

VI. EXAMINATION OF THE HYPERTENSIVE PATIENT

41. CLINICAL EXAMINATION OF THE HYPERTENSIVE PATIENT INCLUDING BLOOD PRESSURE MEASUREMENT

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I. INTRODUCTION

Until quite recently there has been a tendency to perform detailed investigations on all patients with hypertension. Despite a 20% prevalence of this condition, rising to 50% in the elderly [1], only a quarter of hypertensive subjects are on treatment [2]. As public awareness of the dangers of hypertension grows and screening programs are initiated, the numbers attending for assessment will increase. The incidence of secondary hypertension in such a screened population is small, about 2% [3] compared with 5–10% of a referred population [4], and as most of the newly diagnosed patients will have mild hypertension, the incidence of end-organ damage will also be less. Faced with a poor yield from these investigations and a rising population of people identified as hypertensive, detailed laboratory evaluation is therefore no longer feasible nor indeed indicated. We are now more dependent on clinical evaluation as a means of identifying patients who have secondary hypertension and end-organ damage. Only where clinical assessment suggests an underlying cause, or the patient is young with severe or progressive hypertension, should the patient be referred for detailed investigations. Otherwise, routine urinalysis, serum electrolytes and an electrocardiogram are adequate.

Hypertension can only be diagnosed by a sphygmomanometer. The technique gives an indirect assessment of the intra-arterial pressure and the results must be interpreted in the light of the many factors which affect this method. Further, the technique records blood pressure at one particular point in time. Blood pressure varies greatly and so attempts are being made to assess blood pressure behaviour by various methods, including home recording by the patient [5] and ambulatory measurement using automatic blood pressure recorders [6]. While these techniques make us less dependent on single measurements, in general the diagnosis and management of hypertension is based on a few measurements with the conventional sphygmomanometer. We must ensure, therefore, that our technique is accurate and that we avoid the many pitfalls which can affect the results of indirect blood pressure measurement. With increasing accuracy fewer people will be mislabelled as hypertensive and those with mild hypertension will be offered the benefits shown to result from treatment [7].

In this chapter we shall discuss the clinical evaluation of the hypertensive patient with emphasis on identifying possible secondary hypertension and end-organ effects of high blood pressure. We shall also describe in some detail the pitfalls in indirect blood pressure measurement and how they may be avoided.

2. HISTORY

2.1. Symptoms

Elevated blood pressure does not cause symptoms per se unless extremely high or complications are present. There have been descriptions of a characteristic headache, occipital in position, worse in the morning and relieved by rising. However, the incidence of headache is the same in normotensive and hypertensive people [8]. Further, headaches are more common in patients who know they have high blood pressure than in those who are not aware of it [9]. Despite this, the incidence of sustained hypertension among patients with headache in one study was 40% higher than in a normal population [8]. Raised intracranial pressure with stretching of pain sensitive structures is the most likely cause of the headache of malignant hypertension, which is generally worse in the morning and may be associated with vomiting. An association has been described between hypertension and migraine. Migraine sufferers have diastolic blood pressures which are, on average, 10 mm Hg higher than in the normal population [10]. Other symptoms which occur more commonly in hypertensive subjects are breathlessness and tinnitus. Epistaxis is frequently considered a symptom of hypertension, and although in one study there was a higher incidence of hypertension when no local cause was found for the epistaxis than when a nasal lesion was detected [11], it is more probable that epistaxis is a chance occurrence which brings the high blood pressure to the attention of the doctor. The frequent finding of epistaxis and hypertension in the same patient is due to the fact that both conditions are common. Many symptoms of hypertension result from end-organ damage. Angina, dyspnoea, intermittent claudication and cerebrovascular symptoms all indicate the presence of end-organ disease. Urinary symptoms are rare and, in patients with little other evidence of end-organ involvement, suggest primary renal or urinary tract disease.

2.2. Causative factors

A positive family history of raised blood pressure occurs more commonly in essential than secondary hypertension, though in some secondary causes,

such as multiple endocrine adenoma syndromes and polycystic renal disease, it can nearly always be elicited. A positive family history emphasises the importance of the disease to the patient and may encourage him to persuade family members to attend for screening.

