THE JESSE PATT PRIZE IN SURGERY.

THE ROLE OF THE SURGEON IN THE TREATMENT OF ARTERIAL HYPERTENSION.

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Introduction:

In his classical description of cases connected with albuminous urine, Bright (1836) recognized that hypertrophy of the left ventricle was probably due to the greater action necessary to force blood through the distant subdivisions of the vascular system. When it became possible to measure arterial blood pressure, this supposition was confirmed. Knowledge of hypertension was initially derived from Post Mortem evidence of left ventricular hypertrophy without obvious cause. However, with the introduction of instruments of von Basch and Riva Rocci (1896), accurate measurement of arterial blood pressure became possible and evidence obtained was more quantitatively based.

The need to treat hypertension:

From the figures below in Table 1, taken from a report by the Actuarial Society of America & the Association of Life Insurance Medical directors 1941, it may be seen that whether systolic or diastolic arterial blood pressure is considered, mortality increases with pressure, and that the relation is quantitatively. There is no sudden break. The excessive mortality among the hypertensives is primarily due to the 4.5 times greater incidence of cardiovascular-renal disorders.

<table>
<thead>
<tr>
<th>Systolic Reading mm Hg</th>
<th>Diastolic Reading mm Hg</th>
<th>Systolic - 83%</th>
<th>84-89%</th>
<th>90-93%</th>
<th>94-103%</th>
<th>All %</th>
</tr>
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<tbody>
<tr>
<td>118 - 132.</td>
<td>91</td>
<td>118</td>
<td>112</td>
<td>97</td>
<td>9.2</td>
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<tr>
<td>133 - 142.</td>
<td>100</td>
<td>118</td>
<td>112</td>
<td>134</td>
<td>11.0</td>
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<tr>
<td>143 - 152.</td>
<td>137</td>
<td>118</td>
<td>112</td>
<td>173</td>
<td>14.8</td>
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<tr>
<td>153 - 162.</td>
<td>178</td>
<td>118</td>
<td>112</td>
<td>237</td>
<td>21.0</td>
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</table>

MORTALITY RATIOS for Men According to Groups of Systolic and Diastolic (5th Phase) Blood Pressure Readings:

There is however, considerable controversy over the defining lines between Normotension and Hypertension.

A generally accepted guide is given by Pickering et al 1964:

<table>
<thead>
<tr>
<th>Age</th>
<th>Male</th>
<th>Female</th>
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<tbody>
<tr>
<td>Under 40</td>
<td>105</td>
<td>115</td>
</tr>
<tr>
<td>40 - 59</td>
<td>110</td>
<td>120</td>
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These figures would be modified up or down by other factors affecting the prognosis. E.g. in an obese male with elevated serum cholesterol, there would be a strong case for reducing the arterial pressure persistently of the order of 100 mm Hg.

**Classification of arterial hypertension:**

Arterial hypertension may be broadly classified into two major groups, Primary or Essential hypertension and Secondary hypertension.

Primary hypertension is responsible for 85% of all cases of arterial hypertension, and in these cases, the etiology of the hypertension is unknown. It is a disease of the middle aged and elderly with its main incidence in the 40-60 yr age group. Although so common, it is nonetheless a serious condition, and is the direct cause of 20% of all deaths over the age of 50.

It has some familial connections, if both parents suffer from the disease, then siblings stand a 45% chance of developing it, and if only one parent has the disease, the siblings' chance falls to approximately 30%. Primary hypertension may be divided into two categories, Benign and Malignant.

Benign essential hypertension is the most common, accounting for 90% of all cases of essential hypertension. The arterial blood pressure rises very gradually over years in most cases to moderately high levels e.g. 160/110 mm Hg. The rise normally always starts before the age of 45. About 60% of deaths in patients with benign essential hypertension are from left ventricular or total heart failure if this is due to the increased workload thrown upon the left ventricle. 30% die from cerebrovascular disorders & the remaining 10% from causes unrelated.

The etiology of benign essential hypertension is obscure. There is an increase in peripheral resistance—normally controlled by the muscular tone of arterioles throughout the body. The renal vessels are particularly affected. It is unlikely that arteriosclerosis initiates the hypertensive state, as such changes are sometimes absent, & moreover structural changes do not develop in arterioles which are protected from hypertension by exclusion. Changes in the larger arterioles supplying the kidney because of these reasons, it is accepted that the observed structural changes in arterioles of arterioles are the result of hypertension and not the cause. The kidney may be important in its pathogenesis, as when its blood supply is impaired due to arteriolar narrowing, there is a vicious circle of arteriolar spasm followed by arteriolar thickening, with the development of renal ischaemia and the release of vasoactive substances such as renin which increase the hypertension. Thus there is a progressive increase in the severity of the condition.
Malignant essential hypertension develops in about 10% of cases of benign essential hypertension. It is characterized by a very high arterial blood pressure which progresses rapidly, eye changes such as retinal haemorrhages, exudates, and papilloedema. There is rapidly progressive renal injury terminating in uraemia and by hypertensive encephalopathy. Unless treated, patients with malignant hypertension will die in about 6 months, usually of renal failure.

