Gabriele Bronzetti

Atlas of Pediatric and Youth ECG



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Gabriele Bronzetti Pediatric Cardiology Policlinico S.Orsola-Malpighi Bologna Italy

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This Springer imprint is published by Springer Nature The registered company is Springer International Publishing AG The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland *To Angelita, Francesco, and Chiara, for all your heartbeats that I wasn't there 4*

Foreword I

One of the most remarkable transformations in life is the growth of an infant into a mature adult. Human infants are helpless when born and have one of the longest periods of dependence on their parents of any creature. Every organ of the body goes through a constant process of growth and development ultimately culminating in the emergence of a mature adult. Among the organs that change significantly in the process of growth is the human heart. The development of electrocardiography as a method of recording the electrical activity of the heart gave medicine dramatic new insights into both normal cardiac physiology and its pathophysiology. These initial investigations focused almost exclusively on adult men, with little appreciation of the ways in which electrocardiogram may differ in women and the developing child. The emergence of pediatric cardiology as a distinct discipline has emphasized the need to establish the unique electrocardiographic characteristics of heart in child and adolescent in health and in response to illness. While there have been several attempts at producing comprehensive texts to explore the wide range of electrocardiographic findings in children and adolescents, the subject has not received the attention that it so richly deserves. The current book by Gabriele Bronzetti admirably helps fill this need. Using the insights and experiences gained over a career spent tending to the cardiac needs of children and adolescents, he has produced a remarkable atlas of electrocardiograms that cover the entire spectrum of disorders found in clinical practice. The book provides a rich and invaluable resource for those who wish to explore the fascinating and complex world of ECG findings in younger patients. One of the book's strengths is the meticulous attention to a detailed analysis of each ECG presented, as well as the clarity of the recordings themselves. Bronzetti's work will prove an indispensable resource for anyone interested in the complex and fascinating world of pediatric electrocardiography.

> Blair P. Grubb, M.D., F.A.C.C. Distinguished University Professor of Medicine and Pediatrics The University of Toledo Medical Center Toledo, OH, USA

Foreword II

"A doctor walks into a bar and says to the bartender..."

No, this is not a joke. In many ways it was a pivotal moment of Dr. Bronzetti's life. The rub: Gabriele Bronzetti wasn't the doctor; he was the bartender. Thus began (not even arguably) the most important of Dr. Bronzetti's many passions: his love for his amazing wife Angelita, who he eventually followed into the study and practice of medicine. Perhaps someday she'll tell me the story of what drew her as a young neonatologist into that bar on that day.

Dr. Bronzetti's second passion, which also may have had its origins that day, is for medicine and eventually pediatric cardiology and its heart rhythm disorders. His third passion is for teaching, as is aptly demonstrated in this book on pediatric electrocardiography. His next passion (of which I am aware) is for the arts and written word, which is what makes this *Atlas of Pediatric and Youth ECG* uniquely special. His final passion is for his own children, but also extends to the patients and families that he cares for so expertly.

I met Gabriele Bronzetti when he came to train with me in pediatric electrophysiology at The Hospital for Sick Children, Toronto, in 2004. Gabriele was adored by staff and patients alike and excelled in both learning our discipline and teaching it to the core fellows, residents, and students within our program. His appreciation for the fine arts, as well as other important aspects of life, became obvious to me one evening when he invited me home for dinner. With two of my younger and more boisterous children in tow, I arrived at his home to realize he was staying in the Toronto residence of a renowned art expert, and I spent the evening envisioning and avoiding priceless pieces being destroyed by my offspring. As dinner was being prepared, I heard what to a non-Italian sounded like the most passionate of arguments coming from Gabriele and Angelita in the kitchen. I eventually realized this was all about whether the pasta was done just right. It is with this background that I introduce you to Dr. Bronzetti, passionate physician, teacher, husband, and father, whose grasp of language and the written word, whether in English or Italian, defines the word "art."

Electrocardiography is to a great extent part of the art of medicine. While the statistical norms have been finely calculated for adults and children by giants such as Drs. Pentti Rautaharju and André Davignon, the application of electrocardiography to diseases has primarily relied on translation of empiric observations into defined set of rules. Thus, we "interpret" the results of an ECG. Since only normal values are well described, this can lead the casual observer to consider an ECG as the "sedimentation rate" of the heart. It's great if it is normal, but if not, you'd better do a battery of other tests to find the cause of the abnormality. This simplistic approach is hazardous, as the ECG contains many measurements, some of which, when defined at the 98th percentile, may be statistically abnormal in any individual. On the other hand, certain patterns can be recognized for specific cardiac lesions and can provide the astute clinician with a likely diagnosis while awaiting more definitive studies such as echocardiography. These classic findings in congenital heart disease are beautifully described and illustrated in Chap. 18 of this atlas.

