Modern Cardiology

John D. Cantwell, M.D.

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Director, Preventive Cardiology Clinic, Atlanta;
Co-Director, Cardiac Rehabilitation, Georgia
Baptist Hospital, Atlanta; Clinical Assistant
Professor of Medicine, Emory University

The Butterworth Group

United States
Butterworth (Publishers) Inc
19 Cummings Park
Woburn, MA 01801

Canada
Butterworth & Co. (Canada) Ltd.
2265 Midland Avenue
Scarborough, Ontario M1P 4S1

England
Butterworth & Co. (Publishers) Ltd.
88 Kingsway
London WC2B 6AB

New Zealand
Butterworths of New Zealand Ltd.
26–28 Waring Taylor Street
Wellington 1

Australia
Butterworth Pty Ltd
586 Pacific Highway
Chatswood, NSW 2067

South Africa
Butterworth & Co. (South Africa)
(Pty) Ltd.
152–154 Gale Street, Durban

Graphic Design: Audrey Sichel
Printed in the United States of America.

Cataloging in Publication Data

Library of Congress Cataloging in Publication Data
Cantwell, John D
Modern cardiology.

Includes bibliographical references and index.
1. Cardiology. 2. Cardiovascular system—Diseases. I. Title. [DNLM: 1. Cardiovascular diseases. WG100 C234m]
RC681.C175 616.1'2 77-22824
ISBN 0-409-95007-6

Preface

This is not a textbook of cardiology, nor was it meant to be. Rather, it is a practicing cardiologist's attempt to gather recent concepts and data in the rapidly expanding field of cardiovascular disease as a means of keeping abreast of the specialty and of organizing this information so it can be shared with the reader. It was a large task, but also a pleasant one. I will accept whatever success this book achieves with humility for, like Socrates, I am well aware of how little I really know.

I have had the opportunity to learn cardiology under outstanding physicians such as Eugene Braunwald and J. Willis Hurst. From Dr. Braunwald I learned the vast capabilities of the human intellect although I do not profess to have but a fraction of his. I also learned to look for ways to apply recent discoveries and observations in a personal, practical sense. From Dr. Hurst I learned the problem-oriented approach to clinical medicine, the mental discipline of "touching all the bases" in diagnostic possibilities and therapeutic considerations, and the necessity of making the most out of available data — no more or no less than is justified. Dr. Gerald Fletcher has been a constant source of inspiration and encouragement throughout my career in cardiology. To him I am grateful for the example of compassion and understanding, both to patients and to members of allied health fields.

JoAnn Bryant did an excellent job deciphering my handwritten scrawl and typing the manuscript. Her efforts are greatly appreciated. Martha Tarrant and Bob Beveridge did most of the illustrations and photography, both of which are of high quality. Dr. Fred Rose provided figures for chapter 24.

I have attempted to write this book without neglecting my wife and three children in the process, for their love and companionship "lights my fire."

Introduction

This book was written primarily for the physician engaged in the private practice of internal medicine and cardiology. As a cardiologist who divides his time between a solo practice and teaching duties in a medical center, I can appreciate how difficult it is for the practitioner to keep abreast of the rapidly changing concepts in clinical cardiology. *Modern Cardiology* provides an up-to-date analysis of most clinical problems that arise in the field of cardiology. Over 90% of the references are concerned with information that has been published since 1970.

This book is not an encyclopedia of cardiology, but rather a source of current information for the physician who deals with common problems such as coronary disease, valvular heart disease, hypertension, and pulmonary embolism. As such, it should be useful to all physicians in the field whether they are house officers or members of the university staff.

The book begins appropriately at the patient's bedside, where new concepts in physical diagnosis are considered. Since ischemic heart disease is by far the number one cause of death in this country, five chapters deal with its etiology, medical and surgical therapy, complications, and variations. Practical aspects of endomyocardial disease and valvular disease are considered, with emphasis on advances in the understanding of etiology and classification and featuring new delineation of diverse entities such as mitral valve prolapse and asymmetric septal hypertrophy.

The proper use of new and old cardiovascular drugs, ranging from digitalis to vasodilators and agents used in cardiac emergencies, occupies several chapters. The practitioner is updated as to the value of serum drug levels in guiding antiarrhythmic therapy.

The sections on laboratory techniques, such as echocardiography and exercise stress testing, should help the practicing physician to decide when such tests are indicated for his patients. I have not gone into basic elec-

trocardiography or radiographic and cardiac catheterization techniques, as these topics are vast and well covered in separate texts.