Though there is still some doubt regarding the effectiveness of salt restriction in hypertension, recent evidence suggests that sodium restriction combined with increased potassium intake reduces blood pressure in mild hypertensives [12]. Patients with a high salt intake should therefore be advised to limit its use. A rough estimate of salt intake may be made by grading the patient according to whether he adds salt to his food, adds it only after tasting the food or adds it routinely to all food. A history of prescribed and non-prescribed medications that elevated blood pressure, such as steroids, sympathomimetics, carbonoxalone or other liquorice derivatives, and analgesics, which may damage the kidney, should be determined. Oral contraceptives are probably the most common cause of secondary hypertension [13]. The blood pressure rises in most women taking oestrogen preparations, in 4% of cases to hypertensive levels [14]. Other risk factors, such as smoking, alcohol intake and a history of diabetes should be noted. Patients should be questioned regarding symptoms which may give clues to other secondary causes of hypertension. Intermittent symptoms of sweating, palpitation and nervousness suggest a phaeochromocytoma, though anxious patients and those with the hyperkinetic heart syndrome may have similar complaints [15]. Weakness may result from hypokalaemia secondary to Conn's syndrome.

3. CLINICAL EXAMINATION

3.1. *General examination*

The general appearance of the hypertensive patient may suggest a possible cause or aggravating factor. There may be features indicating a poor life style such as obesity and nicotine staining of the fingers. Evidence of diabetes, such as glycosuria, fundal changes and peripheral vascular disease may be present. Xanthelesma and xanthomata suggest hyperlipidaemia. Cushing's syndrome, chronic renal failure and polycythaemia are usually self-evident. Skin lesions, such as neuromas and pigmented patches suggest neurofibromatosis which is occasionally associated with phaeochromocytoma and hypertension due to renal artery compression [4]. Ischaemic leg ulcers due to dermal end-artery occlusion are an uncommon complication of severe hypertension which resolves with treatment [16]. Sweating, tremor and nervousness may be due to anxiety or may indicate an underlying phaeochromocytoma.

Evidence of vascular disease suggested by bruits in large arteries, tortuous vessels, absent or reduced peripheral pulses and aneurysm of the abdominal aorta should be sought. A careful neurological examination may detect occult neurological signs due to a previous minor cerebrovascular accident.

Secondary causes of hypertension are detected in the majority of cases by careful physical examination and urinalysis [13]. Coarctation of the aorta is suggested by an ejection murmur at the left sternal border radiating under the left clavicle, in association with a delayed and reduced femoral pulse. Confirmatory signs include a palpable arterial pulsation and bruit over the medial scapular border. Abdominal examination may detect enlargement of one or both kidneys due to polycystic disease, hypernephroma or hydronephrosis. Dipstick urinalysis is in our view an important part of the physical examination. Routine culture and microscopy is not indicated unless the urinalysis is abnormal.

3.2. Cardiovascular assessment

3.2.2.1. The pulse

Examination of the pulse in hypertensive patients is generally unhelpful. Earlier writers emphasised the increased tension of the pulse in patients with strokes prior to there being any method of blood pressure measurement [17]. Palpation of the radial artery as a means of detecting arterial wall disease has often been stressed though it bears no relationship with pathological findings [18]. Atrial fibrillation and ectopic activity may be detected. Their occurrence probably reflects underlying ischaemic heart disease.

3.2.2. Taking the blood pressure

This forms the most important aspect of the evaluation of the hypertensive patient and is discussed in greater detail later. In general, an inflatable rubber bladder of suitable size enclosed in a cloth cuff is wrapped around the upper arm so that its lower edge lies above the antecubital fossa with the tubing facing upwards. The bladder is inflated rapidly to a pressure 30 mm Hg above the point of disappearance of the radial pulse. During slow deflation, at a rate not exceeding 2–3 mm Hg per pulse, the examiner auscultates over the brachial artery below the cuff edge. The appearance of the first Korotkoff sound corresponds to the systolic pressure, while both the point of muffling and disappearance (phases IV and V) should be recorded as the diastolic pressure. The time of day, the position of the patient, whether lying, standing or sitting, and whether or not the patient appears anxious or distressed are all factors which should be recorded. For example, the blood pressure might be recorded as 142/94/90 (lying, relaxed) [19].

