מכבי שירותי בריאות

סניף כרמיאל

Signature:_____

ID: 314195512

Last Name: כונין

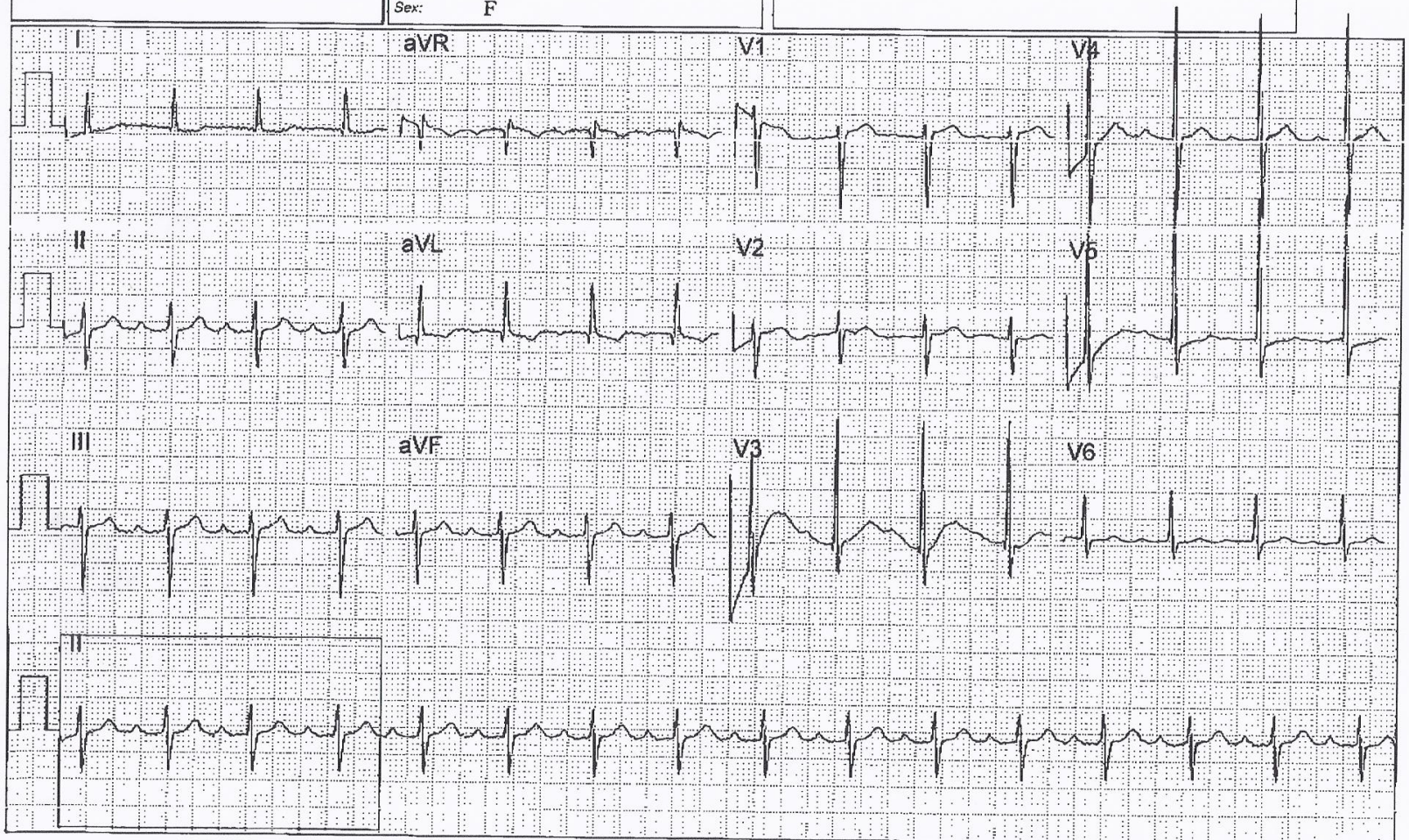
First Name: גניה

Birth Date: 12/03/1906 Age: 105

Sex: F

Physician's Notes:
Left anterior hemiblock
Inverted T wave in aVL
ST depression,lateral leads

HR: 94



25 mm/sec 10 mm/mV Filters: 50 Hz -on; BL -on; 0.05-35 Hz

October 31, 2011 10:53

Norav Medical Ltd. rev. 4. 6. 1

THE THREE ACUTE ISCHEMIC PHASES

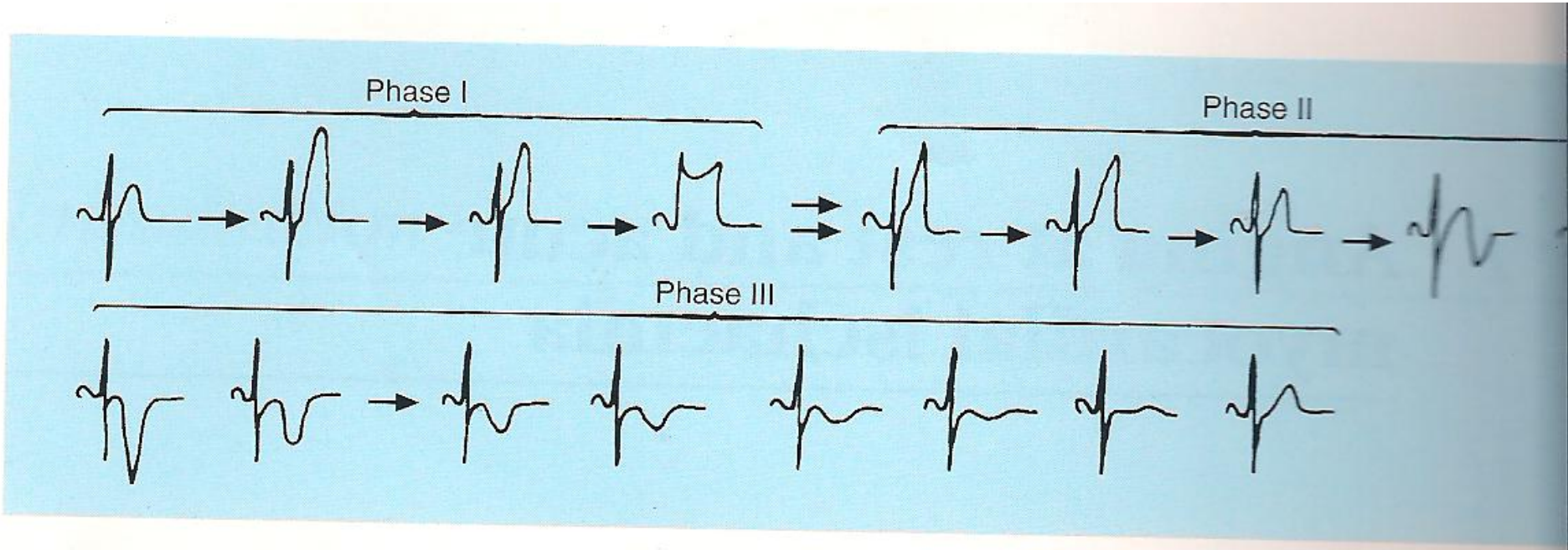
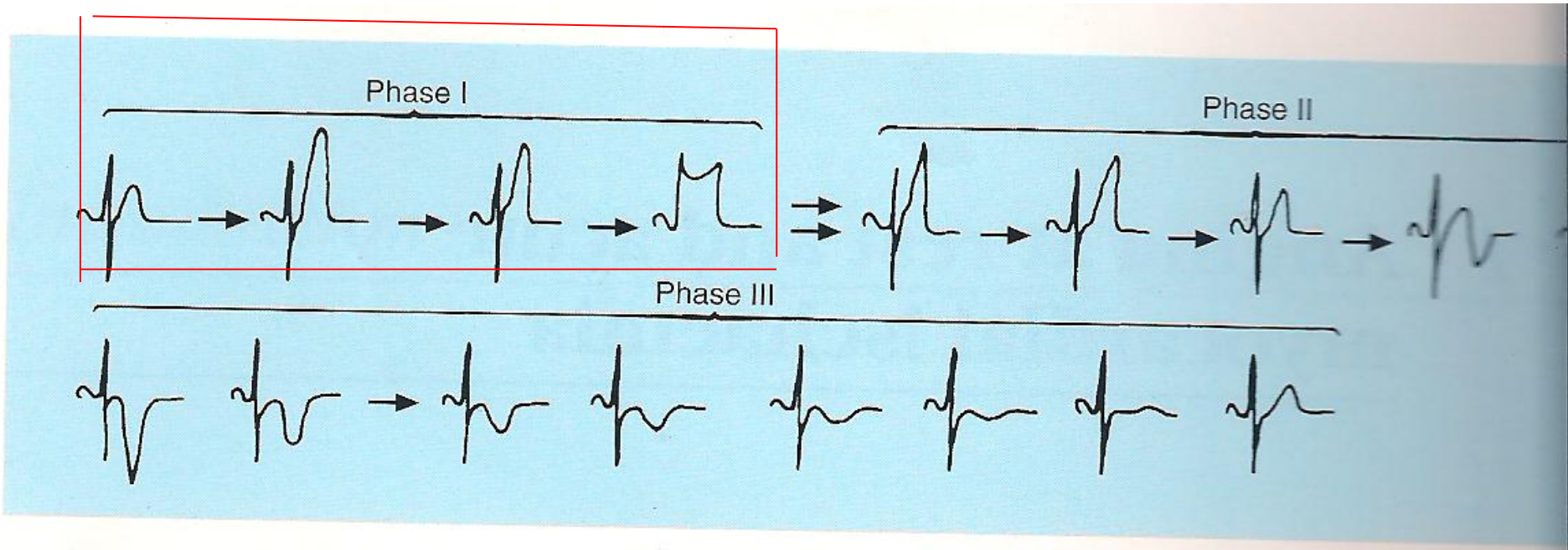


Figure 1.1 *A depiction of the ECG patterns in the evolution of acute myocardial ischaemia.*



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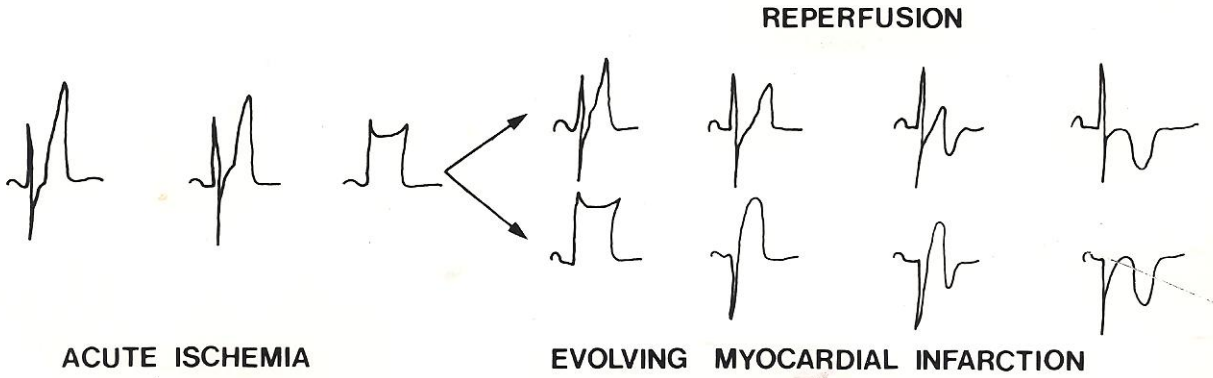
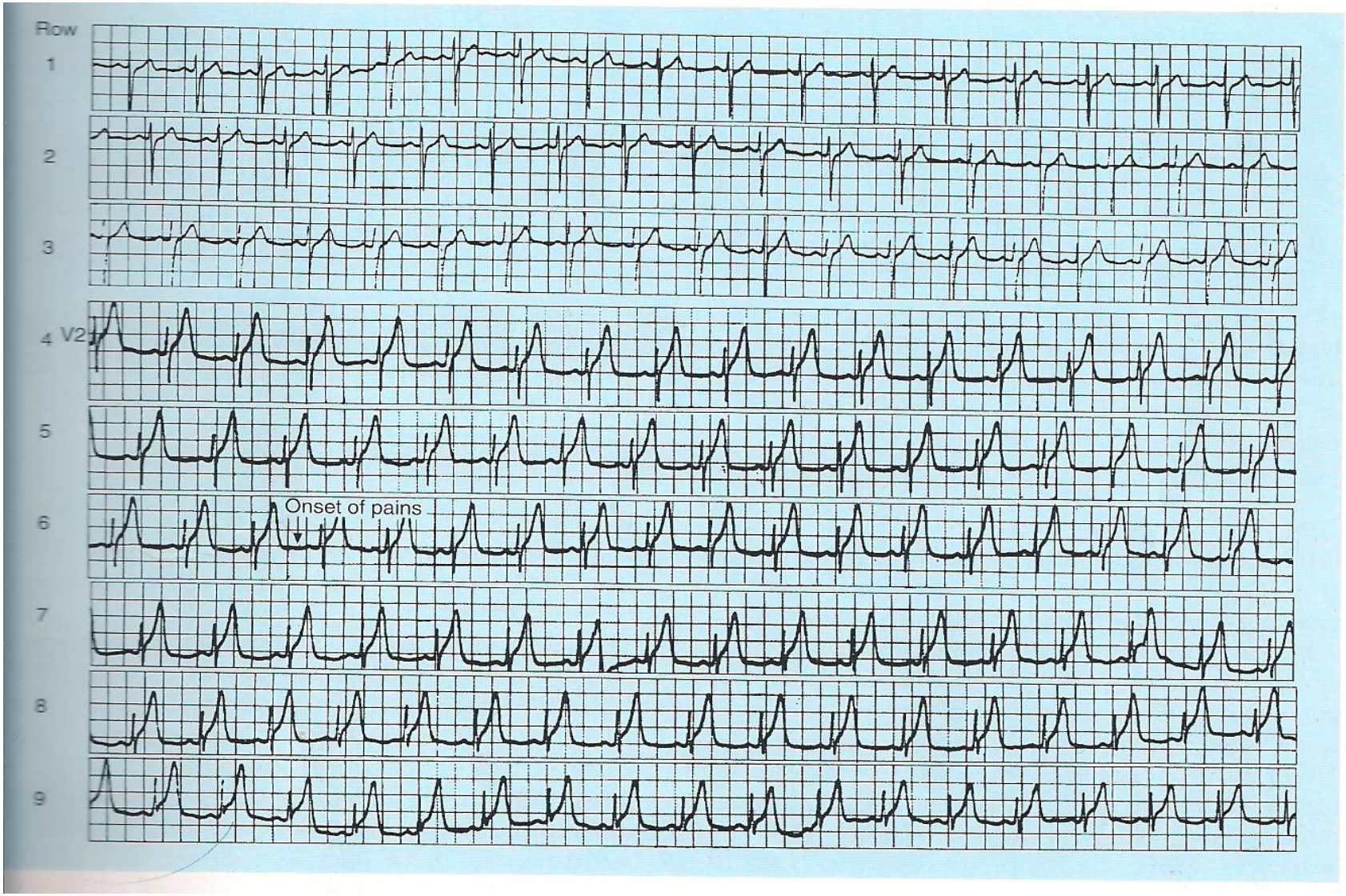