Under "Miscellaneous Cardiac Topics," I have included several additional concepts that are important to the clinician: a practical approach to preventive cardiology, an understanding of the ocular manifestations of cardiovascular disease, and a consideration of disease and pseudodisease that may be recognized in the schoolboy or professional athlete. Familial cardiac dysrhythmias will interest some readers more than others. The chapter on cardiovascular problems in airline pilots emphasizes how the step-wise use of clinical and laboratory techniques can resolve most dilemmas in this patient population. For the physician involved in clinical medicine this book should serve as a current reference for commonly encountered problems in cardiology, from midsystolic click to a case of progressive angina pectoris.

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Contents

New Concepts in Physical Diagnosis

In an era of computers and electronic gadgetry, it is refreshing to the bedside clinician to reflect upon recent advances and evolving concepts in the physical diagnosis of the cardiovascular system. These advances and concepts will be reviewed using categories of inspection, palpation, percussion, and auscultation. Additional remarks will cover the assessment of cardiac function at the bedside of patients with acute myocardial infarction, heart failure, and cardiac dysrhythmias.

INSPECTION

The importance of inspection is evident in the picture of the master clinician, Sir William Osler, poised at the bedside, intently observing the patient. Clues to clinical diagnosis can be everywhere — the furrowed brow of neurocirculatory asthenia, the Roth spot of endocarditis, and the unusual thumb of Holt-Oram Syndrome (Fig 1–1). Inspection of the precordium may reveal the systolic bulge of an acute myocardial infarction or the sternoclavicular pulsation of a dissecting aortic aneurysm. This section is primarily concerned with one important area of inspection: observation of the jugular venous waveform.

Although the author was privileged to receive medical training in some of the cardiovascular meccas of America, it was not until the third year in private practice that the correct examination of the venous pulses was mastered. The patient's head should be elevated to 45 degrees or to the height at which venous pulsations are the most obvious. The head should not rest on a pillow. One should concentrate on the internal jugular pulses, ignoring for the moment the external jugulars. (The latter show delayed appearance of waveform because of narrow vessel diameter and can be otherwise altered by the presence of venous valves which are not found in the internal jugular system.)

With the patient's head at a 45-degree angle, one locates the sternal angle of Louis and projects an imagi-

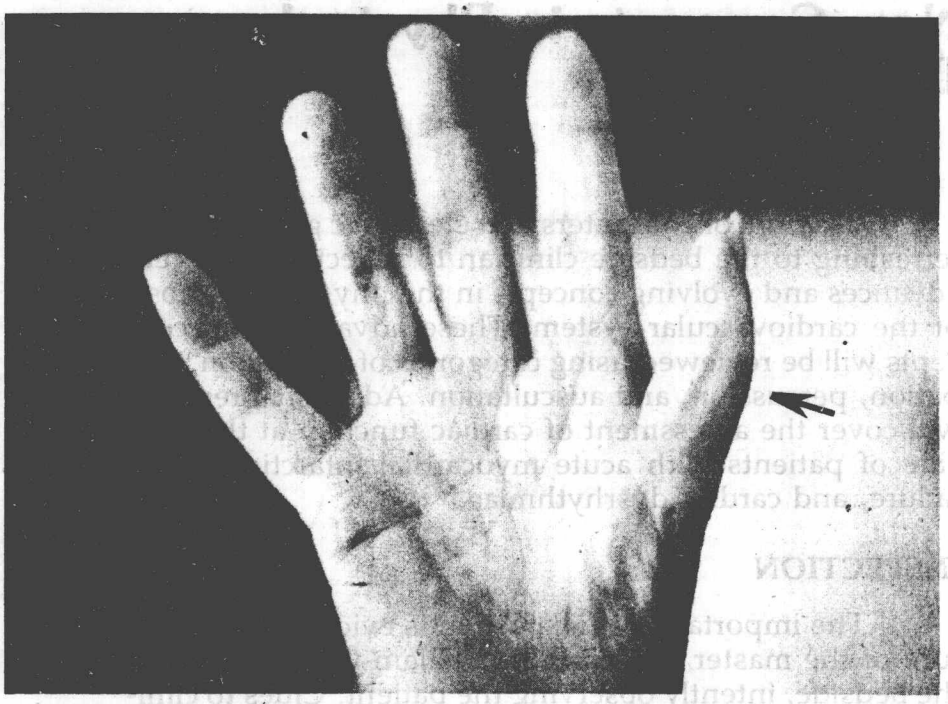

Figure 1–1 The thumb of a patient with the Holt-Oram syndrome resembles a finger. The underlying cardiac disorder is an atrial septal defect.

nary line from the angle to the neck, keeping the line parallel to the floor or flat part of the bed. The midright atrium is located an average of 7.5 cm below this line, and this distance is added to the distance that the internal jugular venous column extends above the line. For example, if the top part of the venous column extends 4 cm above the projected line from the sternal angle of Louis, the venous pressure is 4 cm plus 7.5 cm or 11.5 cm. Normally, the venous column should not extend more than 4 cm above the sternal angle. Three of the positive venous waves — the A, C, and V components — are well known (Fig 1–2). The fourth positive wave — the H wave — is less well appreciated. The negative waves include the X and the Y descents with the X descent having two components, X and X'. According to Constant, the mechanisms of these components, are as follows:[1]

A wave — caused by right atrial contraction.