3.2.3. *The apex*

A heaving apex beat is the most common sign of left ventricular hypertrophy. Mechanical methods of assessing the cardiac impulse have shown that in normal subjects the outward movement of the apex is short and does not extend into the latter one third of systole. In hypertensive patients with left ventricular hypertrophy, the outward deflection is prolonged, sometimes beyond the second sound. This is referred to as a heaving apex and is best appreciated by simultaneous auscultation and palpation of the apex beat with the patient supine [20]. The area occupied by the cardiac impulse may also be increased beyond the normal one rib space. The least sensitive sign of left ventricular hypertrophy is displacement of the apex relative to the midclavicular line. Factors which interfere with the proper assessment of the apex include obesity and emphysema. A cardiac aneurysm produces a prolonged and enlarged cardiac impulse. Although usually distinguishable by its position, above and medial to the apex beat, it may occupy the ventricular apex.

3.2.4. *Auscultation*

Much emphasis has been placed on the loudness of the second heart sound in hypertension. This may reflect a period when blood pressure measurement was of doubtful accuracy and a loud second sound was used as an aid to diagnosis [21]. Although quoted as being present in as many as 70% of hypertensive patients [23], there is no evidence that this sign gives any information as to the state of the heart or the patients prognosis. The origin of the second sound has been variously attributed to vibrations in a column of blood or myocardium after valve closure, sudden tension in a flaccid membrane and apposition of valve cusps. Part of the sound is due to closure of the pulmonary valve and opening of the atrioventricular valves [24]. More recently, in the laboratory study of porcine and human valves mounted in a transparent medium, the second heart sound has been shown to be the result of diastolic vibration of the valve cusps. The amplitude of the sound produced depends on the amplitude of the valve motion which is directly related to the rate of change of pressure across the aortic valve [25]. This in turn depends on the aortic diastolic pressure and the rate of diastolic isovolumic relaxation of the left ventricle. Therefore, in a hypertensive patient with a normal left ventricle, the rate of change of pressure across the valve in early diastole is high, producing a loud second heart sound. However, in heart failure where the rate of isovolumic relaxation is poor and there is a fall in the rate of pressure change across the valve, the amplitude of the second sound becomes diminished [26]. Rarely, paradoxical splitting of the second sound may occur in which the aortic component of the second sound is delayed maximally during expiration. It is more common when hypertension is complicated by left bundle branch block.

Another common auscultatory finding in hypertension is a presystolic or fourth heart sound, present in 50% of cases [23]. It correlates well with electrocardiographic P-wave abnormalities and is probably due to decreased left ventricular compliance. It may also be due to underlying myocardial ischaemia and is frequently first heard after beta blockade. A protodiastolic or third heart sound is heard in 30% of hypertensives, frequently in association with other signs of left ventricular failure or accelerated hypertension.

Systolic murmurs are common in hypertensive patients, present in up to 70% of cases [23]. They are usually ejection type murmurs best heard at the apex or base of the heart. Amyl nitrate inhalation increases their intensity though aortic valvular lesions are rarely found at post mortem. In contrast, the regurgitant murmurs of mitral incompetence are rare in hypertensive patients and are usually due to organic disease. Apical mid-diastolic murmurs are occasionally heard and must be distinguished from a presystolic or protodiastolic sound. They are most commonly due to organic mitral stenosis, a condition associated with an increased incidence of hypertension. Rarely they occur in the absence of organic disease and are probably the result of increased flow across the mitral valve. Finally, functional aortic regurgitant murmurs may occur in patients with severe hypertension [23, 27].

3.2.5. Renovascular hypertension

The presence of a renal artery bruit is the only sign of discriminatory value between renovascular hypertension and essential hypertension. In the Co-operative Study on Renovascular Hypertension, 12% of patients with renal artery stenosis and 1% of patients with essential hypertension had an abdominal bruit [28]. However, in other studies abdominal bruits were found in 88% of cases due to renal artery dysplasia and 36% of cases due to atherosclerosis [29]. Further, they have been detected in 18% of normal subjects [30]. Careful auscultation is as successful in the detection of abdominal bruits as phonarteriography [31]. The patient should be examined in a quiet room without undue compression of the abdominal wall by the stethoscope. Renal artery bruits are best heard above the umbilicus on or lateral to the midline. Occasionally, the murmur is heard well up into the epigastrium. It frequently radiates to one or other side, and this might help to lateralise the lesion, though it can be misleading. Higher pitched murmurs and those with a diastolic component are more significant, the latter predicting a successful surgical outcome in patients with renal artery dysplasia [32]. By far the commonest cause of abdominal bruit is coeliac artery stenosis. Other causes include stenosis of mesenteric, splenic and hepatic arteries, collateral vessels around complete vascular occlusions, aortic aneu-

rysm and vascular tumours. A history of worsening hypertension in a young person with an abdominal bruit suggests renovascular hypertension secondary to renal artery dysplasia. In older subjects, atherosclerosis is a more common cause and there is frequently widespread vascular disease [4].

