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Special edition

Simplified Electrocardiogram:
A practical guide for all

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When there is a will,
there is a way ..

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Why dedicate a special issue to Electrocardiogram interpretation now when there are endless publications on this topic in the market?

Since its launch in 2009, Med Emergency, MJEM was designed to be the journal of the practitioner and for practitioner use. Therefore, this special issue on how to read an electrocardiogram does not resemble any other publication not because it is the best, but because it is the outcome of daily practice of its authors who after scrutinizing the smallest squares of the electrocardiogram sheet, managed to summarize more than two hundred charts and offered the widest possible interpretation of an ECG in emergency.

In their approach, authors try to make the reading of the ECG look easy in a bid to reassure the reader that he/she too can easily read an ECG. But behind this attempt to make things look easy, are two major difficulties. The first difficulty is in the sequence of the information. We need to clarify that the sequence of the various sections that strictly follows the different phases of depolarization - repolarization of the myocardium is there in order to facilitate the reading but in practice the whole picture has to come together for a holistic interpretation. Thus, the interest of the decision tree that is suggested in conclusion. The second difficulty is to be able to establish clinical correlations on the basis simple electrical charts. This is possible because authors are basing their findings on real clinical cases that they came across and followed up personally. The reader who does not know the clinical case may interpret some strips differently. This is why this work shows us that it is sometimes impossible to master all the subtleties of an ECG reading be it for regular physicians, or specialists in this field.

In brief, this work remains a very practical tool for those who wish to perfect their knowhow and avoid erring or missing serious things that threaten the life of a patient. One needs to remember that an ECG reading is not always enough to diagnose the problem and in case of doubt one needs to seek the advice of a cardiologist. This is reflected in our daily practice and nowadays general practitioners, if faced with chest pain, do not perform an ECG rightly so but rather refer the patient to the ER to get a more enlightened opinion.

For us, simple messages need to be disseminated. Firstly, one needs to know the normality and know how to measure PR, QRS and QT intervals….. One needs to know and analyze heart rhythm and conduction disorders. Last but not least, one needs to master the electrical interpretations of ischemic heart diseases namely myocardial infarction and frequent and serious medical conditions. In sum, the reader has to come up with simple and clear ideas. Either ECG is abnormal and constitutes an immediate threat to the patient's life, or ECG is abnormal and requires a non urgent opinion by a cardiologist. Or ECG is normal or shows a benign disorder in which case the patient may leave.

The idea of this issue is very appealing. We leave it to you to judge how pertinent it is and wish you a pleasant reading.

Nagi Souaiby, MD, MPH, MHM
Chief Editor

Continuous Education: Special ECG (outline)

SIMPLIFIED ELECTROCARDIOGRAM : A PRACTICAL GUIDE FOR ALL
GOTTWALLES Y, KEMPF N, SAVINEAU-RAETH JR.

General information

SIMPLIFIED ELECTROCARDIOGRAM
A PRACTICAL GUIDE FOR ALL

GOTTWALLES Y. KEMPF N. SAVINEAU-RAETH JR. Simplified Electrocardiogram: a practical guide for all. Med Emergency, MJEM 2014; 18:3-54.
Keywords: ECG, rapid reading, daily practice.

ABSTRACT

Objective: ECG is a simple tool that could be performed easily. Its contribution in the diagnosis can be of major importance especially if properly read and correlated to the clinical diagnosis. This work aims at providing a rapid and easy interpretation of an ECG based on clear landmarks or point of reference allowing the practitioner to detect ECG anomalies especially those predictable serious disorders requiring immediate attention. This simplified method is of great sensitivity and can be applied with certainty and provide a diagnosis in 98% of the cases.

Design and methods: this work is the result of reviewing the literature and proposed practical guides. But most importantly, it is mainly the result of daily practice in emergency departments. A systematic approach is proposed. Separate sections will be discussed consecutively in order to facilitate the reading flow. The following topics will be tackled: Anatomical and electrophysiological pre-requisites, P wave, PR interval, QRS complex, T wave, rhythm conduction and repolarization abnormalities, ECG data analysis and traps to be avoided. Practical exercises and a summary of the main points to be remembered are suggested at the end of each section. An algorithm and practical exercises will be suggested at the end of each section. Last but not least, a summary of the main points to be remembered is presented.

Importance: every professional practicing in the emergency field finds himself facing these two simple questions: is ECG normal? If not, is it serious? The importance of this method relies in providing a rapid and easy reading. It is more sensitive for approaching with near certainty the positive diagnoses and is specific to severe pathologies because it does not miss some rare but relatively severe diagnoses such as Brugada syndrome or the Wolff Parkinson White syndrome. The clinical correlation is always remembered and highlighted.

Conclusion: this special edition will undoubtedly be the "bedside book" for every professional wishing to leverage all information ECG reading can provide in order to have an efficient diagnostic approach. This long journey with small squares and electrical impulses will be concluded with a simple algorithm.

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INTRODUCTION

Similarly to the measure of arterial pressure or of capillary glycemia, the surface electrocardiogram (ECG) must be a common tool for all physicians, independent of their specialty. However, its routine versatility collides with certain pitfalls such as the tremendous variability in pattern between individuals, the variability among electrical leads, and the lack of practice or training on how to read an ECGs.

For a long period of time, the teaching of ECGs and its use have remained standardized, based on certain conventions, and limited access have repelled practicing physicians who are not used to reading 25 to 30 ECGs per day.

Throughout these following sections, we will propose a new simplified approach to interpreting and reading ECGs, an approach based on restricted number of guidelines and principles, allowing for the classification of various strips into 3 categories:
• The strip appears normal
• The strip is abnormal, but it is not worrisome in the immediate future
• The strip is abnormal, and the patient requires immediate attention.

Whether the interpreter has a medical or paramedical background, by respecting a few basic principles, 10 seconds are enough to interpret an ECG strip. Nevertheless, a warning is necessary: in Medicine, 100% certainty does not exist. This method, forcibly restrictive in its approach, does not demean the rule, but rather allows it to be applied with certainty and allows for a diagnosis to be made in 98% of the cases.

The following topics will be successively tackled:

I. Anatomical and electrophysiological pre-requisites
II. Atrial depolarization: P wave
III. Ventricular depolarization: QRS complex
IV. Repolarization: ST segment, T wave and QT interval
V. Rhythm disturbances
VI. Conduction abnormalities
VII. Repolarization abnormalities
VIII. Traps to be avoided
IX. Electrical / Clinical correlation
X. Conclusion: Algorithm

I - ANATOMICAL AND ELECTROPHYSIOLOGICAL PRE-REQUISITES

The ECG strip is a recording of action currents produced by the cardiac muscle. It is generally interpreted by a strip that regroups 12 to 18 conventional leads.

Action currents are the summation of each myocardium cell's membrane potentials (electrical gradient on the cell membrane) the electrical graph records the variations of this summation, and according to the location of the recording cell, this sum will have a different resulting aspect.

Several observations are therefore necessary:

• The electrical activity of the heart is separate from myocardial cell. This will be discussed further in the following sections.

• Myocardial cell is endowed with several properties:

- Excitability: an electrical stimulus will induce a reaction, that is the depolarization of the cell which will be followed by a contraction of the heart muscle,

- Conductibility: the cell has the ability to transfer electrical impulses,

- Automaticity in certain conditions: if the cell is not energized, it has the ability to be spontaneously activated in order to preserve its function, that is myocardial contraction.

• ECG is the result of the electrical activity in the myocardium and not in the conducting tissue of the heart.

In order to obtain a recording of quality, one must remember that an action potential is electrical, thus electricity implies conduction. Therefore, one must:

• Improve the conductivity of interfaces (use a conductive fluid),

• Eliminate potential artifacts (decrease muscular artifact, immobilized subject, with no muscular activity, patient must breath slowly and avoid talking),

• Decrease main interference (earthing, recording with filters activation),

• Place the leads on a clean shaved skin and in very precise locations of the body.

The propagation of the electrical wave in the heart creates a vector loop, which is the combination of the direction and the amplitude of each cardiac activation vector; the electrical axis of the heart is the main direction towards which the electrical loop is oriented.

Birth of currents of action, automaticity and conduction

All processes of activation originate from the sinus node under normal conditions [1]. Spontaneous activation is produced in the sinus node found above the right atrium, and subsequently extends to the adjacent auricular myocardium, while diffusing from cell to cell (cell conductivity), in order to reach the atrioventricular node (under the right atrium). The propagation of electrical impulses in this nodal tissue occurs uniquely from cell to cell, before reaching the sino-atrial node (Keith and Flack) where conduction becomes filamentous (it follows the electrical wire) followed by the atrio-ventricular node (Aschoff-Tawara), bundle of His, the branches of this bundle, and the Purkinje network that ramifies under the endocardium. For any myocardial cell that is in contact with these nervous conducting structures, propagation from cell to cell is once again the rule.

The 12 leads

Their use is purely conventional with no logical or scientific basis. We distinguish the following leads **(Strips I-1 and I-2)**:

- The extremity leads:
  - Augmented unipolar extremity leads (aVR, aVL, aVF),
  - Bipolar extremity derivations leads (DI, DII, DIII).
- The precordial or chest leads:
  - Precordial leads (V1, V2, V3, V4, V5, V6), and
  - Other precordial leads (right, epigastric, upper precordial, posterior…)

The three bipolar leads form the sides of the Einthoven triangle, a theoretical equilateral triangle where the heart occupies the center. The bipolar and unipolar derivations of the extremities study the cardiac electrical activity on a frontal plane, and are depicted on the Bayley triple axis.

Morphology

The baseline wave is either positive (the propagation of electrical activation moves towards the electrode), or negative (the electrical activation moves away from the electrode), or isoelectric (zone of transition or inactivity). The morphology varies according to the variations in apparent amplitude of the activation wave: Each lead is an eye that watches the displacement of the electrical vector **(figure I-1)**; it is the same effect as watching a train:

- When the action potential comes towards the eye, then it is shown as positive,
- The more it comes towards the eye, the greater is the amplitude,
- When the action potential goes further from the eye, the wave becomes negative,
- The further it goes from the eye, the greater is the amplitude,
- The morphology varies according to the location of the recording electrode.

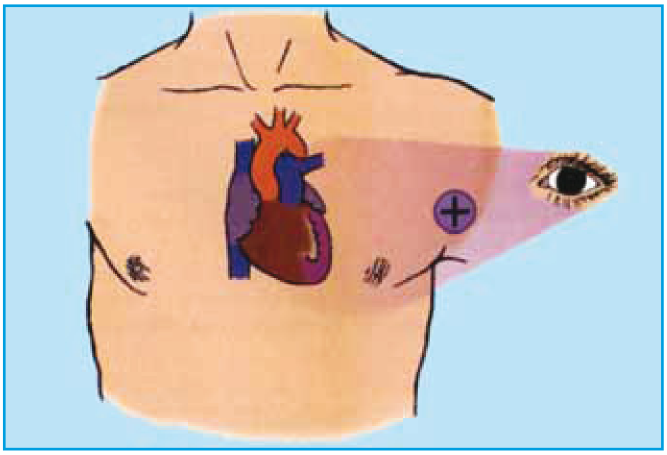


Figure I-1

Duration

Each deflection of the baseline wave is characterized by its duration and the duration of each wave is determined by the properties of the conducting cells. A prolonged duration is a sign of pathology in conduction, either organic or functional.

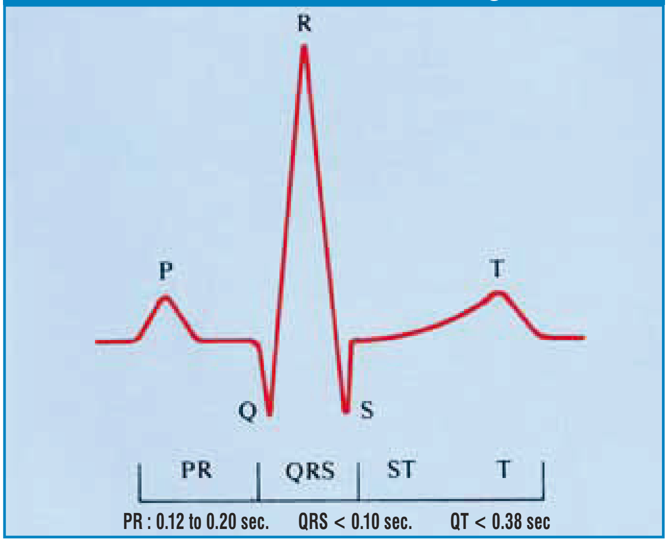
Amplitude

It is the result of the amount of material in action, which is a function of the sum of the amount of the stimulated material and of the amount of tissue that was crossed.

Baseline wave

Three deflections are recognizable, the P wave, QRS complex and the T wave followed by a U wave.

Elements of the electrocardiogram



P : Atrial depolarization. QRS : Ventricular depolarization.
T : Ventricular repolarization.

Nomenclature of the QRS complex

The presence and the relative size of different components are designated by the letters q, r, s, Q, R, S, T, and U. the deflections of large amplitude are designated by the appropriate upper case letter. The deflections of small amplitude are designated by the appropriate lower case letter. If the primary wave is positive, it is termed r or R. If the primary wave is negative, it is designated by q or Q. the secondary negative wave is termed s or S. Any strictly negative wave is termed qs or QS. All second upward deflection positive waves are termed r' or R' (R prime).

An important observation concerning aVR: aVR measures the cardiac cavity from its endocardial side, and/thus the depolarization occurs from the right atrium towards the ventricles. The current moves away from aVR, therefore aVR is always negative, thus a QS aspect can be normal in aVR.

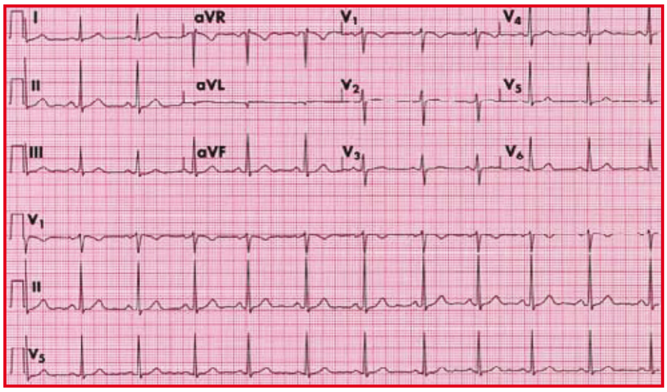
Normal strip

Two major obstacles are potentiated when interpreting an ECG: first, a tremendous variety of different electrocardiographic images exist for the 12 derivations leads of the same subject. Furthermore, a large variety of electrocardiographic images contain a random lead within a population of normal subjects.

Normality Criteria

There are two types of leads: extremity leads and precordial leads. Duration, amplitude, morphology, area of interest will be successively analyzed [2].

However, this analysis is narrowly linked to technical recording conditions. The calibration of the instrument must be such that a test of 1 mV must provoke a rectangular signal of 1 cm in height. The speed of the course is usually 25 mm/sec, each mm corresponds to 0.04 sec, 5 mm at 0.2 sec, and 25 mm at 1 sec.



Strip I-1. 12 leads strip with long strip in V1, DII and V5.

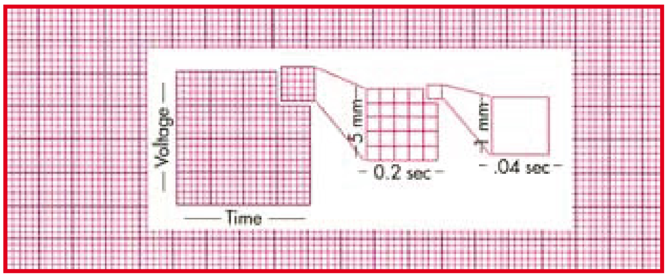
Sometimes, a standard tracing is inconclusive, and the recording must be either repeated or extended.

The isoelectric line must be as horizontal and clear as possible, exempt from parasites, and the cables must be plugged in the right order.

The graph must contain at least 12 main leads, in the following order: DI, DII, DIII (bipolar leads) then aVR, aVL, aVF (unipolar extremity leads) then the 6 precordial leads from V1 to V6.

Basic rules for a rapid reading

Electrical depolarization and repolarization successively follow each other, but: the repolarization depends on the depolarization, which in turn depends on the conduction, which in turn depends on heart rate, which in turn depends on the rhythm. It is therefore necessary to read an ECG in a logical sequence, in order to notice all the anomalies and offer an overview. This overview will be all the more precise given the precision of the clinical context.



Strip I-2. Time unit in the horizontal axis and voltage amplitude in vertical axis.

Main points to be remembered

The ECG tracing is:

1. The electrical activity of myocardial cells.

2. The sinoatrial node is endowed with automaticity and conductivity that predominates the activities of the underlying chambers.

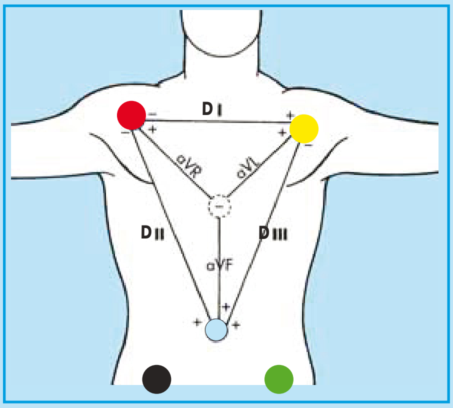
3. 12 leads: 6 peripherals (3 unipolars and 3 bipolars) and 6 precordial leads.

A lead keeps an eye on the current:
- If the current comes towards it: positive,
- If the current moves away from it: negative,
- If the current is flat: isoelectric.

4. A morphology (direction, duration, and amplitude):
- Direction: main direction of the current,
- Duration: conduction,
- Amplitude: amount of stimulated material plus amount of material that was crossed.

5. A graph where 1 cm = 1 mV and 25 mm = 1 sec.

6. A succession of PQRS ± U waves, in which aVR is always negative.



Red electrode **right arm**
Yellow electrode **left arm**
Black electrode **right thigh**
Green electrode **left thigh**

II – ATRIAL DEPOLARIZATION: P WAVE

The anatomical and electrophysiological prerequisites being acquired, the only way to be efficient in reading and interpreting an ECG is a regular practice, using a systematic interpretation, since the logic of the reading remains the chronological order. Please note that contrary to the other sections, captions are placed above the strips for a better understanding.

P wave

In principle, the first visible wave on a strip is the P wave. It corresponds to the depolarization of the atria, the right atrium first, the activation occurring from the sinus node, then the left atrium after the propagation of the impulse.

The P waves have a varied morphology, which depends on:

• The size of the atria: The deflection should be monophasic, however in case of auricular hypertrophy, a biphasic or a hypertrophic aspect are possible,

• The derivation of the reading of the graph: The best leads to recognize P waves are DII, DIII then V1,

• The localization of spontaneous activity or natural pacemaker:

- Under normal physiological conditions, the sinusoidal node is located above and at the right of the right atrium, the propagation of the impulse being from cell to cell toward the bottom, the front and the left.

- The global electrical axis of the heart connects the right medioclavicular point to the left elbow.

- In case of low, atrial extrasystole or a spontaneous activation taken up by another node, the morphology will be altered.

• The type of cardiac rhythm:

- Under normal physiological conditions, the rhythm is regularly sinusoidal.

- When the natural pacemaker node is defective, its responsibilities can be taken up by another node located outside of the atrium.

- When the rhythm disturbances originate in the atria, the morphology of the wave is altered [3].

Normality Criteria

The P wave has a small, rounded deflection. It is always positive from V3 to V6 and can be positive or diphasic in V1 and V2. Its amplitude is less than 2.5 mm in all leads.

It is normally followed by an isoelectric segment which separates it from the beginning of the QRS complex, falsely named PR. This space is measured from the beginning of the P wave to the beginning of the Q wave. It should be constant from one cycle to the next, and has a duration ranging from 0.12 to 0.20 sec, this duration being proportional to the heart rate.

Any P wave should be followed by a QRS complex, and the same P wave should be identical during each depolarization, guaranteeing the normality of the sinus function.

The frequency of stimulation of the sinusoidal node is variable depending on the physiological state, physical activity, training of the individual, gender [4], and living conditions. A frequency between 50 and 120 bpm triggers the persistence of a sinus rhythm. A frequency lower than the aforementioned must evoke a disturbance in conduction associated with the absence of obvious physiological causes (training, chronotropic treatment). A frequency that is higher must localize the startup of the rhythm, a thin QRS complex being in favor of a supraventricular tachycardia, widened complexes are suggestive of rather a ventricular tachycardia, in the absence of aberrant conduction of the branch.

P wave analysis will allow answering the following questions:

• Can the rhythm be defined? Is it sinusoidal, regular, suggestive of bradycardia or tachycardia?

• Did the rhythm originate from the sinusoidal node? Is its origin supra-ventricular? Does another ectopic source exist?

• Is the supra-ventricular rhythm arrhythmic, suggestive of atrial tachycardia, flutter or fibrillations?

• In the absence of a P wave, the lower the cardiac heart rate, the more the escape rhythm will be triggered.

• In the presence of a pacemaker, the type of stimulator can be recognized (AAI, VVI, DDD,…)

All aforementioned analytical elements will have a therapeutic implication that will sometimes be immediate or short-termed according to its clinical impact.

Types of P-wave graphs

During any ECG interpretation, two major questions must be tackled:

1. Does a P wave exist?

2. Is every P wave followed by a QRS complex?

The rhythm is considered to be supra-ventricular when it originates above the ventricular layer.

The morphology of P waves will be either rapid dispersion, or a dome evoking an organization in wide intervals, or a variation in the isoelectric line which evokes a more anarchic organization.

Strip II-1. P exists, and is always followed by a QRS complex.

This is a regularly sinus rhythm. P waves are identical amongst themselves followed by a thin QRS. Although the rhythm is slightly fast, it is considered normal.

Strip II-2. P exists and it always followed by a QRS complex.

The rhythm is sinusoidal, regular, and the P waves are identical, the PR is normal, the QRS complexes are thin. The rhythm is normal.

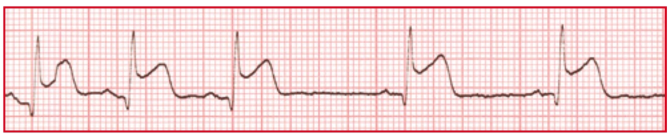
Strip II-3. P exists, it is always followed by QRS complex, but the rhythm is irregular and the morphology of P wave is variable.

The rhythm is sinusoidal, regular on complexes 1,2,4 and 5. On the 3rd complex, P wave exist but is negative, and QRS complex remains identical to the others. This is the case of an atrial extrasystole that originates in the bottom side of the atrium, ascends against the current to the sinusoidal node. There is a compensatory pause.

Strip II-4. P exists but not always, and the QRS have different aspects.

In the first 3 complexes, the rhythm is sinusoidal, regular, with thin complexes. Complexes 4 and 5 are aberrant, widened, with an opposite axis to that of the first QRSs. Complex 6 slims down, resembling a bit more to the morphology of the first QRSs. Complex 7 is identical to the first QRSs and is also preceded by a P wave, and complex 8 is similar to the others although less wide. This is the case of a regularly sinus rhythm on the first 3 complexes, followed by a triplet of premature ventricular contractions (PVCs), a normal QRS, and then another PVC (the QRS is quasi-identical but there is no P wave).

Strip II-5. P exists and the number of P waves= the number of QRS waves.

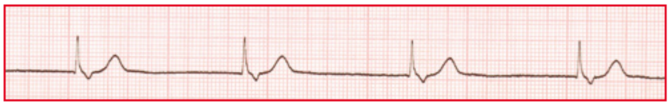


The rhythm is sinusoidal, with clearly visible P waves. Each wave is followed by a complex; the rhythm is irregular. In the context of a myocardial infarction, a bradycardia takes place after the 4th complex, with a PR that remains subtly identical.

Strip II-6. P exists and is always followed by a QRS.

The rhythm is sinusoidal, regular, with visible P waves, an equal number of QRSs but a prolonged PR, well above 0.20 sec (5 small units or 1 large unit): this is the case of a first-degree atrioventricular block.

Strip II-7. P wave is not seen.



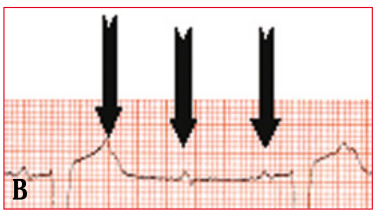
The rhythm is slow (30 bpm), the QRS complexes are thin and there is no P wave. But, at the end of S, there is a small negative wave: this is a ventricular escape rhythm with a retrograde P wave.

Strip II-8. The P waves exist, there is always a QRS that follows, the rhythm is irregular and the P waves are negative.

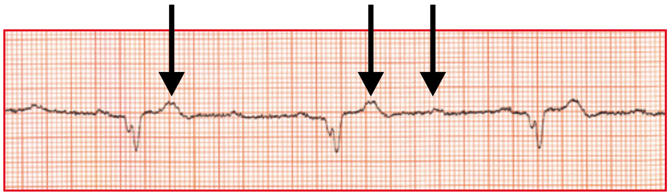
The presence of negative P waves in inferior leads is a sign of their origin being close to the coronary sinus. It is called “coronary sinus rhythm”.

Strip II-9 A and B. P wave exists but there are more P waves than QRS complexes.

The P waves are clearly visible. We can locate 11 of them on the strip, associated to 4 wide QRS complexes. The PR between the second P wave and the first QRS complex is identical in the following complexes: this is a sinus rhythm with an atrioventricular block and a conduction of type 3/1. For every three stimulations, a complex is generated. It is important to note that P waves are sometimes overlapped by the peak of the T waves.

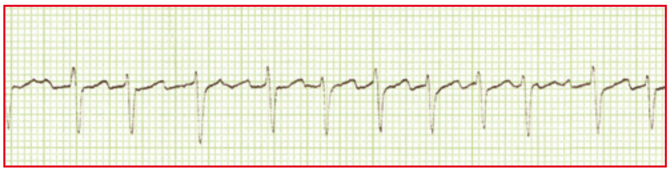


Strip II-10. P exists but there are more Ps than QRS complexes.



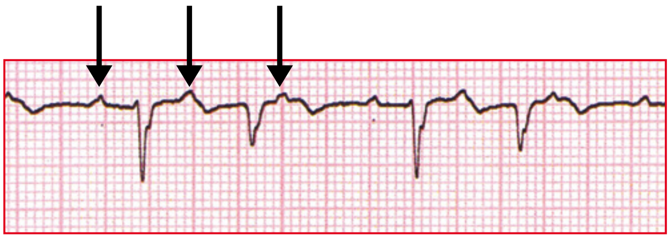
P waves are a little less visible compared to the former strip, but the same observations can be made about this strip: disturbances in conduction of type 3/1, in addition to a PR at 0.24 sec when P is generated.

Strip II-11. It is difficult to see the P waves, the rhythm is irregular.



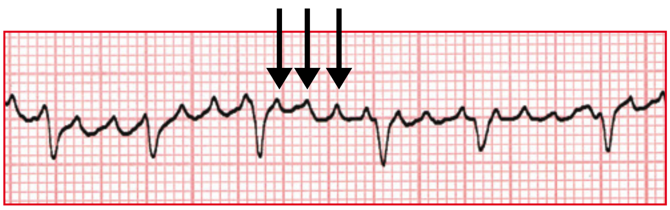
When looking at this graph, it is important to concentrate on the zones where the heart rate is the slowest, namely during the largest space between two QRS complexes (between the 3rd and 4th complexes or between the 9th and 10th QRS complexes). If a P wave appears between the 9th and 10th, we tend to see two P waves between the 3rd and 4th. When reporting this period between the two supposed P waves, we see P waves at a frequency of more than 300 bpm, with a variable conduction of QRS complexes. This is the case of supraventricular tachycardia.

Strip II-12. We can see P waves, QRS complexes are irregular and there are more P waves than QRS complexes.



There are P waves. They are sometimes followed by QRS complexes but not always. The first two P waves are followed by a QRS, but not the third. This pattern is repeated. The QRS complexes remain thin but are heterogeneous. This is the case of an atrial tachycardia at 150 bpm with a conduction of type 3/2.

Strip II-13. We do not see a P wave, but rather a tremor that seems slightly regular, with thin but irregular QRS complexes.

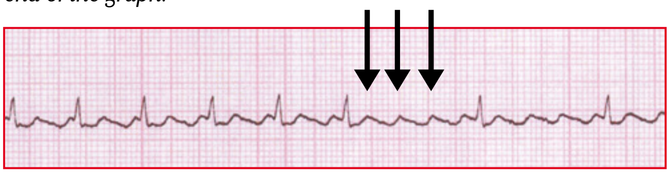


The rhythm is variable, reaching 120 bpm, with semblances of narrow and rapid P waves of 450 bpm. The QRS complexes remain thin. This is a supraventricular tachycardia with variable conduction. The structure of the P waves appears to exist when compared to an atrial fibrillation.

Strip II-14. We do not see a clear P wave, but rather a dome between every QRS.

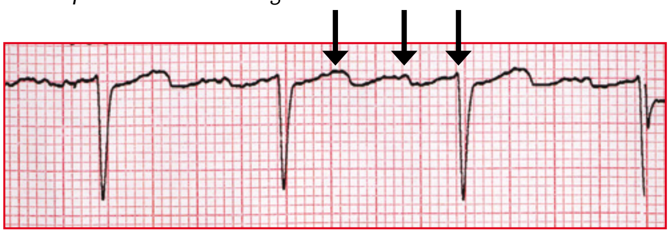
The rhythm is rapid, in the order of 150 bpm, with thin QRS complexes and rounded saw-like teeth structures instead of P waves. The base of each QRS complex is widened. It is actually a second dome superimposed on the QRS complex. This is a supraventricular tachycardia of artial flutter type, with a conduction of type 2/1.

Strip II-15. We do not see a P wave, but rather a clearer tremor at the end of the graph.



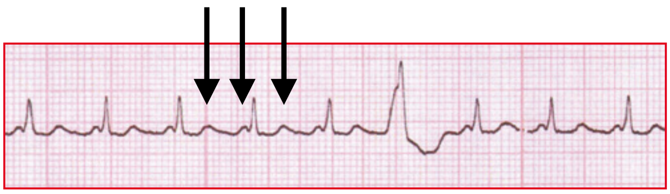
In the first part of the strip, the rhythm is rapid, in the order of 140 bpm. In the second part, the rhythm is slower and reveals a tremor with a sawtooth waveform, typical of an atrial flutter. The deceleration was provoked by a carotid compression.

Strip II-16. We do not see a clear P wave, but rather a slow tremor with two slopes with different angles.



The rhythm is slow, the QRS complexes are thin and there are slow tremors between the QRS complexes, three times more numerous than the QRS complexes, with a sawtooth waveform. This is an atrial flutter of conduction of type 3/1.

Strip II-17. We do not see a clear P wave…



The complexes are thin, except the 6th one. There is a deflection before each QRS complex, but is practically attached to it. This deflection is so close that it cannot allow physiological conduction. As such, this means that another wave exists before that particular one that is conducting (complexes are too thin to be ventricular, a ventricular extra-systole confirms it, and the rhythm is too fast to have originated at the level of the ventricle): this is therefore a supraventricular tachycardia and, when having a closer look, P wave that is conducting is confounded with the preceding T wave, and there is a conduction of type 2/1.

Strip II-18. We do not see a P wave, and the complexes are rapid and thin.

There is no visible P wave, the rhythm is rapid in the order of 180 bpm and the complexes are thin. This is a tachycardia of Bouveret type.

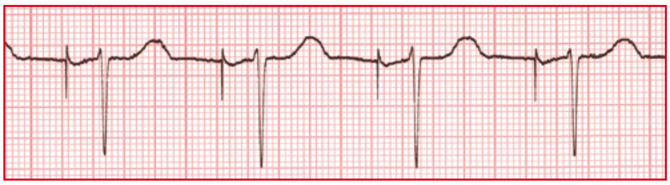
Strip II-19. There is no clear P wave, but rather a tremor in the baseline.

This is an auricular fibrillation with an average ventricular frequency in the order of 100 bpm, suggestive of a complete arrhythmia by atrial fibrillation.

Strip II-20. There is no clear P wave, but rather an overall tremor of the line, intercepted with wide QRS complexes, preceded by a micro-spike.

This is atrial fibrillation with a VVI pacing mode.

Strip II-21. There is no natural visible P wave.



Permanent auricular electrostimulated rhythm: the auricular spike is clearly visible, followed by a discretely lengthened PR at 0.22 and by a spontaneous ventricular complex: this is either an AAI pacing mode (auricular detection, auricular stimulation) or a DDD pacing mode (auricular and ventricular detection, possible auricular and ventricular stimulation) for which there was no ventricular stimulus, the QRS complex being triggered by nature.