C wave — thought to reflect bulging of the tricuspid valve upon closure into the right atrium; MacKenzie attributed it to carotid pulse artifact as well.

V wave — reflects atrial filling from the venae cavae with a closed tricuspid valve.

H wave — late diastolic filling of the right atrium, perhaps due to a sphincterlike contraction of the superior vena cava.

X descent — due to atrial relaxation.

X′ descent — due also to continued atrial relaxation plus downward movement of the tricuspid valve during right ventricular contraction.

Y descent — due to rapid flow of blood across the opened tricuspid valve into the right ventricle.

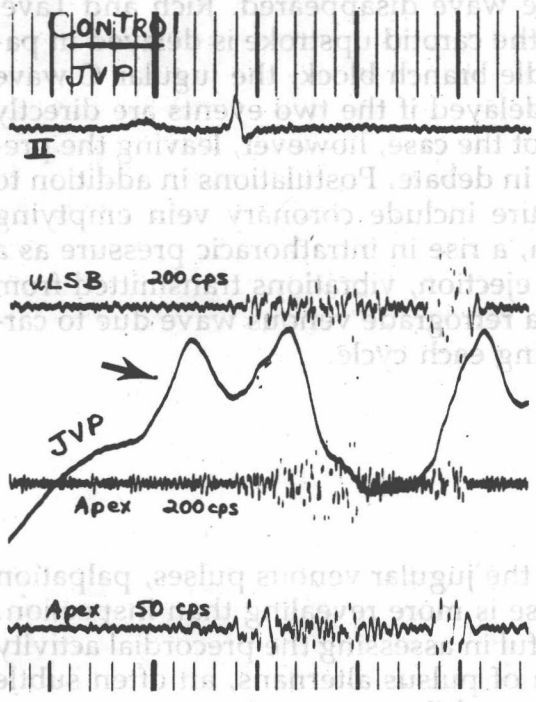

Figure 1–2 Recording of a jugular venous pulse, showing A, C, and V waves with negative X and Y descents.

Reflecting upon whether the X descent represents ventricular contraction, atrial relaxation, or both, Cheng observed a patient in which these two events were separated during periods of complete atrioventricular dissociation.[2] He concluded that the X descent did not depend upon ventricular systole (with its downward pull on the tricuspid valve) but rather seemed related to atrial relaxation. Constant believes that it is important to separate the X descent from the X′ descent, indicating that the latter seems related to ventricular systole since it was present in atrial fibrillation (which lacks the "atrial relaxation" phase).[3] It may be that the X descent corresponds to atrial relaxation while the X′ descent corresponds to ventricular systole (during which the tricuspid valve and cardiac base move downward).

As stated earlier, Sir James MacKenzie believed that the jugular venous C wave represented carotid artifact, for when he dissected the carotid artery away from the jugular vein, the wave disappeared. Rich and Tavel reasoned that since the carotid upstroke is delayed in patients with left bundle branch block, the jugular C wave should likewise be delayed if the two events are directly related.[4] This was not the case, however, leaving the precise mechanism still in debate. Postulations in addition to tricuspid valve closure include coronary vein emptying into the right atrium, a rise in intrathoracic pressure as a result of ventricular ejection, vibrations transmitted from the aortic root, and a retrograde venous wave due to cardiac movement during each cycle.

PALPATION

Carotid Pulse

In contrast to the jugular venous pulses, palpation of the carotid impulse is more revealing than inspection. Palpation is also useful in assessing the precordial activity and in the detection of pulsus alternans, an often subtle sign of congestive heart failure.

In a review of the arterial pulse in health and disease, O'Rourke discusses the confusing descriptions of the pulse in modern clinical literature.[5] He thinks that the

terms "percussion wave," "tidal wave," and "diastolic" or "dicrotic wave" should be abandoned, to be replaced by terms referring to timing or mechanism of the wave.