Secondary hypertension may be caused by a number of conditions which will merely be listed here, as the conditions will be considered in detail later.

Causes of Secondary Hypertension:

1. Renal:
   i) Renal artery stenosis.
   ii) Acute or chronic glomerulonephritis.
   iii) Chronic pyelonephritis.
   iv) Polyarteritis.
   v) Tumours.
   vi) Tuberculosis.
   vii) Miscellaneous conditions e.g. amyloidosis, polyarthrites, diabetes.

2. Endocrine:
   i) Phaeochromocytoma.
   ii) Cushing's Syndrome.
   iii) Cushing's Syndrome.

3. Coarctation of the Aorta:

4. Toxaemia of Pregnancy:

A Systematic Consideration of the Surgical Treatment for Hypertension:

Essential Hypertension:

The hypothesis that hypertension of this primary or essential type was due to overactivity of the sympathetic nervous system was popular for a long time, and led to the operation pioneered by Rutherford and Adson in 1925, namely Subdiaphragmatic Sympathectomy and Excision of the abdominal sympathetic ganglia. Since a significant fall in blood pressure was not invariably, the operation was extended, culminating in the total excision of the paravertebral chains by O'Brien.

Reganomacenic cervicodorsal sympathectomy may be performed by two main routes:

a) Supraclavicular method — Through a supraclavicular incision, the clavicular part of the sternomastoid, the posterior belly of the omohyoid, and the scalene anterior muscles are divided, the phrenic nerve being displaced upwards. The subclavian artery is exposed and depressed so that the supracleural fascia can be divided and the dome of the pleura displaced downwards. The stellate ganglion may be identified as it lies on the neck of the first rib. The
Sympathetic trunks is traced downwards and divided below its 3rd thoracic ganglion. All the rami communicantes associated with the 2nd and 3rd ganglia and its rami of 3rd, a grey ramus running upwards from the 2nd thoracic ganglion to the 4th thoracic nerve are also divided. The procedure is then repeated on the contralateral side.

Fig. 1.

Exposure of the Right Sympathetic Chain

b) Transthoracic Method: This gives a greater exposure and facilitates the removal of the sympathetic chain from the 5th ganglion up to the lower fringe of the stellate ganglion. It tends to give better results than a) and can be employed when all has failed. Through a posterolateral thoracotomy via the 4th intercostal space, the sympathetic chain is easily seen and after dividing the pleura, it is dissected out, care being taken to avoid the intercostal vessels.

Chisla is as to the effects of these operations have varied widely. Characteristic of its favourable results is that of Smithwick (1956) who studied 1118 males and 1104 females, some of whom were treated medically, and some surgically. The comparison shows a substantial increase in survival in the patients treated by operation. Unfortunately the matching of patients was poor. However, Evelyn et al. (1966) have followed up 100 of Smithwick's sympathetomised patients for 10 years and compared them with 100 unoperated controls who were matched as closely as possible. The results after 10 years slightly favoured the sympathectomy operation; 48% of males and 34% of females in the operated series were dead, compared with 50% and 40% respectively in the control series.

According to Hessels J.B. Macekod & D.C. Carter EJ 1974, Sympathectomy has not been performed as a routine operation for primary hypertension since the early 1950s, in Edinburgh at least. Thus, no further revision will be made except to note that:

1. There is no doubt that the operation has achieved some striking and lasting successes.
2. Such successes seem to be more frequent in the younger patient and the more recent the hypertension.
3. Unfortunately there are some patients in whom the blood pressure is not reduced. In those with malignant hypertension, sympathectomy does not guarantee a blood pressure fall adequate to reverse the malignant to the benign phase, though this may happen.
4. No test has yet been devised that will distinguish those who will react favourably from those who will not.

5. The effects of sympathectomy tend to wear off after some years.

Thus, nowadays sympathectomy is not the treatment of choice in primary hypertension. Chemotherapy, using sympathetic-blocking drugs carries less risk and is more certain. If chemotherapy fails or cannot be carried out for some reason, then sympathectomy is probably the best choice in most patients, especially if they are young and the hypertension recent.

Renal causes of secondary hypertension:

1. Renal artery stenosis:

   This is prominent as a cause of arterial hypertension due to the possibility of total cure offered by operative measures such as arterial reconstruction, or reversal of the hypertension by nephrectomy. (Nelson et al 1947, Younger et al 1966, McKusick et al 1969)

   The disease occurs in two main age groups:

   a) Relatively young people, especially females, in which the cause appears to be thickening of the intima of media by hyperplasia of collagen and muscle in the vessel wall - Fibromuscular hyperplasia. (Tyng and Wellington 1960, Foster et al 1969)

   b) The elderly, in whom the cause of narrowing is atherosclerosis, a plaque is frequently found close to the origin of the main vessel.