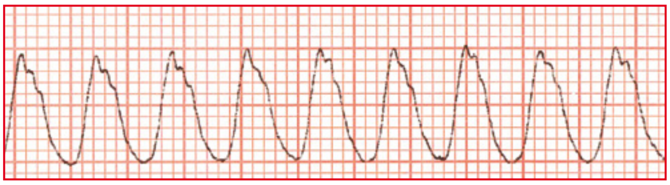
Strip II-22. We do not see a natural P wave.

The rhythm is permanently electrostimulated, there are no P waves or atrial spikes, there are only ventricular spikes. This is an electrostimulated rhythm in VVI mode with an AF background.

Strip II-23. There are no clearly visible natural P waves, but rather double spikes.

The rhythm is regular, permanently electrostimulated in the atrium and ventricle: this is a DDD stimulator, with auricular stimulation (first spike), a PR equivalent at 0.12 sec, then a ventricular spike followed by a ventricular complex.

Strip II-24. We do not see a P wave, and then complexes are very large.



There are no P waves, the rhythm is rapid in the order of 280 bpm, and the complexes are very wide. This is supposedly a ventricular tachycardia, to be confirmed by other leads.

Main points to be remembered

Everything depends on the P wave:

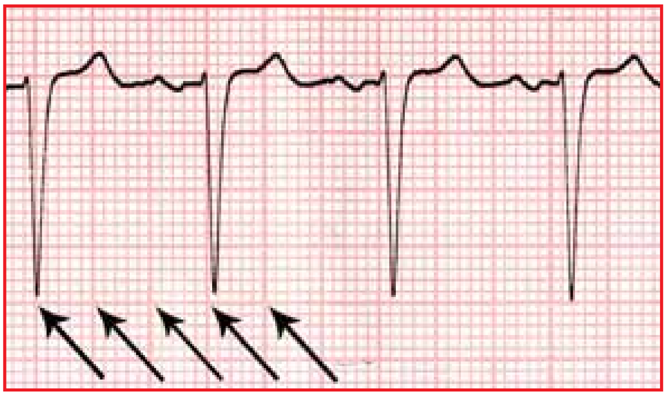
1. P is most visible in DII.
2. P wave should exist.
3. All P waves should be followed by a QRS complex.
4. There can only be one identical P wave before each QRS complex.
5. The PR interval is of constant duration, between 0.12 and 0.20 sec (3 to 5 small units).
6. The usual sinus frequency is between 50 and 120 bpm.
7. A high frequency is suggestive of a SVT.
8. A tachycardia with wide complexes is suggestive of a VT.

III – VENTRICULAR DEPOLARIZATION: QRS COMPLEX

Following the P wave on a normal graph, QRS complex reveals the propagation of electrical stimulation across the ventricular myocardium. It is generally, but not always, the largest deflection on the electrocardiogram. It usually has a pointy shape that varies based on the analyzed lead.

Heart rate

The heart rate is determined by the frequency of QRS complexes, whether they are preceded by P wave or not. It is approximately calculated according to the number of large squares between two consecutive QRS complexes, abiding by the 300/150/100/75/60/50 rule **(Strip III-1)**.



Strip III-1

5 large squares = 1 sec = frequency at 60 bpm.

1st arrow = one complex.

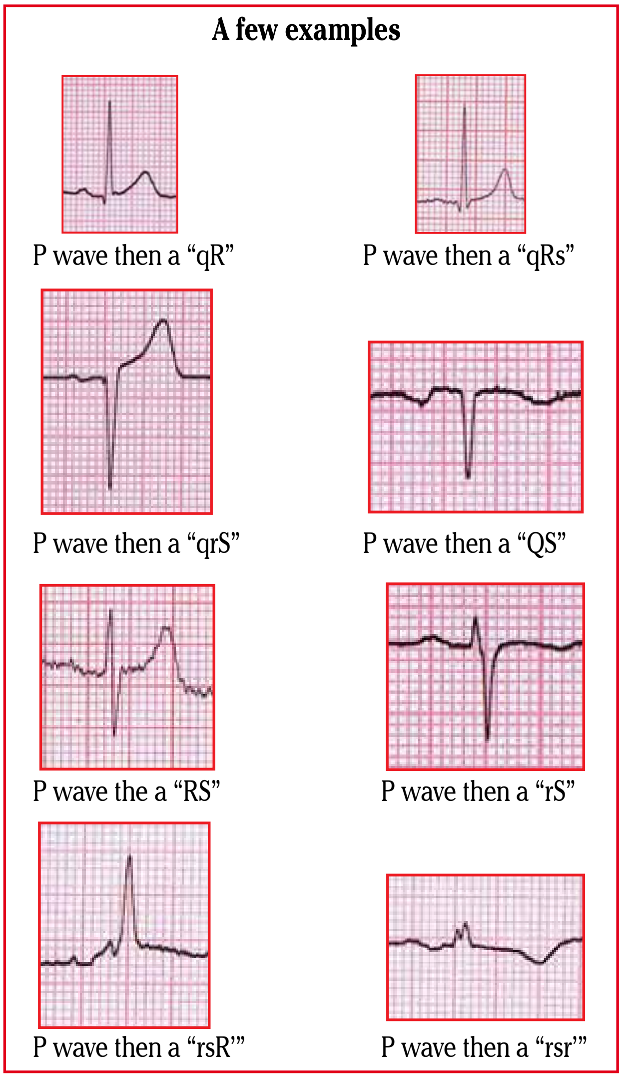
2nd arrow = 1 large square = 300 bpm if the following QRS complex is found at this level.

3rd arrow = 2 large Square = 150 bpm if the QRS complex is found here.

4th arrow = 3 large Square = 100 bpm and the following QRS complex is found slightly after this arrow; the cardiac frequency is found between 100 and 75 bpm, very close to 100 (95 bpm on the complete strip).

Nomenclature

A specific nomenclature exists for the QRS complex: the occurrence and the relative size of the different components are designated by the letters q, r, s, Q, R, S. the first positive wave is designated by r or R, a negative wave is designated by q or Q if it precedes r or R, s or S if it follows r or R. any strictly negative wave is designated by qs or QS. Deflection of large amplitude are designated by the appropriate capital letter, Deflection of small amplitude are designated by the appropriate lower case letter, and all secondary positive waves are termed r' or R' (R prime) **(Strips III-2)**.



Strips III-2

Normality of Q

Its duration should always be below 0.04 sec. Its depth cannot surpass one fourth of the height of the R wave that it precedes, when it involves the precordial region. In fact, it should not exist.

Normality of QRS

The amplitude of the largest R wave in the precordial region must be between 8 and 27 mm. The sum of the largest R wave and the deepest Q wave must be below 40 mm. The maximal duration of QRS is below 0.10 sec. The size of R cannot surpass 13 mm in aVL and 20 mm in aVF. Finally, the normal cardiac axis is between -30° and 90°.

Electrical tracing and ischemia: electrical correlation

The action potentials reflect the viability of all aspects of the myocardium and the conductive tissue [5]. This viability depends on satisfactory perfusion of the tissues. The coronary atheroma can cause an ischemic attack in any part of the myocardium or the conductive tissue, leading to alterations of P waves, QRS complexes, ST segments, or T waves in any lead of the ECG;

any form of irregularities in the rhythm are possible in the case of ischemia of the conductive tissue. This result in a myriad of widespread electrical anomalies, with one challenge and two paradoxes:
• Challenge: ischemic irregularities are added onto preexisting anomalies on the strips.
• Two paradoxes: a widespread atheroma and a normal ECG can co-exist, much like infarcts of the epicardial coronary arteries that appear healthy on an angiogram.

The ECG is not an infallible diagnostic tool of ischemic cardiomyopathies. However, there are two anomalies that can hint to ischemic cardiomyopathy, such as an abnormally low voltage of the R wave in a localized zone or abnormal Q waves. These anomalies depend on and are a consequence of the reduction in the quality of the living myocardium or of its total absence in comparison with the exploratory electrode.

Loss of R wave voltage

Under normal circumstances, the progressive increase in thickness of the underlying myocardium can cause a progressive increase in the positive or upward deflection: the R wave increases, S decreases in parallel, and V6 grossly mirrors V1 **(figure III-1).**

In case of an ischemic attack, there is a decrease in the thickness of the viable myocardium in a localized zone. This criterion is nevertheless not applicable to V1 and V6, and two leads ranging between V2 and V5 are required. A preceding comparative strip is sometimes necessary. The R wave cannot progress normally **(figure III-2).**

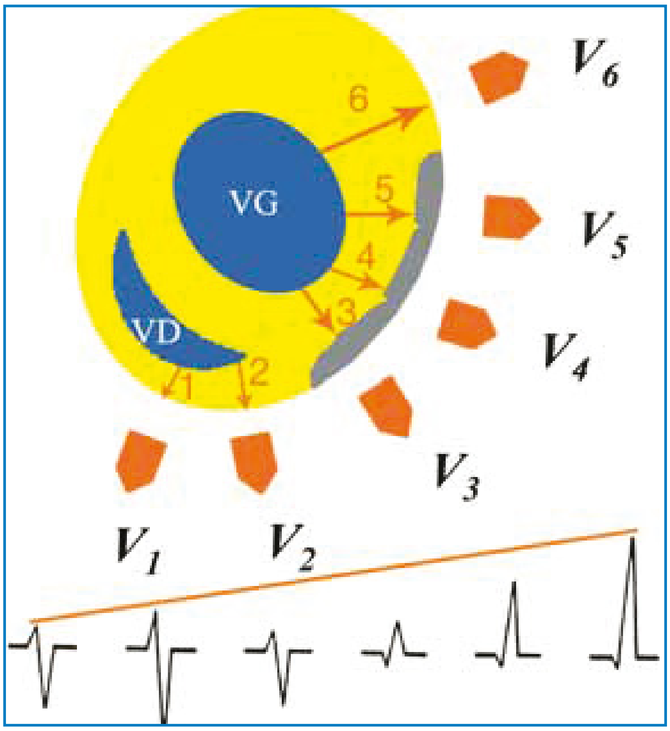


Figure III-2: A nontransmural infarct with regards to V3 to V5. The scar muscle is electrically inert, there is not harmonic progression of the R wave, and a small Q wave could appear.
The strip will record successively the following:
- In V3, a rS aspect following the planing of the R wave,
- In V4, a qr or a qR aspect,
- In V5, a qR aspect,
- In V6, a normal aspect of the complex.

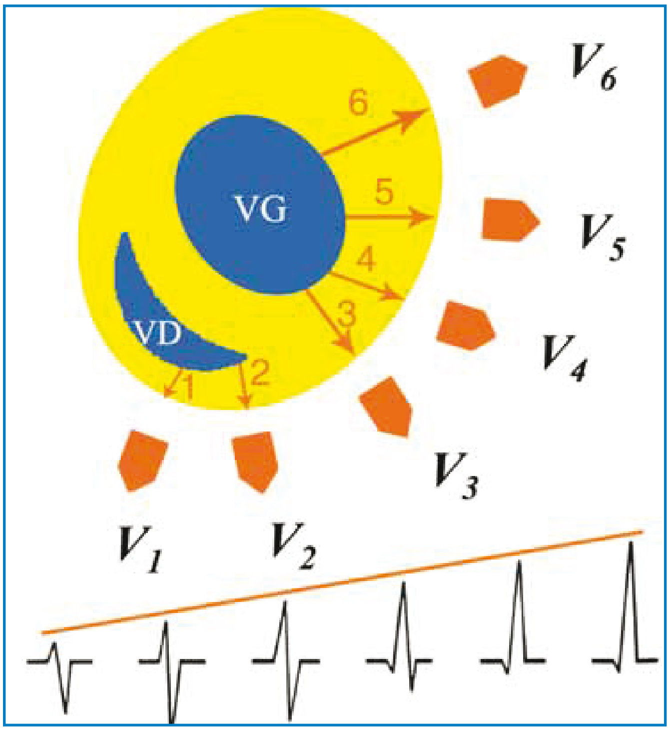


Figure III-1: Transverse cross-section of the myocardium with muscle walls, the left ventricle (LV) and the right ventricle (RV). The evolution and normal progression of the R wave from V1 to V6, with respect to the thickness of the dissected muscle.

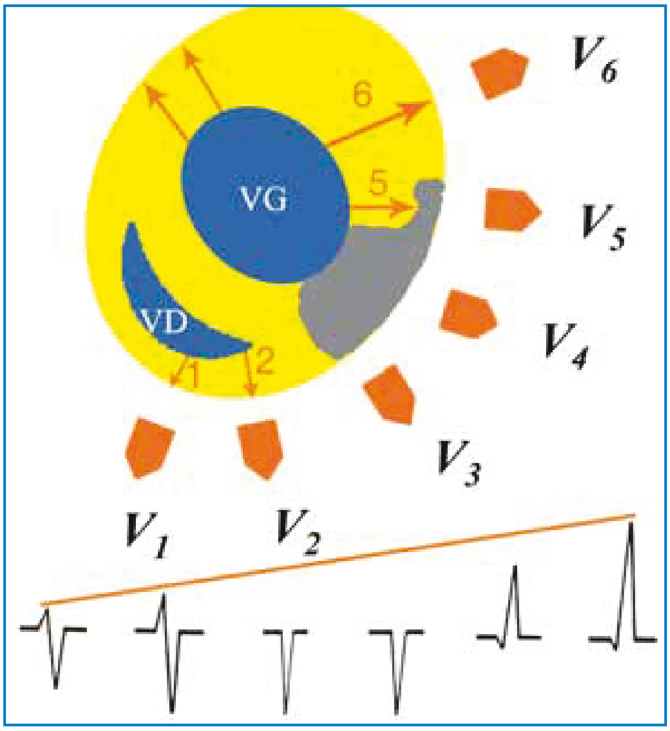


Figure III-3: During a transmural infarct with regard to V3 and V4, and non-transmural with regards to V5, there is a QS aspect in V3 and V4, and a qS aspect in V5. The QS aspect corresponds to the posterior side of the myocardium, which is the only electrically active part with regards to V3 and V4. It is actually a mirror image of a posterior R wave, V3 being the mirror image of V9.

For a transmural infarct, there is a total disparity amongst the R waves. The electrical graph is not influenced by the underlying electrically inert myocardium, but it remains influenced by the opposite wall, which means that the action potential is moving away from the electrode, which explains the QS aspect **(figure III-3)**.

Depending of the thickness of the affected myocardium, we can note a sequential reduction in the voltage of the R wave, an abnormal Q wave, a Q wave, and a reduction in the R wave, followed by a QS complex.

Bundle Branch blocks

We can distinguish the right bundle branch block (RBBB), the left bundle branch block (LBBB), the hemiblocks amongst which we can find the left anterior hemiblock (LAHB) and the left posterior hemiblock (LPHB) and finally the possible associations RBBB+LAHB and the RBBB+LPHB.

Right Bundle Branch Block or RBBB

There is a problem in conduction in the right branch of His. The impulse will propagate through the left branch, and is then transmitted via Purkinje fibers to the right ventricle. As such,the conduction is slowed down across the myocardium, with a delay in depolarization of the right ventricle and a delay of repolarization in the right ventricle. As a result, the entire duration of the QRS complex is prolonged (≥ 0.12 sec or three small squares) and a second depolarization appears. We also note the appearance of a secondary R wave in V1 that is wide and plump, and the appearance of a deep S wave in V5, V6: this is the typical appearance of V1 in M or bunny ears shape **(Strips III-3)**. It can be either complete or incomplete if the duration is less than 0.11 sec.

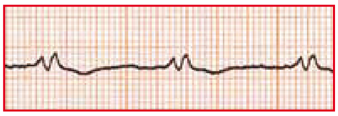
The initial segment of the QRS complex is not modified in the case of an RBBB, and the criteria of normality or abnormality of voltage, of the progression of the R wave, and of the Q wave can be applied. Consequently, we can diagnose an infarct, a ventricular hypertrophy or any other cardiomyopathy. It can be either complete or incomplete if the duration is less than 0.11 sec.

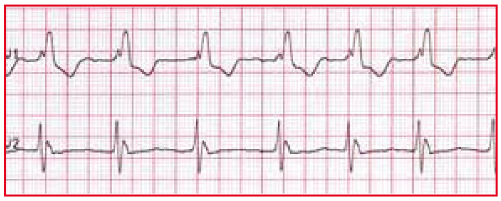
A RBBB can be seen in ischemic cardiomyopathies, respiratory pathologies with a cardiac repercussion, arterial hypertension, pulmonary embolism, cardiomyopathies, Chagas disease and congenital caridomyopathies (CIA, Fallot, etc.). A RBBB is not synonymous to an underlying cardiomyopathy and can rather be physiological in an athlete with no clinical or pathological implications.

Left Bundle Branch Block or LBBB

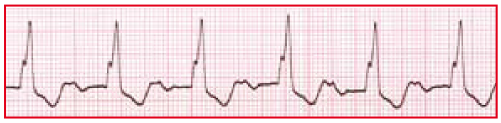
There is a problem in conduction in the left branch of the bundle of His, with a septal depolarization occurring in the inverse direction. The depolarization and return to normal in the left ventricle are delayed; the conduction takes place through the Purkinje fibers originating from the right branch of the bundle of His. Given that the left ventricle is the most electrically active, there are major alterations in the QRS precordial complexes with a prolonged overall duration, with the absence of the R wave in V1, the absence of the septal Q wave in V5, V6, and an M or crocheted aspect in V5, V6.

Various aspects of RBBB can be found; below are a few examples with their aspects in V1.


RBBB with arsr' aspect


RBBB with rsR' aspect


RBBB with rSR' aspect

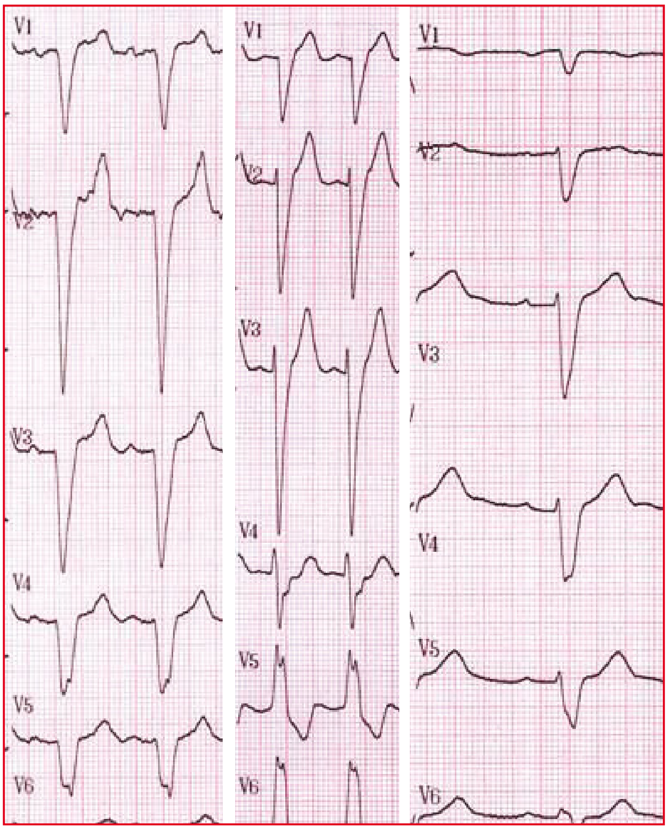

RBBB with RsR' aspect


RBBB with RSR' aspect

Strips III-3

The initial segment of QRS is produced in the opposite direction of the normal in all leads. The criteria of normality or abnormality of voltage, the progression of the R wave, and the Q waves can no longer be applied. As a result, it is no longer possible to diagnose neither an infarct nor a ventricular hypertrophy… according to the standard criteria.

A left bundle block branch always indicates the presence of serious cardiac injury, such as ischemic cardiomyopathies, post-hypertensive cardiomyopathies, shrinkage of a tight and calcified aorta, fibers degeneration of the conductive tissue fibers, congestive and hypertrophic cardiomyopathies, myocarditis after cardiac surgery, and congenital cardiomyopathies.



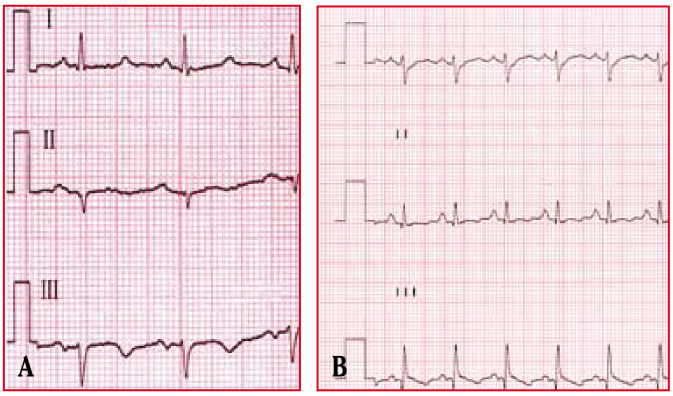
Strips III-4

Here are 3 aspects of the LBBB: wide QRS complex well above 0.12 sec, falsely evoking a QS aspect for the first strip, or an important planing of the R wave in the extended anterior **(Strips III-4)**.

Hemiblocks

There is a rupture either in the LAHB or the in the LPHB of the left trunk of His bundle. The major functional disturbance that follows is a spectacular axial deviation of the QRS axis in the frontal plane **(Strips III-5 A and B)**.

There is an initial R wave in DII, DIII and aVF. Consequently, DII is negative.



Strips III-5 A and B

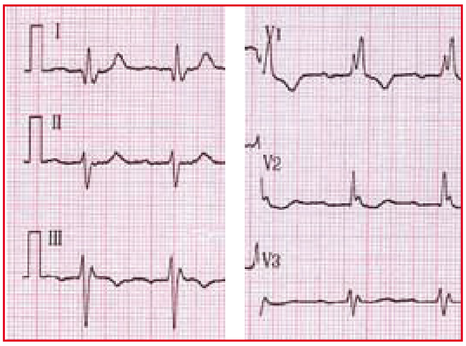
For an LAHB, the average axis on the frontal plane is above -30°

For a LPHB, the average axis on the frontal plane is deviated to the right and lies between +90° and +120°. Consequently, DI is negative.

The possible associations are of two types:

1. Either RBBB+ LAHB or bunny ears in V1 and DII is negative:

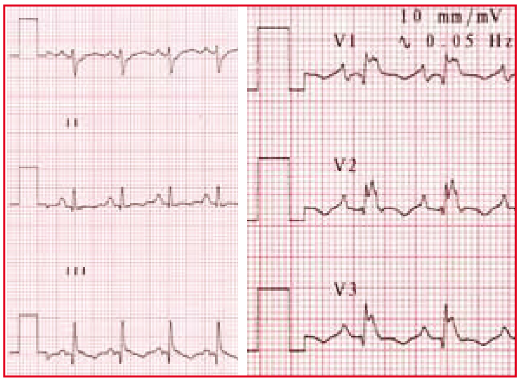
- This association is common,
- However, only 5 to 15% will become a complete atrioventricular block (third-degree artioventricular block or complete heart block) **(Strips III-6)**.



Strips III-6

2. Either RBBB+ LPHB or bunny ears in V1 and DI is negative:

- This association is much less common,
- However, 60 to 70% will become a complete atrioventricular block and its discovery during an episode of malaise, faintness, or loss of consciousness must precipitate this outcome **(Strips III-7)**.



Strips III-7

Main points to be remembered

1. The heart rate is defined by the number of QRS complexes.
2. The QRS is usually positive in DI-DII.
3. DII is negative= LAHB.
4. DI is negative= LPHB.
5. The QRS complexes are very thin, less than 0.10 and normalized in voltage.
6. A normal aspect of QRS is rS in V1, qR in V6, and R wave increases from V1 to V4.
7. A Q wave does not exist, except in the case of a microQ in the frontal plane (<1/3 of R) and a microQ in V5-V6.
8. Bunny ears aspect or M shape in V1 is a RBBB.
9. RBBB is not pathological until proven otherwise.
10. A wide aspect of QS with M shape in V6 is an LBBB.
11. LBBB is always pathological.
12. RBBB + LPHB is a precursor for a complete AVB.

IV- REPOLARIZATION: ST SEGMENT, T WAVE AND QT INTERVAL

The ventricular systole begins with the QRS complex and ends at the end of the T wave. Therefore it encompasses the phases of depolarization and repolarization of the ventricles.

In this section, the phases of repolarization will be analyzed, along with the ST segments, the T wave, and the QT interval.

These three elements are, or can be, severely affected, with possible immediate complications, requiring an immediate patient care, such as coronary ischemia which is considered the most frequent case among the most serious ones.

ST segment

It represents the initial phase of ventricular repolarization, or the plateau phase. The transition between the S wave (or the ascending slope of the R wave in case the S wave is absent) and the ST segment is called point J.

This segment is located on the baseline under normal conditions; it is horizontal and flat, and by definition, is on the same level as the other baseline zones. The problem that is often encountered is the ability to determine this isoelectric line when these strips are performed in emergencies.

In order to be considered normal, ST segment cannot deviate more than 1mm above or below that isoelectric line in any lead. 1mm is significant when considering extremities leads, and 2mm is significant when considering precordial leads.

A deviation above or below ST segment corresponds to a lesion that could be subepicardial (elevation) or subendocardiac (depression).

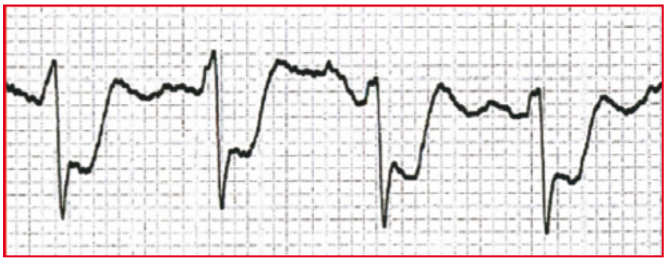
The modifications of the ST segment can have multiple origins: ischemia, ventricular hypertrophy, bundle branch block, medical, or non-specific.

Finally, a ST segment can be (Strips IV-1 to IV-10):

- Isoelectric/flat,
- Shifted below/ depressed,
- Shifted above/ elevated,
- A downward deflection and an upward deflection can be:
  - Elevated,
  - Depressed,
  - Horizontal or rigid.
- Cup-shaped.

Strip IV-1 A and B. Isoelectric ST segment.

Strip IV-2. Downward deflection of ST segment.

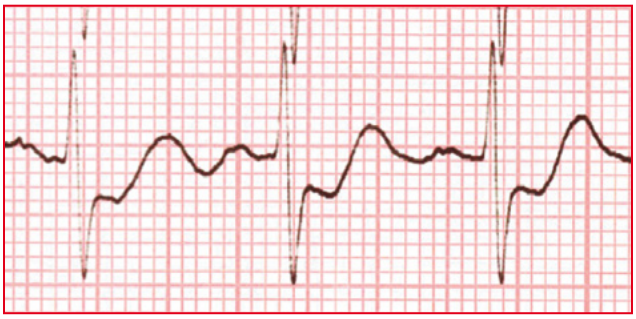


Strip IV-3. Major downward deflection, horizontal, of ST segment; it is the case "Pardee wave" that is inversed, a true mirror, indicative of a menacing coronary lesion.

Strip IV-4. Upward deflection of ST segment.

Strip IV-5. Ascending Segment ST.

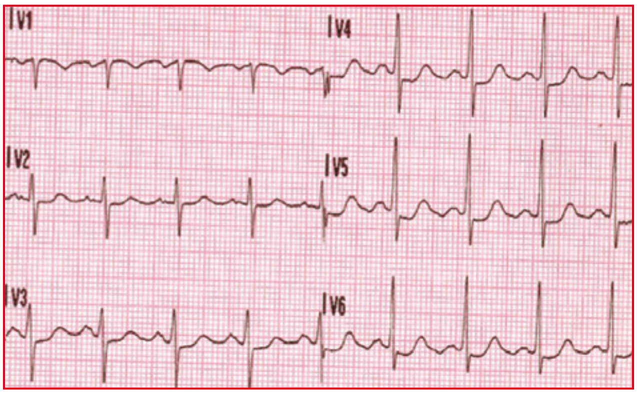
Strip IV-6. Depressed downward deflection of ST segment.



Strip IV-7. Depressed downward deflection straighter than the preceding one.

Strip IV-8. Flat/horizontal, rigid ST segment.

Strip IV-9. Cup-shaped ST segment, on a digital printing.

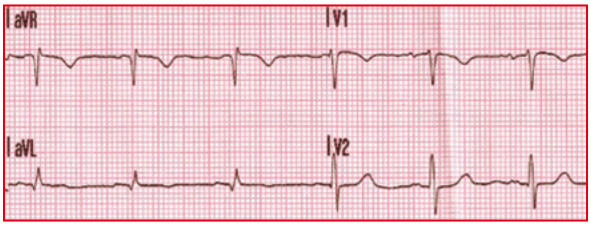


Strip IV-10. Subendocardial injury current extending to the anterior walls, with a ST elevation from V2 to V6.

T wave

This wave represents the final phase of ventricular repolarization, and this phase is rapid and efficient, with the myocytes returning to their regular resting state. Usually it is an asymmetric, rounded, smooth and positive wave.

A normal T wave is positive in all leads, excluding the aVR **(Strip IV-11)**. Variations of the normal T wave include the negative or flat T wave in V1 in 20% of the cases, inverted T wave in V1 and V2 in 5 to 10% of the cases. It is important to remember that a positive T wave in V1 and a negative T wave in V2 is always abnormal, a negative T wave in V4, V5, or V6 is always abnormal and a normal T wave is always asymmetrical. A T wave can be physiologically negative exclusively in DIII, hidden by a concordance with a QRS complex that is highly negative (axial deviation, hemiblock,…).



Strip IV-11. Normal aspect of a T wave with negative T wave in aVR, and negative T wave in V1, positive in V2 (in 20% of the strips).

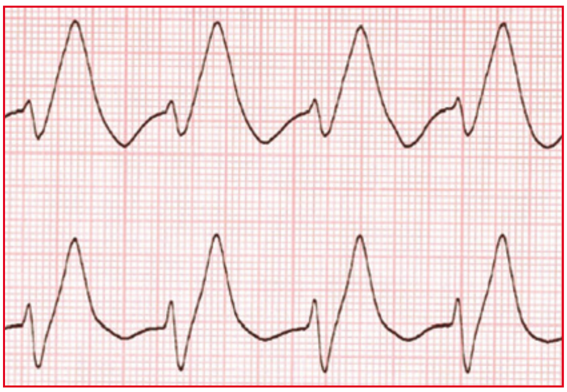
A modification of the T wave corresponds to ischemia, with common significant symetric inversions of this wave. This corresponds to a subepicardial ischemia in case of a negative T, a subendocardial ischemia in case of a positive T.

Finally, a T wave can be (Strips IV-12 to IV-19):

- Of normal appearance,
- Isoelectric or flat,
- Large, in case of pronounced parasympathetic tone,
- Large and peaked in case of hyperkalemia,
- Large, peaked and symmetrical (and most often negative) in case of myocardial ischemia.

Strip IV-12. Normal T wave, positive, asymmetrical, corresponding to roughly 1/3 of the amplitude of QRS complex.

Strip IV-13. Flat T wave, even isoelectric.



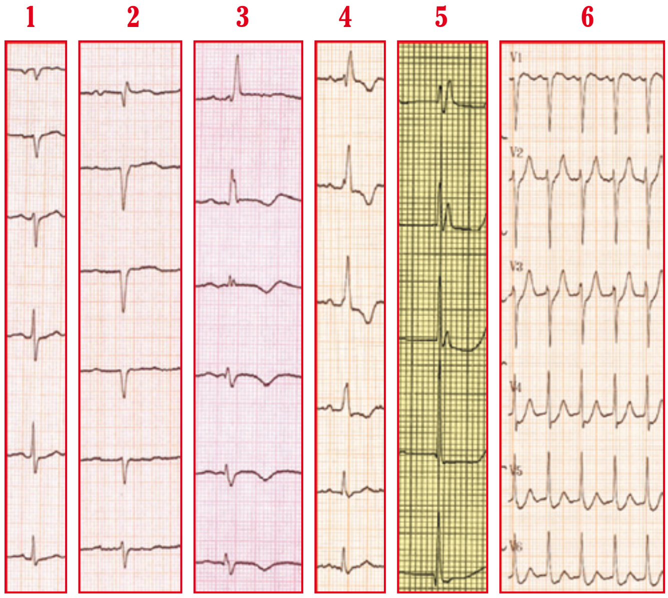
Strip IV-14. Large T wave, very large, peaked, with widened QRS complex, in major hyperkalemia.

Strip IV-15. Large T wave, peaked, symmetrical in myocardial ischemia; note the rigid characteristic of ST segment that is especially visible after the 1st complex.

Strip IV-16. Negative T wave (note the first- degree AVB), normal graph in V1.

Strip IV-17. Aspect of a subepicardial ischemia with a large T wave, peaked, symmetrical, in myocardial ischemia (atrial fibrillation).

Strips IV-18 A and B. Other aspects of negative T waves, asymmetric on ventricular overload, with no myocardial ischemia.



Strips IV-19. Different possible aspects of the T wave are presented in the graphs below.