3.2.6. Fundoscopy

Fundoscopy indicates that there is end-organ damage elsewhere [33] and are of prognostic significance in treated and untreated patients. Moreover, their resolution with treatment offers objective evidence of adequate control. Three disorders are manifested in the fundal changes of the hypertensive: hypertensive neuroretinopathy, arteriosclerosis and atherosclerosis [34]. Hypertensive neuroretinopathy presents as generalised arteriolar narrowing with later focal narrowing (grades I and II). This is difficult to assess as generalised narrowing is common, occurring in 33% of normotensive men aged 40–60 years [35] and attempts to derive an index of arteriolar narrowing have failed to distinguish normal and hypertensive subjects. Focal narrowing may also occur in the absence of hypertension. It has been postulated that narrowing of arterioles is an autoregulatory response to increased pressure. This compensatory mechanism may fail resulting in endothelial damage and arteriolar occlusion presenting as flame shaped haemorrhages and cotton wool exudates (grade III). At higher pressures, ischaemia of the optic nerve head results in papilloedema due to interruption of axoplasmic flow (grade IV). Grades III and IV hypertensive retinopathy occur in malignant hypertension.

Arteriosclerotic changes result in an increased light reflex from the arteriolar wall and venular compression by crossing arterioles. Though considered to reflect the duration rather than the degree of hypertension, the relationship of arteriosclerotic changes to hypertensive disease is unknown. An abnormal light reflex occurs in 30% of middle aged normotensive men [35] and venular compression may also be seen in the absence of hypertension. Further, these changes may resolve on treatment of hypertension casting doubt on their proposed underlying pathological cause.

Atherosclerotic changes of the central retinal artery may present as ischaemic neuropathy of the optic nerve and central retinal artery thrombosis. Retinal vein occlusion may occur due to an atherosclerotic plaque eroding into the vein from an adjacent artery as both lie within a single sheath [36]. In patients with transient ischaemic attacks embolic material may be seen at arteriolar bifurcations [34].

Resolution of vascular changes usually occurs with treatment. Haemorrhages and exudates may resolve over 2–10 weeks, the latter becoming granular and later hard and waxy in appearance before complete resolution. Papilloedema may take some months to resolve.

BLOOD PRESSURE MEASUREMENT

4.1. Equipment

Indirect blood pressure measurement is a valuable clinical technique frequently taken for granted. Students and nurses are poorly trained, equipment is badly maintained and attention to detail is often lacking. Further, failure to standardise the technique and changing recommendations from authoritative bodies have created confusion. These factors tend to make blood pressure measurement more inaccurate than need be.

The two devices most frequently used in blood pressure measurement are the mercury-in-glass and aneroid sphygmomanometers. Both aneroid and mercury manometers are accurate but the mercury manometer retains its accuracy longer and is easier to maintain [37]. Mercury may be lost from the reservoir of the mercury device so that the meniscus lies below zero with no pressure applied to the cuff. Dirty tubing and oxidised mercury may obscure the meniscus. A blocked air vent at the top of the mercury column may cause sluggish movement of the mercury during inflation and deflation and result in overestimation of blood pressure [38]. Calibration of each new device must be corrected for the fall of mercury in the reservoir as the mercury column rises so that the scale indicates the differences between the levels of mercury in the tube and in the reservoir. This error is small, 1 % or less, if the diameter of the reservoir is over ten times the diameter of the tube [39].

An aneroid manometer utilises a metal bellows which elongates when pressure is applied. A gear sector transmits the movement to an indicator needle. In some devices there is a stop at the zero position of the scale so that zero drift cannot be detected. The device should be tested regularly throughout the entire pressure range against a mercury-in-glass manometer by connecting both devices to a single cuff using a T-tube connector. In one study, 32 % of all hospital aneroid sphygmomanometers deviated from the limits laid down by the American National Bureau of Standards (± 3 mm Hg) [40]. Such deviations are less frequent where there is a policy of regular servicing. Aneroid manometers are more convenient to use but must be calibrated against a mercury standard every six months and returned to the manufacturers for servicing if inaccurate. Moreover, errors, and their causes, are less easily identified and corrected than with mercury sphygmomanometers.

