Acuut Coronair Syndroom & Acuut Myocard Infarct

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www.ecgpedia.org

Cardionetworks

Begripsbepaling

- Begripsbepaling/Definities
- Pathofysiologie
- Klachten & symptomen
- Diagnostiek (m.n. ECG)
- Therapie
- Complicaties in het acute stadium
- Complicaties later in het beloop
- Prognose

Definities

- **Hypoxie** (*Grieks*) verminderde zuurstof in de weefsels
- **Anoxie** (*Grieks*) zeer laag/afwezig zuurstof in de weefsels
- **Perfusie** (*Latijn*) weefseldoorstroming
- **Ischemie** (*Grieks*) inadequate bloedtoevoer van weefsel of orgaan

  soorten: - supply

  - demand $\rightarrow$ dysbalans = ischemie

  in principe *reversibel*
- **Angina** *(Latijn)* pijn
- **Pectoris** *(Latijn)* van de borst

Definities

Necrose (*Grieks*) accidentele, door externe factoren veroorzaakte, celdood

Infarct (*Latijn*) irreversibele (orgaan)schade

DUS:

<u>Angina pectoris</u>

*"Pijn op de borst (letterlijk) veroorzaakt door reversibele, maar inadequate bloedtoevoer van (een deel van) de hartspier (→ **dysbalans** tussen **supply + demand)**."*

Angina Pectoris =

Hartkramp

Begripsbepaling

Stabiele angina pectoris

Chronische verminderde supply als gevolg van coronair stenosen. In rust is er voldoende perfusie. Indien demand hoger wordt, treedt pijn op de borst op. Zodra de demand minder wordt, verdwijnen de klachten.

Instabiele angina pectoris

Acute maar reversibele inadequate bloedvoorziening (supply) van (een deel van) de hartspier ten gevolge van het tekort schieten van de bloedtoevoer (kan irreversibel worden = myocard infarct)→ CCU indicatie. CAVE: dreigend infarct.

Begripsbepaling

Instabiele angina pectoris

Acute maar *reversibele* inadequate bloedvoorziening van (een deel van) de hartspier ten gevolge van het tekort schieten van de bloedtoevoer (kan irreversibel worden = myocard infarct).

Acuut myocard infarct

Acute en *irreversibele* beschadiging van (een deel van) de hartspier ten gevolge van het tekort schieten van de bloedtoevoer, leidend tot necrose en verlittekening van het desbetreffende deel van het myocard.

NYHA-klasse

- New York Heart Association
- Bij stabiele AP of hartfalen
- Functionele klasse I-IV

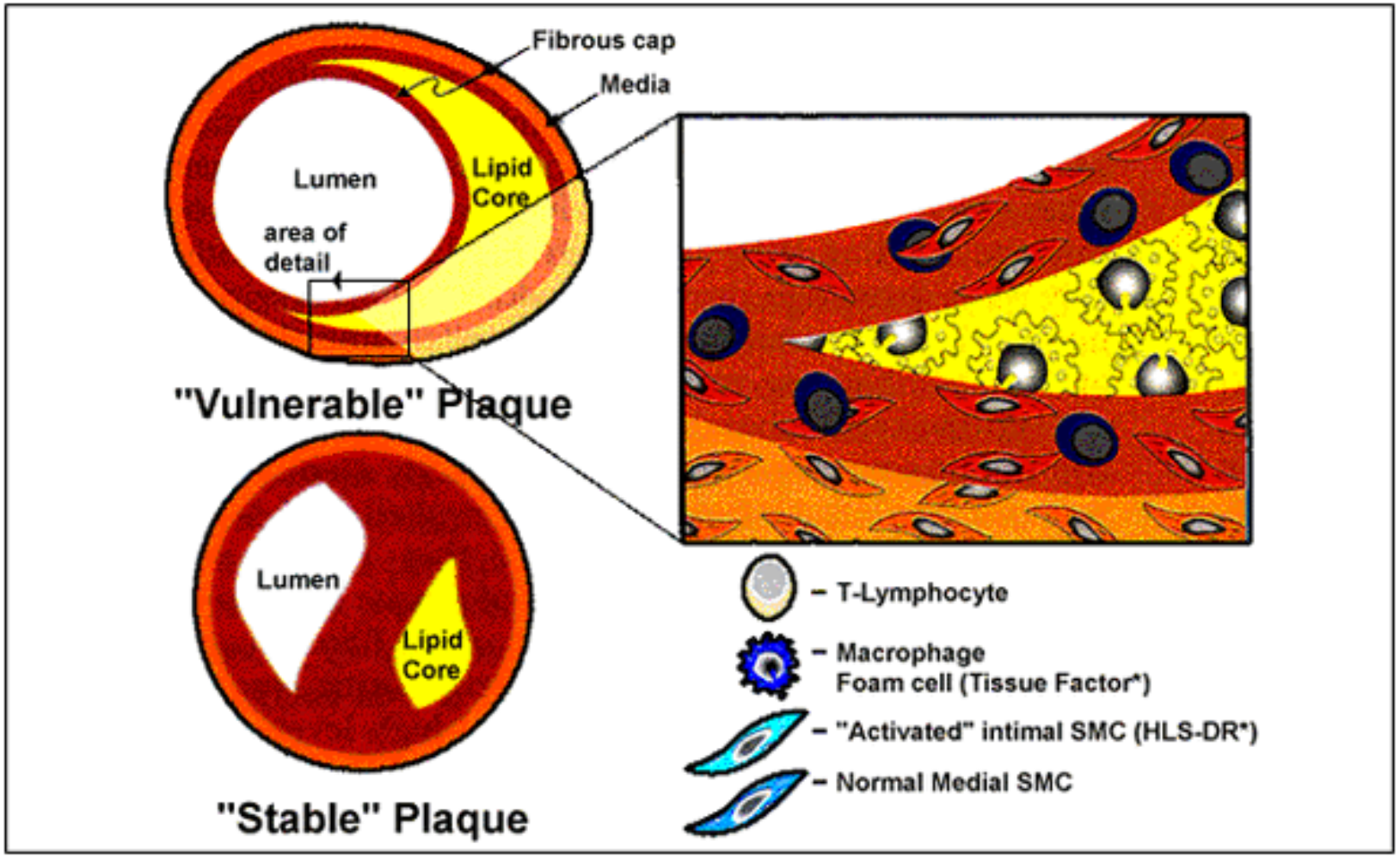
  - I Geen of sporadisch klachten
  - II Klachten bij zware inspanning
  - III Klachten bij lichte inspanning
  - IV Klachten in rust

CCS

- Canadian Cardiovascular Society
- Uitsluitend voor angina pectoris
- Functionele klasse I-IV

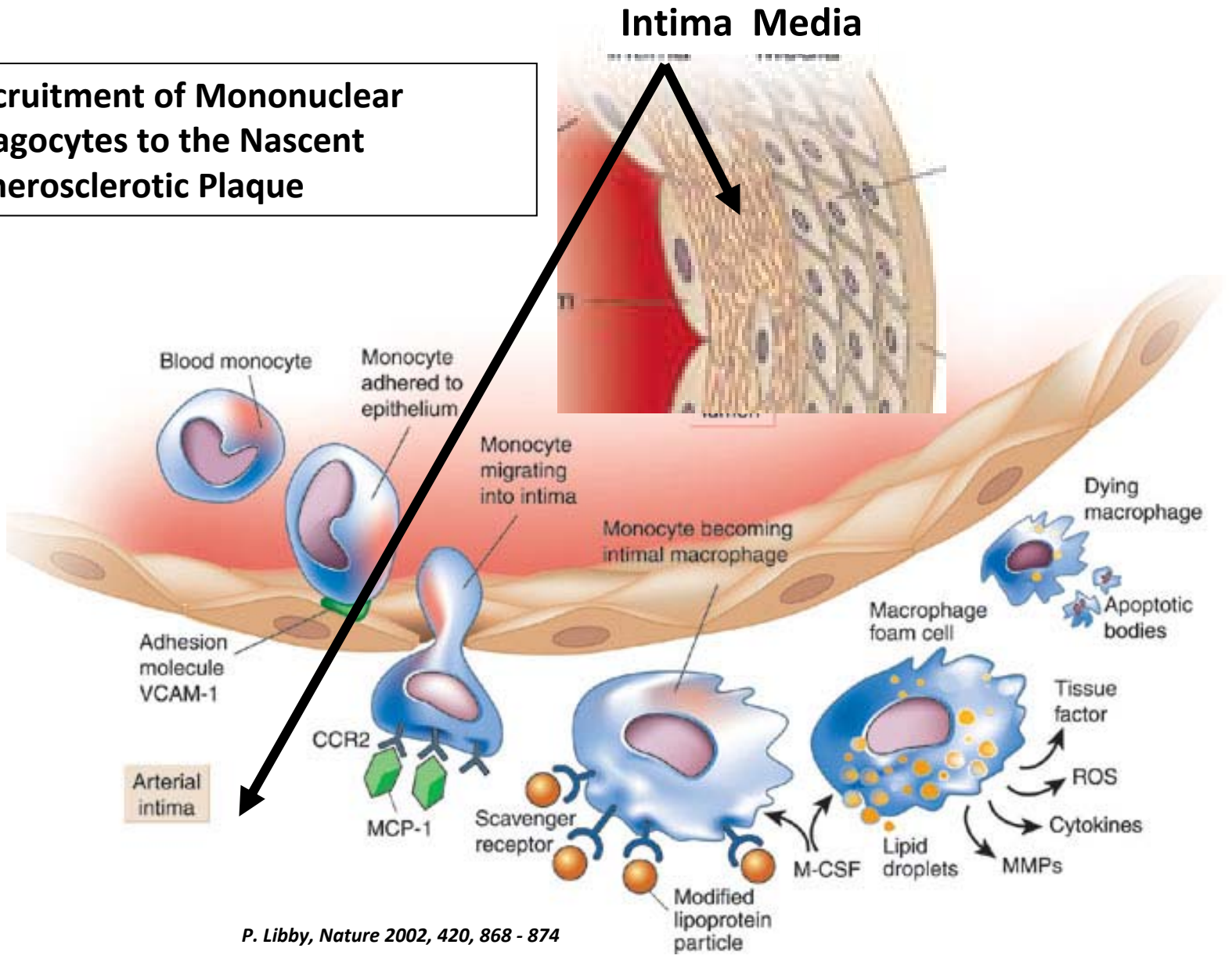
  - I Geen of sporadisch klachten
  - II Klachten bij zware inspanning
  - III Klachten bij lichte inspanning
  - IV Klachten in rust

Pathofysiologie



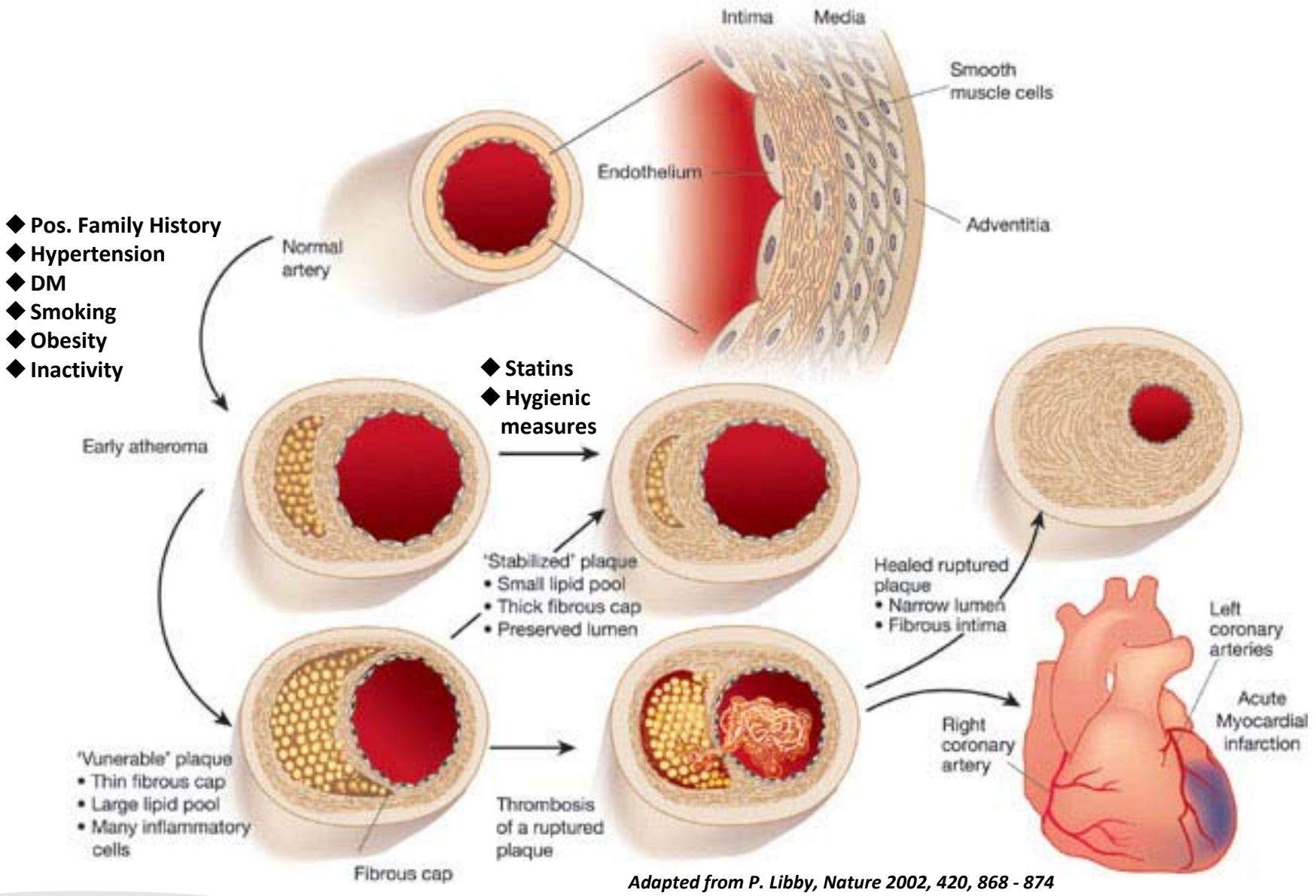
Inflammatory Mechanisms Couple Dislipidaemia to Atheroma Formation

Recruitment of Mononuclear Phagocytes to the Nascent Atherosclerotic Plaque



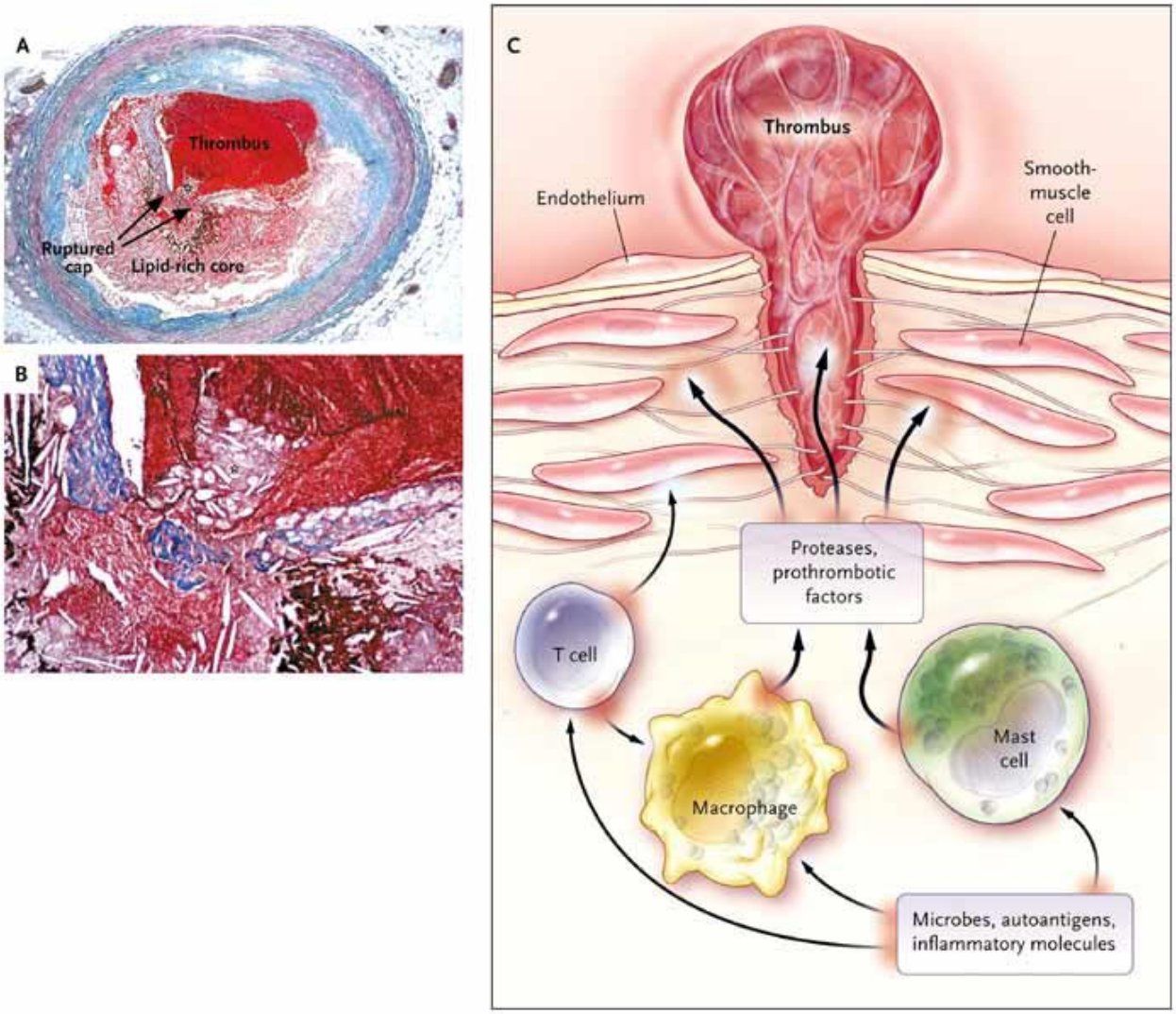
P. Libby, Nature 2002, 420, 868 - 874

Schematic of the Life History of a Human Coronary Atheroma



Adapted from P. Libby, Nature 2002, 420, 868 - 874

Atherosclerotic Lesion in a Human Artery



N Engl J Med 2005; 352:1685-1695

Pathofysiologie

Voornaamste oorzaken AP:

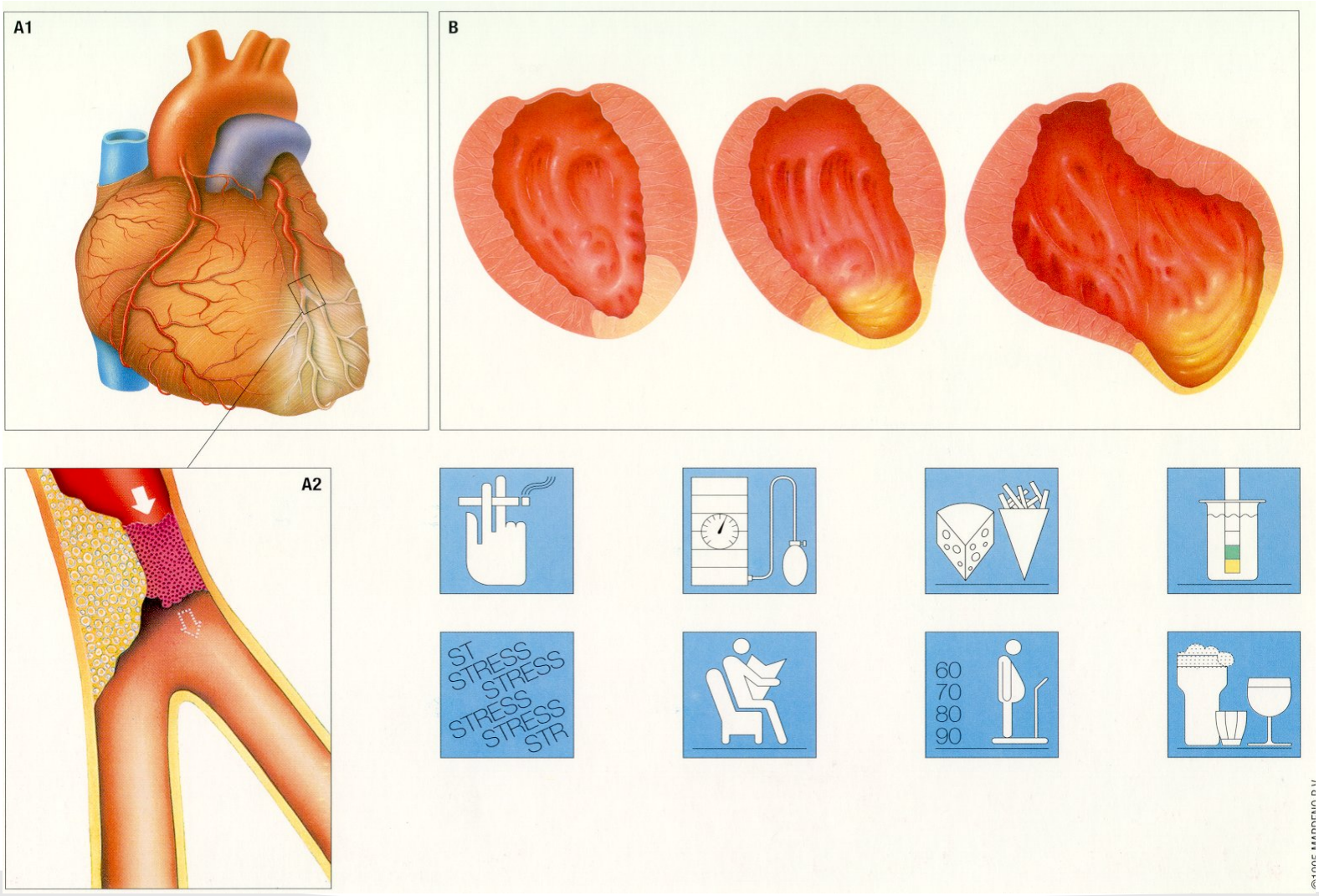
- Atherosclerotische stenose (fixed / stable plaque)
- Coronair spasme (atherosclerotische plaque)

Pathofysiologie

Overige oorzaken AP:

- Refractair coronair spasme (cocaïne, allergische reactie)
- CO-intoxicatie
- Anemie (!)
- Hypoxaemie door welke oorzaak dan ook

Risicofactoren



Klachten en symptomen

Pijn op de borst:	- drukkend/snoerend/bandvormig
	- uitstraling (li>re) arm of kaak
	- inspanningsgebonden
	- goede reactie op NTG
Dyspnoe	- als POB equivalent
	- inspanningsgebonden
	- Als gevolg van pompfalen
Vegetatieve verschijnselen?:	- Vrijwel nooit

Klachten en symptomen

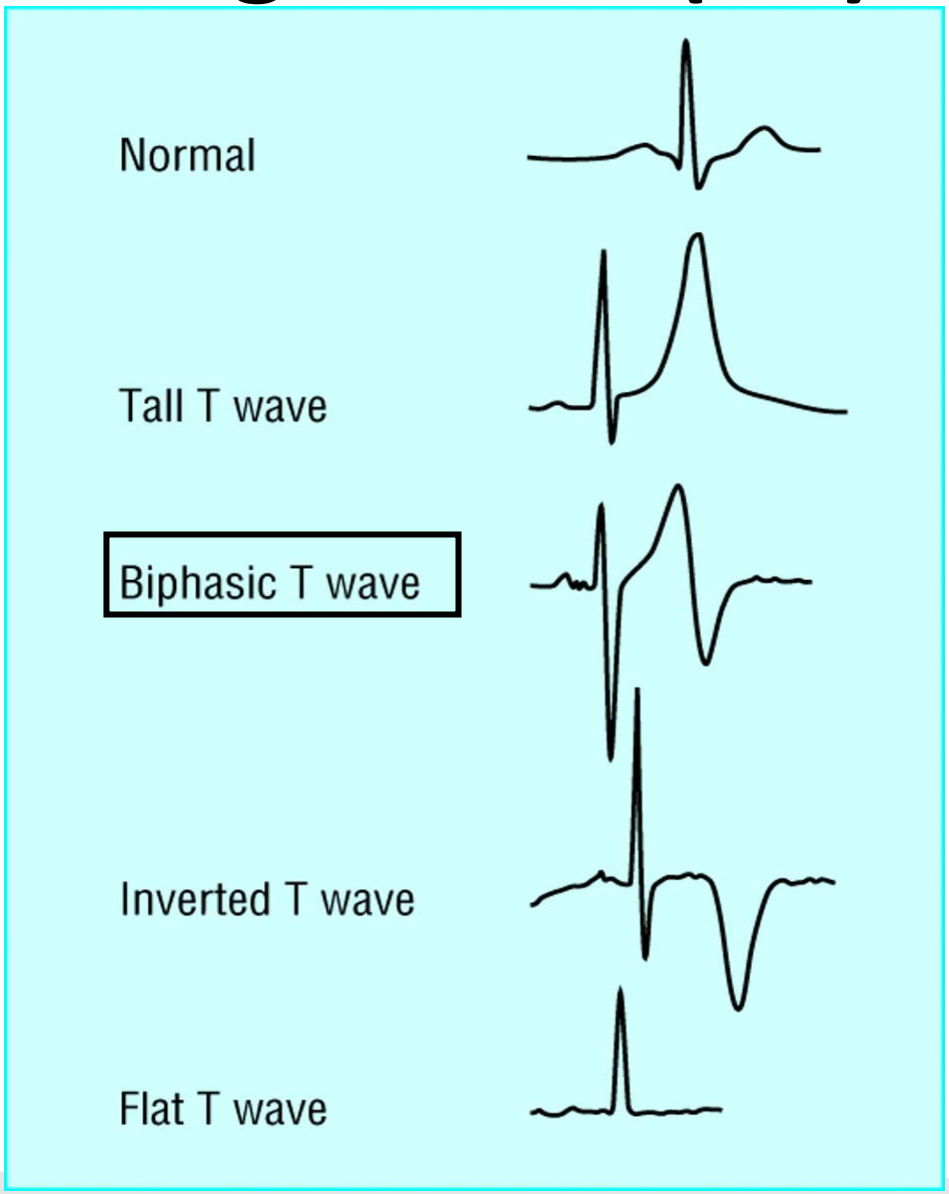
Complicaties bij AP:

- Ritme/geleidingstoornissen
- Verminderde LV functie door ischemie. Decompensatio cordis → *CAVE* Asthma cardiale (m.n. bij patiënten met een reeds pre-existent gecompromitteerde LV functie.)

Diagnostiek

- De Anamnese is de hoeksteen van de diagnostiek bij AP!! (inspanningsgebonden POB?)
- ECG (ST elevaties/depressies/T-top inversie)
- Lab: uitsluiten ACS/AMI (Troponine T/I, CK-MB etc.)

Diagnostiek (AP)

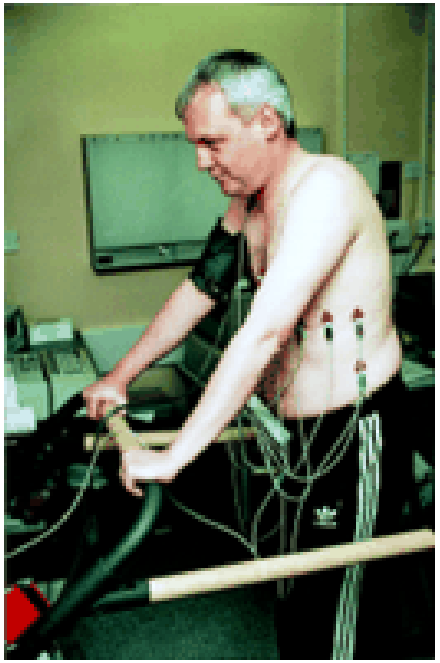


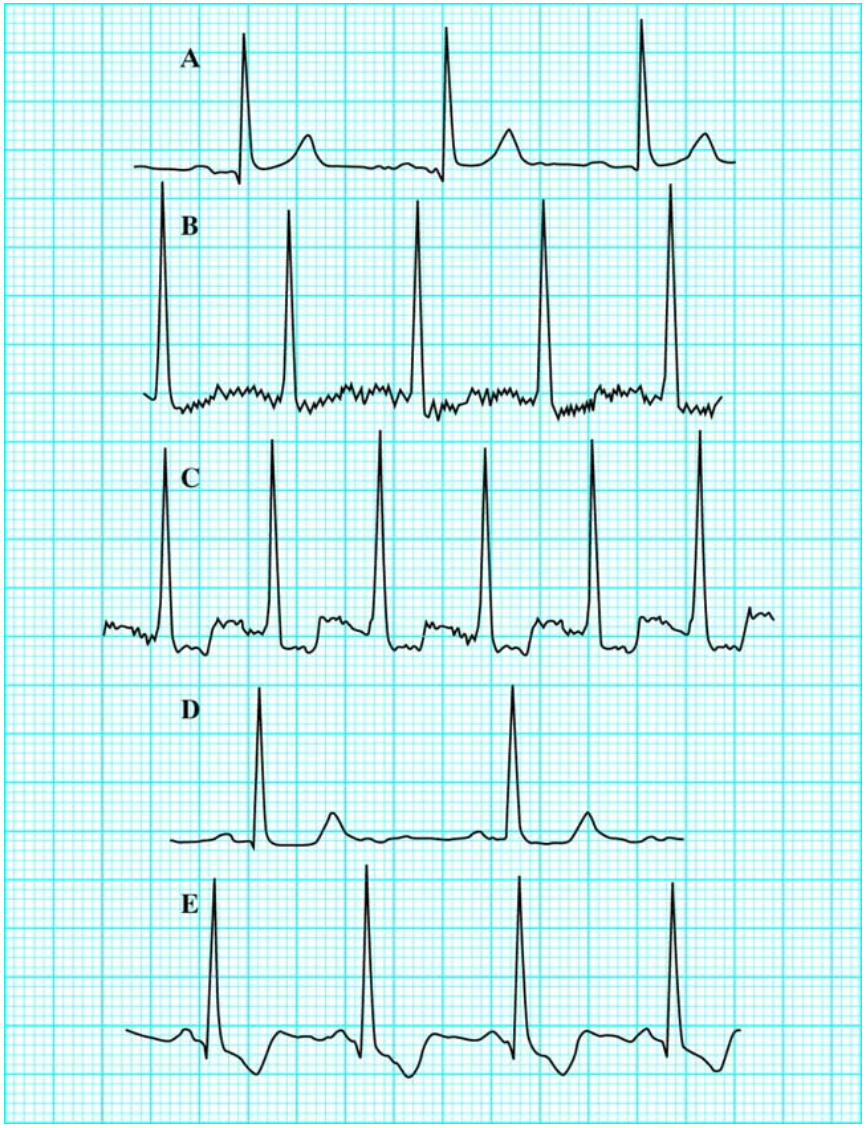
Diagnostiek

Polikliniek

- **Anamnese** (inspanningsgebonden POB)
- **ECG**
- **Inspanningstesten (uitlokken klachten)**
    - **X-ECG**
    - **MIBI scan**
    - **dobutamine echo**