1. Normal strip.
2. Flattened T wave in precordial leads.
3. Aspect of subepicardial ischemia in anterior leads.
4. Inversed T wave in right bundle branch block; in case of RBBB, an inversed T wave is normal up to V3, it is important to monitor if it surpasses V4 and is pathological starting V5.
5. RBBB with an associated cup-shaped digitalis effect, and a T wave remaining positive.
6. Per-tachycardial angina with an ample T wave that is symmetrical as well as a apical lateral subendocardial lesion current (elevated ST segment in V2-V3, downward deflection from V4 to V6).

Ischemia or lesion

An easy mnemotechnique is to place an "O" symbol in lesiOn, and an "X" symbol in iXemia on opposite sides of the isoelectric line, the subepicardial situated above the line, the subendocardial situated below the line. The negative T wave will be a subepicardial ischemia **(Figure IV-1)**.

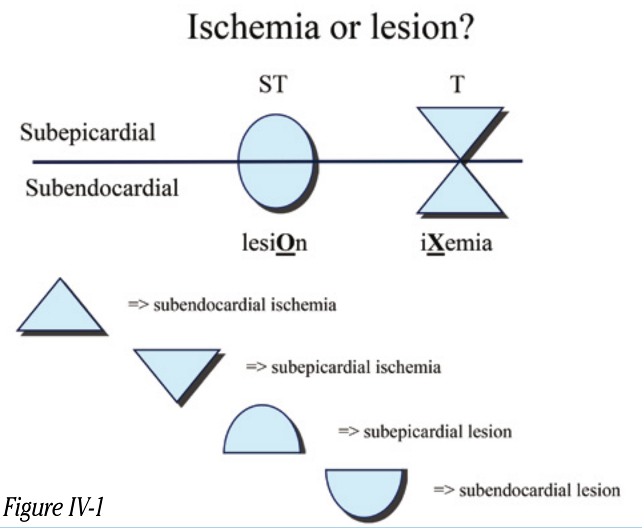


Figure IV-1

QT interval

It is of considerable clinical importance since it is a reflection of the ventricular systole, encompassing both the ventricular depolarization and repolarization.

It is measured between the start of QRS complex and the end of the T wave **(Strip IV-20).** This point indicates that this beginning and this end are not always visible on the same lead, and theoretically, one should compare the various leads in order to look for the earliest and latest phases of returning to the isoelectric line.

Research teams are quite interested in the dispersion of the QT interval: these are the differences in QT from one lead to another, differences that could possibly reflect heterogeneity in the repolarization at the center of the VG. The dynamic quality of the QT interval, which depends, amongst others, on the autonomous nervous system, is also being studied. The dynamic quality is a function of time, physical activity, etc. Dynamicity and dispersion will probably opens new doors to researching the treatments of ventricular arrhythmia, as they are both predictors of ventricular arrhythmia that could potentially be lethal.

Strip IV-20.

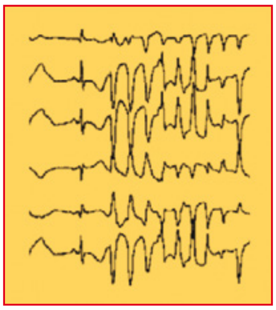
The QT is frequency-dependent, since during an acceleration of the cardiac rhythm, depolarization and repolarization are faster in order to increase their respective efficiencies. As a result, QT interval decreases as frequencies increase.

Its highest normal value is 450 ms in men, 460 ms in women, for a heart rate equal to 60 bpm. It can be calculated either automatically from the ECG registering machine, or by using Bazett's formula QTB = QT/root square of RR (measured in seconds). Its prolongation could be congenital, acquired, or sporadic.

Recently, a study including over 24 000 ECGs demonstrated that a seasonal variety in QT with a significant prolongation is present in the fall in men [6].

The discovery of a lengthened QT could be fortuitous in asymptomatic patients, or reveal evidence of pathologies or the progression of medical treatment. The issue at hand is that the lengthened QT can be silent, or lead to symptoms such as vertigo, lipothymia, syncope or sudden death due to malignant ventricular disturbances. Any discovery of a lengthened QT necessitates a specialized consultancy.

The list of contradicted, ill-advised molecules or those that should be used with precaution is updated on a regular basis, and can be consulted on multiple websites (CHU of Rouen, Doccismef, University of Louvain, University of Toronto, Orpha.net, etc.).



Triggering a wave birth arrhythmia on a lengthened QT interval.

The danger of a lengthened QT resides in the possibility of triggering a malignant disturbance in ventricular rhythm, upon the occurrence of an extrasystole in the terminal phase of the repolarization, known as active phase.

Strip IV-21 [8].

Acute coronary syndromes or ACS

The ECG is a direct reflection of the propagation of the current through myocardial cells; this propagation is independent from cell viability, which itself is dependent from the resulting cell vascularization and oxygenation. As such, ECG becomes an indirect reflection of myocardial vascularization and gives us an approximation of the anatomical state of the coronary arteries. Other than its ability to be a diagnosing factor of coronary ischemia, it also allows for the establishment of the duration and chronology of the former.

The classification of ACSs is regularly being modified, just like the diagnosis of myocardial infarction, recently revolutionized. We will restrain our analysis to the purely electrical aspects of this myocardial ischemia [7] **(Strips IV-22 to IV-30)**.

In any case, one must verify the anomalies noted on the graph that correspond to a specific anatomical site (refer to the next section). The ischemia exclusively disturbs the end of the ventricular repolarization that it was responsible for delaying.

The repolarization remains interpretable in case of right bundle branch block or hemiblock, but the usual criteria retained are not transposable in case of a left bundle branch block or ventricular electrostimulation, whether internal or external.

Ischemia, lesion or cell necrosis will progress into advanced stages in the case that treatment is not delivered [9].

Ischemia, linked to cellular hypoxia, correlates to a symmetrical inversion of the T wave. The precordial leads, being closest to the ventricles, more specifically to the left ventricle, have more pronounced and frequent modifications.

The lesion, or lesion current, corresponds to a shift above or below of the ST segment.

Any shift below correlates, until proven otherwise, to a drop in cardiac output, and this is even more applicable when the shift remains horizontal.

The aspect of the curve evolves progressively with time; the electrical signs usually combine waves of necrosis, lesions and ischemia that appear, develop and regress according to a defined yet variable chronological order:

• A giant T wave, positive, peaked, symmetrical, of subendocardial ischemia (stage 1); there is no Q wave nor a sign of a lesion,

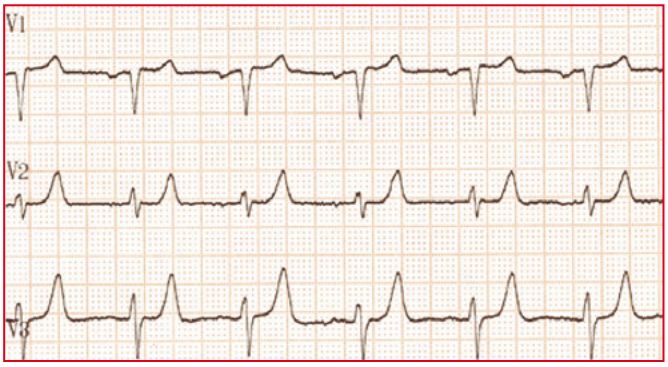
• Then, a shift above of the ST segment progressively appears and amplifies, overshadowing the T wave, making up the Pardee wave (stage 2),

• A beginning Q wave with a decreasing Pardee and an inversion of T wave (early stage 3),

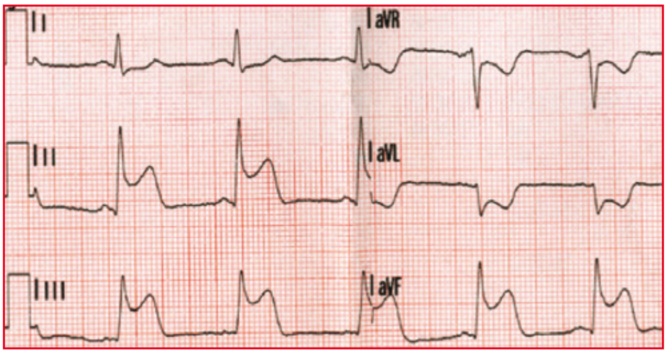
• The Q wave increases in duration and depth and the T wave deepens, ST segment progressively decreases (late stage 3),

• A Q wave, an isoelectric ST segment and a T wave that is positive or remaining negative (stage 4),

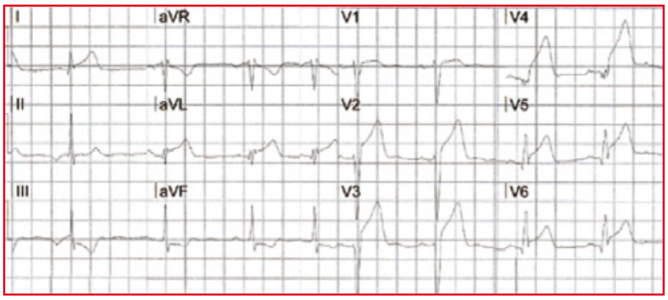
• A Q wave that frequently remains constant over time, or can be spontaneously modified or under the effect of new electrical alterations (bundle branch block, recurrence of infarcts, etc.).



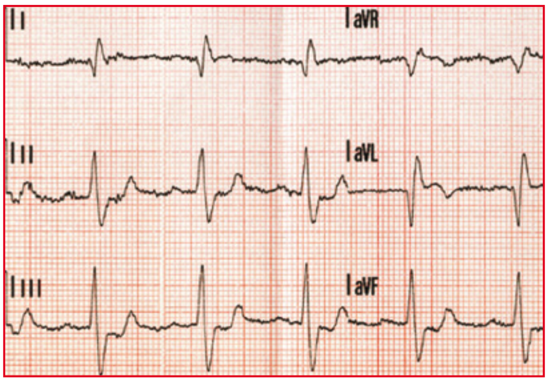
Strip IV-22. Giant, peaked, symmetrical T wave, with a rigid ST segment; there are neither Q waves, nor significant lesion currents; we are at stage 1 of ACS.



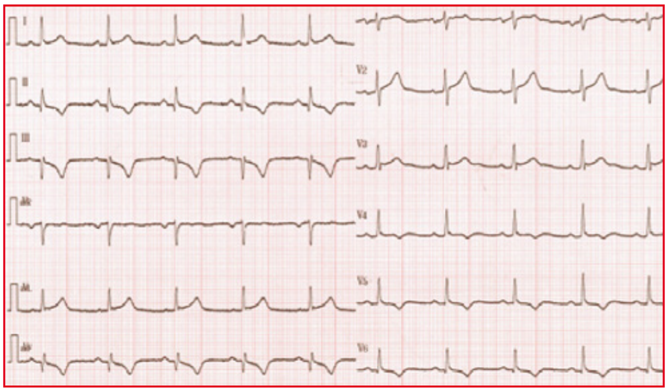
Strip IV-23. Appearance of a Pardee wave, dome-shaped, in the inferior derivations, with a high lateral mirror (DI, aVL); there are no Q waves in the first moments; this is a ACS ST+ in stage 2.



Strip IV-24. ACS ST+ a giant T wave that is symmetrical with an elevation of the ST segment in the extended anterior, with an inferior mirror, the shift below is still not in dome-shape. Pardee wave is actually a result of the increase in the T wave that, while increasing, will overshadow and incorporate the terminal part of the QRS.

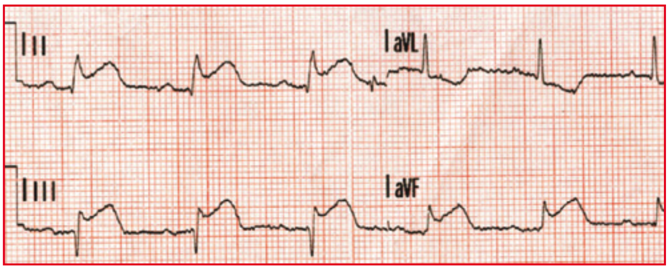


Strip IV-25. ACS ST+ in high lateral (DI, aVL) with an inferior mirror (DII, DII, aVF), in the former stage, with a regressing Q wave shifted above and an inversion of the T wave; late stage 3.

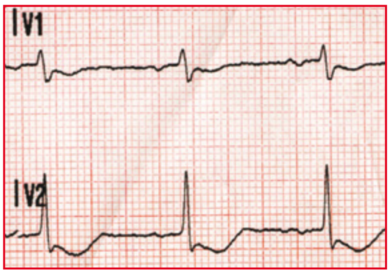


Strip IV-26. Sequellae of inferior ACS ST+, with a Q wave, an ST that reverted to its isoelectric properties, and the persistence of a subepicardial ischemic aspect with a negative T in the inferior lateral; stage 4 of an ECG graph of a myocardial infarct.

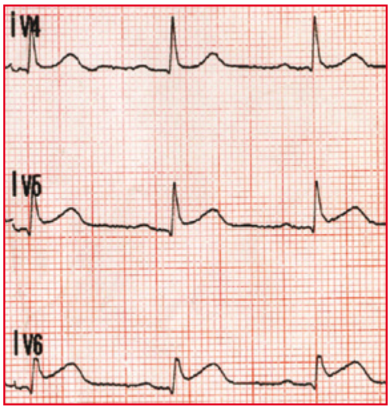
Strip IV-27. Subendocardial lesion current in DIII with a growing R wave; this is a mirror image of a Pardee wave with the beginning of a Q wave.



Strip IV-28. ACS ST+ with lateral mirroring in the inferior phase.



Strip IV-29. Subendocardiac current of lesions in VI and V2 with important R wave starting from VI: this is a mirror image of a posterior infarct with a large R wave in V2 corresponding to a posterior Q wave.

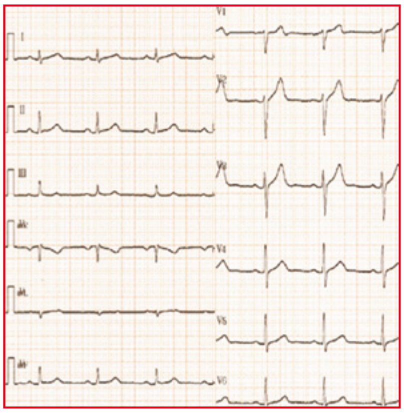


Strip IV-30. ACS ST+ in lateral view, 2 mm are enough in V5.

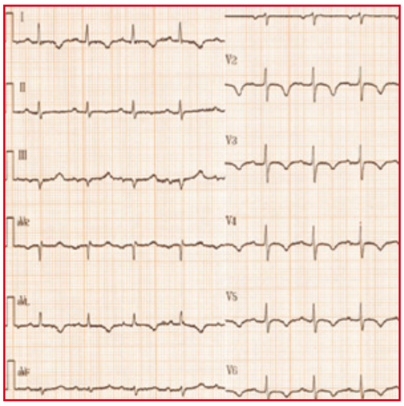
Main points to be remembered

1. All modifications of ST segment of the T wave or in the lengthening of QT translate, until proven otherwise, into a pathology with a possible unfavorable evolution in the short term.
2. Modification of ST = lesion; Modification of T wave = ischemia.
3. All sub shifts of the ST segment translate, until proven otherwise, into a decrease in the coronary debit.
4. A sub shift in the ST is even more representative of ischemia whether it is horizontal or non-elevated.
5. A normal T wave is always asymmetrical; a T wave denotes increasing ischemia with symmetry and sharpness.
6. A normal T wave is positive in the overall derivations, while excluding aVR.
7. A T wave is negative or flat in V1 in 20% of the cases, a T wave is inverted in V1 and V2 in 5-10% of the cases.
8. A T wave that is positive in V1 and negative in V2 is always abnormal.
9. A T wave that is negative in V4, V5 or V6 is always abnormal.
10. A T wave can be physiologically negative exclusively in DIII, when transposed with its QRS complex.
11. The QT is a function of cardiac frequency, gender and seasonality in men.
12. An elongation of the QT interval necessitates a specialized consultation and forbids a certain number of therapeutic molecules, some of which are freely sold.

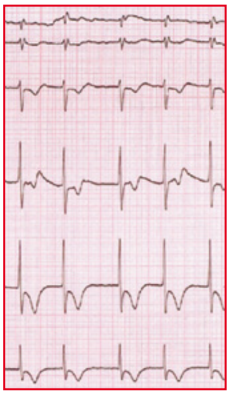
THREE EXERCISES ALONG WITH THEIR INTERPRETATION ACCORDING TO "OUR GUIDE"



Interpretation according to our guide	Normal interpretation
The P wave is present; each one is followed by a QRS. The PQ interval is identical from one complex to the next	Regular sinus rhythm
Frequency of 62 bpm	62 bpm
QRS are thin, DI is positive, DII positive, no bunny ear aspects in VI	No bundle branch blocks, no hemiblocks
ST is isoelectrical, with no elevation or depression	No lesions
T wave is positive in all leads except aVR	No ischemia
QT is normal	No elongated QT interval
Conclusion: regular sinus rhythm at 62 bpm, normal	



Interpretation according to our guide	Normal interpretation
The P wave is present; each one is followed by a QRS. The PQ interval is identical from one complex to the next	Regular sinus rhythm
Frequency of 90 bpm	90 bpm
QRS are thin, DI is positive, DII is positive, no bunny ear aspects in VI	No bundle branch blocks, no hemiblocks
ST segment is isoelectrical, with no elevation or depression	No lesions
T is negative in the entire precordium and in DI-aVL in all leads except aVR	Subepicardial ischemia
QT interval is normal	No elongated QT interval
Conclusion: regular sinus rhythm at 90 bpm with subepicardial ischemia in the anterior layer	



Interpretation according to our guide	Normal interpretation
No visible P wave, irregular frequency, tremors at the baseline	Rhythm in atrial fibrillation
Average frequency of 80 bpm	80 bpm
Thin QRS, aspect of bunny ears in VI	Right bundle branch block
Depressed ST from V3 to V6	Subendocardiac lesion
Negative T wave in the entire precordium	Subepicardial ischemia
QT interval is normal	No elongated QT interval
Conclusion: Auricular fibrillations at 80 bpm on average, with right bundle branch block and disturbances in repolarization of the subendocardial apicolateral lesion current type, and septo-apico-lateral subepicardial ischemia	

V- RHYTHM DISORDERS

ECG interpretation starts by the determination of the heart rate and the baseline rhythm. Normally, electric impulses are generated in the sinoatrial node (SA node), a natural pacemaker, localized at the upper right side of the right atrium (RA). Impulses propagate from cell to cell, reach the atrioventricular node (AV node), pass through the bundle of His, its right & left branches, the ramifications of Purkinge network then again from cell to cell.

The heart rate is determined by the 300/150/100/75/60/50 rule as already made explicit. A heart rate is considered **normal** between 60 and 100 bpm, **bradycardia** if lower than 60 and **tachycardia** if higher than 100.

The rhythm is usually called sinusoidal when generated by the Sinoatrial Node (SA node), it is called ectopic when generating from a different source. In the case of sinus failure, another source of electrical automatism will take over. It is rather a safe stimulation. The lower the source is located on the electrical pathway; the lower is its stimulation frequency **(table V-1)**. Every chamber (source of automatism) with inhibit an underlying slower one, which explains that in the presence of normal automaticity of the NA, lower chambers are inhibited **(figure V-1)**. Finally, by definition, an electrical automatism will discharge into a sensitive regular rhythm, dependent from the automatic nervous system among other factors.

The rapid determination of the heart rate also gives an idea on the diagnostic of heart rhythm disorders in regards to certain frequencies, the latter being prone to being slowed down by a chronotropic negative treatment or antiarrhythmic drugs already in place.

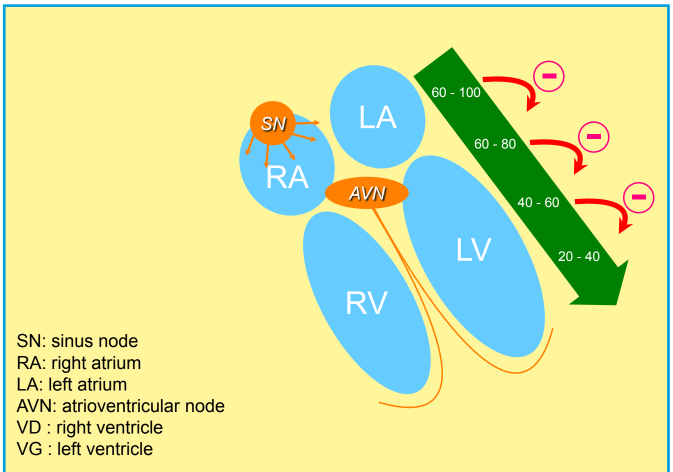


Figure V-1. Inhibition of the underlying cardiac automaticity sites.

Rhythm	Birth location	Frequency / rate bpm
Sinus	Sinus Node	60-100
Non sinus	Atria	60-80
	Atrioventricular Junction	40-60
	Ventricles	20-40

Table V-1.

Rhythm		Conductrive P wave	Fréquence cycles / mn	Possible diagnoses
regular	slow	present	≤ 60	sinus bradychardia
		absent	20- 40	junctional escape beat
				ventricular escape beat
				AVB III
	normal	present	60-100	normal rhythm
				atrial escape beat
		absent	60-100	junctional escape rhythm
	rapid	present	≥ 100	sinus tachycardia
		absent	150	atrial flutter with 2.1 AV conduction
			180-200	junctional tachycardia
			180-250	ventricular tachycardia
			250-350	Polymorphic ventricular tachycardia
irregular	slow	present	≤ 60	sinus bradycardia interspersed with extra-systoles
		absent	≤ 60	atrial fibrillation with bradyarrhythmia
	normal	present	60-100	sinus arrhythmia
		absent	60-100	atrial tachycardia with variable conduction
	rapid	present	≥ 100	sinus tachycardia with extrasystole
		absent	≥ 100	atrial tachycardia with variable conduction
				atrial fibrillation
				atrial flutter
				ventricular fibrillation

Table V-2.

Heart Rhythm Disorders

Heart arrythymias are by definition abnormal rhythms. Some may be physiological, such as respiratory sinus arrhythmia, others may be pathological.

For better understanding these arrhythmias, one must understand the physiology of the nervous impulses among other cardiac electrical components, as well as some major standards:

1. At every level (atrium, atrioventricular junction and ventricle), the heart beat could be either too slow, normal or too fast,

2. Each heart cavity can act as a automation center, always regular especially when its upper chamber fails,

3. Atrioventriculor node is a physiological filter of electrical conduction and automaticity; in the case of upstream very slow rhythm, it will only allow the transition of impulses necessary to preserve a consistent hemodynamic activity in the ventricles;

carotid compression (not to be performed unless in the absence of carotid bruit, always under recording) will stimulate the blocking power of AVN, and will help to uncover the underlying activity when it exists,

4. When determining 3 characteristics; the presence or not of conductive P wave, the regularity or not of complexes and the encrypted frequency, diagnoses can be made **(table V-2)**.

Various rhythm disorders [10]

1 – Regular rhythms

A. Escape rhythm

In the case of failure of conductivity of the sinoatrial node (SA node), an escape beat is self-generated to ensure electrical discharge. This escape, by definition of a regular/steady rate, can be atrial, junctional or ventricular.

Atrial escape beat or rhythm

When sinus node fails to ensure its function, an atrial site takes over. On the strip, this can be shown by PQRS complexes at a rate of 60 to 70 bpm, but at a lower rate from the regular one for a given patient, and the morphology of P wave is slightly different from the "natural" P wave in the same patient. Recorded on the same strip or on a holter, the distinction is easy, but in the case of no prior strip, an atrial escape can be mistaken for a sinus strip.

Junctional escape beat or rhythm

Automatism is located at the ANV level, and is inhibited by a higher heartbeat of the sinus node or atrium. The conduction starts at the supraventricular site/stage and ECG morphology will show a strip with regular QRS complexes for the patient, without a P wave ahead.
A retrograde P wave can take place because the electrical fibers remain functional; it is only the starting battery that fails **(Strip V-1)**.

Ventricular escape beat or rhythm

Active stimulation sites are situated at the very lower side of the ventricles, and take birth/initiate from within the ventricles; QRS complexes are prolonged. There are two clinical cases where these can be observed:

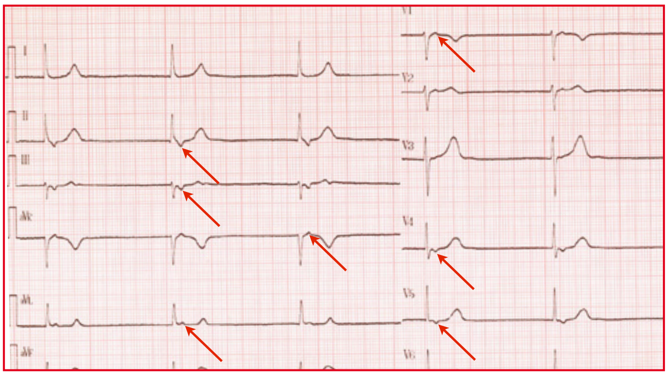
Either a ventricular escape beat with a complete block :in the case of failure of all other upper sites (sinus, atrial, atrioventricular node) , the strip will shows prolonged QRS complexes (the lower the site, the wider the complex) intercepted with an isoelectric line;

Either a ventricular escape beat with a third-degree atrioventricular block: the strip will show wide QRS complexes at a natural frequency, and a atrial activity also at a natural frequency, both independent from one another **(Strip V-2)**.

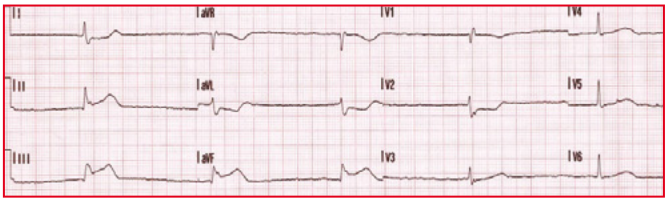
B. Rapid rhythms

Atrial Tachycardia (AT)

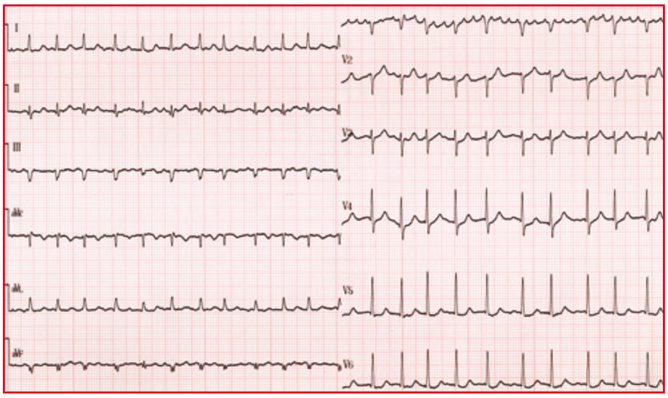
A discharge site, at a regular frequency, faster than that of the sinoatrial node will stimulate the ventricles. The transmission will



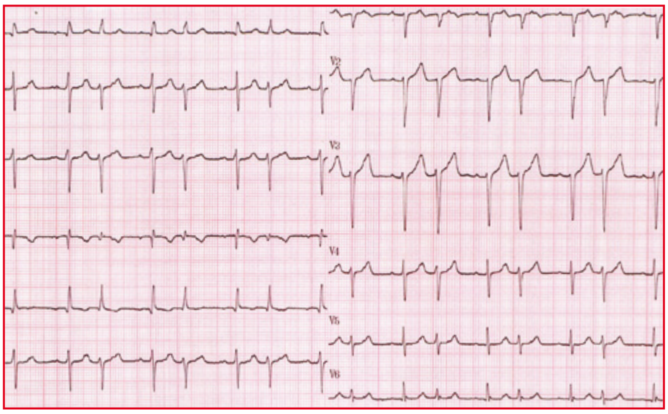
Strip V-1. Junctional escape rhythm at 50 bpm, with a retrograde P wave (arrow) clearly visible at the beginning of T wave in V2 and V3, thin QRS complexes.



Strip V-2. Ventricular escape rhythm at 34 bpm, over ACS ST+ in inferior leads; QRS complexes remain thin with no P wave.



Strip V-3. Rapid AT (at an atrial rate of 350 bpm), clearly visible in V1, with variable conduction and pseudo- P waves that remains well organized.



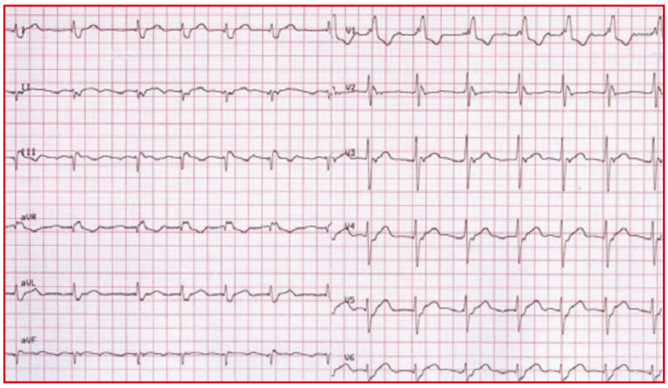
Strip V-4. A much slower AT (at an atrial rate of 148 bpm) with a "normal" conduction of two cycles, followed by a P wave block, then a resumption of the diagram; the block is in fact due to its occurrence in the refractory period of the previous complex, so it may not stimulate; Interestingly, the isolated analysis of V3 could say sinus complex followed by a bigeminy supraventricular extra-systole atrial, which is rectified on V1.

be filtered through the atrioventricluar node. The latter will allow one over 2,3,4 or 5 impulses to pass through, in order to preserve a compatibility between ventricular rhythm and hemodynamic efficiency.

The ratio of P wave to the QRS complexe will result in the heart rate. The blockage may be regular or not **(Strips V-3 or V-4)**.

Atrial Flutter It is a macro-reentry process, giving the aspect of "sawtooth pattern" at an atrial rate of 300 bpm. The conduction towards the ventricles is usually regular, after being filtered by the AVN, with one impulse over 2, 3, or 4 transmitted: resulting ventricular frequency at 150, 100 or 75 bpm **(Strips V-5 and V-6)**.

Strip V.5. Atrial flutter with a flutter rate of 300 bpm, a slow conduction at the AVN in 2:1, with a residual frequency of 150 bpm.



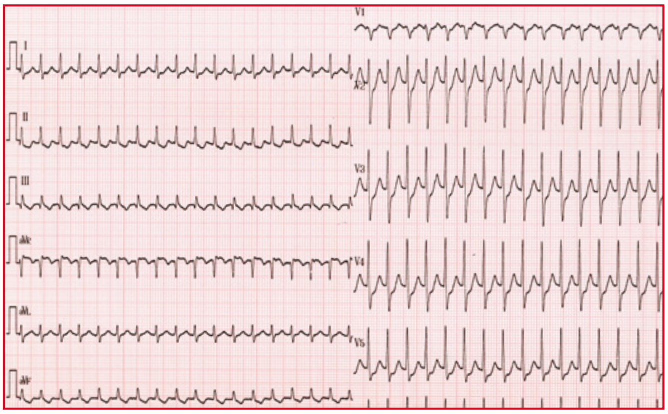
Strip V-6. Atrial flutter visible in DII, slowed down by amiodarone treatment, at a frequency of 190 bpm, with a variable conduction of type 2:1 (ventricular frequency of 95 bpm) or type 3:1 (ventricular frequency of 65 bpm).

Junctional Tachycardia

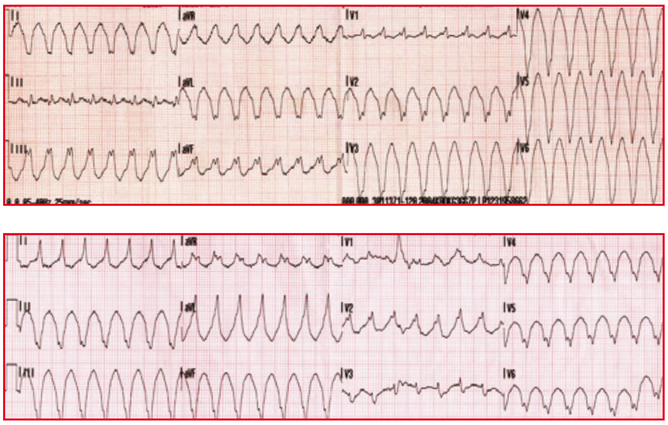
Stimulations start at the atrioventricular node level, the atrioventricular node reentry, an auto-induced short-circuits. The heart beat is between 150 and 250 bpm. Complexes are usually thin and retrograde P waves can sometimes take place **(Strip V-7)**.