Old Term	New Term (based on timing)	New Term (based on mechanism)
Percussion wave	Early systolic	Impact
Tidal wave	Late systolic	Cephalic reflected
Dicrotic wave	Diastolic	Caudal reflected

Interpretations of impedance curves suggest that the percussion wave is due to impulse arrival, or impact, from ventricular ejection. The tidal wave is the echo from the lower part of the body. The anacrotic notch in aortic stenosis and the bisferiens pulse in a mixed aortic valve lesion probably have the same mechanism, namely the sudden vascular recoil after peak ventricular ejection (Fig 1–3). In idiopathic hypertrophic subaortic stenosis, however, the bisferiens contour probably reflects the unusual pattern of ventricular ejection (rapid initial outflow, then a late systolic rise in outflow). In palpating the neck of a child, one must be aware that the innocuous venous hum can be accompanied by a thrill.[6]

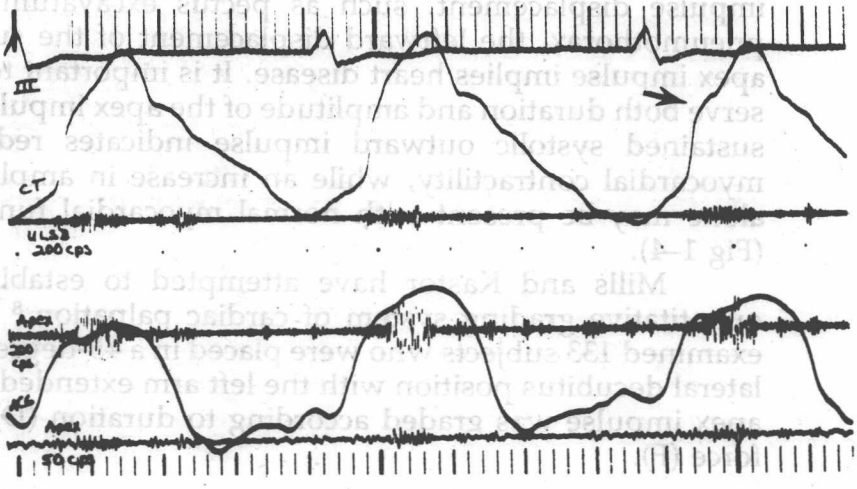

Figure 1–3 The anacrotic notch and delayed upstroke of the carotid pulse in an elderly patient with calcific aortic stenosis.

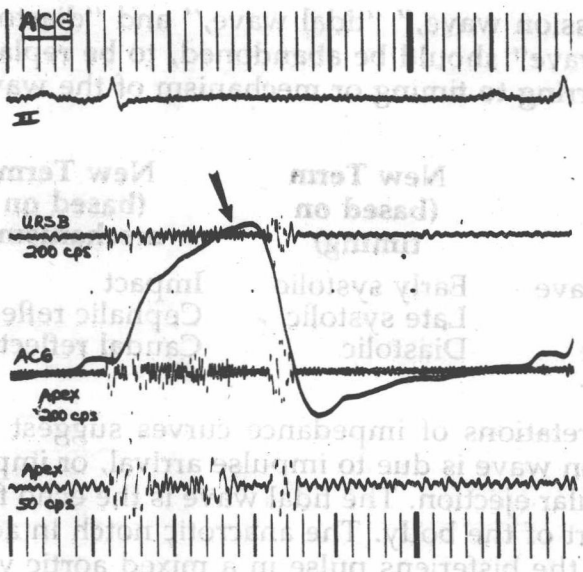

Figure 1–4 A sustained apex impulse in a patient with aortic stenosis.

Apex Impulse

Important information about left ventricular function can be gathered by simple, but careful, palpation of the cardiac apex impulse. Stapleton and Groves discussed the various nuances of precordial palpation in an extensive review.[7] If one excludes extracardiac causes of apex impulse displacement, such as pectus excavatum and pneumothorax, the leftward displacement of the supine apex impulse implies heart disease. It is important to observe both duration and amplitude of the apex impulse. A sustained systolic outward impulse indicates reduced myocardial contractility, while an increase in amplitude alone may be present with normal myocardial function (Fig 1–4).

Mills and Kastor have attempted to establish a quantitative grading system of cardiac palpation.[8] They examined 133 subjects who were placed in a 45-degree left lateral decubitus position with the left arm extended. The apex impulse was graded according to duration (D) and force (F).

Duration (requires simultaneous palpation and auscultation)

D_1 — The apex impulse retracts immediately after the first heart sound.

D_2 — The impulse retracts in the first half of systole.

D_3 — The impulse retracts in the latter half of systole.

D_4 — The impulse retracts with or after the second heart sound.

