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HYPERTENSION

A Case-based Approach



Elizabeth W Edwards
Donald J DiPette

Foreword
C Venkata S Ram

2nd Edition

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CHAPTER 2

Hypertensive Target Organ Damage: Mechanism and Recognition

Tatsuo Shimosawa

INTRODUCTION

It is well-recognized that hypertension is a major but modifiable cardiovascular risk factor. This enhanced cardiovascular risk disease burden is largely attributable to hypertension-related target organ damage. The major manifestations of target organ damage affect the cardiac, central nervous, renal, and peripheral arterial systems. There is a long-standing controversy regarding whether hypertension is a disease or an abnormal population-based biomarker. In general, reference values for biomarkers are defined as the mean or median ± two standard deviations. For example, a recent survey in Japan identified the reference value for blood pressure as 165/97 mm Hg[1] under the assumption that blood pressure follows a Gaussian distribution; however, the general consensus is that a threshold blood pressure 130/80 mm Hg or more is hypertension. This definition is based on the observation that the risk for coronary artery disease, heart failure, stroke, and renal failure increases substantially above this threshold. Further complicating this debate is that the blood pressure above which target organ damage substantially occurs varies from patient to patient.

The mosaic theory on the cause of primary hypertension by Dr IH Page[2] has been accepted since the 1950s. The mosaic theory proposes that primary hypertension does not have one unifying etiology; rather, it has a combination of a number of etiologies that can vary from individual to individual. Clinically, it is difficult to identify the cause or causes of primary hypertension (also defined as essential hypertension) in a single particular individual since genetic, hemodynamic, humoral, endocrine, neural, renal, environmental, dietary factors, and many other factors may be involved. This difficulty differs in individuals with secondary hypertension who often have a single etiology for elevated blood pressure. Regardless of whether hypertension is primary or secondary, the elevation of blood pressure increases the risk of target organ damage in a given individual.

In order to develop a clinical plan to prevent and potentially treat not only the elevated blood pressure but also the target organ damage from that elevated blood pressure, it is essential to recognize and monitor the potential target organs of the hypertensive process. Clearly, early in the treatment plan

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clinicians should screen for the presence of secondary hypertension by history, physical, and laboratory modalities if indicated.

This chapter presents a case of an elderly woman diagnosed with hypertension due to primary hyperaldosteronism from an adrenal adenoma and treated appropriately with adrenalectomy. Following the adrenalectomy, the patient followed up for an additional 15 years. This chapter focuses on the clinical and laboratory approach to the evaluation of hypertension-related organ and vascular damage.

CASE

A 69-year-old Asian woman presents with uncontrolled hypertension and a slight increase in serum creatinine from 1.0 to 1.2 mg/dL. Her physician initially diagnosed her with hypertension at the age of 45. Her blood pressure was controlled with a calcium channel blocker (CCB), a diuretic, and an angiotensin-converting enzyme (ACE) inhibitor (ACEi). At age 54, she was hospitalized for a myocardial infarction. After a successful percutaneous transluminal coronary angioplasty, she was then treated with a beta-blocker, an ACEi, and a CCB. At age 56, her outpatient physician noted an extremely high blood pressure (180–190/110 mm Hg) and admitted her to the hospital with hypokalemia, low plasma renin activity (PRA), and high plasma aldosterone concentration (PAC). Adrenal scintigraphy and bilateral adrenal venous sampling revealed a right adrenal adenoma and she was diagnosed with primary hyperaldosteronism.

Surgeons removed her right adrenal gland. After the adrenalectomy, her blood pressure remained controlled to a level of approximately 130/80 mm Hg with only a CCB and a beta-blocker. However, at the age of 69, her blood pressure had gradually increased to approximately 156/85 mm Hg. She presented to the outpatient clinic for a routine visit.

Physical Examination

Height: 162 cm
Weight: 56 kg
BMI: 21.3 kg/m^2

The temperature was 36.6°C, the blood pressure 169/101 mm Hg, the pulse 73 beats/min, the respiratory rate 18 breaths/min, and the oxygen saturation 99% while breathing ambient air. She was thin, in no distress, and appeared well. Her funduscopic exam showed focal narrowing and arteriovenous crossings (Keith Wagener II). She had no bruits over her carotid arteries. Her lungs were clear and there were no cardiac murmurs. She had no organomegaly. There was an abdominal aortic bruit after squatting. Her extremities had no edema. The reflexes were 2+ bilaterally and her sensation and strength were intact.