REPERFUSION
ACUTE ISCHEMIA
EVOLVING MYOCARDIAL INFARCTION


Row
1
2
3
4 V2
5
6
Onset of pains
7
8
9

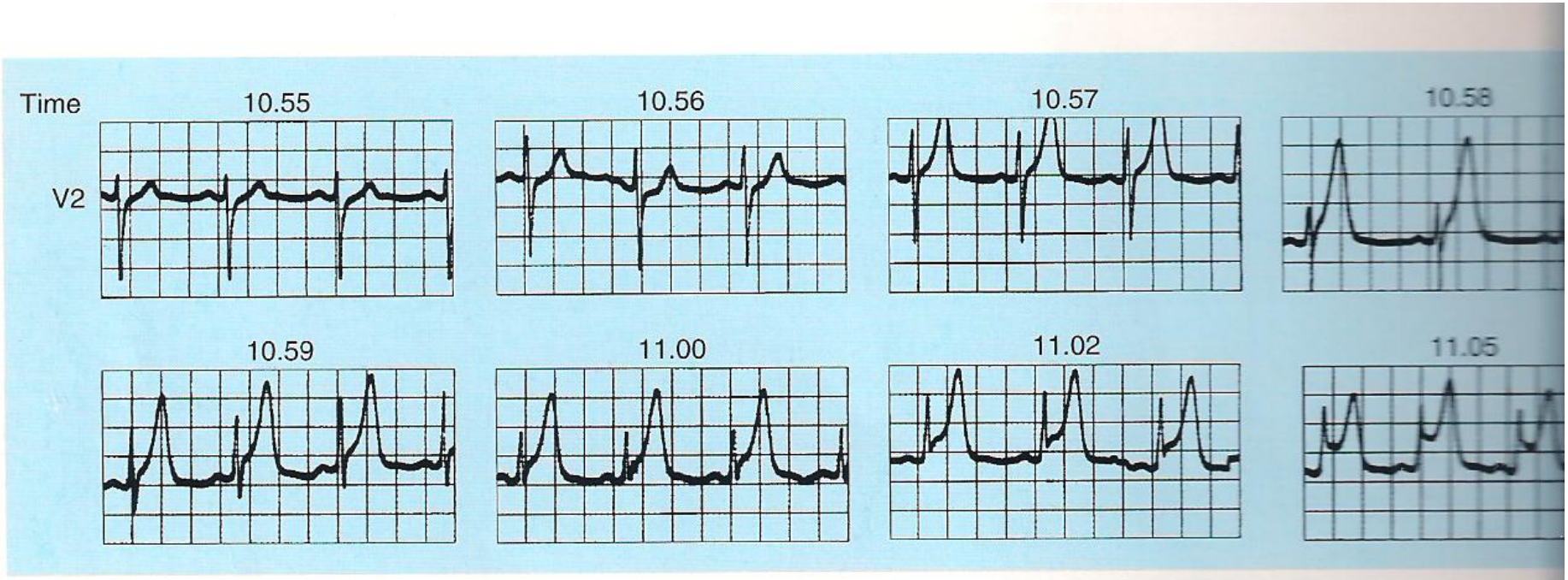
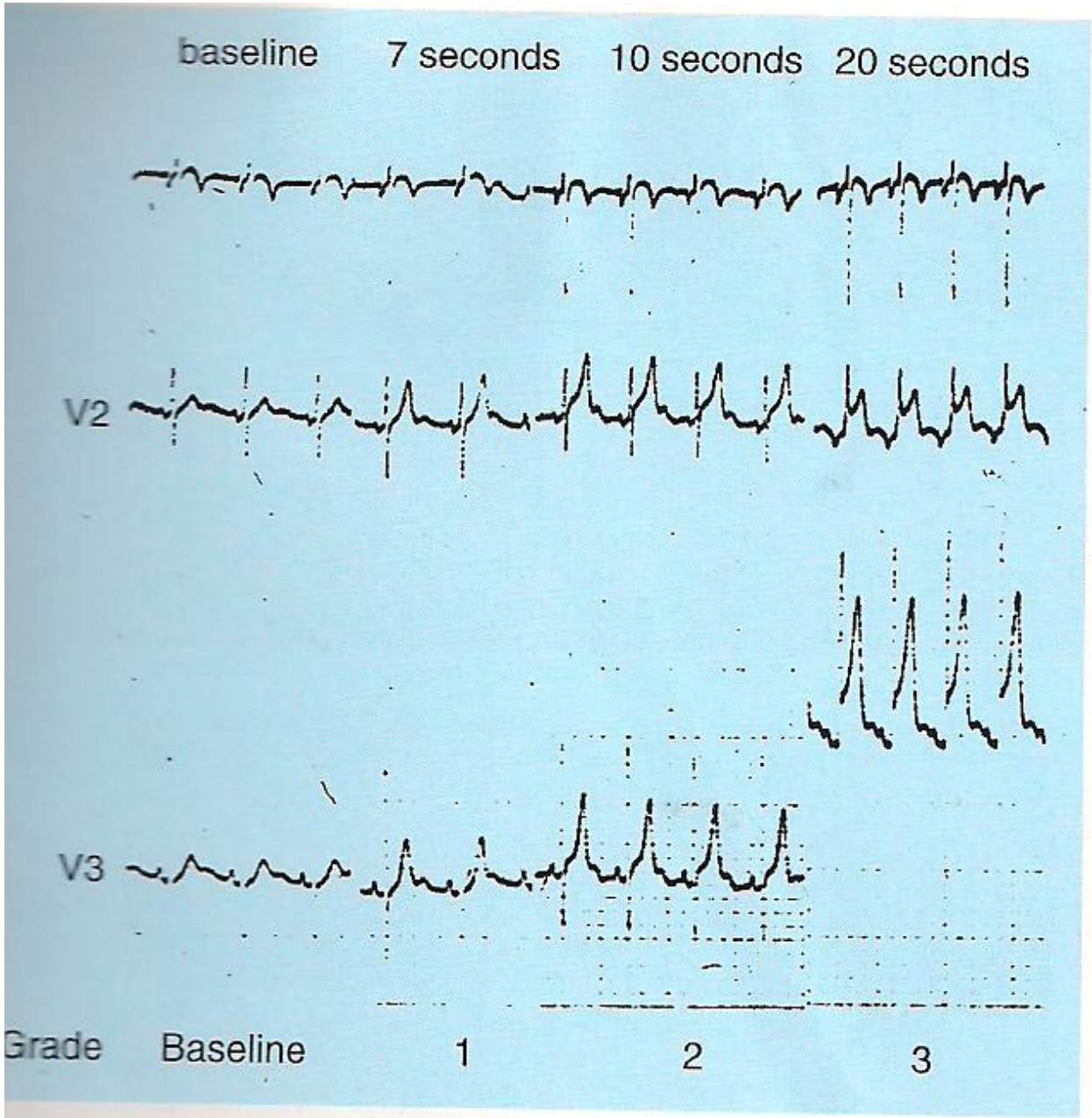


Figure 1.5 *Ten minutes of continuous ECG recording in lead V2 in a patient with regional transmural ischaemia. T wave becomes more prominent over time until 10.57 (ischaemia Grade 1). Thereafter, the ST segment begins to rise (*


baseline
7 seconds
10 seconds
20 seconds
V2
V3
Grade
Baseline
1
2
3

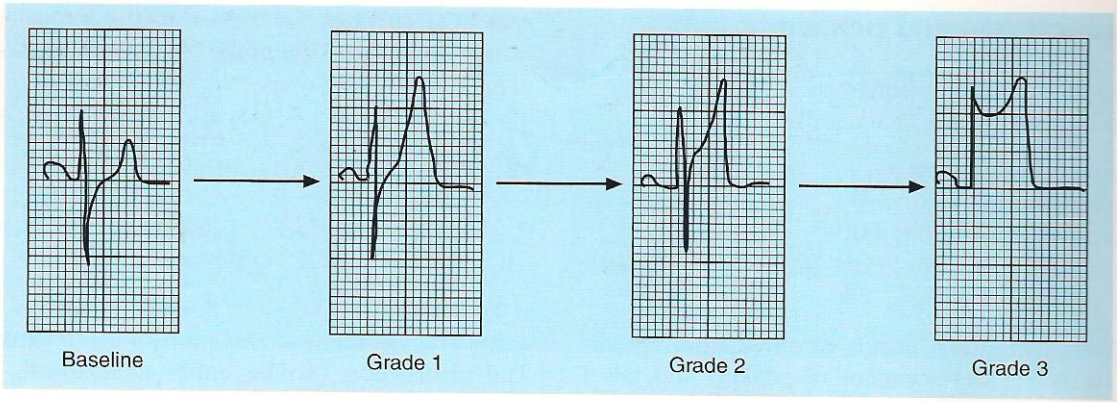
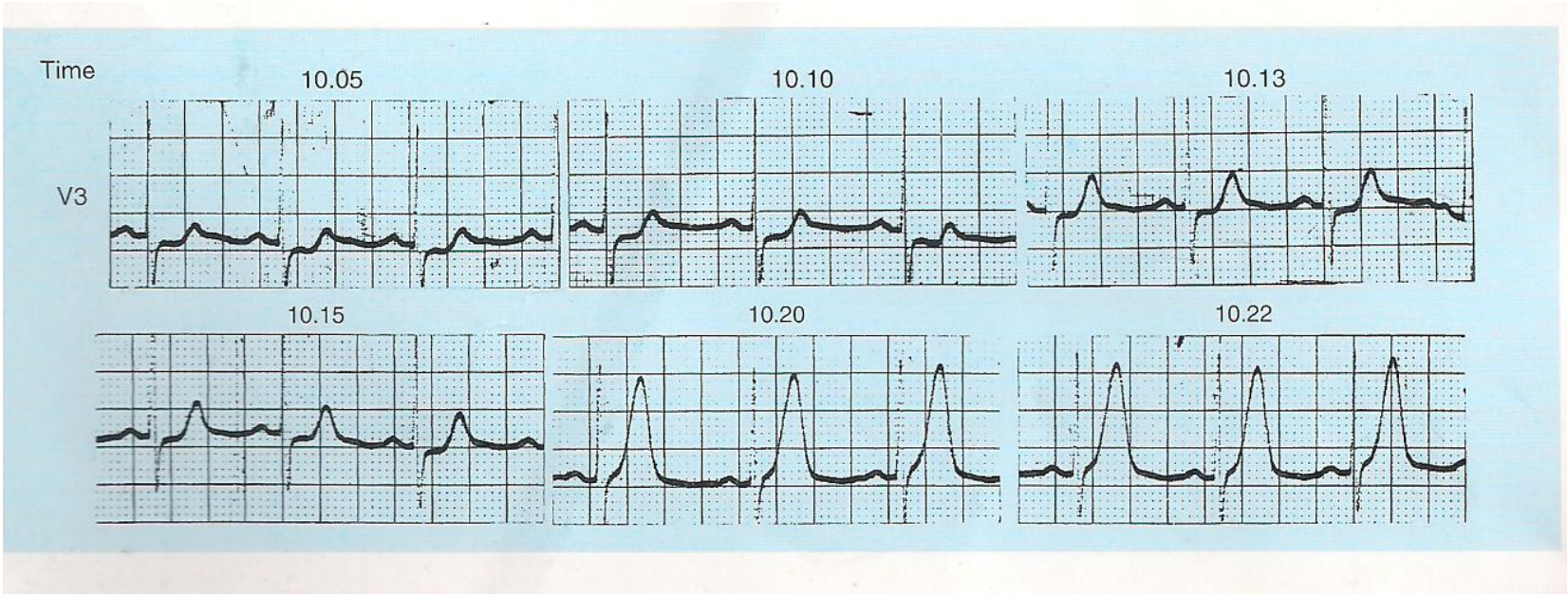


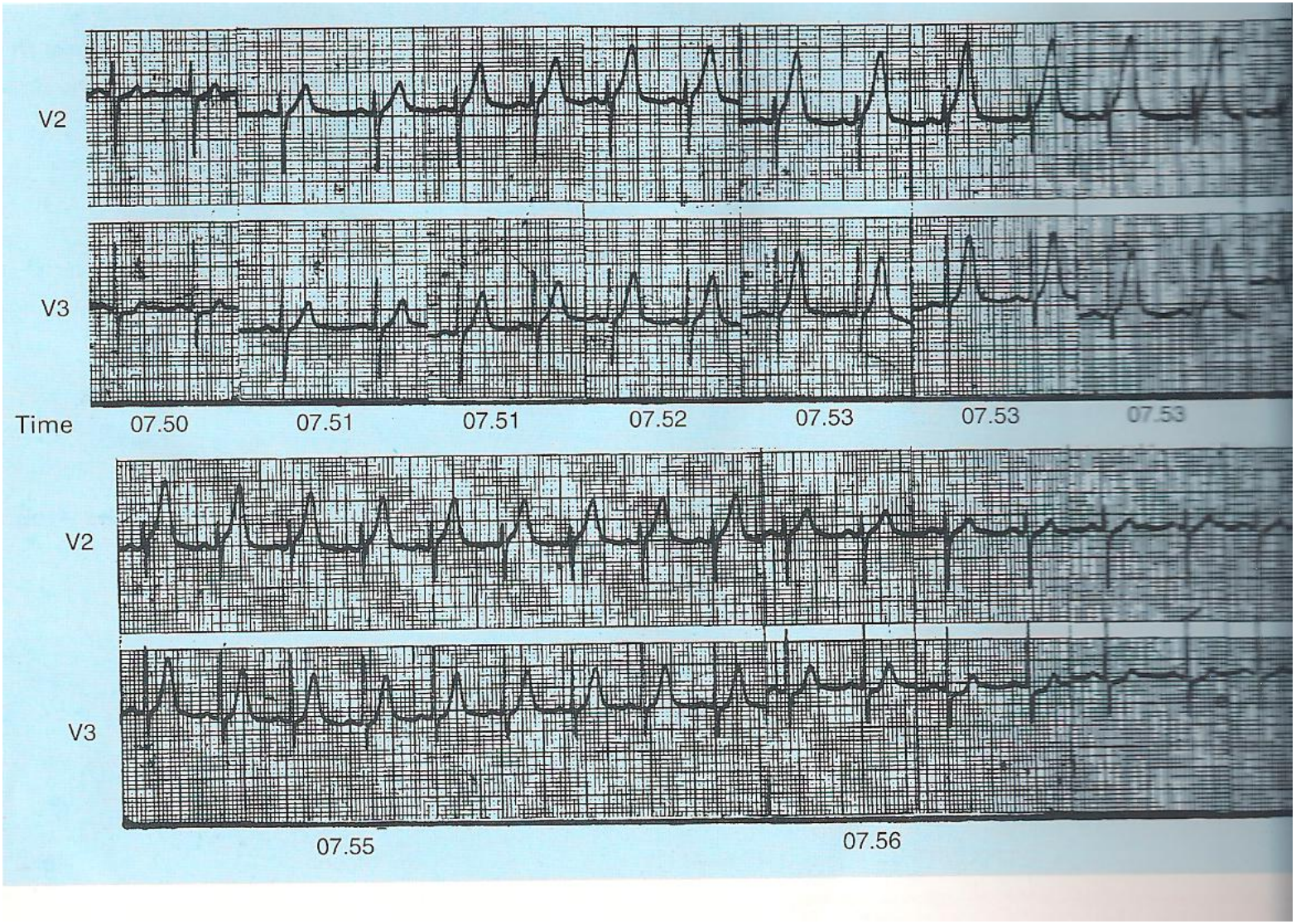
Figure 1.4 *The different ECG grades of ischaemia during regional transmural ischaemia.*