Force

F_1 — The faint apex impulse does not lift the lightly held finger above the chest wall.

F_2 — The lightly held finger is lifted, but a firmly held one is not.

F_3 — The firmly held finger is displaced a few millimeters.

F_4 — The markedly increased apex impulse makes it difficult to maintain the finger or stethoscope immobile over the cardiac apex impulse.

The authors found that 35 of the 38 normal subjects (92%) had impulses with both force and duration graded 2 or less; the remaining 3 (8%) had increased force or amplitude and were considered to be hyperkinetic. Of 29 patients with left ventricular dysfunction due to cardiac disease, 26 (90%) had abnormal apex impulses. It might be argued that the impulses were assessed in the left lateral decubitus position rather than the more standard supine position, possibly inducing false positive readings. This was not the case in the normal subjects evaluated. On the other hand, nine subjects with left ventricular disease had impulse abnormalities detected in the lateral position which could not be detected when supine.

Conn et al studied the cardiac apex impulses in 50 patients who underwent subsequent left ventricular angiographic evaluation. The physical examination proved more sensitive in detecting left ventricular hypertrophy than did either the electrocardiogram or the chest roentgenogram.[9]

Form of Study Assessing LVH	Sensitivity (% as compared to angiography-proven LVH)
Palpation of apex impulse	88
Chest roentgenogram	81
ECG	69

The authors summarized three normal aspects of the apex impulse: (1) It does not exceed half the distance between the midsternal and midaxillary lines. (2) It occupies only one rib interspace in size. (3) Its outward movement does not persist beyond early systole. As previously mentioned, a precordial bulge can often be observed after a transmural anterior wall infarction. This bulge or thrust can be palpated in up to 70% of such patients during the acute event and may persist permanently in up to 60%.[10]

Paradoxical Pulses

Kussmaul coined the term "paradoxical pulse" over a century ago in reference to the clinical situation in constrictive pericarditis in which there is an incongruity of regular heartbeat and irregular pulse. The latter, in spite of the apparent irregularity, "decreases and disappears with repetitive regularity."[11] In modern days, the term is used to describe a reduction in systolic blood pressure of at least 10 mm Hg during inspiration. The term is confusing since a lesser decrease of blood pressure is physiologic, whereas the term "paradoxical" would suggest that the normal response was an increase in blood pressure during inspiration. Adding to the confusion in terminology, Massumi et al. have described such an inspiratory rise in arterial systolic and diastolic pressures in certain clinical conditions, calling it "reversed pulsus paradoxus."[12] This entity may be observed in idiopathic hypertrophic subaortic stenosis, isorhythmic ventricular rhythms, and during intermittent positive-pressure breathing in subjects with heart failure.

The mechanism of the physiologic fall of systolic blood pressure during inspiration is related to a reduced left ventricular stroke volume during inspiration. This reduction comes from the pooling of blood in the pulmo-

nary vasculature because of the reduced intrapleural and intrathoracic pressure (which offsets the augmented venous return and increased right ventricular volume). In pulsus paradoxus (≥10 mm Hg fall in systolic blood pressure during inspiration), right ventricular filling and left ventricular emptying are interfered with because of a nonyielding pericardial sac, accentuating the physiologic inspiratory imbalance of stroke volumes of the two ventricles. Pulsus paradoxus can also be observed in severe asthma (in which left ventricular performance is hindered by the increased intrapulmonary pressure) or in right-sided heart failure, when venous return is hampered.[13]

Reversed pulsus paradoxus (or paradoxical pulsus paradoxus, to go from the sublime to the ridiculous) may have the following mechanisms, varying according to the underlying clinical situations.[13] In idiopathic hypertrophic subaortic sentosis (IHSS) a Valsalva-like effect may decrease left ventricular volume on expiration, hence lowering the blood pressure as compared to systolic levels. In intermittent positive pressure breathing (IPPB) the blood is squeezed out of the pulmonary capillaries and venules during positive pressure inspiration, augmenting left ventricular stroke volume and systolic blood pressure. With isorhythmic ventricular rhythm perhaps the accelerated heart rate during inspiration permits atrial "capture" for a few beats, increasing left ventricular volume and output during inspiration.

PERCUSSION

Although percussion of the lung fields continues to be a routine part of the pulmonary examination, cardiac percussion is rarely mentioned. It has not proven to be a useful means of assessing cardiac size, although it can be a clue to underlying pericardial effusions. A precordium quiet to palpation, in the face of enlarged area of cardiac dullness on percussion, should stimulate a search for pericardial effusion.

AUSCULTATION

Heart Sounds

Luisada et al have reviewed the changing concepts about the mechanism of the first and second heart