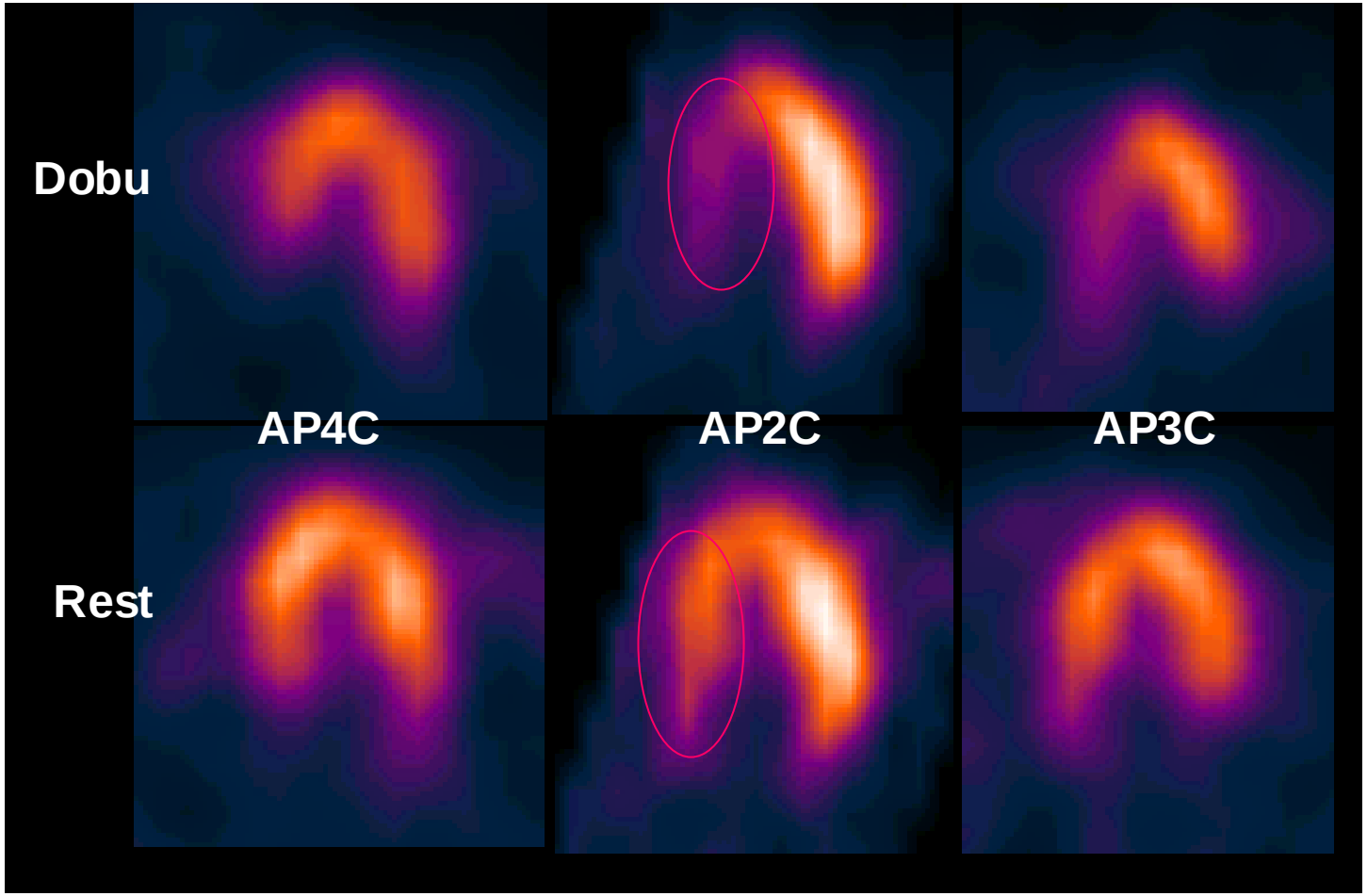
Diagnostiek





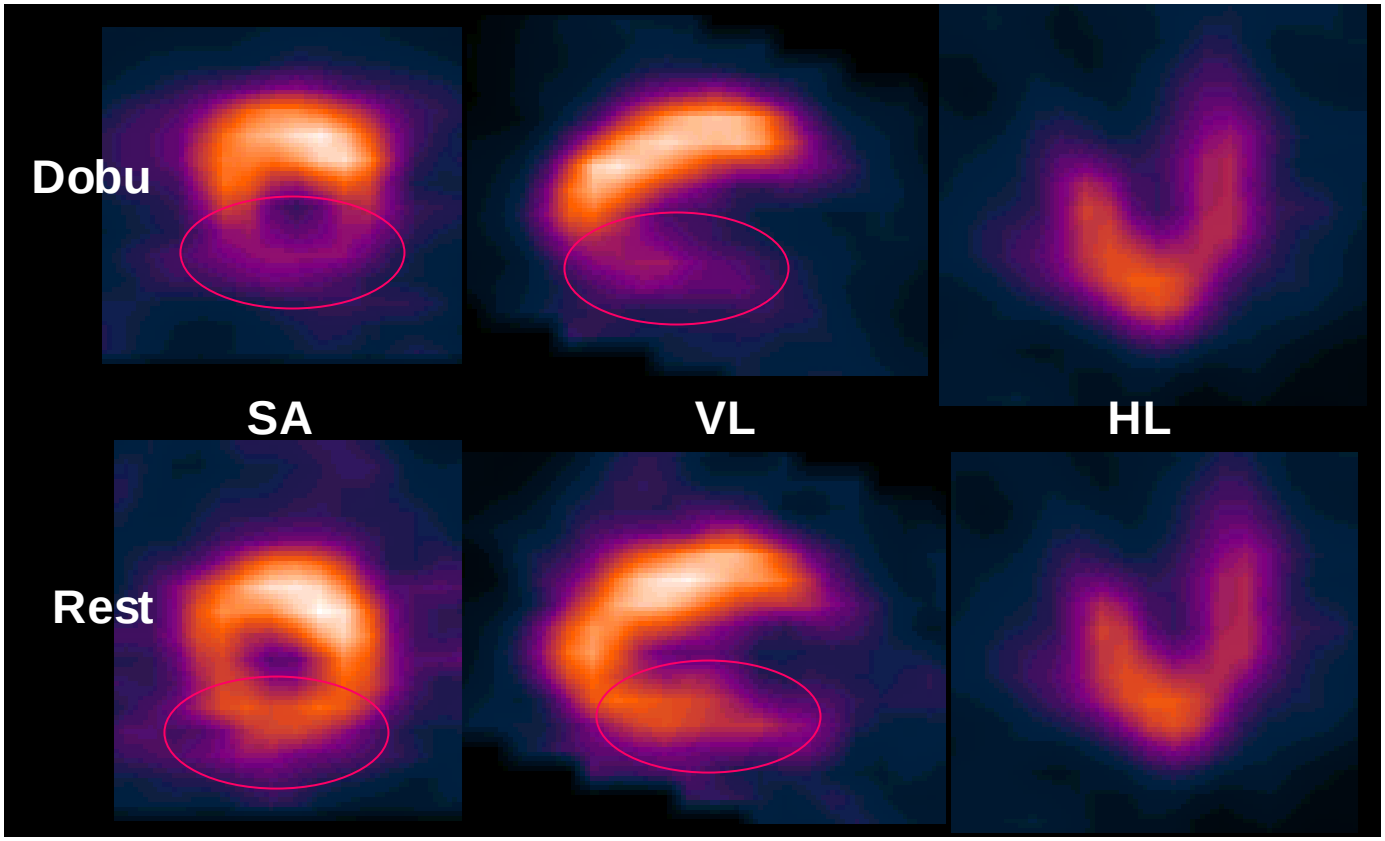
Diagnostiek

Dobutamine/inspannings



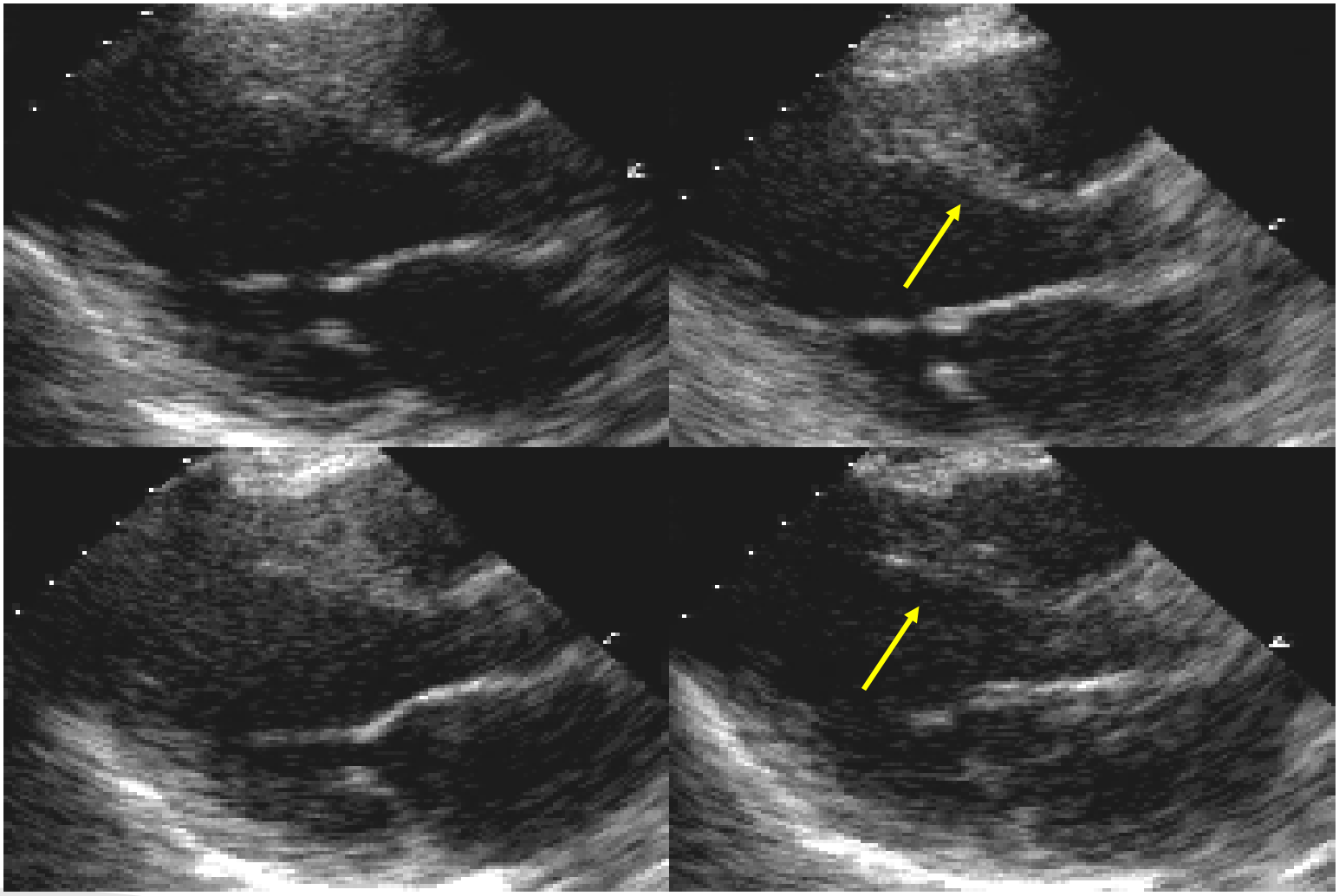
Diagnostiek

Dobutamine/inspannings



Diagnostiek

- **Dobutamine stress echocardiogram**



Therapie

- **Doel therapie**

  - optimaliseren balans tussen **demand** en **supply**

- **Verbeteren supply**

- voorkomen thrombusvorming op stenose
  - ASA(Ascal)/Clopidogrel(Plavix) remt plaatjesaggregatie
- 'remmen' progressie stenose(s)
  - Statine (cholesterol ↓, verbeteren endotheeldysfunctie)
- voorkomen vaatspasmen met Ca-antagonisten

Therapie

- **Verminderen demand**

- vertragen hartfrequentie: beta-blokkade

- bestrijden hypertensie: nitraten, beta-blokkade, ACE

- verminderen contractiliteit: beta-blockade, nitraten (preload ↓)

Therapie

- **Refractaire AP (medicatie schiet tekort)**

  Revascularisatie procedures:

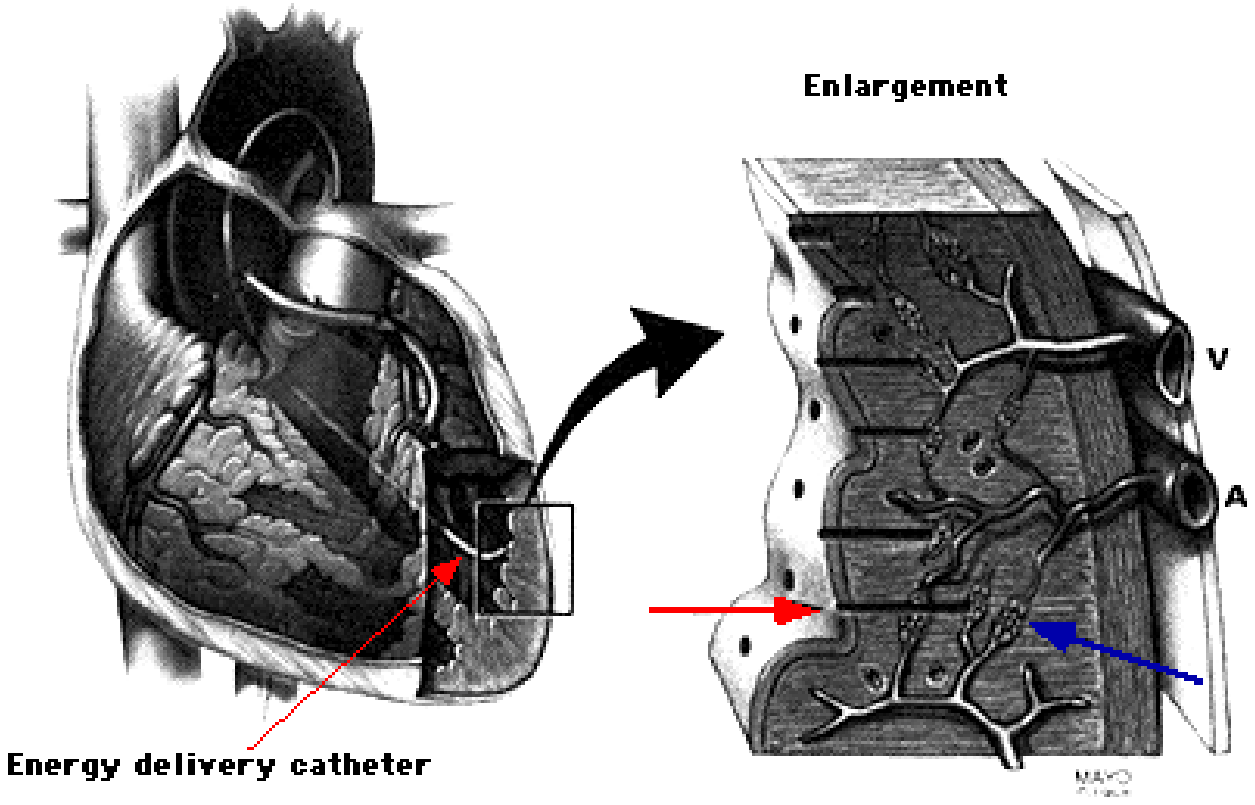
  - PTCA
  - CABG

- **Revascularisatie onmogelijk**

  - anatomie coronairen accepteren en evt. pijn behandelen
  - Angiogenese
  - Transmyocardiale laser revascularisatie (TMLR)
  - TENS (transcutane electrische neurostimulatie)
  - Medicatie toevoegen : bv. Vastarel, Nicorandil, etc.

Therapie

- **transmyocardiale laser revascularisatie (TMLR)**



Dus....

- Myocardinfarct
- Non-Q-wave infarct
- Instabiele Angina pectoris

3 uitingen van in principe dezelfde ziekte

Coronarialijden

Risicofactoren voor atherosclerose

- Leeftijd
- Mannelijk geslacht
- Positieve familie anamnese
- Verhoogd cholesterol
- Diabetes Mellitus
- Overgewicht
- **Roken!**

Klachten

- Drukkende pijn op de borst
- Uitstralend naar de kaak of linker arm
- Bij IAP toename bij inspanning
- Niet houdingsafhankelijk
- Neemt niet toe bij druk op thorax
- Misselijkheid, braken, transpireren
- Bij IAP reactie op NTG
- Benauwdheid (bij decompensatie)
- Collaps (bij ritmestoornissen)

Lichamelijk onderzoek

- Bleek, zweterig (met name bij infarct)
- Doodsangst (met name bij infarct)
- Bloeddruk vaak afwijkend
- Pols kan afwijkend zijn (ritmestoornis)
- Koorts na infarct (38º)
- Gewichts toename (bij decompensatie)

Beleid:

- NTG (iv)
- Ascal
- Heparine
- Telemetrie / CCU
- Bij infarct: eventueel trombolyse
  eventueel Primaire PTCA
- Ischaemie detectie

Beleid:

Preventie!!!!

- Stoppen met roken
- Minder vet eten
- Bij DM suiker goed reguleren
- Afvallen
- Medicatie: Simvastatine
  Ascal
  B-blokker

Myocardinfarct

Definitie

Acuut myocard infarct

Acute en irreversibele beschadiging van (een deel van) de hartspier ten gevolge van het tekort schieten van de bloedtoevoer, leidend tot necrose en verlittekening van het desbetreffende deel van het myocard.

Pathofysiologie

- **Oorzaken AMI**

- meestal thrombotische occlusie t.g.v. plaque ruptuur

  $\rightarrow$ plaatjesaggregatie

  $\rightarrow$ vasoactiva $\rightarrow$ spasmen

  $\rightarrow$ thrombine $\rightarrow$ stollingsactivatie

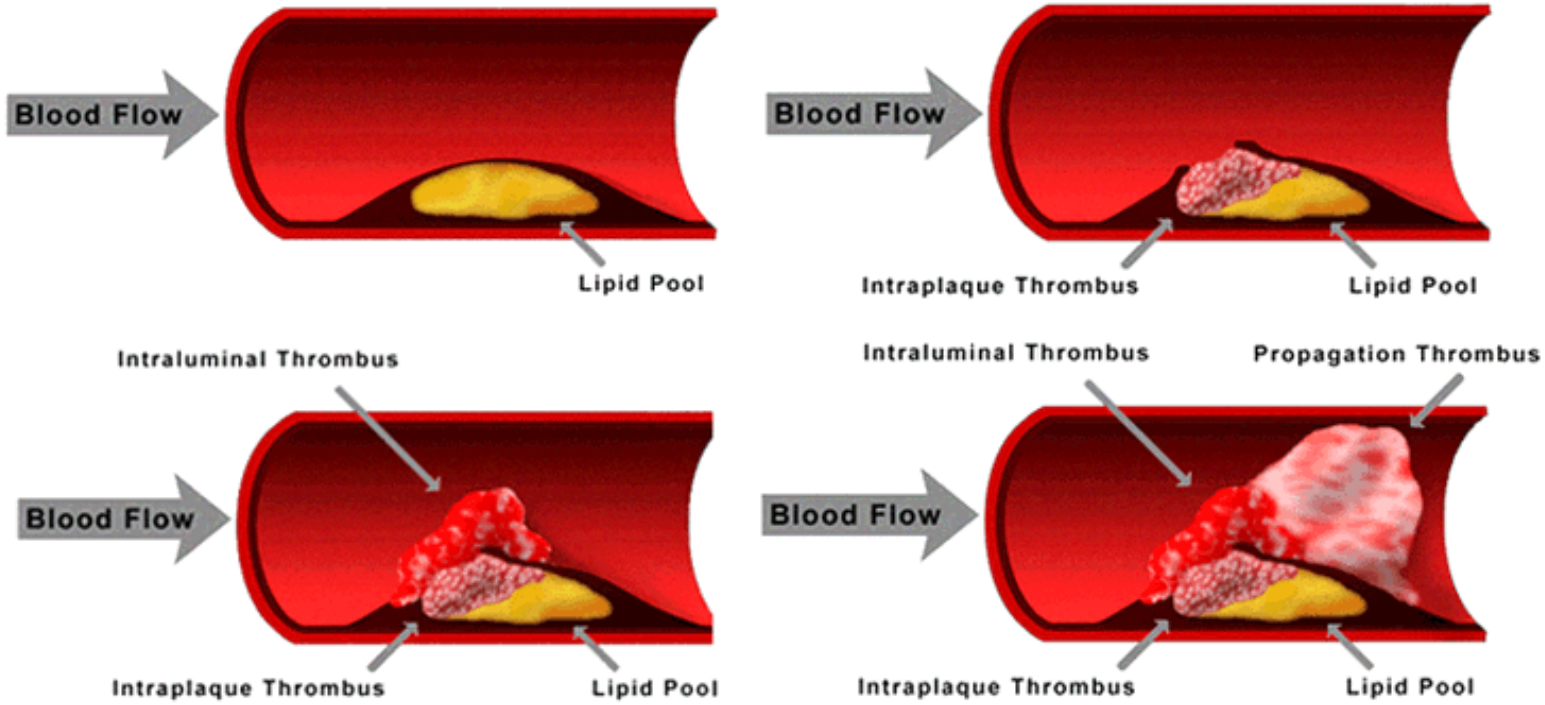
  $\rightarrow$ bevordering aggregatie

Pathofysiologie

- **Oorzaken AMI**

- zelden:
  embolus (endocarditis!)
  coronair spasme (cocaine, allergische reactie)
  CO-intoxicatie
  anemie
  hypoxaemie

Pathofysiologie



Pathofysiologie

Grootte AMI

afhankelijk mate verstoring evenwicht demand-supply en hoe lang deze verstoring aanhoudt.

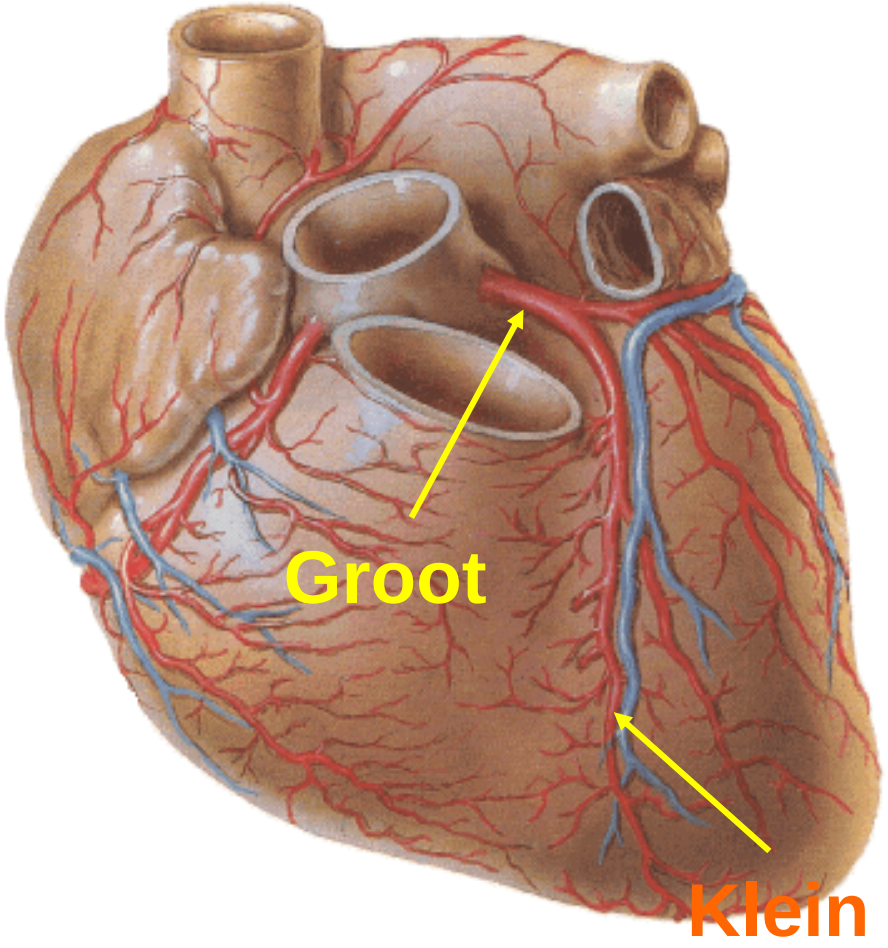
supply:

- plaats afsluiting
- duur afsluiting - irreversibel na 20 - 30 min ischemie
- collaterale doorbloeding
- (Hb, PO_2)

demand

- zuurstof behoefte myocard
- frequentie, RR, contractiliteit

Pathofysiologie



Pathofysiologie

- **Tijdsbeloop AMI**

begin infarcering: 20-30 min

volledig na ongeveer 6 uur

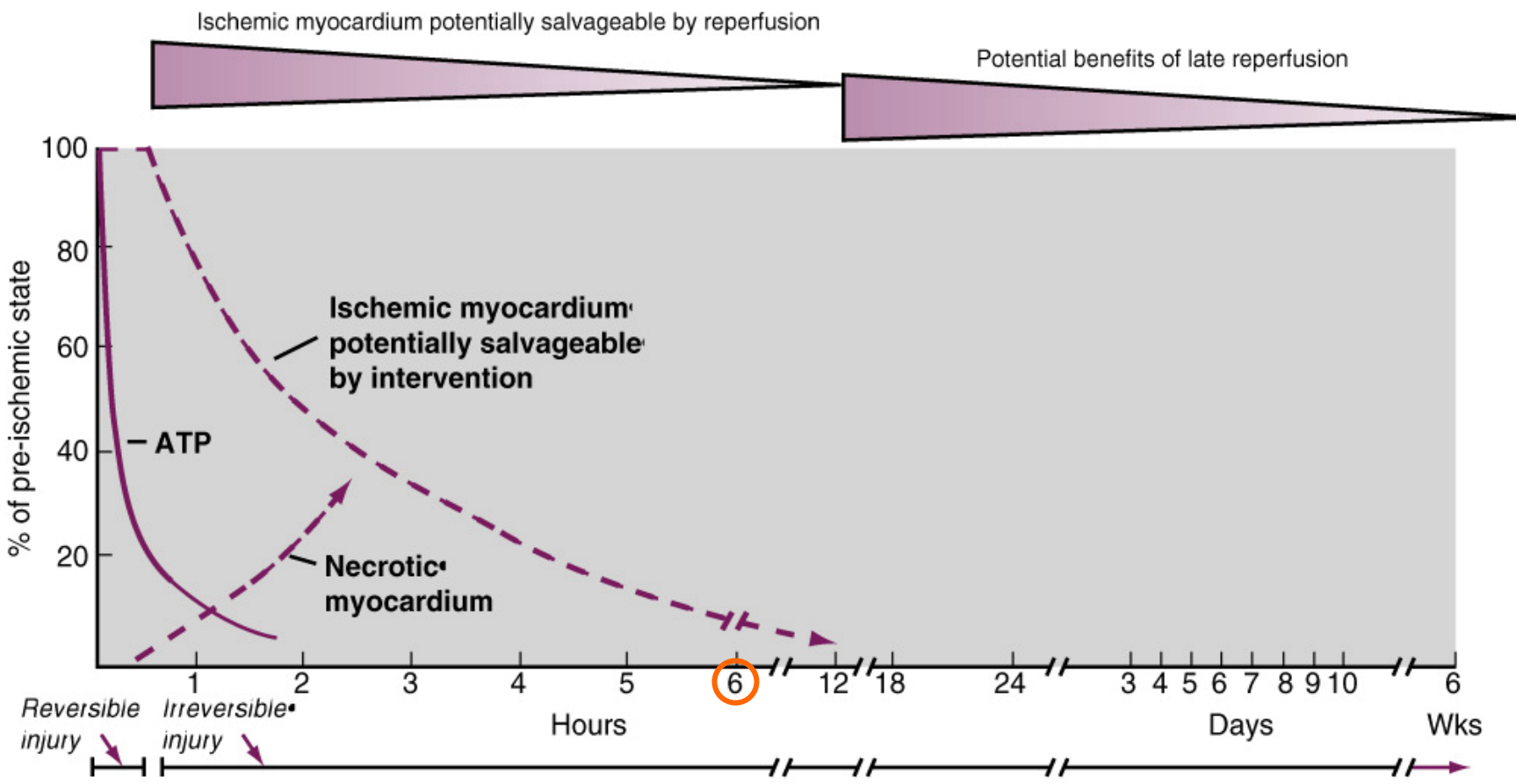
NB: stuttering infarction: - afwisselend occlusie/reperfusie

- geprotraheerd beloop

ischemie meest uitgesproken endocardiaal

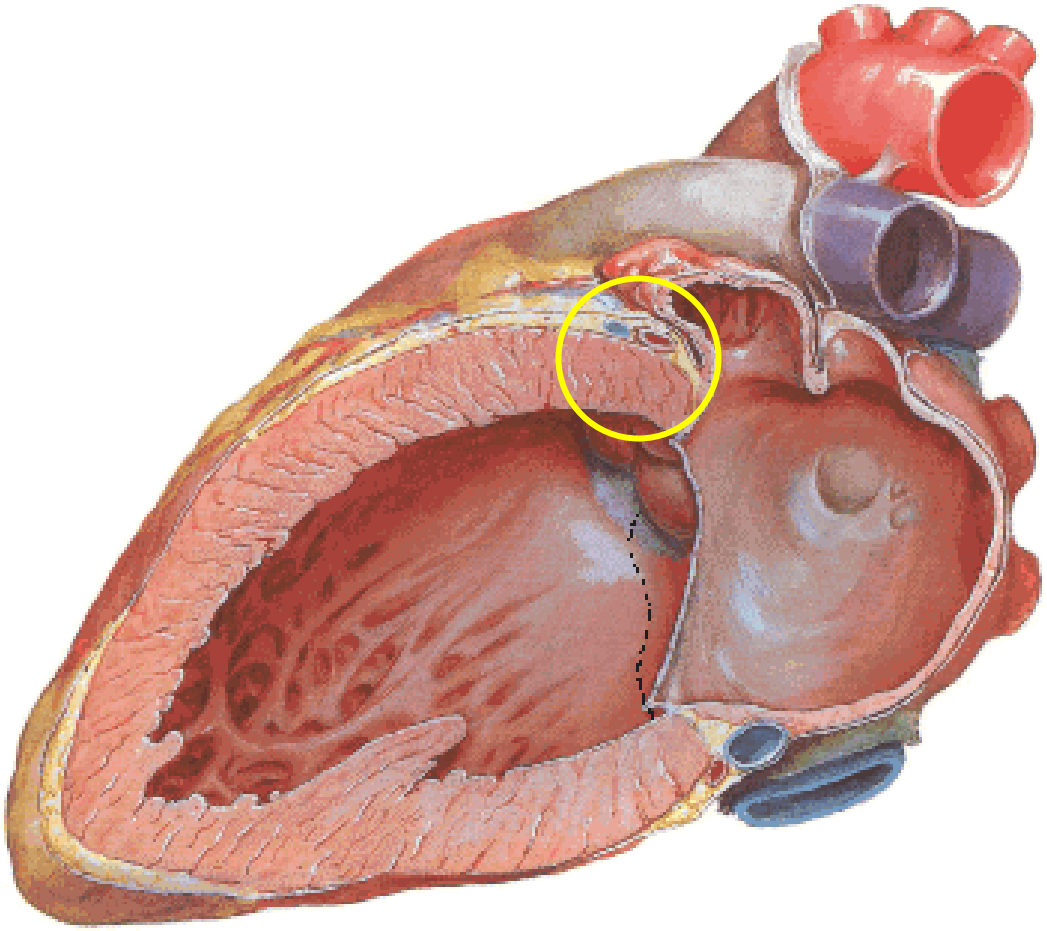
infarcering **endo** → **epicard** (uren)

Pathofysiologie



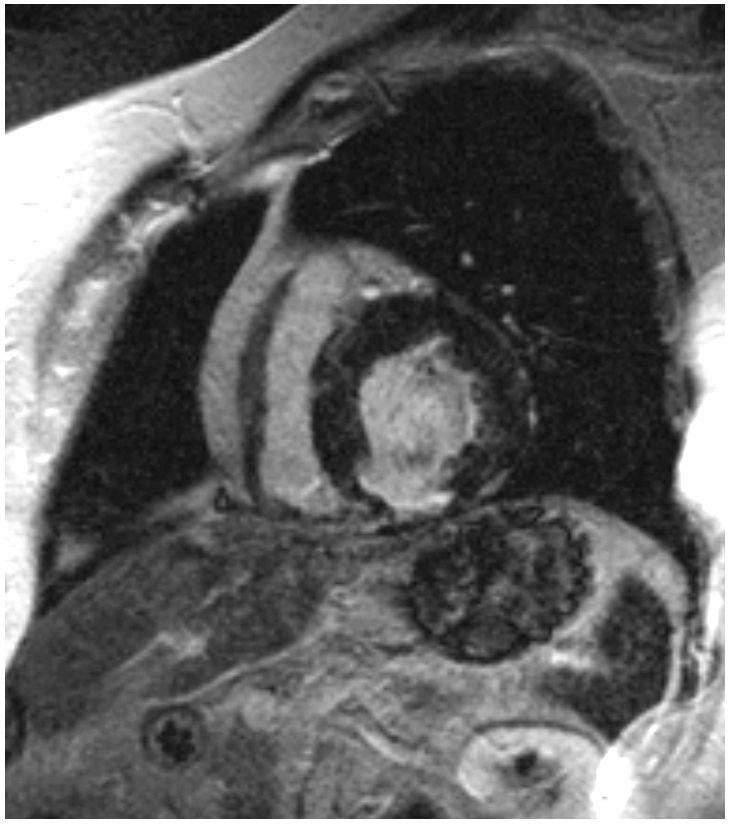
Pathofysiologie

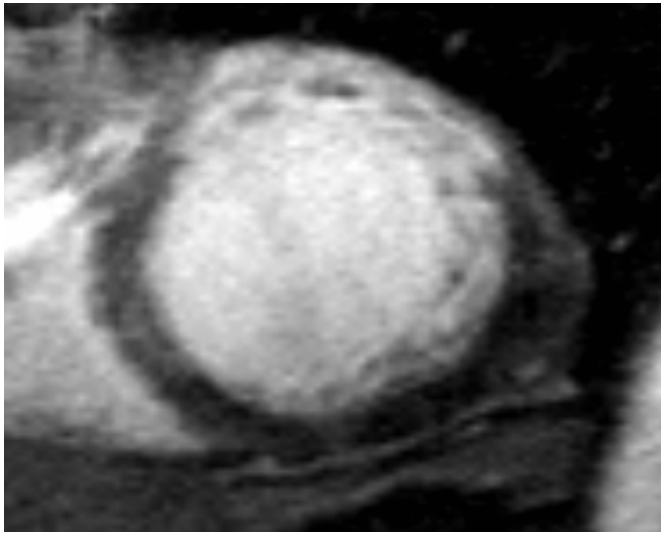
Coronairen van epicardiaal → endocardiaal

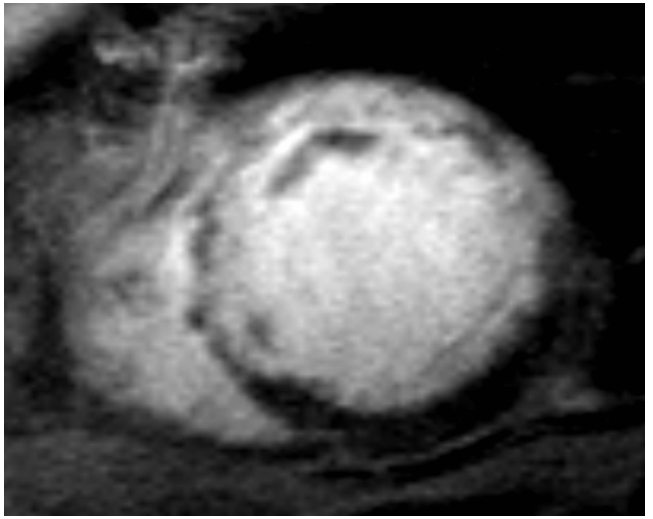


Pathofysiologie

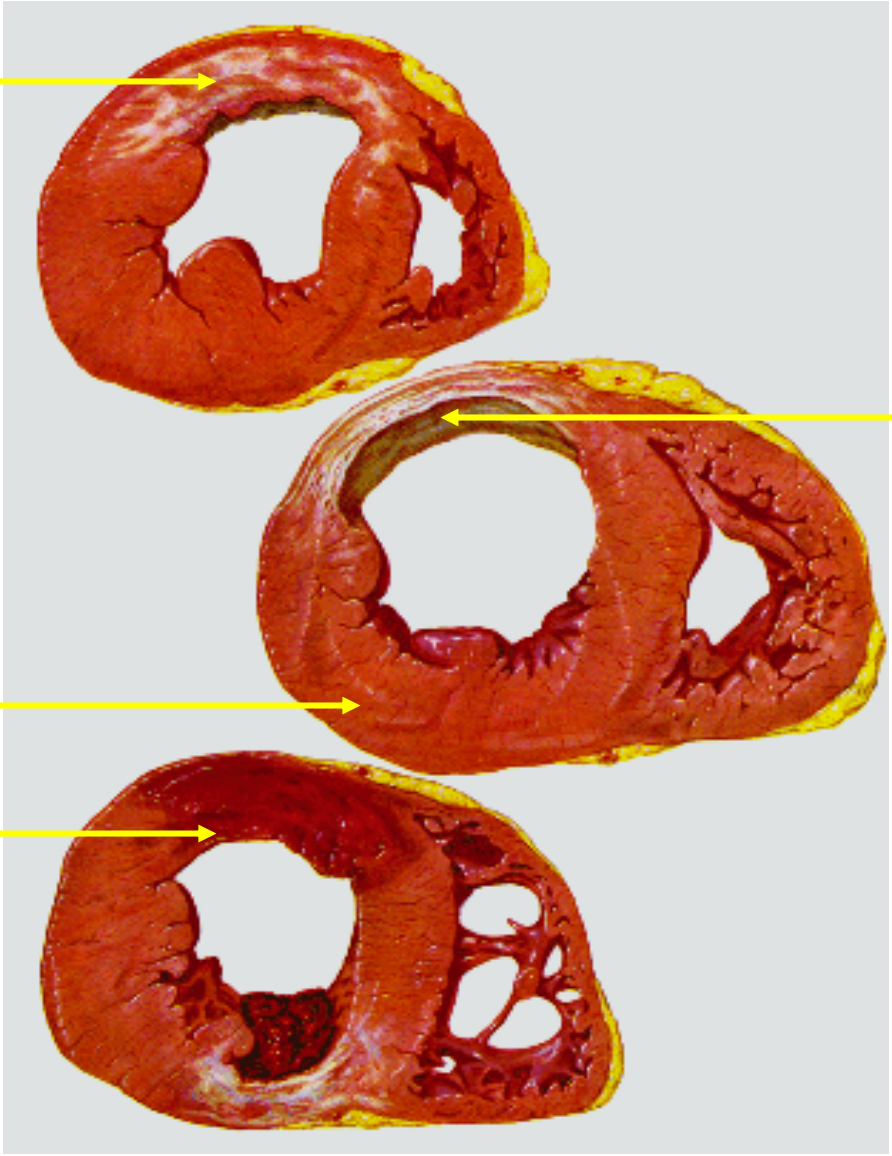
Transmuraal
Subendocardiaal



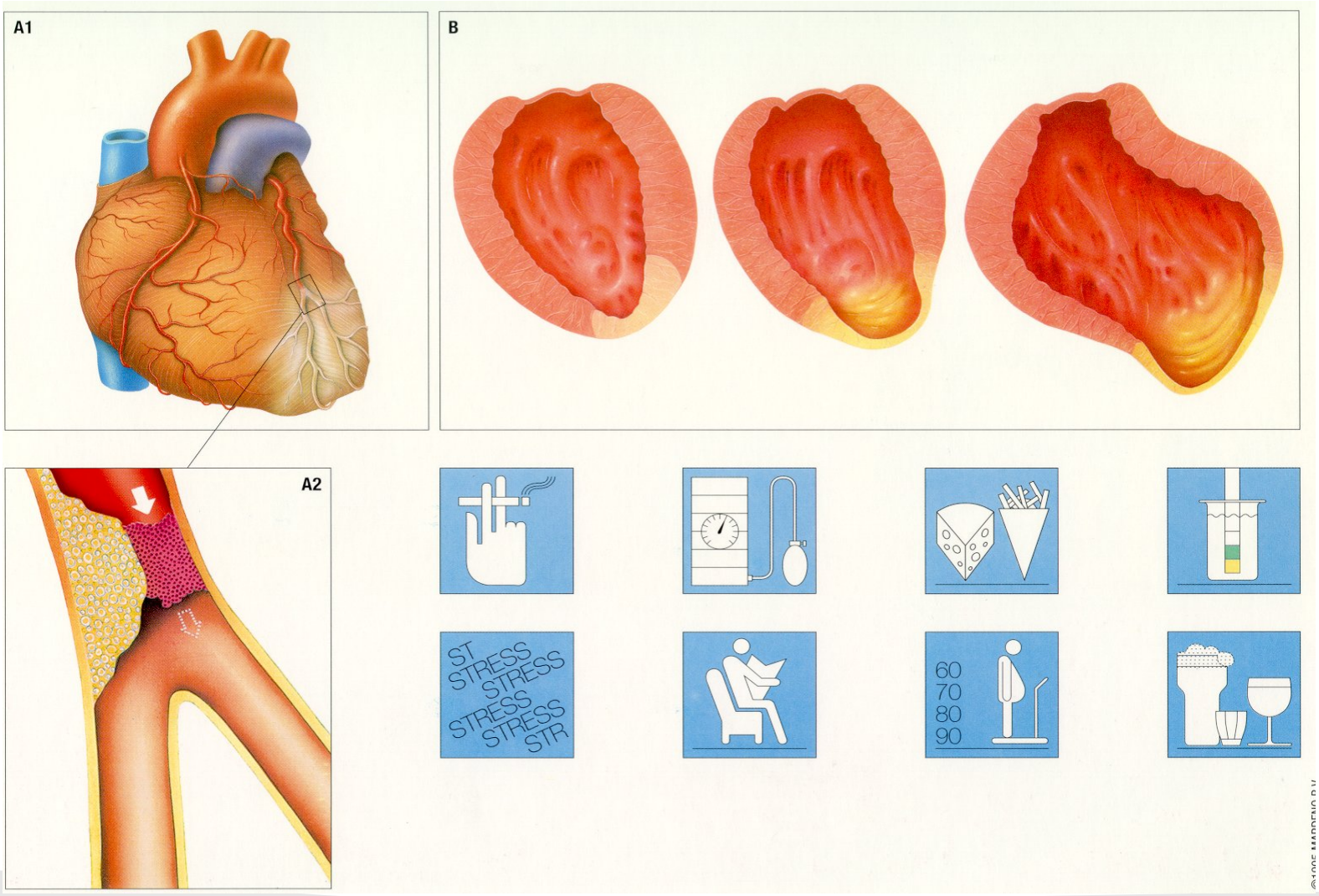




Pathofysiologie



Risicofactoren



Klachten en symptomen

- **Klachten door AMI zelf**

Pijn op de borst: - drukkend/snoerend/bandvormig
- uitstraling (li>re) arm of kaak
- in rust onstaan (!0400 AM)
- weinig of geen reactie NTG

Vegetatieve verschijnselen:
- misselijk, braken, transpiratie, gapen

Klachten en symptomen

Mogelijke gevolgen van AMI

Dyspneu:	backward failure
Shock:	forward failure
duizelig/collaps:	ritme-/geleidingsstoornissen (VF!)
CVA:	LV thrombus/AF

NB:

frequent geen (duidelijke) klachten → ouderen, diabetici!!