   The diagnosis of renal artery stenosis is suggested by an audible murmur on either side of the midline above the level of the umbilicus. An intravenous pyelogram may help considerably in diagnosis. There is usually delay in excretion of the dye on the affected side, but the actual intensity of the shadow produced by the dye when excretion occurs is greater than on the normal side due to an impaired glomerular filtration, but relatively less impaired tubular function, so that reabsorption approaches normal.

   Angiography - the injection of a radiopaque dye into the aorta just above the renal arteries by means of a catheter fed in from the femoral artery - should be performed in younger patients with severe hypertension so that the actual stenosis of the renal artery may be directly demonstrated.

   Luteten catheterisation studies can also be performed. If kidney whose artery is stenosed forms a smaller volume of glomerular filtrate, and a higher proportion of the filtered water, Na⁺ and Cl⁻ ions are reabsorbed. Samples from the affected side will be more concentrated.

   Treatment for renal artery stenosis falls into 4 main categories: Nephrectomy, Partial nephrectomy, Reconstructive vascular surgery, and Autotransplantation.
Nephrectomy

This is usually chosen for the treatment of poor risk patients when there is gross damage of the kidney in the presence of a normal or hypertrophied contralateral kidney. (Burkebund 1959)

The kidney lies in a deep recess beneath the right thoracic cage, so no single incision will meet all requirements, but the oblique lumbar incision (see Fig. 4) is usually adequate.

The patient is placed on the operating table in the lateral kidney position to avoid the pleura, the incision commences 1.2 cm below and lateral to the angle made by the 12th rib with the outer border of the scapula, and it passes downwards and outwards towards the anterior superior iliac spine. All the muscular layers are incised until the extraperitoneal fat is reached. If the access obtained is inadequate, the 12th rib can be dislocated upwards or resected. The renal pedicle is cleared of fat so as to display its vascular components. After isolating the ureter, it is divided between haemostats as far from the kidney as is convenient. The clamped end is ligatured commencing inferiortly. A portion of the vascular pedicle is caught in a long haemostat, care being taken to avoid the renal pelvis. A short haemostat is placed in juxtaposition nearer to the kidney and the tissue between the haemostats is divided. The process is repeated until the kidney is free. It is removed, and after ligation of any small haemorrhaging vessels, the tissue layers are stitched up again.

Partial Nephrectomy

This has become more popular in recent years, and its obvious advantage is that it conserves as much functioning renal tissue as possible. It is indicated for the treatment of lesions of branches of the renal artery. (Farquharson 1966).

The operation is necessarily very much easier when the lower pole rather than the upper pole is required to be removed. Control of haemorrhage, the most difficult part of the operation, may be achieved by compression of the vascular pedicle, using a light occlusion clamp, which should not be left on for more than 10 mins. The capsule is incised around the affected pole, and reflected back beyond the proposed level of section. Rickles (1959) considers that the kidney tissue should be divided using a flat transverse cut (rather than a V-shaped incision (Farquharson 1966)) and to give this a double covering with the overlapped capsular flaps. Before the repair is commenced, the assistant relieves pressure on the vessels at intervals, so that bleeding points on the cut surface can be identified and undermined with fine caligus sutures. Any available flaps of renal fascia or fat are utilised to reinforce the suture line, and to assist in the control of haemorrhages.
c) Direct vascular reconstruction:

Renal artery stenosis possibly suitable for renovascular surgery is uncommon, probably representing only 3-5% of the hypertensive population (Kenedy et al. 1965). In addition, there is no evidence that reconstructive vascular surgery is more likely to be associated with a good blood pressure response than nephrectomy (Maxwell, 1966), but every effort should be made to conserve functioning renal tissue, and reconstructive vascular surgery should be carried out wherever possible. These procedures are however often protracted, and in the older patient especially, may carry significant morbidity and mortality. Furthermore, reconstructive surgery is not possible in a significant proportion of patients with fibromuscular hyperplasia because of the involvement of the distal renal artery by the disease process (Fenton et al. 1965).

Policy for the selection of patients for surgery in unilateral renal artery stenosis, with hypertension:

a) Assess the significance of the hypertension, renal function and fitness for possible surgery.

b) In suitable cases, proceed to transvenous pyelograms and renography.

c) If the IVU and renogram imply unilateral renal artery stenosis with renal ischaemia, and if the opposite kidney is normal, then for young patients with significant hypertension, proceed to angiography.

d) If these tests and features are in agreement, and there are no contraindications, then renovascular surgery is indicated.

e) In older patients, there should be an attempt to control the hypertension using antihypertensive drugs, but if they are not effective, or have too many adverse effects, then a surgical approach can be reconsidered.

If the stenosis is fairly proximal, and the distal vessels relatively healthy, removal of the stenosed portion of the artery with a prosthetic replacement, or a bypass procedure e.g. an omental bypass, or a bypass grafting may be done. Often this may improve the blood pressure, often with an improvement in renal function and symptoms of hypertension.