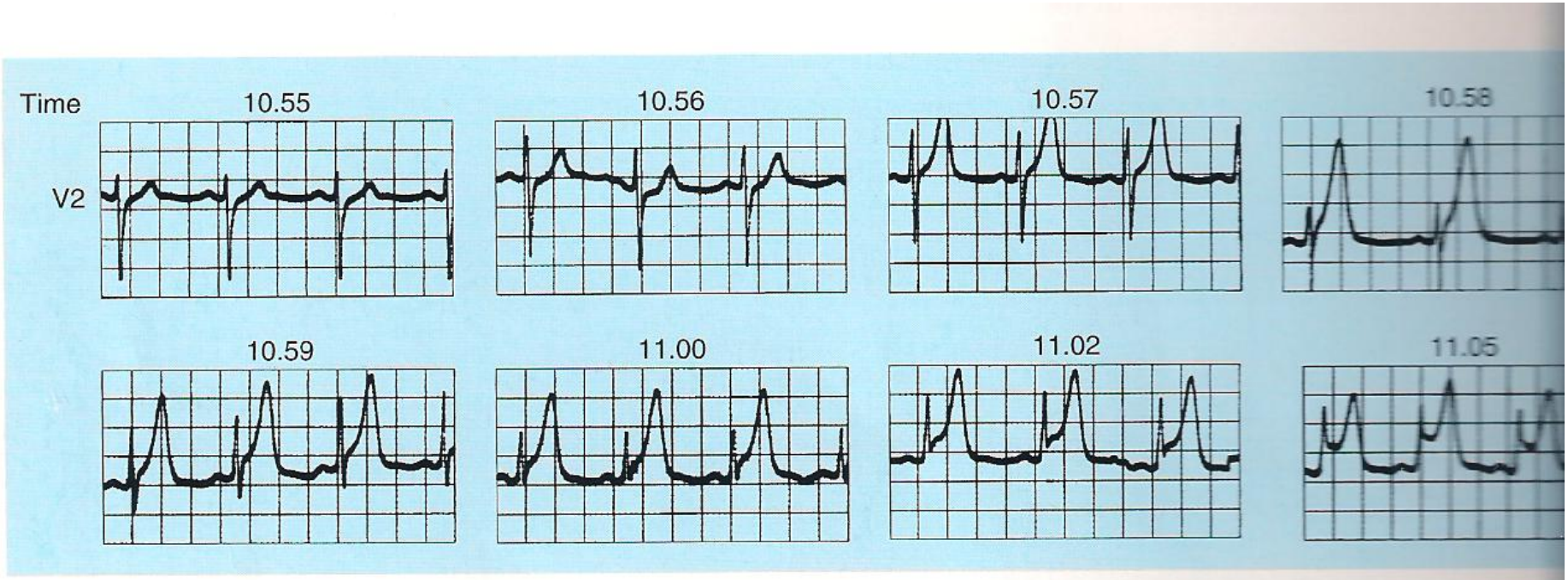
FIRST DEGREE OF ISCHEMIA



Saguie A, Sclarovsky Sa acute myocardial anterior wall infarction presenting with positive Twaves and without ST e;evation electr coronary correlation Chest 95;1211;1989

SECOND DEGREE OF ISCHEMIA





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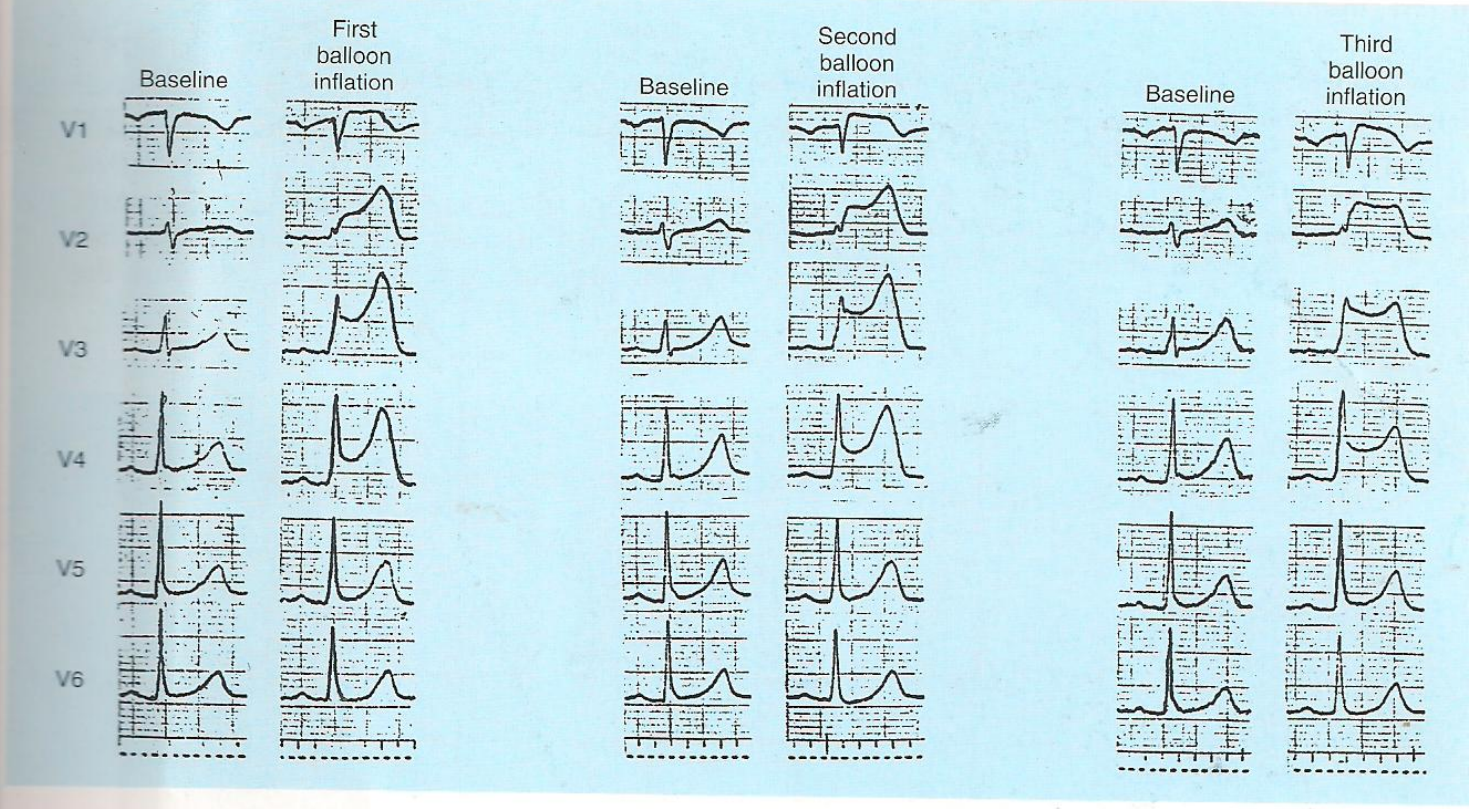


Figure 1.10 *ECG recordings during angioplasty during three balloon inflations and the preceding baselines. The patient developed the same ECG pattern of ischaemia Grade 3 during each*

developed the same ECG pattern of ischaemia Grade 3 during each one of the three balloon inflations.

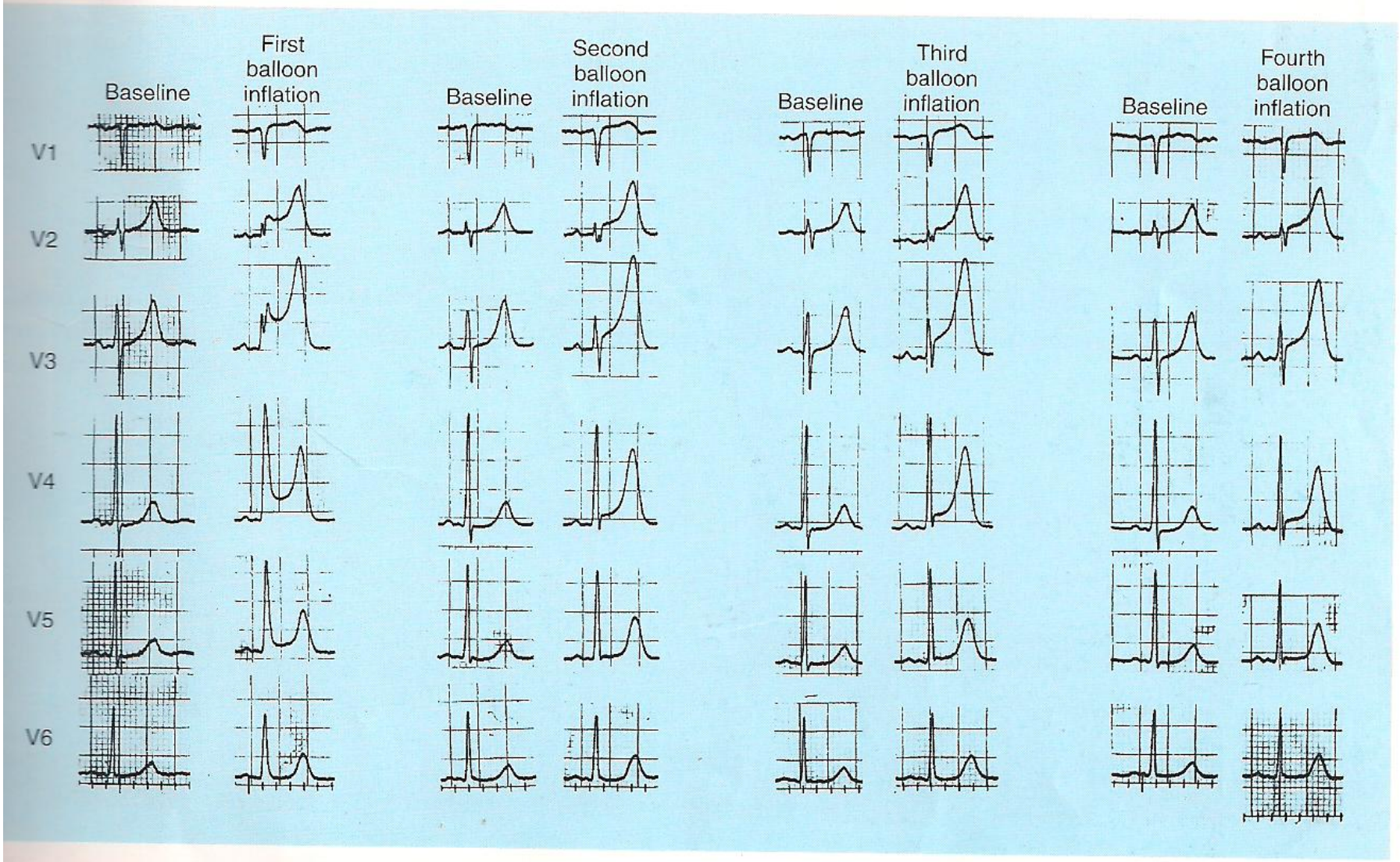


Figure 1.11 *ECG recordings during angioplasty during four balloon inflations and the preceding baselines. During the*

Figure 1.12 *ECG recordings during angioplasty during three balloon inflations and the preceding baselines. The patient developed only Grade 2 ischaemia during each of the three inflations.*