In the first chapter, Dr. Bronzetti provides philosophical approaches to the evaluation of the ECG, invoking classics from children's books to weighty novels, and reviews the indications for electrocardiography. The second chapter introduces the reader to basic concepts of electrocardiography, this time using classic sculptures, and provides further philosophy on the

reporting of ECGs. Chapters 3 and 5 introduce the reader to the development of electrocardiographic waves (both normal and pathological) and their ontogeny through childhood, this time with some biblical references for interest. Chapter 4 takes a magical tour of the "numerology" of ECGs, including calculation of rate and axis.

Chapter 6 defines normal ECGs and normal variants. Those entrusted with ECG interpretation, hopefully also provided with context, should remember to highlight findings deserving further assessment and minimize variations that might lead to unnecessary investigation or worry. Chapters 7 through 9 highlight ECG findings of metabolic derangements, artifacts and devices, and bradycardias and conduction delays, respectively. Each of these chapters is beautifully illustrated with dozens of high-quality electrocardiograms demonstrating these specific findings.

Electrocardiography remains the mainstay for diagnosis of cardiac rhythm disorders. Chapters 10, 11, and 12 describe the electrocardiography of preexcitation syndromes, supraventricular tachycardia, and ventricular tachycardia. Given our renewed understanding of the implication of preexcitation in children, the reader is appropriately referred to the 2012 Guideline document. The electrocardiography of SVT subtypes is described, an information which may be crucial in guiding long-term management. Ventricular tachycardia, albeit rare in children, is described in detail due to the importance of determining its etiology.

As might be expected from an Italian cardiologist, the electrocardiography of cardiomyopathies is beautifully described and differentiated. Similarly, the identification and differentiation of channelopathies are well described and illustrated. Finally, Dr. Bronzetti rounds out with chapters on cardiac tumors, pulmonary hypertension, and ischemia.

This atlas provides beautiful illustrations, current references, and a philosophy and style that is a pleasure to read. I congratulate Dr. Bronzetti and "whole-heartedly" recommend his work to you, the reader.

Robert M. Hamilton, M.D., M.H.Sc., F.R.C.P.(C) The Labatt Family Heart Centre and Translational Medicine Program and Department of Paediatrics The Hospital for Sick Children & Research Institute and University of Toronto Toronto, ON, Canada

Preface

One of the most fascinating and mysterious aspects of an ECG is the wealth of information hiding behind a seemingly innocent tracing. The ECG is a technically simple examination that produces familiar writing on a piece of paper, in a language that has stayed the same since it was invented more than a century ago. While echocardiography now uses machines that produce images that even 20 years ago were unimaginable, an ECG performed today displays the same signs seen by the founding fathers of electrocardiography, from Einthoven onward.

In the space of a century, we have discovered the many diseases that can lie concealed between the waves, from channelopathies to cardiomyopathies, from arrhythmic syndromes to congenital heart diseases. In the meantime, the ECG has been supported but not replaced by the new imaging techniques and has earned a precise role in screening programs for various conditions, in particular in sports preparticipation screening for prevention of sudden cardiac death, a program that is already strongly supported in Italy but which is now spreading throughout the world. Without getting into any discussion, it is known that the "Italian" position considers the ECG to be a cost-effective method. The ECG is generally satisfactory in terms of specificity and sensitivity; in other words, a normal ECG reassures the cardiologist and the families of young sports players. The most important thing, however, is to recognize when an ECG is normal, avoiding false-positive results and all related implications in terms of additional tests and examinations as well as the risk of discouraging sports activities. Sports activities aside, ECG still plays a key role in the diagnosis of arrhythmias and ischemic syndromes. Nevertheless, the interpretation and deductive analysis of an ECG is no easy task, but requires curiosity, clinical experience, and culture. There are no shortcuts, and the quick routes on a cardiac map are usually accessory pathways and should not be followed. The ECG seizes and stops the anatomy of an instant, a hermeneutic process even more difficult in babies and in the young where the heart is changing day by day. This continuous alteration during developmental age takes the cardiologist by surprise: in the attempt to keep up with the new methods and the enormous number of publications, he or she has been obliged to neglect electrocardiographic semeiotics and now struggles to interpret the tracings of a newborn, of a youngster with normal variants, or of a 60-year-old who has had cardiac surgery and suffers from hidden congenital heart disease. One of the particular features of this book is that all the tracings come from the same tertiary center. They were selected from thousands of tracings that I have personally collected over the last 20 years and which allowed me to make or confirm a diagnosis. The part for electrophysiology experts is kept to a minimum in order to leave room for examples and tracings that everyone working in the medical sector can understand. The ECGs that a pediatric cardiologist sees in a lifetime, the ones that a pediatrician imagines in his or her patients and that a cardiologist of adult patients fears when he or she is on night duty, are all included and commented on. This atlas can be also of interest to other players in the field of medicine – anesthetists, students, and nurses – who may be the first to see the mysterious pink paper emerging. No one should feel left out since, as Tolstoy would say, all normal ECGs look alike, but every ECG is in its own way abnormal.