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Laboratory Data (Tables 2.1 to 2.5 and Fig. 2.1)

Table 2.1: Complete blood count.

	Age 54	*Age 56*	*Age 69*
White blood cell count (mm^3)	4,800	3,800	4,500
Hemoglobin (mg/dL)	12.7	13.4	12.8
Hematocrit (%)	39.9	39.7	38.9
Platelets (thousands)	201	189	199

Table 2.2: Chemistries.

	Age 54	*Age 56*	*Age 69*
Sodium (mEq/L)	140	139	142
Potassium (mEq/L)	4.2	4.4	4.0
Chloride (mEq/L)	102	105	101
Blood urea nitrogen (BUN) (mg/dL)	22	28	23
Creatinine (mg/dL)	0.98	1.01	1.28
Finger stick blood sugar (mg/dL)	104	108	102
Aspartate aminotransferase (AST) (U/L)	15	18	12
Lactate dehydrogenase (LDH) 267 (U/L)	240	237	267
Calcium (mg/dL)	9.4	9.3	9.4
Uric acid (mg/dL)	5.8	6.1	6.9
Hemoglobin A_1C (%)	6.1	6.0	6.0

Table 2.3: Cholesterol.

	Age 54	*Age 56*	*Age 69*
Total cholesterol (mg/dL)	240	246	245
Triglycerides (mg/dL)	135	108	124
HDL (mg/dL)	76	75	78

Table 2.4: Urinalysis.

	Age 54	*Age 56*	*Age 69*
pH			5.5
Protein	Negative	Equivocal	Positive
Glucose	Negative	Negative	Negative
Occult blood	Negative	Negative	Negative
Casts	Negative	Negative	Present, 1–10 granular casts per high powered field

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Table 2.5: Hormonal studies.

	Age 54	*Age 56*	*Age 69*
Plasma aldosterone	ND	240 pg/mL	100 pg/mL (normal value 30–159 pg/mL in supine position)
Plasma renin activity	ND	<0.1 ng/mL/hour	2.3 ng/mL/hour (normal value 0.2–2.3 ng/mL/hour in supine position)

Questions

1. What information is required to determine her target blood pressure?
2. How does hypertension damage end organs?
3. How do we evaluate for the presence of end-organ damage?

DETERMINATION OF TARGET BLOOD PRESSURE

The aim of blood pressure control is to prevent damage to end organs, namely the kidneys, heart, vasculature, and brain. Numerous observational studies and several interventional trials have shown that organ damage from hypertension confers greater risk of coronary disease, heart failure, stroke, and renal failure and therefore influence a particular patient's blood pressure target. Furthermore, because risks vary over time, close clinical follow-up is required to evaluate end organ damage.

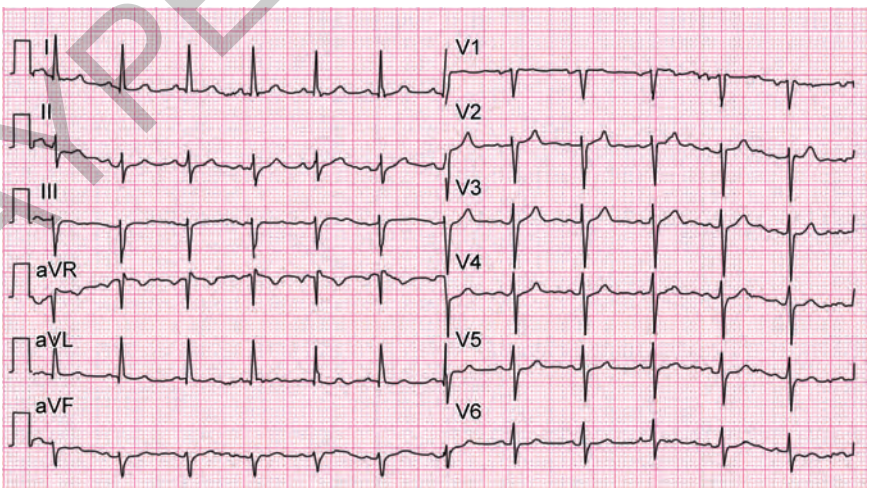

Figure 2.1: Electrocardiogram.