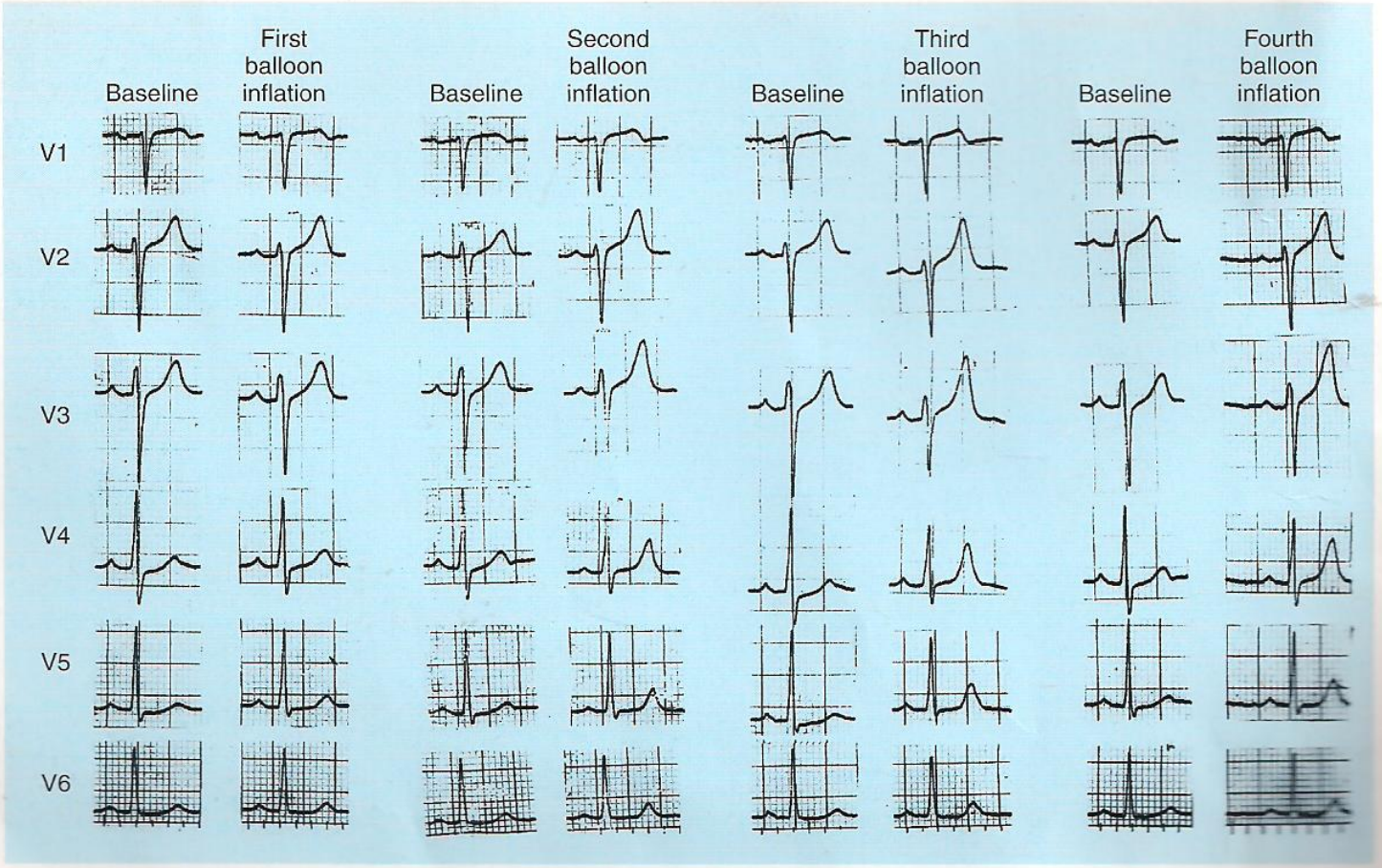
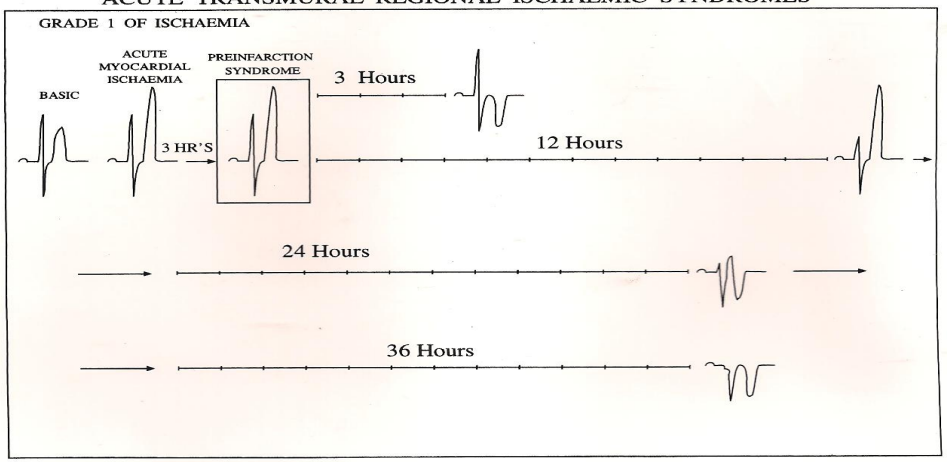
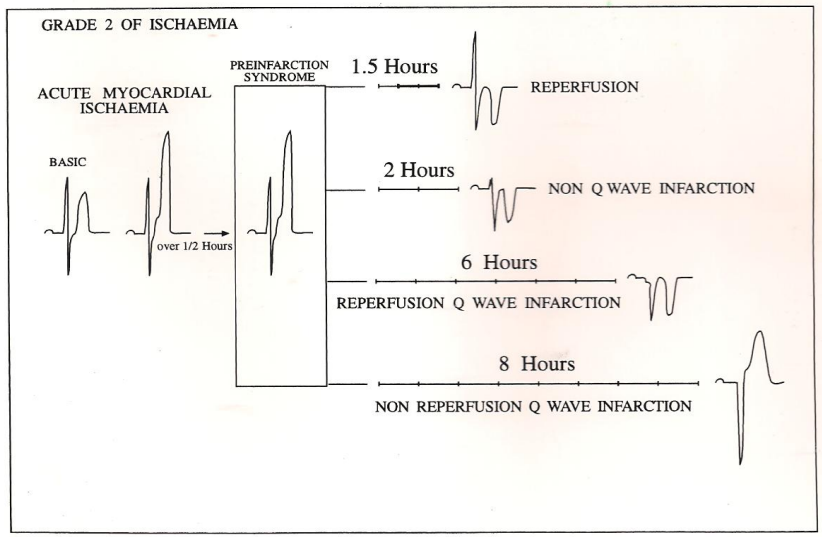


Figure 1.13 *ECG recordings during angioplasty during four balloon inflations and the preceding baselines. Note that th*

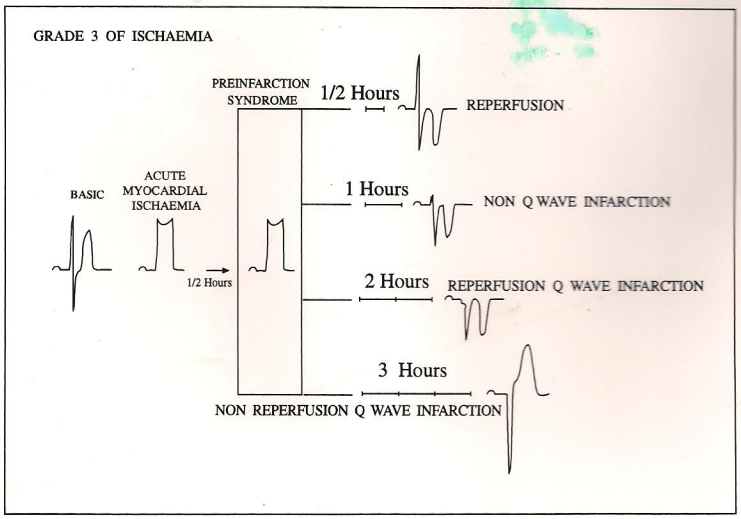
ACUTE TRANSMURAL REGIONAL ISCHAEMIC SYNDROMES



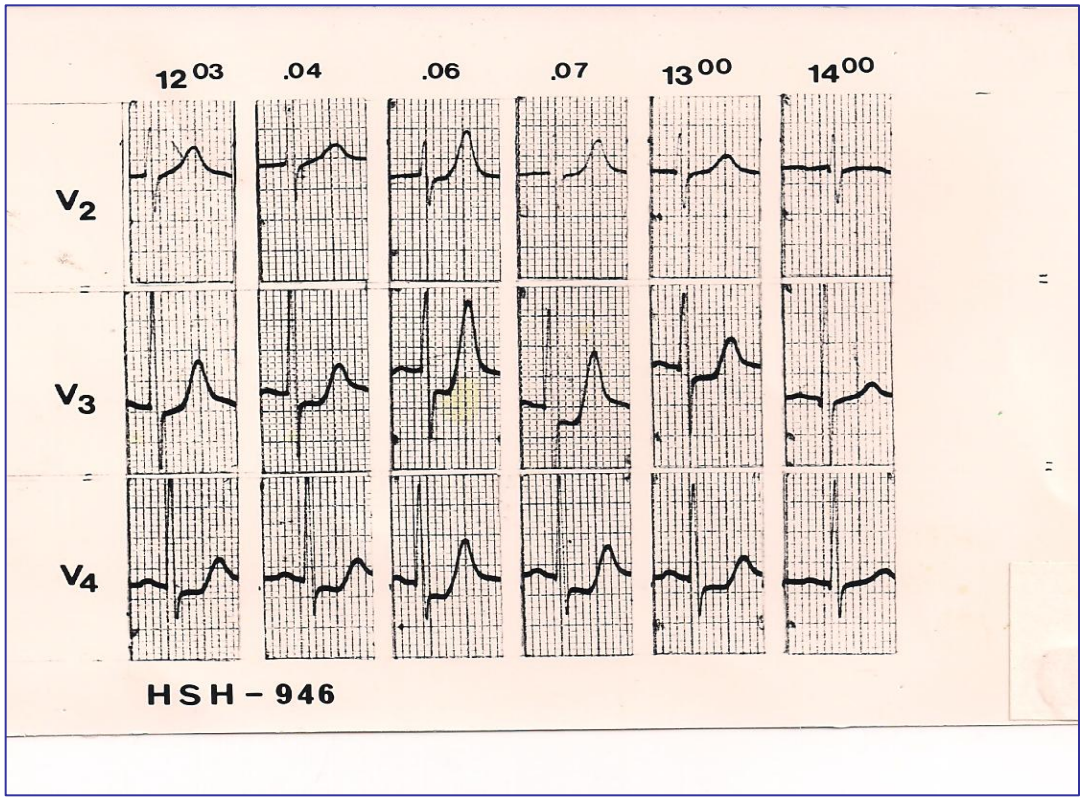
ACUTE TRANSMURAL REGIONAL ISCHAEMIC SYNDROMES

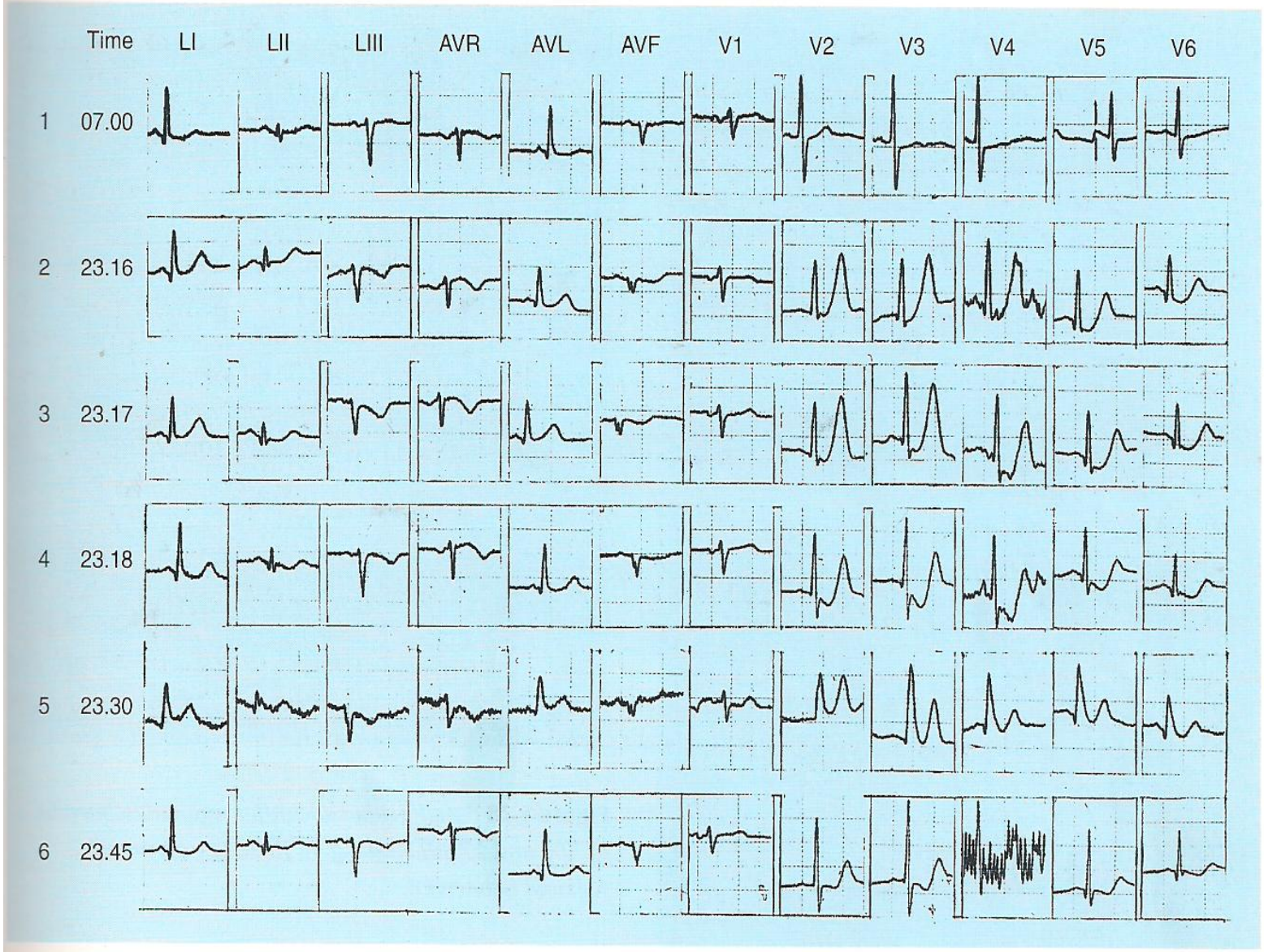


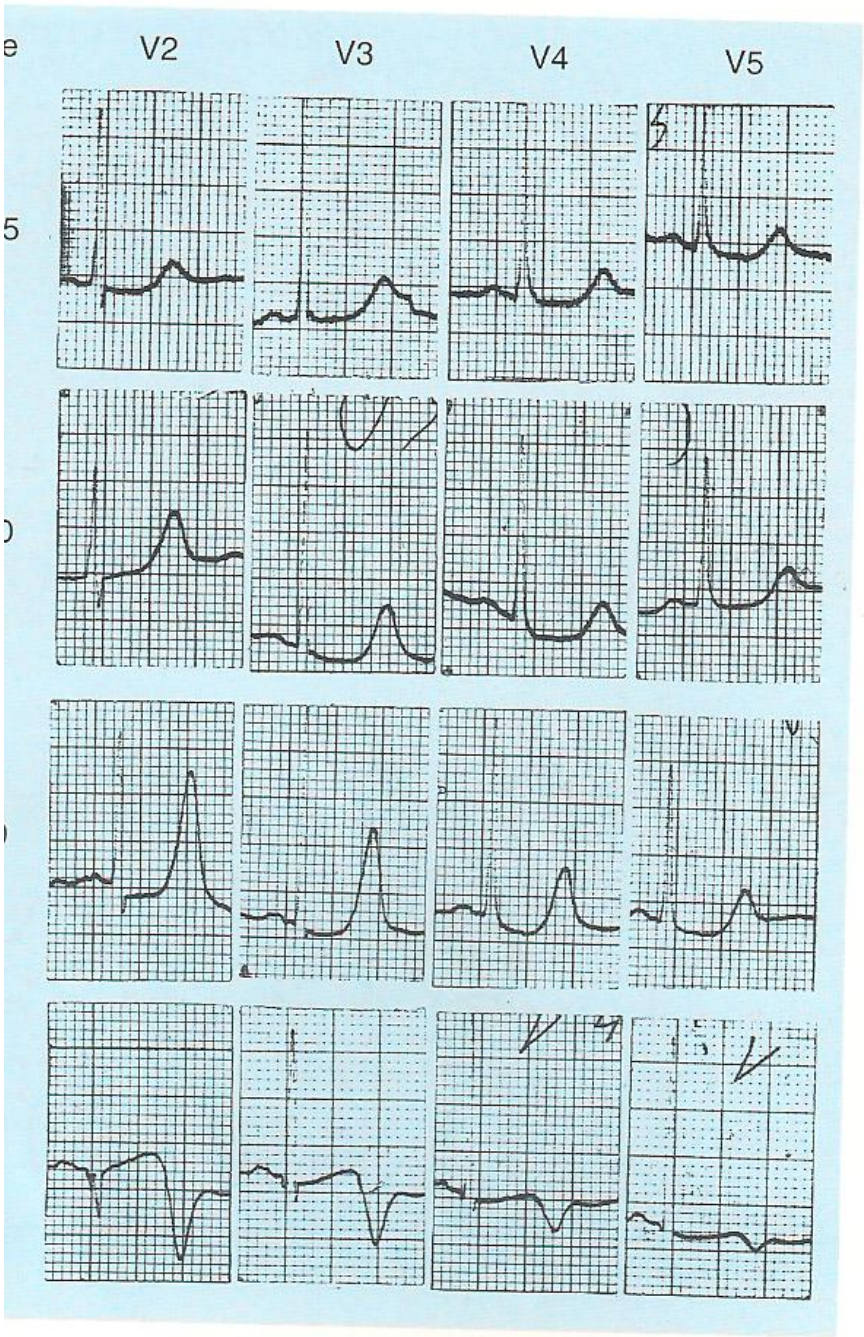
ACUTE TRANSMURAL REGIONAL ISCHAEMIC SYNDROMES