Bologna, Italy

Gabriele Bronzetti

Acknowledgements

"I would like to thank all the Maestros we have had and would like to have had and all the young doctors that actively contributed to the writing of this book and whose thirst for knowledge made it necessary.

My thanks also to Susan West for improving my English here and there (her words, not mine!)."

Special thanks for the invaluable collaboration of Tanya Bodnar, Maurizio Brighenti, Cristina Ciuca, Alessandro Corzani, Cinzia D'Angelo, Junio Massimiliano Durante, Elisabetta Mariucci, and Monica Urru.

A special mention in memory of Gabriele Cristiani who contributed fundamentally to the construction of some images.

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Abbreviations

ACAOS	Origin of the coronary artery from the opposite sinus
AF	Atrial fibrillation
Aflutter	Atrial flutter
ALCAPA	Anomalous left coronary artery from pulmonary artery
ARCAPA	Anomalous right coronary artery from pulmonary artery
ARVC	Arrhythmogenic right ventricular cardiomyopathy
ASD	Atrial septal defect
AV	Atrioventricular
AVB	Atrioventricular block
AVNRT	Atrioventricular nodal reentrant tachycardia
AVRT	Atrioventricular reentrant tachycardia
BAV	Bicuspid aortic valve
BER	Benign early repolarization
BS	Brugada syndrome
CAVC	Complete atrioventricular canal
CCTGA	Congenitally corrected transposition of great arteries
CPVT	Catecholaminergic polymorphic ventricular tachycardia
DCM	Dilated cardiomiopathy
EAT	Ectopic atrial tachycardia
ECG	Electrocardiogram
ECMO	Extracorporeal membrane oxygenation
ERP	Early repolarization pattern
HCM	Hypertrophic cardiomiopathy
HR	Heart rate
JET	Junctional ectopic tachycardia
LBBB	Left bundle branch block
LGL	Lown-Ganong-Levine syndrome
LQTS	Long QT syndrome
LVH	Left ventricular hypertrophy
PAC	Premature atrial contraction
PAPVR	Partial anomalous pulmonary venous return
PMK	Pacemaker
PJRT	Permanent junctional reciprocating tachycardia
PSVT	Paroxysmal supraventricular tachycardia
PVC	Premature ventricular contraction
QTc	Corrected QT
RBBB	Right bundle branch block

RVH	Right ventricular hypertrophy
RVOTVT	Right ventricular outflow tract tachycardia
SVT	Supraventricular tachycardia
TAPVR	Total anomalous pulmonary venous return
TGA	Transposition of great arteries
VSD	Ventricular septal defect
VT	Ventricular tachycardia
WPW	Wolff-Parkinson-White

Introduction

The real voyage of discovery consists not in seeking new landscapes, but in having new eyes
Marcel Proust

1.1 A Literary Premise to a Practical Book

Is another book on the electrocardiogram (ECG) really necessary if there are already so many? One could venture a literary answer, stating that those who know Anna Karenina should not ignore Madame Bovary. We do not know and will never cease to talk about the heart enough, the heart that follows the rules and the heart that is forced to betray them because of congenital or acquired reasons, because of hemodynamic or electrical reasons. More prosaically, one can add that among the many published texts, only a few are dedicated to children and the young.

This book would like to share a simple, fresh look at a set of more or less orderly signs on pink paper, which reveals and hides a treasure chest of 12 leads in 12 interminable seconds. The ECG is crystallized time like a Jurassic butterfly in a drop of amber. Faced with the pink paper, any person may feel like an entomologist watching an insect during its metamorphosis and a scientist, like an artist, who does not invent but discovers. The truth is in the detail: almost everything is in the almost nothing of a small or hidden wave. Physicians should seize tiny clues and as Proust says "we must break the spell that holds things captive, bring them down to us and prevent them from falling forever into emptiness." We must learn to see the detail that reflects or deflects from *normality*, sure only of its vast and elusive nature. We have to dig up the past, stop the present, and foresee the future using various arts and cultures, even the "intermittence of the heart." These are arrhythmias of Proustian memory, revealing beats that open up time with the ephemeral and irrevocable grace of a butterfly. If growing up means becoming what we already are, we can only really know the ECG of an adult by looking at the ECG of the baby that the adult once was.

Ferdinand Céline was a French physician who became a famous writer. He said that adults are children gone bad. So even the doctor with little or no specialization in pediatrics eventually feels the need to know what the patient's ECG was like before the deterioration. In "The Metamorphosis" by Franz Kafka, Gregor Samsa becomes an insect and dies unrecognized even by his family. Culture and science, on the other hand, should serve to discover the man hiding in an insect.

To start off on the right foot, we should rely on Tolstoy, and citing the opening words of his "Anna Karenina," we have to recognize that *all normal ECGs look alike but every ECG is abnormal in its own way*.