Both devices are subject to leaks due to worn valve washers, old tubing or ruptured bladders. These can be detected by inflating a cuff to 250 mm Hg. With the valve closed, the leak rate should not exceed 2 mm Hg in 10 s after the system has been allowed to equilibrate [37]. By clamping the circuit in sections, the site of the leak will be identified. Difficulty in inflating the cuff

may be due to a blocked air vent within the valve apparatus. This is easily removed and cleaned.

4.2. Blood pressure measurement

An adult sized cuff should be placed snugly round the patients arm above the antecubital fossa. Ideally the bladder should completely encircle the arm and if it does not the bladder centre must be over the brachial artery [41]Ç. A loosely applied cuff acts as a narrow cuff and gives a falsely high measurement [42]. Inflation should be rapid to 30 mm Hg above the point of disappearance of the pulse. Slow inflation causes congestion of the arm resulting in attenuation of Korotkoff sounds and false elevation of the diastolic blood pressure [43]. Deflation should not exceed 3 mm Hg per pulse if underestimation of systolic blood pressure and overestimation of diastolic blood pressure are to be avoided. Tilting of the mercury column causes underestimation of blood pressure. The pressure should be read to the nearest 2 mm Hg from the top of the mercury meniscus which should be at eye level to avoid parallax error.

The patient should be in a resting position with the arm supported by the observer to prevent the elevation in blood pressure due to the isometric exercise of maintaining posture [44]. The arm should be at heart level to avoid the affect of hydrostatic pressure. In most subjects, the intra-arterial pressure is equal in both arms [43]. There may, however, be a difference of 10 mm Hg between arms when simultaneous indirect recordings are compared; this difference occurs more frequently when the pressures in the two arms are recorded sequentially because of the additional effect of physiological variation. Therefore, though a difference in pressure recordings between arms occurs in patients with widespread atherosclerosis, dissecting aortic aneurysm and coarctation of the aorta, it commonly occurs in patients with no vascular lesion. The pressure should be recorded in both arms initially and the higher measurement accepted as the true pressure. The arm with the highest pressure should be used for future measurement. In general, the pressure is highest in the right arm.

The frequency range of Korotkoff sounds in 40–120 cycles per second with maximum amplitude centred about 100 cycles/s. Upon muffling (Phase IV), the higher frequencies are attenuated and the maximum amplitude then centres about 60 cycles/s [46]. The diaphragm of the stethoscope is usually made of stiff linen bakelite designed to attenuate lower frequencies. The open chest piece, or bell, however, has a lower natural frequency and is four times more sensitive to low frequency sounds. The acoustic range of Korotkoff sounds is therefore better suited to the bell of the stethoscope. However, it is usually easier for nurses and trained technical staff to use the

diaphragm and stethoscopes used by nurses may not have a bell attachment. A single tubed stethoscope avoids extraneous noise caused by two tubes rubbing against each other and, by reducing the volume of the device increases the efficiency of sound transmission to the ear. The stethoscope head should be placed as near as possible to the point of compression of the artery, below the lower edge of the cuff.

4.3. Korotkoff sounds

In his thesis on indirect blood pressure measurement, Korotkoff described three phases in the arterial sounds heard during cuff deflation [47]. The appearance of the first sound corresponded to systolic pressure. This was followed by a louder murmur and finally disappearance of the sound which corresponded to the diastolic pressure. In the subsequent decade, five phases of Korotkoff sounds were described.

- Phase I – First appearance of a clear tapping sound.
- Phase II – Period during which a swishing quality is heard.
- Phase III – Period during which sounds become crisper.
- Phase IV – Period of abrupt muffling of the sounds.
- Phase V – Disappearance of sound.

Another phenomenon that may occur during cuff deflation is the auscultatory gap when the sounds disappear temporarily, reappearing at a lower pressure. If auscultation is not continued beyond this gap, a falsely high diastolic pressure will be recorded.