Diagnostiek

- **ECG bij ischemie:**

  - subendocardiaal: hoge symmetrische, spitse T's
    ST depressies
  - Transmuraal: negatieve T-top

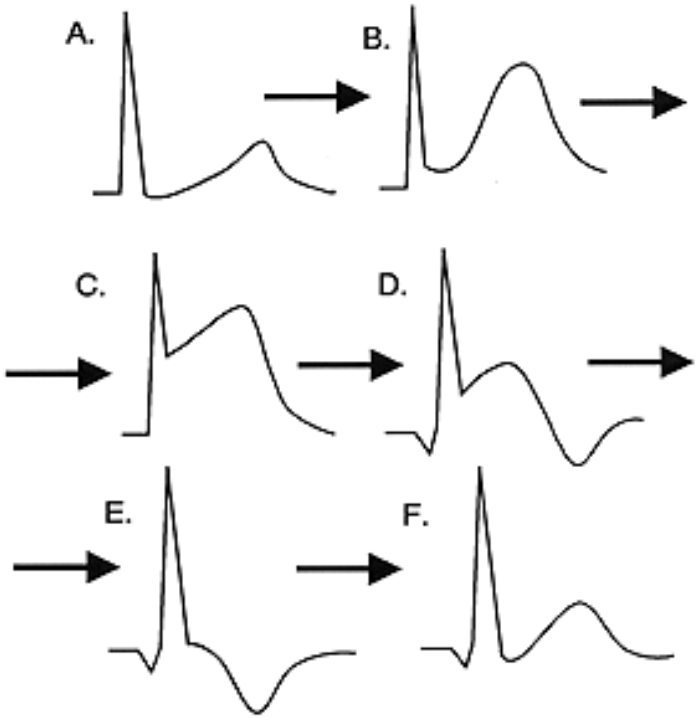
- **ECG bij (transmurale) infarcering**

  repolarisatie :

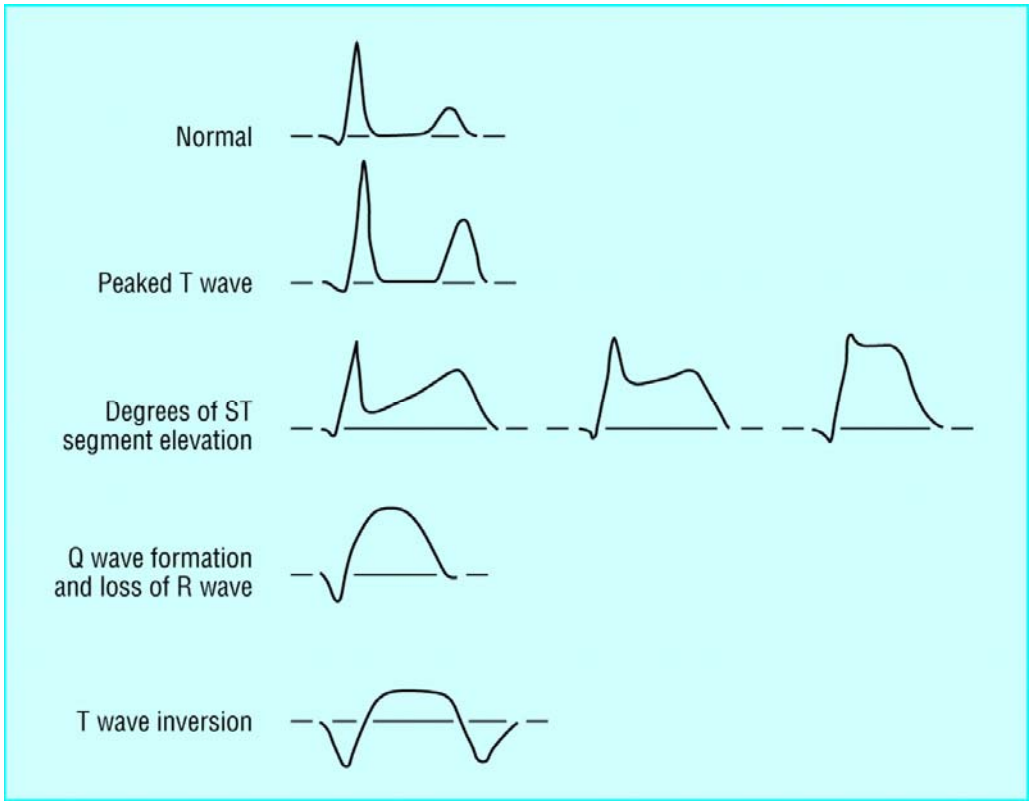
begin	ST-elevatie > 1mm;vaak reciproke depressie
	hoge, spitse positieve T
Later	afname ST elevatie en depressie, (ontstaan Q-golven),(terminaal) negatieve T toppen

Diagnostiek

- **ECG en AMI (evolutie)**



Evolution of Acute MI



Diagnostiek

- **AMI zonder ECG veranderingen**

  - klein AMI (non-Q-wave)
  - electrisch ‘stil’ gebied - RCx
  - groot circulair AMI - ‘cancellation’

Diagnostiek

- **ECG ischemie/infarct**

  Echter:

  - kan weer geheel normaliseren
  - pre-existente repolarisatie stoornis:
    - LBTB
    - pre-excitatie (WPW)
    - Ventrikel pacing

QRS complex:

- afname R toppen/ontstaan Q toppen

NB: Q-golf ***niet*** bewijzend voor irreversibiliteit

Diagnostiek

- **Grootte AMI/area at risk op ECG**

- som ST elevatie en depressie (‘totale ST-shift’)
- bepaalt prognose, agressiviteit therapie

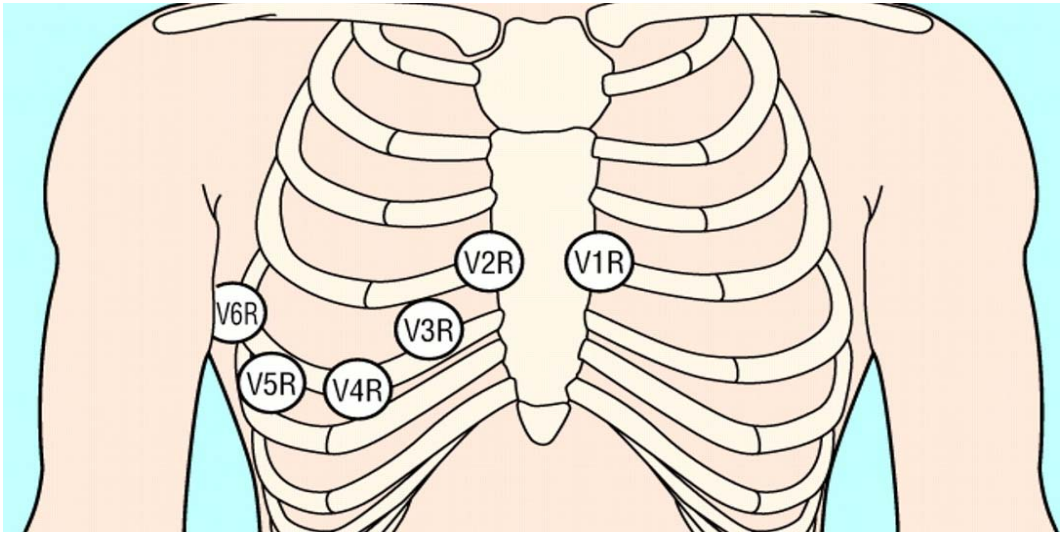
Diagnostiek

- **Localisatie AMI op ECG**

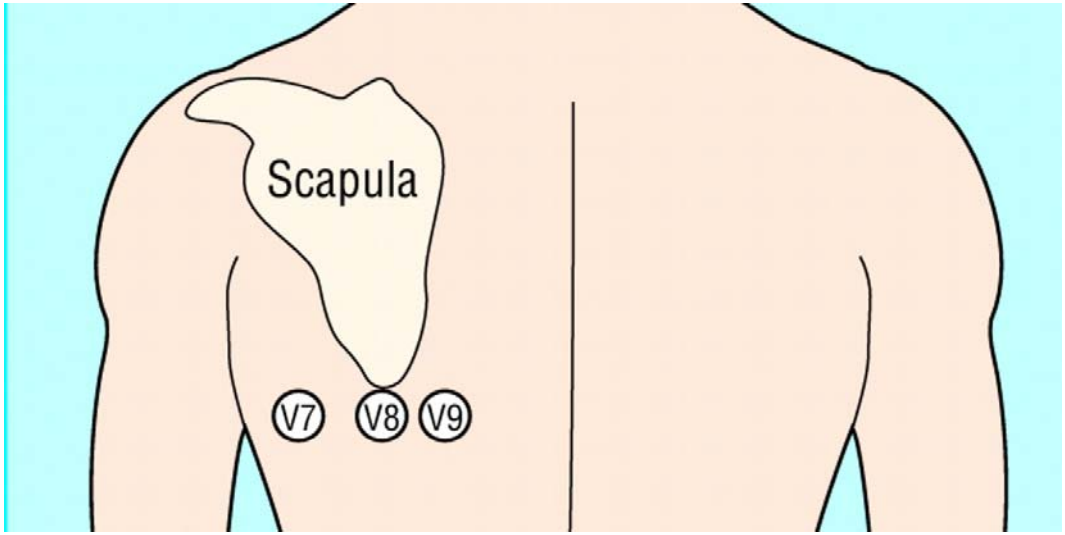
anterior infarct	
- anteroseptaal	ST↑ in V1-3,4
- zuiver anterior	ST↑ in V2-5
- anterolateraal	ST↑ in V4-6, I aVL
lateraal infarct	ST↑ in I aVL V6
hooglateraal infarct	ST↑ in I aVL
inferior infarct	ST↑ in II, III, aVF
posterior infarct	acuut: ST↓ in V1-3 chron: hoge/brede R en hoge T-top V1-2, ST↑ in V7-9
RV-infarct	ST↑ in V4R,V5R

Diagnostiek

RV leads

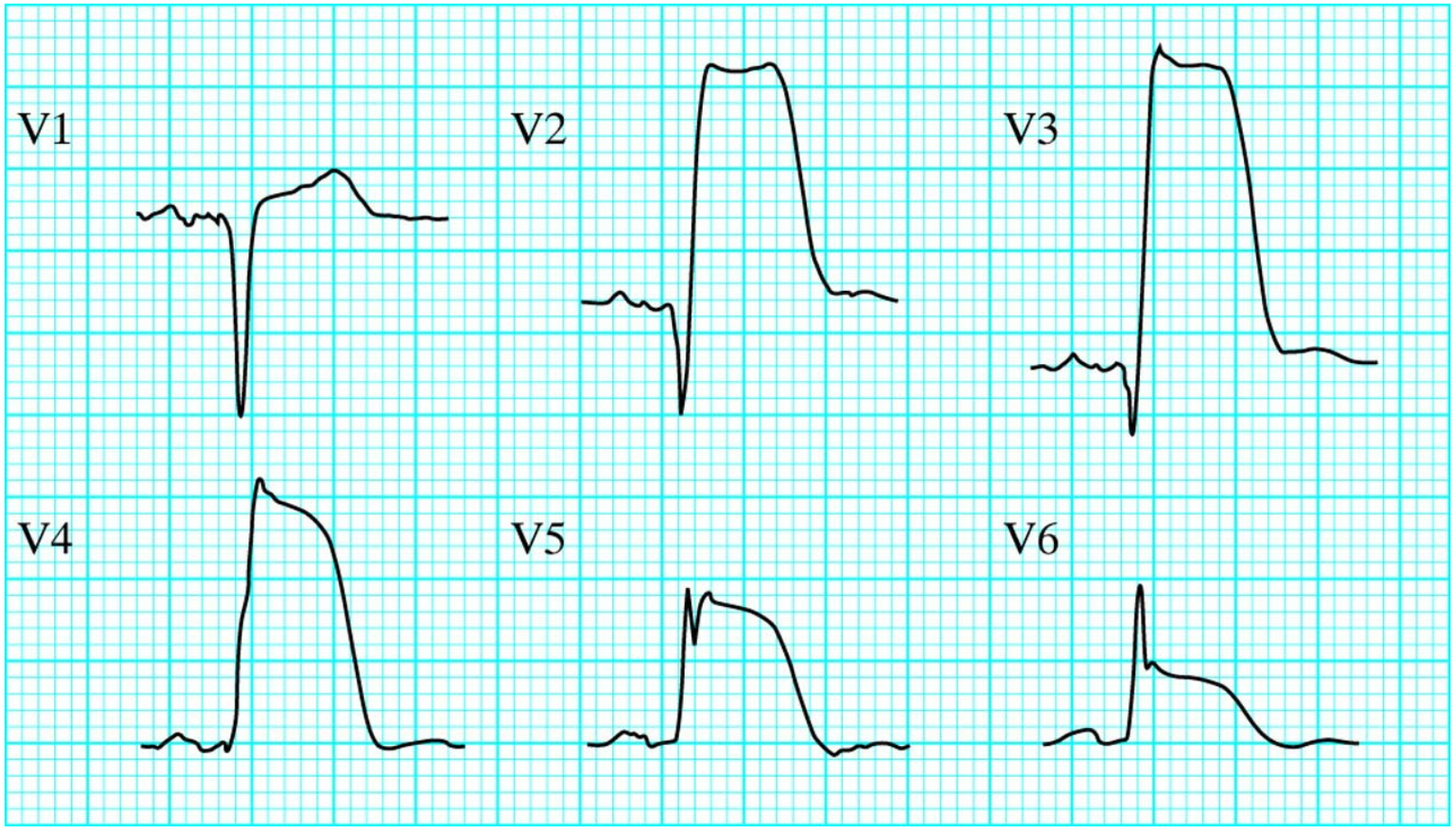


Posterior leads



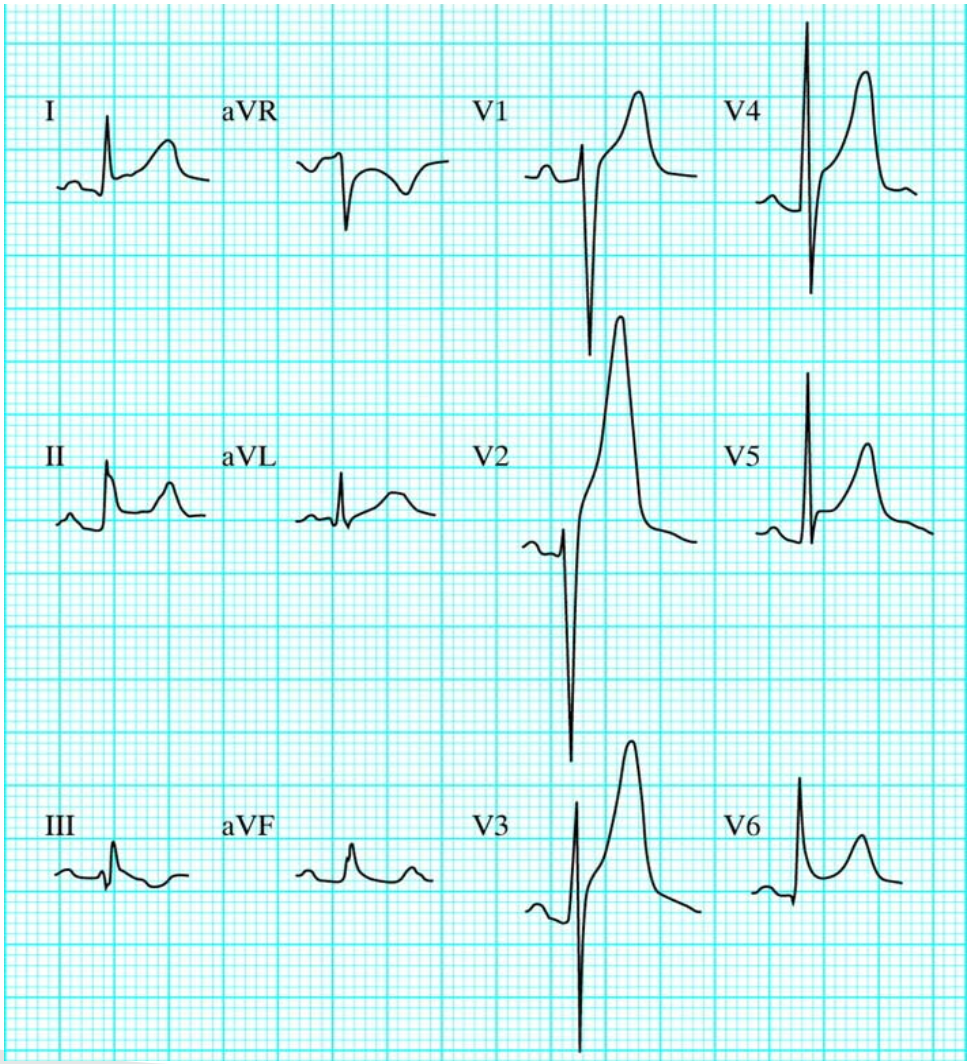
Diagnostiek

Anterior



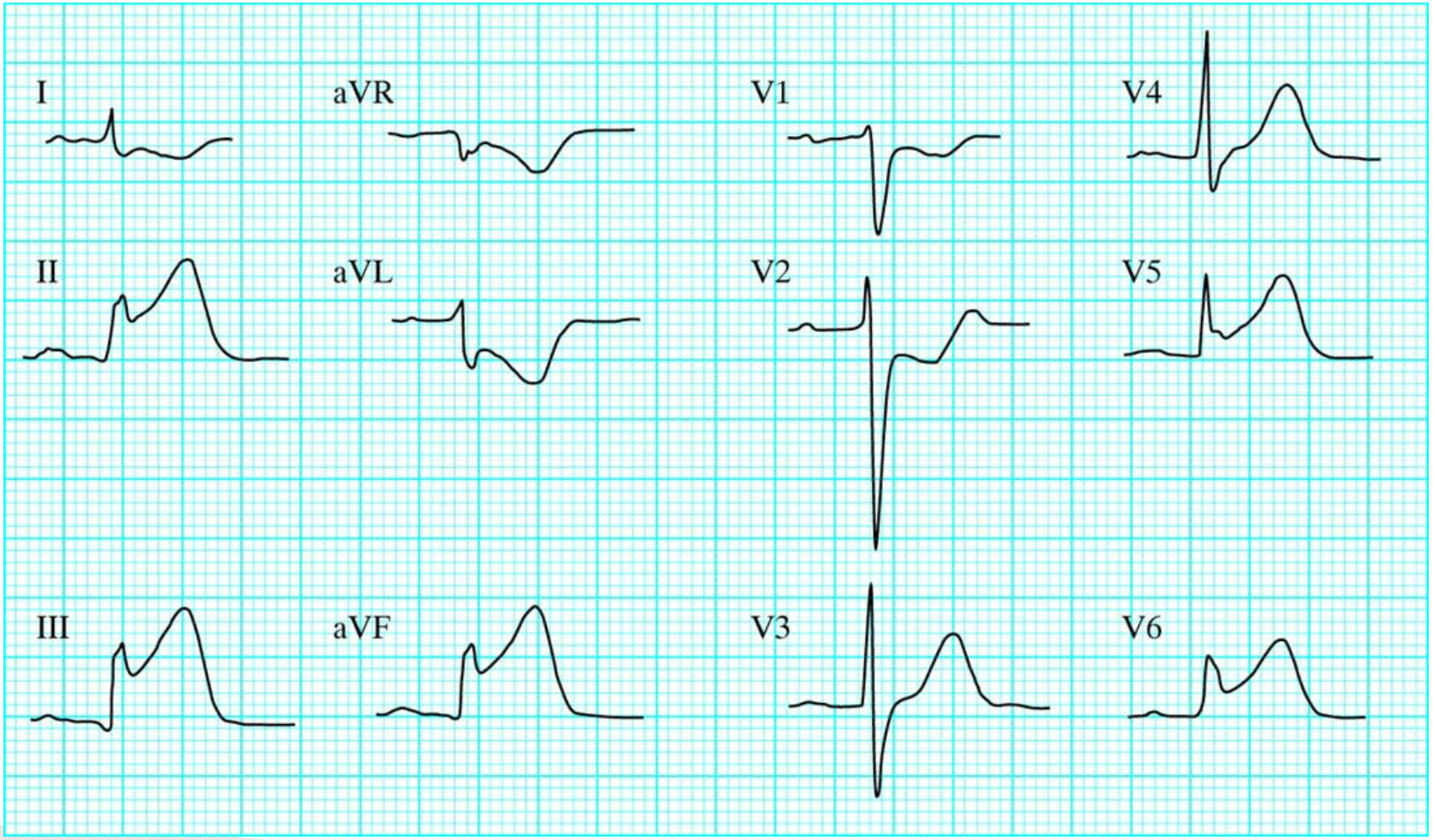
Diagnostiek

Anterolateraal infarct



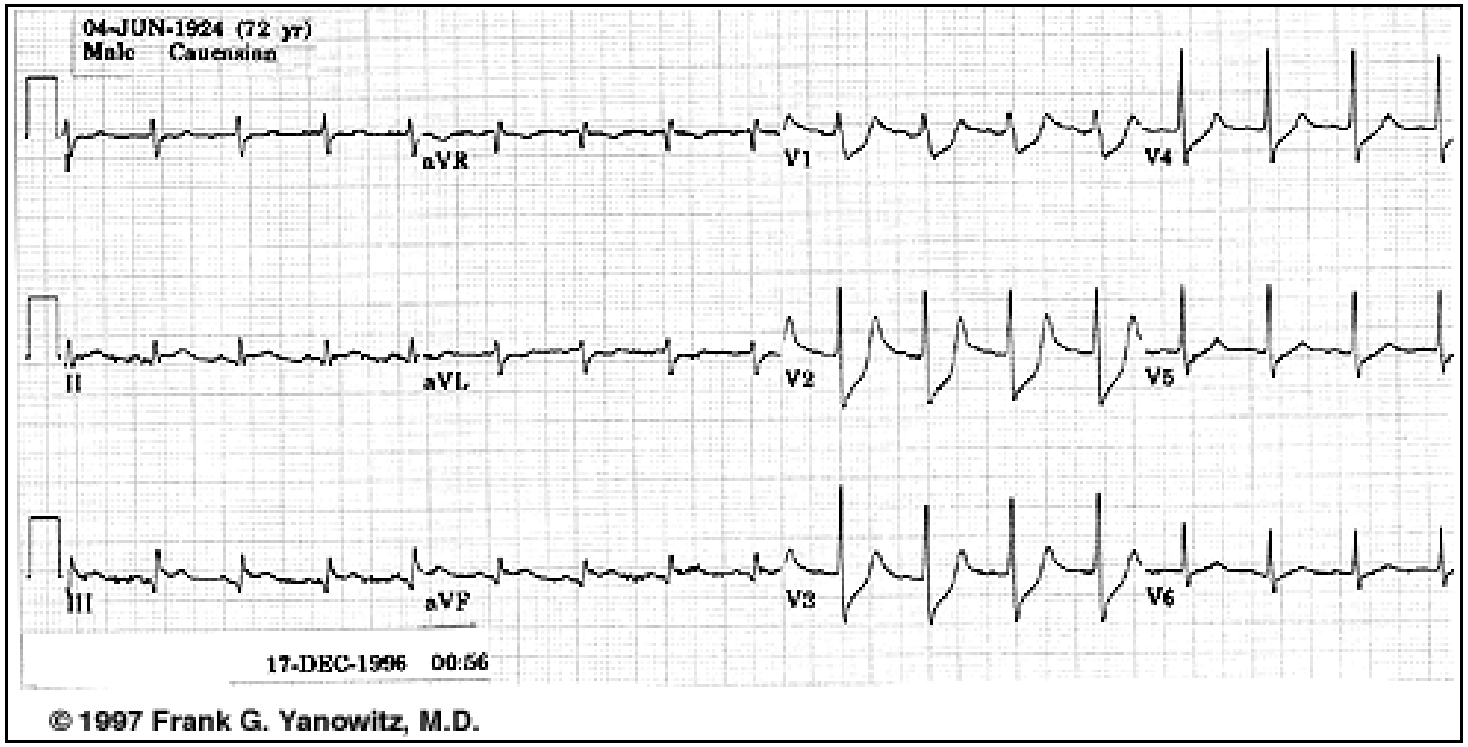
Diagnostiek

IPL infarct



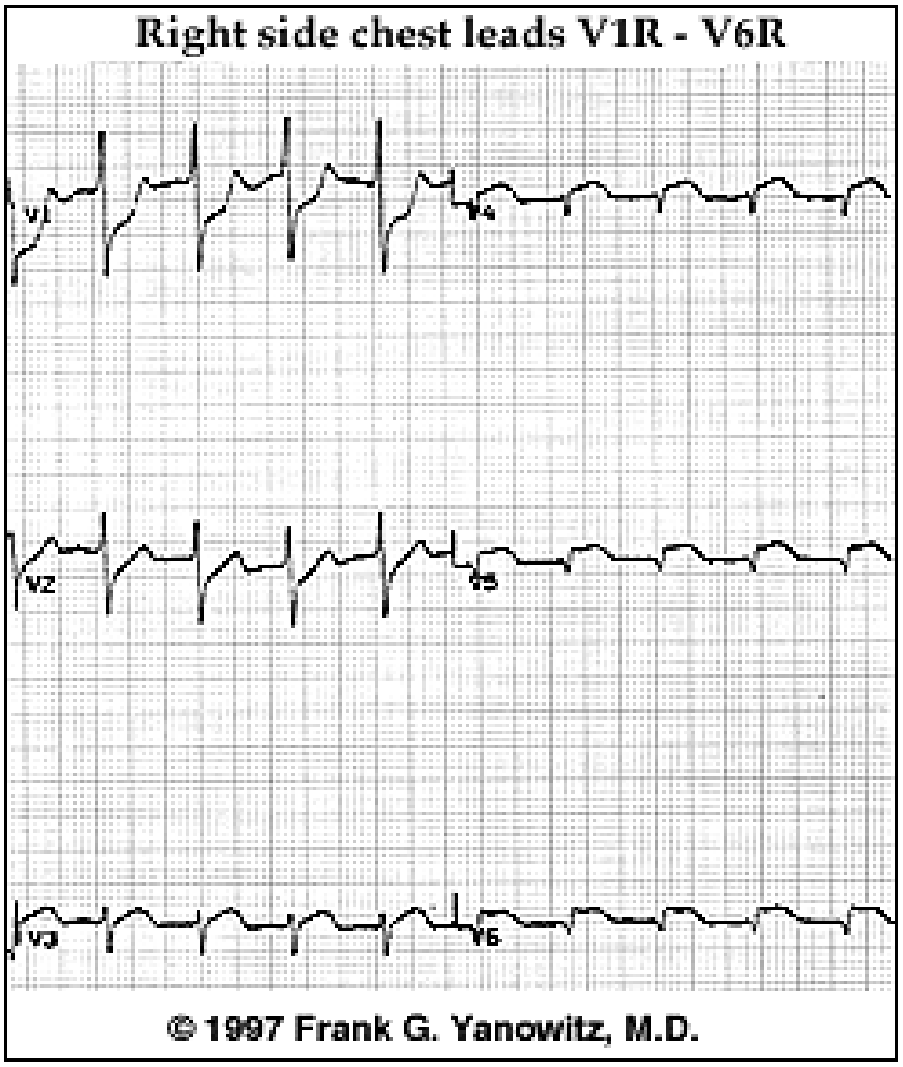
Diagnostiek

‘True’ posterior infarct



Diagnostiek

RV infarct



Diagnostiek

- **Enzymbepalingen**

Infarct = necrose

Integriteit celmembraan verloren $\rightarrow$ intracellulaire stoffen ontsnappen naar extracellulair

biochemische markers van **infarcering**

Diagnostiek

- **biochemische markers**

CK/creatine kinase: aspecifiek

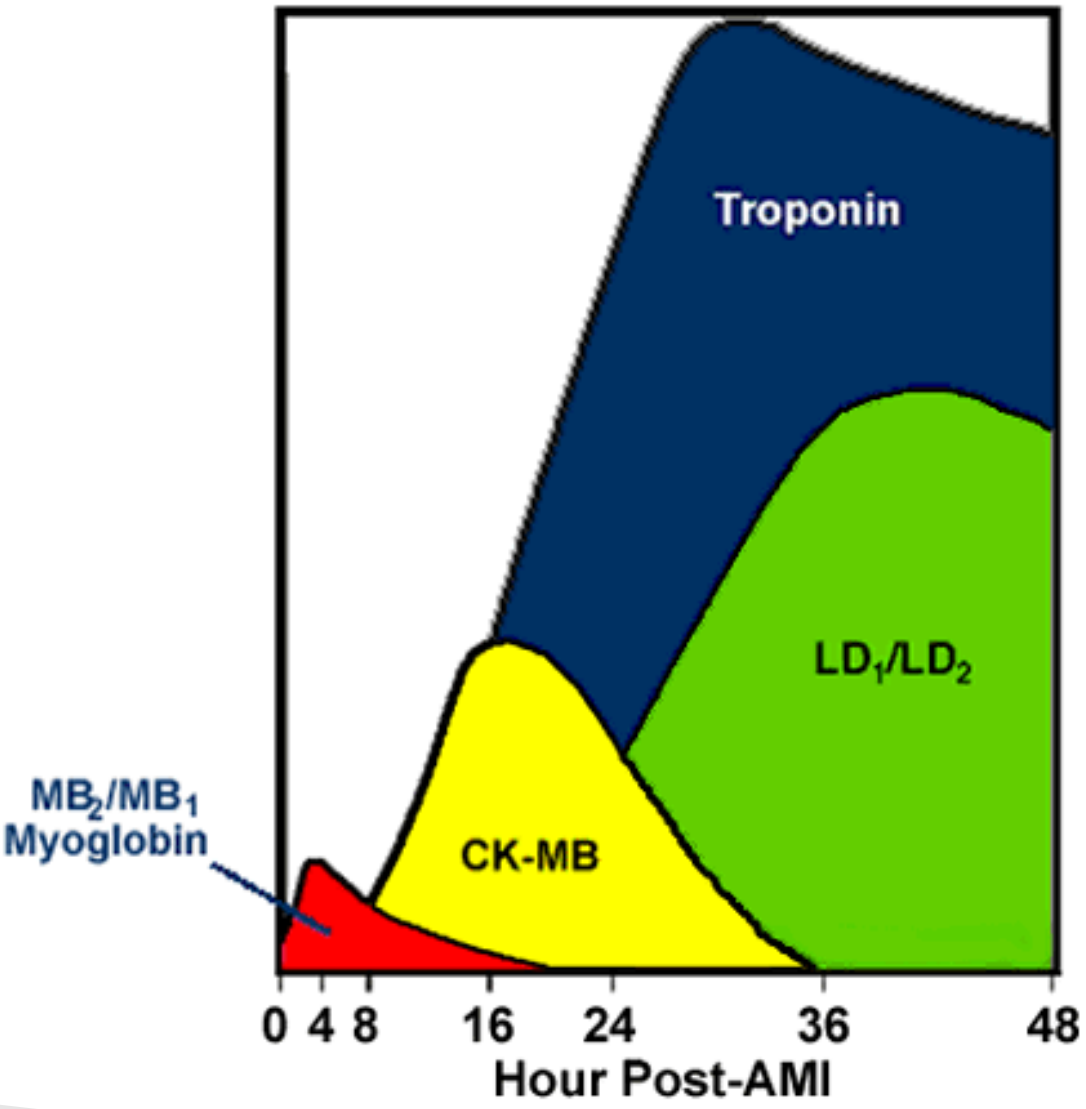
CK-MB: myocardiale fractie (10% van CK = infarct)

ASAT*/aspartaat aminotransferase*: aspecifiek

LDH*/lactaat dehydrogenase*: aspecifiek

Troponines: onderdeel contractie mechanisme spiercel (zeer gevoelig en specifiek)

Diagnostiek



Diagnostiek

- **Differentiaal diagnostiek**

Cardiaal

- Instabiele AP geen irreversibele schade

  klacht als bij AMI, vaak niet vegetatief

  vaak subendocardiaal:ST-depressie, neg T, wel reactie NTG

- Pericarditis houdings- en ademhalingsafhankelijk

  ST-elevatie ‘all-over’; geen reciproke depressie

Diagnostiek

- **Differentiaal diagnostiek**

Vasculair

- Aneurysma Dissecans ‘scheurende’ pijn tussen schouderbladen

  RR verschillen li/re arm; arm/been

  neurologische uitval!!