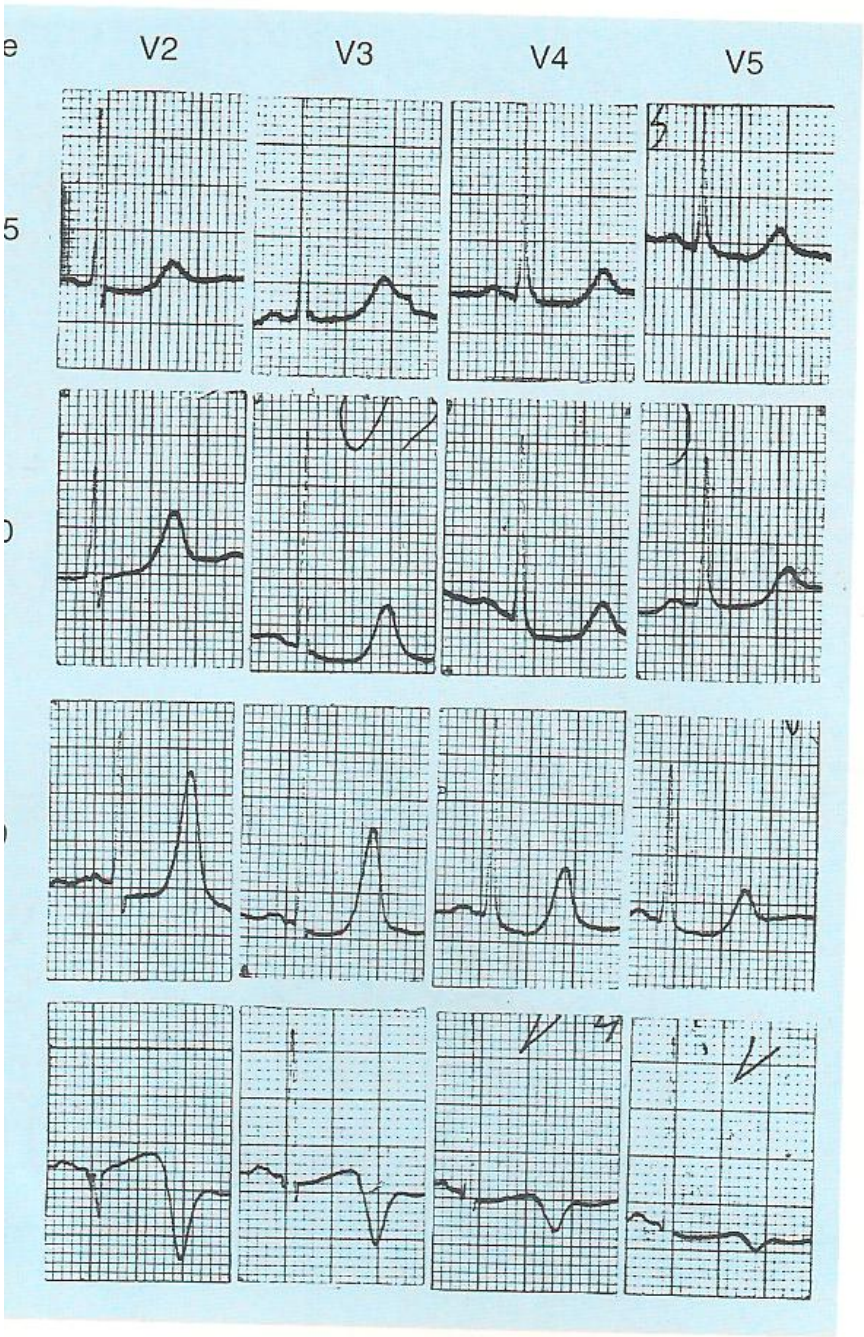


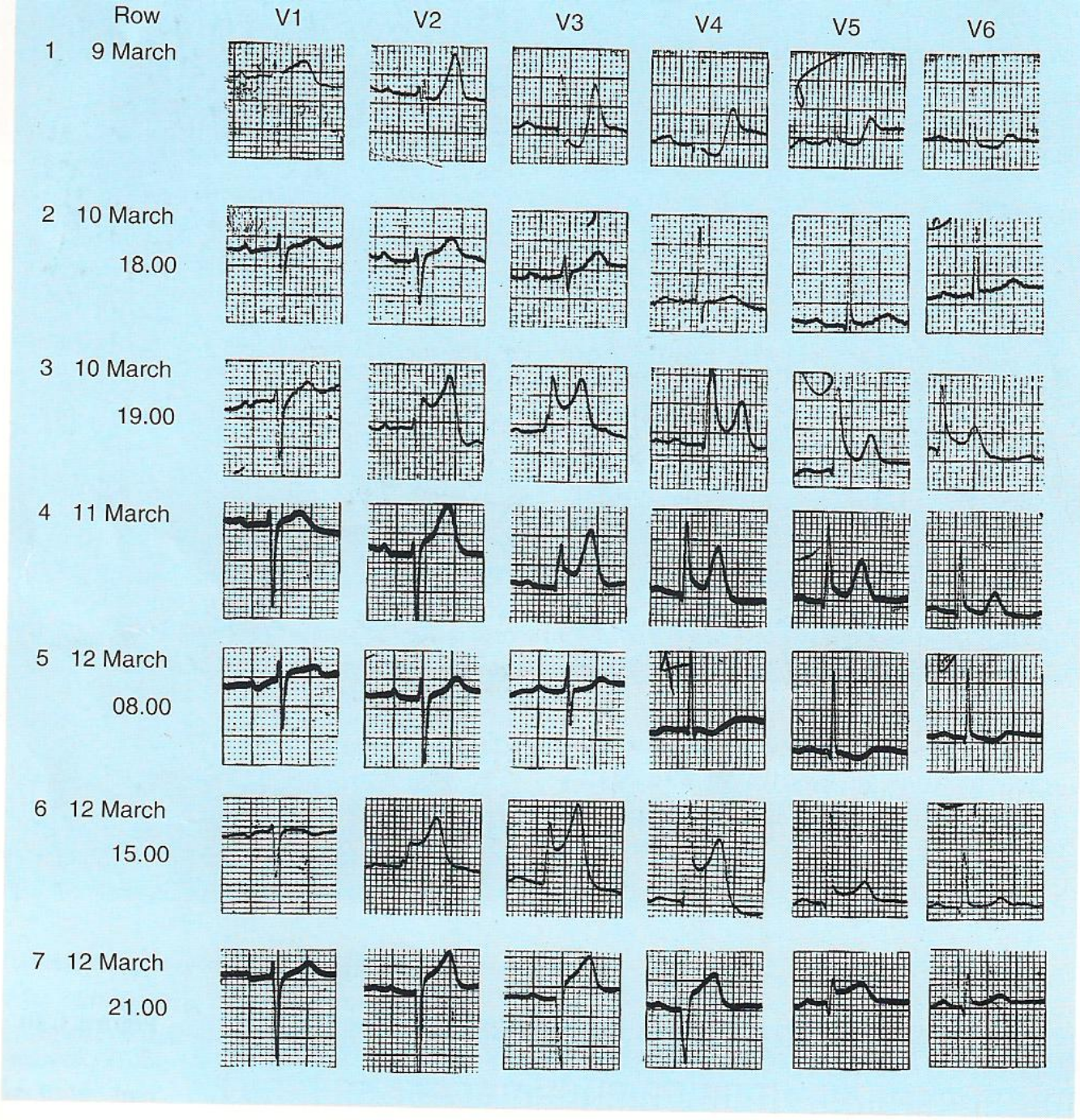
ACUTE SUBENDOCARDIAL REGIONAL ISCHEMIA

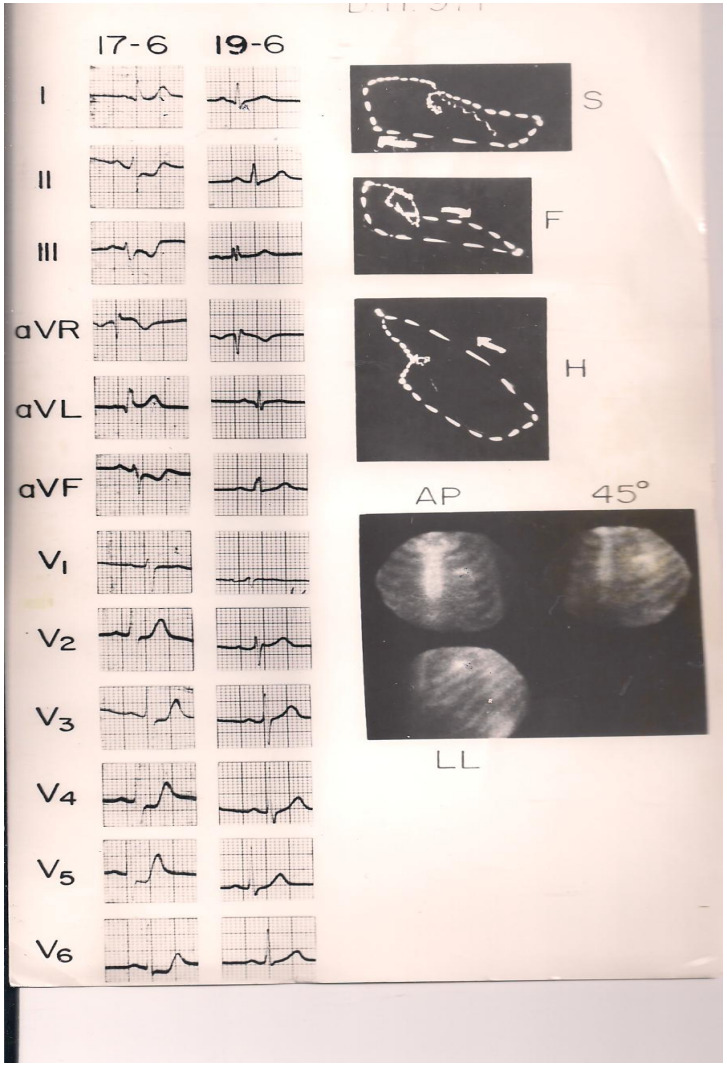



Time
LI
LII
LIII
AVR
AVL
AVF
V1
V2
V3
V4
V5
V6
1
07.00
2
23.16
3
23.17
4
23.18
5
23.30
6
23.45

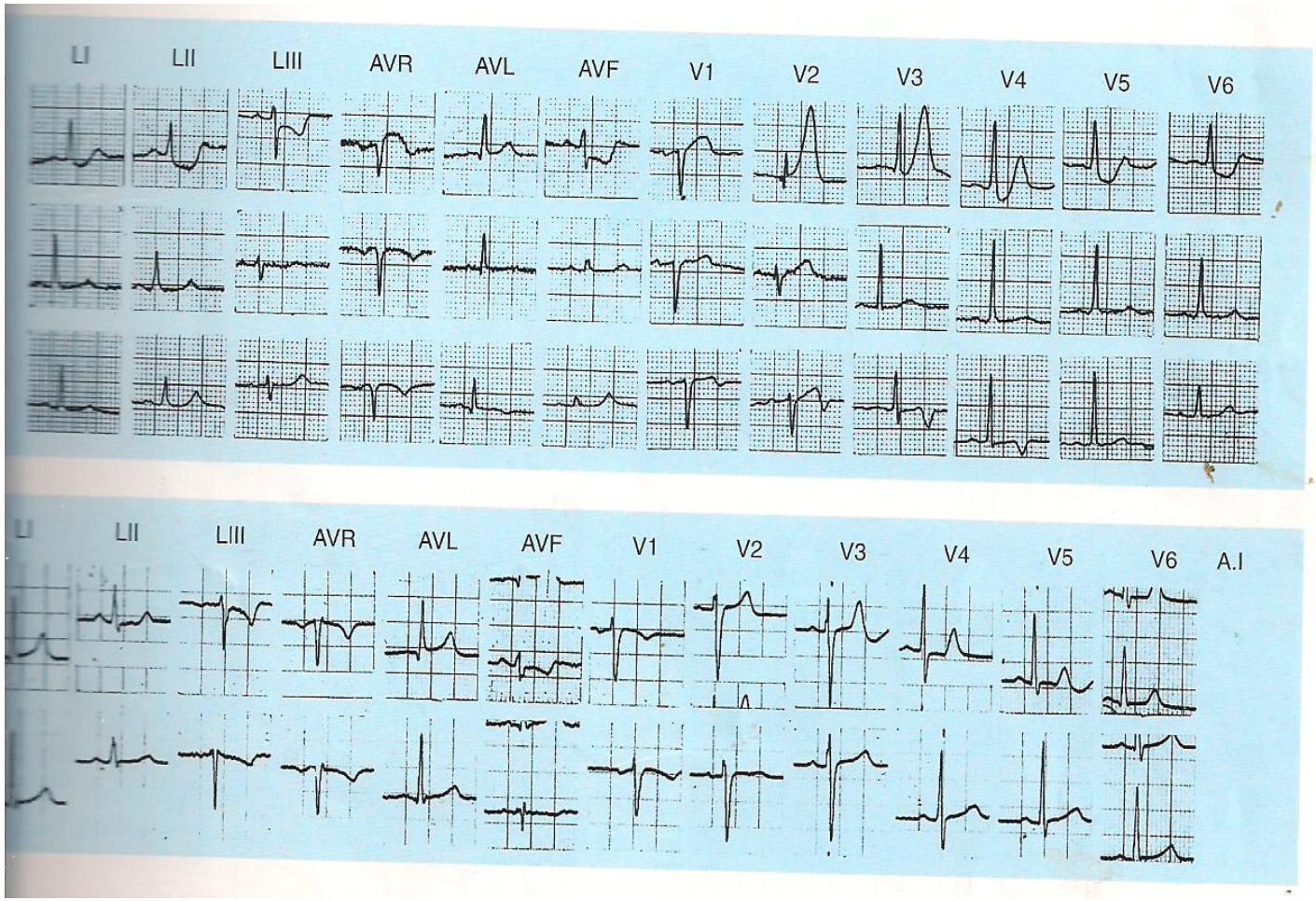

V2
V3
V4
V5

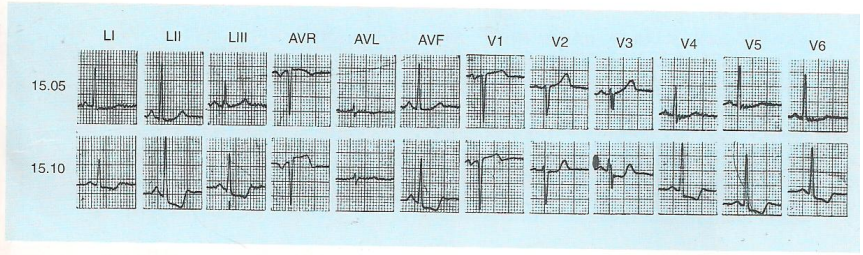

V2
V3
V4
V5

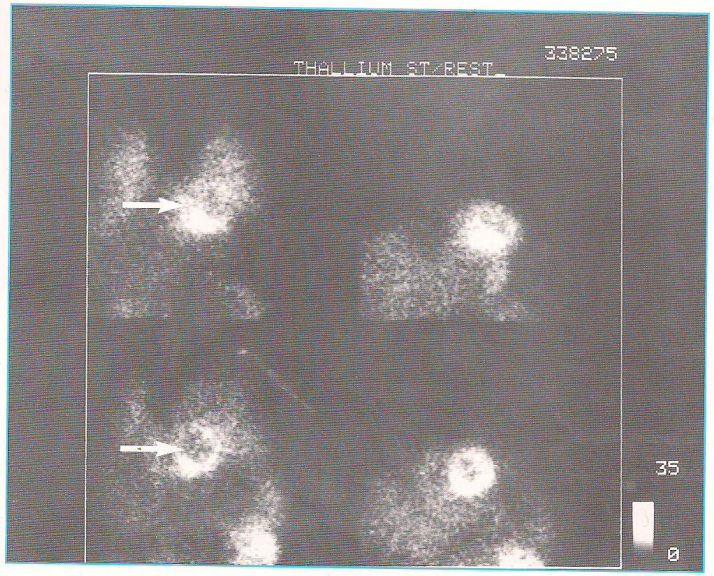

Row
V1
V2
V3
V4
V5
V6
1 9 March
2 10 March
18.00
3 10 March
19.00
4 11 March
5 12 March
08.00
6 12 March
15.00
7 12 March
21.00



Sclarovsky S et all isolated mid anterior wall infarction;a spetial ecg subtype with with AMI presenting with 2 non consecutive ST elevation and two different morpho;ogy ofST depression Int J cardiol 46;.37;1994


LI
LII
LIII
AVR
AVL
AVF
V1
V2
V3
V4
V5
V6
LI
LII
LIII
AVR
AVL
AVF
V1
V2
V3
V4
V5
V6
A.I





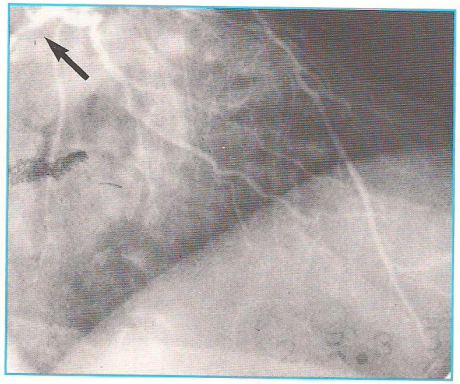