No one hypothesis for the production of Korotkoff sounds has found general acceptance. Theories have included a sudden localised change in the shape of the compressed artery, a water-hammer affect of the wave front striking a stationary column of blood below the cuff, fluid turbulence and resonance of air within the cuff. Studies using a Doppler technique [46] to differentiate arterial wall movement and fluid turbulence suggest that the Korotkoff sounds consist of a higher frequency component due to sudden arterial wall movement, and a lower frequency component due to fluid disturbance. At the point of muffling, the higher frequency component disappears. This point corresponds with the period in which the artery first remains open throughout the cardiac cycle, though the relationship is not consistent. Theoretically, therefore, this would appear to be the most logical end-point for diastole because in order for the artery to remain open throughout the cardiac cycle, cuff occlusive pressure must be lower than diastolic pressure. According to the analysis of Anliker [48], the production of Korotkoff sounds results from dynamic instability of the vessel wall induced by the cuff pressure. At this point oscillations induced in the arter-

ial wall by the wave front are amplified and are detected as Korotkoff sounds. The limits of cuff pressure which induce dynamic instability correspond to the systolic and diastolic auscultatory pressures. Outside these limits, above the systolic or below the diastolic pressure, no sounds are heard.

4.4. Diastolic dilemma

It is not surprising, therefore, that as the origin of the Korotkoff sounds is not satisfactorily explained, their relationship to intra-arterial pressure has been studied in detail. Many of these studies are not comparable because of differences in technique. Some authors have compared direct and indirect recordings from the same artery. However, it seems probable that an indwelling catheter would alter sound production from an artery. In many earlier studies, the transducer-catheter systems for intra-arterial pressure measurements were of doubtful accuracy and in some the frequency-amplitude characteristics were not given. This varies greatly depending on length and bore of tubing, transducer type and volume of transducer chamber [49]. In some studies, the direct radial pressure in one limb has been compared with indirect recordings from the opposite brachial artery. The systolic pressure in the radial artery, however, is about 6 mm Hg higher than in the brachial artery and there may be a difference in indirect pressure recordings between arms.

The sphygmomanometers used as standard for comparison with intra-arterial recordings have varied between studies and this may alter results. The London School of Hygiene Sphygmomanometer [50] has been used in two studies which favoured phase IV Korotkoff sounds as the diastolic end-point [51, 52]. We have found that this device tends to underestimate blood pressure for two reasons. Firstly, a calibration error in the device leads to an underrecording of 4 mm Hg at higher pressures. Secondly, there is a difference in decision end-points between this device and the standard method of blood pressure measurement. During deflation of the London School of Hygiene Sphygmomanometer, three mercury columns hidden from the observers view monitor cuff pressure. The fall of mercury in each column may be halted independently so that separate recordings for systolic and diastolic phase IV and V pressure are made. In order to ensure that the systolic end-point has been reached, the second Korotkoff sound must be taken as the systolic end-point. Similarly, the diastolic end-point is some variable pressure below the last sound, that point where a sound would be expected but fails to occur. Further, the London School of Hygiene Sphygmomanometer has been shown to give lower recordings of indirect blood pressure than other methods, including the standard mercury device [51, 53]. This may

explain why in these two studies indirect blood pressure measurement with the London School of Hygiene Sphygmomanometer was lower than direct intra-arterial measurement.

In general, most authors have found that phase V corresponds more closely with the intra-arterial diastolic pressure. The American Heart Association has variously recommended phase V [54] and phase IV [55] as the diastolic end-point, whereas the World Health Organisation recommends that both phase IV and phase V be recorded. There is greater agreement between observers using the silent rather than the muffled end-point, a matter of great importance in training observers. We recommend that phase V be taken as the diastolic pressure. To avoid confusion, however, we support the suggestion that the fourth and fifth phases should always be noted. In patients with a high velocity of blood flow, Korotkoff sounds may be heard right down to zero pressure. In such cases the fourth phase should be taken as the diastolic end-point.

Many factors influence the difference between the indirect and direct pressure recordings. The mercury or aneroid sphygmomanometer measures cuff pressure and it is assumed that this reflects intra-arterial pressure. In general, however, the indirect method tends to underestimate the intra-arterial systolic pressure and the fifth phase diastolic end-point tends to have a variable relationship to intra-arterial diastolic pressure. The relationship between indirect and direct blood pressure recording depends on the elasticity, wall thickness and radius of the artery under compression [56]. It might be expected, therefore, that in elderly subjects the pressure required to collapse the artery would be increased. This would lead to an overestimation of the true intra-arterial pressure by the indirect method in the elderly, a phenomenon referred to as 'pseudohypertension' [57].