Ventricular Tachycardia (VT)

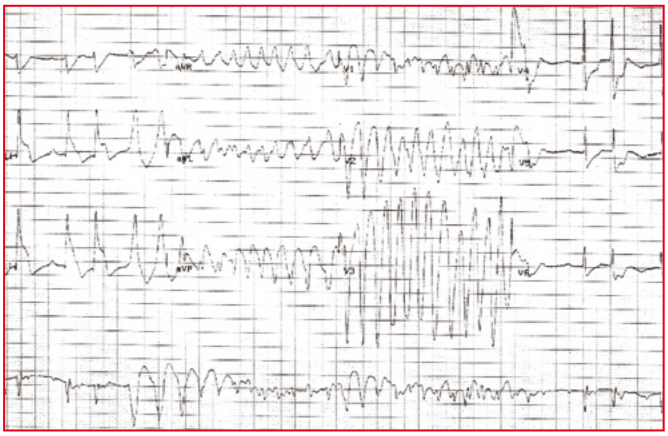
Characterized by a rapid heartbeat, it is either caused by an irritable spot that will discharge rapidly (from 150 to 250 bpm), or by a reentry phenomenon, that will induce a variable tolerance of the heart based on its previous condition. Complexes are wide, with a thin complex sometimes: it is the case of fusion complex between sinus activity that persists and extends towards



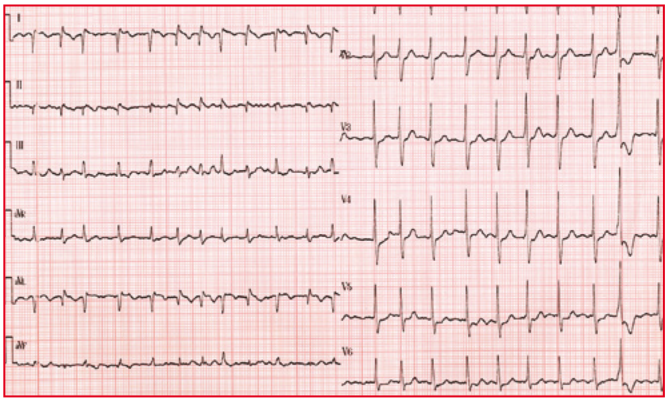
Strip V-7. Junctional tachycardia with thin complexes, at 214 bpm, Bouveret disease.



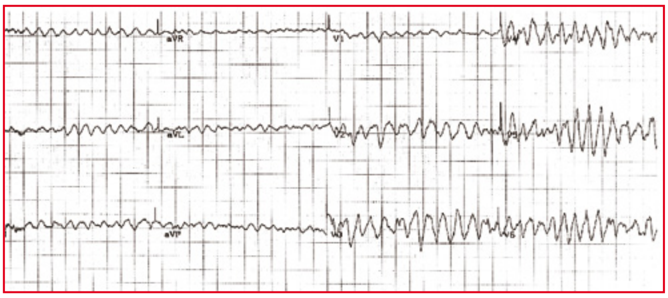
Strip V-8 and V-9. Two types of VT, at 200 and 190 bpm, regular; a positive aVR is almost certainly a sign of ventricular origin of the disturbance in adults, a strip showing a post reduction will confirm this hypothesis.



Strip V-10. Interpretation on the first and last complexes: sinus strip, with a STEMI in lower and side mirror – at least on the last line, two sinus QRS followed by a supraventricular extrasystole, then a PVC that launches a burst of "torsade de pointes" for 6 seconds with a spontaneous reduction and return to the sinus rhythm (one complex), then one PSVC, then one sinus rhythm.



Strip V-11. AF with rapid ventricular response of 140 bpm, with thin complexes and a ventricular extrasystole at the end of the strip; the difference with AT in clear in V1, where there is no longer an organized aspect of P waves. An inversion of extremity electrode is to be noted.



Strip V-12. Totally anarchic strip of ventricular fibrillation.

the ventricles. The final frequency can be low based mainly on treatment being followed by the patient. The distinction between a ventricular tachycardia and a branch block is not always easy but an often overlooked criterion is the negativity aVR; if aVR is positive, a ventricular origin is almost certain. VT can be sustained (≥ 30 complexes), a burst (to keep in mind that 3 consecutive PVCs correspond to a burst of VT). Risks, other than the hemodynamic failure, are mainly the potential evolution of towards an atrial fibrillation **(Strips V-8 and V-9)**.

Polymorphic ventricular tachycardia (PMVT) or torsade de pointe

It has a very particular strip, with a very rapid heartbeat between 150 and 250 bpm. Two distant sites are competing, giving in sum the aspect of “spikes” around the isoelectric line. Torsades are secondary to a prolonged QT interval acquired and/or congenital.

PMVT may be induced by a variety of clinical scenarios, including acute ischemia, reperfusion, and organic heart disease. Less frequently, PMVT with a normal QT interval can occur in patients without apparent structural heart disease resulting from several underlying electrophysiological abnormalities [11].

Some torsades occur on a thin QT (idiopatic or digital). They are usually associated with a high incidence of sudden cardiac arrest. Same as the VT, the major risk of Polymorphic ventricular tachycardia is the evolution towards a ventricular fibrillation **(Strip V-10)**.

2 – Irregular rhythms

They are defined by intervals between every irregular cycle.
Multifocal atrial tachycardia Many discharge sites are located in the atria; every site will stimulate at its own regular frequency; having a number of discharge sites, at different regular frequencies, the summation will result in an irregular rhythm exceeding 100 bpm. At a given lead, many morphological differences of P waves will take place.

Sinus Arrhythmia

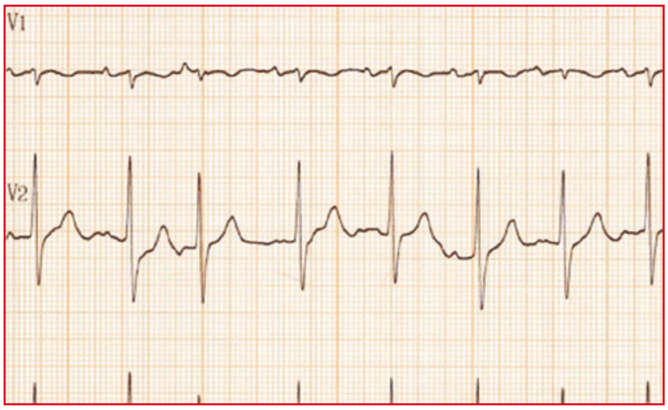
There is an automatic site near the sinus node that discharge. P wave will slightly differ from other P waves.

Atrial Fibrillation (AF)

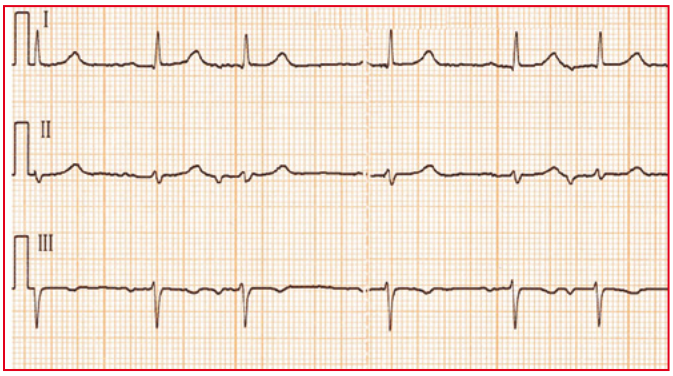
It is the summation of multiple discharge sites that will lead to anarchy in atrial depolarization. Hemodynamic activity is amputated of 10 to 30%, based on the presence or not of cardiomyopathy history. A bradyarrhythima, a total arrhythmia or a tachyarrhythmia can take place **(Strip V-11)**.

Ventricular Fibrillation

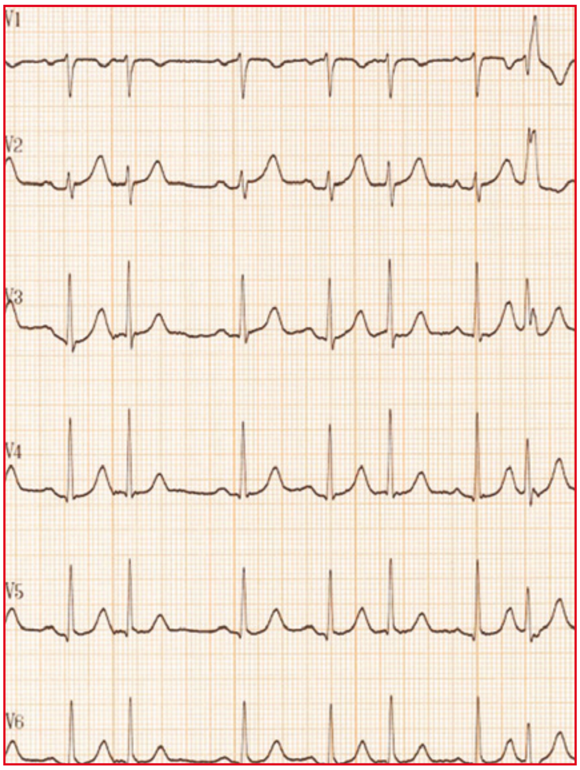
It is the case of an anarchic rhythm, where many sites discharge, with a frequency higher than 400 bpm. There is a total hemodynamic inefficiency; the patient is suffering from a cardiorespiratory arrest. It is an absolute emergency requiring immediate defibrillation **(Strip V-12)**.



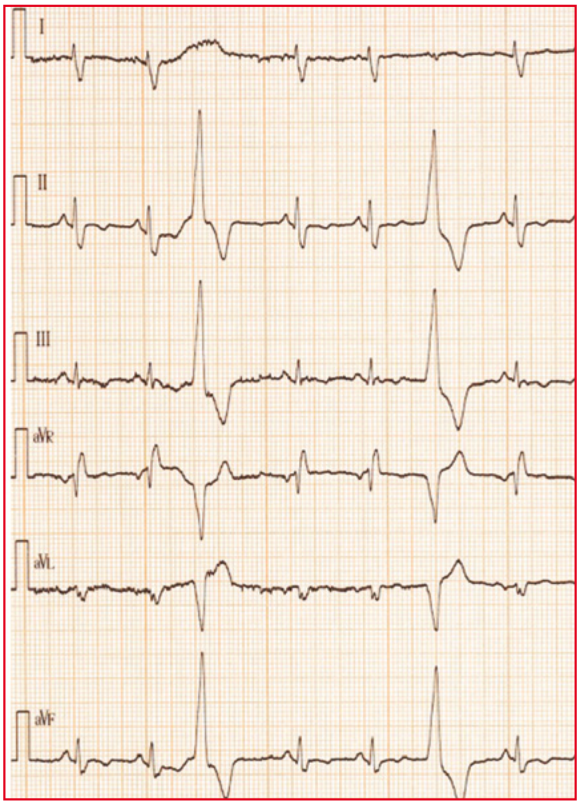
Strip V-13. The 3rd complex is an AES: P wave slightly different, QRS identical to the others, short compensating pause.



Strip V-14. The 1st and the 4th complexes are AES, originating near coronary sinus (negative P wave in DIII), followed by normal QRSs.



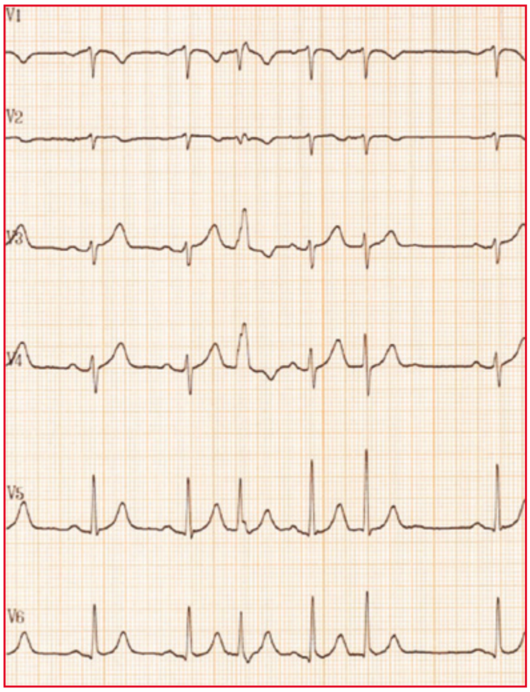
Strip V-15. Sinus rhythm, PSVC corresponding to complexes 2 and 5 (no P wave, QRSs are identical) and one PSVC (7th complex) of right delayed type (right bundle branch block aspect).



Strip V-16. Sinus rhythm with one PVC at the 3rd and 6th complex; it is a trigeminal PVC (one PVCevery three complexes).

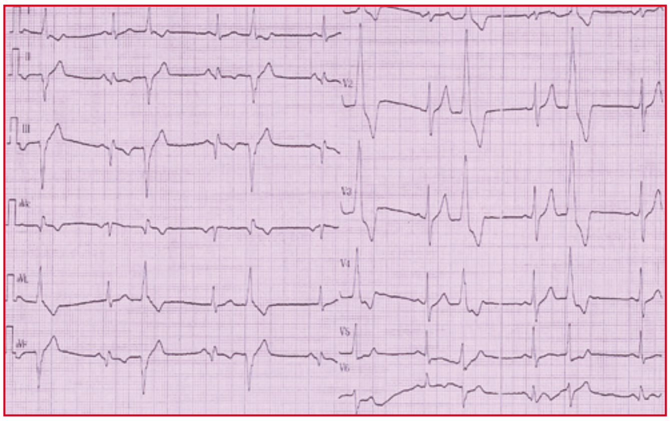
3 - Premature heart contractions (PHC) or Extrasystoles (ES)

They can appear at any level, atrial, junctional or ventricular. It is a site where, under certain conditions, will become irritable and will discharge. It is the case of a premature heartbeat, leading to a general compensating rest period. They cannot be felt them but the heartbeat following an extra systole can be felt (following this rest period, diastole will be prolonged, the intra cardiac blood flow is increased, and since the next heartbeat is sinusal with a normal physiology and hemodynamics, increase blood outpout will lead to this squat sensation).



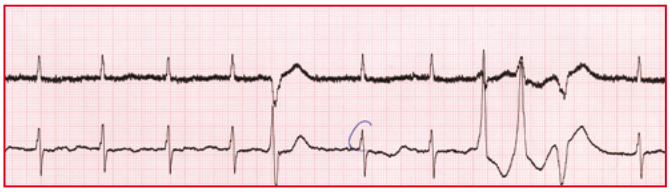
Strip V-17. Sinus rhythm, one PVC of right delayed type (3rd complex), one PSVC (5th QRS).

Strip V-18. Monomorphic PVC with a compensating pause.



Strip V-19. Strip with monomorphic bigeminal PVC (one PVC every two complexes).

Strip V-20. Non sinus strip, with relatively wide complexes; the 1st complex, very thin, reveals an major ST depression (inferior subendocardial current), with a negative DI (left posterior hemiblock); the 2nd and the 3rd are polymorphic PVC; in aVR, aVL and aVF, there are two complexes with one very early PVC, then in V1-V3, there is a QRS in wave crochet pattern (right bundle branch block), then a PVC bigamy, polymorphic, coupled, early with a right bundle branch bloc, followed by a major subendocardial current in the apical lateral walls.



Strip V-21. Strip showing a sinus rhythm with one PVC (5th QRS) then three polymorphic PVCs after two sinus QRSs.

Aspect of extrasystoles

• Atrial level (AES or PAC): P wave differ from other ones, QRS complex will take the same electrical pathway and is similar to others; if the induced current takes the same pathway, ventricluar conduction can vary and QRS aspect can differ;

• Junctional or supraventricular level (SVES or PSVC): there is no P wave, QRS complex is identical or very similar to sinus QRS;

• Ventricular level (VES or PVC): there is no P wave, QRS complex are enlarged and varies depending on the starting point of sinus QRS.

Characteristics of an extrasystole: criteria of gravity for PVC **(table V-3).**

• Its birth level
• The presence or not of compensatory rest
• For ventricular extrasystoles (PVCs)
  - The number of PVCs: every patient suffering from extra systoles on a daily basis, generally "not felt"; a benign criterion is PVC disappearance during stress test.
  - The number of ectopic sites : PVC aspect will determine whether PVC originates from the right ventricle (left delay aspect at the left branch block) or the left ventricle (right delay aspect at the right branch block), whether it is a unifocal (identical aspect of all VES on the same lead or monographic aspect), or whether there are multifocal (polymorphic aspect)
  - Earliness or not with regards to T wave: the nearer the PVC is to T wave, the more susceptible it is to fall under the vulnerability period of fibers repolarization, capable of launching a PT, a VT or a VF
  - Periodicity: unique PVC, without periodicity, occurring after a regular sequence, intercepted by a number of regular sinus complexes…
  - Coupling intervals: isolated PVC (uni, ventricular bigeminy or ventricular trigeminy, or bursts; a succession of 3 PVCs is considered as VT burst.

PVC characteristics		
		criteria of gravity
Numbre of PVC	counted	≥ 6 per min
Number of ectopic sites	morphologic aspects = 1 foyer	morphologic aspects = multiple sites
Compared to previous T wave	late PVC	early PVC (R on T phenomenon)
Periodicity	isolated, bigeminy, trigeminy, quadrigeminy ...	
Coupling	successive number of PVC	Always; if ≥ 3 PVC = VT

Table V-3

Main points to be remembered

1. Each level (auricular, junction, ventricle) is endowed with automaticity and takes in charge in the case of failure of the underneath site.
2. Automatism are regular rhythms.
3. Every level can be the source of the same rhythms problems (too slow, normal, too fast).
4. The heart rate will lead the diagnosis.
5. Frequency, regularity and the presence or not of P wave will allow for precise diagnosis.
6. Carotid compression (in the absence of carotid murmur) will allow to discern an underlying problem.
7. A rhythm of 150 bpm is an atrial flutter or atrial tachycardia type 2/1 until proven otherwise.
8. A rhythm of 180 bpm is indicative of Bouveret disease until proven otherwise.
9. A rhythm higher than 160 bpm with prolonged complexes is a ventricular tachycardia until proven otherwise.
10. A PVC is as dangerous as multiple (≥ 6/mn), polymorphic, coupled and premature.

VI- CONDUCTION DISORDERS

Disturbances in cardiac conduction usually deal with the nodal tissue. This can be defined as the wholecardiac electrical system, comprising an impulse generator to which conductive structures allowing the movement of the electrical current towards the myocardial cells are fixed. It is easily comparable to a "natural" battery linked to a few electrical wires and this comparison often allows us to understand the pathophysiological disturbances in the cardiac conduction system.

It is important to distinguish between rhythm and conduction disorders, although conduction greatly interferes with cardiac rhythm. The latter is equally resulting from conduction and from spontaneous automatism of the heartcomponents. Rhythm disorders were analyzed in the Vth section. The P wave remains a crucial element: all P waves must be followed by a QRS complex, and each QRS must be followed by a P wave. In reverse order, it is common to see disturbances in the conduction.

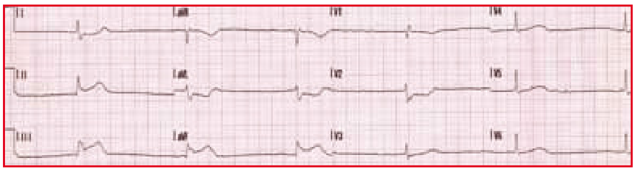
What causes conduction disorders?

- Every cell of this tissue has conductive properties, whether it is a tissue, a node, a bundle, a fiber, or an isolated cell.
- Every deflection of QRS is the property of the underlying tissue that can be projected on its surface.
- Every deflection of QRS is therefore a reflection of the conductive properties of underlying cells, which can be measured by the duration of that deflection. As a result, all lengthening of the duration is a sign of pathology in the conduction whether structural or functional.
- Pathology in conduction will either slow down the nervous impulse in a given structure or block its passage.
- Every layer of the nodal tissue can be affected by conduction disorders which can result in pathology/disturbance of its own conduction.
- When the disturbance concerns the layer above His bundle, its impact is benign; when it originates under His bundle layer, its evolution is dangerous and requires, in addition to a specialized opinion, additional test and often a pacemaker.
- Knowing the source of vascularization of certain structures allows one to explain that conduction disorders are more frequent during an infarction localized in inferior structures (upon occlusion of the right coronary artery), or localized in lateral structures (upon occlusion of a circumflex artery), and that these almost never occur in anterior infarcts unless in poorer prognostic (upon occlusion of the anterior interventricular artery).

Components of nodal tissue are described in **table VI-1**.

Various conduction disorders [12]

In case of decelerating impulse, measures of duration will be lengthened. In case of blockage, the nodal tissue of the underlying layer can take over if necessary in order to preserve sufficient hemodynamic activity **(Strip VI-1)**.



Strip VI-1. Ventricular escape on ACS ST+ inferior with high lateral mirror

Table VI-2 indicates various possible pathologies depending on the tissue layer concerned.

	Blockage	Deceleration
PN	Atrial escape	Ist degree ABV, 2nd degree ABV, 3rd degree ABV
AVN	Junctional escape	
His	Ventricular escape	
Branches	Bundle branch block ; right, left, and hemiblocks	

Table VI-2

Nodal tissue	Function	Observations
Pacemaker node (PN) or sinoatrial node or node of Keith and Flack	Primary physiological center for production of excitatory signals or natural pacemaker	Vascularization: artery of the pacemaker node coming from the right coronary artery in 50% of cases, from the circumflex artery in 20% of cases, and from both in 30% of cases
Auriculoventricular node (AVN) or node of Aschoff-Tawara	Impulse is transmitted from the atrium towards the bundle of His	Vascularization : artery of the auriculoventricular node coming from the right coronary artery in 90% of cases, and from the circumflex artery in 10% of cases
Bundle of His	Excitatory stimuli transmitted from the auriculoventricular node towards Tawara'sbranches	
Branches of Tawara or the branches of the bundle of His	Impulse conduction all the way to the myocardium	Are subdivided into: - right branch - left branch, giving in turn a posterior-inferior branch and anterior-superior branch
Network of Purkinje fibers	Impulse conduction from cell to cell	

Table VI-1: Generation and excitation conduction systems.

1. Sinoatrial blocks (SAB)

They are often difficult to diagnose using automatic recordings. A long recording, of 20 to 30 sec or more is necessary. The main property of the ECG is a pause without a P wave. This pause can be irregular (SAB type Wenckebach) or regular (SAB type Mobitz), with the duration of the pause being equivalent to exactly twice the interval of PP normal. A similar classification of SAB to atrioventricular block (AVB) is possible.

2. Atrioventrical blocks (AVB)

A. First degree AVB

There is a delay in the atrioventricular conduction. The PR space is lengthened above 0.2 sec thus larger than a large unit. In the majority of the cases, this disorder is mild as it remains above His level **(Strip VI-2)**.

Strip VI-2. Sinus rhythm, 1st degree ABV with PR equal to 0.3 seconds.

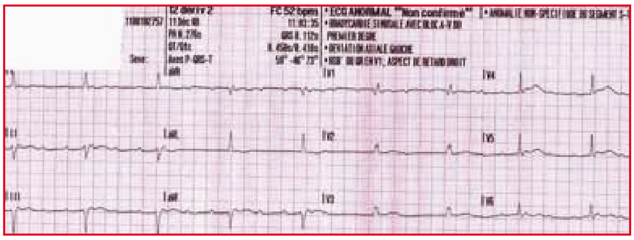
B. Second degree AVB

There is an intermittent interruption in conduction at the level of the AVN. Some P waves are not followed by QRS. We notice:

- Mobitz I or 2nd degree AVB or Wenckebach's block:
  - Progressive lengthening of the PR interval until a blocked P wave is reached, then an identical sequence is repeated,
  - This disorder takes place above His bundle.
- Mobitz II:
  - Occurrence of an unexpected blocked P wave,
  - The affected area can be either above or below bundle of His.
- AVB II of type 2/1 (or 3/1):
  - Only an atrial contraction on 2 (or 3) will be transmitted to the ventricles. It is the ratio between the P waves and the QRS that determine the type of AVB,
  - The affected area is most frequently below bundle of His **(Strip VI-3)**.

C. Third degree AVB

The connection between the atria and ventricles has totally disappeared. No P wave can be linked to a QRS complex in a logical manner. We distinguish a properatrial rhythm and a ventricular rhythm, recording a true atrioventricular dissociation. The shape of the QRS complex can vary depending on the localization and the origin of the new ventricular pacemaker; the slower is the latter, the more the QRS will be enlarged and slow, but will remain regular.



Strip VI-3. The strip is too short to be able to affirm the diagnosis with certainty. P waves are visible, and more numerous when compared to the QRS, with at least two P waves between each QRS complex. We can thus assume that this is an AVB of type 2/1 with a lengthened PR. However, upon more refined measurements, we see that the RR intervals are not fixed, which is by definition contradictory. Therefore, there must be an additional invisible P wave that is included in the QRS, and in this case, the RR spaces are constant. It is therefore either a 2nd degree AVB of type 3/1 with a lengthened PR when the P wave is conducted, or a 3rd degree AVB, of which the auricular frequency is close to three times the ventricular frequency. It is interesting to note the automatic interpretation of the recorder, because if the anterior hemiblock is well recognized, as for the pacemaker rhythm, we shall revisit…

D. How to recognize and understand AVBs

One only needs to imagine two batteries, A and V, linked by an electrical wire. Progressively, we will think about an increasing deceleration of conduction in the electrical wire.

- The electrical wire is "pulled", the conduction decelerates but remains constant, PR interval is lengthened; this is the 1st degree AVB.
- The electrical wire "ears out as it warms up" while conducting electricity, the PR is progressively lengthened until it blocks after a certain sequence, the system reboots, this is the 2nd degree AVB of type Mobitz I or Wenckebach's block;
- The electrical wire is saturated at times and blocks, P wave is no longer followed by a QRS complex, this is the 2nd degree AVB of type Mobitz II;
- One time out of two or more, as the saturation progresses, so do the blockages. This is the 2nd degree AVB of type 2/1 or more
- Finally, the wire between the two batteries is "fried", and each battery functions separately, this is a 3rd degree AVB

The presence of P wave, its regularity, and the ratio between the number of P waves and QRS waves allows the progression into a simplified approach of the electrocardiogram. Every P wave should be followed by a QRS, P=QRS is the normal situation. The rhythm is either sinusoidal, or containing a lengthened PR interval, thus a 1st degree AVB.

If there are more QRS than visible P waves, it can only mean that there is an escape beat, the level of which is to be determined. If there are too many P waves compared to QRS, we are in the 2nd or 3rd degree AVBs.

The absence of a P wave will make us look for regularity in rhythm. In the case of a regular rhythm without a P wave, this will be a 3rd degree ABV (or ventricular escape rhythm). In case of an irregular rhythm, there will be a disturbance in the rhythm of various types, namely, atrial fibrillation, flutter, or atrial tachycardia. The **table VI-3** summarizes these elements.

P wave	Ratio P / QRS	Possible diagnosis
Present	P = QRS	Normal pacemaker rhythm, 1st degree AVB
	P < QRS	Escape beat
	P > QRS	If bradycardia => 2nd degree AVB, Mobitz I, Mobitz II, of type 2:1, 3rd degree AVB If tachycardia => AT
Absent	Regular pulse	3rd degree AVB

Table VI-3

3. Bundle Branch Blocks

These were analyzed in the 3rd section. To recapitulate, we note:

• RBBB: typical aspect from V1 in M, or in bunny ears, with a possible diagnosis of infarct, ventricular hypertrophy, etc.

• LBBB: wide QRS, no R wave in V1, M aspect in V5-V6,

• ALHB: DII negative,

• PLHB: DI negative,

• Associations:

- RBBB+ALHB: M in V1 and DII negative, 5% will evolve to AVB III
- RBBB+PLHB: M in V1 and DI negative: 70% will evolve to AVB III

Naturally, AVBs and Bundle branch blocks may be associated.

Treatments of conduction disorders

The etiological treatments remain essential, but the natural evolution of the nodal tissue and its aging imply the use of cardiac pacemakers. After having treated the metabolic causes as well as the iatrogenic causes of the disturbances in conduction, implementing artificial pacemakers remains crucial.

Properties of a pacemaker

The international nomenclature [13] for these pacemakers consists of three letters and an additional letter that defines them. The first three letters characterize the pacemaker, and the fourth indicates its mode of programming. The 1st letter corresponds to the chamber being paced, the 2nd letter corresponds to the chamber being sensed, and the 3rd letter indicates the response to sensing: A = atrium, V = ventricle, D = dual (A+V) or I = Inhibited. The 4th letter is used to qualify an additional characteristic: Rate modulation. R is used (modulation rate) for a programming that respects the acceleration of heart rate that is particularly necessarily during physical activity.

These pacemakers can be either **(Strips VI-4 to VI-7)**:

• Single chamber: a single chamber can be sensed (atrium or ventricle), the same chamber is paced in case of a noted defect. For example: AAI that senses and paces the atrium when it is not inhibited by a sufficient spontaneous base frequency. Another example is with a VVI that listens to and stimulates the ventricle except in cases of inhibition,

• Dual chamber: both layers are sensed (atrium and ventricle), and the two layers can be paced in case of spontaneous inactivity. For example: for a DDD-R that senses, paces one or both cavities and adapts to physical activity,

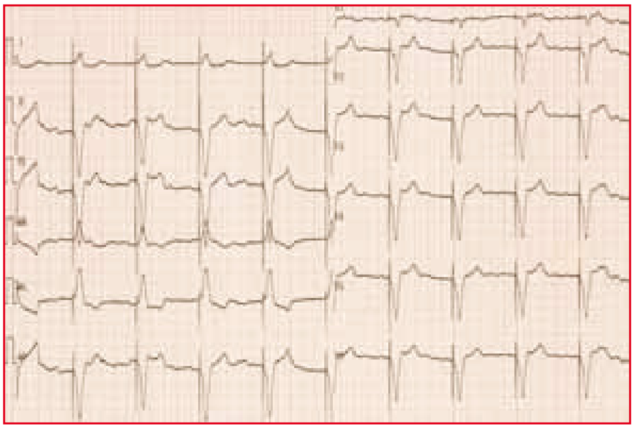
• Triple chamber: it has the same characteristics as a dual chamber, with an additional third probethat allows, in the case associated with acute heart failure, for resynchronization of ventricles. A third spike can be seen in the middle of the paced QRS.

The electrical translation of pacemakers is the presence of stimulation spikes. When a spike is present in the atrium, it either means that we are dealing with an AAI pacemaker (the atrium is sensed, the atrium is paced when needed), or that we are dealing with a DDD pacemaker (both cavities are sensed, the atrium is paced when it is unresponsive). We do not pace the ventricle because the provoked artificial P wave follows the normal path and activates a normal QRS. Implanting an AAI pacemaker is rare because this implies perfect integrity of the atrioventricular conduction and its stability with time.

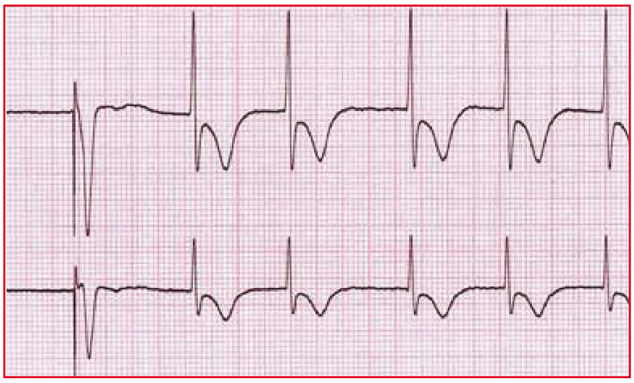
When a spike is present in the ventricle, then we are dealing with a VVI pacemaker (only the ventricle is sensed, only the ventricle pace if there are no spontaneous QRS complexes, and we do not take note of the eventual activity of the atria) or we are dealing with a DDD pacemaker (both cavities are sensed, a natural P wave exists but the atrioventricular conduction is blocked, furthermore, we only pace the ventricle if necessary).

When two spikes are present, in the atrium and in the ventricle, it can only mean that we are dealing with a DDD pacemaker or a triple chamber.

Finally, the repolarization is no longer capable of being interpreted when a pacemaker is worn, except in the rare cases where there is only a strict auricular stimulation with respect to the downstream conduction path (for example AAI).



Strip VI-4. Electro stimulated rhythm in VVI mode with an atrial fibrillation background, the second complex being nevertheless preceded by a wave raising a P wave.



Strip VI-5. Electro stimulated rhythm with intermittence in VVI mode, with an atrial fibrillation background. The rhythm remains totally irregular despite the absence of required stimulation. Note the disturbances in repolarization that exists for the non-stimulated complexes. They are interpretable in these conditions; this is an electrical memory effect of the myocardial cell.

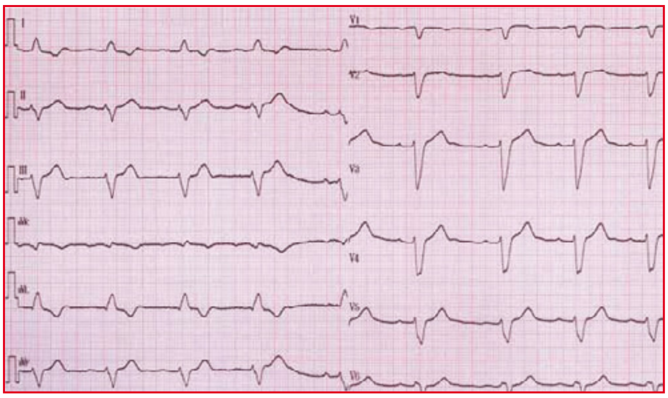
Strip VI-6. Permanently electro stimulated rhythm in DDD mode: this can only mean that we are dealing with a dual chamber since the pacemaker senses the atrium and paces the ventricle. A VVI would not sense the atrium and would pace its own frequency.