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Table 2.6: Risk factors for cardiovascular disease other than blood pressure.

Risk factor	*Indicator*
Advanced age	>65 years
Social habits	Smoking, excessive alcohol
Dyslipidemia	Low HDL, high LDL and TG
Obesity/metabolic syndrome	Visceral fat
Diabetes mellitus	Blood sugar
CKD	Proteinuria and eGFR
Past medical history	Stroke, ischemic heart disease, LVH, heart failure, peripheral arterial disease
Family history	Juvenile CVD (<49 years)

Specifically, clinicians should evaluate for the presence of obesity, especially abdominal obesity as in the metabolic syndrome; diabetes, hypercholesterolemia, and chronic kidney disease (CKD) (Table 2.6).[3] In this case, the patient is not obese as her BMI is 21.3 kg/m^2 and she is described as thin. When evaluating a patient's hemoglobin A_1C to determine the presence of diabetes, we must ensure the patient is not anemic and does not have an abnormal hemoglobin mutation such as that seen in sickle cell disease as these conditions influence the hemoglobin A_1C. Genetic mutations in the globin genes result in structurally altered hemoglobin chains and lead to diseases such as sickle cell disease with hemoglobin S and thalassemia with impaired production of globin chains. In this case, she is not anemic and her fasting glucose levels and hemoglobin A_1C are within normal limits. She is diagnosed as free from diabetes. Low-density lipoprotein (LDL) cholesterol is a well-known risk factor for cardiovascular disease; however, its measurement is not standardized. Therefore, clinicians should calculate the LDL cholesterol level with the formula:

LDL cholesterol = Total cholesterol – HDL cholesterol – (Total triglycerides/5).

In this case, LDL cholesterol = 245 – 78 – 124/5 = 142.2. Note that this formula does not apply when the triglyceride level is above 400 mg/dL. An LDL cholesterol of 142 does not necessarily lower the target blood pressure. Finally, the presence of CKD affects the target blood pressure. Evaluation of renal function should include not only serum creatinine and glomerular filtration rate (GFR) but also urinary protein and sediment. In this case, the patient had granular casts and the spot urine albumin-to-creatinine ratio was 106 mg/g.

According to Kidney Disease: Improving Global Outcomes (KDIGO) guidelines, a GFR <60 mL/min/1.73 m^2 (GFR categories G3a-G5) for at least three months or the presence of markers of kidney damage such as proteinuria

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and abnormal urinary sediment indicates CKD. A GFR of 60 mL/min/1.73 m^2 is less than half the normal value in young adults of approximately 125 mL/min/1.73 m^2 (see further for calculation formula for eGFR).

This patient's GFR has been <60 mL/min/1.73 m^2 since at least age 54 and she has casts and proteinuria; therefore, it is safe to diagnose her with moderate CKD.

This patient has no obesity, diabetes, or hyperlipidemia, but she does have CKD and therefore her target blood pressure is 130/80 mm Hg.

MECHANISM OF ORGAN INJURY FROM HYPERTENSION AND EVALUATION OF END-ORGAN DAMAGE

The organs of interest in hypertensive patients are the kidneys, the heart, the blood vessels, and the brain. Those organs are also targets for damage from diabetes, hypercholesterolemia, and autoimmune diseases. We have several tools to evaluate the morphological changes and function of these organs but these tools do not always tell us the cause of damage.

Evaluation of End-organ Damage (Table 2.7)[3]

Renal Damage

Mechanism of injury: Systemic hypertension damages the kidneys in several different ways. In addition to increased intraglomerular pressure, hypertension may lead to interstitial ischemia and oxidative stress, both of which could damage tubular cells. Furthermore, an increase in angiotensin II may induce cellular proliferation and further augment oxidative stress that damages mesangial cells, podocytes, and tubular cells. High dietary salt intake has also been shown to induce inflammation and oxidative stress as well as activate mineralocorticoid receptors, which may lead to further damage of several components of the kidney cells.[4]

Evaluation of damage: As mentioned earlier, routine clinical evaluation of kidney damage is via measurement of serum creatinine over time; estimation or direct determination of GFR; and examination of the urine including the presence of protein, albumin, and sediment. Urinalysis is cost effective, quick to respond to therapy, and has a high prognostic value. In contrast, the serum creatinine level is slow to respond to therapy.