By-pass Grafting:

The by-pass may either be a simple one as in Fig. 2, with both ends of the graft attached to the renal artery, or alternatively if the stenosis is due to a plaque close to the origin of the renal artery, the graft by-pass may run from the aorta to the renal artery distal to the stenosis as in Fig. 3.
Grafts may either be autogenous or synthetic, with Dacron being a common type. Arterial autografts which have been prepared by freeze-drying are preferred (McNeill hose 1966), and sections of the splenic artery are often used, without sacrifice of the spleen (Bonhomme 1970). If a synthetic graft is used, it should be about twice the size of the renal artery to avoid failure of the graft from fibrosis (Kaufmann & Naoney 1967), as after division, the graft becomes incorporated by an invasion of fibroblasts and becomes fixed within the lumen by endothelial cells.

d) Autotransplantation:

Autotransplantation of the kidney in renal artery stenosis has considerable merits, especially where in situ repair is difficult or the aorta diseased, rendering by-pass grafting difficult. It permits anastomoses to normal pelvic vessels with optimal exposure and predictable technical success. E.g. The kidney may be repositioned in the right iliac fossa, and end to end hypogastric artery to renal artery and end to end renal vein to common iliac vein anastomoses are made using interrupted suture techniques.

The principal technical limitations and complications resulting from renal autotransplantation have been chiefly involved with arterial problems, but with the ureter left intact, albeit with a redundant cause, common urological complications can be overcome.

Kaufmann (1972) has performed several operations of this type, mainly in the treatment of hypertensive children where the hypertension has been caused by congenital bands compressing the renal artery, or aberrant renal arteries. His results are very encouraging with patients remaining normotensive for the follow-up period of 2 years.
The results of surgery of all the above-mentioned types have been evaluated by Justan (1963) and Sakah (1967) and their conclusions indicate that after surgical treatment of renal stenosis, overall, 65% of patients are rendered normotensive for a follow-up period of 1-6 years, and another 18% have their hypertension "greatly improved".

2. GLOMERULONEPHRITIS, PYELONEPHRITIS, TUBERCULOSIS OF KIDNEY, REINAL AMYLOIDOSIS

All forms of renal parenchymal disease are liable to cause arterial hypertension. Especially common in this respect are chronic glomerulonephritis and chronic pyelonephritis (which may arise from a lower urinary tract infection which was not adequately treated).

The only possible surgical treatment for longstanding renal disease which is bilateral or bilateral nephrectomy followed by renal transplantation - somewhat drastic surgery, especially in view of the efficacy of modern chemotherapy in the treatment of infections and hypertension.

However, much attention has centred upon the small number of hypertensive patients suffering from unilateral renal disease, because of the possibility that an operation on the affected kidney might reduce the hypertension permanently. (Luker 1968). In a review of 102 patients suffering from various disorders, mostly pyelonephritis, tuberculosis & hypoplasia, complete relief of hypertension could be obtained in 33% of those patients treated surgically (Luker 1968). A significant proportion of hypertensive patients have unilateral renal disease as the aetiological factor causing their hypertension.

The most amenable lesion to surgical treatment is sclerosing pyelonephritis which, if unilateral, can be cured by nephrectomy. Unfortunately, pyelonephritis is usually bilateral, and unilateral disease only occurs if the kidney has been the site of previous trauma, congenital malformation, or if the ureter on that side has been blocked by eg. a calculus, or unilateral hydronephrosis has occurred.

Little else will be considered in this discussion except to note that the operations performed are essentially similar to those in nephrectomy or partial nephrectomy for renal artery stenosis (p.w.). Partial nephrectomy is usually undertaken if the lesion is well localised in one pole, eg. a single tuberculous lesion. An outline for the policy of selection of patients for surgery in unilateral parenchymal renal disease is given below:

1. Assess the severity of the hypertension, total renal function and fitness for possible surgery.
2. In suitable cases, intravenous pyelograms and renograms should be carried out.

3. Often an infusion IVP with nephrotomography permits more precise diagnosis and shows more clearly the anatomy of the contralateral kidney.

4. If the diseased kidney is contributing less than 20% of the total renal function, which is itself maintained, and the contralateral kidney is healthy, nephrectomy is advised provided the hypertension is significant. The best results are likely to be obtained in younger patients with a short history of hypertension.

**Poly cystic Disease of the Kidney**

About 75% of patients with poly cystic disease have some degree of hypertension, why the other 25%, do not is unknown, possibly it is the development of hypertension is due to a separate genetic factor, as it is well established that poly cystic disease is hereditary. The condition is slightly more common in women but in both sexes the prognosis is poor, only one quarter of all patients survive the age of 50.

Excretion Pyelography is the best method of confirming the diagnosis. The shadows are enlarged in all directions, the renal pelvis is elongated, the calyces are stretched over the cysts and are often narrow.