1.2 Why Does Everything Begin with P?

There are mathematical explanations dating back to Descartes that put the P at the beginning of the ECG glossary, and not the A as would be more intuitive [1]. However, if the smallest wave, the one that first comes out of the heart and breaks through the skin to land on the pink paper, is called P, there must be other reasons. Many childhood heroes begin with P: "Le Petit Prince (The Little Prince)," to tell us that what is essential is invisible to the eye and can only be seen with the heart. "Pinocchio," to remind us that the ECG is sincere but does not always tell the truth. "Peter Pan," with his desire never to grow up, just like the P wave. Le Petit Poucet or Pollicino (Tom Thumb), the cunning P, able to disguise and hide. The Dickensian Philip Pirrip "Pip" of "Great Expectations." "Pippi Longstocking" by Astrid Lindgren, herself an orphan, an example of power to the imagination. The "Pink Panther" of seraphic guile. Schulz's cartoon strip "Peanuts," with the gang including the dynamic "Peppermint Patty." Not to mention Walt Disney's "humanized" dog Pluto, an example of candor and levity. Then we have the dreamy Pimpa, with its four paws firmly planted in the clouds. Finally, a sign of the times is the very current "Peppa Pig," which sums up the ECG with porky pragmatism. In fact, pork is pink and cheap and can be used up to the very last bit. In German culture there is the "Eierlegende Wollmilchsau," a mythic pig that lays eggs like a chicken, gives milk like a cow, wool like a sheep, and ends up in ham and chops, practically like an ECG, which sometimes acts as an echocardiogram, a catheterization, and an MRI all in one.

1.3 ECG Waves: An Entomological Vision

On an ECG and on the palm of a hand, the body of a beat can be dissected into three parts like a beetle (from the Greek "en temnein," cut into pieces) (Table 1.1, Fig. 1.1), namely, the head, the P wave and PR segment, where everything starts; the chest, the QRS complex, where everything starts; the chest, the QRS complex, where everything starts; the abdomen, the ST-T segment, restoring ionic balance and where everything returns to baseline status. We start off on our campaign with a butterfly net of graph paper to capture the enchanting winged creature and study it; the subjects of our research could be at different stages of development. In insect metamorphosis there are four stages: egg, larva, pupa, adult insect (the latter also called imago, or imagine). The ECG is the only image, the only visible phase of the electrical activity of the heart. More precisely, we can recognize four ECG stages: neonatal age up to 1 month, early infancy up to 3 years, second and third periods of infancy up to 12 years, and adolescence and adulthood after the age of 12 years.

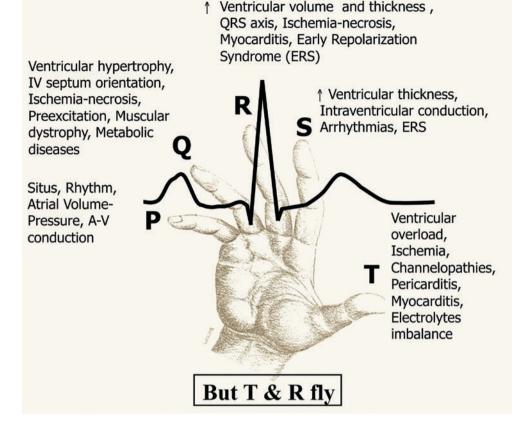
The ECG approach cannot ignore the knowledge of its diachronic development, the real objective of the pages that follow.

Childhood is endless and over the years, and in the absence of disease, the P waves remain surprisingly unchanged, while the T and R fly. To condense the ineffable concept, having cited Tolstoy we must mention Nabokov. The Russian writer was not only a great expert on butterflies;

Table 1.1 ECG's dissection and dedu	ction
-------------------------------------	-------