- Longembolie sterke ademhalingsafhankelijkheid pijn,

  dyspneu meer uitgesproken,

  na bevalling, fractuur, immobilisatie, veneuze thrombose,CVD verhoogd, pleurawrijven, tachycardie rechter as, RBTB, SIQIIInegTIII

Diagnostiek

- **Differentiaal diagnostiek**

Oesophagusspasme

reageert ook op NTG!!!

Bovenbuikspathologie

ouderen!

(Inter)costaal Pathologie (Tietze)

Diagnostiek

Diagnose hartinfarct *vermoed* als:

- typische klachten
- typische ECG afwijkingen

Diagnose hartinfarct *bewezen* als:

- typisch beloop ECG afwijkingen
- enzymen positief (CK 2x bovengrens normaal, CK-MB 10% hiervan, positieve Troponines)

Diagnostiek

- [illegible]

Definition of Myocardial Infarction

ACC/ESC Working Group 2000

- Typical rise and gradual fall (troponin) or rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis.

  With at least one of the following:

  A. Ischemic Symptoms
  B. Development of Q-Waves on the ECG
  C. ECG Changes Indicative of Ischemia (↑ ST or ↓ ST)
  D. Coronary Revascularization (i.e. PCI)

- And/or pathological findings of an acute myocardial infarction.

Therapie

Doel therapie

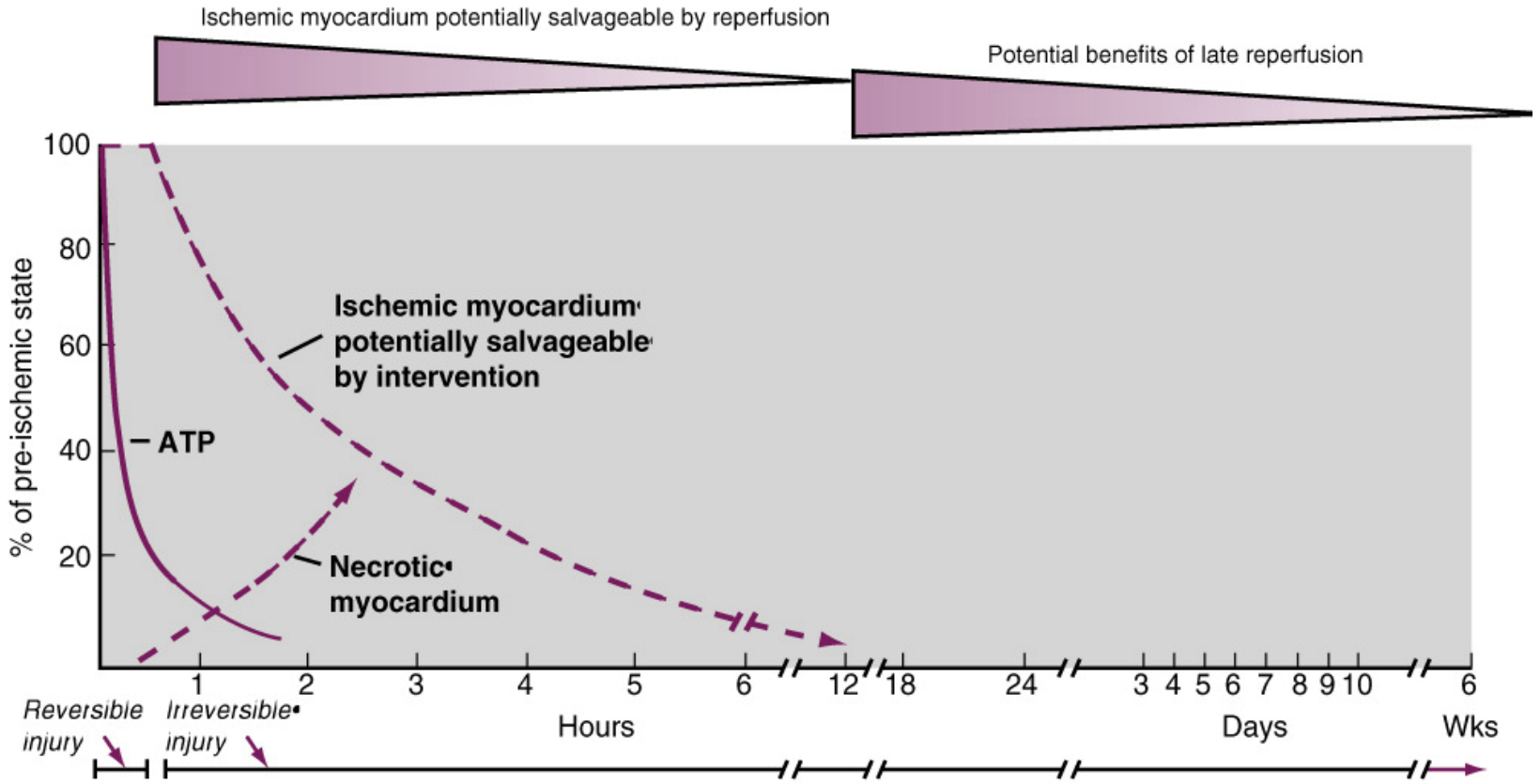
- herstelen balans tussen vraag en aanbod

→ beperken infarctgrootte!

"TIME IS MUSCLE": snelheid is geboden

"TIME TO REPERFUSION" bepaalt een groot deel van de prognose → mogelijkheden thrombolyse en PCI

Therapie


Ischemic myocardium potentially salvageable by reperfusion
Potential benefits of late reperfusion
100
80
60
40
20
% of pre-ischemic state
Ischemic myocardium potentially salvageable by intervention
ATP
Necrotic myocardium
1
2
3
4
5
6
12
18
24
3 4 5 6 7 8 9 10
6
Hours
Days
Wks
Reversible injury
Irreversible injury

Therapie

Verbeteren supply:

- zuurstof per neusssonde

- doorgankelijkheid coronair herstellen (reperfusie)
    - ASA remt plaatjesaggregatie
    - chemische recanalisatie: i.v. Thrombolyse
    - mechanische recanalisatie: PTCA

- inotropie, I.A.B.P. bij cardiogene shock

Therapie

Verminderen demand:

- vertragen hartfrequentie: beta-blokkade
- bestrijden hypertensie: nitraten, beta-blokkade
- pijnbestrijding/sedatie: Fentanyl
- bedrust

Therapie

Recanalisatie

Algemeen	< 3 h:	50 % risk area gered
	3-6h:	25 % risk area gered
	6-12h:	12.5% risk area gered

"TIME IS MUSCLE!"

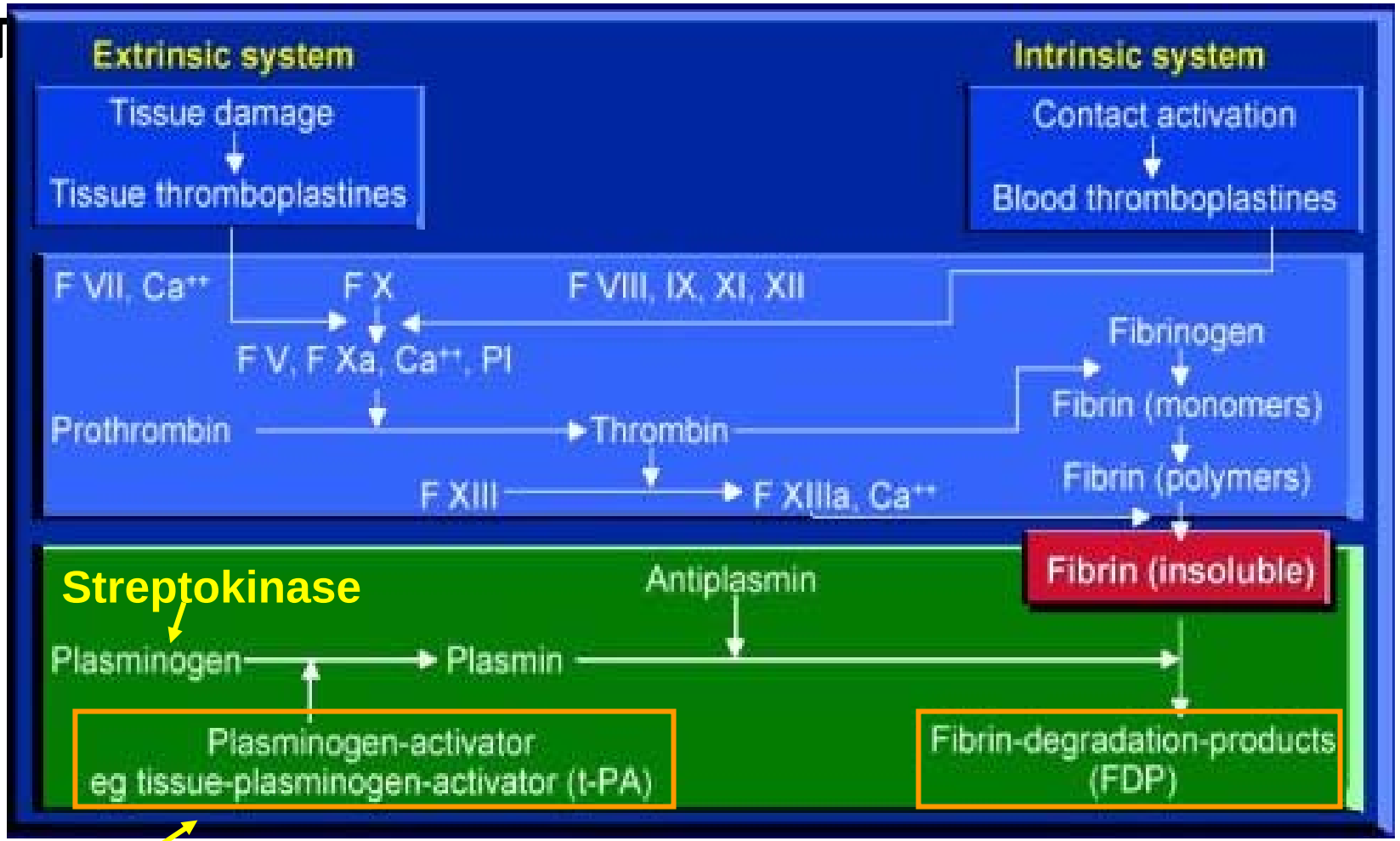
Pre-thrombolyse tijdperk:

Hartcatheterisatie: (De Wood et al.1980, De Feyter et al. 1982)

4 uur na infarct:	87% occlusie
1 dag - 8 wkn:	65% occlusie

Therapie

T


Extrinsic system
Intrinsic system
Tissue damage
Tissue thromboplastines
Contact activation
Blood thromboplastines
F VII, Ca++
F X
F VIII, IX, XI, XII
F V, F Xa, Ca++, PI
Fibrinogen
Fibrin (monomers)
Prothrombin
Thrombin
F XIII
F XIIIa, Ca++
Fibrin (polymers)
Fibrin (insoluble)
Streptokinase
Antiplasmin
Plasminogen
Plasmin
Plasminogen-activator
eg tissue-plasminogen-activator (t-PA)
Fibrin-degradation-products
(FDP)
r-tPA

Therapie

Thrombolyse (GISSI 1986, 11.000 ptn behandeld)

Streptokinase:	na 90 min 50-60% reperfusie
(Urokinase)	na 180 min 80% reperfusie
	geen effect CVA's
	CAVE: allergie/antistoffen/ bloeddrukdaling
r-tPA:	na 90 min 80% reperfusie
(Actilyse®, Metalyse®,	meer bloedige CVA's
Rapilysin® , etc.)	geen allergie/antilichamen/ bloeddrukdaling
	duur

Therapie

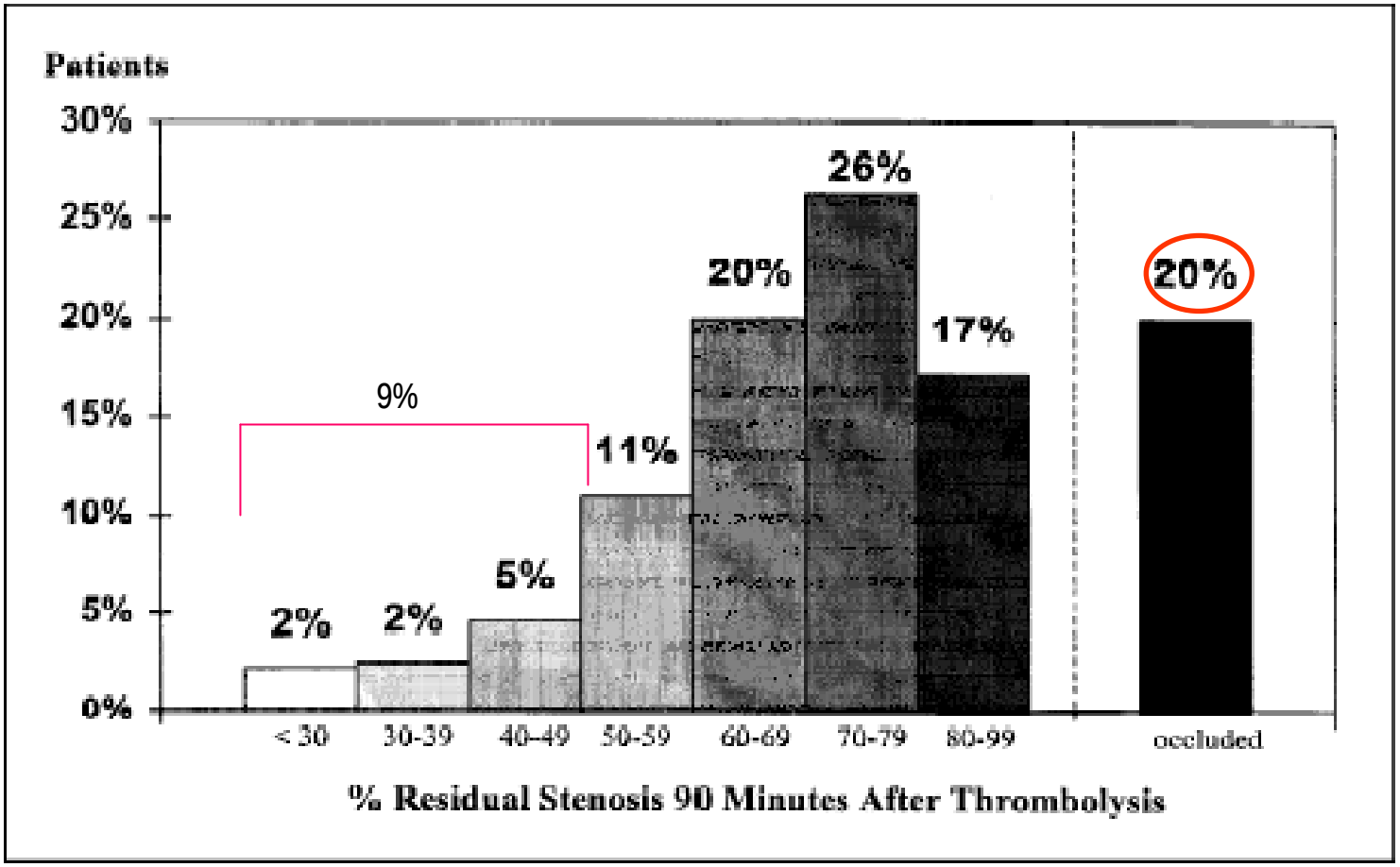


FIGURE 1. Distribution of residual stenosis in the infarct-related artery at 90 minutes after thrombolysis in 2,119 patients in the TIMI 4, 10A, 10B, and 14 trials.

Llevadot et al, AJC 2000

Therapie

- **Keuze Thrombolyse versus P.C.I.**

PTCA ‘waarschijnlijk’ superieur

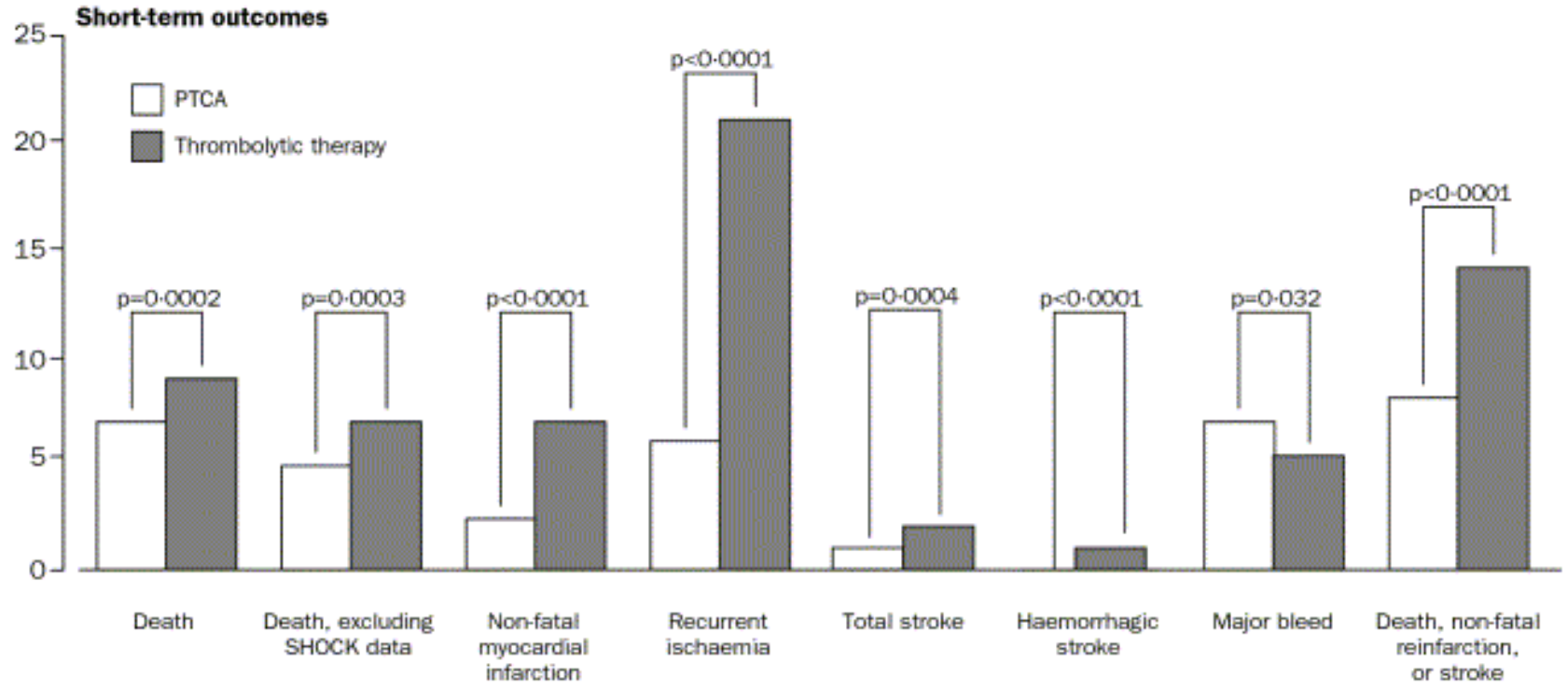
Uiteindelijke keuze afhankelijk : winst vs. risico
logistiek
financiën

Situatie regio Amsterdam:

Indien klachten <6 uur → P.C.I. → **LIFENET**

Therapie

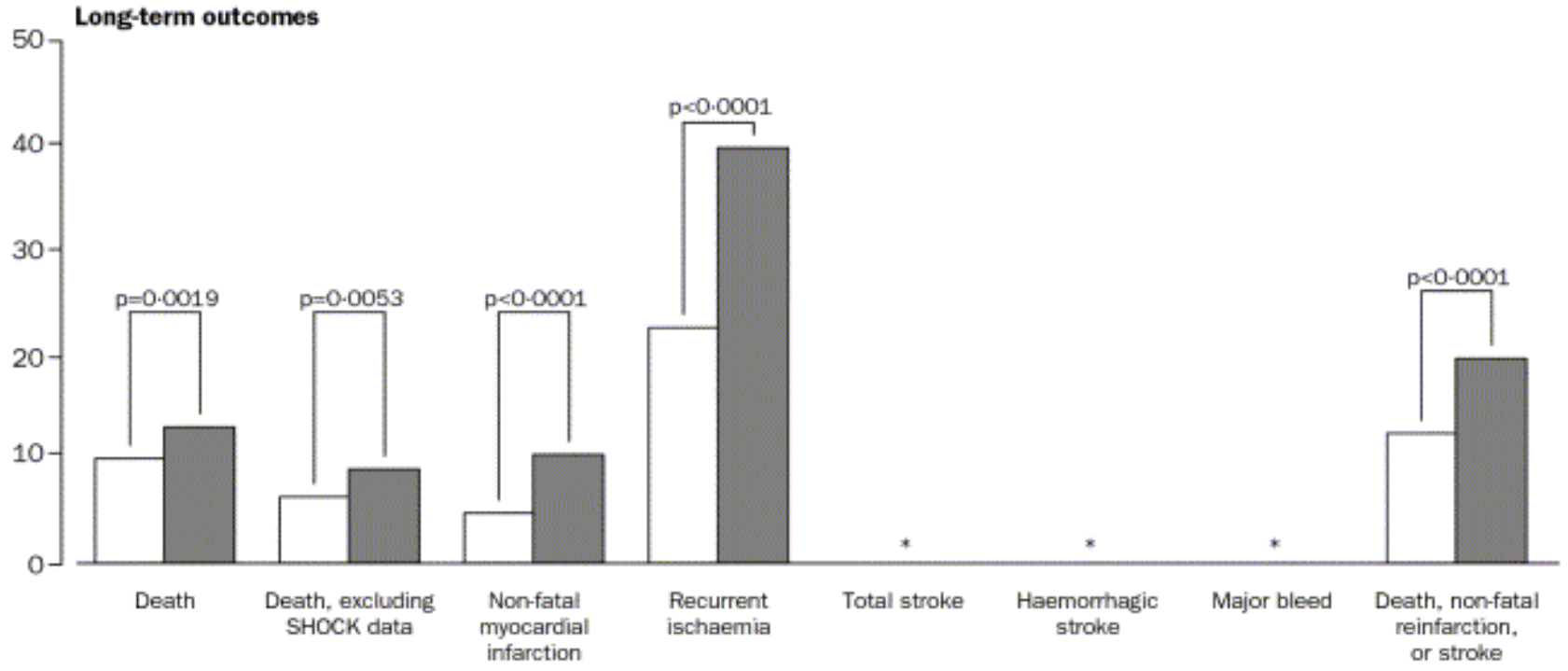
PTCA vs Thrombolysis Korte termijn



Keeley, The Lancet 2003

Therapie

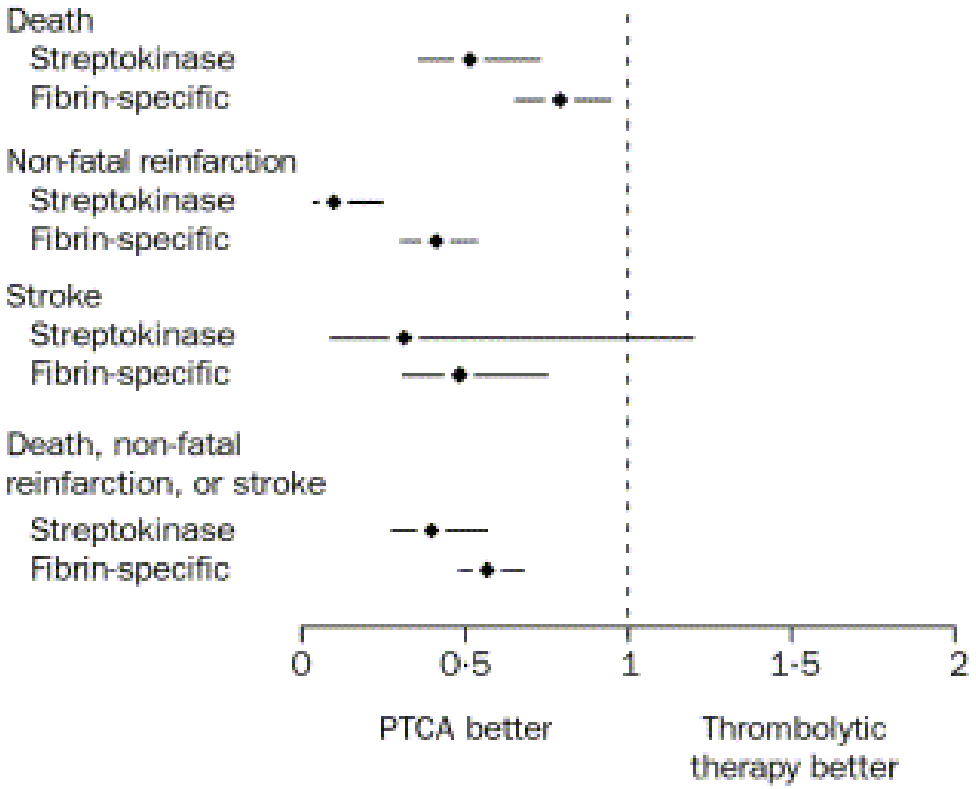
PTCA vs Thrombolysis lange termijn



Keeley, The Lancet 2003

Therapie

PTCA vs Thrombolysis



Keeley, The Lancet 2003

Therapie

PTCA (occlusie)

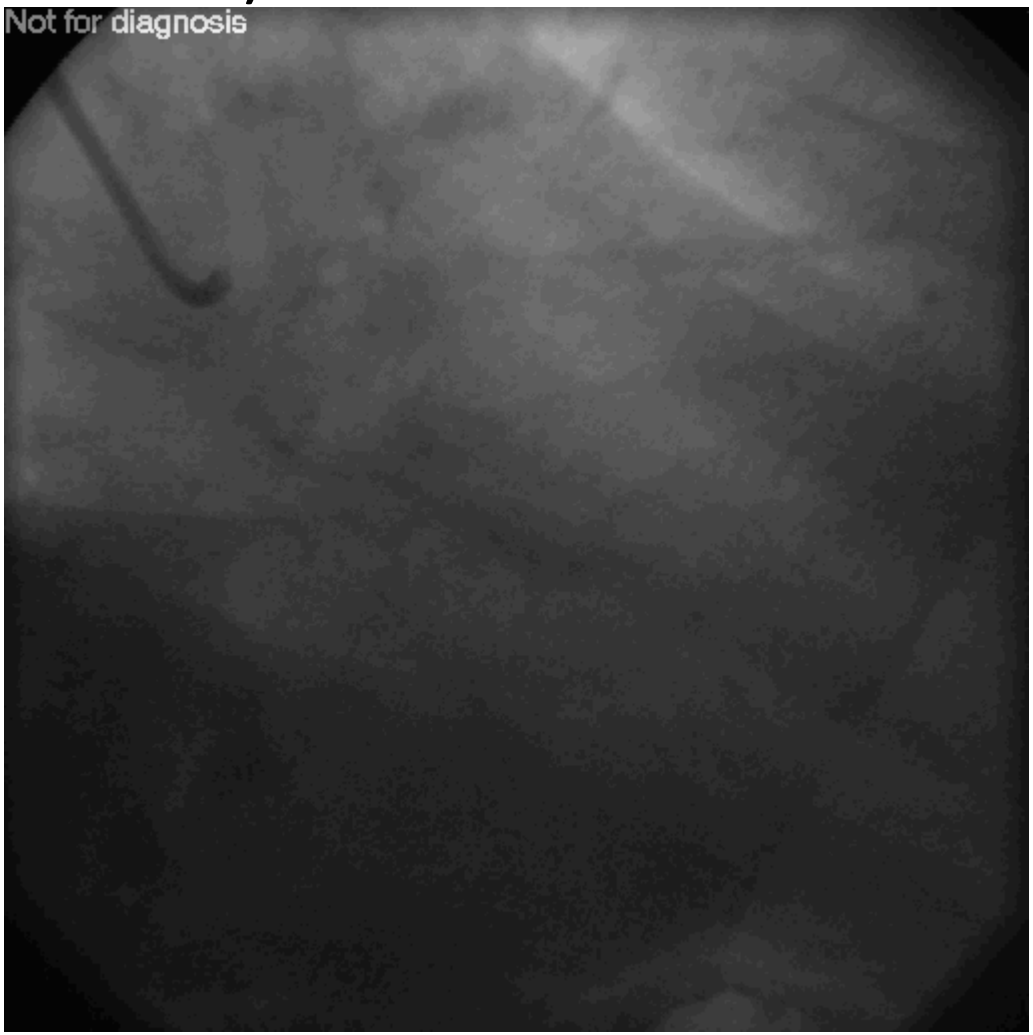
Therapie

PTCA (ballon-dilatatie)

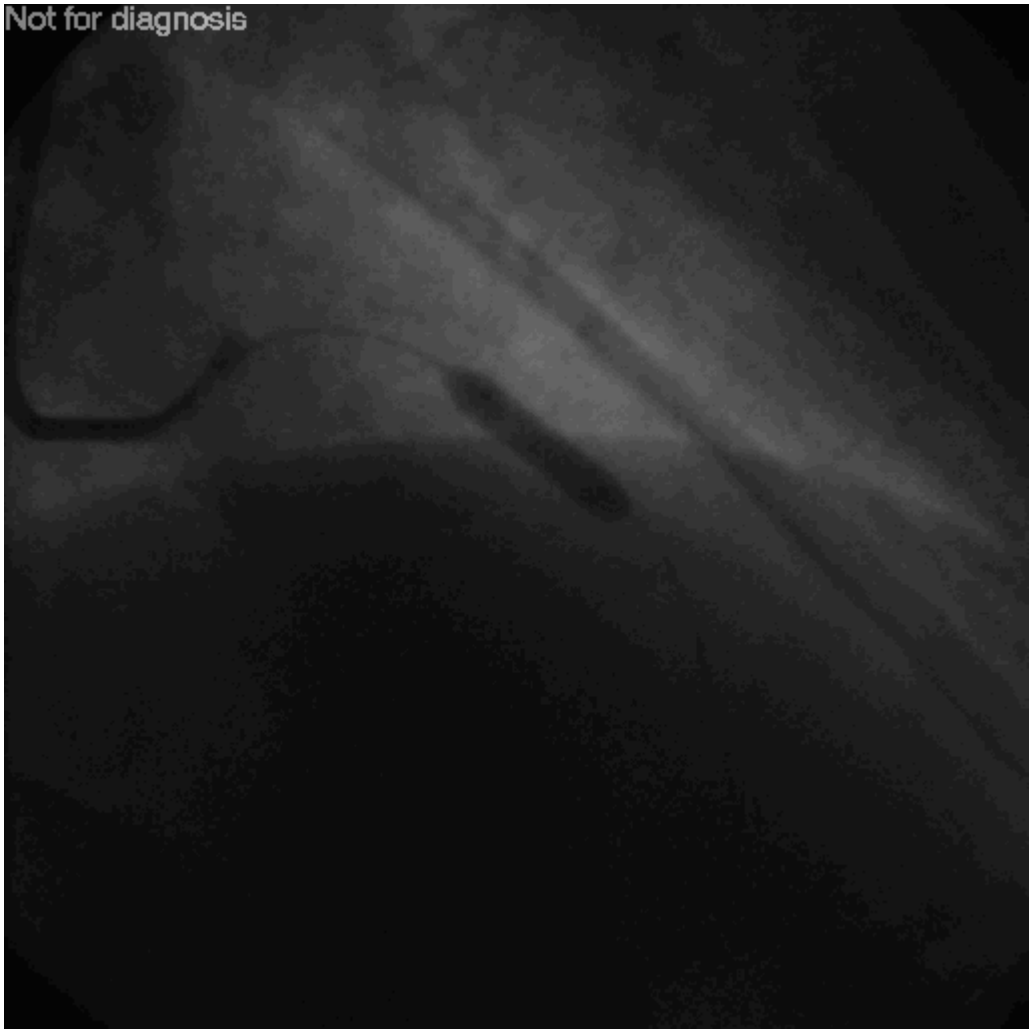
Therapie

Therapie

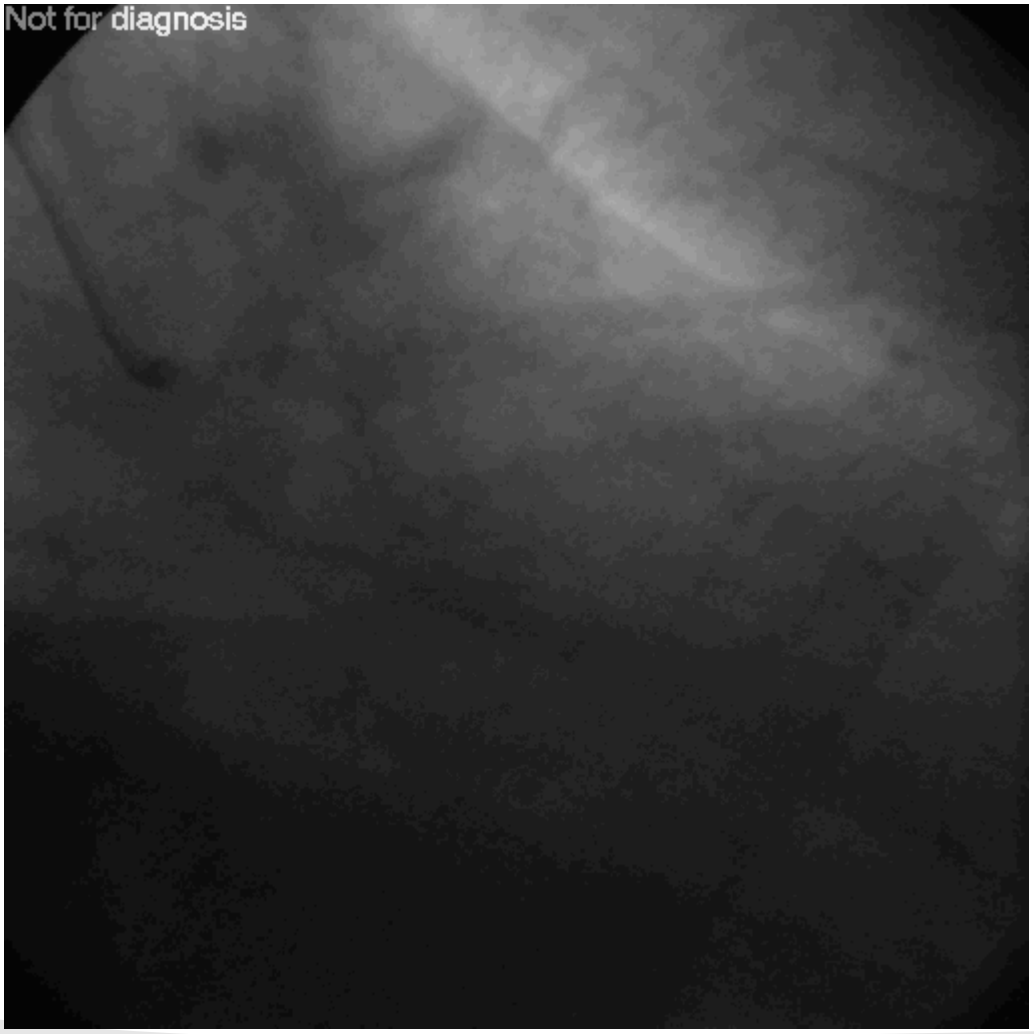
PTCA (subocclusie)