Surgical treatment is not primarily aimed at controlling the hypertension but at relieving pressure upon any normal renal parenchymal tissue and in preventing renal failure. The operation pioneered by Rovsing (1862-1927), if performed early, improves renal function; both kidneys are exposed and the cysts are incised first on the convex border, then on the posterior surface, and as the kidney becomes more manageable in size, the peritoneum is peeled away and the anterior surface dealt with similarly.

However, some authors (Smith, Schulte & Smart 1970) do not advocate surgical treatment unless complications supervene. (Nikan et al 1963) These include spontaneous rupture, obstruction etc.

**A. Renal Tumours:**

In a series of 491 patients with renal tumours of various types (Morlock & Horton 1936), the group as a whole showed no significant rise in arterial pressure, and there was no consistent change in blood pressure after nephrectomy. However, in children with Wilms' tumour, severe hypertension is often found, and also in children with Gravitz (adenocarcinomas) tumours. Here are cases where its blood pressure has risen from 100/60 mmHg to 180/140 mmHg within one month, and by this time, renal exudates and papilloedema were present.
That such humours are the initiating factor in the development of hypertension is shown by the fact that if the lesion containing the humour is removed, the hypertension subsides. There are two theories accounting for the hypertension: a) possibly the expanding humour impedes the renal arterial blood supply and acts like a Goldblatt (1937) clamp. This is substantiated by evidence supplied by Koons & Rich who showed that the hypertension was relieved by nephrectomy even although secondary deposits remained. b) The second theory is that the humour releases pressor substances, such as renin, into the circulation. This is supported by the fact that the hypertension is relieved by preoperative radiation therapy. However, as yet no such substance has been detected in extracts from within humours.

In cases of small humour, a preoperative course of deep X-ray therapy is given which usually causes diminution in the size of the humour, but also causes anaemia and leucopenia. Therefore blood transfusion is carried out in the 4 weeks elapsing before the operation. Nephrectomy is performed. If the humour has not greatly decreased in size, the abdominal route is chosen, otherwise humoral nephrectomy is chosen. As soon as the wound has healed, postoperative irradiation is given. However, its prognosis is poor, and metastases prove fatal within 2 yrs in 80-100% of cases.

For adenocarcinomas of the kidney in adults, the procedure is very similar. The abdominal route offers certain advantages, notably that the pedicle can be ligated before the organ is handled, thereby minimizing dissemination of the growth. Whatever route is chosen, the renal vein should be ligated as closely as possible to the inferior vena cava, as in 10% of cases, there is humour extension into this vein. The prognosis is poor, about 60% of patients suitable for operation are alive after 3 yrs.

ENDOCRINE CAUSES OF SECONDARY HYPERTENSION:

1. PHAECHEMOCYTOPHOMA:

Phaeochromocytoma is a benign humour of chromaffin cells in the adrenal medulla which secretes and liberates into the circulation, noradrenaline and adrenaline in proportions up to 20:1. About 10% of these humours are malignant and 10 to 20 are multiple. Any age may be affected, but the humour is especially found in young adults of both sexes. The catecholamines which are released from the humour produce hypertension which is severe and either paralytic, or if at a late stage, persistent. As surgery offers a complete
cure for this condition, it is essential that all patients under 60 yr who suffer from sustained arterial hypertension have routine tests to confirm or exclude a phaeochromocytoma, while nationwide it is considered that not more than 0.5% of all cases of hypertension are caused by a phaeochromocytoma, at the Mayo Clinic, where routine diagnostic procedures are undertaken, its percentage is 3% - an important fact when considering that untreated, the condition progresses to a fatal termination solely as a result of its effects of hypertension.

The tumour has a number of associations. It is associated with pregnancy (Hume 1969) and if undiagnosed, its prognosis is poor - about 45% mortality (Peelen & Degroot 1953). Unfortunately its diagnosis is easily confused with hypertensive disease of pregnancy (Pickering 1955) especially when the typical paroxysms are absent. Carcinoma of the Thyroid is also 14 times commoner in patients with phaeochromocytomas than in the general population (Sipple 1961).

The clinical features include a definite group of symptoms produced by its oversecretion of noradrenaline and adrenaline, e.g. Hypertension which is initially paroxysmal but later becomes persistent. It is arteriosclerosis even in young patients. Increased sweating, palpitations, glycosuria, hypoglycaemic attacks and headaches.

Investigations: An intravenous pyelogram may reveal displacement of the upper pole of the kidney. Paraesophageal injection of oxygen into the retroperitoneal tissues is free from danger and combined urography is helpful in defining the tumour. Angiography often reveals a vascular "blush" at the tumour site, and, in addition, shows the vascular state and size of the kidneys. Examination of the urine for vanillymandelic acid, a metabolite of noradrenaline, and blood tests for noradrenaline and adrenaline confirm its diagnosis.