Strip VI-7. Permanently electro stimulated rhythm in DDD mode with permanent atrial and ventricular stimulation.

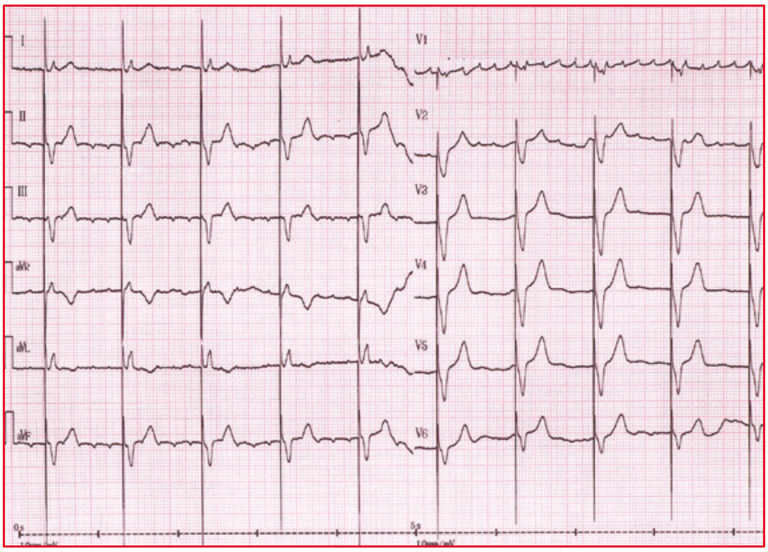
Main points to be remembered

1. Any P wave should be followed by a QRS, and every QRS should be followed by a P wave
2. All lengthening in the duration is a sign of conduction disorder. This could be either structural or functional;
3. A conduction disorder will either slow down nervous impulse in a given structure, or block its passage
4. Every layer of nodal tissue can be affected by a conduction disorder and can result in a proper conduction disorder.
5. The name and classification of AVB is done according to increasing severity
   a. Lengthened PR= 1st degree AVB
   b. PR is elongated then gets blocked= 2nd degree AVB of type Mobitz I
   c. P wave occasionally blocked= 2nd degree AVB of type Mobitz II
   d. Regularly blocked P wave= 2nd degree AVB of type 2.1
   e. Independent P wave and QRS= 3rd degree AVB
6. Treatments of conduction disorders rely on pacemakers, outside of the current etiological treatment (when it exists)
7. Pacemakers are characterized by three letters (+/- 1), indicating the cavity that is sensed, the cavity that is paced, the type of response that is generated and a last letter that qualifies the mode of stimulation (rate of modulation).
8. The interpretation of disturbances in repolarization remains possible in conduction disorders. This is not the case when the left branch is blocked or when there is ventricular pacemaker.

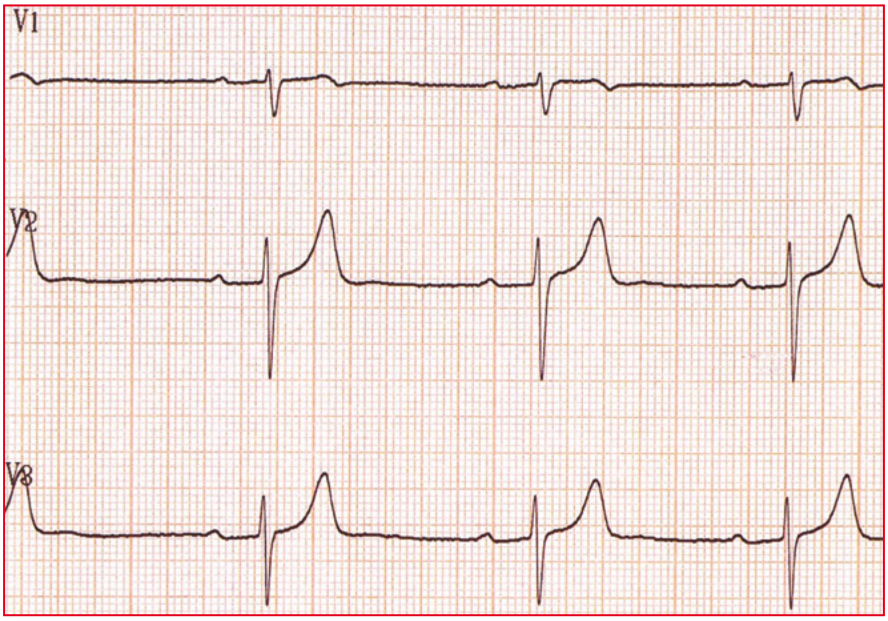
THREE EXERCISES ALONG WITH THEIR INTERPRETATION ACCORDING TO "OUR GUIDE"



Interpretation according to our guide	Normal interpretation
P wave is present; each is followed by a QRS. The PQ interval is identical from one complex to the next	Regular pacemaker rhythm
Frequency of 58 bpm	58 bpm
PR greater than 1 large unit	1st degree AVB
QRS are large. No R wave in V1	Blockage of the left branch
ST cannot be interpreted due to left blockage	BBG
Conclusion: regular pacemaker rhythm of 58 bpm, with 1st degree AVB and blockage of the left branch	



Interpretation according to our guide	Normal interpretation
P wave is absent and replaced by multiple organized waves, each at 350 bpm	Atrial tachycardia
Ventricular frequency of 65 bpm	65 bpm
QRS are large, preceded by a ventricular spike	Permanent electro stimulation in VVI mode
ST cannot be interpreted due to electro stimulation	
Conclusion: Permanently electro stimulated rhythm in VVI mode, of 65 bpm on atrial tachycardia background	



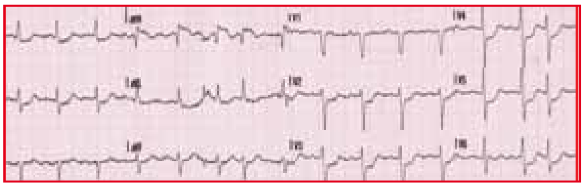
Interpretation according to our guide	Normal interpretation
P wave is visible, with slow and regular frequency	Regular pacemaker rhythm
Mean frequency of 38 bpm	38 bpm
PR greater than 1 large unit	1st degree AVB at 0.28 sec
Thin QRS, with no bunny ears in V1	No blockage of branches
Ascending ST in visualized leads	No lesions
Positive T in visualized leads	No ischemia
Conclusion: sinus bradycardia at 38 bpm, with 1st degree AVB, without repolarization disturbances. It could be due to a sinoatrial block of type Mobitz (one absent P wave out of two). But the graph is too short to be conclusive, and in this case we would need a graph with complexes at 76 bpm, followed by a recorded bradycardia.	

VII- REPOLARIZATION DISORDERS

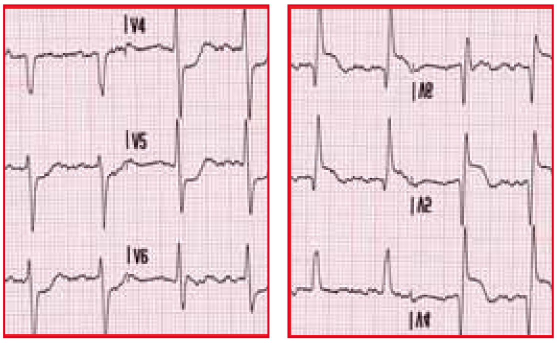
The myocardial repolarization often gives us more information on the myocardial cell than the QRS complex alone. The given information is not only on the cell's viability, but also on its functionality. The viability depends on a satisfying perfusion of the cell, which is mainly influence by the presence or absence of a coronary artery disease (CAD): ischemia or anoxia will have different representative aspects. Cellular function depends on multiple factors (overload, morphology, metabolism, iatrogenic property, specific location, etc.) and the electrical expression will be very close; the interpretation will have to consider the clinical context more than ever. Finally, it should not be forgotten that, in the cardiac cycle, the repolarization depends on the depolarization that depends on the conduction that depends on the heart rate that depends on the cardiac rhythm.

Ischemic cadriomyopathies

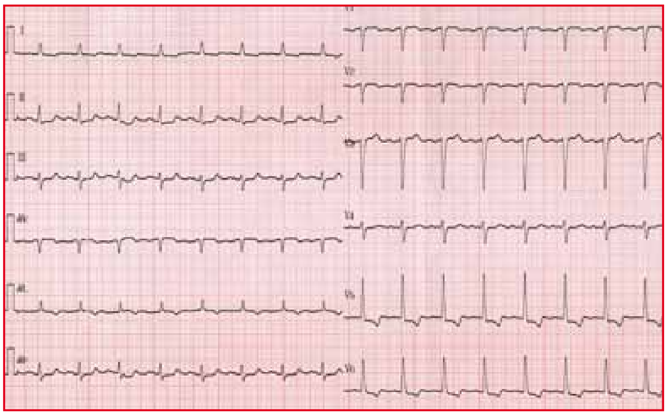
The typical anomalies are either the lesion currents or the coronary ischemias (reference to section IV). It is crucial to remember the dynamic of the ST segment as the ischemic cardiomyopathies and their decompensation wane, may they be acute or sub-acute **(Strips VII-1 to VII-3)**.



Strip VII-1. Regular sinus rhythm, with one ESSV (3rd complex in V4), shift below of ST in stretched inferior anterior plane, with a large R wave. This is a mirror image of a Pardee wave with a Q wave. Note the shift above of the ST in the isolated aVR, shift above of the isolated aVR with a stretched shift below = lesion of the common trunk until proven otherwise, which was the case in this patient.



Strip VII-2. To the left, recorded graph with the aspect of a large R wave and a shift below of the ST in the anterior stretched plane. To the right, same graph mirrored with the Q wave and shift above of the ST, which is a lot easier to recognize.



Strip VII-3. Graph of a 75 year old man, referred due to chest pain. Eeveryone recognizes the P wave, a QRS following every P wave and preceding every other P wave, thin QRS, without hemiblocks. There is a straight shift below of ST in the inferior plane, but also inversions of the T waves in the top lateral plane. If there is no doubt about the CHD in the inferior plane (ST that is more than rigid and shifted below), the disturbances that can be seen laterally are linked to a left post-hypertensive ventricular hypertrophy (ST shifted below but descending, negative T wave but very asymmetrical).

Left ventricular hypertrophy

Muscular hypertrophy of the left ventricle can have multiple translations on the surface electrocardiogram. Other than ample complexes, with indexes above 35 mm for R5 S1, there are frequent disturbances in repolarization, mainly lateral, similar to ST shifts below or inversion of T waves.

Metabolic disorders

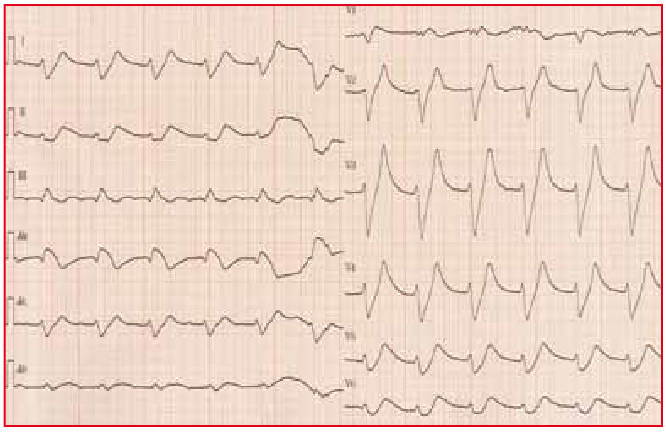
Hyperkalemia

Modifications in repolarization are proportional to the serum potassium level. The T wave becomes abnormally high and narrow, and at the same time there are modifications of the ST segment, a widening and distortion of the QRS, complexes that become excessively large, before transforming into ventricular fibrillations. The aspect is typical and it is life threatening. Calcium gluconate and Bicarbonates which are membrane stabilizatord are the first line therapy before beginning the etiological treatment [14,15] **(Strips VII-4 and VII-5)**.

Hypokalemia may present with complete normal ECG is completely normal. It can lead to a depression of the ST segment, a flattening of the T wave, a projecting U wave that is taller than the T wave. Rhythm disorders are also possible.

Hypocalcemia usually leads to a lengthening of the QT interval, which is responsible for transient torsade de pointe.

Hypercalcemia will shorten the QT interval, but lengthen the PR, which is responsible for causing a sino-auricular bloc.

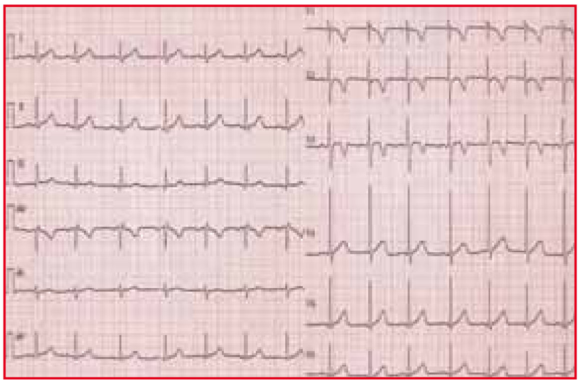


Strip VII-4. Typical aspect of a major hyperkalemia, at 9.8 mEq/L, with a very ample T wave, sharp, and a widening of the QRS. There still appears to be a sinus rhythm.

Strip VII-5. Graph showing extremely enlarged QRS complexes, with a very ample T wave, the rhythm is not easily determined (supposedly FA), preceding ample complexes, only two diagnoses are possible: either a major hyperkalemia, which is life threatening, or an intoxication with anti-arrhythmic medication of class Ic—in both cases, stabilization can be obtained only after intravenous administration of bicarbonates.

Neurodystony

The T wave is ample, especially in precordial, remains asymmetrical, but there is no true ST segment, or the ST segment is directly ascending and directly following the T wave. This is repolarization that is rather early. The ascending ST segment allows us to rule out an ischemic origin.



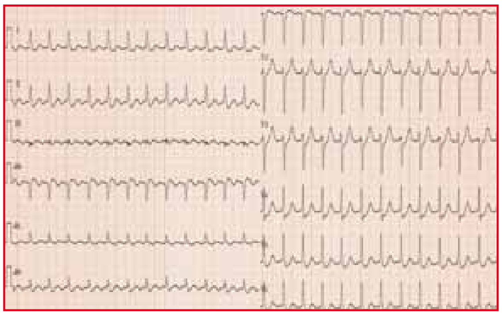
Strip VII-6. Graph of a 16 year old patient, with RSR, incomplete right block, negative T wave from V1 to V3 (V1=normal, V2= probable, V3= possible in 5% of cases), especially, suspended and ascending ST segment clearly visible in DI, DII, V4-V6.

Drug-induced disorders [16]

Amiodarone provokes a lengthening of the QT: it is due to an electrophysiological effect, with prolonged action potentials, and anterior subepicardial pseudo-ischemia with giant T waves. Bradycardia with lengthened QT can promote clever transient disturbances in ventricular rhythm.

Cardiac glycosides: there is reduced amplitude of the T wave, a pseudo shift below of the ST that remains concave, a cup-shaped aspect, a shortening of the QT, and an increase in amplitude of the U. The appearance of disturbances in rhythm is indicative of toxicity in these molecules and is a criterion of severity.

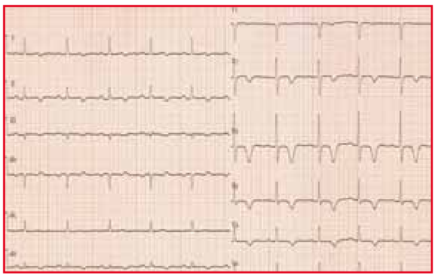
Dysthyroidism: Hypothyroidism, resulting from edema, cellular myocardial or pericardial effusion of the interstitium, will translate into a weak voltage of complexes, flattened T waves, and disturbances in conduction. This will exceed the cellular threshold of excitability, inducing disturbances in, mainly, supraventricular rhythm (sinus or atrial tachycardia, A-fib, etc.). **Strip VII-7** shows SVD due to hyperthyroidism.



Strip VII-7. There is no visible P wave, QRS are regular, thin, at 150 cycles/min, with a doubly woven aspect in inter-QRS, notably in the inferior plane: it is a auricular flutter at 300 cycles/min of conduction type 2/1. It exists in per-tachycardia, an aspect of subendocardial lesion current that is 3 to 4 cm shifted in lateral. This tachycardia is supraventricular and secondary to a hyperthyroidism.

The women

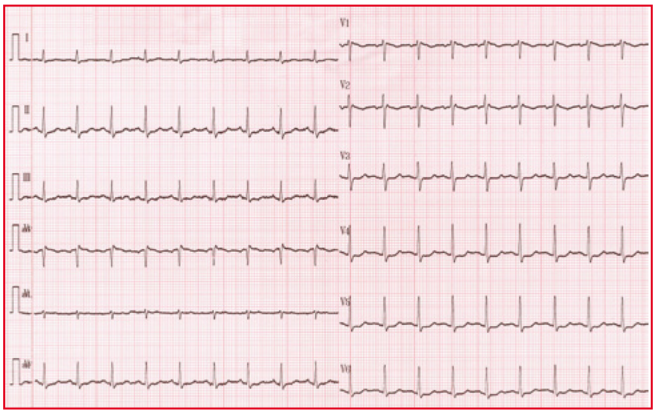
Repolarization disorders are very frequent, without necessarily meaning that there is an underlying cardiomyopathy [17, 18]. The disturbances can correspond to a flattened and diffuse ST segment, and aspect of subepicardial anterior ischemia with a negative T wave that theoretically remains asymmetrical, but could potentially be a diffuse subendocardial pseudo-lesion current. The difficulty remains to determine the absence of cardiomyopathy. A fluctuation with age is possible **(Strips VII-6 and VII-8)**.



Strip VII-8. 83 year old woman without notable previous history of cardiomyopathy, systematic pre-op ECG prior to a total hip replacement. Regular sinus rhythm, with negative T wave in over almost all of the derivations. The t wave remains asymmetrical (clearly visible in the anterior plane). The biology is normal as well as echocardiography. Angiography shows normal coronary arteries.

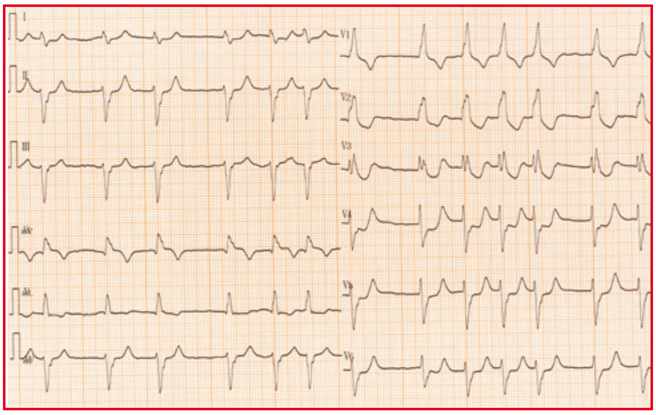
Thermal disturbances

Hyperthermia will lead to a reactionary tachycardia (+10 bpm/ degree above 37°C). This reactionary tachycardia will shorten the intervals between the QRSs, with possible disappearance of the ST, with the T wave getting closer to the preceding QRS. The ST segment will remain nevertheless ascending in the absence of a cardiomyopathy. At a more advanced stage, the tachycardia could mimic a stress test, with a subendocardial lesion current per tachycardia, the most common being in the lateral **(Strip VII-9)**.



Strip VII-9. Young 17 year old female, with flu-like symptoms, hyperthermia at 39.9°C, frequency at 111 cycles/min, that, when adjusted to 37°C, would equal to a frequency in the order of 80 bpm. Diffuse disturbances in unspecific repolarization.

Hypothermia will lead to a deceleration in conduction across all the conductive tissue, with a sinus bradycardia which could be a block, a lengthening of the PR, a widening of the QRS, a proportional lengthening of the QT… Below 32°C, we can note the appearance of an additional dispersion at the end of the QRS that corresponds to the J of Osborne wave **(Strip VII-10)**.

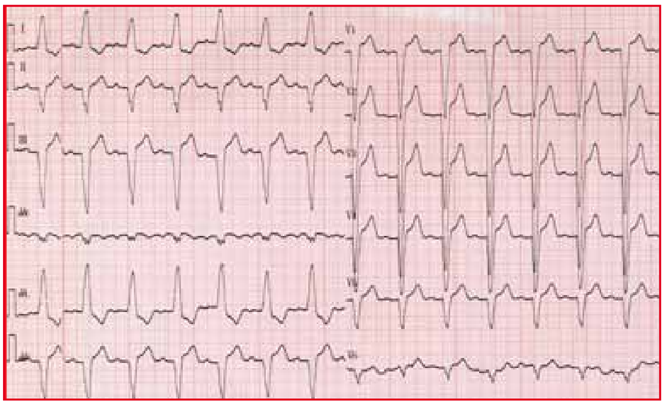


Strip VII-10. 42 year old man, hypothermia at 28.2°C, with previous history of ischemic cardiomyopathy, with auricular fibrillation, taking Digoxin. The graph records a rhythm in FA, with RBBB and ALHB. On the ascending ST segment, notably in DII, DIII, aVF, V4 and V5, there is a weaving that corresponds to the J of Osborne wave. The first two complexes in V3 have a "super digital cup-shaped aspect".

Conduction disorders (section III)

Right bundle branch block: The RBBB induces disturbances in repolarization in negative T waves from V1 to V2 or sometimes V3. The remainder of the repolarization stays perfectly interpretable, with the possibility of diagnosing acute coronary syndrome (ACS).

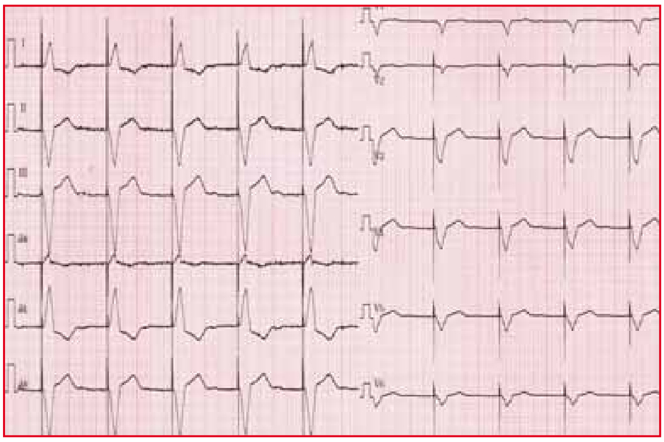
Left bundle branch block: The LBBB induces major modifications in the intraventricular depolarization circuits, reason for which it persists in addition to widening the QRS, a false aspect of shift above of the ST segment in the anterior leads. The presence of an LBBB does not further contribute to the interpretation of repolarization, and the classical criteria of ACS are no longer applicable **(Strip VII-11)**.



Strip VII-11. There is a P wave, a QRS after each P wave and before each P wave, the PR is constant, borderline at 0.2 seconds, the QRS is wide with a woven aspect in M inversed in V6 (or the absence of the R wave from V1 to V3). It is a typical LBBB. The repolarization is not interpretable, and from the start there is a false aspect of a permanent shift above of the ST in the inferior and anterior leads.

Cardiac pacemakers

Similarly, the modifications and disparities induced by cardiac pacemakers render, under normal conditions, the interpretations of repolarization disorders other than those induced by the pacemakers, impossible. The only exception is the pacemaker of type AAI, with conserved auriculo-ventricular conduction where, the physiological repolarization is conserved (refer to section VI) **(Strip VII-12).**

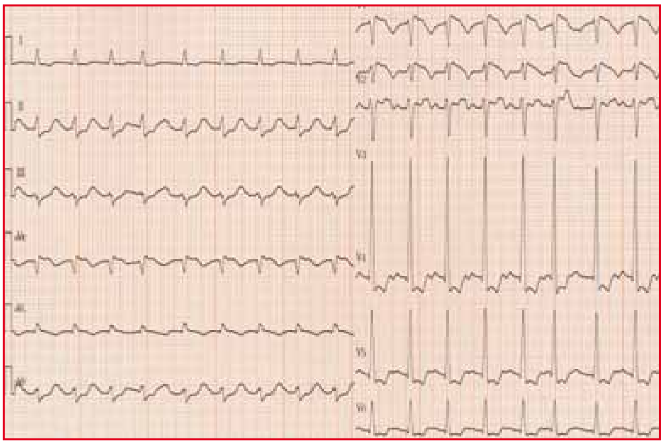


Strip VII-12. There is no visible P wave; the QRS are large, regular, preceded by an ample spike. It is a permanently electro stimulated rhythm according to mode VVI. Note the half-amplitude of the calibration in the anterior plane. We can notice similarities with the former LBBB.

Others

Pericarditis: A shift above in the ST segment, but seems suspended, remaining concave towards the top, with or without an association to the shift below of the PQ segment in inferior derivations. A microvolts aspect is also possible.

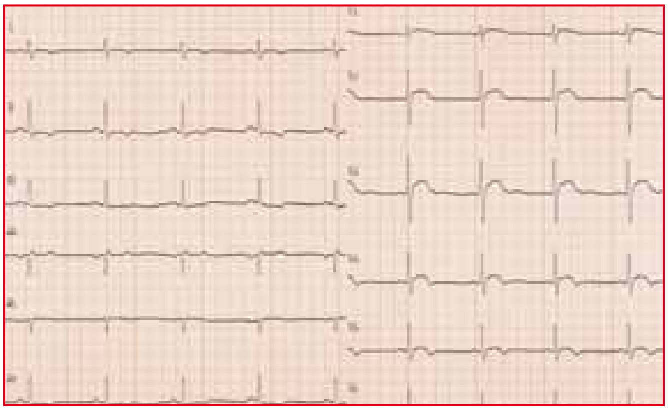
The Brugada syndrome: Described in 1992 by the Brugada brothers, this syndrome is associated with genetic inheritance, symptoms of lipothymia, loss of consciousness or sudden death, and anomalies in the ECG of type RBBB with an ST segment that is shifted above in the right precordial **(Strip VII-13)**.



Strip VII-13. Graph of a 37 year old patient admitted for cranial trauma with loss of consciousness. It is the 3rd episode of sudden and abrupt loss of consciousness. Other than the RSR, there is a RBBB aspect with shift below of the ST in V1-V2, an aspect of ventricular hypertrophy (ample complexes, depressed in lateral). It is a Brugada syndrome type 1.

Myocardial contusion: Rhythm and conduction disorders are more frequent. The appearance of a RBBB, ventricular or auricular extra systoles are present. Repolarization disorders of a type to flatten or negative T waves are possible.

The noncompaction of the left ventricle: Being rare, the electrocardiographic disturbances are characterized by a dome-shaped aspect of the ST segment in the anterior leads. They are secondary to a hypertrophic cardiomyopathy with intraventricular trabeculations noticeable on echocardiography. Clever ventricular rhythm disorders and heart failure are the major complications [19] **(Strip VII-14)**.

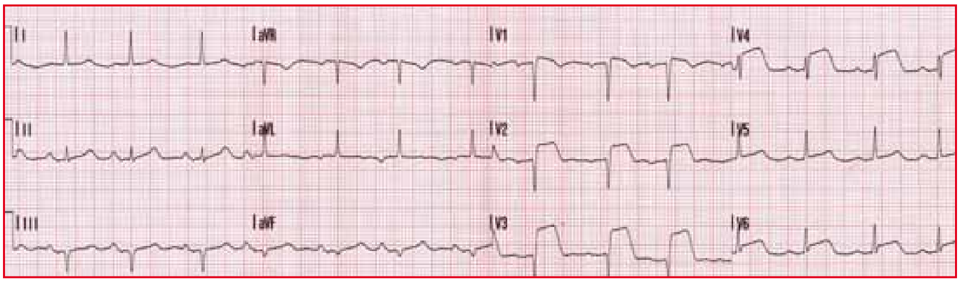


Strip VII-14. 48 year old man, graph was ordered because of an iterative loss of consciousness. Dome-shaped aspect of repolarization in the anterior leads, with a sub-epicardiac ischemic aspect in the apico-lateral and inferior. Also note the dome-shaped aspect that is concave towards the bottom in DII. This is a non-compaction of VG, confirmed by echography. The treatment, in addition to beta-blockers, can consist of the implantation of a defibrillator.

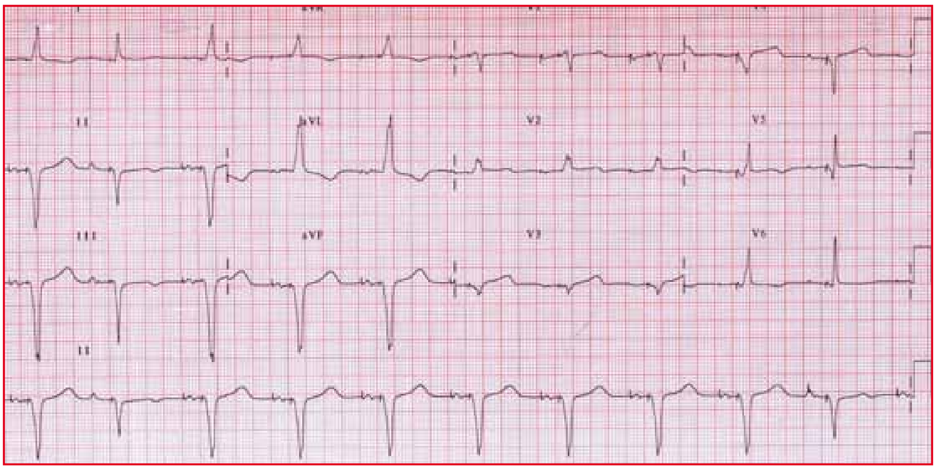
Main points to be remembered

1. Repolarization disorders are frequent, they represent, until proven otherwise, a cardiomyopathy or an underlying pathology;
2. A shift above of the ST segment is a STEMI until proven otherwise, except the LBBB or ventricular electrostimulation;
3. A shift below of the ST segment is a non STEMI until proven otherwise, more so than the shift below is straight;
4. The localization of abnormalities leads to suspect the affected coronary artery;
5. A negative T wave, except in aVR and V1, is abnormal until proven otherwise;
6. A symmetrical T wave is abnormal
7. With severe hyperkalemia, the vital risk appears in the short term, noticeable on the ECG graph.
8. With possible Brugada syndrome, the vital risk appears in the short term
9. In women, a comparative graph is regularly necessary.

TWO EXERCISES ALONG WITH THEIR INTERPRETATION ACCORDING TO "OUR GUIDE"



Interpretation according to our guide	Normal interpretation
P wave is visible, with regular frequency, with a QRS after and before each P wave	Regular sinus rhythm
Average frequency of 80 bpm	80 bpm
Constant PR	No 1st degree AVB
Q wave is present in V1, V2, and V3	Necrosis sequellae
Thin QRS, with no bunny ears in V1 or V6	No bundle branch blocks
ST shifted above from V1 to V4, less in V5, and again in V6—no visible mirror image in the inferior or in lateral	Sub-epicardial lesion current or Pardee wave in the extended anterior plane
T positive in all derivations except aVR and V1	No ischemia
Conclusion: Regular sinus rhythm at 80 bpm with no conduction disorders, with STEMI? Is it really due to a myocardial infarct in the acute phase? The acute phase can be confirmed at the clinic, here, in the absence of chest pain, but past history of infarct dating back to eight weeks. The fact that there is absolutely no mirror image, despite the amplitude of the shift above, must perplex the reader; it is an aspect of sequellae in a myocardial infarct that is anterior and extended, at the 8th week, with the presence of a left ventricular aneurysm.	

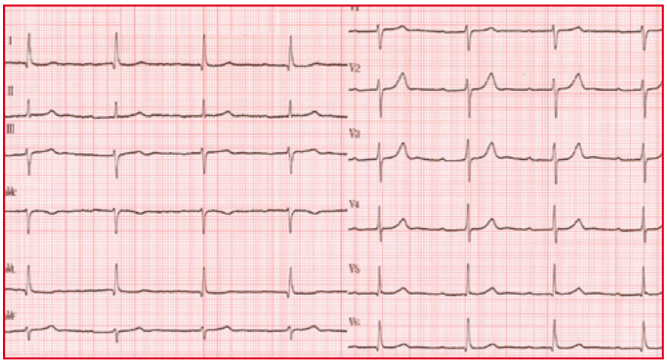


Interpretation according to our guide	Normal interpretation
P wave is only visible in the 2nd and 10th complexes	Intermittent sinus rhythm
Presence in the 2nd complex of ventricular spikes→ it is a stimulator that "listens" to the atrium and "stimulates" the ventricle	Cardiac stimulator of type DDD
Average frequency of 60 cycles/min	60 bpm
PR is greater than one unit in the 2nd complex, with PR at 0.24 seconds	When there is a negative P wave, the auriculo-ventricular is surpassed in order to economize the battery's energy
The other QRS are preceded by a wave that is itself preceded by a spike, with an inter-spike distance of 0.2 sec → in addition to listening to the atrium, it also stimulates it	In the absence of the native P wave, auriculo-ventricular delay is 0.2 sec
Wide QRSs, post-electrical stimulation	Permanent ventricular electro stimulation
The repolarization is non-interpretable due to electro stimulation	
Conclusion: The electrically stimulated rhythm according to the DDD mode, with the intermittent occurrence of a native P wave - with no possible interpretation of repolarization in this context. In the 10th complex, the naïve P wave is not extended enough in time by the pacemaker, reason for which an auricular spike exists, but the spike occurs at the peak of the native P wave, during the refractory period of the atrial myocytes, reason for which we can say that it is not the spike that is conducting, but rather the native complex.	