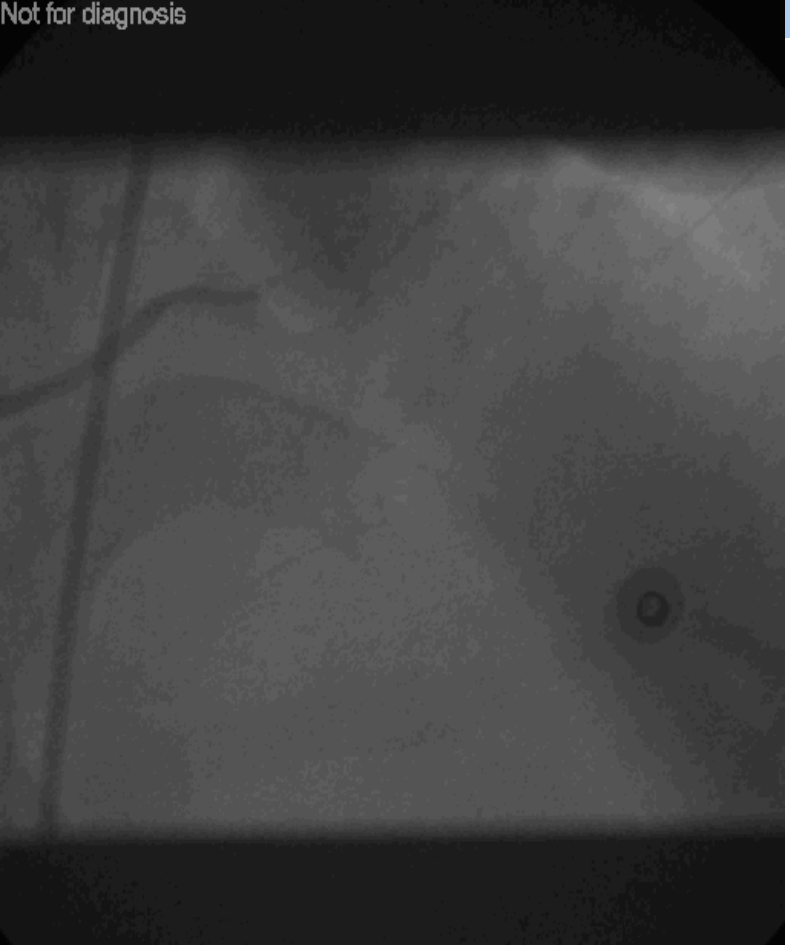


Therapie



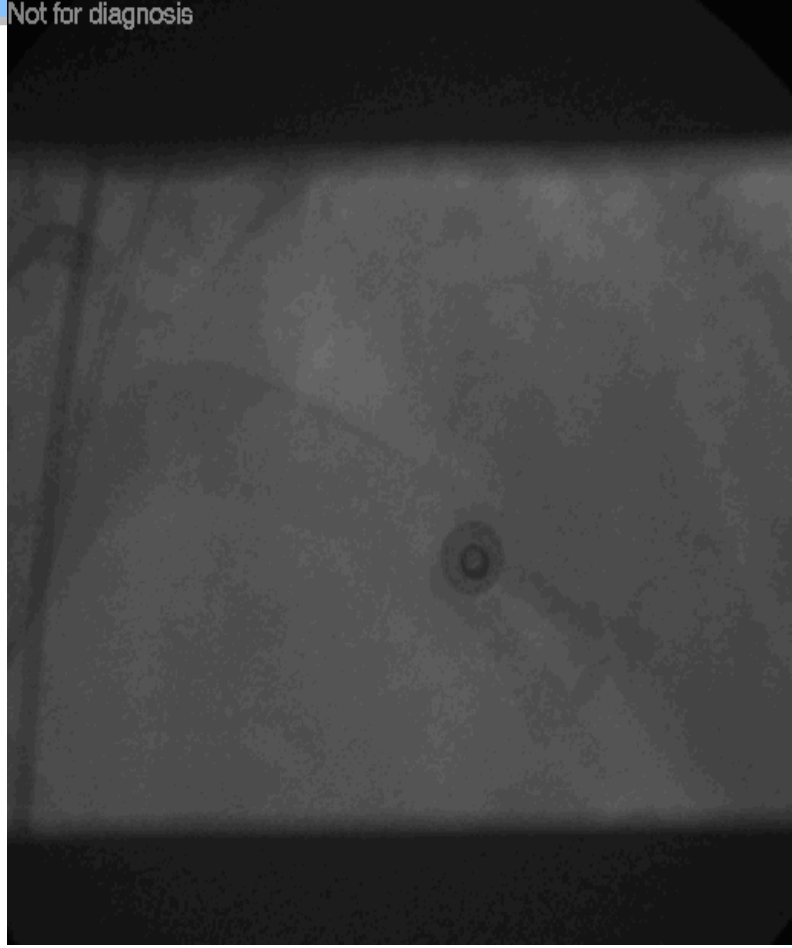
Therapie





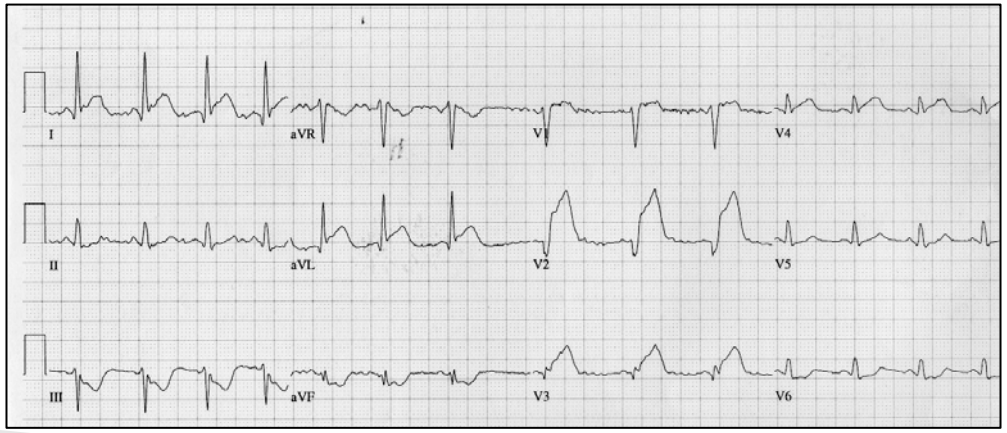
Anterior

AMI



occlusie

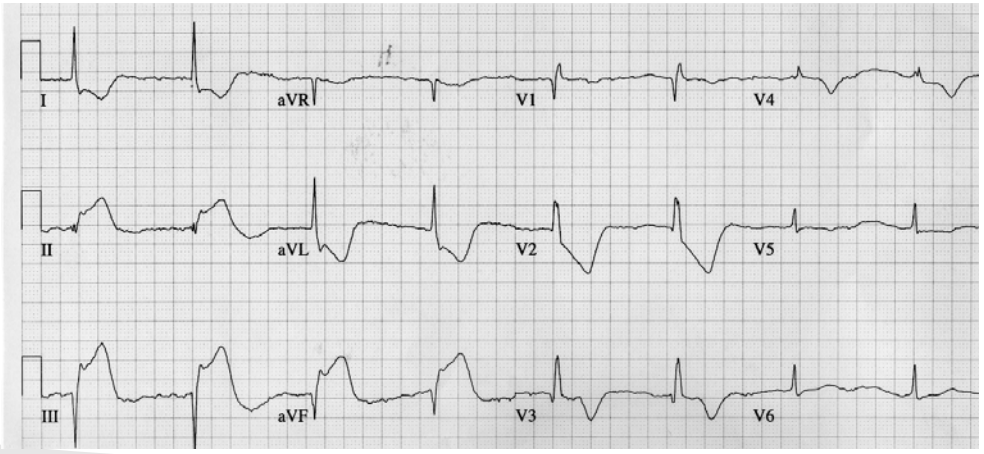
LAD



Inferior AMI

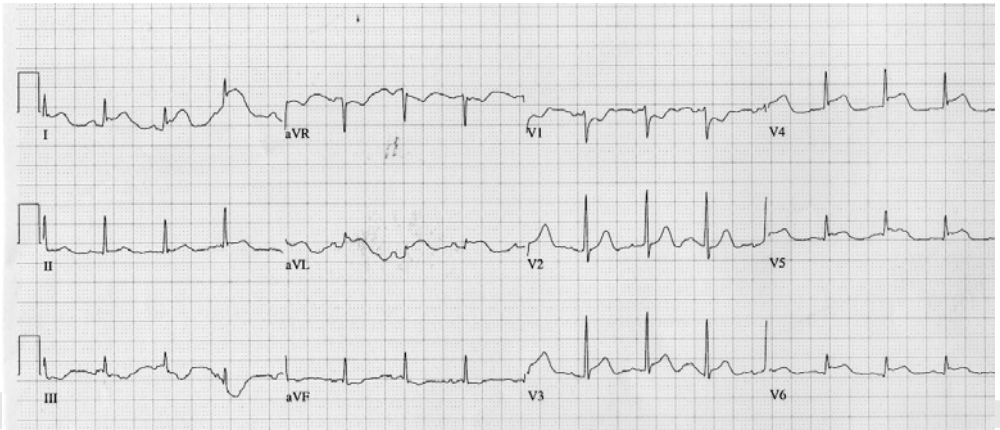
occlusie

RCA




Not for diagnosis


Not for diagnosis


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Reperfusie

- **Aanwijzingen reperfusie**

  - afname klachten **NB**: pijnstilling, sedatie
  - afname ST-shift op t=90 minuten

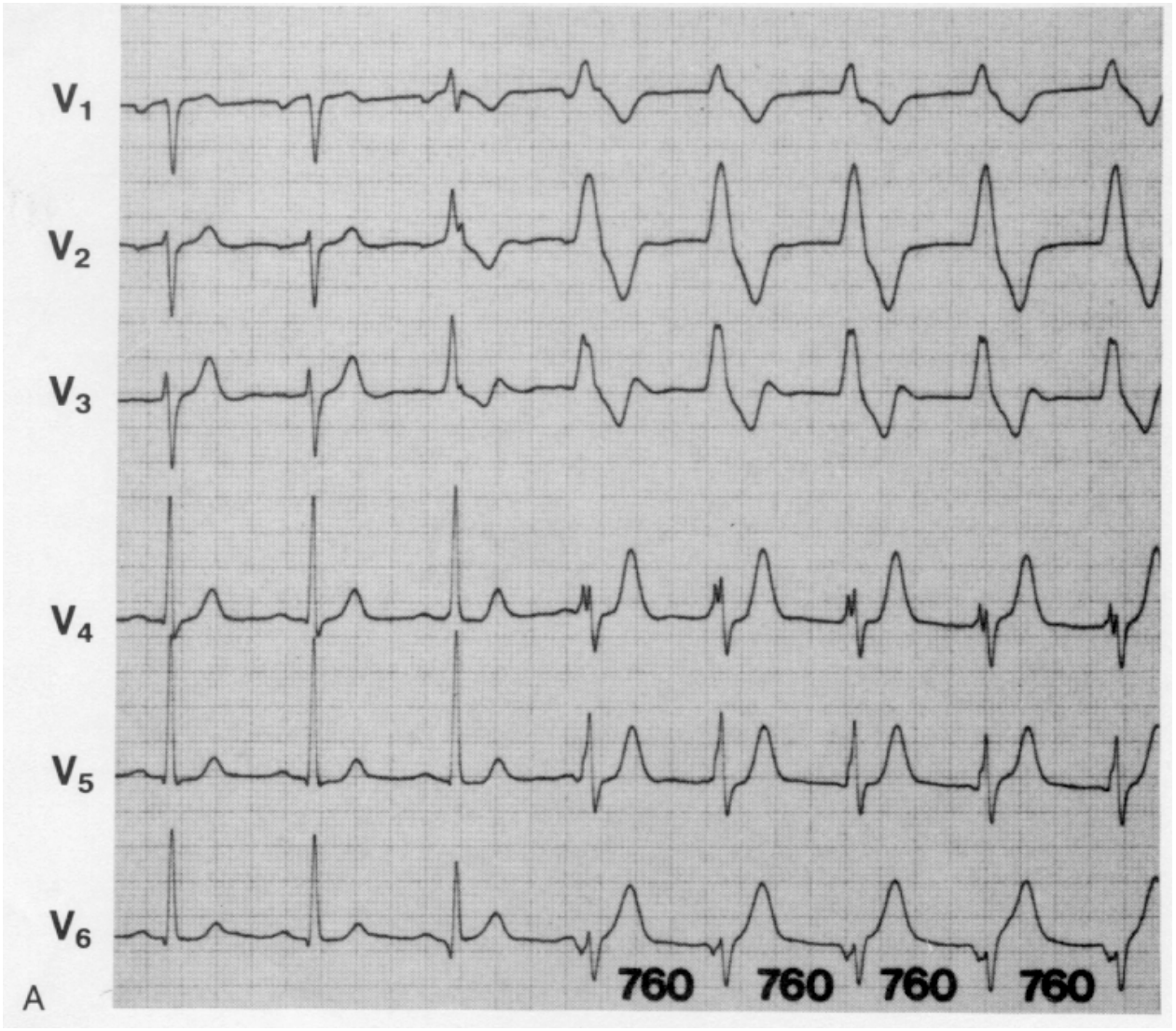
  ST-shift(t=90) < 50% ST-shift(max)

  - optreden aritmie → **A.I.V.R.**
  - combinatie bovengenoemde factoren

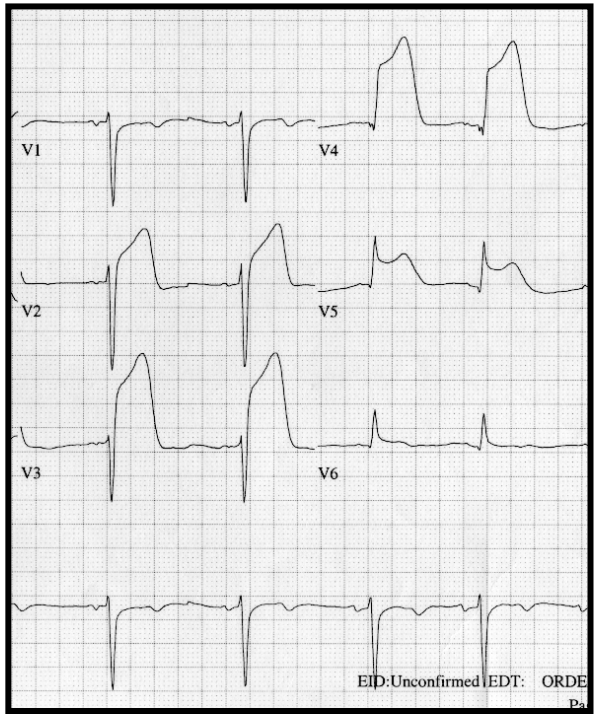
- **Geen aanwijzingen reperfusie**

  - Bij afwezigheid bovengenoemde factoren overweeg rescue PTCA
  - Boezemfibrilleren

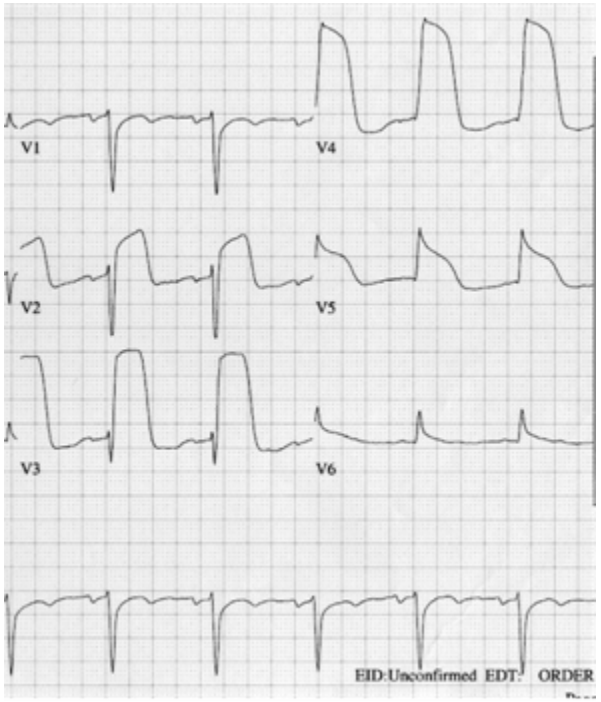
AIVR



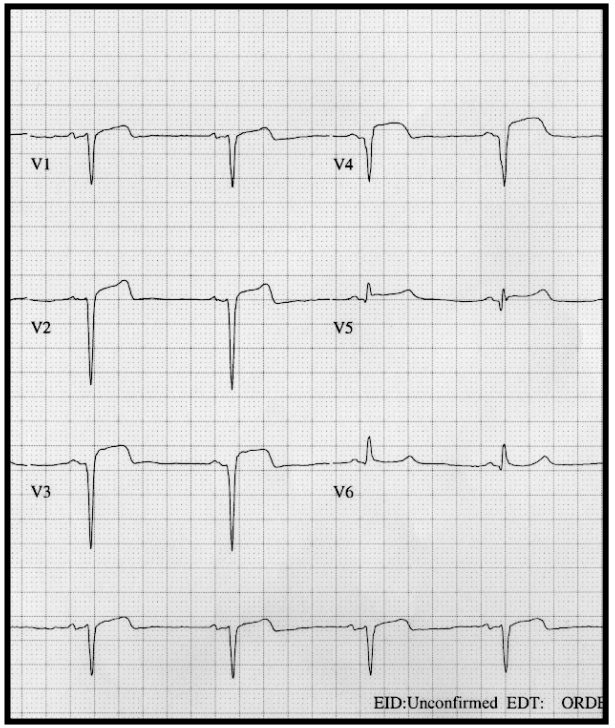
Reperfusie



before reperfusion



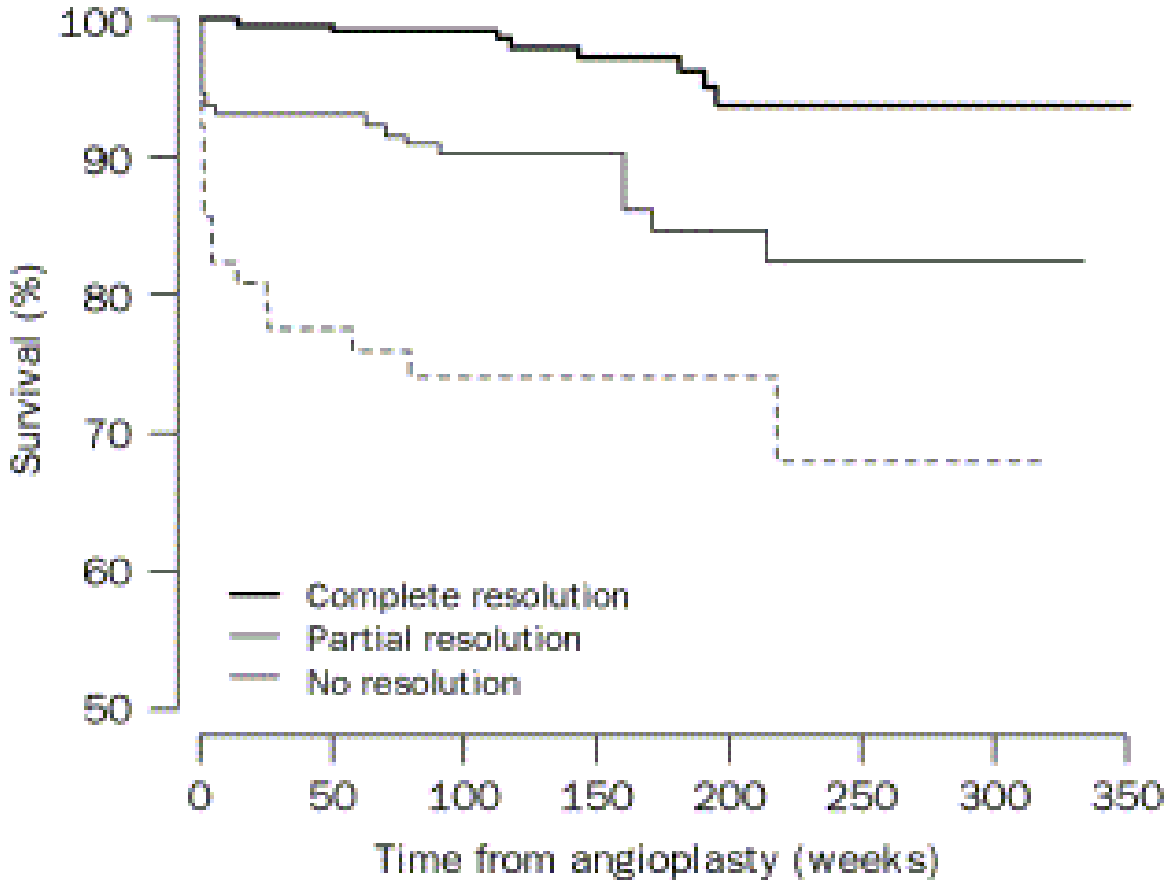
during reperfusion



after reperfusion

Reperfusie

ECG en ST resolutie



> 70% resolutie

30 - 70% resolutie

< 30% resolutie

Therapie

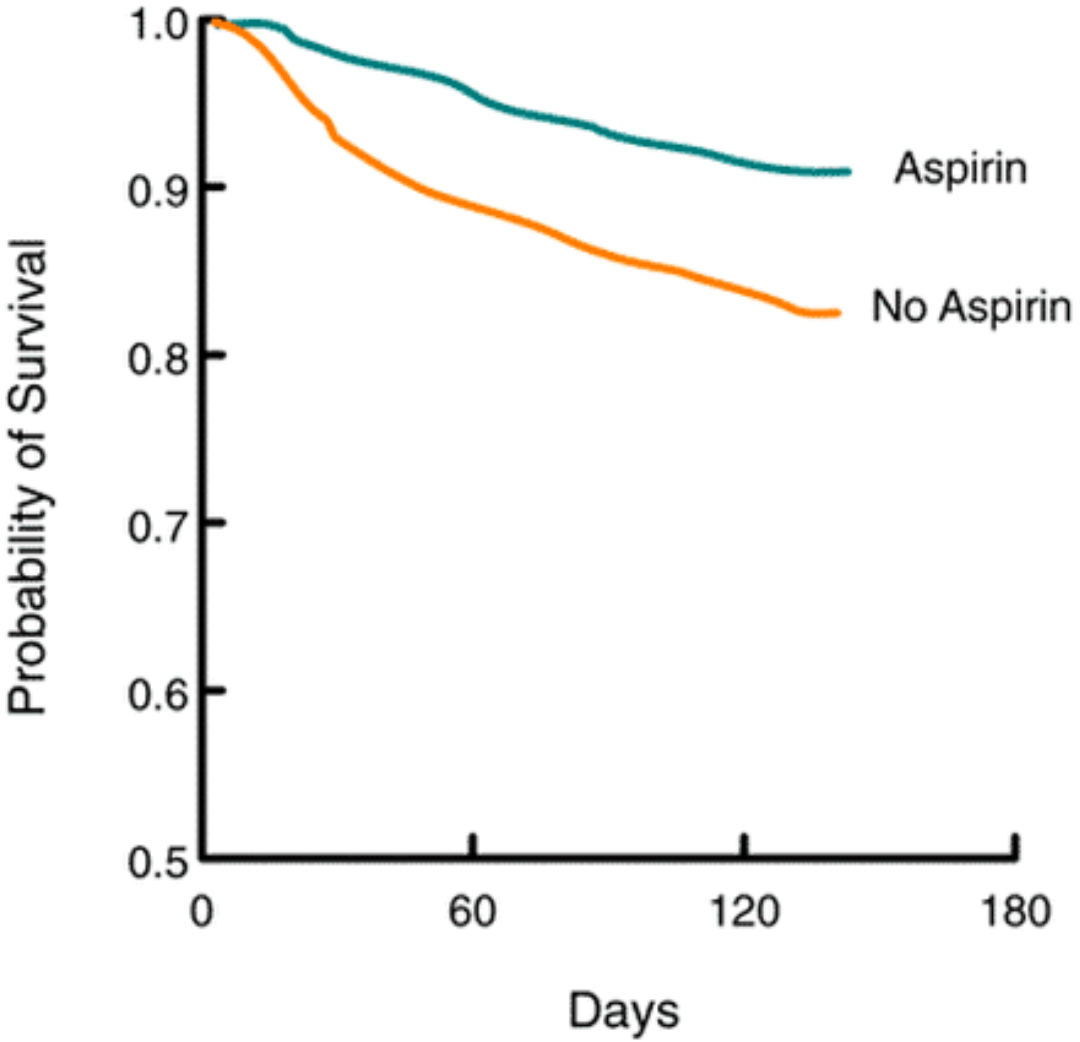
- **Basistherapie**

Aspirine, betablockers + statines

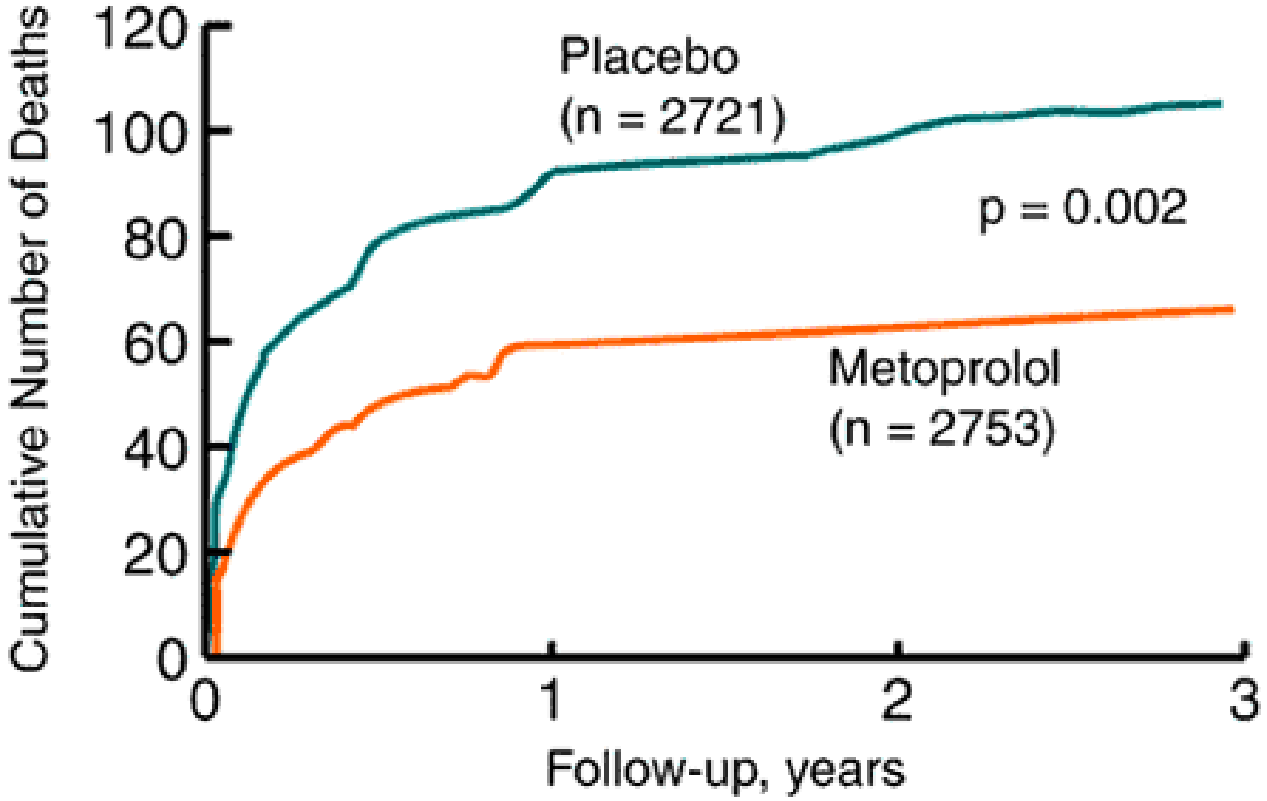
- **Eventueel**

ACE remmers:	(verwacht) hartfalen
Diuretica:	idem
Antiarrhytmica:	bij (ventriculaire) aritmie
Antistolling:	bij bewezen LV thrombus of profylactisch bij slechte LV restfunctie
Nitraten:	(post AMI) angina pectoris

Therapie

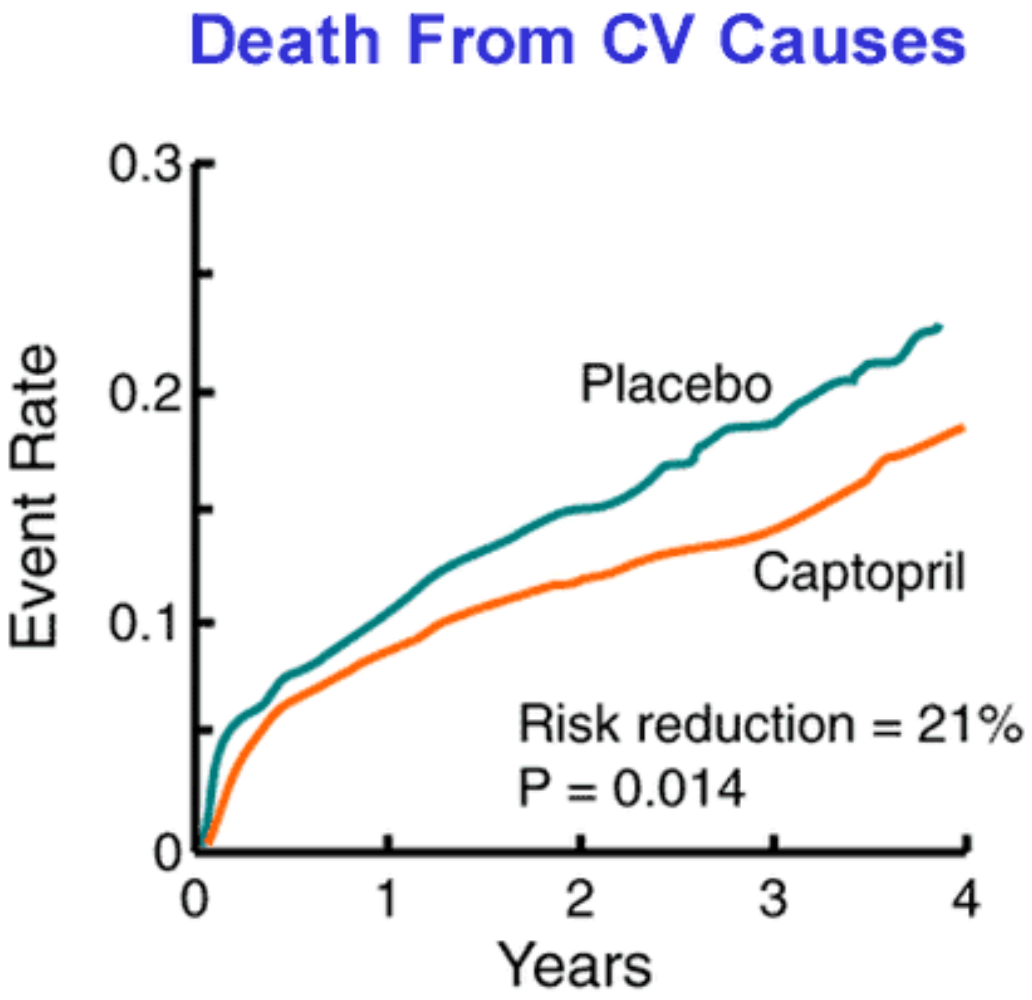

1.0
0.9
0.8
0.7
0.6
0.5
Probability of Survival
Aspirin
No Aspirin
0
60
120
180
Days

Therapie



Cumulative number of sudden deaths reported in five postinfarction trials.

Onderhoudstherapie



Therapie

Controversieel

Antistolling:	iedereen post AMI
Ca blockers:	Overstefte?
Digoxine:	falen - alleen effect aantal heropnamen

Tevens

Stoppen met roken

Hypertensie behandelen

Diabetes goed regelen

Overgewicht bestrijden

Bij jonge mensen (<50 jaar): hyperhomocysteïnemie!