What to look	Finding	Suspected diagnosis					
Atrial wave							
Rhythm	A-V dissociation, bradycardia A-V dissociation, tachycardia	III degree AVB JET (narrow QRS)—VT (wide QRS)					
P wave morphology	Negative P wave D_2 - D_3 -aVF, tachycardia Abnormal P wave axis	Incessant arrhythmia (PJRT, AT) Ectopic atrial rhythm, atrial isomerism, dextrocardia					
	Peaked P wave >2.5 mm P duration >120 ms	PS, TA, PAIVS, Ebstein, PEX, RCM Mitral steno/insufficiency, HCM, RCM					
PR interval							
	Short PR—Delta wave	Metabolic (Pompe, Danon, Fabry, PRKGA 2), mitochondrial (LHON, MELAS), TS, Duchenne-NMD, pre-excitation AVC, CCTGA, RF, KD, NMD, Brugada, HCM					
QRS interval	C						
Electrical axis	Left axis deviation Right axis deviation (extreme)	AVC, TA, ostium primum ASD, VSD, CCTGA, Steinert Noonan syndrome, severe RVH					
Q waves	Q in right precordial leads (always pathologic) Anterolateral, inferior pathologic Q waves Absent Q wave in V6	Severe RVH, CCTGV, univentricular heart ALCAPA, Duchenne,-NMD CCTGV, LVH/RVH, normal variant					
Delta wave	Delta wave + short PR	Pre-excitation/WPW, Ebstein, CCTGV, HCM, TS					
Epsilon wave	Small positive wave in the end of QRS	ARVC					
J wave	Slurring/notching of terminal part of QRS	Hypothermia, early repolarization (Brugada)					
Bundle branch block	SR—VT (RBBB—LBBB morphology)	ASD/RV overload, idiopathic VT, myocarditis, neoP, CMPs, normal variant, PM					
Repolarization ((S-T, T, QT, U)						
	 ↓ S-T right precordial leads (left) 0–7 days ↓ S-T right precordial leads >7 days Positive T wave in V1: 7 days–7 years of age Negative T wave V2–V4 < 12–14 years Negative T wave >14 aa ↓ left precordial-inferior leads ↑ S-T multiple leads ↑ S-T V1–V3 Prominent U waves (>50% of T wave) 	PPHN RVH RV overload Juvenile pattern Overload, ischemia, electrical memory, neoplasms, CMPs ALCAPA, CMPs Pericarditis, myocarditis, MI, BER, PNX Brugada, BER Hypokalemia, long QT, bradycardia					

ASD atrial septal defect, ARVC arrhythmogenic right ventricular cardiomyopathy, AT atrial tachycardia, AVB atrioventricular block, AVC atrioventricular canal, CCTGA congenitally corrected transposition of great arteries, BER benign early repolarization, CMPs cardiomyopathies, HCM hypertrophic cardiomyopathy, JET junctional ectopic tachycardia, LVH left ventricular hypertrophy, KD Kawasaki disease, LHON Leber's hereditary optic neuropathy, MELAS myopathy, encephalopathy, lactic acidosis, and stroke-like, MI myocardial infarction, NMD neuromuscular diseases, neoP neoplasms, PAIVS pulmonary atresia with intact ventricular septum, PEX pectus excavatum, PKRGA 2 protein kinase AMP-activated non-catalytic subunit gamma 2, PJRT permanent junctional reciprocating tachycardia, PMK pacemaker, PNX pneumothorax, PPHN persistent pulmonary hypertension of the newborn, PS pulmonary stenosis, RCM restrictive cardiomyopathy, RF rheumatic fever, RVH right ventricular hypertrophy, SR sinus rhythm, TA tricuspid atresia, TS tuberous sclerosis, VSD ventricular septal defect, VT ventricular tachycardia **Fig. 1.1** The ECG in the palm of the hand and at your fingertips



he was also able to write masterpieces directly in his adopted language, English. The P remains the same, but T and R fly. In a word, BUT T & R FLY.

Immediacy and low cost are the features that make the ECG absolutely modern and, combined with a skillful "imagination," able to be ahead of the more expensive cardiac imaging. In emergency situations such as arrhythmias or chest pain, when it's just you and the patient and you cannot delay the diagnosis by asking a colleague or performing another examination, the ECG remains an unavoidable tool of bearable lightness (Table 1.2).

The book is addressed to everyone, students, nurses, and doctors (mindful cardiologists, inquisitive pediatricians, and alert anesthesiologists) more or less familiar with young and older patients. We have carefully avoided an introductory section on electrophysiology, not essential to the understanding of the text and which in any case can be found in infinite variations, both in the attic and on the web. The book collects

Table 1.2 Common indications for pediatric ECGs

Screening	Neonatal (nonmandatory), mother with systemic lupus erythematosus, sports
Symptoms	Palpitations, syncope, chest pain, congestive heart failure, cyanosis, epilepsy
Conditions	Rheumatic disease, Kawasaki disease, pericarditis, myocarditis, hypertension, neuromuscular disease, electrolyte imbalance, infectious disease (Lyme disease, etc.)
Family history	Congenital heart disease, cardiomyopathy, primary electrical diseases, sudden death
Therapy	Antiarrhythmic, pacemakers, anticancer, diuretic, QT-prolonging drugs, poisoning

the essence of exams carried out in a reference center over the past 20 years. At this point the good teacher would admonish her/his pupils with "mind your p's and q's" which, in the ECG's world, is literally the most appropriate advice I can give to the reader.

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Basic Concepts of Electrocardiography

We did not start from the action potential, and we have deliberately not got bogged down in vectorcardiographic analysis. So we can say that Willem Einthoven is our *notable absentee*. The "child" vision requires an intuitive approach which nevertheless seeks to translate the deep, unconscious electrophysiology into a concept of surface: the vector and its symbol, the arrow shot by ions that travel across the membrane proteins.