Both glomerular and tubular damages result in urinary protein excretion. The glomerulus filters protein; the tubules then resorb most of the filtered protein. When the glomeruli are damaged there is increased filtration of protein; when the tubules are damaged, this filtered protein is not fully resorbed, resulting in enhanced proteinuria. Active urinary sediment suggests pathological damage in the glomeruli and/or tubules. Dysmorphic red blood cells

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Table 2.7: Tests for evaluating organ damage.

Potential methods of evaluation	*Indicators of organ damage*
Blood tests	
• Fasting blood glucose	Elevations
• Complete blood count	Anemia
• Lipid profile	Elevations of LDL and/or TG
• Serum creatinine with eGFR	CKD
• Serum sodium and potassium	Elevated sodium, decreased potassium
Urinalysis	Proteinuria, glucosuria, urinary sediment
Chest X-ray	Enlarged cardiothoracic ratio
Electrocardiography	LVH
Cardiac ultrasound	LVH, altered cardiac or valvular function
Cognitive function test, depression assessment, MRI, fundoscopy	Cognitive dysfunction or depression, stroke, retinal damage
Carotid ultrasonography, ankle brachial index (ABI)	Blood vessel damage
Tilt table test	Autonomic nervous system dysfunction
Specific examinations by specialists	Diagnostic tests for secondary hypertension Coronary CT

imply bleeding in the glomerulus. Cellular casts suggest inflammation in the kidney. Characteristic round cells point to a poor prognosis of renal function.[5]

Measurement of serum creatinine over time: The measurement of serum creatinine is essential to estimate GFR. Serum creatinine is proportional to muscle mass and therefore, in the elderly or in thin females, may be unexpectedly low and not reflect true glomerular function. In specific circumstances when estimated GFR (eGFR) based on serum creatinine may be inaccurate, cystatin C is an option. Most nucleated cells produce cystatin C and its rate of production is relatively constant and not affected by changes in muscle mass or diet, although this concept is not proven.

In addition to monitoring serum creatinine chronically, acute increases in creatinine are important in that they reflect acute kidney injury. In this case, the patient's PRA is inappropriately high despite treatment with a beta-blocker since blockade of the beta-1 receptors suppress renin secretion. These data suggest potential renal ischemia, possibly due to atherosclerotic bilateral renal artery stenosis.

Estimation of GFR: The most widely used formulas for calculating eGFR in persons aged 18 and over are the modification of diet in renal disease (MDRD) study equation and the chronic kidney disease epidemiology collaboration (CKD-EPI). Both the MDRD study and CKD-EPI equations include variables

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for age, gender, and race (Black vs. White) and both equations are equally accurate with eGFRs below 60 mL/min/1.73 m^2. However, racial and ethnic populations other than Blacks are underrepresented in the development or validation cohorts. In this case, the patient is a 69-year-old Asian female.

Using the MDRD study equation

$$eGFR = 175 \times (S_{cr})^{-1.154} \times (age)^{-0.203} \times (0.742, \text{if female}) \times (1.212, \text{if African American})$$

where:

S_{cr} is serum creatinine in mg/dL.
eGFR = 41

Using the CKD-EPI equation

$$eGFR = 141 \times \min(S_{cr}/\kappa, 1)^{\alpha} \times \max(S_{cr}/\kappa, 1)^{-1.209} \times 0.993^{Age} \times 1.018 \text{ (if female)} \times 1.159 \text{ (if Black)}$$

where:

S_{cr} is serum creatinine in mg/dL,
κ is 0.7 for females and 0.9 for males,
α is –0.329 for females and –0.411 for males,
min indicates the minimum of S_{cr}/κ or 1, and
max indicates the maximum of S_{cr}/κ or 1.
eGFR = 43

The online calculator is available from https://www.niddk.nih.gov/health-information/health-communication-programs/nkdep/lab-evaluation/gfr-calculators/Pages/gfr-calculators.aspx.