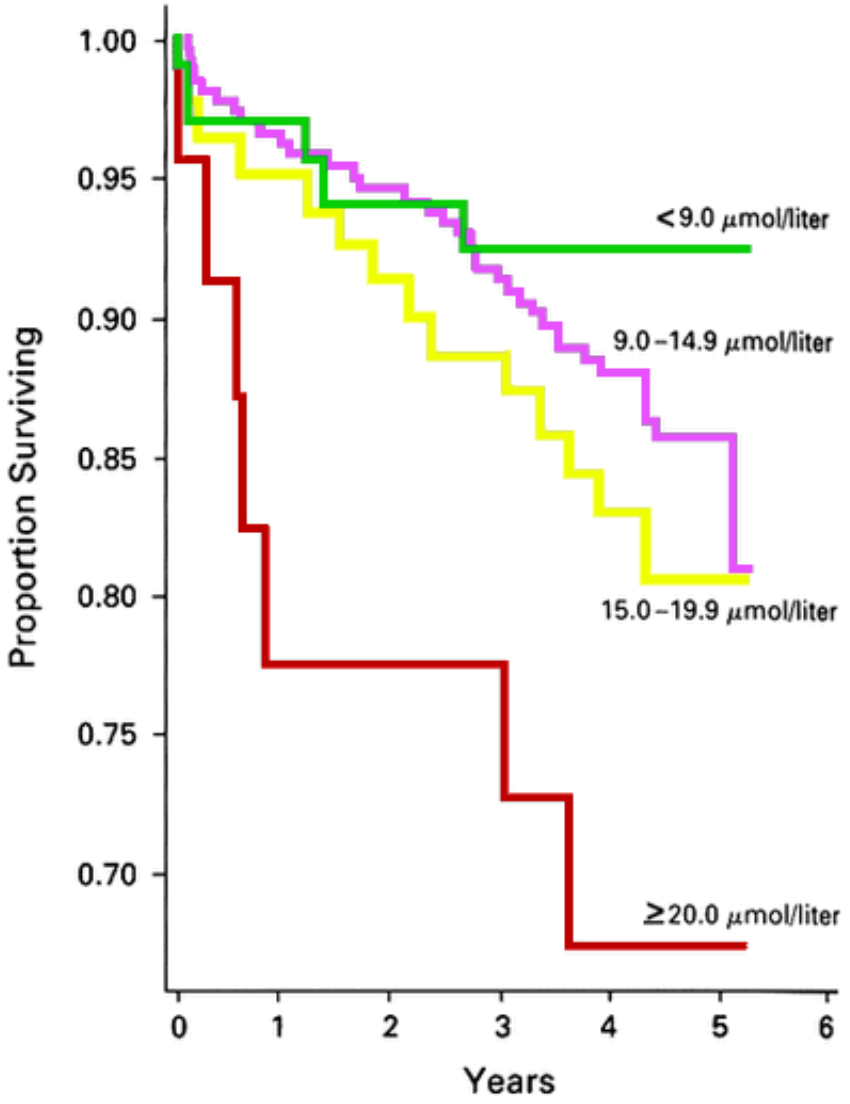
Therapie

Summary Of Large Observational Studies Of The Effect Of Smoking Cessation On Mortality After Acute Myocardial Infarction With At Least 5 Years Of Follow-up

Study	Patients Studied (no.)	5-Year Mortality Rate	
		Quitters	Smokers
Sparrow, 1978	365 (269 men, 96 women)	12%	25%
Aberg, 1983	983 (men only)	16%	22%
Daly, 1983	498 (men only)	20%	30%
Johansson, 1985	156 (women only)	15%	27%
Perkins, 1985	119 (90 men, 29 women)	21%	47%
Hedback, 1987	305 (258 men, 47 women)	16%	31%

Therapie

Homocysteïne



Acute complicaties

Electrisch

ritmestoornissen abnormaal automatisme

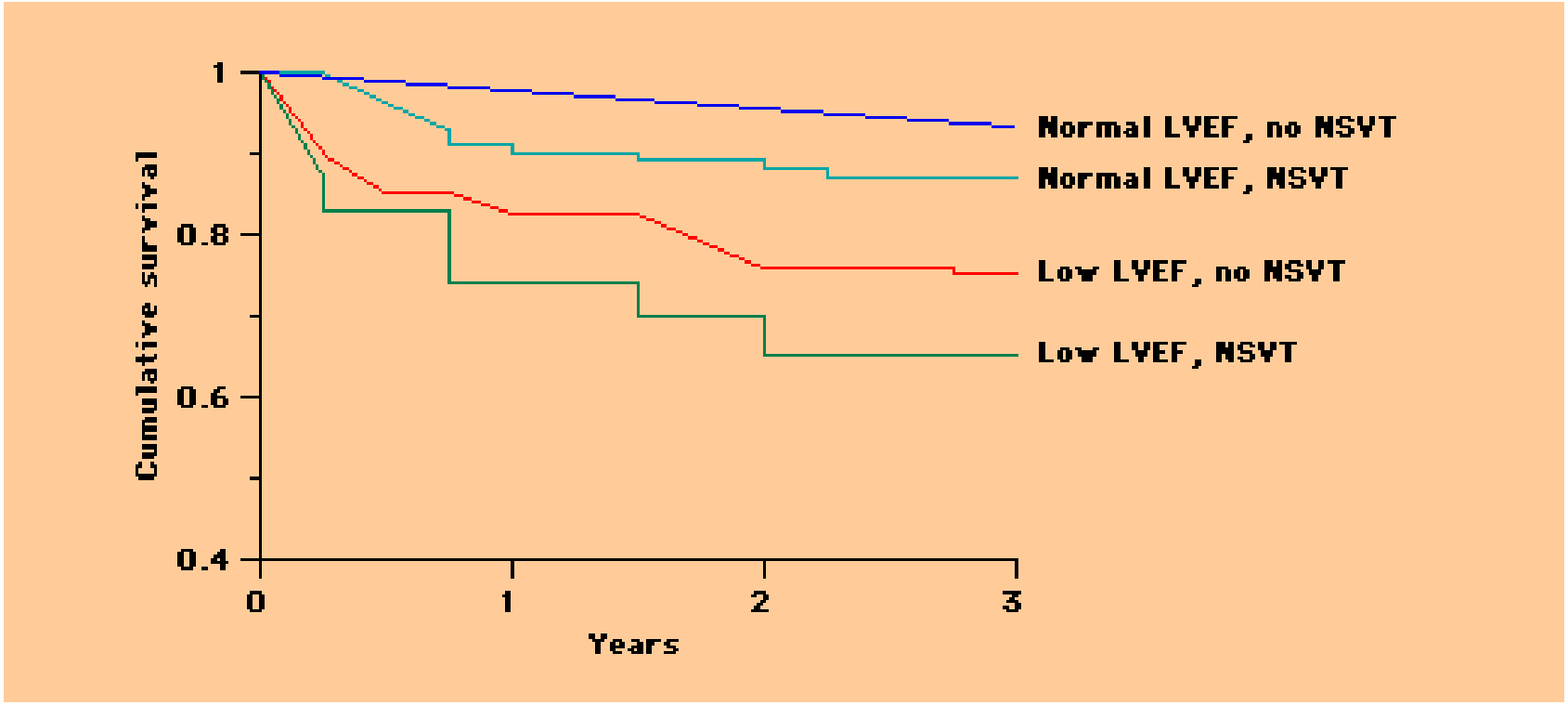
re-entry circuits

geleidingsstoornissen AV block (OWI - RCA)

RBTB e.v.t. met LAFB (VWI - LAD)

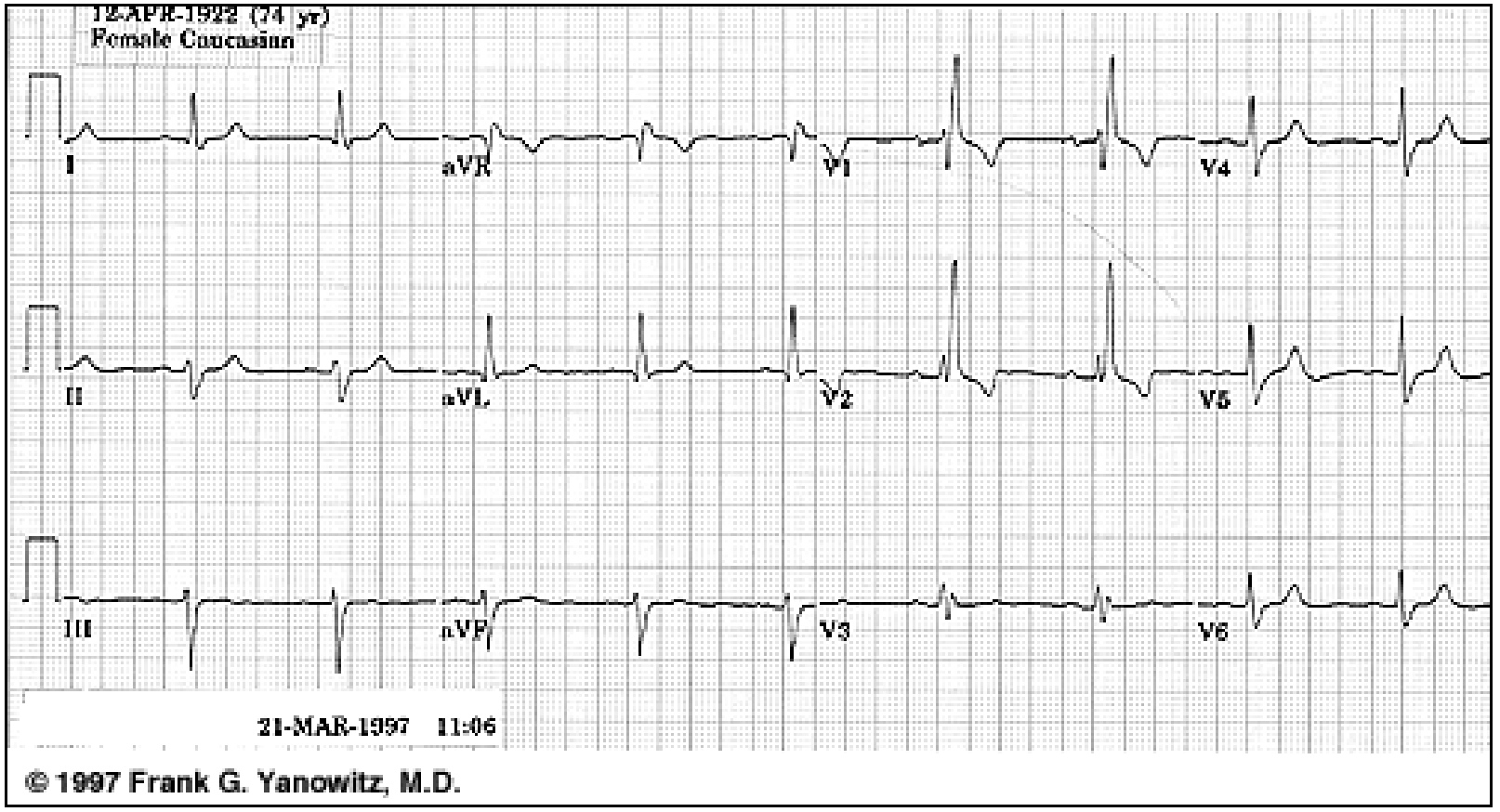
Complicaties (acuut)

- VT’s na AMI



Complicaties (acuut)

- **RBTB en LAFB**



Complicaties

Mechanisch

- papillairspier ischemie: mitralisklepinsufficientie — ischemische
- papillairspier ruptuur: verslechtering, — plots massale MI → acute
  - » astma cardiale
- VSR: — li-re shunt
  - » volume overbelasting
  - » acute verslechtering, vnl rechts falen
- vrije wand ruptuur: shock/EMD — hemopericard, tamponade,
  - » meestal fataal

Therapie

Rechter Ventrikel infarct:

RV falen: laag slagvolume bij verhoogde RV vullingsdruk cq. CVD,
vullen o.g.v. Swann Ganz catheter

Pericarditis

typisch 4-7 dagen post AMI (Dressler)
recidief ‘POB’

LV Thrombus

kans op ischemisch CVA
Coumarines

Complicaties

- **Functioneel**

  hartfalen remodelling - vergrote eindiastolische diameter

  recidief ischemie

- **Electrisch**

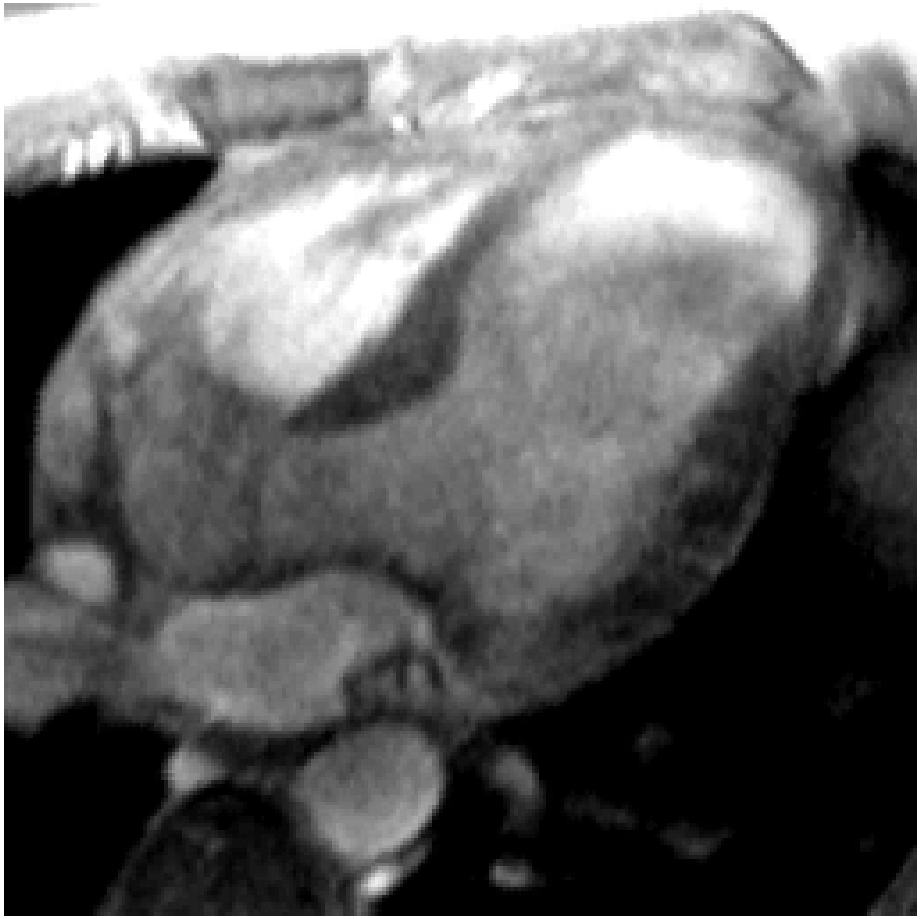
  aritmie tgv. falen

  ischemisch

- **recidief AMI**

Therapie

- **Remodelling (aneurysma)**

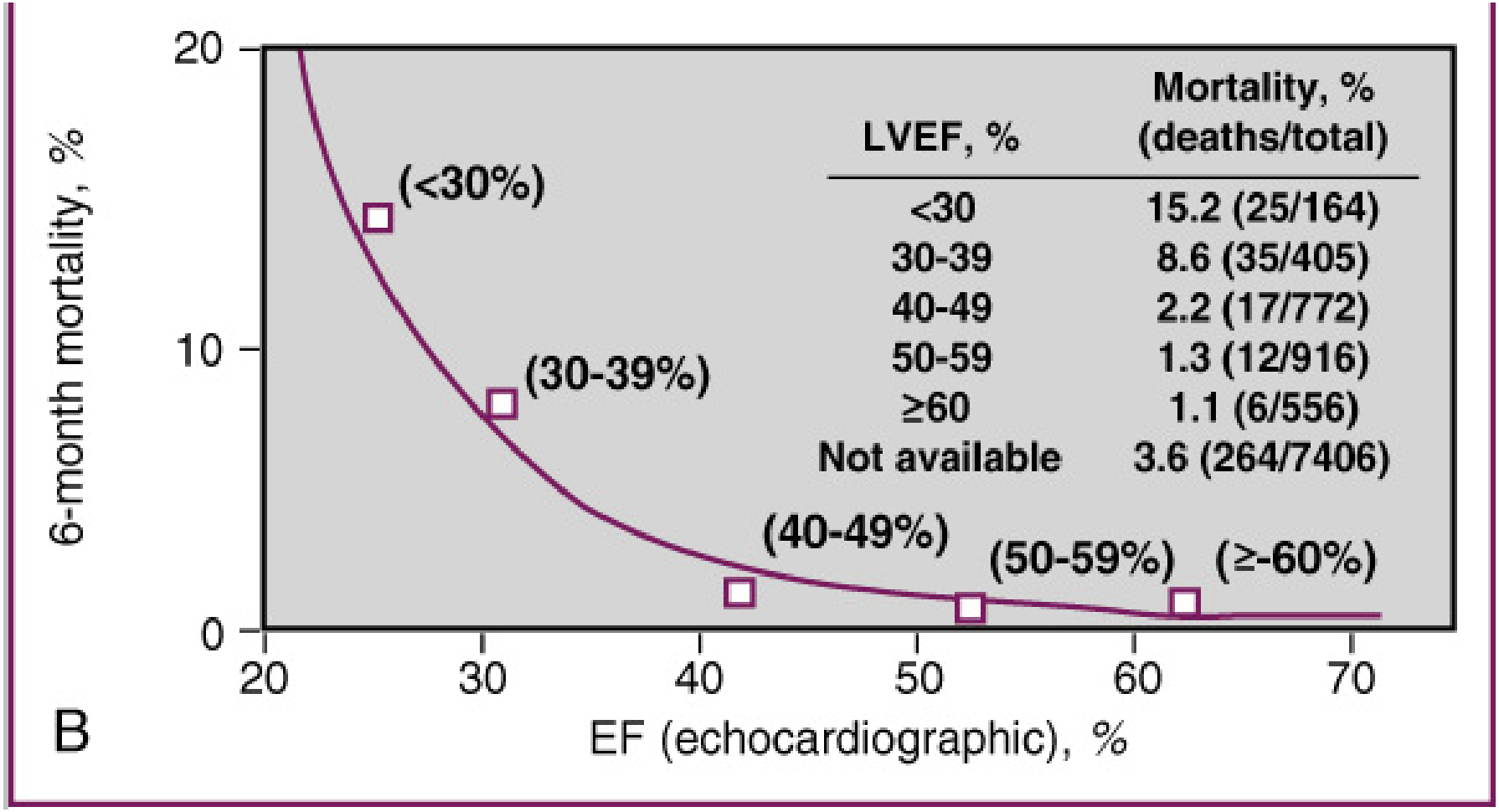


Prognose

Prognose post-AMI

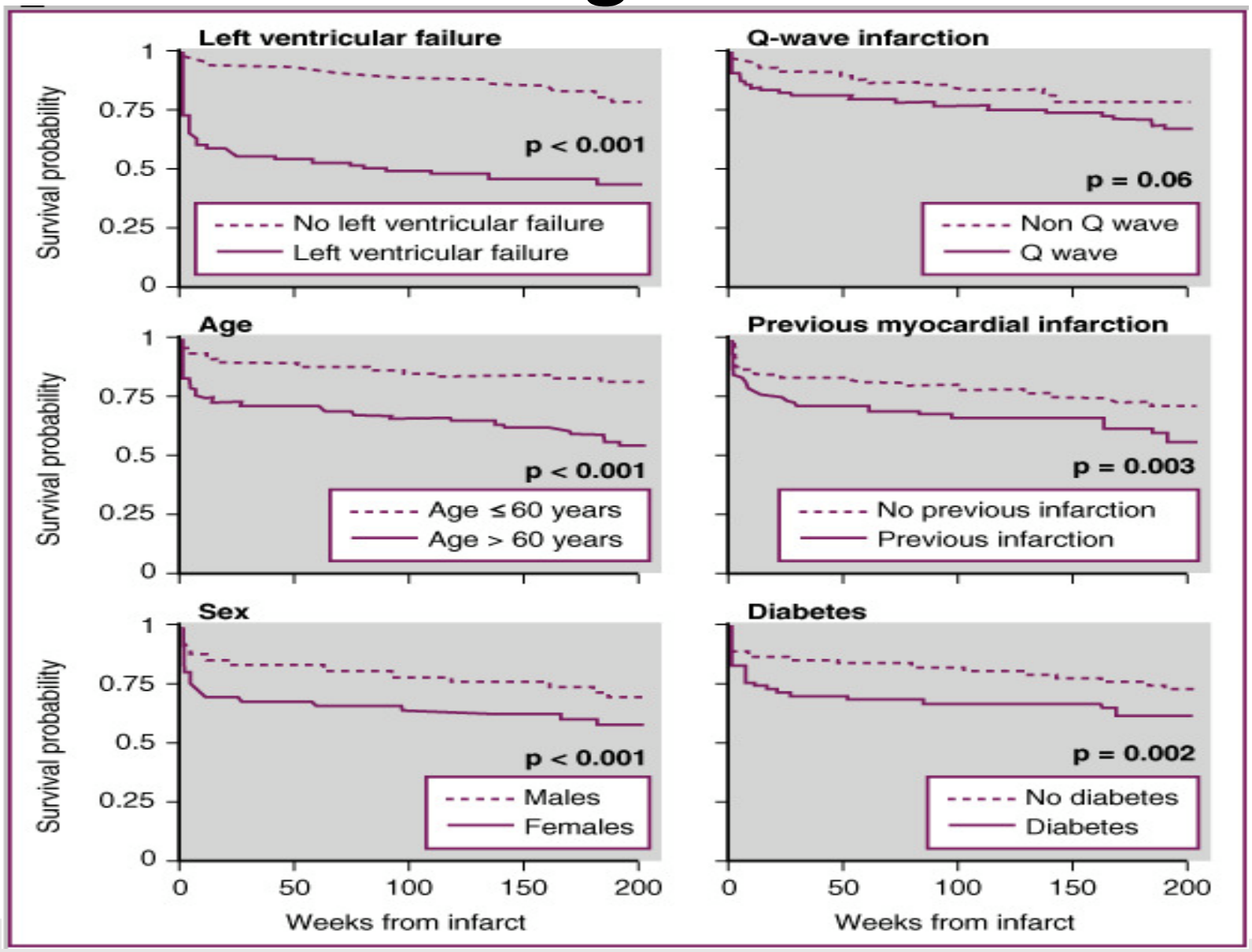
- LV rest functie
- uitgebreidheid coronaria lijden
- ischemie
- vrouw
- leeftijd
- % resterende stenose

Prognose



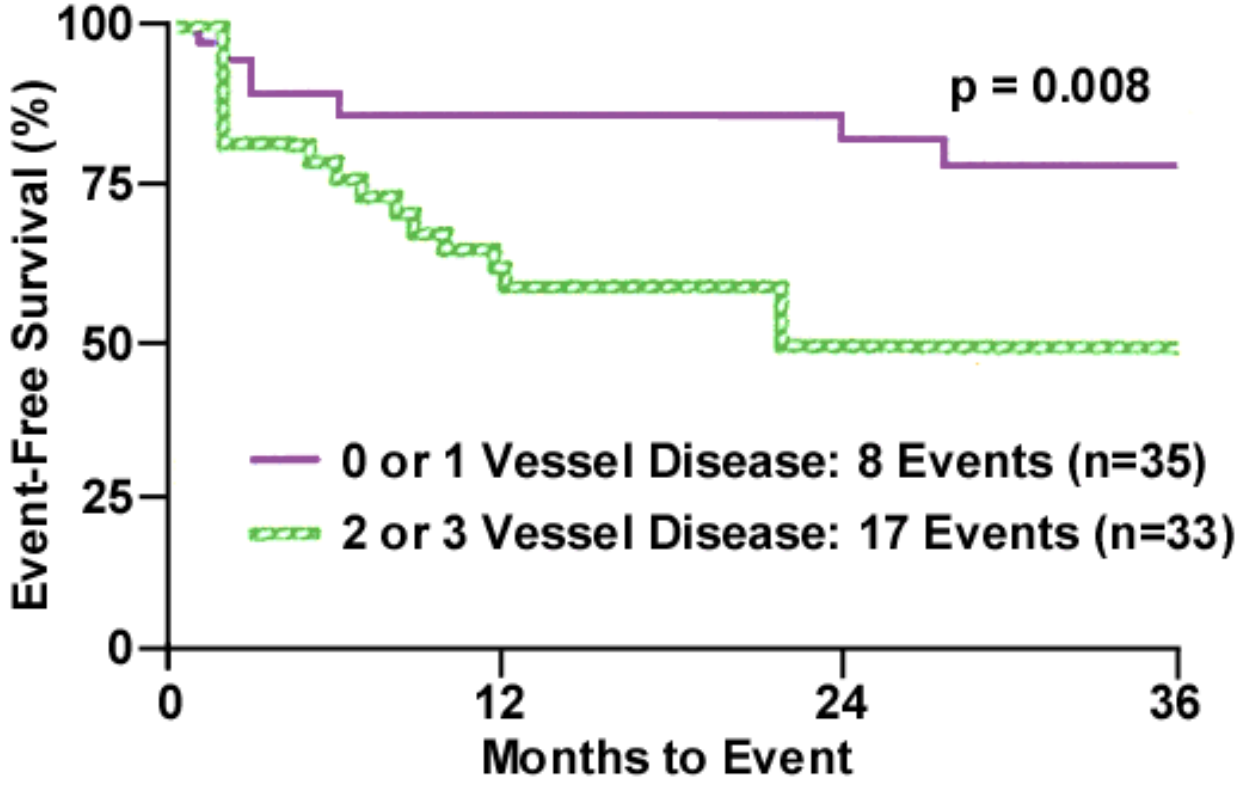
GISSI-2, 1993

Prognose

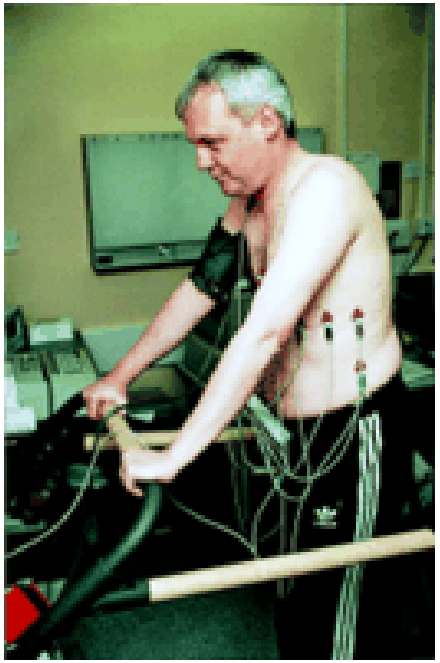

Left ventricular failure
p < 0.001
No left ventricular failure
Left ventricular failure
Q-wave infarction
p = 0.06
Non Q wave
Q wave
Age
p < 0.001
Age ≤ 60 years
Age > 60 years
Previous myocardial infarction
p = 0.003
No previous infarction
Previous infarction
Sex
p < 0.001
Males
Females
Diabetes
p = 0.002
No diabetes
Diabetes
Survival probability
1
0.75
0.5
0.25
0
0
50
100
150
200
Weeks from infarct