The activity of the heart creates an electric field that can be detected by surface electrodes. This electrical activity may be assimilated to a dipole, a system consisting of two electric charges of equal and opposite sign and separated by a constant distance in time. When the atria (or ventricles) undergo depolarization, the wave that spreads across the muscle takes many different directions simultaneously and can be represented by arrows. Each arrow represents a different individual vector, and the set of these individual vectors can be summarized by the mean (or resulting) electrical vector. It is best to keep in mind that the forces generated in opposite directions can neutralize each other. The most intuitive way to read the ECG is to think of this vector (which applies to every wave from P to T) as an arrow that we see from our vantage points and that is the standard 12-lead ECG. The direction of this arrow relative to the electrode determines the magnitude of the recorded voltage (the voltage of course also depends on the mass of the cardiac tissue involved). When the arrow is approaching, the electrode which sees the tip will record a positive voltage and then a positive wave; conversely, when the arrow moves away from the electrode and this sees the bottom, it will react with a negative voltage and a negative wave (Fig. 2.1).

The arrow that we imagine on the ECG goes a long way and travels across a sky called conduction system. This skyway begins in the pale cells of the sinoatrial node, located in the upper right atrium where the superior vena cava ends. Hence, the impulse that arises from spontaneous depolarization regulated by the autonomic nervous system and by circulating hormones will go toward the left atrium through the muscle cells but also past the Bachman bundle and toward the ventricles with internodal pathways and through the working myocardium. The AV junction consists of a proximal part, the AV node, and a distal part, the bundle of His. From the His bundle, the two main branches will finally start and will end in the ventricles to the Purkinje fibers. Now ventricles are activated and the electromechanical coupling can move the blood. The main ventricular vector can be divided in three parts, taking into account that the right ventricle starts to depolarize shortly after the left ventricle: the first part represents the resultant force of the septum (vector 1), moving from the left toward the right and triggering the q wave in V6 (and lead I and aVL); the second represents the activation of the free wall of ventricles (vector 2); the third represents the activation of the postero-basal aspects of ventricles (vector 3).

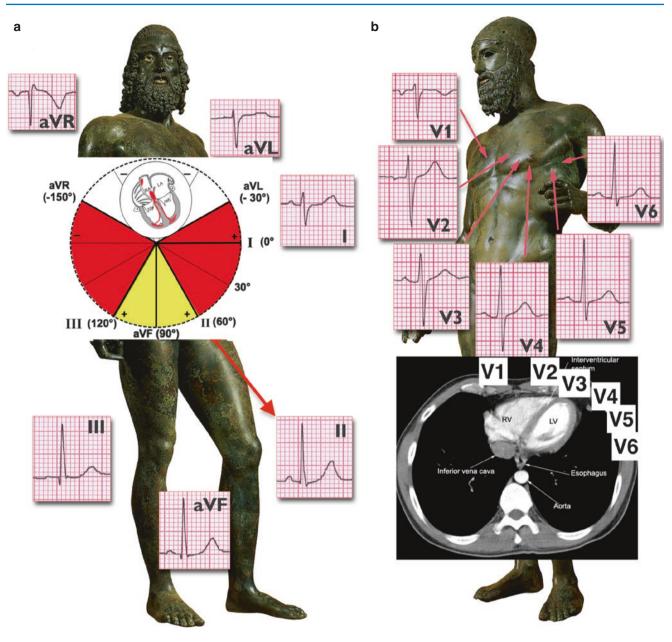


Fig. 2.1 The Riace Bronzes as models for a 12-lead ECG in a 12-yearold boy. **Bronze (a)** the one with the more restless style. Frontal plane, standard limb leads (I, II, III), and augmented unipolar (aVR, aVL, aVF); like the style of the statue, these leads are sensitive to movements and artifacts. **Bronze (b)** horizontal plane, precordial leads V1 (fourth

intercostal space, right parasternal), V2 (fourth intercostal space left parasternal), V3 (equidistant between V2 and V4), V4 (fifth intercostal space midclavicular line), V5 (fifth intercostal space anterior axillary), V6 (fifth intercostal space, axillary media); like the style of the statue, these leads are more stable and relaxed.

2.1 Technical Aspects

Before moving on to atrium and ventricular dissection, it is worth remembering some technical aspects. You should always check that paper speed and calibration are standard and thus respectively 25 mm/s and 10 mm = 1 mV. An increase in speed or amplitude may be required when greater precision in estimation of intervals or small amplitude waves is important. True dextrocardia must be distinguished from misplacement of the limb leads (the so-called nurse's dextrocardia).

When faced with an improbable ECG, you have to question the correctness of lead placement, sometimes poorly positioned due to staff carelessness, thoracic deformity, dressings, etc. [1, 2] The surface ECG is a simple technique, but that does not mean it can be performed superficially. Just like cooking, it is the simplest recipes that require perfection of the few necessary ingredients. Every laboratory should comply with the standard placement of the electrodes, and nurses should be skilled in being able to put children of all ages at their ease, resorting to various arts ranging from soap bubbles to smartphones.