Therefore, her eGFR is 41–43 mL/min/1.73 m^2.

Examination of the urine including protein, albumin, and sediment: In this case, proteinuria is present; for further evaluation, urine protein should be quantified. Determination of the degree of protein excretion is a central part of the evaluation of patients with CKD. However, because the measurement of urinary albumin is not standardized, special caution must be paid.[6] The gold standard for quantification of urine protein excretion is a 24-hour urine collection. However, this is a cumbersome test for patients and prone to error. Therefore, KDIGO supports using a spot urine protein-to-creatinine ratio to quantitate proteinuria. In this case, the spot urine albumin-to-creatinine was 106 mg/g creatinine, which is abnormal and indicates microalbuminuria.

Based on the eGFR and urinary albumin level, she has G3aA2, moderate CKD (Table 2.8).

Cardiac Damage

Mechanism of injury: Hypertensive cardiac damage is life-threatening and prevention of both ischemic heart disease and heart failure is important. Cardiac hypertrophy is one of the most prevalent causes of ischemia and heart

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Table 2.8: Frequency of monitoring (number of times per year) by GFR and albuminuria category.

				Persistent albuminuria categories (Description and range)		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
GFR categories (mL/min/ 1.73 m Description and range)	G1	Normal or high	≥90			
	G2	Mildly decreased	60–89			
	G3a	Mildly to moderately decreased	45–59			
	G3b	Moderately to severely decreased	30–44			
	G4	Severely decreased	15–29			
	G5	Kidney failure	<15			

The numbers in the boxes are a guide to the frequency of monitoring (number of times per year).
Source: http://kdigo.org/guidelines/ckd-evaluation-and-management

failure in hypertensive individuals. In a hypertensive individual, the heart must overcome greater afterload. It increases cross-bridge formation according to the Frank-Starling mechanism, augments muscle mass to bear the extra load, and recruits neurohormonal mechanisms to increase contractility. The increase in mass is due to the hypertrophy of cardiac myocytes rather than hyperplasia and the parallel addition of sarcomeres causes an increase in myocyte width, which in turn increases wall thickness. This remodeling results in concentric hypertrophy. According to LaPlace's Law, the load on the myocardium = (pressure × radius)/(2 × wall thickness). Therefore, an increase in wall thickness can reduce the load on the myocardium, which helps maintain a normal ejection fraction. Although this type of remodeling rescues cardiac failure in the acute phase, long-standing hypertrophy can result in complex changes in gene reprogramming. Among them genetic reprogramming that modifies intracellular ion homeostasis such as downregulation of sarcoplasmic reticulum calcium ATPase (SERCA-2), variable upregulation of the Na^+/Ca^{2+} exchanger, downregulation of beta-1 adrenergic and M2 muscarinic receptors, and increases in the ratio of angiotensin II receptor subtypes (AT2 to AT1) account for cardiac dysfunction. In addition

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to changes in cardiomyocytes, increases in connective tissue induced by angiotensin II, aldosterone, or other humoral factors also play a role in the deterioration of cardiac function.

Evaluation of damage: The chest X-ray (CXR) and electrocardiograph (ECG) are often screening tests for hypertension-induced cardiac damage including hypertrophy.

LVH: Chest X-ray is helpful to see enlargement of the heart but does not necessarily reflect hypertrophy. The routine examination of the ECG is more accurate to assess for the presences of left ventricular hypertrophy (LVH). There are several sets of criteria used to diagnose cardiac hypertrophy by ECG. None is perfect but the Cornell voltage criteria and Cornell product are widely used in clinical settings and clinical trials as they have higher sensitivity for LVH than the Sokolow-Lyon criteria.[7,8]

The Cornell product is calculated as follows:

$$[\text{S in } V_3 + \text{R in aVL, with 0.80 cm added for women}] \times \text{QRS}$$

Left ventricular hypertrophy is defined as > 2,440 mm × msec

In this case, the Cornell product is (1.2 + 1.3 + 0.80) × 80 = 2,640 mm × msec and she is diagnosed with LVH.