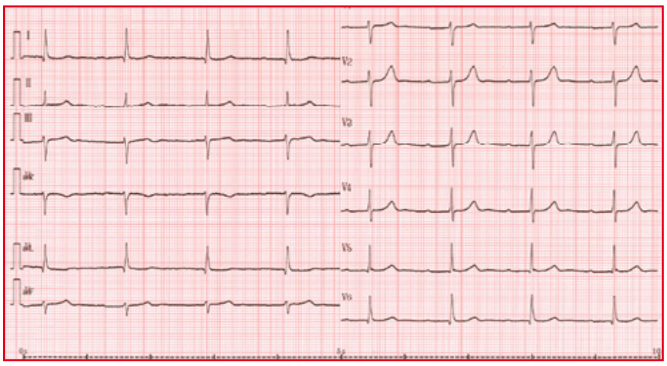
VIII-ELECTRICAL TRAPS

As we have seen in the previous sections, the interpretation of an electrocardiogram calls for knowledge of anatomy, physiology, electrophysiology, pathology, and must be integrated in a diagnostic approach revolving around the patient

However, this procedure depends on the quality of the recording of the strip and on respecting the basic rules of producing the strip. These elements are easily acquired with little practice; however one must not forget the following: more than ever, our logic will depend on a machine. To illustrate this, let us look at **strip VIII.1**: a priori, there is a P wave in front of each complex, the rhythm seems regular, the PR is constant but greater than one large unit, the QRS are thin, there is no M aspect in V1, DI and DII are positive, the ST is isoelectric, and the T waves are all positive (except in aVR which is normal). The strip can therefore be considered to be within the limits of the normal, but with a lengthened PR. However, is it a normal sinus rhythm (NSR) at 46 bpm with a 1st degree ABV (PR at 0.40 sec), or is it a patient in NSR at 92 bpm with no 1st degree ABV but with a PR at 0.20 seconds recorded at 50 mm seconds? The two answers are valid, and no one can confirm the answer without having instructions and inscriptions of the recordings. The therapeutic implications can be important depending on the context.



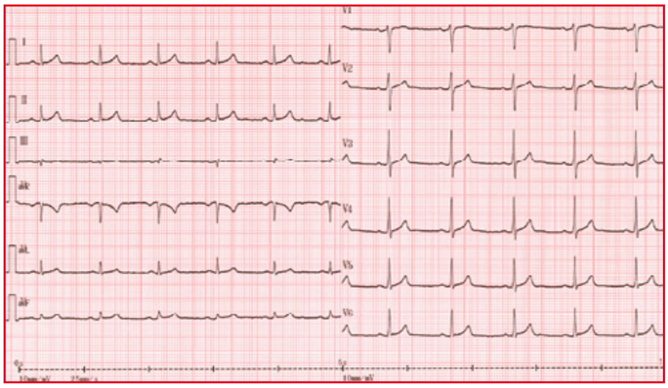
Strip VIII-1. 1st degree ABV or NSR with no ABV recorded at 50mm/sec?



Strip VIII-2. Same recording as strip VIII.1 with speed and calibration that define the strip.

Strip VIII.2 provides the solution, depicting the speed of the recording and the calibrations used. It is a NSR with bradycardia at 46 bpm, and 1st degree ABV, the PR being 0.40 seconds.

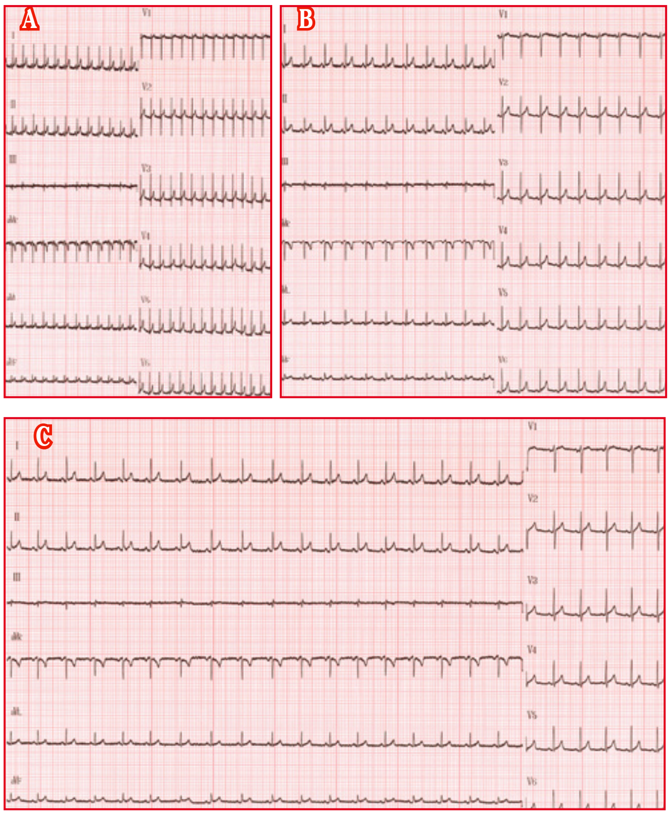
For the rest of this section, **strip VIII.3** will be the strip of reference, depicting a healthy subject; the inferential analysis will be based on this strip: the various possible incidents in recording and their repercussions on the recording will be analyzed.



Strip VIII-3. Reference strip, of a healthy subject.

Traps in cardiac rhythm

The cardiac rhythm determination is essential in the chronology of the interpretation of a strip. The standard strip uses a scrolling speed of 25 mm/sec, allowing for the calculation of cardiac frequency following the rules of 300/150/100/75/60 (section III).



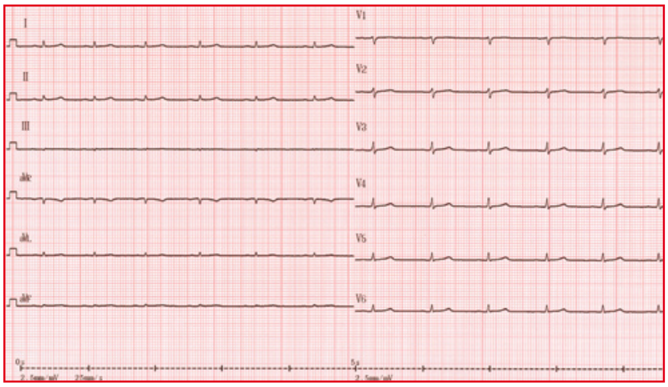
Strip VIII-4. A, B, and C: What do you see?

What do the **strips VII-4 A,B and C** show? For VII-4 A, do you agree with the following interpretation: supraventricular tachycardia with thin complexes ant 325 bpm? Does it suggest a Bouveret disease? For strip VII-4 B, is it a sinus tachycardia at

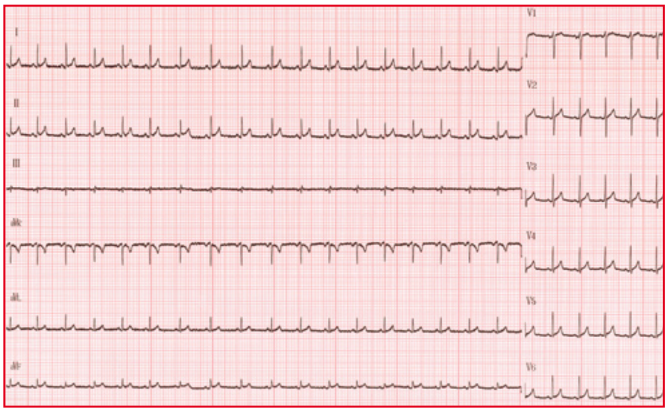
162 bpm? For strip VII-4 C, is it a sinus tachycardia at 130 bpm? All of these answers are correct and false at the same time since neither strip notes the speed of the recording. Actually, it is the same strip recorded with a speed of 5 mm/sec in A, 10 mm/sec in B, and 12.5 mm/sec in C. The frequency is still the same, 65 bpm; it is just the scrolling that varies. Obviously, the handling of Bouveret disease or a sinus tachycardia, or a NSR is not at all identical.

Traps in the amplitude of the strip

A standard recording is done at 10 mm for 1 mV, with a noted calibration at the start of the strip. If this calibration differs from the former or is not adjusted to this value, disturbances can occur, either to flatten the complexes or to stretch them. **Strip VIII-5**, recorded with an mplitude of 2.5 mm for 1mV, reveals microvolted complexes evoking pericardial effusions similar to a cardiac tamponade. Practically flat derivations appear, notably in the peripheral leads (DII or aVF). The same applies for **strip VIII-6** with a calibration of 5 mm/mV.

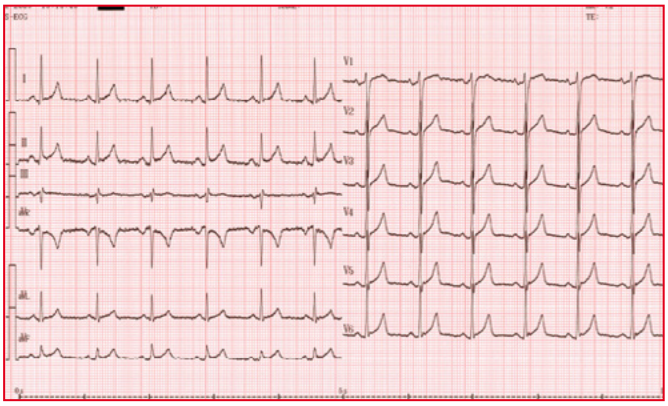


Strip VIII.5. Recording with an amplitude of 2.5 mm/mV.



Strip VIII-6. Recording with an amplitude of 5 mm/mV.

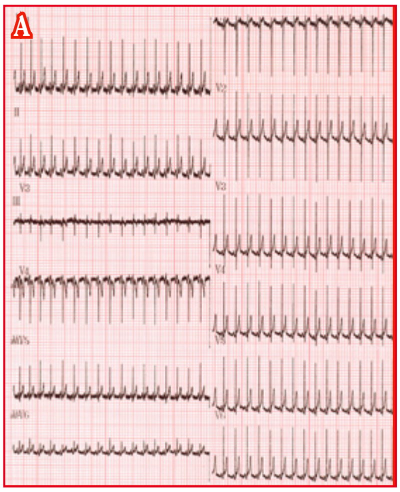
On the contrary, amplitudes greater than 10 mm/mV will produce strips with overlapping QRSs, especially in the precordial leads, and will wrongly evoke ventricular hypertrophies, similar to shifts above that are found in young patients with a particular cardiac erethism **(Strip VIII-7)**.



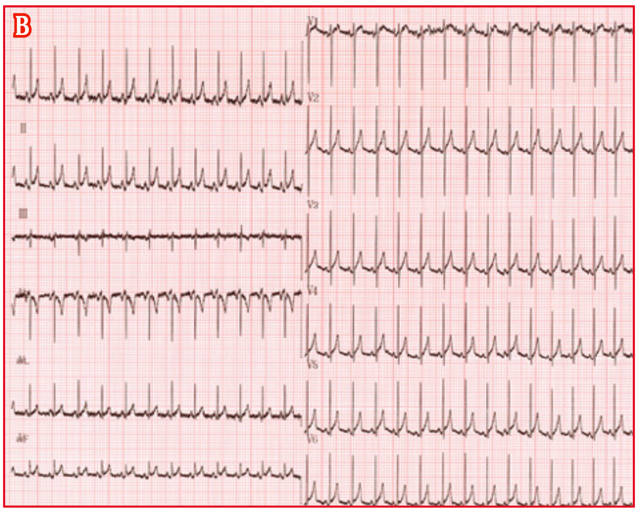
Strip VIII-7. Amplitude of 20 mm.

Traps associated with speed and amplitude

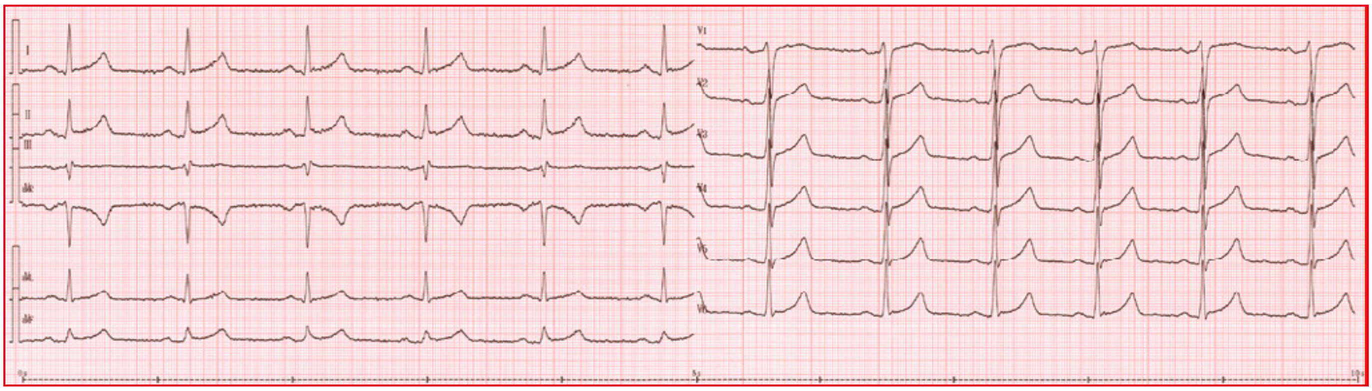
These two mistakes in calibration can clearly potentiate, as can be noted in the following **strips (VIII-8 A and B)**. strip VIII.8 A is recorded at a speed of 5 mm/sec, with a calibrated amplitude at 20 mm/mV. This results in a rapid tachycardia aspect, with very thin complexes, greater than 300 bpm, evoking a very rapid supraventricular tachycardia. Such a strip is possible under certain conditions (young patient, newborn). Strip VIII.8 B seems more plausible and could correspond to an adolescent after a bit of physical effort.



Strip VIII-8 A. Speed of 5 mm/sec and amplitude of 20 mm/mV.



Strip VIII-8 B. Speed of 10 mm/sec and 20 mm/mV.



Strip VIII-9. Speed of 50 mm/sec and an amplitude of 20 mm/mV.

Sometimes, the aspect of a strip could be less shocking, and it is just its observed frequency that is surprising. **Strip VIII-9**, where the speed and amplitude are doubled, records QRS complexes of a normal aspect, but with a possible 1st degree ABV, and a bradycardia at 33 bpm. The repolarization seems halted notably in the inferior-lateral plane.

Traps in electrode positioning

The inversion of electrodes is frequent and common, but is easily discernible by respecting, and consequently looking for, some basic rules that we can call ground rules. The complexes must be consistent amongst each other, notably in the explored coronary territories.

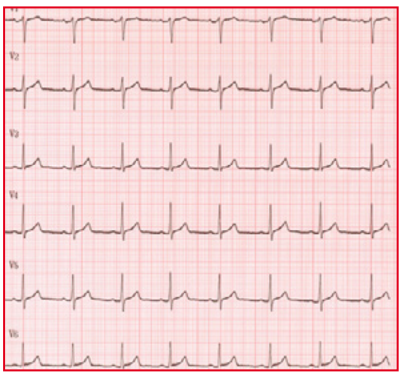
The anterior interventricular artery that supplies the anterior wall of the left ventricle corresponds to the precordial territory: from V1 to V5 (possibly V6) there must be a regular progression of R, and in parallel but slightly delayed, a decrease in the depth of S. A planing of R in the anterior is possible in specific cases of sequelae of infarct, but the logic persists and is only delayed with a booting of R in V3 or V4 (Section III).

To be clear,

• R in V1 is inferior to R in V2, which is inferior to R in V3, which is inferior to R in V4, which is inferior to R in V5, which is inferior to R in V6, and

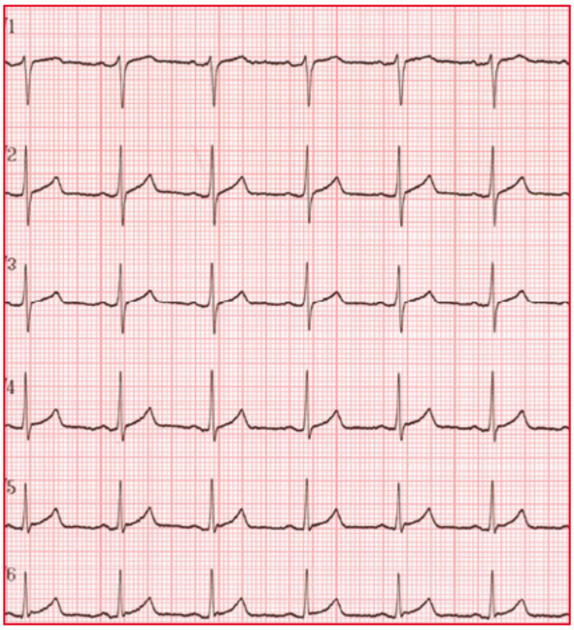
• S in V1 is deeper than S in V2, which is deeper than S in V3, which is deeper than S in V4, which is deeper than S in V5, which is deeper than S in V6

• Or more schematically speaking [R1<R2<R3<R4<R5<R6] and [S1>S2>S3>S4>S5>S6].



Strip VIII-10. Inversion of V3 and V5.

Strip VIII-10 does not respect this logic: R1<R2<R3<R4=R5>R6 and S1>S2>S3<S4<S5>S6. These anomalies are due to an inversion of V3 and V5 that disturb the logic of the progression of R and the regression of S.



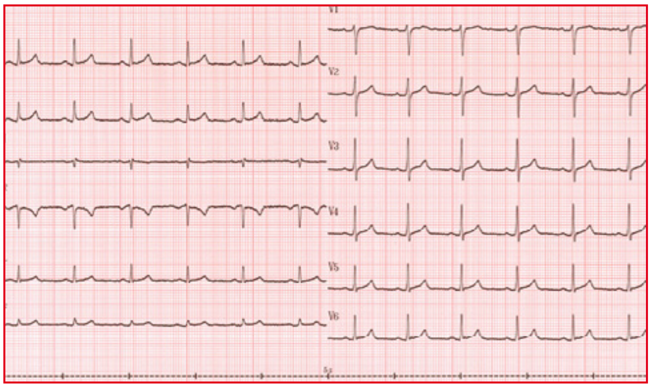
Strip VIII-11. Misalignment of V3.

Strip VIII.11 reveals a misalignment of an electrode that is a bit more difficult to visualize but very frequent to occur, knowing R2>R3. If a vertical line, parallel to the vertebral column is drawn, the correct position of the electrodes can be verified by a progression of V1 to V6 (section I). The lines passing through V2 and V3 are very close to one another, but V3 remains between V2 and V4. This is not a true inversion of electrodes but rather V3 is placed between V1 and V2, this most often occurs due to the anatomy of the patient (breast, breast implant…). On the other end, an isolated Q wave in V3 is possible.

In the peripheral derivations, DI and aVL (upper lateral territory, supplied by the circumflex artery), the QRS complexes must closely resemble each other, with the same aspect. Positive DI and negative aVL (or the inverse) is not physiologically possible.

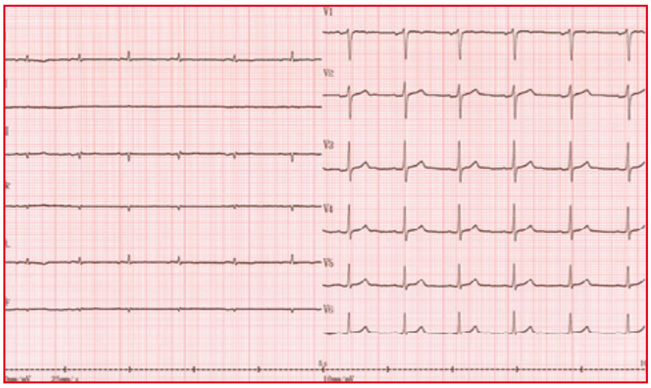
Always in the peripheral leads, DII, DII, and aVF (inferior territory, generally supplied by the right coronary artery). strip VIII.12 to 14 reveal the inversion of electrodes due to the failure to abide by the colors RBGY (red, black, green, yellow).

An inversion between the green and the black (lower right and left extremities) barely alter the strip **(Strip VIII-12).** The strip is within normal limits.



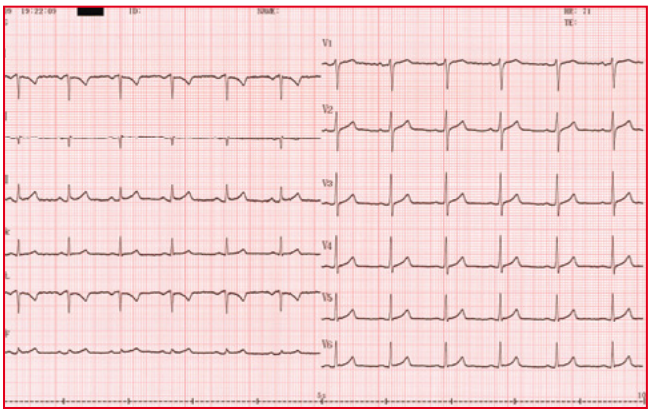
Strip VIII-12. Inversion of G and B.

An inversion of the red and black electrodes (right extremities, both upper and lower) is more flagrant with a recording that is quasi-inexistant in DII, DIII, aVF, and microvolted in DI-aVL, as can be seen in **strip VIII-13.**



Strip VIII-13. Inversion of R and B.

The inversion that is most easily recognized is the one that can be seen on **strip VIII-14**, with an inversion of the red and yellow electrodes (both right and left upper extremities), because this appears when there is a discord between DII-negative-, DIII-positive-, but especially when aVR becomes positive.

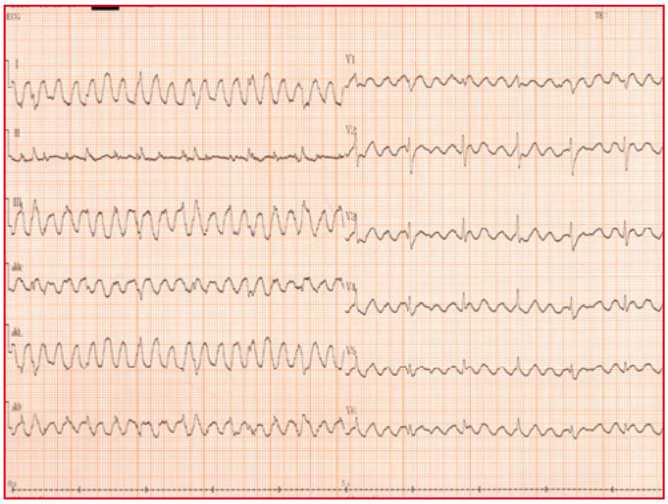


Strip VIII-14. Inversion of R and Y.

Traps associated with recording devices

The two main types that should be recognized in this category: artifacts and errors in interpretation (for devices that have a help menu to help you interpret the strip).

The artifacts can have multiple causes. Muscular contractions, comitiality, Parkinson's disease, muscular disease, hiccups, electrical interference, defect in grounding the device, type of current, artifacts starting at 60 Hz, etc. The list is far from being exhaustive, and contains potential causes of artifacts that can be seen on the strip. These are generally clearly recognizable by parasites in the baseline, more or less broad, with the persistence of a normal ECG added to these artifacts. The installation of water as a conductor between the electrodes and the skin, the use of electrodes that are gelled in advance, the use of filters, asking the patient to relax and stop talking, are all elements that allow us to minimalize interferences. **Strip VIII-15** is caricatured. It is a strip that is systematically done before surgical intervention in a 70 year old patient presenting with a fracture of the neck of the femur. A diagnosis of ventricular flutter was mentioned. Large meshes were observed at 300 bpm in DI. Is this possible? No, and for multiple reasons. The first is that a ventricular flutter is rare, and usually degenerates after a few seconds to become a ventricular fibrillation. When faced with any difficult interpretation of an ECG, it is essential to go back to the basics. Do we see any P waves? Is the rhythm regular? Are the QRS thin or large? These three questions will give you the answer.



Strip VIII-15. Electrical artifacts with visible and regular QRS complexes.

Do we see P waves? A priori, no at least not at first glance.

Is the rhythm regular? In precordial a priori, yes.

Are the QRS thin or large? In precordial, they are thin, with a frequency at 74 bpm.

Thin complexes, regular at 74 bpm, is too fast to originate from the ventricles **(Section V)**. Therefore this can only be originating from the supra-ventricular area (atrium or node). If the origin is supra-ventricular, the meshes in the baseline are too large and too organized to be a fibrillation. Is this a flutter or an atrial tachycardia? If yes, the ratios of these waves and the QRS in the precordial should be identical, from one complex to the next. However, if on the 2nd QRS in V2, this wave is close to the QRS, it is less so for the 3rd QRS and it is even more distant for the 4th complex, thus this interpretation is incorrect. A regular QRS rhythm with an irregular supra-ventricular activity cannot exist.

Our reasoning is therefore false. Therefore, let us go back to our first question: do we see P waves? The P waves are usually more visible in peripheral derivations, and the only area that fits these parameters is DII. The QRS are regular in the precordial, they must be so in the peripheral. The QRS complexes are therefore broader waves than we would have guessed in DII, interrupted by two smaller sharp waves that also seem regular but have a different frequency (about 160 bpm). If the QRS are regular at 74 bpm, we must find a preceding P wave. When talking a closer look, the 3rd QRS in DII is preceded by a clear P wave, with a PR at 0.16 seconds. This P wave allows us to reveal the other P waves that precede the QRS in DII, and these P waves are added to the other artifacts. Finding a P wave on a regular rhythm means that this is a sinus rhythm. In fact, it is a strip with a normal sinus rhythm at 74 bpm with thin QRS, and if we examine the waves with regards to the detected QRS in DII, we notice on the other peripheral derivations that the QRS are added to the baseline waves.

The device has been turned off, rebooted, the electrodes remained on this patient, the sector was unplugged, the device was used on batteries and a new recording turned out to be normal. The baseline waves were due to an inverter.

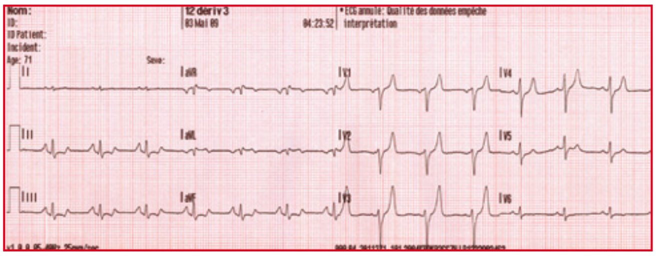
Should we trust the devices' help menus when it comes to interpretation?

I believe that the answer is simple: to the extent that we can trust the measures of duration recorded by the device, we should also question its interpretation. **Strip VIII-16** is another clear and simple illustration. The quality of the values impedes the interpretation!!!

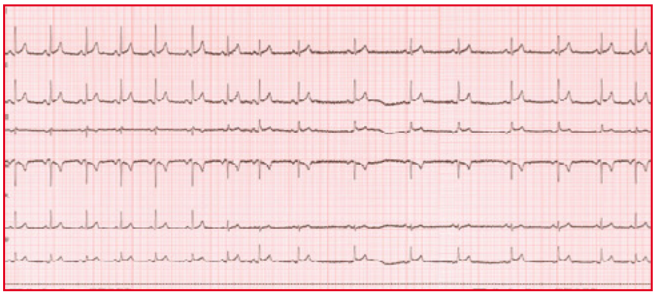
In emergency medicine, it is rare to obtain strips with such a small number of artifacts.

What man can do?

The presence of an isolated Q wave, not very deep in DIII, can be physiological, and will disappear once the subject takes a deep breath. **Strip VIII-17** is a caricature of what man can do by simply mimicking a vagal maneuver. Closely observe DIII. It is a sinus rhythm, regular at 72 bpm. We ask the patient to take a deep breath and hold it, starting from the 7th complex, the QRS becomes positive, the rhythm is originating from the coronary sinus (negative P wave in DIII) and slow down, and we even note a shift above of ST in DII-DIII-aVF that appears but remains ascending. The situation returns to normal upon resuming breathing.



Strip VIII-16. Interpretation is not possible???

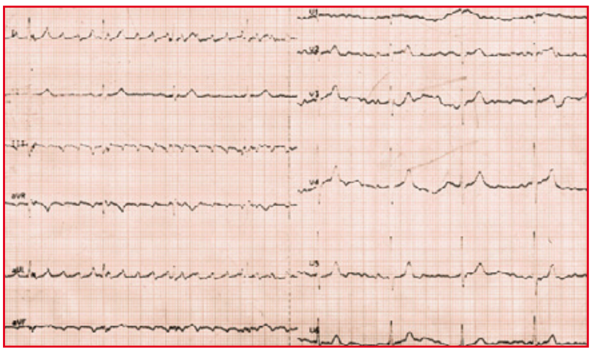


Strip VIII-17. Same subject, with deep inspiration.

Main points to be remembered

1. The interpretation of the strip depends on its quality.
2. The standardization of the strip is 25 mm/s for the speed, with an amplitude of 10 mm/mV.
3. The inversion of electrodes can mimic Q waves.
4. One must be critical, especially when the interpretation seems complex, and one must revert to simple questions.
5. One must sometimes see beyond what our eyes see at first glance.
6. A strip can hide another.

EXERCISE ALONG WITH THEIR INTERPRETATION ACCORDING TO "OUR GUIDE"



Interpretation according to our guide	Normal interpretation
P waves are visible in DII, all are followed by a QRS, and each QRS precedes a P wave	Normal sinus rhythm
P waves are not visible in the other derivations, but there is a jerk at 280/min that seems regular, however the regular sinus rhythm recorded concomitantly on other corresponding derivations is normal, thus, we are dealing with artifacts	
Regular frequency of 50 bpm	50 bpm
Constant PR at 0.20 sec	No, or barely any 1st degree ABV
Thin QRS, no bunny ear in V1 or V6	No bundle branch block
Positive T in all leads, except in aVR and V1	No ischemia
Conclusion: Normal sinus rhythm at 50 bpm, with no conduction or repolarization disorders, but with diffused artifacts that are not visible exclusively in DII. This describes a patient with Parkinson's disease affecting the left hemibody.	

IX- ELECTRICAL AND CLINICAL CORRELATION

When reading an ECG the physician has two questions in mind:

• Is there an ischemia that I'm missing?

• Is there a life threatening arrhythmia?

These questions are the main concern when confronted with a chest pain or discomfort. Answering them should help decrease mortality.

We have seen the importance in doing a proper ECG in the clinical setting, with proper lead placement and standardized proper scale (time and amplitude). Even if by analyzing the 12 lead ECG (ideally 18) that represents three dimensional cardiac chambers morphology, there exist areas that are seldom if not at all examined. The sign are not clearly apparent. We will discuss the indirect signs of ischemic heart disease and threatening rhythm disorders.

The Acute Coronary Syndrome (ACS)

The diagnosis of ST elevation MI (STEMI) is easy, if the ECG didn't show LBBB or pacing leads. However 80% of ACS don't present with ST elevation MI. Most ACS cases are due to subendocardial infracts, there are ECG findings we need to look for that are clinically significant and prompt urgent management.

Normal ECG

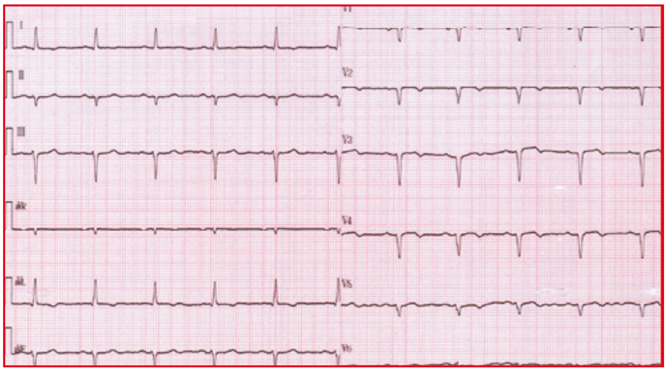
A reminder for normal ECG findings **(table IX-1)**:

ECG finding	Interpretation
Presence of P wave before each QRS which is followed by a P wave	Normal sinus rhythm
PR interval is constant < 1 large square	No AVB
Narrow QRS no M in V1 no W in V6	
Equal shape QRS	No bundle branch block
No PVC	
No Q wave	No old infarct
I is positive	No LAHB
II is positive	No LPHB
ST is isoelectric	No acute lesions
T waves are positive except V1& aVR	No old ischemia

Table IX-1

Adding to those findings, a normal R wave progression in precordial leads, T wave is proportional to R wave and no ST changes over time.