In remote-viewed tracings, whether sent by fax or other means, you have to make sure that the relationship with the original is 1:1; even small differences of scale can lead to blunders, for example in the calculation of the QT interval (if you don't know the scale, you can count the little boxes that at standard paper speed correspond to 40 ms). Sometimes there are funny artifacts: the oscillatory ventilation often used in neonatal intensive care can simulate drug-resistant flutter (diagnosis and therapy are electric, just switch off the oscillator) [3]. Even hiccups can create bizarre waves resembling extrasystoles which are neither supraventricular nor ventricu-

lar, but simply underventricular or diaphragmatic. Congenital heart disease may require nonstandard leads such as V3R, V4R, and V7: the first two increase the sensitivity of the right overload [4], while V4R (positioned on the right midclavicular fifth intercostal space) can be an excellent alternative to V1-V2 which cannot be applied due to dressings or for other reasons. With ECGs that are "RBBB like" or with an "atypical" RBBB, in family members of patients with Brugada syndrome, or in the case of unexplained syncope, in addition to the ECG standard you should do an ECG with right precordial leads (V1 and V2) moved high in the third and in the second intercostal space. In the same conditions, it is also useful to perform an ECG in the course of fever as it is known that hyperthermia can unmask the Brugada pattern. Modern electrocardiographs have sample rate and bandwidth higher, respectively, than 500 samples/s and 250 Hz - they write very well but they don't read quite so well. Automatic diagnosis may be incomplete, petulant, or catastrophic and is not always reliable, especially when the machines do not know they are dealing with a child and see infarction instead of infants [5]. "Manual" diagnosis should avoid as much as possible scary terms like "block," "delayed," "nonspecific alteration," and "anomaly" which are not much help. When these terms are unavoidable, they should be followed by conclusions that are as reassuring as possible. You should also avoid defensive adverbs and phrases such as likely and cannot be excluded followed by a dozen differential diagnoses. The perfect report consists of a single word: "N o r m a l." A broken clock tells the correct time at least twice a day. Cardiologists who are too far forward or too far back never get it right. For all the time and amplitude parameters, refer to the normal value tables; the Davignon and Rijnbeek tables are still useful [6, 7] (Table 2.1).

Table 2.1 Values of normal ECG parameters according to age

	HR	QRS axis	QTc*		PR	R wave*		S wave*		Q wave*	k
	(bpm)	(degree)	М	F	(ms)	V1	V6	V1	V6	III	V6
0–3 day	90-155	60-170	480	480	70-150	27	12	21	10	3	3
3–7 day	90-155	65-165	480	480	80-140	24	12	17	10	3	3
7–30 day	95-180	65-160	440	440	70-130	21	17	11	10	4	4
1–3 month	95-180	30-120	440	440	70-130	(12) 20	(15) 22	13	7	5	3
3–6 month	95-180	5-105	440	440	70-150	(13) 22	(16) 27	17	10	7	4
6–12 month	95-170	5-100	440	440	70-160	(11) 20	(17) 27	18	7	8	6
1–3 year	90-150	5-100	440	450	80-160	(10) 21	(18) 29	21	7	7	6
3–5 year	70-135	5-100	440	450	80-160	(9) 18	(19) 31	22	5	5	6
5–8 year	65-132	5-140	450	460	90-160	(6) 15	(20) 29	23	4	6	7
8-12 year	60-130	5-110	450	460	90-170	(5) 11	(21) 32	25	4	7	7
12-16 year	60-120	5-130	450	460	90-180	(5) 11	(20) 30	22	4	7	7

() = median; $* = 98^{\circ}$ centile

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The Waves During Developmental Age

In the various stages of development, the ECG changes constantly, pursued with cognitive anxiety by the cardiologist. It is a horizon slipping away in front of us, an ontology of waves that knows no end because when it ends, the ECG can no longer be recorded. The handwriting of the heart depends on position, relative mass of the ventricles, physique, thoracic deformity, lung inflation, pneumothorax, and impedance of tissues. Intracellular accumulations increase the voltage and thus the amplitude of the waves on the paper, while extracellular accumulations or fibrosis depresses them. And so a newborn with a 21 g heart can generate a QRS complex as wide as that of an adult with a heart 10-15 times heavier; a young person with anorexia can show widespread low voltage typical of a devastating myocarditis; a massive myocardial hypertrophy with fibrosis can have lower voltage than a heart fivefold less thick or heavy (Table 3.1). The entomologist recognizes the age of bees through sight and smell: the young bee is hairier and more colorful and emits different Nasonov pheromones from an adult bee. To infer the age of a child, we could choose lead V1, look at it, and smell it, leading to enlightening diagnostic inspirations. You need intuition, a nose, and fragrant pink paper is much better suited than an aseptic computer screen. In addition to V1, lead II is also a formidable lead due to dissection of the P wave at baseline and during arrhythmias, to the atrial and ventricular electrical axis calculation, and to the accurate estimation of the OT interval.