To confirm cardiac hypertrophy, ultrasound is essential to measure left ventricular mass (LVM) and evaluate cardiac function. Left ventricular mass is calculated as follows:

$$\text{LVM (g)} = 0.8 \times \{1.04 \times [(\text{LVDD} + \text{LVPWT} + \text{IVST})^3 - \text{LVDD}^3]\}$$

where:

LVDD is the internal diameter of the left ventricle at end-diastole,
LVPWT is the thickness of the left ventricular posterior wall, and
IVST is the intraventricular septal thickness.[9]

Left ventricular hypertrophy is defined as an LVM adjusted for body surface (LVM index, LVMI) of >115 g/m^2 in men and >95 g/m^2 in women (http://www.csecho.ca/wp-content/themes/twentyeleven-csecho/cardiomath/?eqnHD=echo&eqnDisp=lvmlvmi). Increasing LVM is associated with increasing relative risk for cardiovascular disease as well as all-cause mortality. Concentric hypertrophy which is evaluated by (IVST + LVPWT)/LVDD >0.42 suggests a poor prognosis.[10]

Function: In addition to hypertrophy, cardiac function is routinely evaluated by Doppler ultrasound (normal sample is shown in Figures 2.2A to C). In most cases in hypertensive individuals, diastolic dysfunction manifests earlier than systolic dysfunction. Diastolic dysfunction usually results from impaired left ventricular relaxation and increased left ventricular stiffness, which increases filling pressure. The Doppler echocardiogram measures the ratio of early to late diastolic transmitral valve flow velocity (E/A ratio) to assess diastolic

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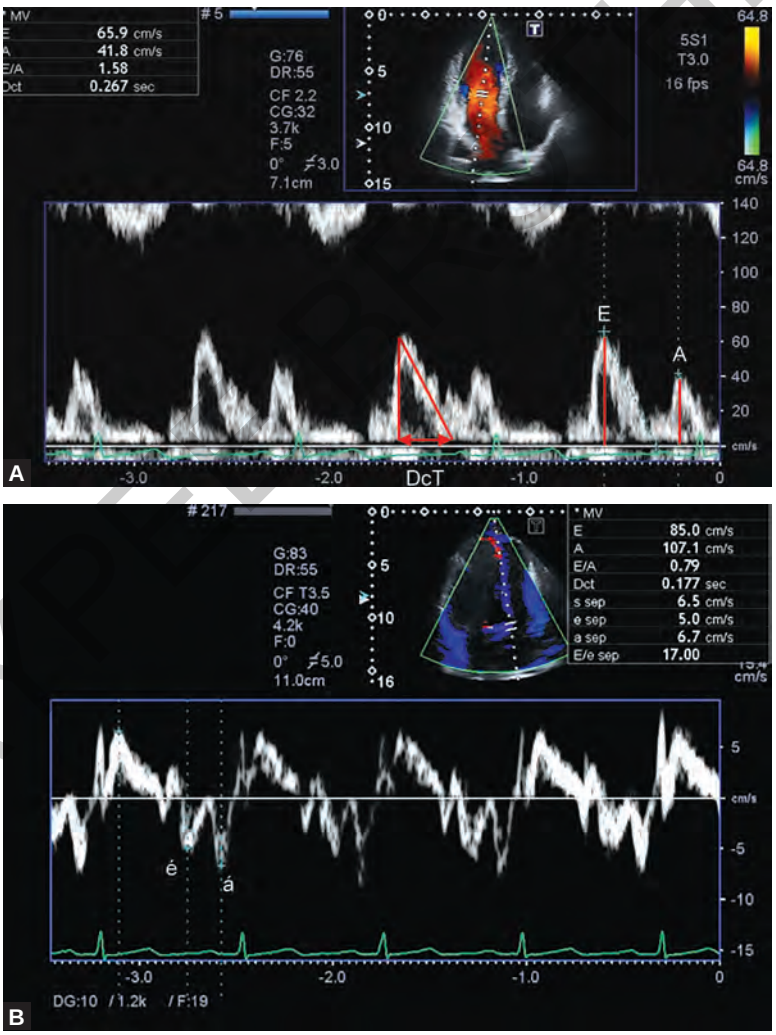
function. In a healthy heart, the E velocity is greater than the A velocity so a normal E/A ratio is one to two. When the left ventricular wall stiffness and filling pressure increases, early transmitral valve flow velocity (E) decreases and left ventricular filling relies more on the atrial contraction (A), lowering the E/A ratio. The reversal of the E/A ratio, when the A velocity exceeds the E velocity, is a marker of diastolic dysfunction.

