Third Edition

BASIC SURGERY

edited by

Hiram C. Polk, Jr., M.D.
Professor and Chairman
Department of Surgery
University of Louisville
School of Medicine
Louisville, Kentucky

H. Harlan Stone, M.D.
Formerly, Professor of Surgery
Emory University
Atlanta, Georgia

Bernard Gardner, M.D.
Professor and Director
Department of Surgery
Hackensack Medical Center
Hackensack, New Jersey

APPLETON-CENTURY-CROFTS/Norwalk, Connecticut
1987
One of six Americans is hypertensive, but approximately half of those affected are unaware of the disease. Surgeons are interested in hypertension because it is an important risk factor in many patients being considered for surgical treatment of diseases unrelated to hypertension and because hypertension is caused by surgically treatable lesions in a significant number of patients. Detection of the latter group is obviously important and continues to be a difficult problem.

CLINICAL PRESENTATION

Hypertension does not lend itself to the case presentation approach because it does not produce a characteristic symptom complex and, in fact, many hypertensive patients are absolutely asymptomatic. Dull occipital headache is commonly described and, because this is often relieved by control of the hypertension, cause and effect are assumed. When the hypertensive process produces functional and structural changes in other organ systems, usually central nervous system (CNS), cardiovascular or renal, a variety of symptom complexes occur. Epistaxis may be a significant problem in hypertensive patients.

PATHOPHYSIOLOGY

Arterial blood pressure is determined by the interrelationship among (1) blood volume, (2) cardiac output, (3) blood viscosity, and (4) peripheral resistance. Blood pressure is controlled by a highly complex system including neural, chemical, and hormonal mechanisms which are not completely understood. The four principal control systems are:

1. The arterial baroreflex. Impulses from pressure sensors in the carotid sinus, aorta, and left ventricle are relayed through neural pathways to the brainstem. The efferent arc is also neural to the sympathetic adrenergic nerves and vagal cholinergic nerves;
2. Regulation of fluid volume. This is a slowly responding system that results in loss of...
fluid with elevated pressure and retention of fluid when pressure falls;
3. Renin and angiotensin. The initiating enzyme, renin, is released from the kidney and splits angiotensin I from plasma globulin. This is converted to angiotensin II which is a potent vasoconstrictor. Angiotensin II also stimulates aldosterone secretion;
4. Vascular autoregulation. In several organ systems, changes in perfusion pressure result in local changes in vascular resistance to keep perfusion volume constant.

DIAGNOSIS

The diagnosis of hypertension is established simply by measuring elevated blood pressure. The clinical significance of hypertension increases with levels of blood pressure but even moderate elevations are associated with decreasing life expectancy. For this reason, determination of the cause of hypertension is important in all patients whose diastolic pressure exceeds 90 mm Hg consistently. Causes of hypertension are listed and illustrated in Table 18–1 and Figure 18–1. An algorithm for the investigation of hypertensive patients devised by Fry is reproduced as Figure 18–2. As with all schemata, this should not be followed slavishly and, because of cost, tests should rarely be ordered in batteries.

ESSENTIAL HYPERTENSION

In the great majority of hypertensive patients, no specific cause for hypertension can be determined and the patients are considered to have essential hypertension. Patients with mild, untreated hypertension are probably not at increased risk for operation but patients with diastolic pressures exceeding 100 should be treated prior to elective operations. The treatment of essential hypertension is a combination of dietary salt restriction and a variety of drugs. These are listed by type of action and drug name in Table 18–2. Adequate control of hypertension by drugs is frequently difficult and combinations of agents are commonly employed. Many drugs used in the treatment of hypertension significantly alter the patient’s response to various anesthetic agents and this becomes an important factor in the surgical management of hypertensive patients. Early consultation with an anesthesiologist is advisable prior to operating on patients who are hypertensive.