Figure 1.17 *(a) ECG tracings from a patient with acute circumferential subendocardial ischaemia. (b) Thalluim scintigraphy; during the acute episode thallium was injected, depicting uptake in the lungs (left panels, see arrows) without filling defects. Subsequent images illustrated that uptake was no longer evident, concomitantly with the resolution of the ECG changes (right panels). (c) Severe stenosis of the left main coronary artery (arrow) was detected during coronary angiography.*

ACUTE CIRCUNFERENTIAL SUBENDOCARDIAL ISCHEMIA

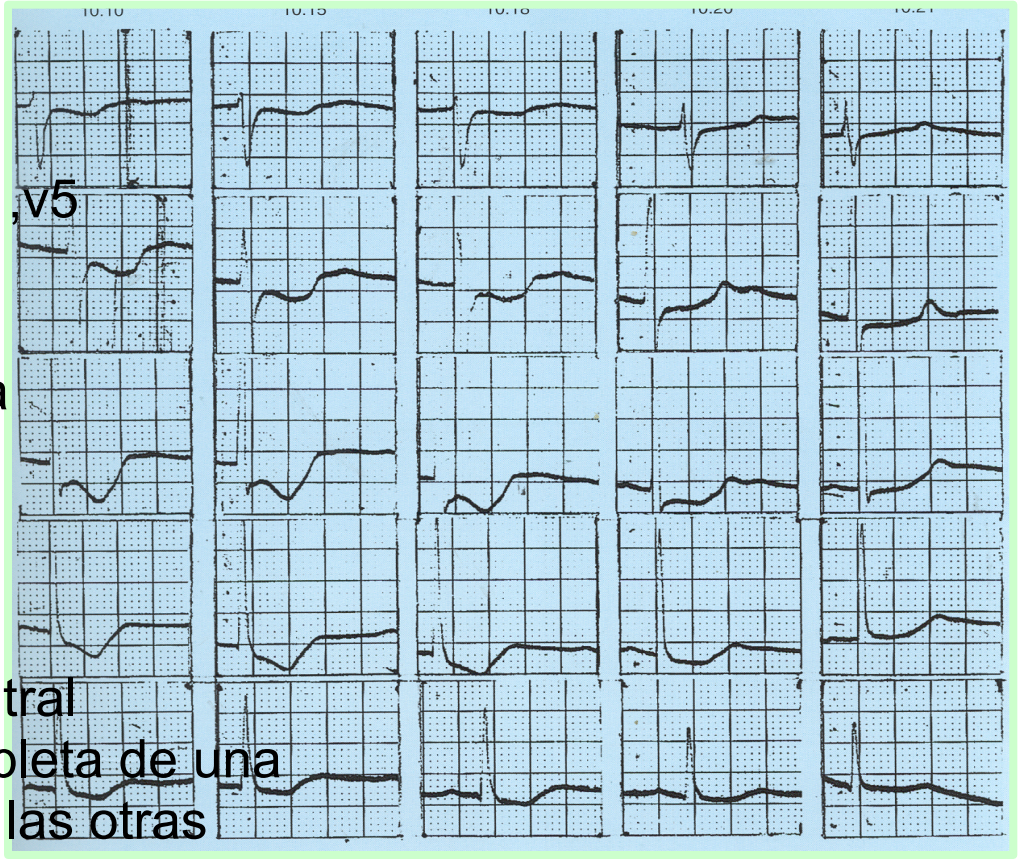
Ischemia aguda subendocardial Circunferencial

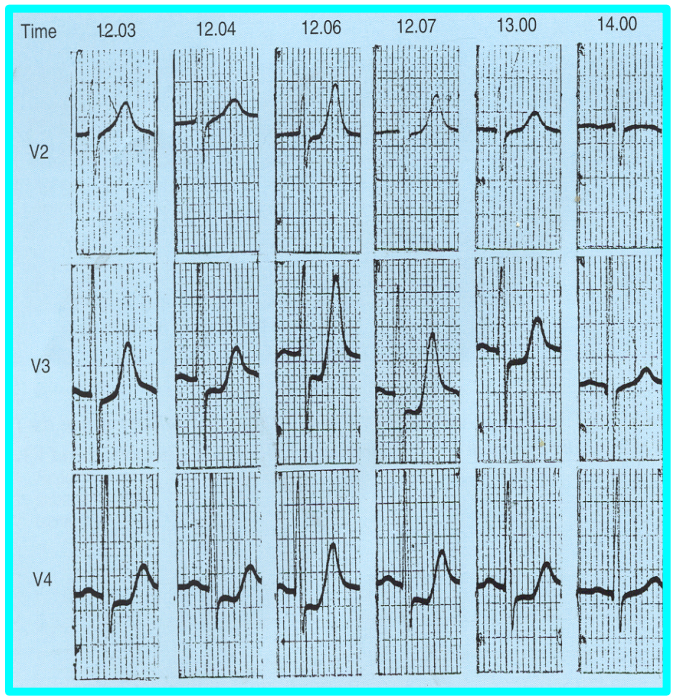
Depresion maxima del ST-T en v4 ,v5

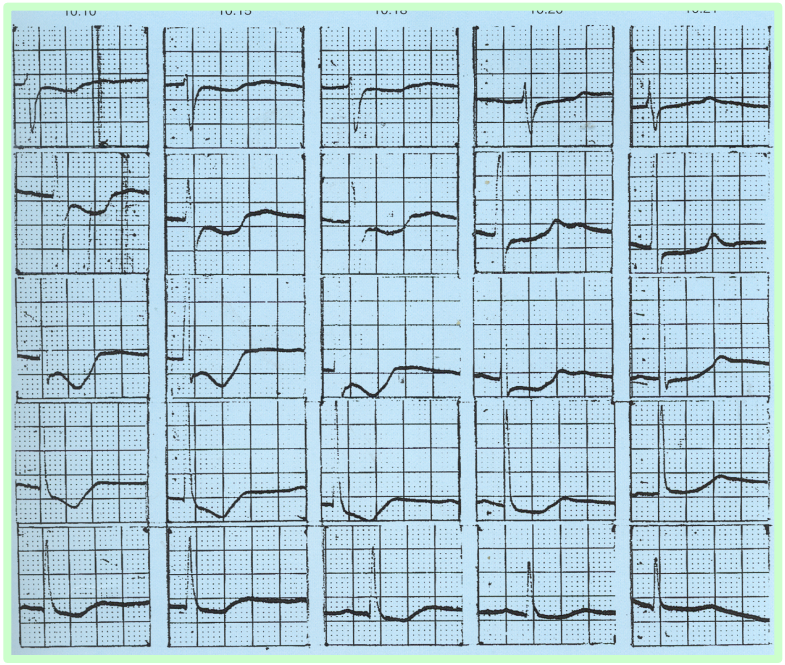
Frecuencia cardiaca < 100 lpm

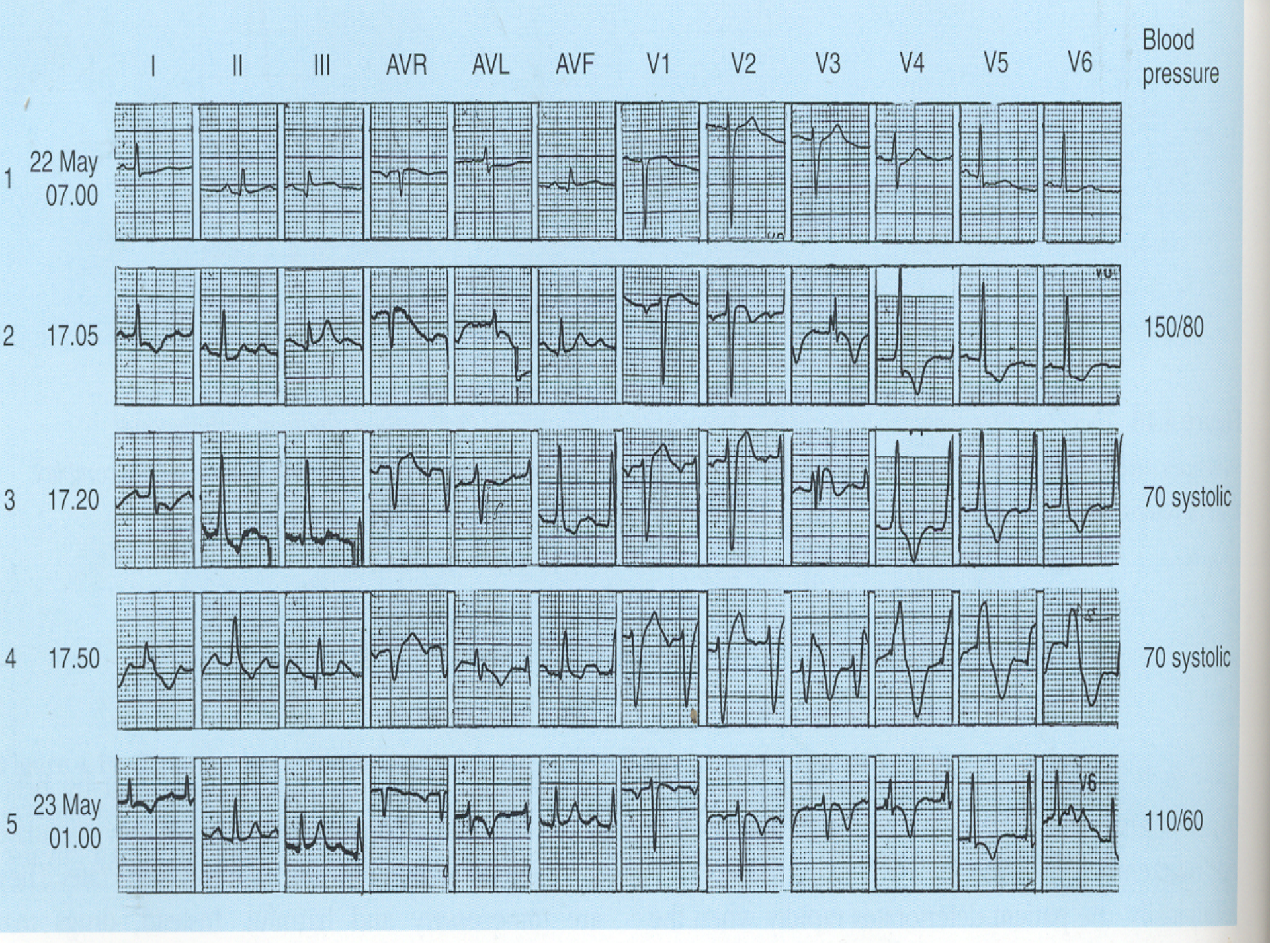
Este fenomeno expresa la maxima presion diastolica sobre la punta Izquierda