Preoperative and operative management:

The hazardous phases during the operation are:

a) During induction of the anaesthetic, as halothane and other anaesthetics stimulate the release of catecholamines. b) The positioning of the patient on the operating table. c) When the tumour is being manipulated d) Immediately after removal of the tumour e) Haemorrhage during the operation can be considerable when a very vascular tumour is being removed.

In these reasons, the operation was considered to be dangerous until the use of adrenergic blockers such as phentolamine or phentolamine has reduced the operative mortality from about 30% to less than 5% - Ross, Prichard (1967).

20-40 mg of phentolamine should be given after meals three times daily for 3 days preoperatively. Prior to
Commencing anaesthesia, 5 mg of pentoxyfresh is given intravenously, and a drip infusion of dextrose and saline is run during the operation.

An ample posterolateral incision is commonly used; after subperiosteal resection of the twelfth rib, the lower border of the pleura is defined and protected. The incision is extended through the bed of the 12th rib to reveal the perinephric fat wherein the adrenal gland is identified. The tumour is usually less than 2 cm in diameter and a brown colour. As some 10% of tumours are ectopic (6% of all pheochromocytomas are extraadrenal and in the organs of Zuckerkandl. Newall 1970), there is a case for exploration via an anterior route through a curved transverse incision. The blood pressure rises sharply when the affected adrenal is handled, and to counteract this, another 5-10 mg of pentoxyfresh is administered. As soon as the adrenal is removed, a considerable fall in blood pressure is to be expected and this is combated by an intravenous injection of Noradrin (which should also be on hand for 1 week postoperatively).

The choice of a adrenergic blocker varies from surgeon to surgeon. The blocking of pentoxyfresh is immediate, but transient. Its hypotensive effect under these circumstances persists for only 5-10 minutes so injections must be repeated frequently, and control of blood pressure is erratic. In addition, pentoxyfresh has the disadvantage of causing tachycardia. Greater control has been achieved by the use of phenoxybenzamine (Ross 1970), which has a much longer duration of action. However, although it is much better in reducing rises of blood pressure than pentoxyfresh, it does not block or counteract the effects of the catecholamines upon the heart, so tachycardia and arrhythmias remain.

Haemorrhage during the operation is serious, and blood loss must be made good rapidly, as the blood pressure of patients under complete or near complete adrenergic blockade is very susceptible to volume changes.

If symptoms persist after unilateral adrenalectomy, a tumour in the contralateral gland is highly probable, and upon subsequent operation, the second tumour (which is usually well-defined) must be dissected from the healthy portion of the gland, as removed would necessitate replacement therapy using glucocorticoids for the rest of the patient's life.

Although surgery offers a complete cure for the condition, if it cannot be performed or if a malignant form of the tumour is present which prevents complete surgical removal, medical management with adrenergic blocking drugs can control.
Symptoms successfully in these patients.

2. CONN’S SYNDROME:

In 1954 J.W. CONN described a clinical syndrome of mineralocorticoid excess induced by adrenocortical synthesis of too much aldosterone, and characterised by greatly increased amounts of aldosterone in the urine, but normal amounts of 17 hydroxy-progesterone and 17ketosteroids. The symptoms include muscular weakness (particularly of the lower limbs) and paralysis, polyuria and polydipsia, headaches etc.

Upon investigation, a moderate degree of hypertension is apparent, with hypernatremia and hypokalaemic alkalosis. The sex ratio is 2.5 female: 1 male and although it can occur at any age, the maximum incidence is 30-60 years. In making a diagnosis of primary aldosteronism, the syndrome must be carefully differentiated from the secondary states of excess aldosterone secretion which occur in e.g. Cushing’s syndrome, congestive cardiac failure, nephrotic syndrome etc. This is facilitated by the double isotope clearance assay technique for aldosterone secretion described by Klibanov & Peterson (1960).

The lesion causing the syndrome is an adenoma of the adrenal cortex. The adenoma is usually single, but bilateral in 10%, benign and well encapsulated. They are small, 1.3g or less, and relatively slow growing. Because of the small size of the adenoma, preoperative procedures to determine the side or side of the tumour are unlikely to be successful, although the larger tumours may sometimes be identified by retroperitoneal pneumography using oxygen or pyelography alone or combined. Isotopic techniques using 131I cholestrol and the gamma Camera appear promising in the detection of the adenomas (CONN 1967).