SURGICALLY CORRECTABLE HYPERTENSION

From 1 to 10 percent of hypertensive patients have a surgically correctable cause for the hypertension. This is a highly significant group of patients and the importance of making a correct diagnosis is obvious. The reader is again referred to the management algorithm (Fig. 18–2).

Coarctation of the Aorta
Congenital aortic coarctation is one of the most common causes of hypertension in children but may be encountered at any age. Patients with significant coarctation have diminished or absent femoral pulses and a combination of

---

**TABLE 18–1. CAUSES OF COMBINED SYSTOLIC AND DIASTOLIC HYPERTENSION**

<table>
<thead>
<tr>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenal hyperactivity</td>
</tr>
<tr>
<td>Cushing’s syndrome</td>
</tr>
<tr>
<td>Hyperaldosteronism</td>
</tr>
<tr>
<td>Phaeochromocytoma</td>
</tr>
<tr>
<td>Renal disease</td>
</tr>
<tr>
<td>Renal artery stenosis</td>
</tr>
<tr>
<td>Fibromuscular hyperplasia</td>
</tr>
<tr>
<td>Dissecting aneurysm (involving renal artery)</td>
</tr>
<tr>
<td>Intrinsic disease—chronic pyelonephritis,</td>
</tr>
<tr>
<td>glomerulosclerosis</td>
</tr>
<tr>
<td>Mechanical</td>
</tr>
<tr>
<td>Coarctation</td>
</tr>
<tr>
<td>Other causes</td>
</tr>
<tr>
<td>Idiopathic—essential hypertension</td>
</tr>
</tbody>
</table>
patients, an be dei-
sidered to
ents with
ably not
ants
should
ons. The
is a com-
ly a v
ction and
ifficult
only em-
ent of
atient’s
s and this
surgical
nts. Early
is advis-
ho are

patients
or the hy-
group of
ring a cor-
again
(Fig.

the most
children
Patients
inished
ation of

hypertension and diminished femoral pulses should instantly suggest the diagnosis. Additional findings suggestive of increased collat-
eral flow, such as interscapular pulsations or rib notching on chest x-ray, corroborate the diagnosis.

The mechanism by which coarctation of the aorta produces hypertension has been de-
bated for years. Evidence suggests that both

mechanical factors and the renin–angiotensin

mechanism are involved.

Patients suspected of having coarctation
of the aorta should have the diagnosis con-
firmed by angiography and, when significant
coarctation is present, it should be corrected
surgically. It is clear that the earlier coarcta-
tion is surgically treated, the better the chance for
blood pressure reduction. The likelihood of

Figure 18-1. Etiology of some forms of hypertension. Note that many of the causes
can be treated by operation.
significant control of hypertension becomes so small that coarctation should probably not be repaired in patients over 50.

Renal Artery Stenosis
Several disease processes may produce extra-renal narrowing of the renal arteries of sufficient degree to activate the renin-angiotensin pressor system. These lesions include arteriosclerosis, fibromuscular dysplasia, dissecting aneurysm of the aorta, and trauma. The clinical picture produced by all of these entities is hypertension, with or without the cardiac, pulmonary, cerebral, or renal mani...