Debido a una obstruccion subita y completa sobre la coronaria central ezquierda o una obstruccion completa de una arteria epicardial en presencia de las otras 2 obstruidas




Time
12.03
12.04
12.06
12.07
13.00
14.00
V2
V3
V4




I
II
III
AVR
AVL
AVF
V1
V2
V3
V4
V5
V6
Blood pressure
1
22 May 07.00
2
17.05
150/80
3
17.20
70 systolic
4
17.50
70 systolic
5
23 May 01.00
110/60

Physiological and pathological remodeling in acute myocardial infarction

- In the early 80ties the concept of remodeling was applied to the structural and molecular changes occurring in the healthy left ventricle regions during and after myocardial Infarction

- Later this concept was extended to the left ventricle hypertrophy induced by systolic or diastolic overloading, metabolic diseases, and genetic mutations

Swyngheadaum B et al, Physiol Rev 1999

- Electrical remodeling is a persistent change in the electrophysiological properties of the myocardium in response to a change in rate or activation sequence.
  These are- Ionic changes but not structural

(Darwin Jeyaraj et al, Circulation 2007)

- Electrophysiological remodeling is a concept applied to the structural and molecular changes in the hypertrophic left ventricle witch promote complicated arrhythmias

(Ruan H et al Circulation 2007)

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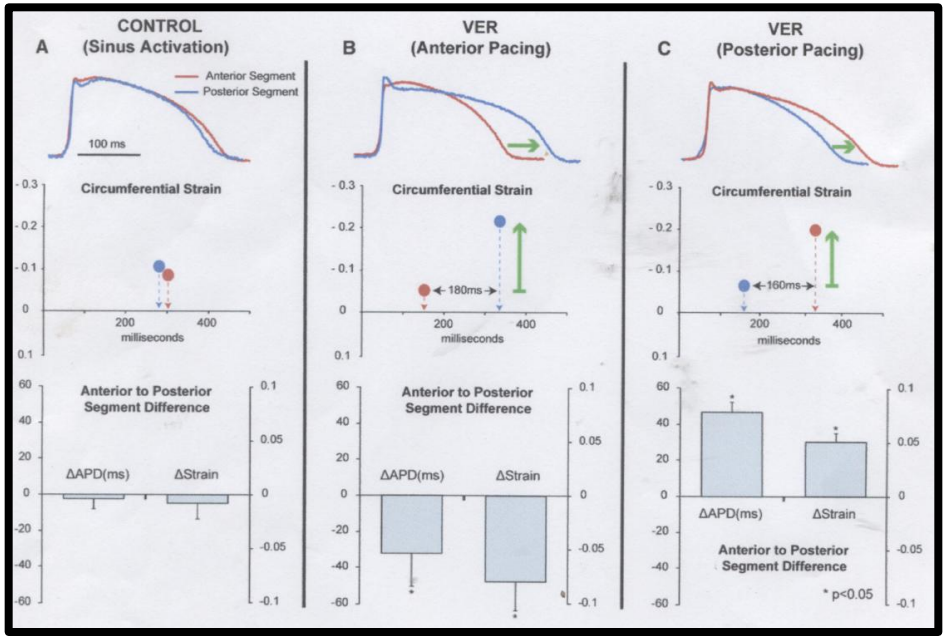
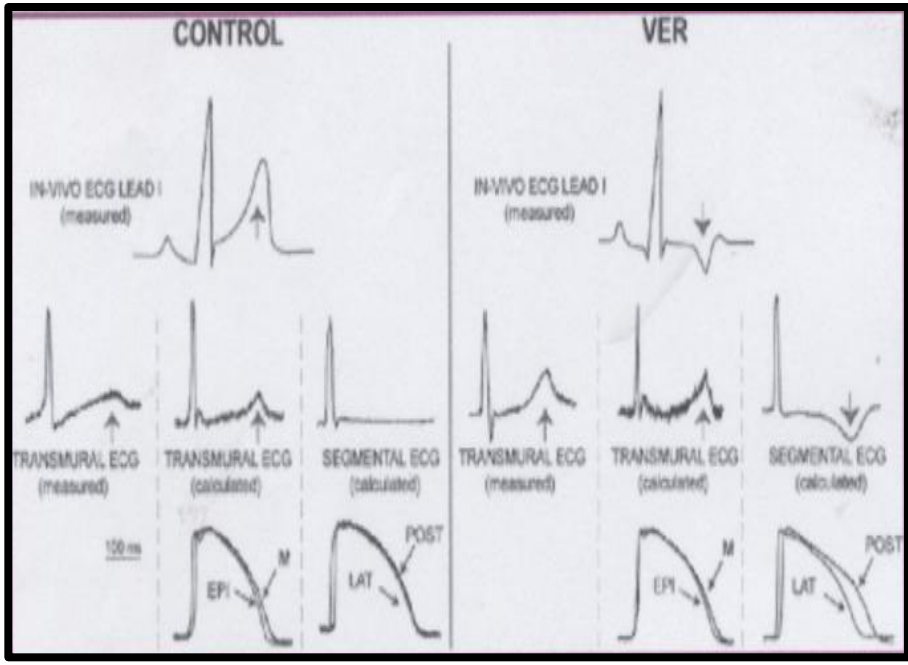
(Ruan H et al Circulation 2007)

Electrical remodeling (cardiac memory)

Mechano-electrical feedback as a novel mechanism of electrical remodeling

Jeyaraj D et al, Circulation 2007





Electrocardiographic remodeling expresses the morphologic cardiac changes occurring in patients with ;1) left ventricular hypertrophy due to systolic or diastolic overloading ,genetic o congenital disease or metabolic causes2) in acute and chronic Ischemic syndromes

ECG physiological remodeling in acute ischemic syndrome expresses the electrophysiological and metabolic changes occuring in the non involved muscle due to an sudden obstruction of a epicardial artery

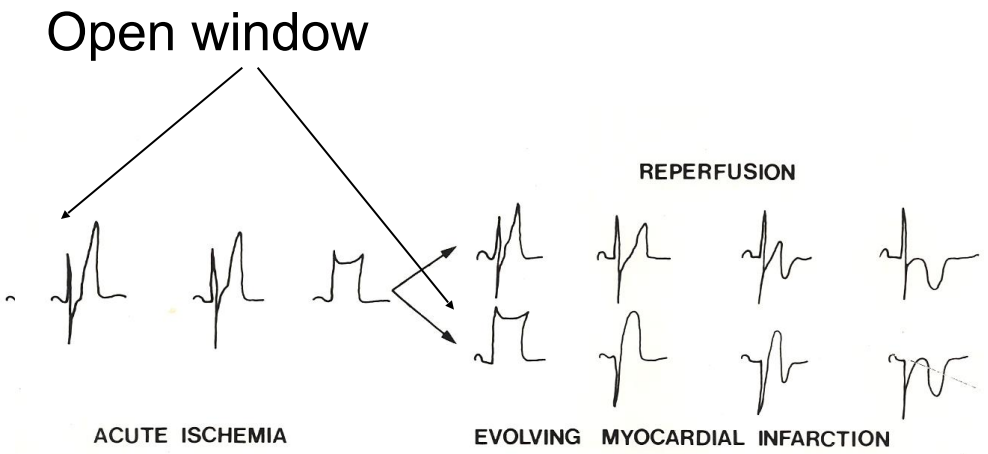
Electrocardiographic pre- infarction is the appearance of a

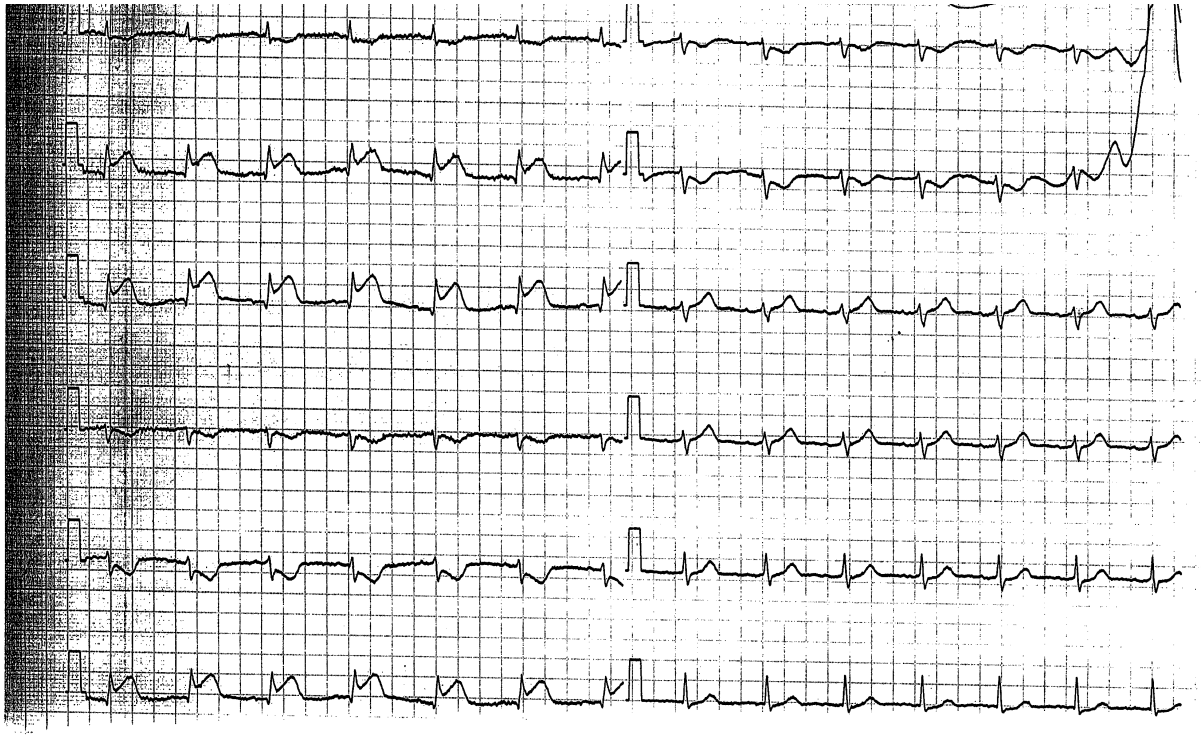
sudden appearance of ST segment elevation with positive T waves

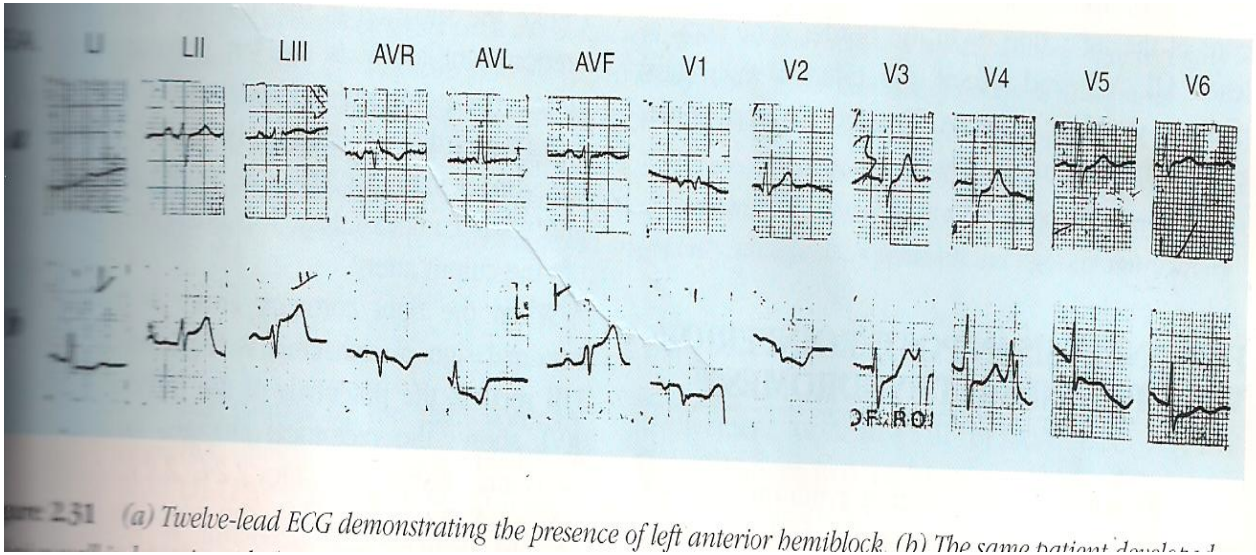
and previous the appearance of Q wave ,due to an sudden obstruction of an epicardial artery