The smooth R wave progression was explained previously. Poor R wave progression is considered a flattening of the R wave sometimes referred to "electric gap" which will lead to formation of a Q wave **(strip IX.1)**.

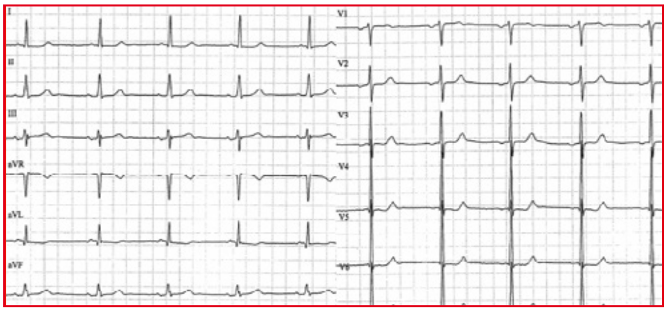


Strip IX-1. NSR with absent R wave in the precordial leads, it is not a Q wave because there is a tiny R wave starting V3. To note that we have a LAHB (II is negative) and an antero-lateral subendocardial infarct (V2-V5, I and aVL).

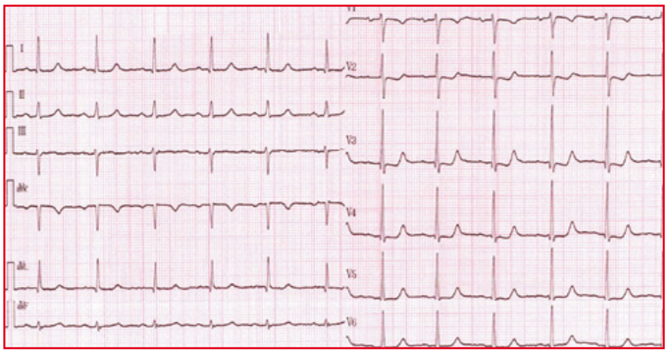
Normally, T wave should be proportional in shape to the R wave. Usually it is supposed to be roughly equal to a third of the R wave.

Finally, in the lead where the R wave is dominant, the QRS and ST segment should be in "concordance" when one is positive the other is positive and vice versa.

We should also not forget that coronary disease is progressive disease whose symptoms are mainly chest pain, which depends on the severity of the lesions, the efforts (severity of activity Vs at rest), the need of oxygen and pathology of the event. So if we have progressive clinical symptoms with no ECG changes rules out of favor of coronary origin. And the opposite is true; if symptoms worsen and we have ECG changes, then this is due to coronary disease **(strip IX-2 and IX-3).**



Strip IX-2. 57 year old patient ECG at rest: NSR with left ventricular hypertrophy.



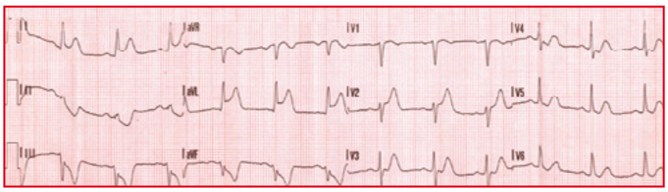
Strip IX-3. Same patient presenting with anginal pain with new an acute sub endocardial lesion in anterior lead. ST depression in V2 and gets clearer in V3. When off pain we have normalization of findings.

Why and how to detect suspicious ECG?

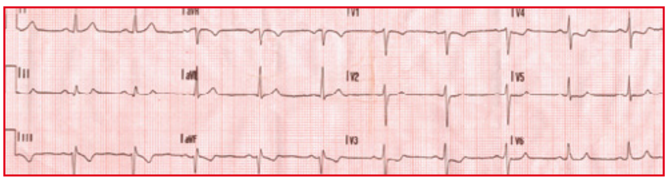
Detecting and diagnosis suspicious ECGs will allow physician to manage ACS accordingly. If the management of patients with STEMI is well structured, It was not until very recently that specific recommendations for the Non STEMI ones have been published, although paradoxically, a higher mortality rate existed in this group [20]. It is well established that the treatment is more efficient when started early specially that the ACS is progressive. So it is crucial to have an early diagnosis and thus decrease morbidity and mortality of non STEMI/ ACS

If there is any doubt on the ECG findings and the clinical symptoms, it is better to repeat it 15 minutes later and follow these changes. It is more cost effective than a serial troponin:

- For the troponin to increase, there must be an ischemia (& pain) of at least 20-30 min; this delay can be shortened with the latest generation markers, but a delay must always exist before an elevation;
- A rise will be noticed at least after 4 hours for classical troponins
- A negative troponin after 3 hours of pain will compared to a second set 3-4 hours later, so a time loss of at least 6 hours
- After 6 hours how many arrhythmia or sudden death can occur?
- ECG changes are apparent after 2 min of ischemia.
- In the case of doubt, a cardiac echography is more susceptible than a bioassay.



Strip IX-4. Patient presenting with intermittent chest pain for the past 24 hours. 5 min later the ECG shows STEMI (Pardee's sign wave) in lateral leads with mirror images ST depression in inferior leads due to circumflex stenosis.



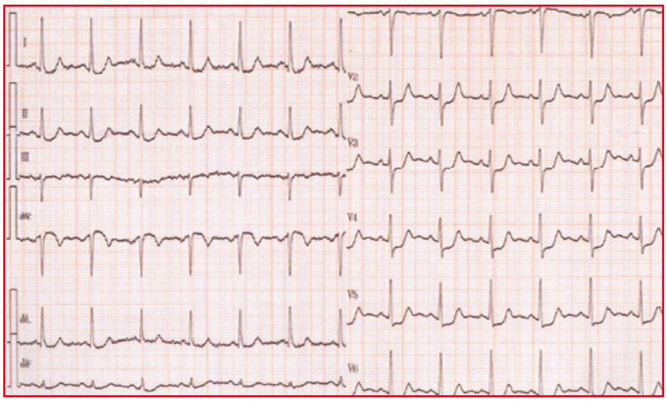
Strip IX-5. Same patient 6 min after the last ECG. The V3-V4 changes in the 4th strip were more appreciated after normalization. This is a normal ECG. And the troponin level will be normal however with occluding the Cx the patient can fibrillate and die.

1. ST segment:

An ST elevation is easily identified, in addition to the mirror image ST depression in opposing lead. An inferior infarct will have anterior mirror ECG changes. An ST depression in V1 and V2 is a mirror image of the posterior wall **(Strip IX-4)**.

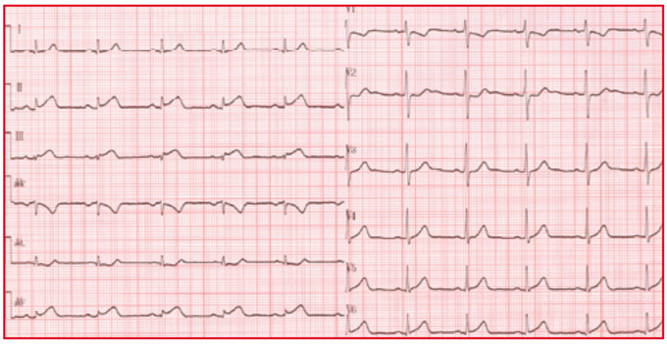
The repolarization of V1 must roughly be the inverse of V6 (unless we have a pacer). V6 is the mirror image of V1 **(Strip IX-5)**. If there is discordance then there is an underlying pathology.

In ECG **strip IX-6**, a subendocardial ischemia is always serious which is seen in the ST depression in lead V3 which can never be physiologic. Similarly in the strip 5, an isolated ST depression in V3 with the presence of clinical chest pain is significant and can only mean an acute ischemic cardiomyopathy and it would be harmful not to refer the patient to a cardiologist.



Strip IX-6. ST depression in the anterior leads: immediate and aggressive management.

The localization of the ST depression is also significant. The Right ventricle is located at 120 degree on the axis III. An inferior ST elevation will be maximal in lead III. And the opposite is true. The mirror axis of 120° is -60° which is aVL so the maximal ST depression will be seen in aVL. This subtlety is important, if the ST elevation in III is larger than the elevation in lead II or the depression in aVL is larger than I, the localization of the ischemia is proven **(Strip IX-7).**



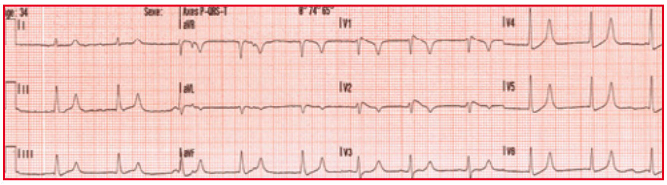
Strip IX-7. Inferior infarct. The largest depression is in aVL (ST depression barely seen in DI) and the largest elevation is in lead III even if the hyperacute T wave in lead II and aVF: it's the ST elevation that matters. Notice the mirror image in V2.

One more thing, don't forget to look at your ECG upside down too, it's easier to visualize and detect an ST elevation rather than a depression. By flipping the ECG we notice ST elevations that we didn't detect (Strip IX-8).

Strip IX-8. On the left, V2 of the strip IX-7, on the right same V2 but the strip is flipped. Some might consider the ST depression due to artifact related to respiration, by flipping the ECG a Pardee's sign of hyperactive ST wave followed by sub endocardial ischemia is appreciated.

2. T wave

We already mentioned that the T wave is proportional to the R wave. And the size of the R wave is always smaller than the T wave. If this is not the case then we are dealing with either ischemia or hyperkalemia which cause a peaked T wave. Both have peaked T wave however the ischemic T wave is of wide base symmetrical. The hyperkalemia peaked T wave is of narrow base **(Strip IX-9)**.



Strip IX-9. 34 year old patient with acute renal failure and hyperkalemia K=7.8 mEq/L: beside the sinus node block, the peaked T wave is of same size of QRS with narrow base with a sharp rise from the ST.

A large T wave compared to QRS is highly suspicious of ischemia like seen in V3-V4 **(Strip IX-9).**

One last point, on a normal strip, the T wave merges with end of ST segment. If there is a net break up point or the beginning of the T wave is well demarcated then the T wave is abnormal **(Strip IX-9)**.

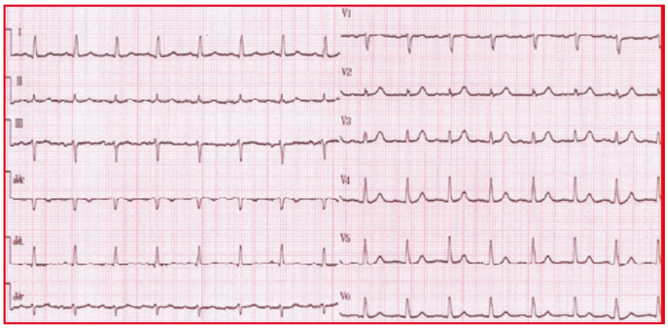
3. Q wave

It represents sequelae of a myocardial infarct. It is also progressive in nature with time and might disappear after an event. Its presence means there was a prolonged cellular anoxia. It might also means that there was no proper management within the time limit of infarct to revascularize but maybe waiting for a serial cardiac enzymes or even worse repeating a positive troponin level.

4. The fragmented QRS

With respect to ischemic and necrosis areas, there is a change in the depolarization [21]. This phenomenon indicates heterogeneity in the action potential with is translated by fragmented QRS. They are referred to as fQRS. They can be seen without a complete bundle branch block, with narrow QRS and when seen on two contiguous leads. We see an R' wave followed with a notch in S followed with R".

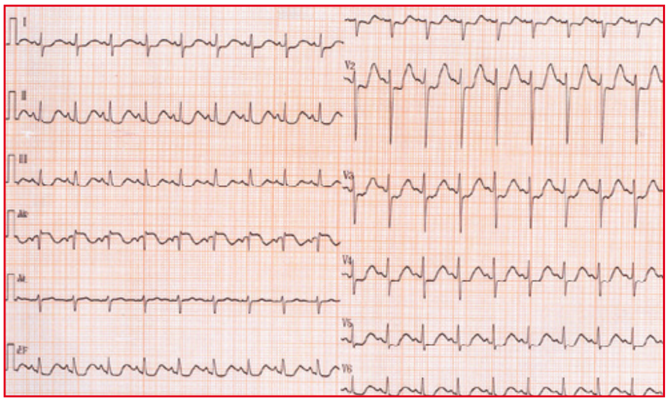
They are signs of scarred ventricle with possibilities of arrhythmias **(Strip IX-10).**



Strip IX-10. Patient with positive troponin, we notice a large peaked T wave compared to QRS in V2-V3, T wave is symmetrical and a notch in QRS (rSr's' in V1, rsr's' in V2).

5. aVR

aVR is not facing the left ventricle, the only chamber with significant muscular efficacy. When it is positive, we know that the origin of the QRS is from the ventricle. If we have an ST elevation of more than 1 mm it means we have a lesion in the left main coronary artery or proximal LAD as it is accompanied with a diffuse subendocardial ischemia. It is a serious finding because patient can have a sudden cardiac death with the occlusion of the left main artery **(Strip IX-11).**



Strip IX-11. A major subendocardial lesion seen in anterior and inferior lead, with a clear Pardee wave seen in aVR. This patient will die or go into cardiogenic shock in the next minutes. There is a subtotal occlusion of the left main coronary artery with an old occlusion in the RCA, which means he lives on a thread. The signs on aVR are less visible on strip IX.6 but are there.

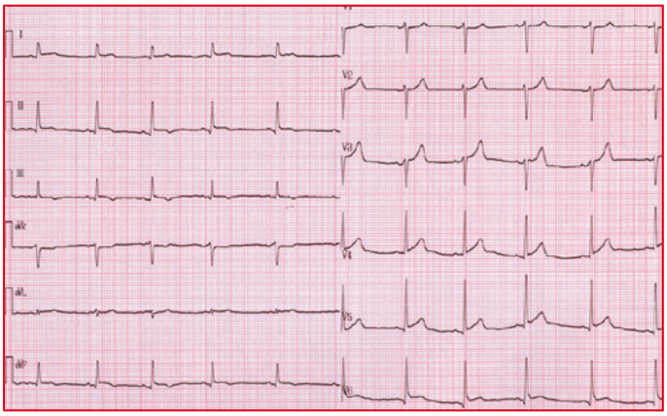
6. Differential diagnosis of ST elevation [22].

Left ventricular hypertrophy is sometimes a difficult diagnosis. There are criteria related to amplitude to diagnose LVH. The most common is sokolow (S in V1 + R in V5 larger or equal to 35 mm. a strip where the complexes overlaps is suggestive of LVH. However the anomalies in their repolarization might be suggestive of other pathologies: if there is discordance between QRS and T waves, whether negative/positive or positive /negative, this discordance is suggestive of LVH. The **strip IX-2** shows an LVH with overlapping complexes and discordance between QRS and T waves (see also **strip IX-16**).

Early repolarization is seen in young male adults, with a concave ST wave, with no mirror image and no progression.

This corresponds to the inability of the R wave descent to join the iso-electric point before beginning the ST segment.

In cases of pericarditis, there are no mirror changes; there is also PR depression that is more significant than the diffuse ST elevations especially in the inferior leads **(Strip IX-12)**.



Strip IX-12. The maximal ST elevations are seen in DII- aVF but not in DIII, maximal in DI and not in aVL. There is ST elevation with no mirror changes even though it seems territorial in the infero-lateral: this is typical found in pericarditis.

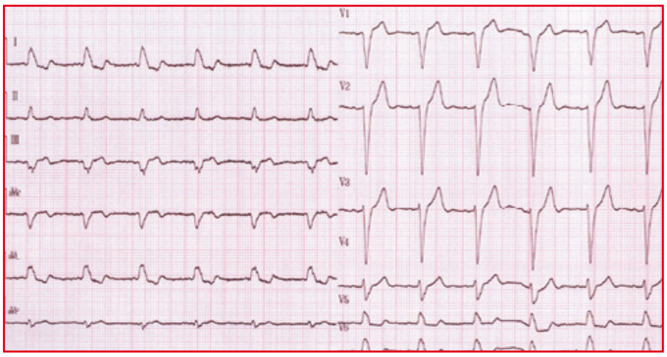
A left bundled branch block has a Pardee's sign on ST wave on a wide complex **(Strip IX-13).** But despite this block it is easy to identified an acute ischemic event on the changes seen with time **(Strips IX-14 and IX-15)**.

Several adages should be kept in mind:

• Chest pain + LBBB = recent infraction

• Chest pain + LBBB of unknown date = infarction until proven otherwise

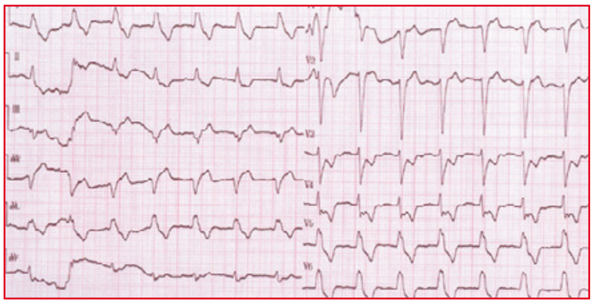
• Chest pain + former BBG old and of know date = look to the clinical context first, infarction until proven otherwise, close monitoring required.



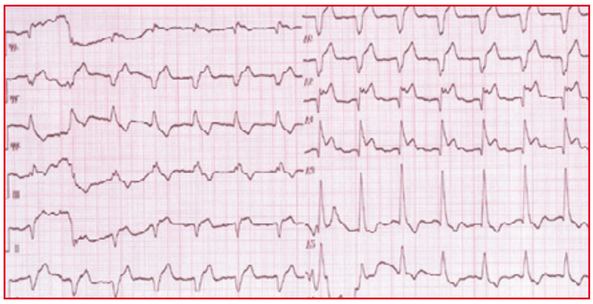
Strip IX-13. Typical left bundle branch block.

A ventricular aneurysm, sequelae of an old infarct will reveal a Pardee's wave without mirror image, stable in time.

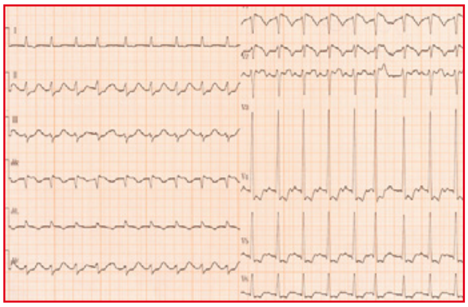
Brugada syndrome, which has a shape of Right bundle branch block with an ST elevation in V1 and V2, has a high risk for fatal arrhythmia, with the positive family history of sudden death or sudden syncope.



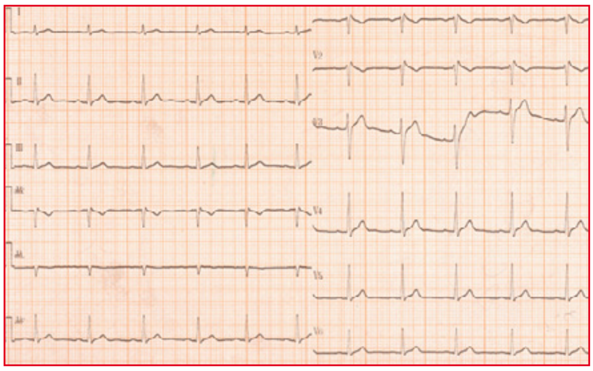
Strip IX-14. Same patient with oppressive chest pain: new anterior ischemic event.



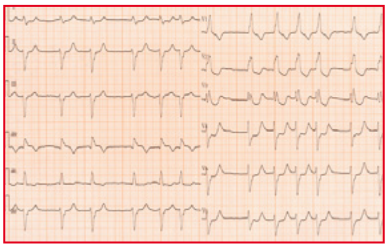
Strip IX-15. Same as strip IX-14 but seen upside down and through the other side. The Pardee's sign is clearly seen in V3-V4 despite the presence of the Left bundle Branch Block.



Strip IX-16. 23 year old patient admitted for a second episode of loss of consciousness, there is a clear LVH (amplitude with discordance of QRS/T) however there is shape of Right Bundle Branch Block with an ST elevation in V1-V2 typical of Brugada syndrome Type1.



Strip IX-17. 19 year old patient presenting with loss of consciousness preceded by palpitation and pre-syncope for over a year. The shape is less clear but highly suggestive of ST elevation in V1-V2. This is Brugada type 2.



Strip IX-18. 72 year old patient brought in for severe hypothermia 32.8° C, on digoxin due to AF with RBBB and LAHB. We clearly see the Osborn wave in V4.

7. Differential diagnosis of ST depression

Physiologic variations: we must keep in mind the ECG registers on an individual 12 different leads whose repolarization can vary in a day and those variations exist between different people. Spooning of ST segment due to digitalis effect, hypothermia or a metabolic disorder (hypokalemia) are all responsible for ST changes **(Strip IX-18)**.

The anomalies that delineate fatal arrhythmias

In current practice, two clinical scenarios can be found:

• A regular screening ECG done for a healthy individual (fitness test) **(table IX-2)**. The anomalies we should look for are Brugada Sydrome, WPW, Premature Ventricular Contractions, Arrhythmogenic Right Ventricular Dysplasia, prolonged QT **(Strip sIX-19 and IX-20)** and atrial fibrillation **(figure IX-1)**.

• The second scenario is the ECGs done in the clinical setting due to symptoms or patient complaints. In these conditions, we look for cardiomyopathies (ischemic, valvular, hypertrophies), left ventricular hypertrophies, arrhtyhmias, QRS abnormalities, etc.

In a healthy individual, we must rule out	
Syndromes	Electric signals
Brugada	RBBB with ST elevation in V1-V2
WPW	Short PR, delta wave in upstroke of QRS
Ventricular beat	Late monomorphic isolated Vs Premature polymorphic multiple, couples
ARVD	Negative T wave in Right precordial leads epsilon wave
Prolonged QT	QT ≥ 0,44
AFib	Absent P wave irregularly irregular rhythm

Table IX-2

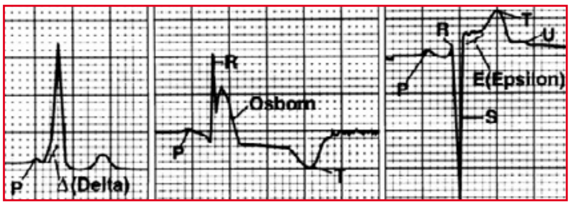
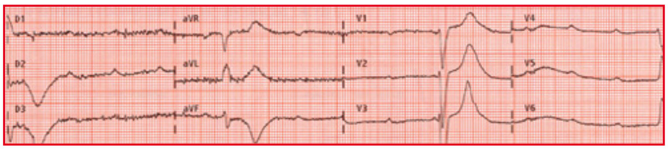
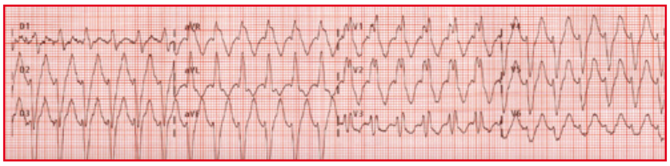


Figure IX-1. The wave to detect [23,24]
- *The delta wave: pre-excitation,*
- *Epsilon Wave: ARVD Arrhythmogenic Right Ventricular Dysplasia,*
- *Osborne wave: hypothermia.*



Strip IX-19. Patient with 3rd degree AVB on amidarone and Beta Blockers. The P waves are visible in V4, Wide QRS, QT is extremely prolonged.

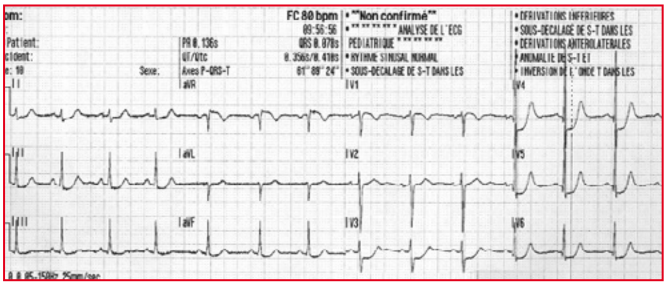


Strip IX-20. Same patient with an extra systolic ventricular contraction in the refractory period of the QRS causing a ventricular Tachycardia at 156bpm. The diagnosis of V-tach is obtained by comparing to the previous ECG. Without it, the diagnosis would have been focus the tachycardia, the frequency, and positive aVR, RBBB, LAHB and not a V-tach.

Main points to be remembered

1. The first sign of an infarct is chest pain, despite some specific contexts (diabetes, hypothyroidism, etc.) where it may be of secondary importance.
2. When in doubt, we must compare to the old ECG.
3. An ischemic ST segment changes with time, with the change in clinical symptoms.
4. An ischemic ST is maximal in III (or aVL) with its mirror image
5. An ischemic ST is always abnormal in V3 (and V2).
6. R wave must progress and S wave must regress.
7. T is proportional to the R wave which is bigger in amplitude.
8. Peaked T wave with wide base = think ischemia. Peaked T wave with narrow base = think hyperkalemia.
9. In case of left ventricular hypertrophy there is discordance between QRS complex and T wave.
10. An ECG should allow to rule out Brugada, WPW, Ventricular extrasystolic beat, ARVD a prolonged QT and asymptomatic AFib.

EXERCISE ALONG WITH THEIR INTERPRETATION ACCORDING TO "OUR GUIDE"



Interpretation according to our guide	Normal interpretation
10 year old patient	10 year old patient
Visible P wave, regular frequency QRS before and after each P	Normal sinus rhythm
Frequency 80 bpm	80 bpm
Constant PR	No 1st degree AVB
Narrow complex large in precordial	It's a 10 year old infant→ the amplitude criteria are not applied
Narrow QRS no rabbit ear in V1 or V6	No bundle branch block
ST depression in V3-V6, I, II II and aVF	Subendocardial insufficiency
Pardee wave in aVR	
T is positive except aVR and V1	No ischemia

Conclusion: normal Sinus Rhythm at 80 bpm with no conduction disorders. With non STEMI in a 10years old infant. Clinically the infant had chest pain while playing Soccer. He has a congenital anomaly of the left main coronary artery compressed by the pulmonary artery during exercise which explains his symptoms.

X- ALGORITHM

This last part is the sum up of all the previous sections, which lead to developing a unique algorithm for the simplified interpretation of an electrocardiogram based on five elements. There is necessarily an approximation, but the rule remain valid in 90-95% of the cases.

1- P wave

How to start an algorithm? What is the first sign to look for in a recording, a sign on which our interpretation will result in a diagnosis? What sign is enough by itself to deduce a number of diagnoses, and guide us in proper and divers therapeutics without the need to look for other confirmatory and complementary testing? You all know it well; it is simply the P wave **(table X-1)**.

Its presence will reassure the proper functioning of the cardiac electrophysiology, at least the initial phase it. Its absence will direct us toward an arrhythmia that most often implies an immediate therapy.

The P wave is present; could the sinus node be functioning?

• The second phrase to remember is: is every P is followed by QRS which is followed by a P wave. There are equal numbers of P waves and QRS. In this case, the rhythm is a normal sinus rhythm and we can move to the second step. We should check that the PR intervals are equal and stable (less than 0.2msec or one big square. If not we will have a 1st degree AVB),

• No QRS after each P wave: either we have more P waves than QRS with low frequency, which implies a 2nd or 3rd degree AVB, either we have more P waves than QRS but with a high frequency and we are dealing with an SVT, or we have less P waves than QRS and we have an escape rhythm,

• P wave is absent→ we move to the next step,

• We must keep in mind the patients with pacemaker where we see a spike that can help us determine the type of pacer, the level of sensing, and the level of trigger. Only the triggers located in the atrium (which mimic the normal electrophysiology) will allow to analyze the QRS complex and repolarization. A reminder: A is atrial V is ventricular D is dual chamber I is inhibitory T is triggered and D is dual sensory and trigger. The first letter is the chamber paced, the second is the chamber sensed and the third is the response to sensing.

<table>
<tr><td colspan="5">P wave present</td></tr>
<tr><td colspan="4">Yes</td><td>No</td></tr>
<tr><td colspan="4">Each is followed by QRS</td><td rowspan="6">Step 2</td></tr>
<tr><td colspan="2">Yes</td><td colspan="2">No</td></tr>
<tr><td colspan="2">Each QRS is followed by P</td><td>P>QRS</td><td>QRS>P</td></tr>
<tr><td>Yes</td><td>No</td><td>If bradycardia: 2nd or 3rd AVB;
if tachycardia: AT</td><td>Escape rhythm</td></tr>
<tr><td>NSR; 1st degree AVB</td><td rowspan="2">Step 2</td><td colspan="2" rowspan="2">Step 2</td></tr>
<tr><td>Step 2</td></tr>
</table>

Table X-1. First element: P wave.

2- Rhythm

• The analysis of the cardiac rhythm is the second most important element when reading an ECG strip. It allows us to known the ventricular response to the atrial stimuli. It can be regular or irregular **(table X-2)**.

• In the presence of a P wave, a regular rhythm where each P wave is followed by a QRS which is followed a P wave is a normal sinus rhythm,

• In the presence of a P wave, an irregular rhythm where we have each P wave is followed by a QRS is a normal sinus rhythm with premature contractions (atrial, supraventricular or ventricular),

• In the absence of a P wave, with a regular rhythm we analyze the frequency,

• In the absence of a P wave and an irregular rhythm, it is a supraventricular arrhythmia.

3- Frequency/Rate

• The rate allows us to have a precise diagnosis based on the result. The rate is essential especially in the absence of P wave. To note that the rate represents the frequency of ventricular contractions, in other words the QRS rate,

• A rate of 150 bpm will most probably imply an atrial flutter with 2:1 block. Not to forget that whether it's a narrow or wide QRS will help with diagnosis,

• A rate of more than 180 bpm with a wide QRS will tilt the origin towards a ventricular **(table X-3)**.

The 4th and 5th step are independent of the presence or absence of the P wave and with absence of pacer.

4- QRS complex

• A normal QRS complex is narrow, always negative in aVR. It can be positive in rare cares of LVH (obstructive or non-obstructive hypertrophic cardiomyopathy),
• Lead II is usually positive. A negative Lead II is due to Left Anterior HemiBlock. Lead I is usually positive. A negative Lead I is due to a Left Posterior HemiBlock,
• An M shaped V1 or rabbit's ear shape is due to Right Bundle Branch Block. A RBBB does not mean an underlying

<table>
<tr><td colspan="4">P wave present</td></tr>
<tr><td colspan="2">Yes</td><td colspan="2">No</td></tr>
<tr><td colspan="4">Rhythm</td></tr>
<tr><td>Regular</td><td>Irregular</td><td>Regular</td><td>Irregular</td></tr>
<tr><td rowspan="2">NSR</td><td rowspan="2">PAC, PVC, SVES</td><td rowspan="2">Step 3</td><td>Supraventricular arrythmia</td></tr>
<tr><td>Step 3</td></tr>
</table>

Table X-2. Second element: P wave.

cardiomyopathy until proving otherwise, however it can be seen in myopathies of the right ventricle,
• A wide complex W shaped or inverted M wave in V6 means we have a left bundle branch block. A new LBBB is always pathological,
• A Q wave is always abnormal until proven otherwise,
• Depending on the clinical presentation, on every strip we must always rule out the possibility of WPW (Wolff-Parkinson-White) with the presence of delta wave, Brugada syndrome (RBBB shape with ST elevation in V1-V2),
• A QRS that change axis between 2 consecutive complexes is an extra systole until proven otherwise. The severity of a PVC depends on its duration, polymorphism, coupling and frequency.

5- T wave, ST segment and QT interval

• A normal T wave is by definition always positive, proportional to the QRS size that precedes it. It is normally negative in aVR and V1,
• A negative T wave means an epicardial ischemia whether the ischemia it is clinically seen or not,
• We must always analyze the ST segment which should be isoelectric. An elevation or a depression are signs of active disease,
• Finally we should be able to:
  - Measure QT interval which should be normally less than 0.4sec,
  - Eliminate RV arrhythmogenic dysplasia.