TABLE 18–2. DRUGS AND DOSES USED TO TREAT HIGH BLOOD PRESSURE

<table>
<thead>
<tr>
<th>Type of Action</th>
<th>Drug Name</th>
<th>Dosage Range in General Use (mg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diuretic</td>
<td>Bendroflumethiazide</td>
<td>5–10</td>
</tr>
<tr>
<td></td>
<td>Hydrochlorothiazide</td>
<td>50–100</td>
</tr>
<tr>
<td></td>
<td>Chlorthalidone</td>
<td>25–50</td>
</tr>
<tr>
<td></td>
<td>Polycarbazine</td>
<td>1–2</td>
</tr>
<tr>
<td></td>
<td>Propranalol</td>
<td>40–640</td>
</tr>
<tr>
<td></td>
<td>Oxprenolol*</td>
<td>40–640</td>
</tr>
<tr>
<td></td>
<td>Atenolol*</td>
<td>50–100</td>
</tr>
<tr>
<td></td>
<td>Metoprolol</td>
<td>200–600</td>
</tr>
<tr>
<td></td>
<td>Methyldopa</td>
<td>500–3000</td>
</tr>
<tr>
<td></td>
<td>Clonidine</td>
<td>0.3–1.0</td>
</tr>
<tr>
<td></td>
<td>Minoxidil</td>
<td>5–40</td>
</tr>
<tr>
<td></td>
<td>Prazosin**</td>
<td>0.5–15</td>
</tr>
<tr>
<td></td>
<td>Reserpine**</td>
<td>0.1–0.25</td>
</tr>
<tr>
<td></td>
<td>Guanethidine**</td>
<td>10–100</td>
</tr>
</tbody>
</table>

*Investigational drug in USA.
**Special care is needed to avoid postural hypotension. Start with a low dose. See manufacturers’ data sheets for full prescribing information and contraindications.
ifestations of hypertension. The stimulus for increased renin production in renal artery stenosis is probably dampening of the pulse pressure in the renal artery distal to the stenotic lesion. Details of the renin–angiotensin I and angiotensin II systems will not be reviewed here.

The hypertension produced by renal artery stenosis has no clinically distinguishing features but, because it is highly amenable to surgical treatment, it is important to be identified. It is estimated to occur in 5 percent of the hypertensive population. Any patient with moderate to severe diastolic hypertension who would be considered a candidate for surgical correction if a renovascular lesion were present should have an intravenous pyelogram (IVP). The rapid sequence excretory urogram is a relatively simple variation which gives added information. Digital subtraction angiography of the renal vessels has been suggested as a method of screening for renovascular hypertension but it is probably no more accurate than IVP and is certainly more expensive. If renovascular hypertension is suggested by the preliminary screening, renal vein renin levels should be measured. In unilateral renal artery obstruction, the involved kidney produces excessive amounts of renin which is reflected in the renal vein assay. When this level of renin is 1.5 times or greater than from the uninvolved side, a significant lesion is almost certainly present. Renal systemic renin indices (RSRI) may be of additional help. This is calculated as follows:

\[
\text{RSRI} = \frac{\text{Individual renal renin activity}}{\text{Systemic renal renin activity}}
\]

Arteriography is the most accurate method of demonstrating renal artery stenosis. Although complications of arteriography have been reduced, it is an invasive procedure and may be replaced by digital subtraction angiography when techniques for that examination are refined.

Surgical revascularization of kidneys with significant arterial lesions results in significant reduction of blood pressure in 90 percent of patients. Aortorenal bypass grafting is employed in most instances. Endarterectomy, with or without patch angioplasty, and reconstruction using the splenic artery may be preferable in some situations. Transluminal dilation of renal artery lesions is of great interest but, to date, has been disappointing in our experience.

Unilateral Renal Parenchymal Disease
Occasionally, a patient is seen who has hypertension, a contracted kidney, and arteriographic findings of no extrarenal stenotic lesions. Historically, removal of the involved kidney has not resulted in reduction of blood pressure in most patients. Recently it has been observed that when the atrophic kidney is behaving physiologically like an ischemic kidney, as determined by split renal function or renin vein assays, nephrectomy will result in blood pressure reduction.

Cushing’s Syndrome
The principal features of Cushing’s syndrome are the “moon” face, central obesity, cutaneous striae, and hypertension. The syndrome is produced by excess circulating glucocorticoids. Excessive glucocorticoid levels may result from iatrogenic administration, pituitary or adrenal lesions, or nonadrenal sources of ACTH production, principally malignant neoplasms.