4.5. Bladder size

Another potential source of error is the bladder size. If the inflatable bladder within the cuff is not matched for arm circumference indirect measurements become unreliable. The zone of effective pressure exerted by the bladder narrows in deeper tissues so that in an obese subjects the pressure within a standard bladder (12 cm × 23 cm) may not be fully exerted on the artery. Therefore, in order to compress an adequate length of brachial artery the bladder must be inflated to pressures higher than in non-obese subjects. Another possible explanation is that in obese subjects the bladder balloons into the soft tissues and therefore acts as an exceptionally narrow bladder [41]. Ideally the cuff bladder width should be 40% of the arm circumference [58]. Bladder width, however, is less critical if a bladder of sufficient length to completely encircle the arm is used and indirect recordings with

such a bladder correlate best with intra-arterial recordings [37]. We recommend that for adults the bladder should be at least 35 cm long and 12 cm wide. If the bladder fails to encircle the arm, the centre of the bladder, often marked on the cuff, must be placed directly over the artery to be compressed.

4.6. Variability of indirect blood pressure measurement

Repeated indirect blood pressure recordings have demonstrated that casual recording may bear little relationship to the patients overall blood pressure behaviour. Raftery [59] has shown the considerable variation in blood pressure over 24 h with direct intra-arterial pressure measurements. This may also be demonstrated using non-invasive technique for ambulatory blood pressure recording (Figure 1). These devices are providing useful especially in patients with borderline hypertension and in those in whom blood pres-

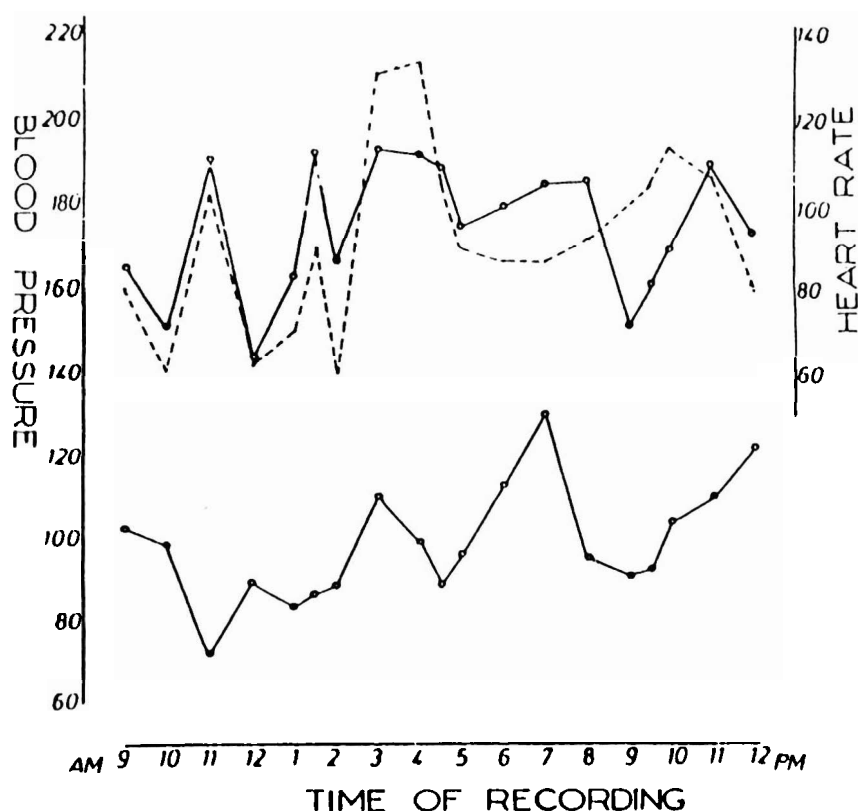


Figure 1. Ambulatory systolic and diastolic blood pressure (—) and heart rate (---) recordings over a sixteen-hour period (Remler M2,000 semiautomatic portable blood pressure recorder).

sure control is difficult. Blood pressure behaviour may also be assessed by home recording of blood pressure by the patient or a relative. This is a simple technique, requiring only a small amount of training and inexpensive equipment [5]. The initial recording of blood pressure tends to be higher especially in the clinical environment presumably due to anxiety. Over 50% of patients found to have high blood pressure at their first visit are normotensive on subsequent examination [2]. Further, repeated measurements on a single occasion may be highly variable, the first recording tending to be highest [60] with a gradual fall to basal levels after four recordings at ten-minute intervals. In practice, the average of three recordings, two at the start and one at the end of the examination, is considered adequate [61]. These should be repeated at one other visit before a decision is made on diagnosis or management. However, in cases of severe or accelerated hypertension, treatment may have to be started earlier.