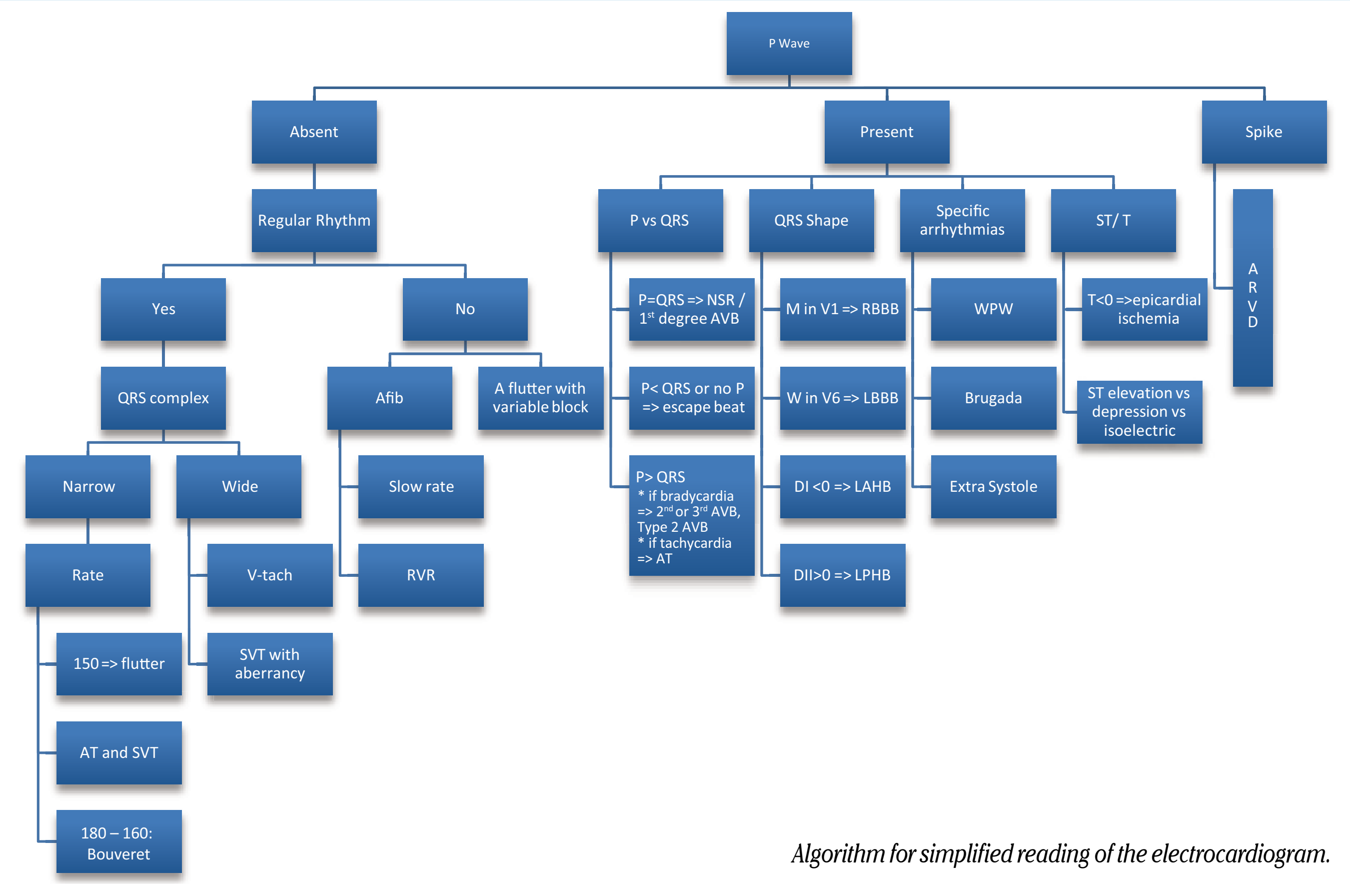
P wave present				
Yes	No			
Calculate the rate/frequency				
Step 4	QRS complex			
	Narrow		Wide	
	Regular	Irregular	Regular	Irregular
	150= Atrial Flutter	AF SR-AF AF with RVR Flutter with variable conduction	VT SVT with BBB	SVT with BBB
	160-180= SVT		Step 4	
	ST			
	Step 4			

Table X-3. Third element: frequency.

Main points to be remembered

Answers we look for in a 12- lead ECG will allow us to know a normal one and detect any abnormality. The questions are:
- Is the P wave Present?
- Is there a QRS after each P wave and each QRS is followed by a P wave?
- Is the rhythm regular?
- What is the rate/frequency?
- What is the shape of the QRS?
- Is the T wave positive? (except V1 and aVR)

The following algorithm will sum up these questions and the previous tables.



Algorithm for simplified reading of the electrocardiogram.

Abbreviations used	
A fib	Atrial fibrillation
A flutter	Atrial flutter
A fib with RVR	Atrial fibrillation with rapid ventricular rhythm
AVB	Atrio ventricular block
RBBB	Right bundle branch block
LBBB	Left Bundle branch block
ARVD	Arrhthmogenic Right ventricular dysplasia
APC	Atrial premature contractions
PVC	Premature Ventricular contraction
LAHB	Left anterior HemiBlock
LPHB	Left Posterior Hemi Block
NSR	Normal Sinus Rhythm
WPW	Wolff Parkinson White
VT	Ventricular Tachycardia

Correspondant author's note

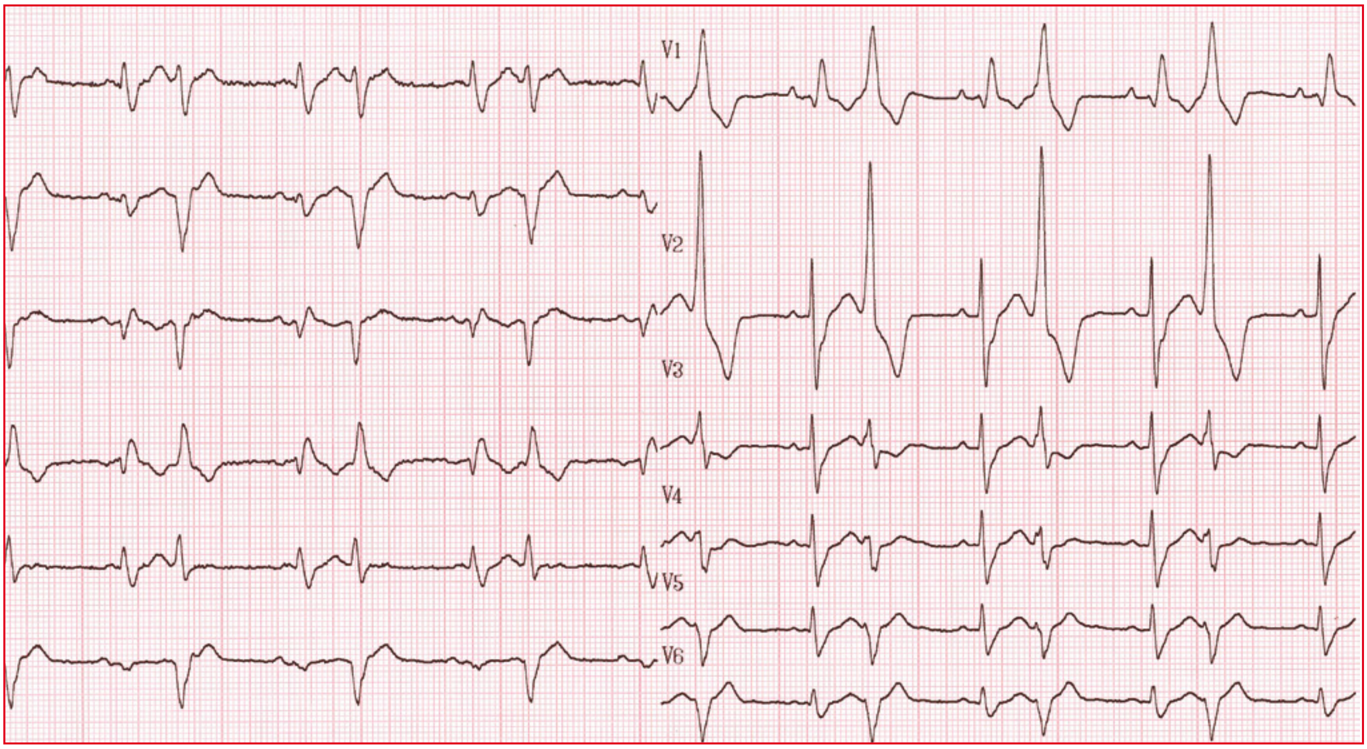
It's with this algorithm that we end a series of 10 sections for a simple interpretation of any electrocardiogram. And this is the right moment to apologize for the professors of ECG who might consider this simplification is a desecration but this is not the case. The simple aim is to provide any person who might finds him/herself in an emergency situation and must interpret an ECG, and not to miss serious life threatening condition for the sake of the patient. And we would like to thank all those who taught me the cardiology material over the years with passion, dedication and devotion without any negligence to patient's care.

I would like to dedicate a special Thank you to Michel Hanssen, a master among teachers and a dear friend. In memories Bertrand Mettauer (1953-2014).

Editor's acknowledgment note

I would like to thank the authors for this audacious work and my colleagues, Karim Farah, Sara Moufarrij and Lina Deghayli, for their strong support. Last but not least special thanks to Ziad Khoueiry, Hugues Lefort, Maria Frangieh and Mireille Srour for their tremendous work.

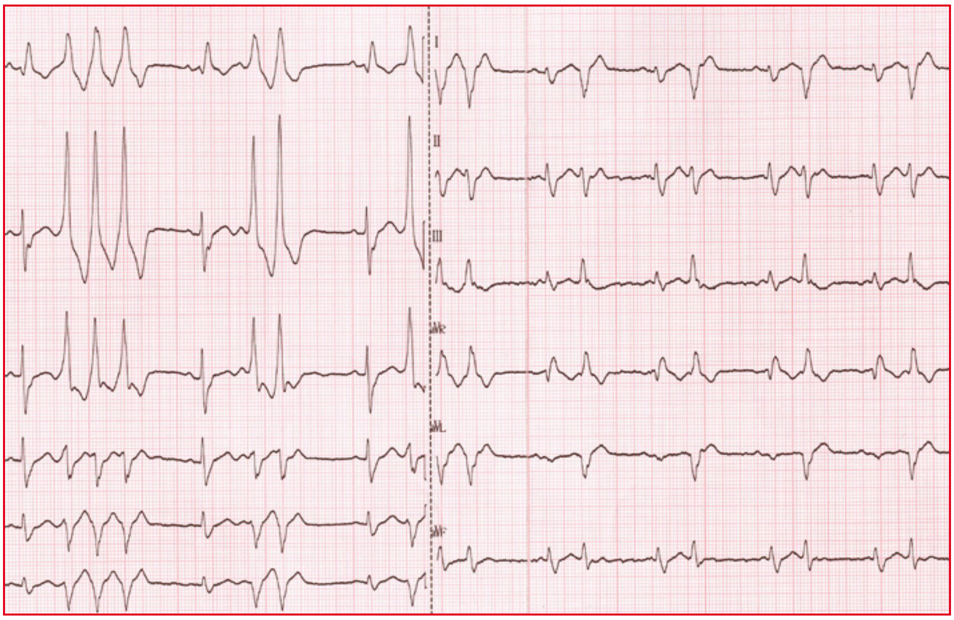
SOME ECGs FOR PRACTICE



ECG 1

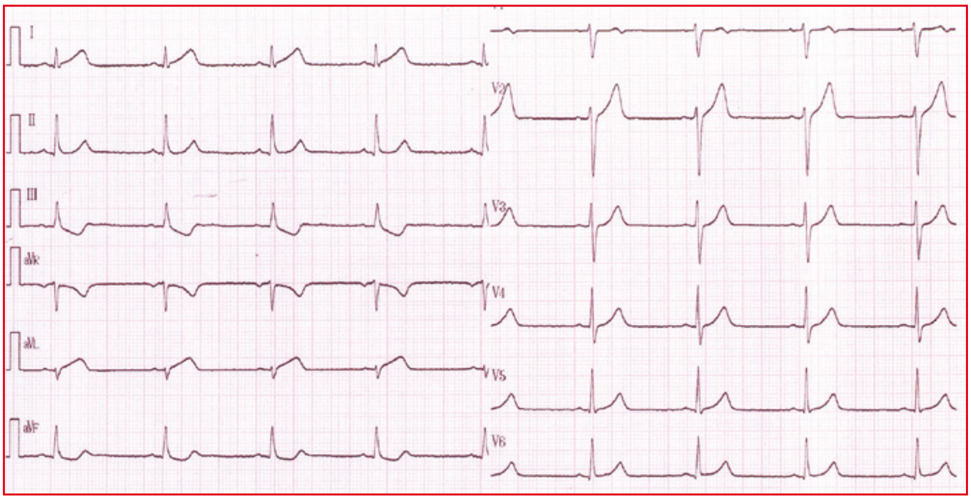
Interpretation according to our guide	Normal interpretation
P wave seen, regular rate with a QRS following every Q wave however no P after each PRS	Sinus rhythm
PR interval is normal	No Ist degree AVB
Narrow complex but 2 consecutives QRS with different morphology	Two bigeminies
AVR is positive	Wrong placement of leads

Conclusion: Sinus rhythm with bigeminies and misplacement of leads.
No further interpretation before repeating the EKG with proper lead positioning.



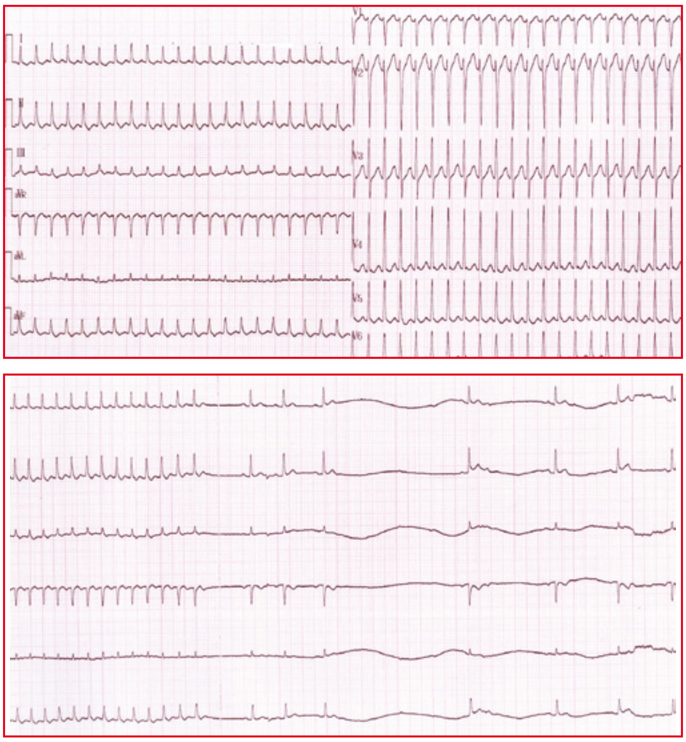
ECG 2 Same patient with repositioning of the electrodes

Interpretation according to our guide	Normal interpretation
P wave seen, regular rate with a QRS following every Q wave however no P after each PRS	Sinus rhythm
PR interval is constant	No 1st degree AVB
Narrow complex but 2 consecutives QRS with different morphology	Two bigeminies
After each P wave the QRS are narrow with negative lead II. No M or W in V1 or V6	LAHB
The extra systoles are monomorphic, sometimes couplets or triplets	Runs of V tach
In V4 there is a small Q wave with T inversion	Isolated apical old infarcts
Conclusion: Sinus rhythm with bigemines (couplets or triplets) with an old ischemia.	



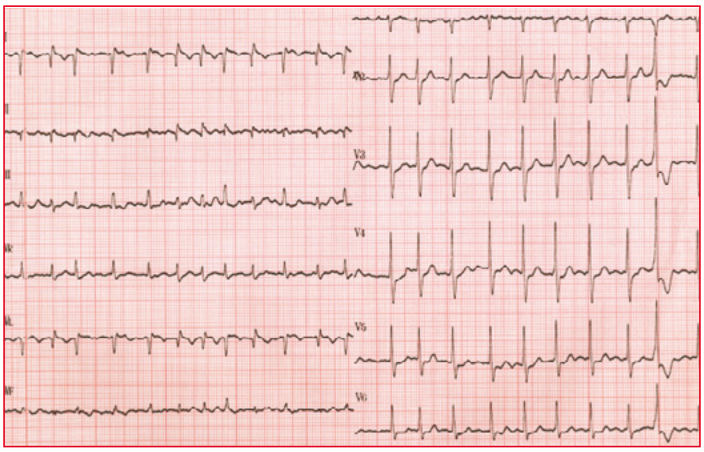
ECG 3

Interpretation according to our guide	Normal interpretation
P wave seen, regular rate, around 54 bpm, narrow QRS	NSR with a rate 54bpm
I and II are positive	No LAHB no LPHB
No M or W in V1 or V6	No bundle branch block
ST elevation in I and aVL and ST depression in III and aVF	Lateral Myocardial infarct with reciprocal changes in inferior leads
Conclusion: NSR with a rate of 54 bpm with acute lateral infarct and reciprocal inferior changes.	



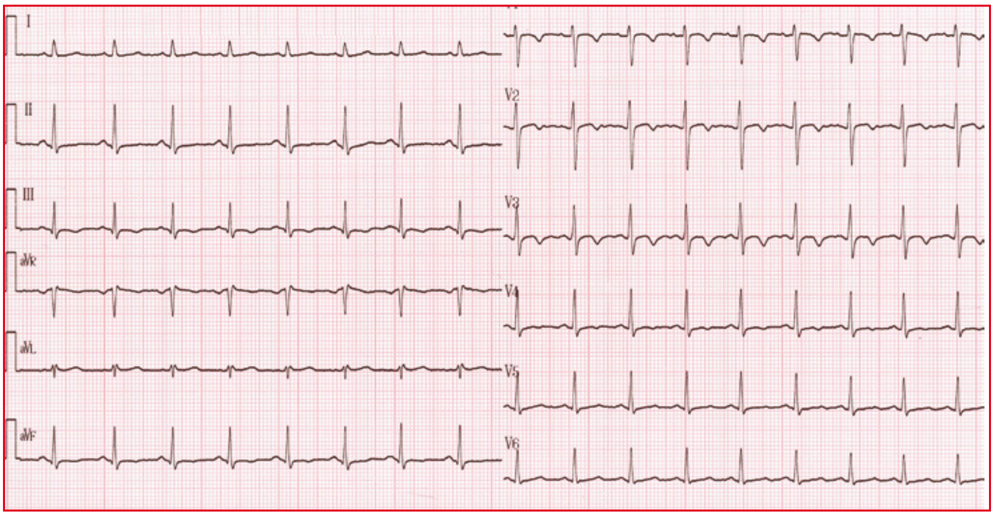
ECG 4 and 4'

Interpretation according to our guide	Normal interpretation
P wave not seen. Irregular rate rapid rate with narrow QRS >280bpm	SVT
I and II are positive	No LAHB or LPHB
No ST changes	No lesion
T waves are positive	No ischemia
On the 2nd strip, rhythm is fast then there is a pause and we see a P wave followed by a QRS of same morphology after 3 complexes then we see a non conducted P wave then P followed with QRS then a non conducted P wave then normal sinus rhythm	Injection of Adenosine that blocks the AVN followed by NSR
Conclusion: Narrow complex junction tachycardia, regular SVT reverted by adenosine with return to NSR.	



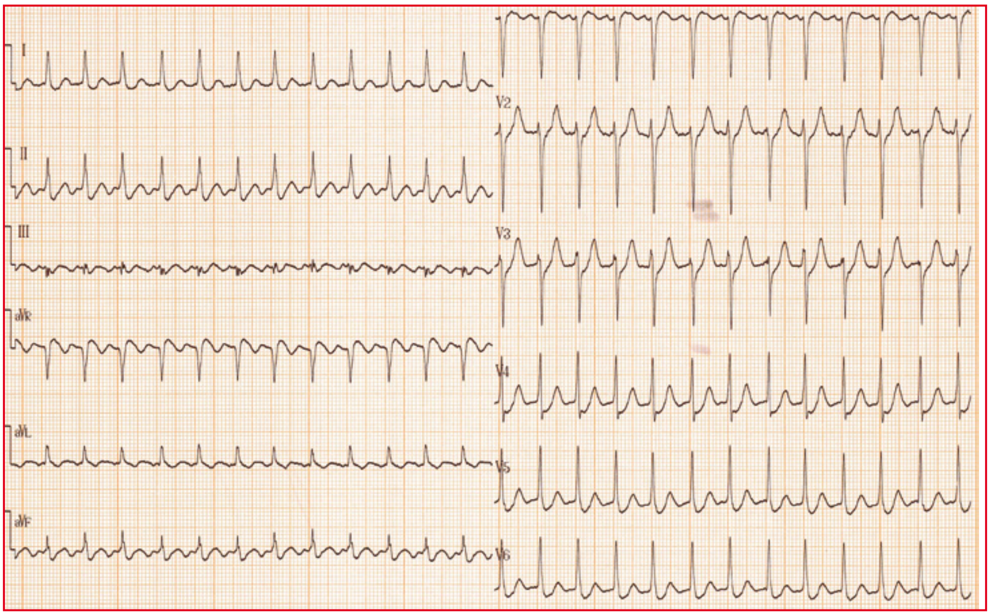
ECG 5

Interpretation according to our guide	Normal interpretation
Absent P wave, irregular rhythm, narrow complex, rapid rate 140 bpm	A fib with RVR
aVR is positive	Error in electrode placement
No ST changes	No acute infact
T waves are positive	No ischemia
1 PVC	PVC
Conclusion: AF with RVR no acute infarct or ischemia. Limb leads need to be readjuste.	



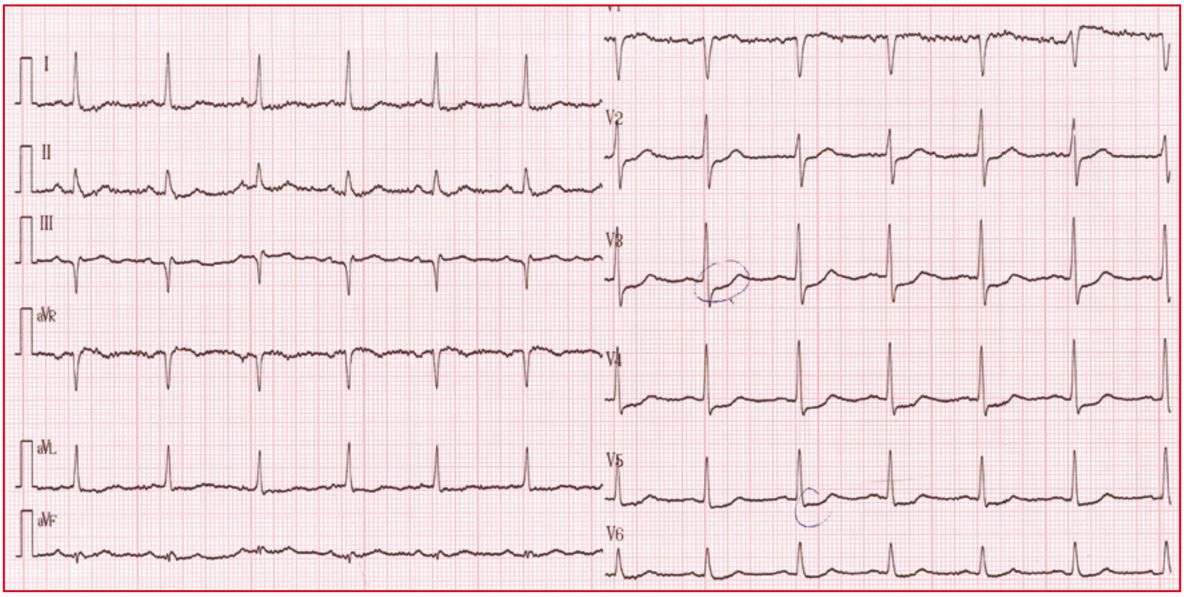
ECG 6

Interpretation according to our guide	Normal interpretation
P wave seen, regular rate at 110 bpm	Sinus tachycardia
I and II are positive	No LAHB no LPHB
No ST changes	No ischemia or infarct
T wave are negative V1to V3, V4	Anteroseptal ischemia
Conclusion: Sinus tachycardia at 110 bpm with no conduction problem with anteroseptal ischemia. The first diagnosis to rule out is a pulmonary embolus, then a coronary ischemia.	



ECG 7

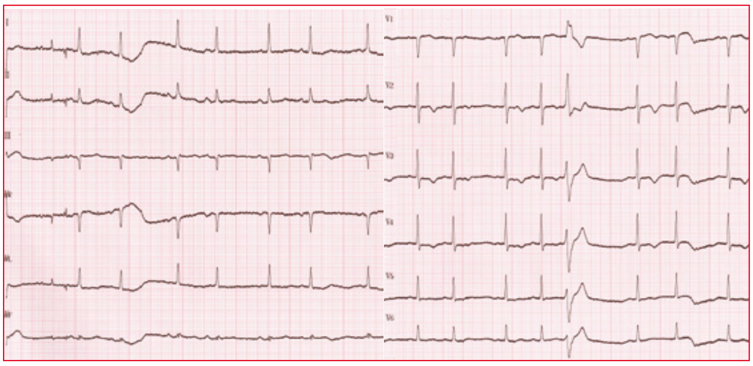
Interpretation according to our guide	Normal interpretation
P wave not seen, regular rate, narrow complex 150 bpm	Narrow complex supraventricular tachycardia
I and II are positive	No LAHB or LPHB
ST depression in V4 V5 V6	Lateral ischemia
T waves are positive	No old ischemia
Sine wave	Atrial flutter
Conclusion: Atrial flutter with 2:1 block with lateral ischemia due to tachycardia.	



ECG 8

Interpretation according to our guide	Normal interpretation
P wave seen, regular rate 75 bpm narrow QRS	Sinus rhythm
I and II are positive	No LAHB no LPHB
ST depression in V3-V6	Anterior acute ischemia
T waves are positive	No old ischemia
Conclusion: NSR 75 bpm with an acute anterior ischemia Non STEMI.	

Same patient 48hours later



Interpretation according to our guide	Normal interpretation
P wave seen, irregular, narrow QRS complex, QRS have same shape except for one.	NSR with APC, PVC
I and II are positive	No LAHB or LPHB
No ST changes	No acute ischemia
T waves are negative V2-V4	Epicaridal ischemia
Conclusion: Sinus rhythm with APC, PVC with a sequelea of an apical non transmural infarct.	

REFERENCES

1- Kirchhof CJ, Allessie MA. Sinus node automaticity during atrial fibrillation in isolated rabbit hearts. Circulation 1992; 86:263-71.

2- Kligfield P, Gettes L, Bailey JJ. AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram and its technology. J Am Coll Cardol 2007; 49:1109-27.

3- Peter M. P-wave morphology in focal atrial tachycardia. J Am Coll Cardiol 2006; 48:1010-7.

4- James AF, Choisy SC, Hancox JC. Recent advances in understanding sex differences in cardiac repolarization. Prog Biophys Mol Biol 2007; 94:265-319.

5- Franz MR. Current status of monophasic action potential recording: theories, measurements and interpretations. Cardiovasc Res 1999; 41:25-40.

6- Arntz HR, Willich SN, Schreiber C, Brüggemann T, Stern R, Schultheiss HP. Diurnal, weekly and seasonal variation of sudden death. Population-based analysis of 24,061 consecutive cases. Eur Heart J 2000; 21:315–20.

7- Goy JJ, Stauffer JC, Schlaepfer J, Christeler P. Myocardial Ischemia, Myocardial Infarction. In Bentham e-Books 144 pages. Electrocardiography (ECG) 2013; 100-5.

8- Escande D, Le Marec H. long QT congenital syndrom. http://www.ifr26.nantes.inserm.fr/u533/people/team1a/qtlong/qtlong/frameset.htm. INSERM – France 2000

9- Zema MJ. Q wave, S-T segment, and T wave myocardial infarction. Useful clinical distinction. Am J Med 1985; 78:391-8.
10- Goy JJ, Stauffer JC, Schlaepfer J, Christeler P. Arrhythmias and Tachycardias. In Bentham e-Books 144 pages. Electrocardiography (ECG) 2013; 53-99.
11- Passman R, Kadish A. Polymorphic ventricular tachycardia, long Q-T syndrome, and torsades de pointes. Med Clin North Am 2001; 85:321-41.
12- Goy JJ, Stauffer JC, Schlaepfer J, Christeler P. Conduction abnormalities. In Bentham e-Books 144 pages. Electrocardiography (ECG); 2013: 27-52.
13- National institute for health and excellence. NICE 2014. Appendix D: Pacemaker nomenclature. http://www.evidence.nhs.uk/search%3Fq%3DBPEG%2520recommendations%2520for%2520pacemaker .
14- Parham WA, Mehdirad AA, Biermann KM, Fredman CS. Hyperkaliemia revisited. Tex Heart Inst J 2006; 33:40-7.
15- Goy JJ, Stauffer JC, Schlaepfer J, Christeler P. Electrolyte Disturbances and QT Interval Abnormalities. In Bentham e-Books 144 pages. Electrocardiography (ECG), 2013, 133-141
16- Liu K, Yang T, Viswanathan PC, Roden DM. New Mechanism Contributing to Drug-Induced Arrhythmia: rescue of a misprocessed LQT3 mutant. Circulation 2005; 112: 3239-46.
17- Papadakis M, Basavarajaiah S, Rawlins J, Edwards C, Makan J, Firoozi S et al. Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. Eur Heart J 2009; 30:1728-35.
18- Okin PM. Electrocardiography in Women: Taking the Initiative. Circulation (AHA) 2006; 113:464-6.
19- Weiford BC, Subbarao VD, Mulhern KM. Noncompaction of the ventricular myocardium. Circulation 2004; 109:2965-71.
20- Hamm CW, Bassand JP, Agewall S, Bax J, Boersma E, Bueno H et al. ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2011; 32:2999–3054.
21- Das MK, Khan B, Jacob S, Kumar A, Mahenthiran J. Significance of a fragmented QRS complex versus a Q wave in patients with coronary artery disease. Circulation 2006; 113:2495-501.
22- Goy JJ. Stauffer JC. Schlaepfer J. Christeler P. Differential Diagnosis of Cardiac Ischemia. In Bentham e-Books 144 pages. Electrocardiography (ECG) 2013; 116-23.
23- Hurst JW. Naming of the waves in the ECG, with a brief account of their genesis. Circulation 1998; 98:1937-42.
24- Segers PM, Lequime J, Denolin H. L'activation ventriculaire précoce de certains coeurs hyperexcitables: étude de l'onde Δ de l'électrocardiogramme. Cardiologia 1944; 8:113–67.

FURTHER READING (non listed sources)

25- Park MK, Guntheroth WG. How to read pediatric ECGs, ed 4, Philadelphia, Saunders, 2006, p 17.
26- Huszar R. Basic dysrhythmias: interpretation and management, ed 3, St Louis, 2007, Mosby.
27- Aehlert B. ECGs made easy, ed 3, St Louis, 2006, Mosby.
28- Miller RD, Eriksson LI, Fleisher LA, et al. Miller's anesthesia, 7th ed, New York, 2010, Churchill Livingstone.
29- Goldberger AL. Clinical electrocardiography: a simplified approach, ed 7, St Louis, 2007, Mosby.
30- Marx JA, Hockberger RS, Walls RM, et al. Rosen's emergency medicine, ed 7, St Louis, 2010, Mosby.
31- Goldman L, Ausiello D. Cecil medicine, 23rd ed, Philadelphia, 2008, Saunders.
32- Dubin D. Lecture accélérée de l'ECG. Ed Maloine, 6th edition, 2007.
33- AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram. Publication of 6 parts in the JACC. J Am Coll Cardiol; 2007 - 2009.
34- Dr. Smith's ECG blog. http://hqmeded-ecg.blogspot.com/ Hqmeded-ecg.blogspot.fr
35- Goldberger AL, Goldberger ZE, Shvilkin A. Goldberger's Clinical Electrocardiography: a Simplified Approach, 8th Ed, Elsevier, Philadelphia 2012.
36- Wang K, Asinger R, Marriott H.. - ST-Segment Elevation in Conditions Other Than Acute Myocardial Infarction. NEJM 2003; 349:2128-35.
37- Shlomo S. Electrocardiogram: Still the Cardiologist's Best Friend. Circulation 2006; 113: e753-6.
38- Ashley EA, Raxwal VK, Froelicher VF. The prevalence and prognostic significance of electrocardiographic abnormalities. Curr Probl Cardiol 2000; 25:1–72.
39- Hampton JR. ECG Made Easy 3rd ed. London: Churchill Livingstone, 1997.
40- Rautaharju PM. A hundred years of progress in electrocardiography. 1: Early contributions from Waller to Wilson. Can J Cardiol 1987; 3:362-74.
41- Klabunde Richard E. Cardiovascular physiology concepts, 2nd edition. Textbook published by Lippincott Williams & Wilkins (2011).
42- Keating L, Morris FP, Brady WJ. Electrocardiographic features of Wolff-Parkinson-White syndrome. Emerg Med J 2003; 20:491-3.

Note of the editor: the authors declared that all strips, figures, tables and graphs are issued from their daily practices except two of them: one on page 18, section IV [8] and the second one on page 46 section IX [23,24].

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