This ECG phase of the acute ischemic syndrome is the

trombolitic or angioplastic open window opportunity

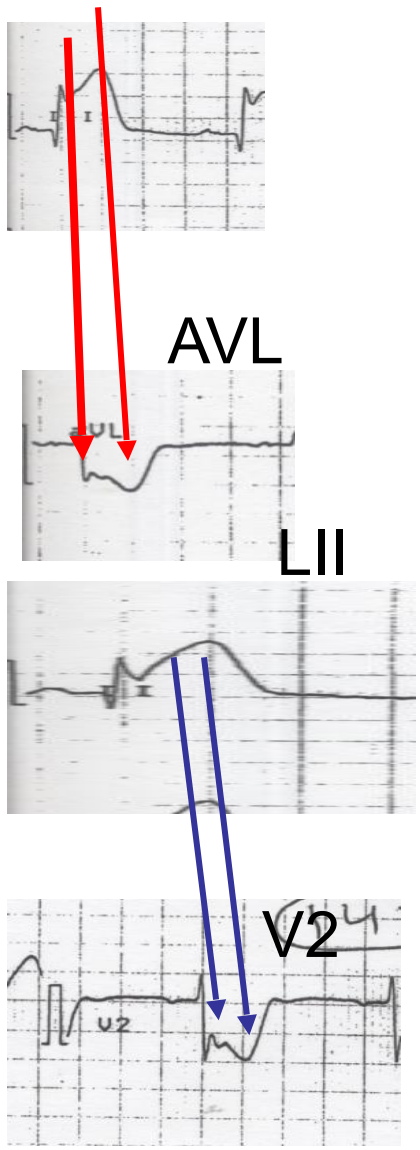


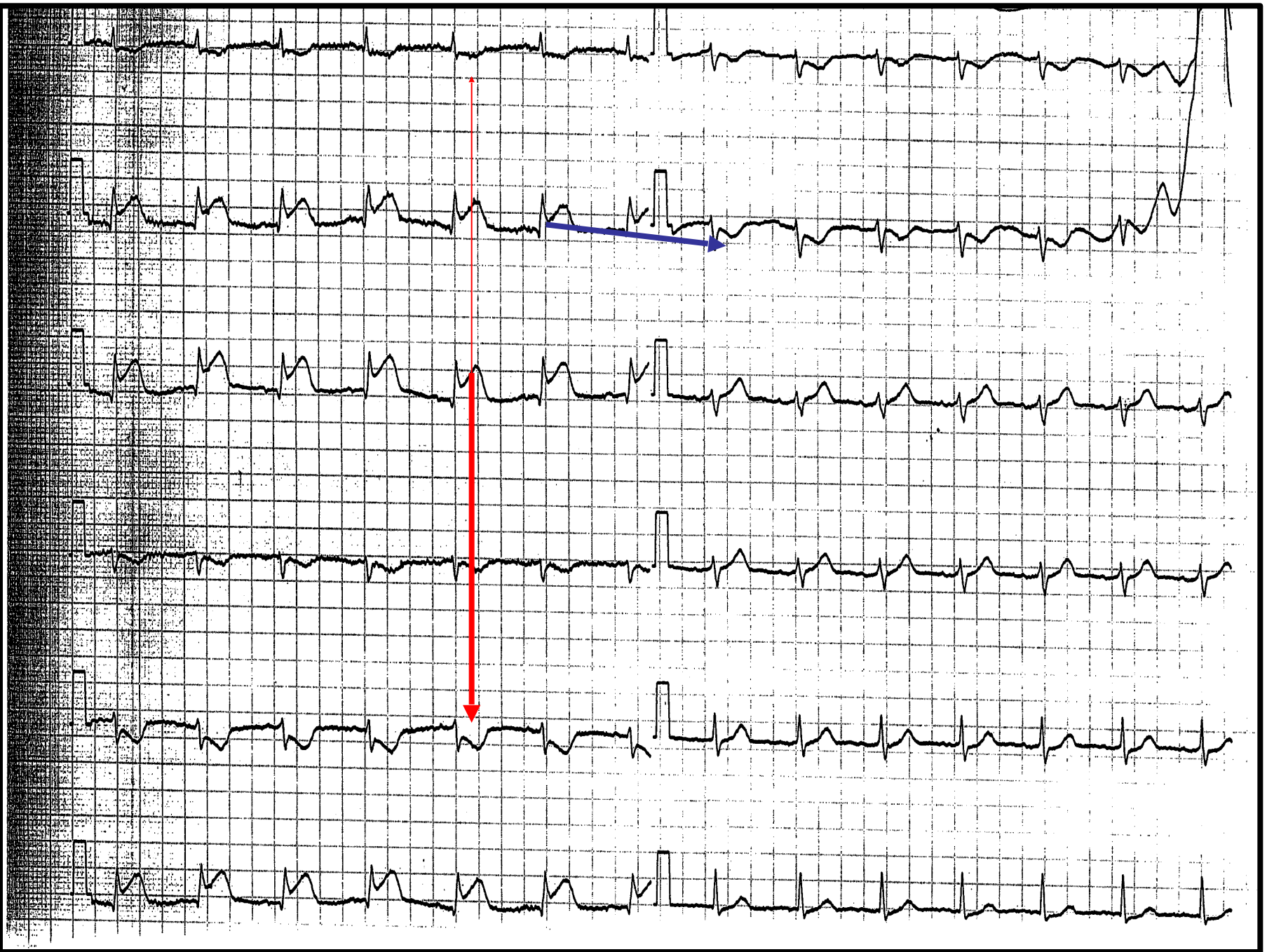


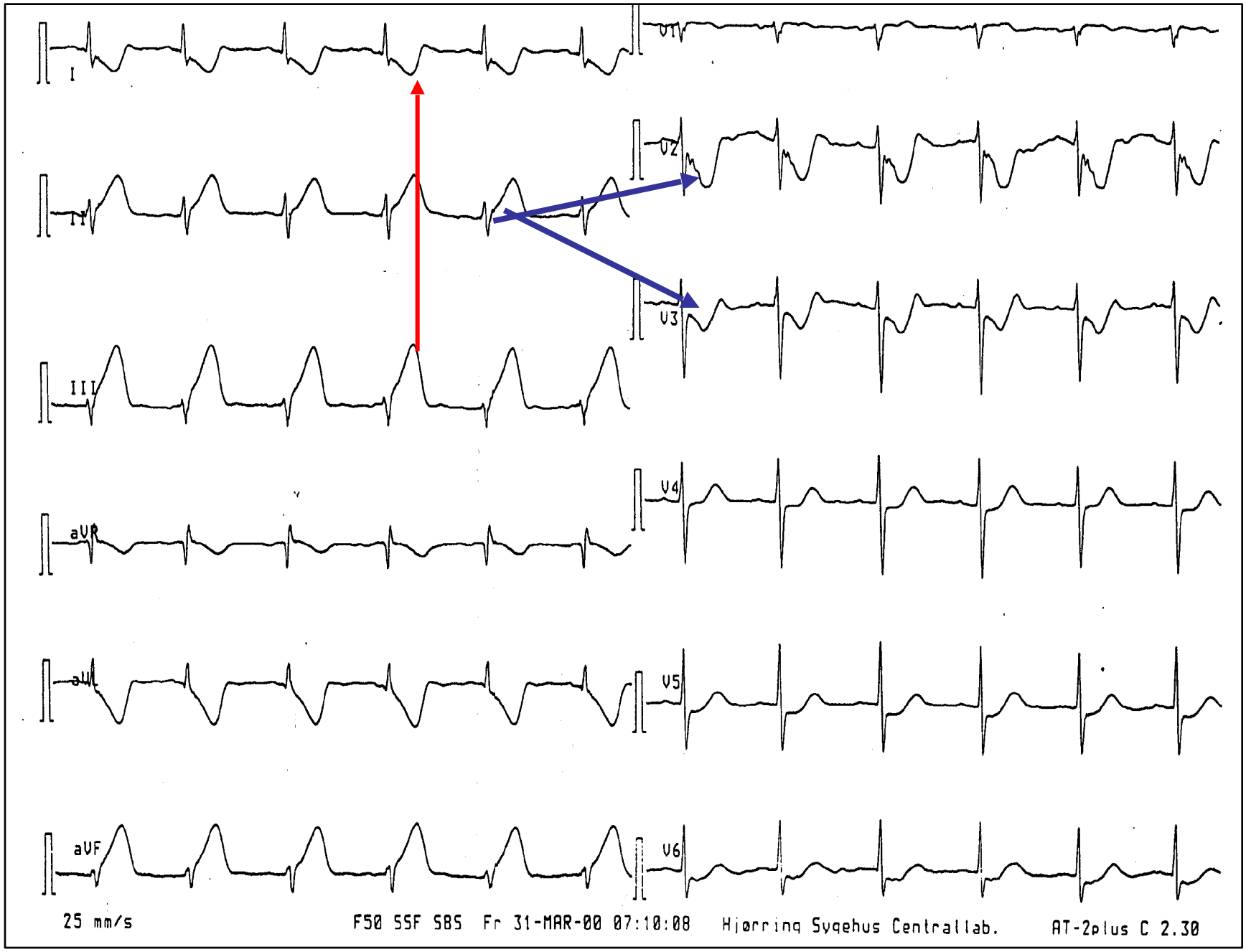


231 (a) Twelve-lead ECG demonstrating the presence of left anterior hemiblock. (b) The same patient developed

The ECG's physiological remodeling rules in acute inferior wall Infarction due to a distal right coronary right coronary obstructio






I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
25 mm/s
F50 SSF SBS Fr 31-MAR-00 07:10:08 Hjørring Sygehus Centrallab.
AT-2plus C 2.30

I
V1
II
V2
III
V3
aVR
V4
aVL
V5
aVF
V6
14-JAN 01 (SØN) 10:27 NYK. MORS SYGEHUS A1
Nyk. Mors Sygehus
25 mm/s 10 mm/mV 35Hz 50Hz MEGACART
*** COPY ***

the pre infarction syndrome, the ST-T depression in the opponent area of the ST-T elevation is due to a double mechanism

- ➢ Electrophysiologic- reciprocal changes
- ➢ Homodynamic- in according with Laplace law the opposite side of the infarction must reduce the radius of the cavity to decrease the wall tension (hyper- dynamic muscle)

The adrenergic substances stimulate the cardiac alfa 1 receptors witch induce hyperkinetic healthy muscle in the presence of acute myocardial infarction

The hyperkinetic opposite area is due to a local releaseof adrenergic subtances activating the alfa receptors adrenergic drive in pathological remodeling

Wood lock E et alCardiac alfa 1 Cardiovascular research 2008;77 ;452

Progress in Biophysics and Molecular Biology
Volume 97, Issues 2-3, June-July 2008, Pages 268-281
Life and Mechanosensitivity

doi:10.1016/j.pbiomolbio.2008.02.010 | How to Cite or Link Using DOI

Review

Inhomogeneity in the response to mechanical stimulation: Cardiac muscle function and gene expression

Rachel Stones[a], Stephen H. Gilbert[a], David Benoist[a] and Ed White[a, ✉]
[a]Institute of Membrane and Systems Biology, University of Leeds, Leeds LS29JT, UK

Available online 13 February 2008.

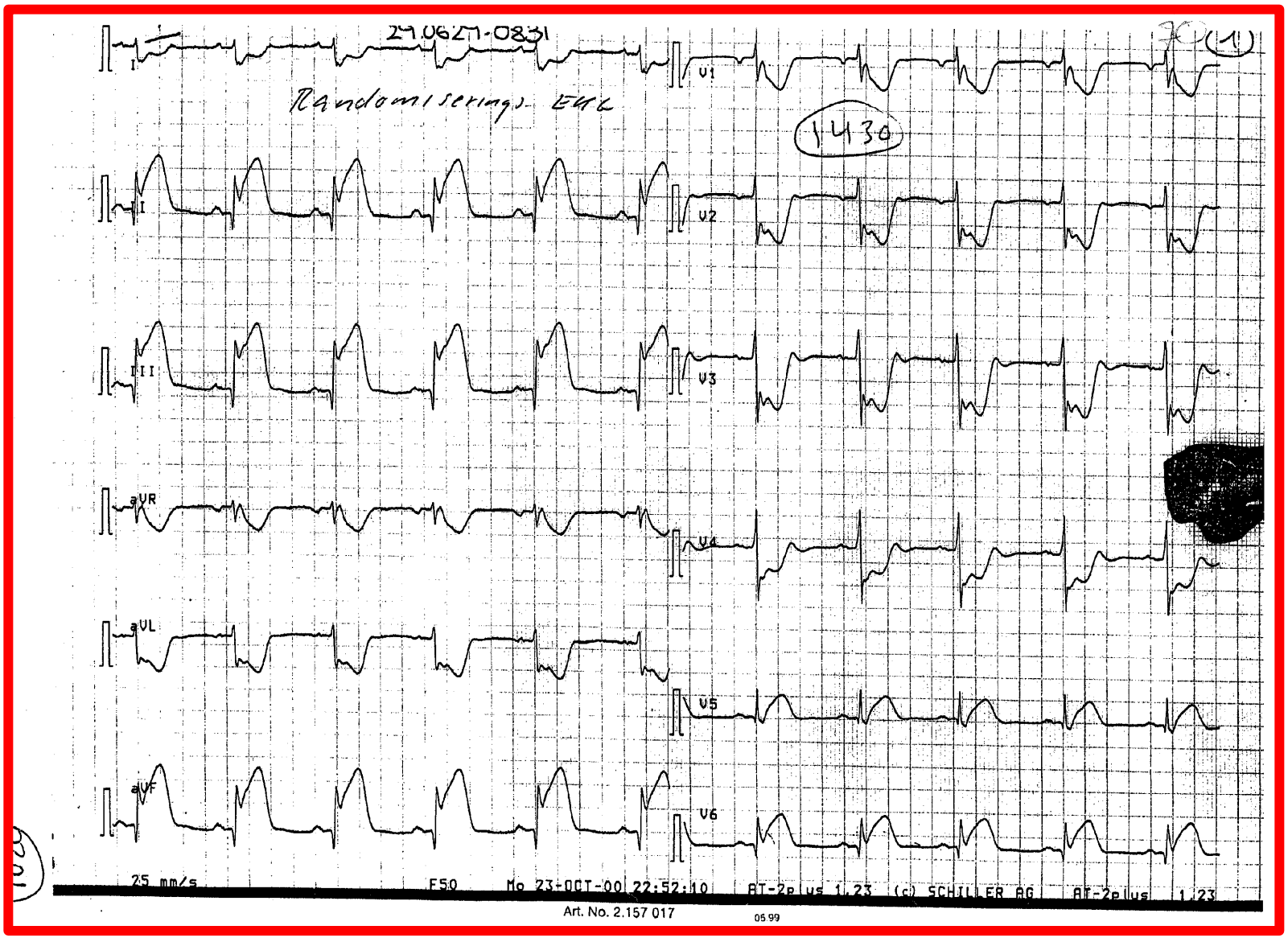
Abstract

Mechanical stimulation has important consequences for myocardial function. However, this stimulation and the response to it, is not uniform. The right ventricle is thinner walled and operates at lower pressure than the left ventricle. Within the ventricles, differences in the orientation of myocardial fibres exist. These differences produce inhomogeneity in the stress and strain between and across the ventricles. Possibly as a result of these variations in mechanical stimulation, there are well characterised inhomogeneities in gene expression and protein function within the ventricular myocardium, for example in the transient outward K^+ current and its associated Kv channels. Perhaps not surprisingly, it is becoming apparent that gradients of expression and function exist for proteins that are intimately involved in the response to mechanical stimulation in the heart, for example in the left ventricle of the rat there is a transmural gradient in mRNA and current density of the mechanosensitive two-pore domain K^+ channel TREK-1 (ENDO>EPI). In healthy hearts it is assumed that these gradients are important for normal function and therefore that their disruption in diseased myocardium is involved in the dysfunction that occurs.

Keywords: Stress; Strain; mRNA; Epicardium; Endocardium; Mechanosensitive channels

Article Outline

1. Introduction



Art. No. 2.157 017

05.99