Preoperatively, efforts should be made to eliminate the severe hypokalaemic alkalosis and up to 300 mg/day should be given for several days. Failure to correct the severe electrolyte imbalance may lead to arrhythmias, respiratory paralysis and irreversible shock. The stress of anaesthesia & operation (Zwirnerman 1960) Careful bilateral exploration is needed and a posterior 12th rib approach is easiest and has the lowest postoperative morbidity. Any grossly detectable adenoma in either gland is removed but if none can be found, the right adrenal gland should be removed (g.v) & sectioned. If still not found, the left gland should be halved - one half must remain to prevent Addisonian conditions ensuing. According to Conn, the operative success is 40-95%. He also considers that at least 25% of patients diagnosed with “Primary hypertension” have primary aldosteronism, as the condition is not always associated with hyperaldosteronism (CONN, SIENMAI 1966), and upon autopsy, what have previously been labelled as “non-functioning adrenal cortical adenomas” are actually aldosterone producing tumours. If so, many patients can in future be cured radically by an essentially simple operative procedure. (Goert 1960, SHINMAN 1958).
Cushing's Syndrome

Cushing's syndrome or adult hypercorticism is a disorder caused by excessive production of glucocorticoids (mainly hydrocortisone) by the adrenal cortex. In 35% of cases, an adrenal neoplasm is present and in 5% of these, half are malignant carcinomas. In 60% of cases, the patient has bilateral adrenal hyperplasia, which is in 4% of these is caused by a basophil adenoma of the anterior pituitary gland. In 5%, there is no discernable structural alteration in the adrenal glands, but tumours of the lungs, liver (benign carcinomas can produce ACTH occasionally), or pancreas.

To Summarise:

- Adrenal Neoplasm
  - 35%
  - Malignant Carcinoma

- Bilateral Adrenal Hyperplasia
  - 60%
  - Basophil Adenoma of Anterior Pituitary
  - Basophil Hyperplasia of Anterior Pituitary
  - Cort-Cell Carcinoma of Lung
  - Thymoma

- Renal Adenoma

The syndrome has a 3:1 sex preponderance towards females, and the commonest age group for it is 15-30 yrs. The clinical features are highly characteristic. The patient has a moon face, protruberant abdomen, thin neck, relatively thin neck, red-purple striae on abdomen, acne and bruises. Males also suffer from impotence and females get amenorrhea. The hypertension is moderately severe, and eventually congestive cardiac failure supervenes. Lab findings include polyglycaemia, a low basal metabolic rate, elevated serum cholesterol and urinary 17-hydroxysteroid.

Operations:

a) If an adrenal tumour has been demonstrated preoperatively by procedures outlined previously, extirpation of that gland alone is carried out.

b) If a tumour has not been demonstrated, the patient is prepared for bilateral exploration.

It is essential that all patients who are to be subjected to extirpation of adrenal cortical tissue be adequately prepared for the operation, and supported postoperatively by adrenocortical hormone replacement, irrespective of the extent of adrenal resection. A typical regime would be:

**Pre-operatively:**

100 mg. Corinone bid intramuscularly for 2 days before operation.

600 mg. Hydrocortisone daily by intravenous drip, then another loading during the subsequent 12 hours.

**During Operation:**

Day 1: Corinone 50 mg. Intramuscularly, 6 am.

Day 2-3: Same 8 am.

Day 4-5: Same 12 am.

**Post-operatively:**

Fig. 6.
Hence, cortisone should be given orally and slowly reduced to maintenance levels in cases of total adrenalectomy, or to zero in subtotal adrenalectomy.

Exploration of the left adrenal is carried out indirectly, as the difficulties are usually less than on the right side. If a tumour is found, adrenalectomy (r.v.) is carried out on that side.

If the left gland is found to be atrophic, it is highly probable that there is a tumour on the right side which should be explored forthwith. If the gland is hyperplastic or normal, subtotal (40%) adrenalectomy is indicated. If, after this has been performed, it is apparent that the patient will not tolerate a bilateral operation well, exploration of the contralateral side should be postponed. The operation is essentially that for resection of a phaeochromocytoma (r.v.); on the right side, the suprarenal vein is short and may be from the Inferior Vena Cava if it is not identified and ligated at an early stage of the dissection.

By finger and gauze dissection, the gland is freed from below and behind, upwards, ligating and dividing bleeding vessels as they are encountered, until it is suspended by only its main vascular pedicle near the apex. If subtotal adrenalectomy is to be performed, the gland is cut across with scissors so as to leave a small triangular fragment of the apex well supplied with blood vessels; bleeding should be controlled by snares and pressure, as diathermy coagulation leads to necrosis.

If the cause of the syndrome is a pituitary basophil adenoma, the tumours are usually small, only a few millimetres in diameter. Their effects are produced by secretion of ACTH which stimulates glucocorticoid synthesis by the adrenals. Treatment is directed to reduction of the secretion of glucocorticoids, either by adrenalectomy or by operation on the pituitary. Surgical removal of the pituitary is a technically difficult and hazardous procedure practised only by neurosurgeons. By contrast, destruction of the gland by pellets of radioactive Iodine 125 inserted into the pituitary fossa by stereotactic approach from above, or implanted across the sphenoidal sinuses through a paranasal incision under X-ray control. This operation is simple to perform and carries little risk. (Forrest.)
3. **Cocartion of the Aorta**

This is a congenital narrowing of the aorta, usually occurring in the descending aorta, just distal to the origin of the left subclavian artery. The stenosis is usually extreme, only a pinpoint lumen remaining. Cocartion can rarely occur in other sites in the aorta. Blood reaches the intercostal arteries from collateral connections between branches of the subclavian arteries and the intercostals and by the anastomoses between its internal mammary and inferior epigastric arteries. In this way, there is a blood flow into the aorta distal to the stenosis.