Observer error accounts for some of the variation in blood pressure recordings and is high for medical staff [62]. This is partly due to a difference between observers which reflects inadequate training and the continuing difference in diastolic decision end-points. Nurses and doctors should be trained in blood pressure measurement using either a film or, more simply, a binaural stethoscope so that teacher and trainee are auscultating the same sounds [63]. A further component of observer variability is observer bias. This describes an unconscious tendency of raising or lowering a patient's blood pressure. For example, there might be observer bias in over-reading blood pressure so that a patient is included in a drug trial. Similarly, we may under-record blood pressure in a young healthy man and so not label him hypertensive. Digit preference describes the tendency to record blood pressures with figures ending in 0 or 5, such as 80 and 95. Adequate discussion during training makes the observer aware of such bias and minimises its affect. Special instruments have been designed to overcome this problem especially during research. The London School of Hygiene reduces both observer bias and digit preference but has disadvantages already referred to and is expensive and cumbersome. The Hawkesley random-zero sphygmomanometer is a mercury-in-glass sphygmomanometer which has the special feature of obscuring the zero point for each recording until after the systolic and diastolic end-points have been read. The results must then be corrected for the zero point which varies between 0 and 60 mm Hg [64]. It has the advantage of being not much larger than the standard mercury sphygmomanometer and is reasonably priced.

4.7. Blood pressure measurement in special situations

Blood pressure determination in children and infants is difficult because of the effect of anxiety, restlessness and crying, and in addition, it is more

difficult to auscultate the Korotkoff sounds. Further, blood pressure is labile and it is especially important to rely on repeated measurements for diagnosis. Proper cuff bladder size is essential for recording accurate pressure readings. Too large a cuff underestimates and too small a cuff overestimates blood pressure. In general, the cuff bladder should be wide enough to cover two-thirds of the upper arm and long enough to fully encircle it [65]. Cuff bladder widths range from 2.5 cm for infants to 9.5 cm for older children. Muffling of sound is recommended as the best index of diastolic pressure [66]. In 50% of children, only one diastolic auscultatory criterion occurs in that there is no discernable muffling before disappearance of the sounds. In neonates, poor sound production may exclude accurate recordings by auscultation and other techniques may be necessary. The Arteriosonde is a device which detects arterial wall movement by an ultrasonic method. It has been shown to give accurate results when compared with both other indirect methods and intra-arterial recordings [67]. Another device, the Infrasonde 3000, detects the low frequency sounds produced by the fluttering of the arterial wall when the pulse wave passes into the collapsed segment. Although outside the acoustic range of the human, this device reproduces these frequencies into an audiosignal. Despite its accuracy in recording systolic pressures, this device is inaccurate for diastolic recordings [67]. Other methods used in children include the flush method, which gives an estimate of the mean pressure [68], and oscillometry [69].

In cardiac dysrhythmias, such as atrial fibrillation, stroke volume, and therefore the blood pressure, varies depending on the preceding pulse interval. The point of onset and disappearance of the Korotkoff sounds vary so that the average of three recordings should be taken [37].

In clinical shock, brachial artery pulsations may be greatly diminished and the blood pressure is difficult to estimate though central pressure may be well maintained. Ultrasonic detection of arterial wall movement (Arteriosonde) or of blood flow (Accoson) and intra-arterial recordings are more accurate in this condition.

The blood pressure should be recorded in the thigh when coarctation of the aorta is suspected. A cuff containing a bladder 18 cm wide and long enough to fully encircle the thigh should be used. The cuff is wrapped around the thigh and the patient lays prone. The pressure is recorded by auscultating over the popliteal artery. Simultaneous intra-arterial femoral and brachial pressures are equal and the tendency to record higher formal systolic pressures by the indirect method reflects inadequate cuff size.

SUMMARY

With a rising population of patients identified as hypertensive through screening programmes, the importance of clinical as opposed to laboratory

investigations is becoming increasingly apparent. A clinical evaluation exploring the areas of secondary causes and end-organ disease combined with an evaluation of blood pressure behaviour will accurately diagnose hypertension in the majority of patients. Further, most cases of secondary hypertension will be identified and the number of laboratory investigations reduced. The inaccuracies of indirect blood pressure measurement are magnified by poor technique, inadequate training of staff, improperly maintained equipment and the continuing debate regarding which phase of the Korotkoff sounds should be taken as the diastolic end point. However, with attention to detail, this technique is accurate except in infancy, marked hypotension and possibly the elderly hypertensive.

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