent work on renin anti-pressor substance,(59), may ultimately give us a means of treating hypertension medically with the benefits that have long been hoped for; hypertension will then become an unfeared, conquered disease. However, until this therapy is realized, surgery must bear the entire load in the treatment of renal hypertension.
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