Although the blood supply to the lower half of the body is reduced, parts seldom suffer from peripheral gangrene, although patients may complain of intermittent Claudication. The real danger of cocartion is due to its effects of hypertension which is often severe and likely to result in cerebral haemorrhage or left ventricular failure. (Blacksie 1964).

Clinically, its diagnosis is usually made upon account of hypertension in a child or young adult. The mechanism of production of hypertension is not simple blockage of the aorta, but is probably due to a large part to the relatively poor blood supply to the kidneys, with renal ischaemia, and release of hypertensive agents such as renin.

On AP chest x-ray, left ventricular hypertrophy and often notching of the ribs by the large intercostal collateral vessels is seen.

Two main types of cocartion are described according to the relationship of the ductus to its cocartion:

a) **Preductal** (Infantile). In this type, a patent ductus enters the aorta below the cocartion. Deoxygenated blood may pass from the pulmonary circulation into the aorta and hence to the lower limbs and extremities. These areas are cyanosed, whilst the head, neck, and upper extremities are pink — differential cyanosis. The stenotic aortic segment may be long and include one or more of the great vessels. 40% of patients have other cardiac lesions, and the mortality in the first year of life is high. (Hermann 1955).

b) **Postductal** (Adult). 60% of all cases are of this type. The ductus is patent in a minority and enters the aorta above the cocartion. Internally the stenosis is severe, and externally, the aorta shows characteristic widening.

The prognosis for both types of cocartion is poor, and untreated, very few patients survive beyond 30 yrs old. Most die from effects directly attributable to the hypertension, e.g. cardiovascular accidents, left ventricular failure.

**Treatment:**

Coarcted and Gross independantly in 1945 demonstrated that the stenosis could be excised and an end-to-end
anastomosis performed safely. This is now a well-accepted operation, for which the mortality is less than 5% (Sellers 1964). Occasionally, a graft is required to bridge a wide defect in cases with a long hypoplastic segment, or with a post-stenotic aneurysm. Aortic homografts were originally employed, but they calcify and narrow with age, and so have been superseded by grafts of drifted Teflon or Dacron. The operation is carried out through a left posterolateral incision through the 4th intercostal space. The large anastomotic vessels in the chest wall are ligated. The aorta is mobilised widely both above and below the constriction, great care being taken not to injure the large thin-walled intercostal and mediastinal vessels arising from the distal segment. The ligamentum arteriosum is divided. The aorta is clamped above and below the constriction, and the stenotic segment is then excised. An end to end anastomosis is performed using fine (10-0) suture material. The clamps are slowly released and any leak from the suture line is controlled by pressure.

The overall mortality for the operation is about 5-7%. In children, it is less than this (Sellers 64). In children, the results are excellent, with blood pressure returning to normal. In adults, some elevation in blood pressure usually persists, but the reduction is important, as symptoms are relieved and risks of serious or fatal complications reduced.

a). Post-stenotic structure in the left subclavian artery is the direct continuation of the aortic arch.

b). A short neck (arrowed) is present which permits cross-clamping without obstructing the flow to the left subclavian artery.

- Operation: resection followed by end to end anastomosis.

- Wedge of aorta excised at levels of the constriction and plastic repair is carried out. This is only applicable if there is adequate dilatation above or below the stenosis.

Fig. 8.
The best age for operation is 5-15 years, as the vessels are healthier, and local vascular complications are less common. In addition, the probability of obtaining normal post-operative blood pressures is greater. In a recent series of 114 such cases, the operative mortality was less than 1%. Morbidity risks include postoperative haemorrhage, subacute bacterial endocarditis, and a dissecting aortic aneurysm formation in the future.

Surgery has no place in the treatment of hypertension caused by toxemia of pregnancy.

Conclusion:

As can be seen from the above discussion, surgeons play an essential role in the treatment of arterial hypertension. Although at present it appears that, in the large number of cases of essential hypertension, surgery has little to offer, this situation is likely to change considerably as specific causes are found for essential hypertension.

In connection with this, the recent studies of Corn (1951) have indicated that up to 25% of patients with what has previously been labelled "essential hypertension" have aldosterone-producing adenomas of the adrenal cortex. If so, radical changes will soon be occurring in the treatment of this condition.

In cases of secondary hypertension, surgery frequently enables complete cure of the raised blood pressure. Conditions especially amenable to such treatment include renal artery stenosis, pheochromocytoma, coarctation, and Cushing's syndrome, and coarctation of the Aorta.

Thus, surgeons already play an important part in the treatment of arterial hypertension, and this role is likely to increase in the future.
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