Having to settle for just three leads—for intellectual challenge or need—you could supplement the abovementioned couple with a left precordial such as V5 or V6. In the majority of cases, 3 out of apostles 12 are sufficient to preach the Table 3.1 Normal pediatric ECG findings

- Marked respiratory sinus arrhythmia (↑ isp↓ esp), physiologic low atrial rhythm
- Short PR and narrow ventricular complex
- Physiologic I degree and II degree AVB (Mobitz 1)
- Atrial and ventricular extrasystoles
- Axis deviation, QRS axis >90°
- Precordial leads mimicking dextrocardia in neonates (lead I is crucial)
- Prominent infero-lateral Q waves
- The repolarization is more important than voltage to diagnose ventricular hypertrophy
- Early repolarization pattern
- Negative T waves in V1–V4 (up to 12 years of age)
- Notched T wave in right precordial leads (V2–V3), prominent
 U wave

electric *word*. Among the ancillary leads, aVR could be considered the most useless, given its persistent negativity. Only at the height of an ischemic calvary, such as proximal obstruction of the left coronary artery, does the ST segment of aVR rise to a sardonic positivity when its 11 colleagues become dramatically negative. In children, these scenarios are fortunately extremely rare (coronary artery dissection, acute thrombosis of an aneurysm in Kawasaki disease, compression of the left main branch of the left coronary artery by the pulmonary artery as can happen in ACAOS or in primary pulmonary hypertension or during balloon angioplasty of the pulmonary artery); in the section that examines the ST segment, the specific ischemic aspects of the first two decades of life will be discussed.

3.1 P Wave

The P wave is fundamental, and unlike other waves, the normal values do not change significantly at different ages. *But T* & *R fly*. In sinus rhythm, the P wave is the sequential activation of the right and left atrium. It can be monophasic, but it is not uncommon for it to be notched or biphasic. The right atrial vector points down and ahead, while the left atrial vector points to the rear, to the left and down (Fig. 3.1). The resulting axis is in the left lower quadrant (0–90°), and the lead that sees it best is lead II, in addition to V1. The P wave can be +/– biphasic in all three inferior leads, while in aVL, it can be biphasic -/+, positive or negative. It must not, however, be negative in lead I; in this case, ectopic rhythm, malposition of the electrodes or dextrocardia should be suspected.

For all age groups, the amplitude limit is 2.5 mm, apart from newborns/infants where it extends to 3 mm up to 6 months. Especially in the very young, left atrial hypertrophy can be missed when a time limit of 120 ms is employed, given the smaller size of the atria and the greater atrial conduction velocity. The diagnosis of atrial enlargement is valid only with ECG in sinus rhythm, as atrial ectopic beats may create an infinite spectrum of amplitude and duration of P waves [1]. Atrial enlargements are a result of a pressure overload (outflow obstruction or ventricular hypertension) or volume (insufficiency of atrioventricular valves [AV] or shunt) of the atria.

In right atrial enlargement (Fig. 3.2), in sinus rhythm, and in situs solitus, there is an increase in voltage of the initial atrial vector, while the voltage of the left atrium is unaffected; since the P wave is the sum of the right and left components and the left follows the right, there is no appreciable prolongation of P wave duration in right atrial enlargement:

- The P wave is high and pointed in the limb leads II, III, and aVF, and the voltage is increased and greater than 2.5 mm (3 mm in infants).
- The P wave in V1 may remain unchanged or may be pointed or diphasic (with a predominant positive component).

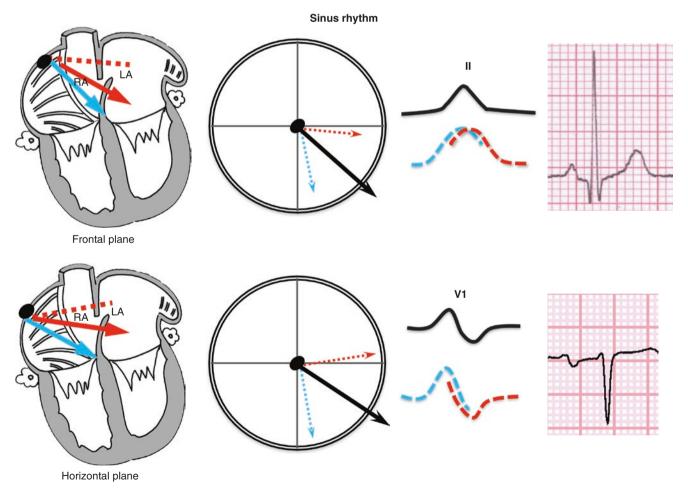


Fig. 3.1 Depolarization of the normal atrium