The Doppler echocardiogram can also measure peak velocities at the mitral annulus, both medially and laterally and designated as é. The E/é ratio is the ratio of E to early diastolic mitral annular tissue velocity at the annulus and estimates left ventricular filling pressures. In normal hearts the E/é ratio is <8. In the presence of elevated filling pressure, E increases; with impaired



Figures 2.2A and B

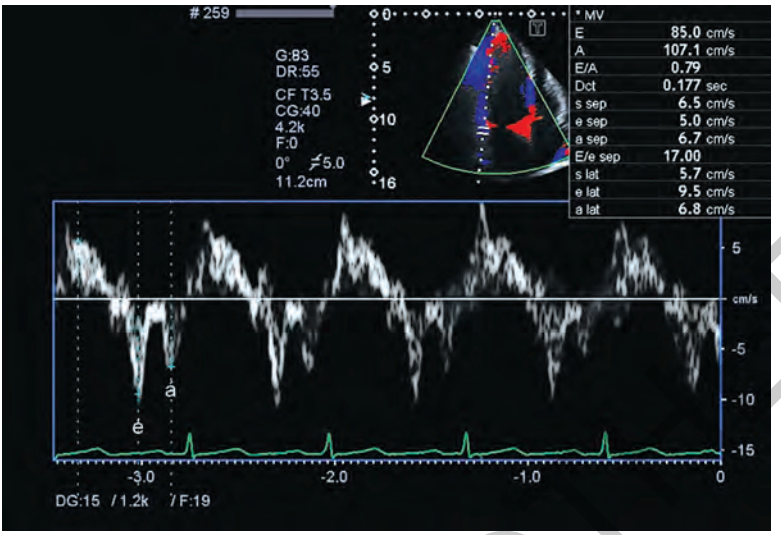


Figure 2.2C

Figures 2.2A to C: Echocardiogram in the evaluation of diastolic function (from normal cases). (A) Mitral valve blood flow; (B) Tissue Doppler, septal; and (C) Tissue Doppler, lateral. The Doppler echocardiogram measures the ratio of early to late diastolic transmitral valve flow velocity (E/A ratio) to assess diastolic function. In tissue Doppler, septal and lateral wall movements are measured to calculate é. The E/é ratio increases in the presence of diastolic dysfunction.

relaxation é decreases. Therefore, the E/é ratio increases in the presence of diastolic dysfunction. Systolic function is mainly evaluated by the percent ejection fraction and fractional shortening.

Multiple modalities exist to evaluate for the presence or absence of cardiac ischemia including resting ECG, cardiac stress testing, and cardiac catheterization when indicated. Recently, coronary computed tomography (CT) and magnetic resonance imaging (MRI) have been used in patients with suspected angina or those at high risk to delineate the coronary microvasculature in place of cardiac catheterization.

In this case,

LVDD = 51 mm

IVST = 11 mm

LVPWT = 11 mm

EF = 70%

E/A = 0.6

Therefore, LVM is 214 g and LVMI is 135 g/m^2 (height 162 cm, weight 56 kg) (Table 2.9).

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It is one of the most practical books to date on the management of hypertension in the community. The book is unique as it employs a case-based format and approach to address the prevention, diagnosis, treatment (both pharmacologic and nonpharmacologic), and control of hypertension, as well as its heavy cardiovascular disease consequences.

Physicians (primary care and subspecialists), healthcare providers (nursing, physician assistants, pharmacists, nutritionists, and social service professionals), and other healthcare professionals in postgraduate and undergraduate training will find the information contained in this book immediately applicable to their clinical practice and educational training in this important and growing field. Not only is this book a fountain of medical knowledge but the principles covered therein have immense practical utility to clinicians all over the world.

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