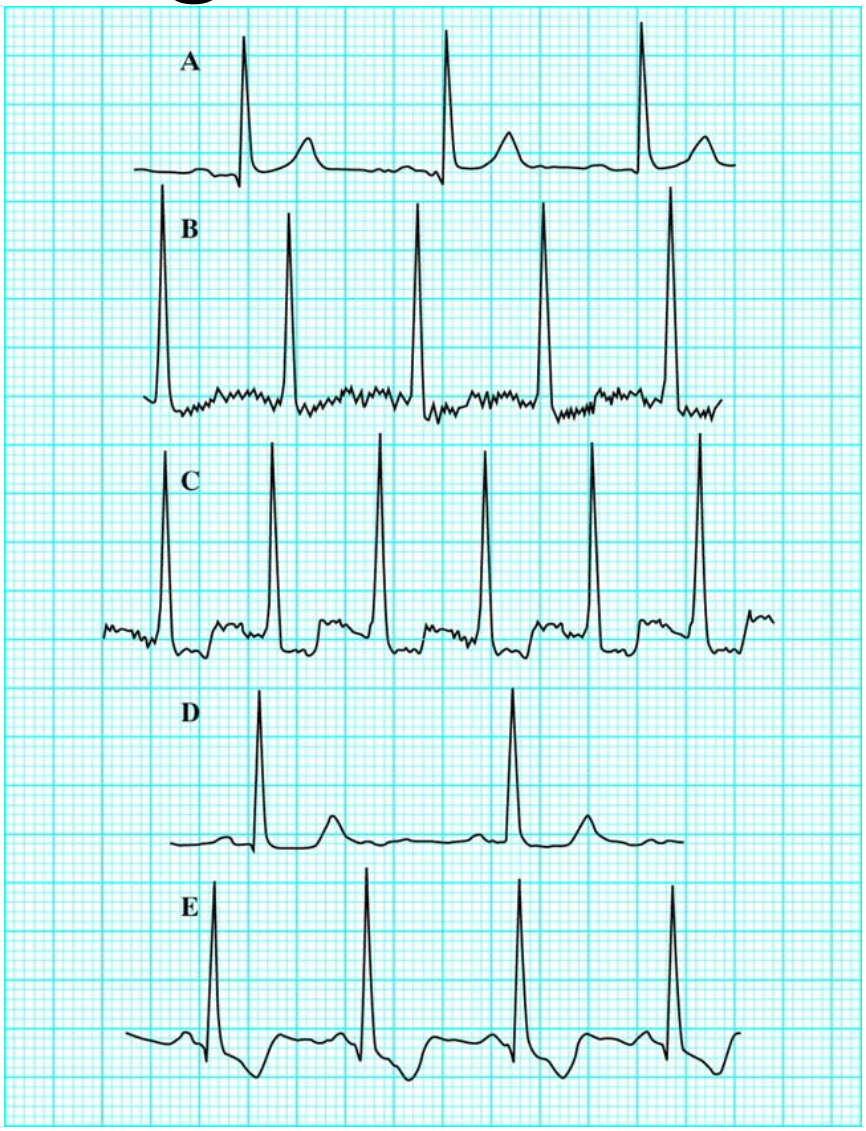
Prognose

Single-Vessel vs. Multiple-Vessel Disease



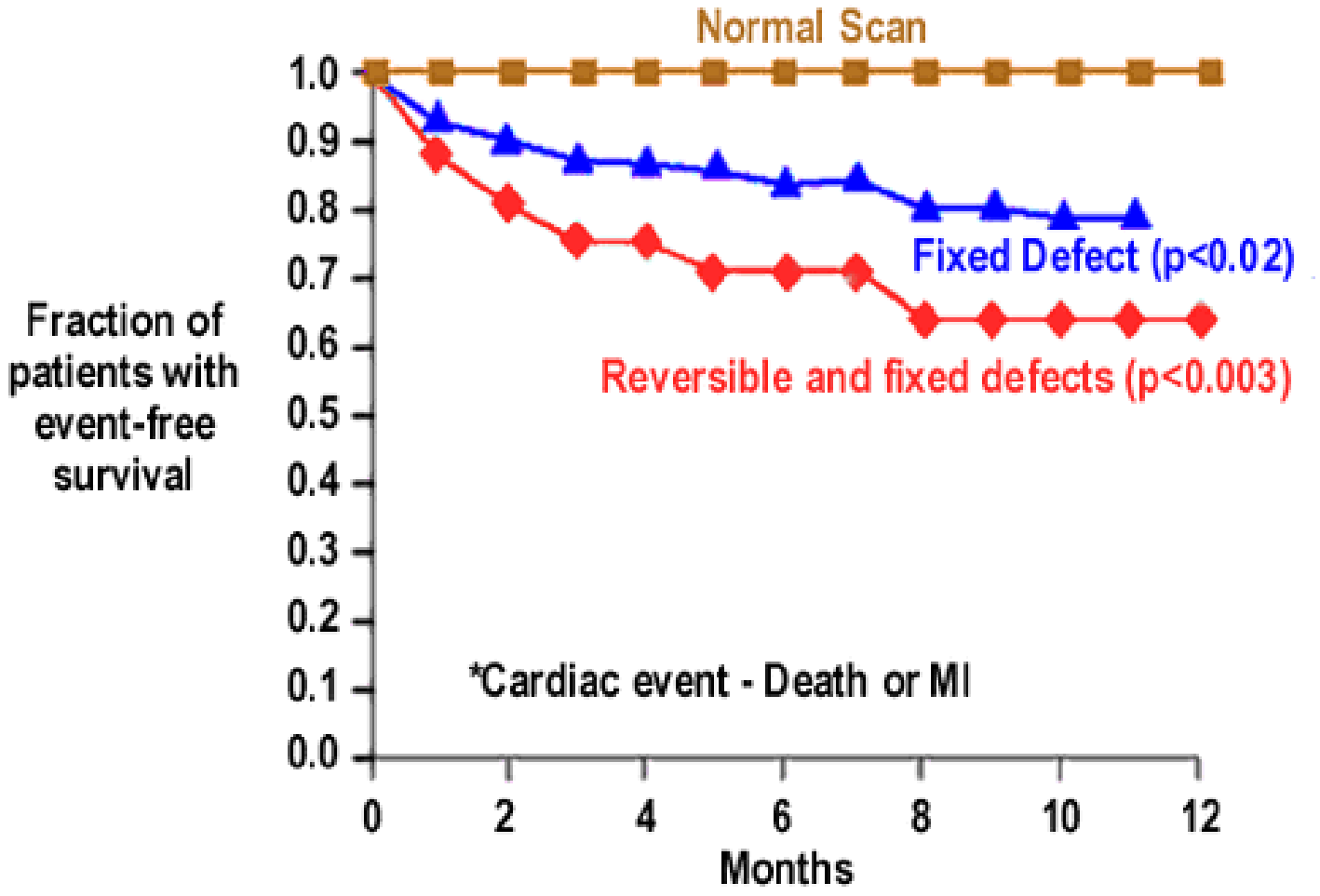
Prognose



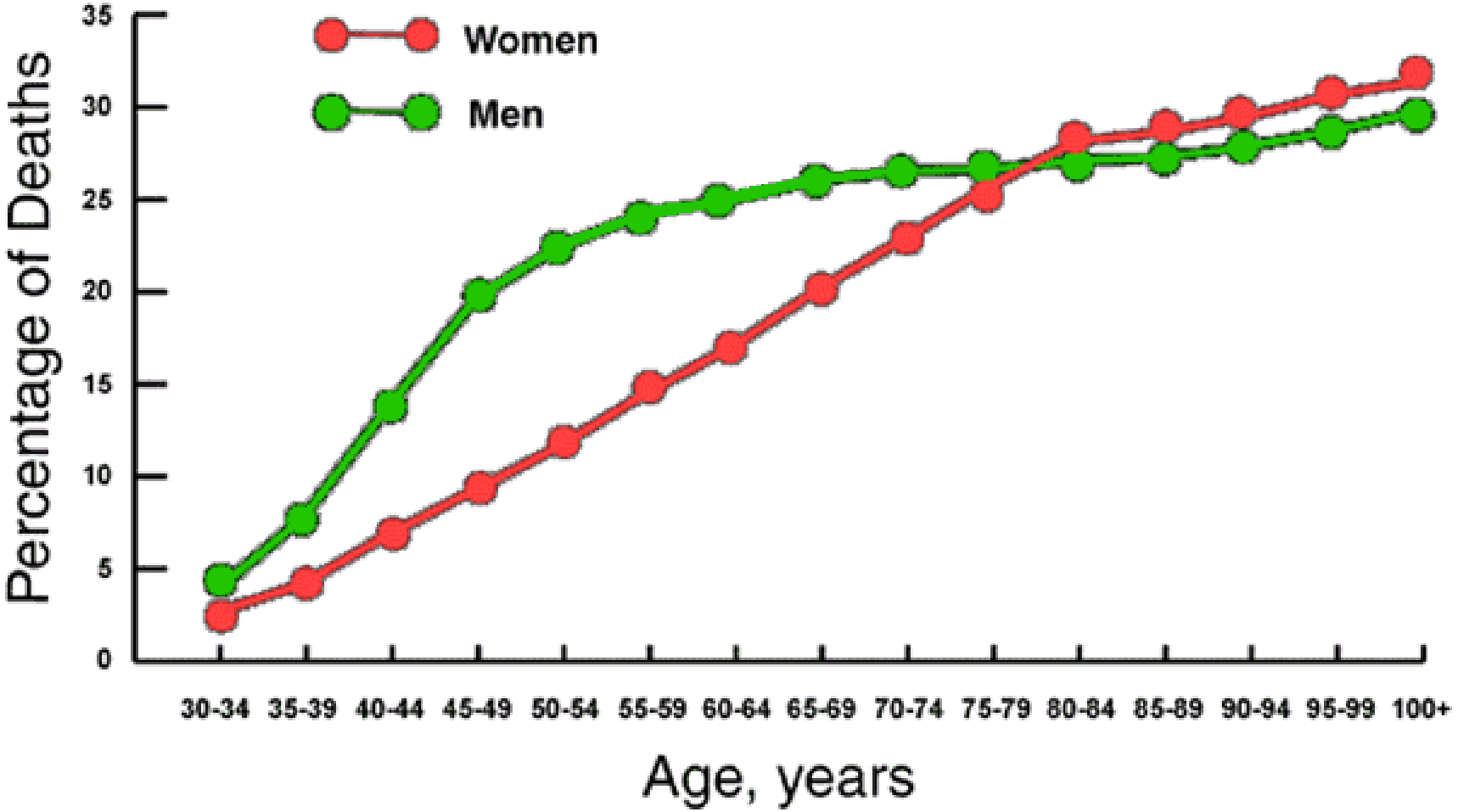


Prognose

MIBI scintigrafie

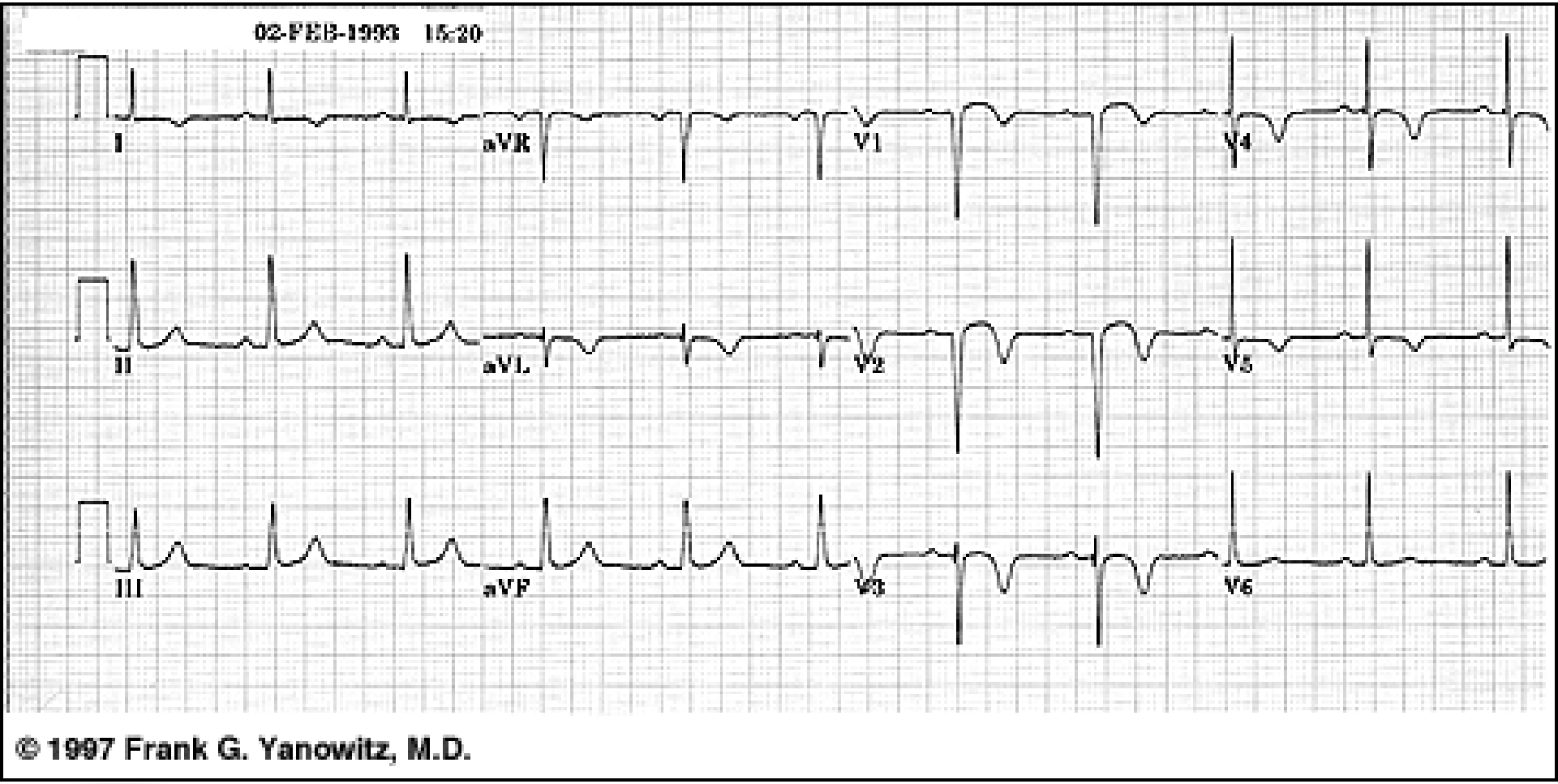


Prognose

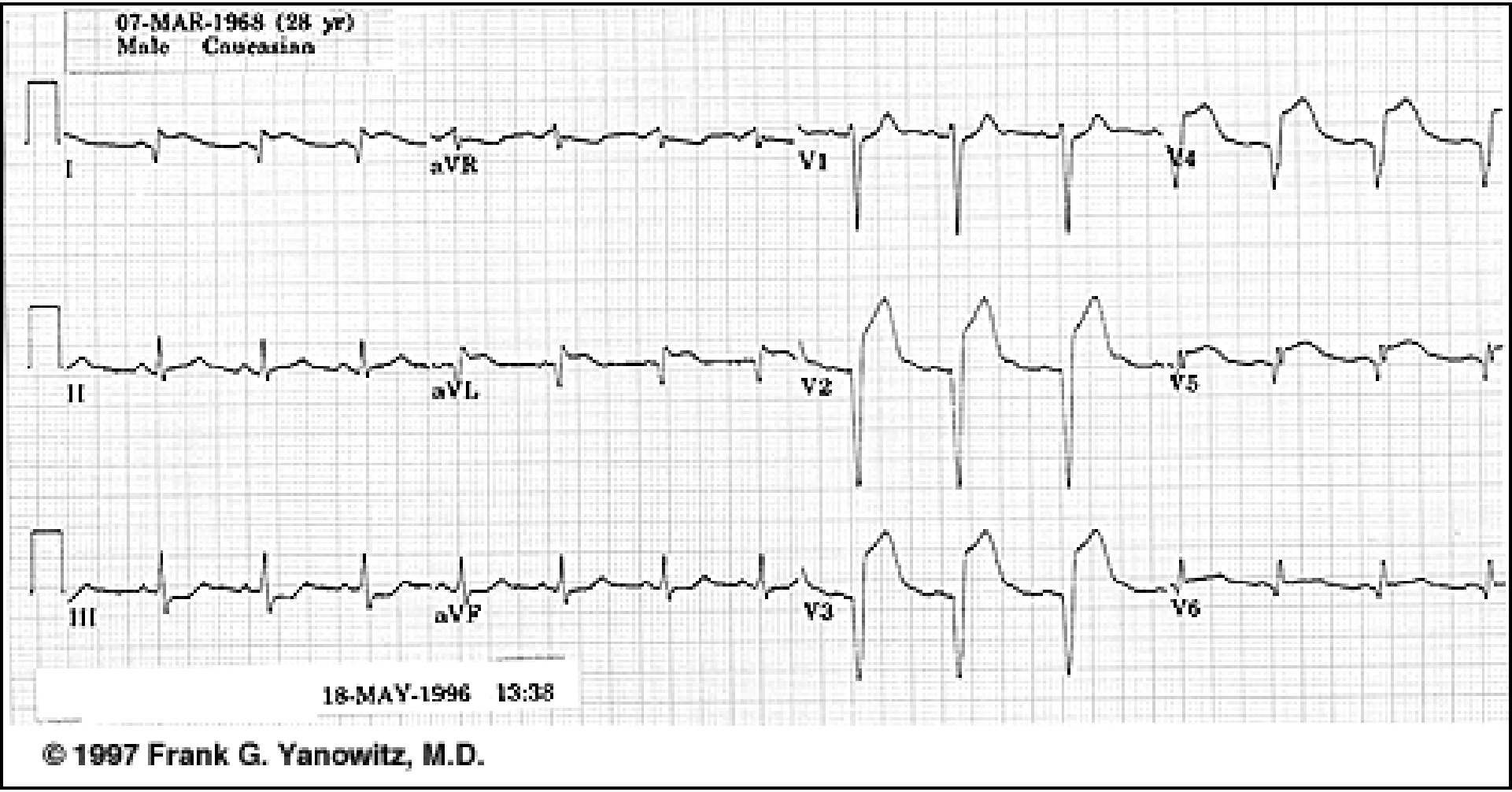

Women
Men
Percentage of Deaths
0
5
10
15
20
25
30
35
30-34
35-39
40-44
45-49
50-54
55-59
60-64
65-69
70-74
75-79
80-84
85-89
90-94
95-99
100+
Age, years

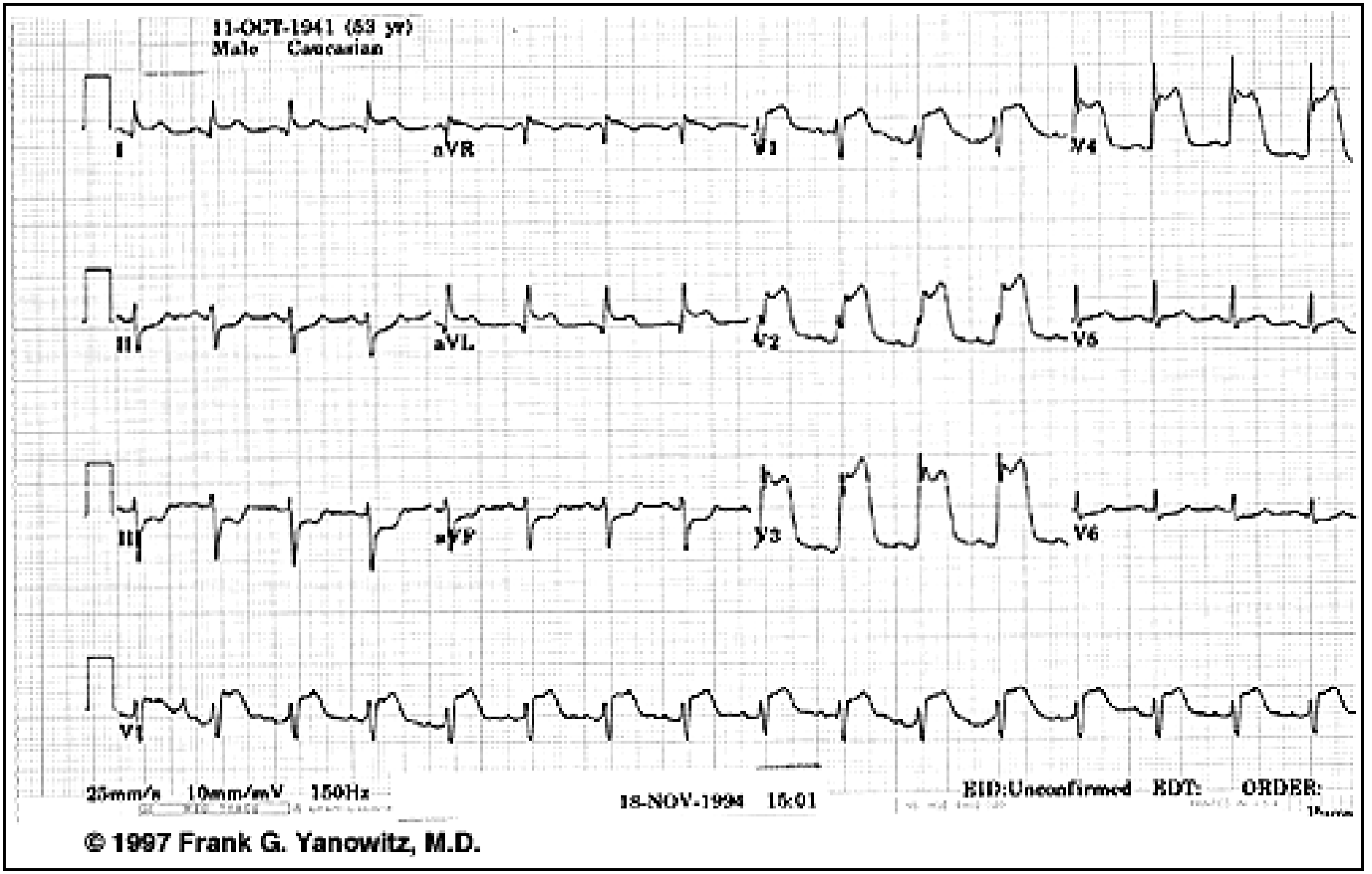
VOORBEELDEN

Doorgemaakt voorwandinfarct

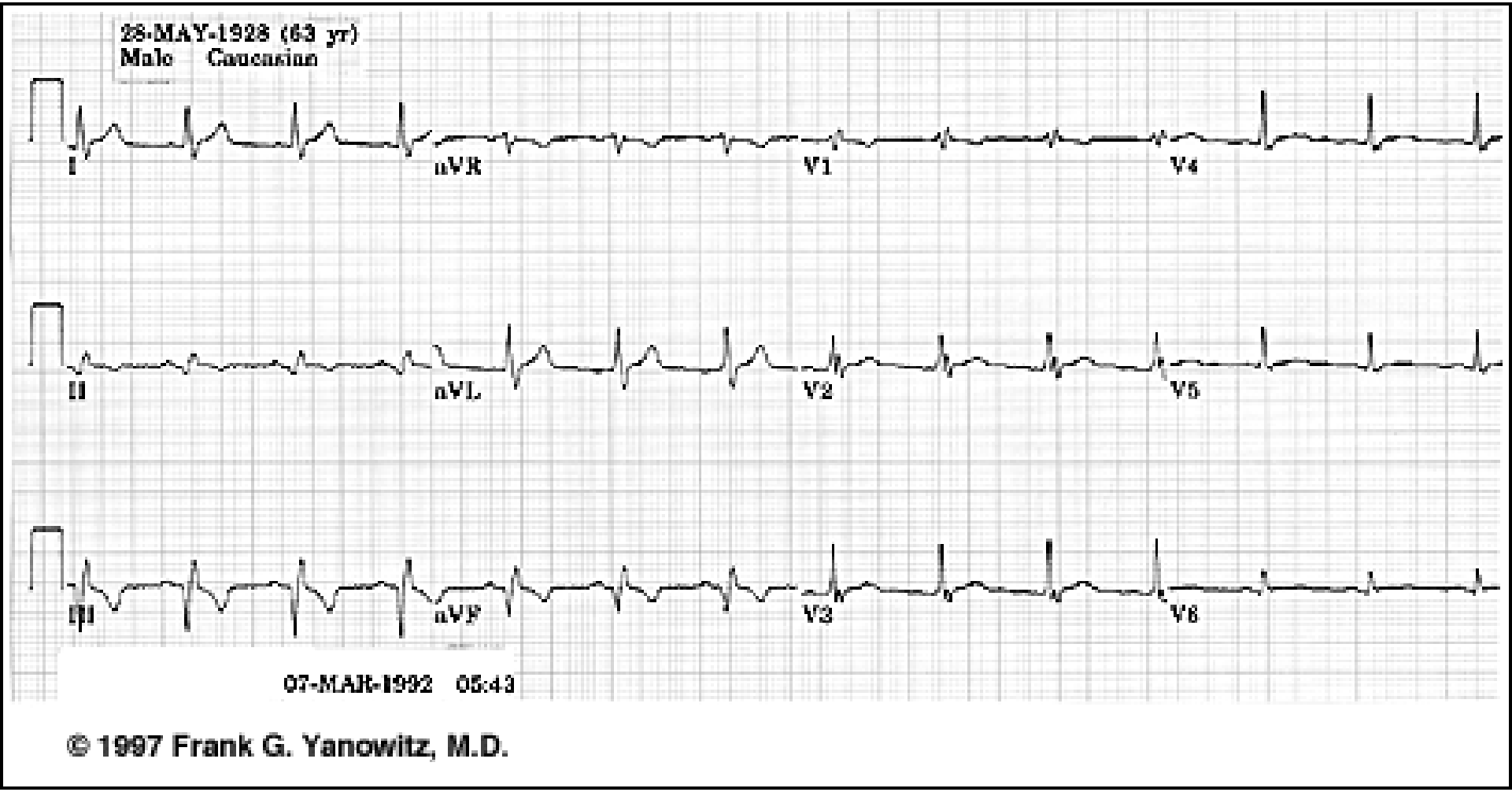


Semiacuut Anterior/lateraal

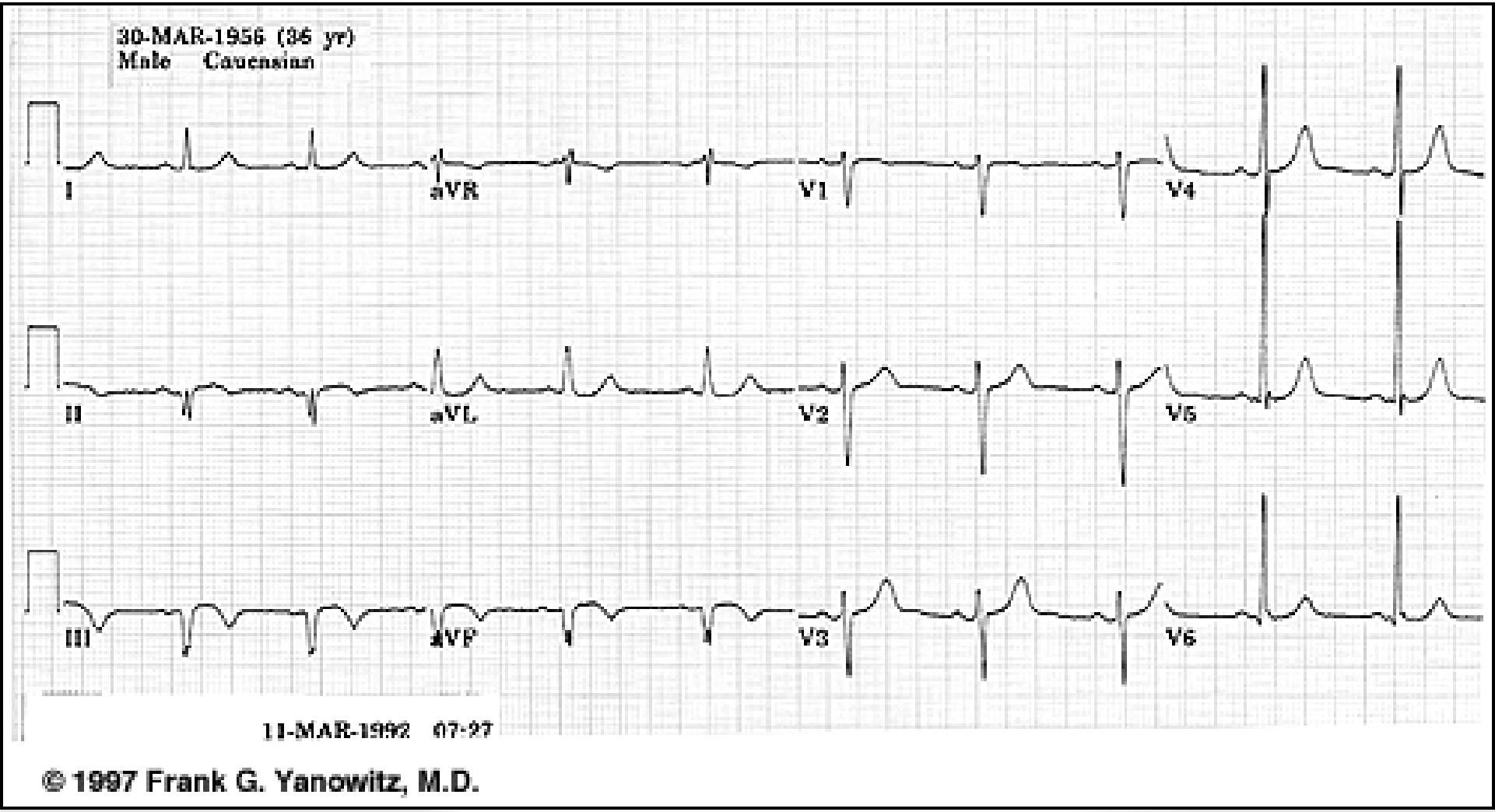


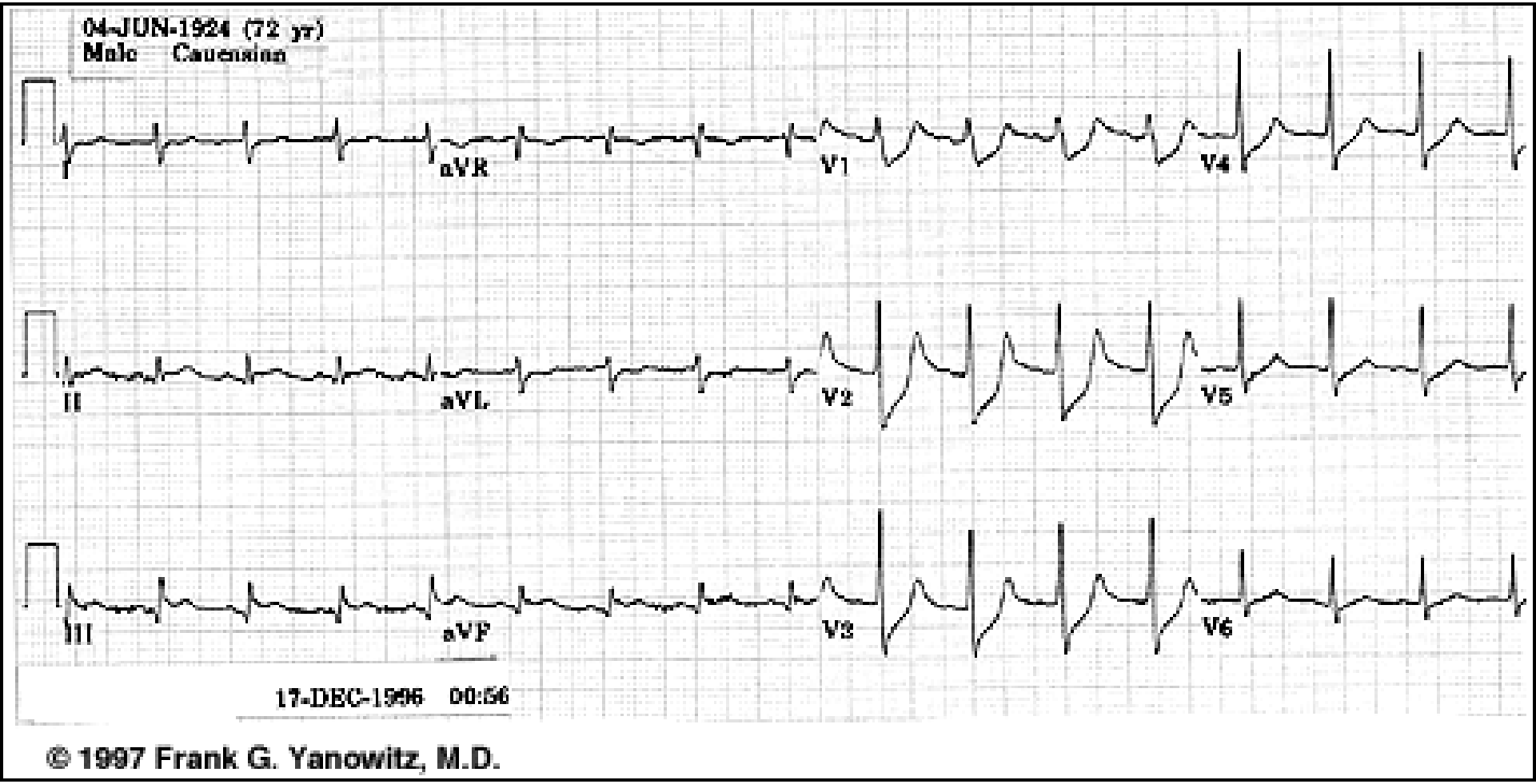

11-OCT-1941 (53 yr)
Male Caucasian
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
25mm/s 10mm/mV 150Hz
18-NOV-1994 15:01
EID:Unconfirmed RDT: ORDER:
© 1997 Frank G. Yanowitz, M.D.

Semirecent inferoposterior infarct

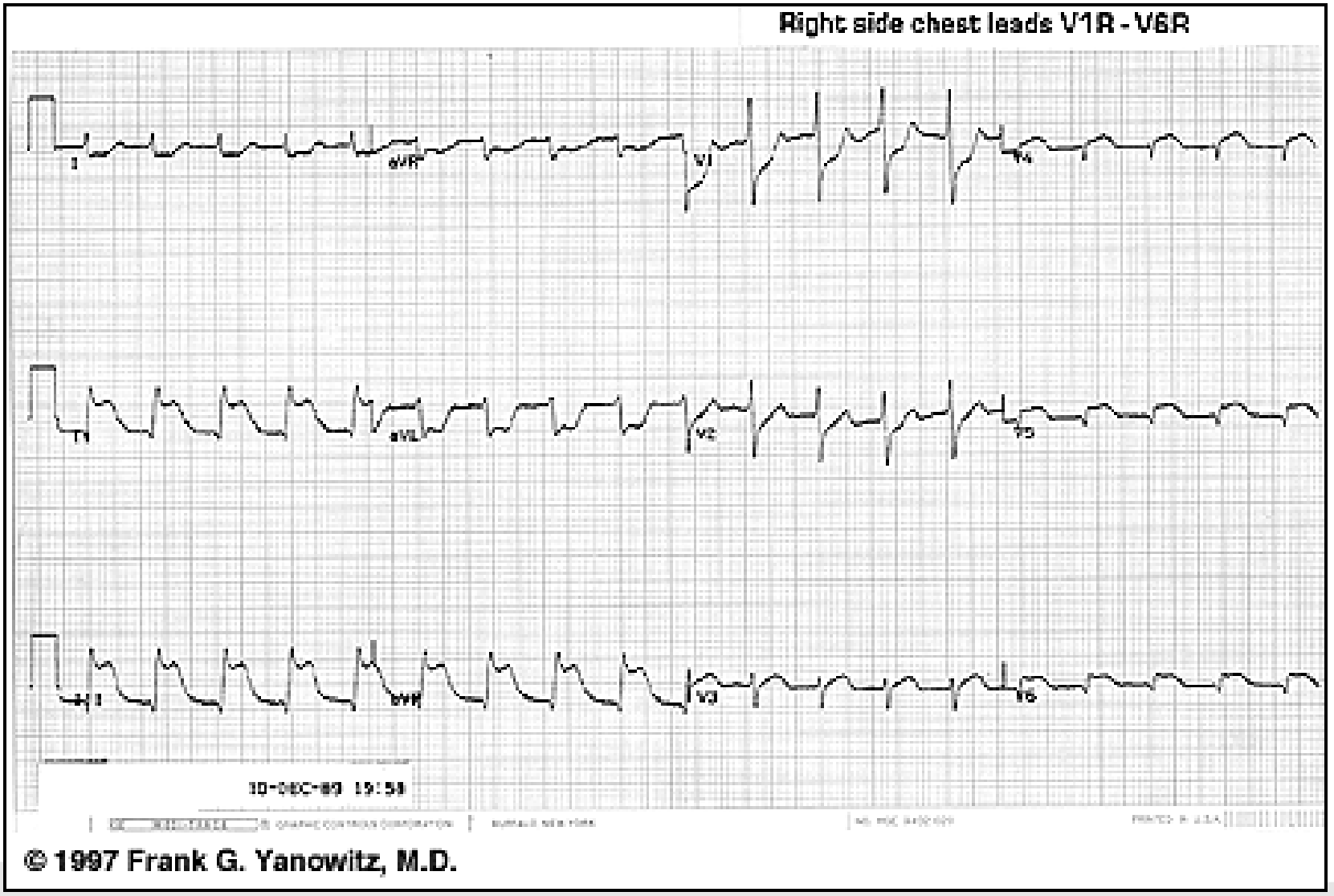


Doorgemaakt onderwand infarct

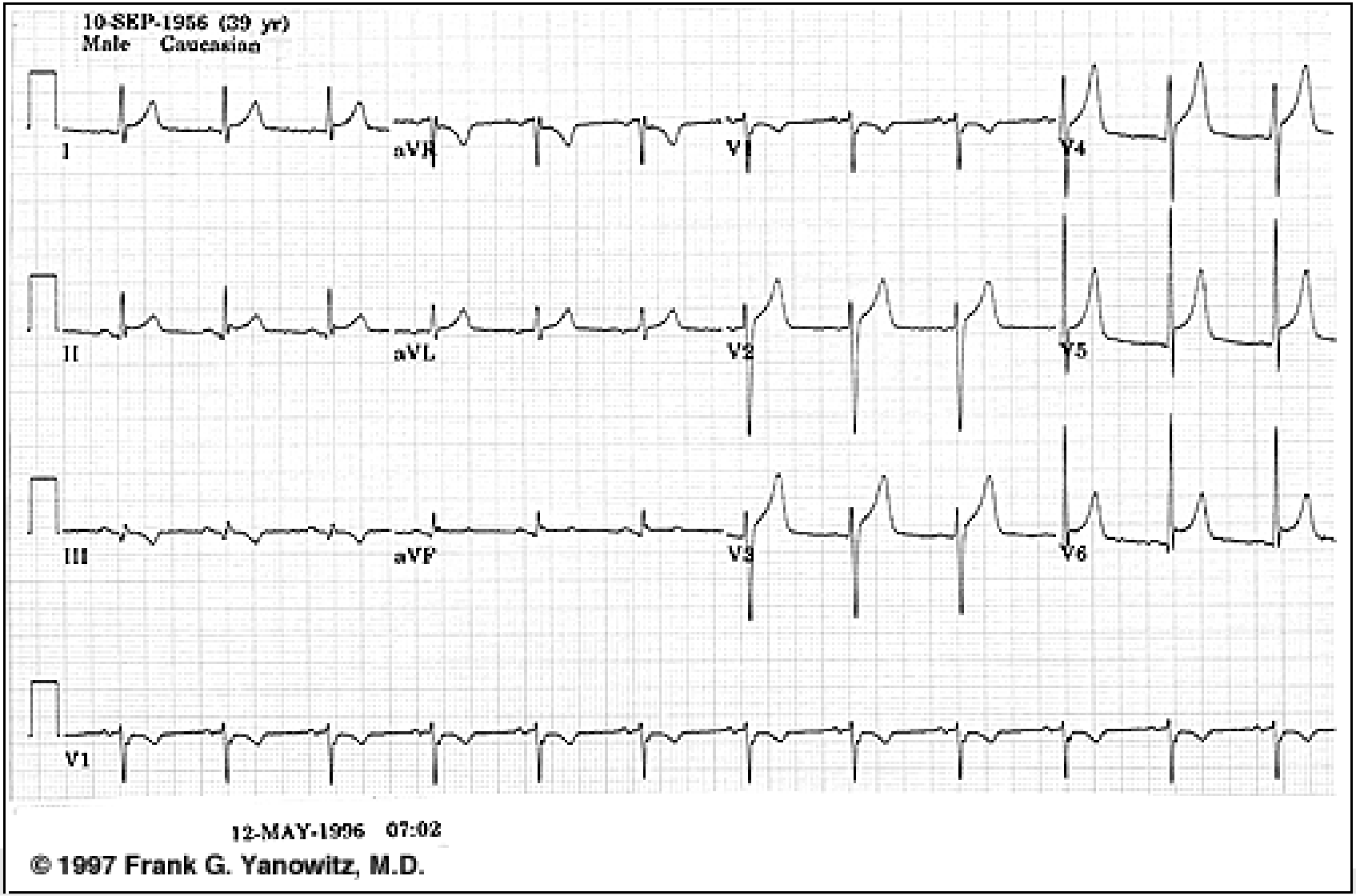




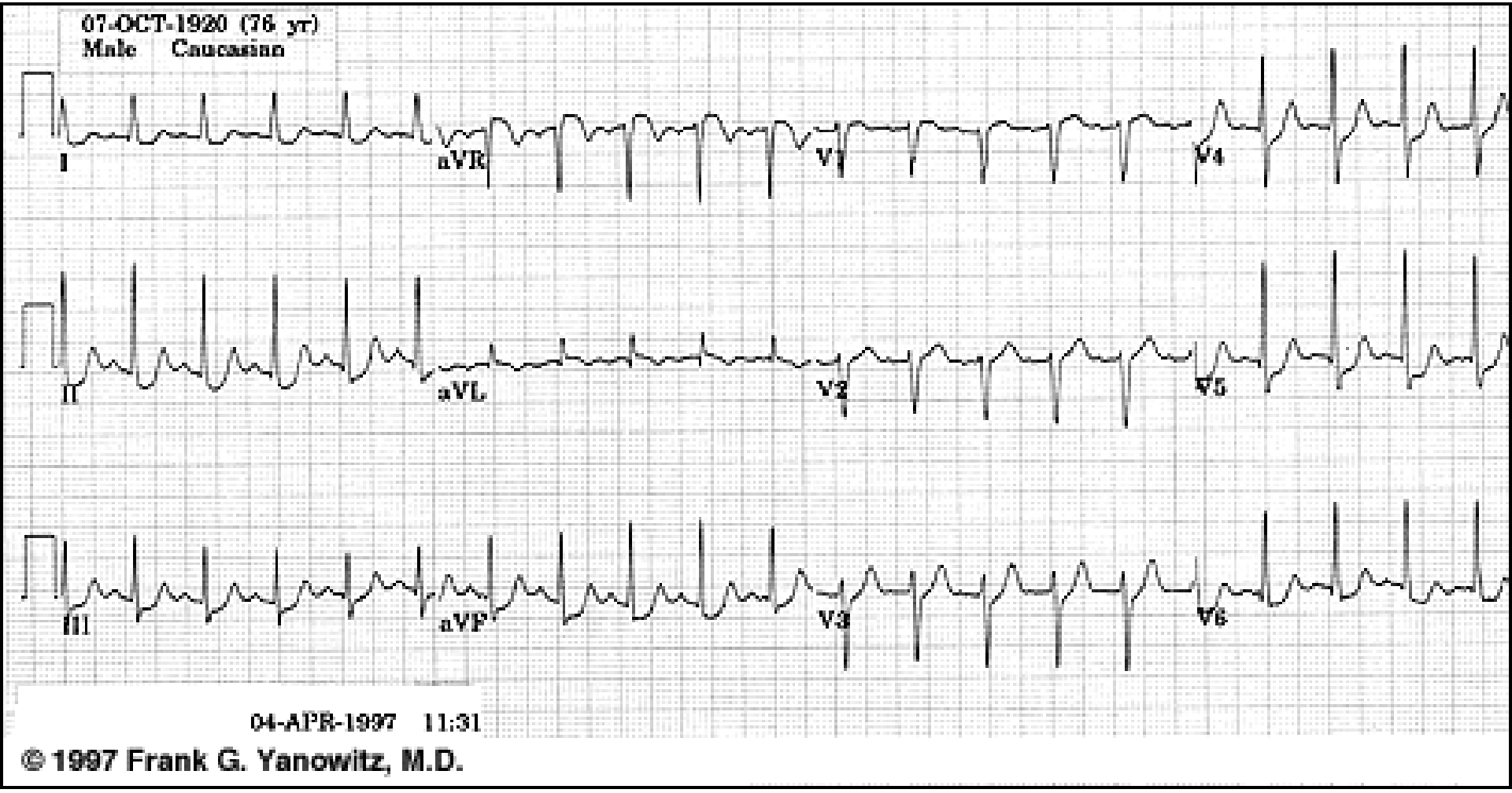
Acuut onderwandinfarct met RV uitbreiding



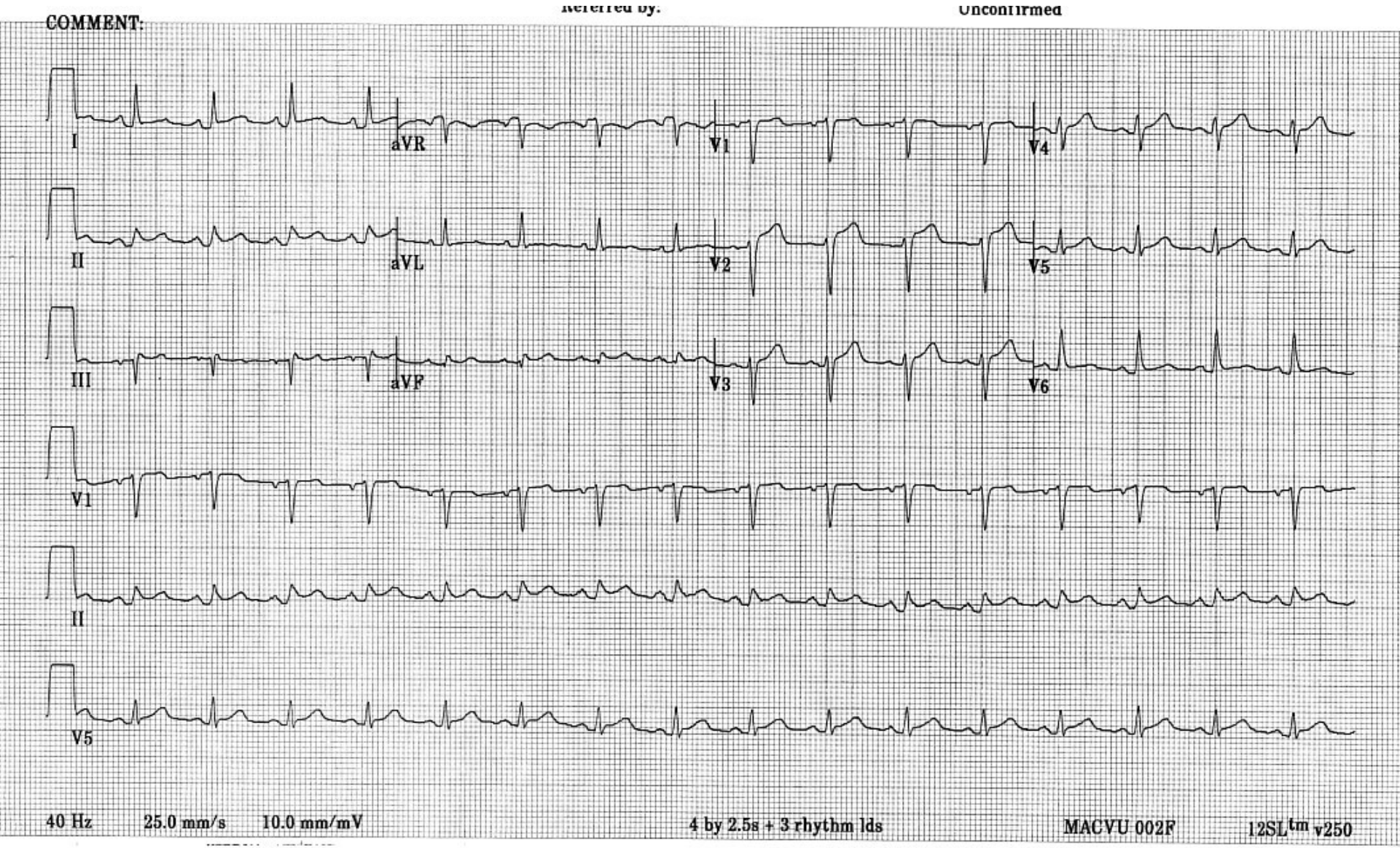
Vroege repolarisatie(normaal)