The diagnosis of Cushing’s syndrome usually begins with clinical observation of the distinctive physical changes. The most widely used screening test is measurement of the urinary excretion of 17-hydroxycorticosteroids. Plasma cortisol levels can be determined and, like the urinary 17-hydroxycorticosteroids, this is usually elevated in Cushing’s disease. Measurement of ACTH levels and its response to dexamethasone suppression may be helpful in differentiating pituitary from adrenal disease. Cranial and abdominal CT scans are currently the most accurate method of demonstrating the lesions anatomically.
The most satisfactory treatment of Cush- 
ing’s disease is surgical removal of the lesion 
responsible for the syndrome. This is more 
completely discussed in Chapter 7.

**Hyperaldosteronism**

Hyperaldosteronism is a condition that 
probably occurs less commonly than was once 
thought. It is caused by hypersecretion of the 
mineralocorticoid, aldosterone, by an adrenal 
adenoma, or by adrenal hyperplasia. Hyper-
tension is present but usually not severe in 
adults. The diagnosis is suspected when per-
sistent hypokalemia and metabolic alkalosis 
are detected in hypertensive patients not re-
ceiving diuretic therapy. Plasma renin activity 
is usually low and urinary aldosterone levels 
are elevated.

Treatment of this condition is discussed in 
Chapter 7.

**Pheochromocytoma**

Pheochromocytoma is a rare, interesting ne-
oplasm arising in the adrenal medulla and pro-
ducing norepinephrine and/or epinephrine. 
The tumor may be unilateral or bilateral, be-
nign or malignant. Patients with pheochrom-
cytomas have episodic or sustained hyperten-
sion. Determination of urinary catecholamine 
and vanillylmandelic acid (VMA) secretion are 
probably the best screening tests for phe-
ochromocytoma. Confirming tests include 
measurement of plasma epinephrine and nor-
epinephrine levels. When biochemical evi-
dence for pheochromocytoma exists, it be-
comes important to localize the responsible 
neoplasm. Intravenous pyelography may 
show downward displacement of the kidneys, 
but a CT scan of the abdomen appears to be the 
most valuable diagnostic tool for locating adre-
nal lesions as well as other retroperitoneal 
mass lesions. The CT scan has superseded to-
mography, arteriography, and retroperitoneal 
gas insufflation.

Operations on patients with unsuspected 
pheochromocytoma can be disastrous because 
anesthesia and operative manipulation of the 
tumor may result in uncontrollable hyperten-
sion or hypotension. Preoperative preparation 
of patients with pheochromocytoma with 
alpha-adrenergic and beta-adrenergic blocking 
agents has decreased surgical risk. Conduct of 
anesthesia is extremely important.

The surgical treatment of pheochromocy-
toma is discussed in Chapter 7.

**REFERENCES**

Dollery CT: Arterial hypertension. In Wyngaarden 
JB, Smith LH Jr (eds): Textbook of Medicine, 16th 

Ernst CB, Bookstein JJ, Montie J, et al: Renal renin 
ratios and collateral vessels in renovascular hyper-

Foster JH: Surgically correctable hypertension. In 
New York, McGraw-Hill, 1979, p 1011

Fry WJ, Fry RE: Surgically correctable hypertension. 


Havey RJ, Krumlovsky F, et al: Screening for hyper-
tension. JAMA 254(3):388, 1985

**CHAPTER REVIEW**

**Questions**

1. Physical findings are suggestive of which 
types of surgically treatable hypertension?
2. Persistent hypokalemia in a patient with 
hypertension suggests what condition?
3. What pathologic lesions are responsible for 
most instances of renovascular hyper-
tension?
4. How are patients with pheochromocytoma 
pharmacologically prepared for operation?
5. List the lesions responsible for develop-
ment of Cushing’s syndrome.

**Answers**
1. Coarctation, Cushing's disease
2. Hyperaldosteronism
3. Adrenal adenoma
   a. Pheochromocytoma
   b. Fibromuscular hyperplasia
   c. Axonal
   d. Exostosis