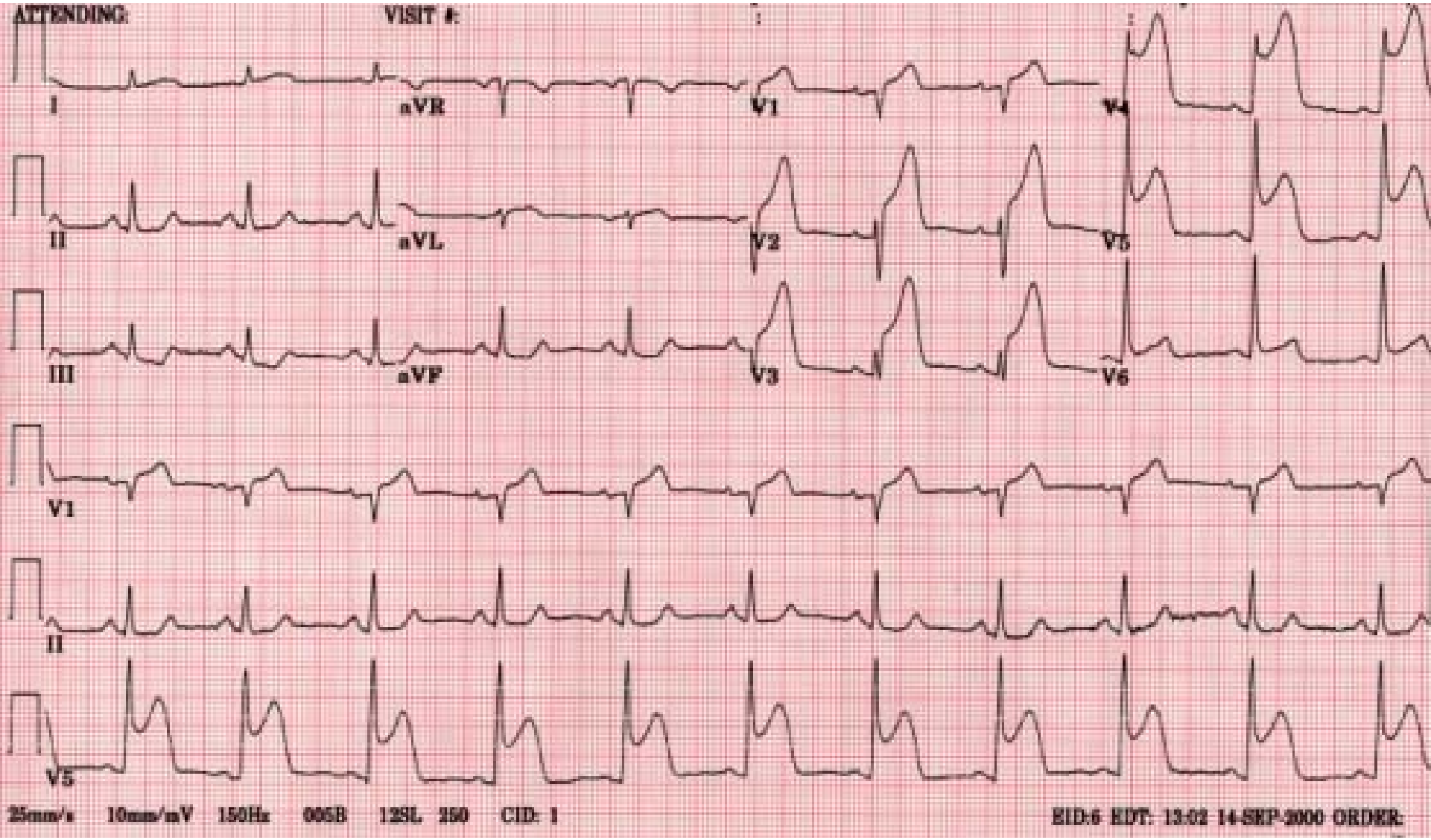


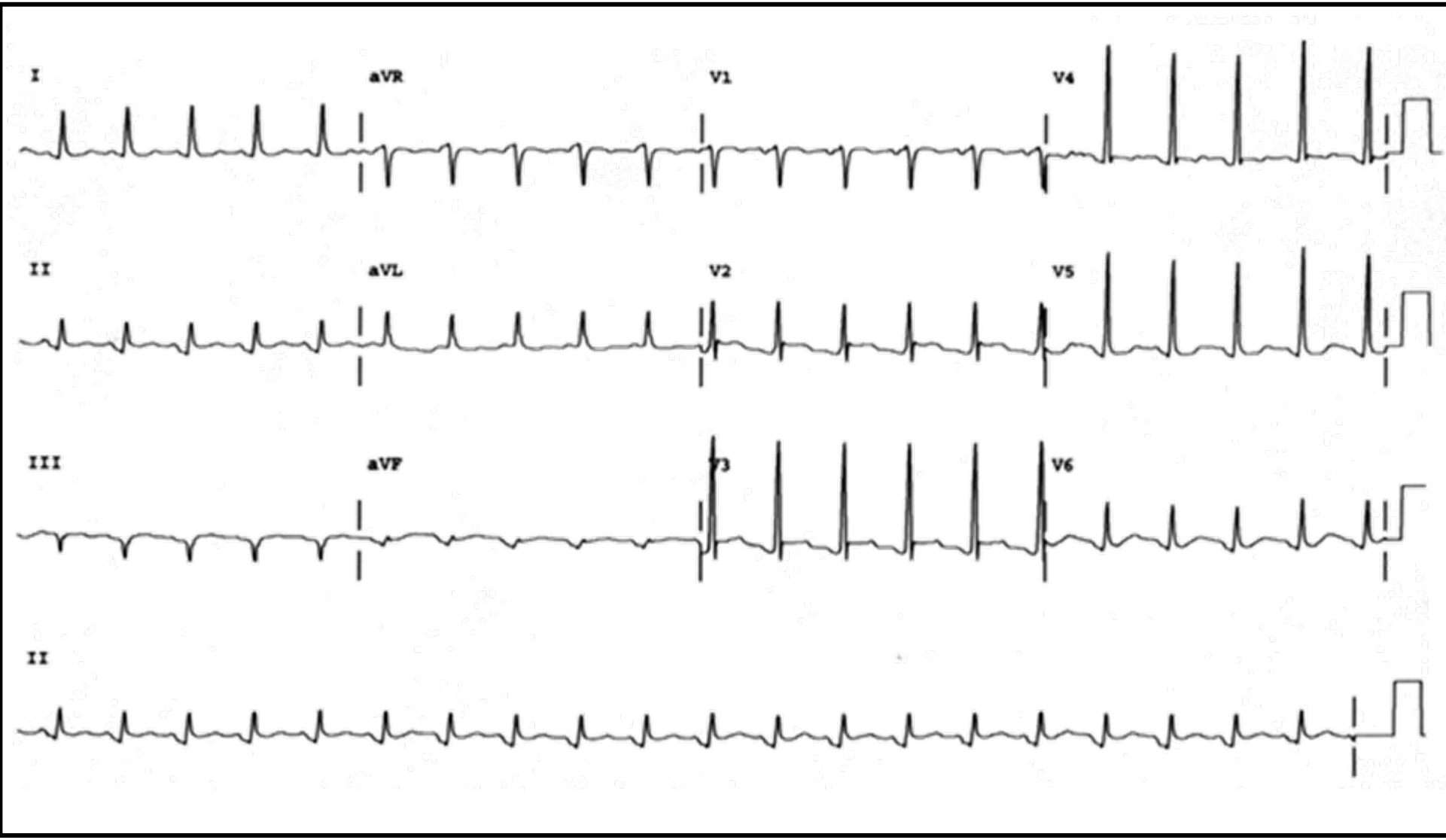
Mogelijk hoofdstamstenose



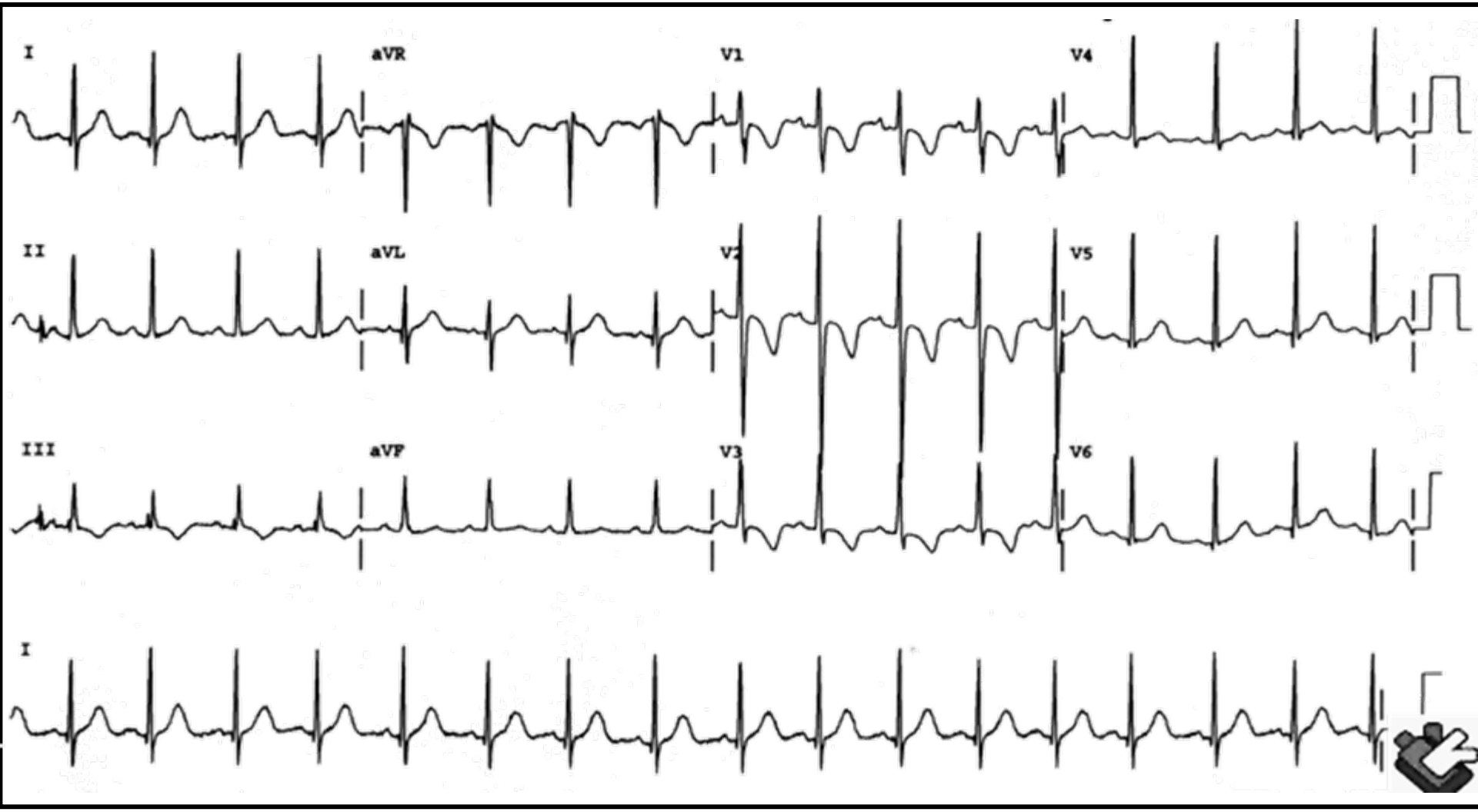
Pericarditis



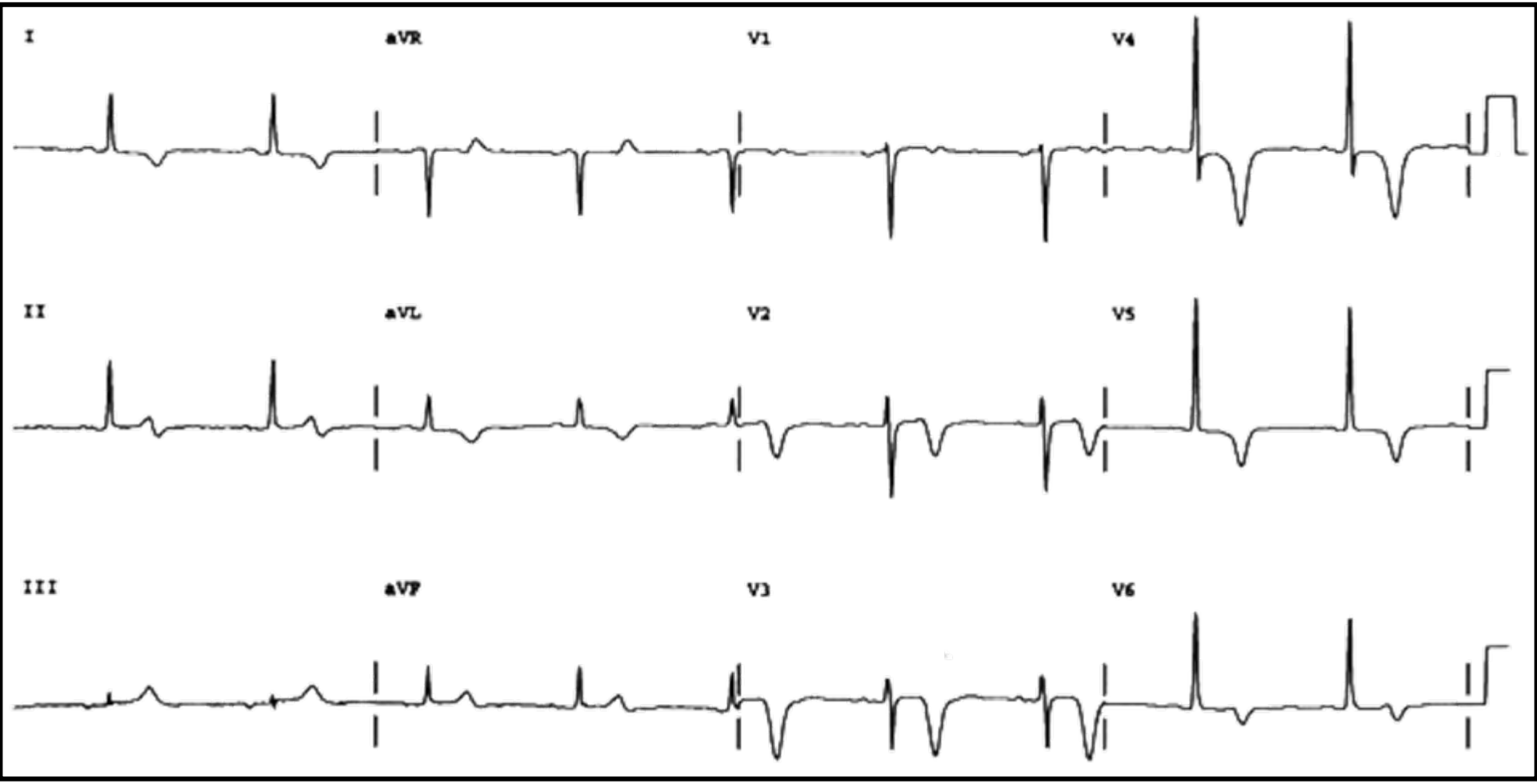

ATTENDING:
VISIT #:
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 150Hz 006B 12SL 250 CID: 1
EID:6 EDT: 13:02 14-SEP-2000 ORDER:


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

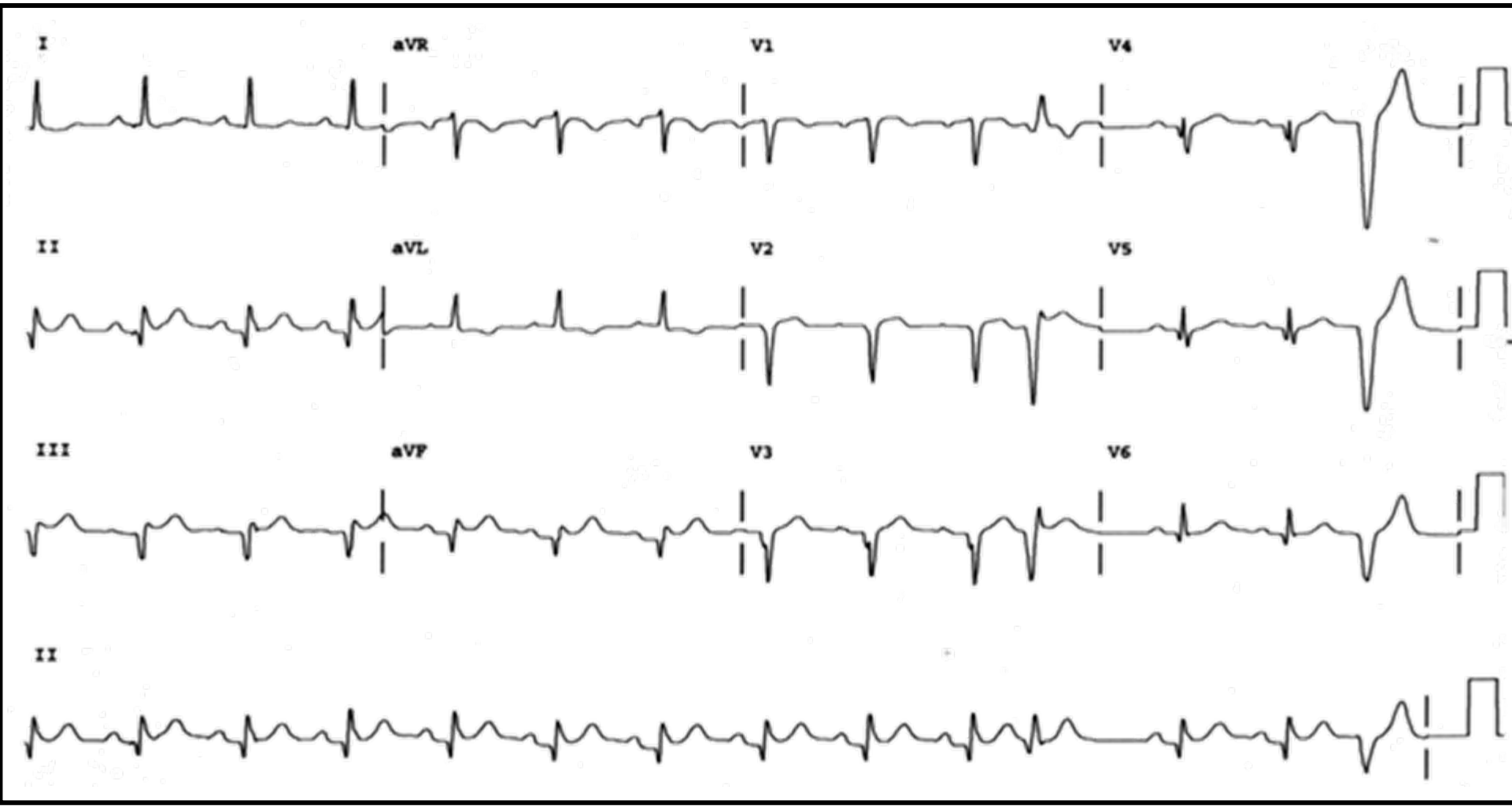
Waarschijnlijk Acuut posterior infarct. Doorpolen !!



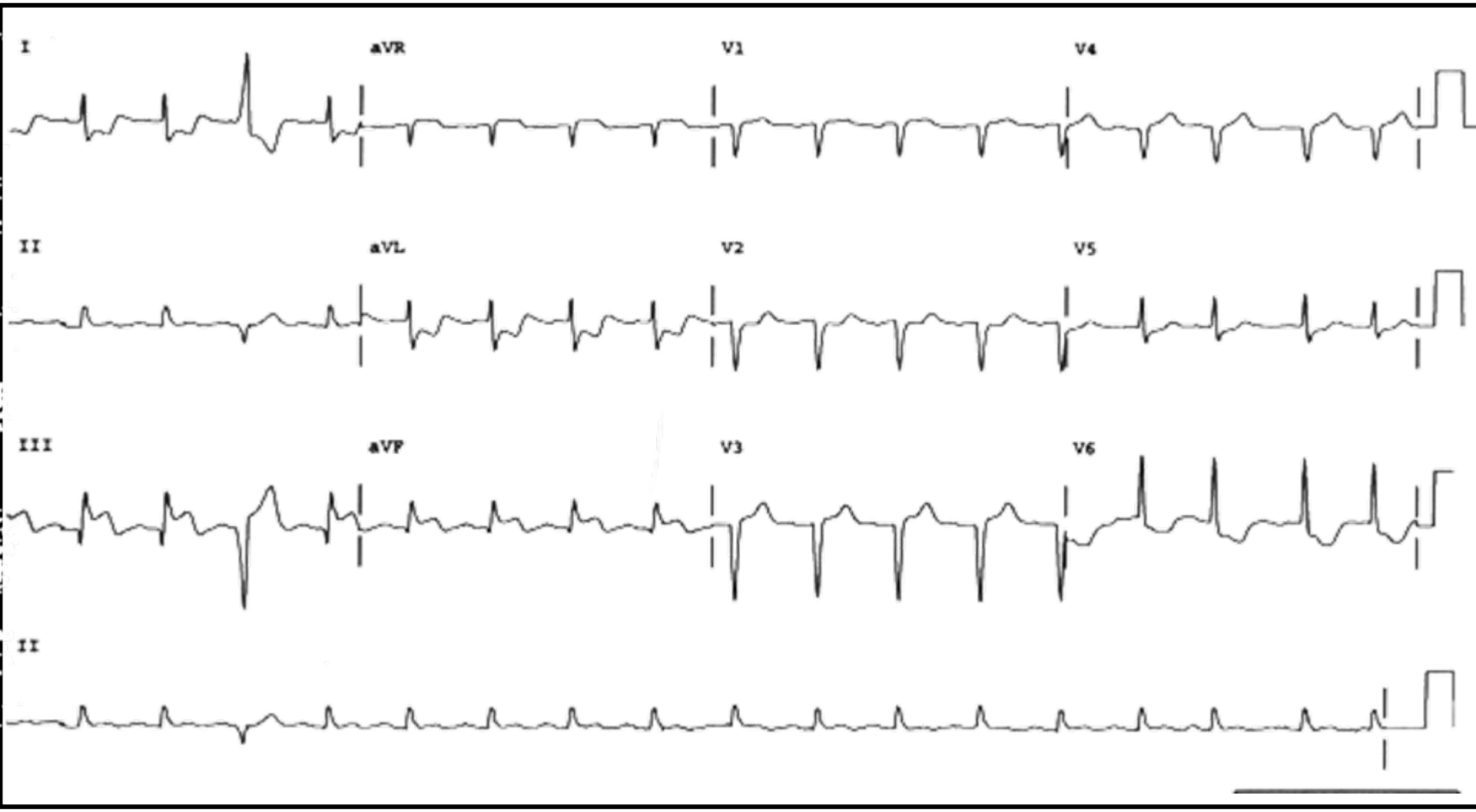
Anterior Non-Q-wave infarct (NSTEMI)



Semirecent inferior infarct, oud anteroseptaal infarct



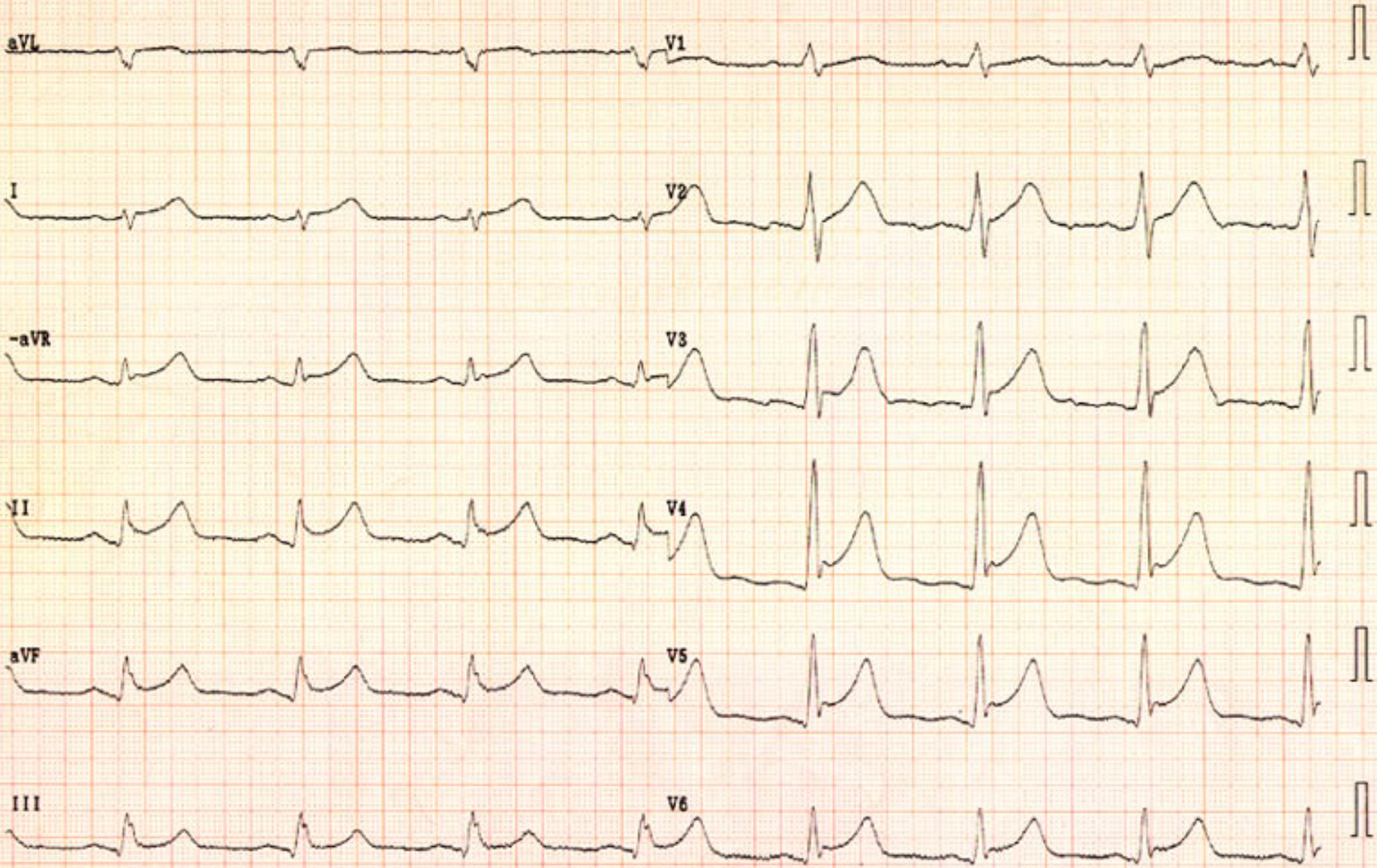
Acuut inferior infarct met reciproke depressies lateraal



ID: 751227- HF: 92 Datum: 97-09-26

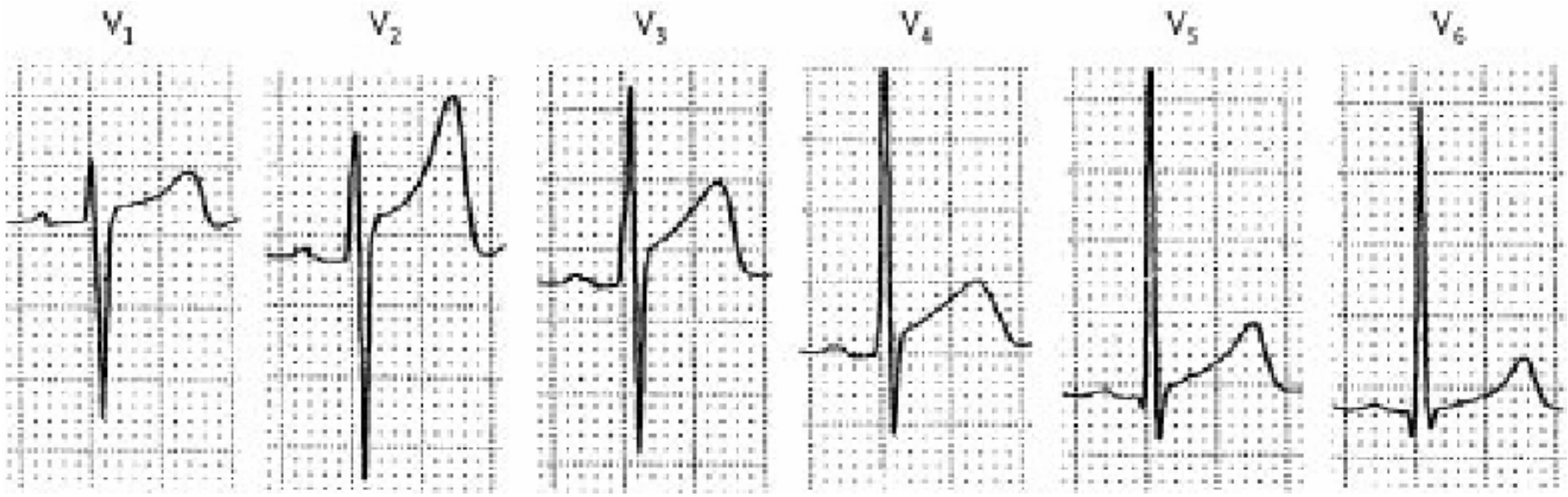
Förnamn: ALEXANDER

28a



50 mm/s 10 mm/mV 50Hz MEGACART

Abnormaal?



Approximately 90 percent of healthy young men have ST-segment elevation of 1 to 3 mm in one or more precordial leads. The ST segment is concave.

ST segment

- Hoe beoordeel je een ST-segment

  – 1)

  Kijk naar ST shift

  – 2)

  Kijk naar ST segment

MAAR…. je kan een ECG beoordeling niet zonder anamnese doen

ST segment

- Definitie van normaal ST segment?

- Definitie van abnomaal ST segment?
  - Wanneer spreek je van een ST elevatie?
  - Wanneer spreek je van een ST depressie?

Normaal ST- segment

- ≤ 1 mm ST shift onder of boven iso-elektrische lijn

MAAR….., wat is de iso-elektrische lijn?

En op welk punt meet je het ST-segment?

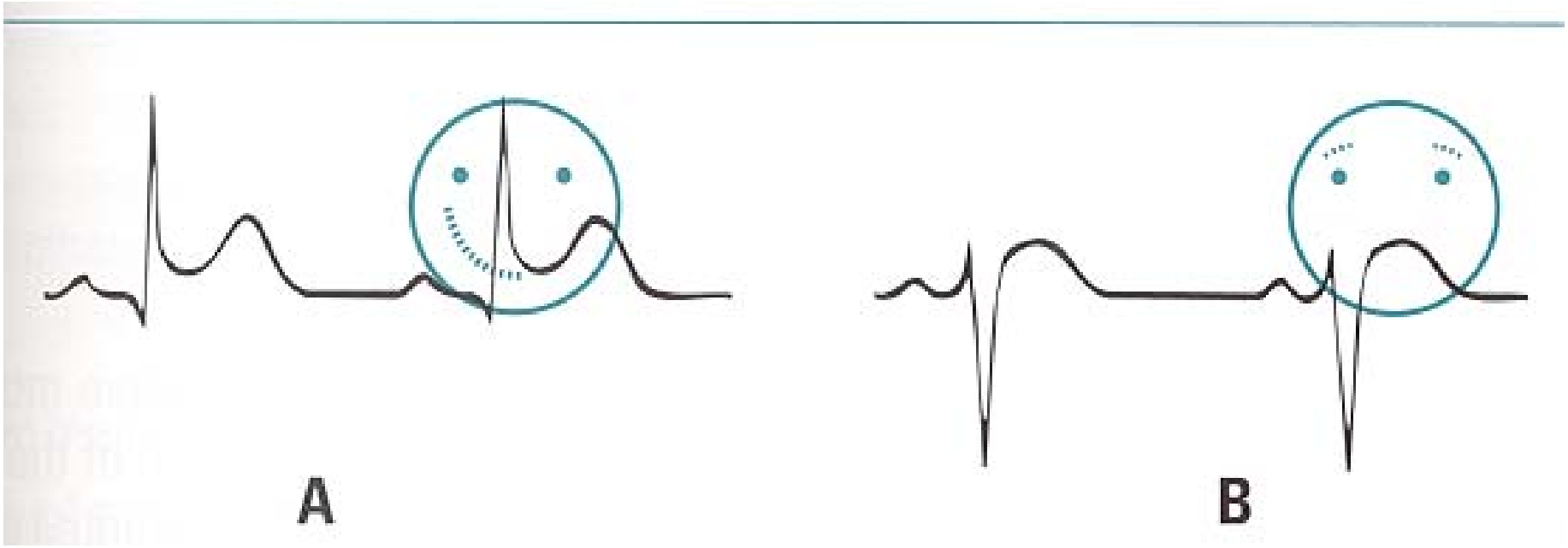
PR segment	60-80 msec na punt J

Normaal ST segment

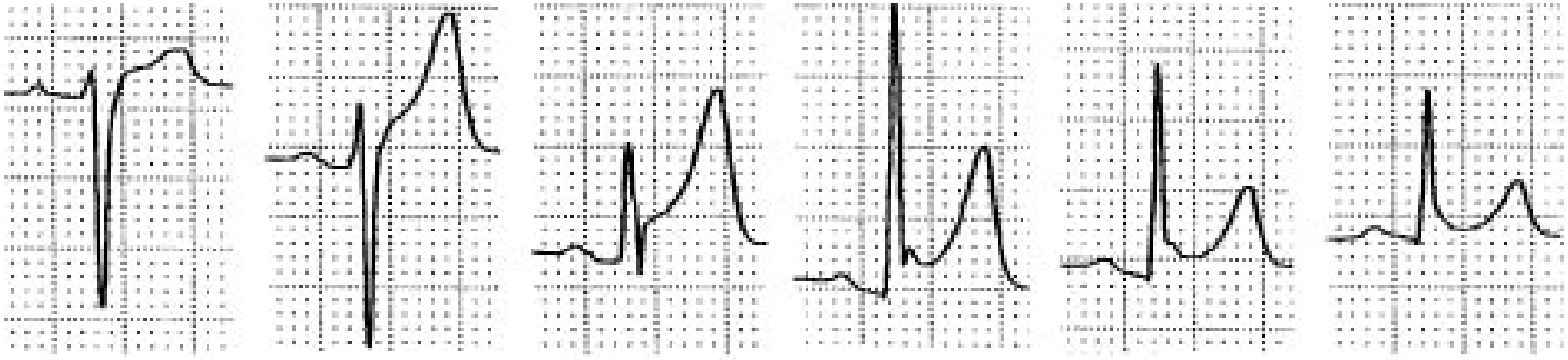
Prevalence of Male and Female Patterns of Early Ventricular Repolarization in the Normal ECG of Males and Females From Childhood to Old Age

J Am Coll Cardiol 2002;40:1870–6

ST - segment elevatie
Vorm (concaaf of convex)

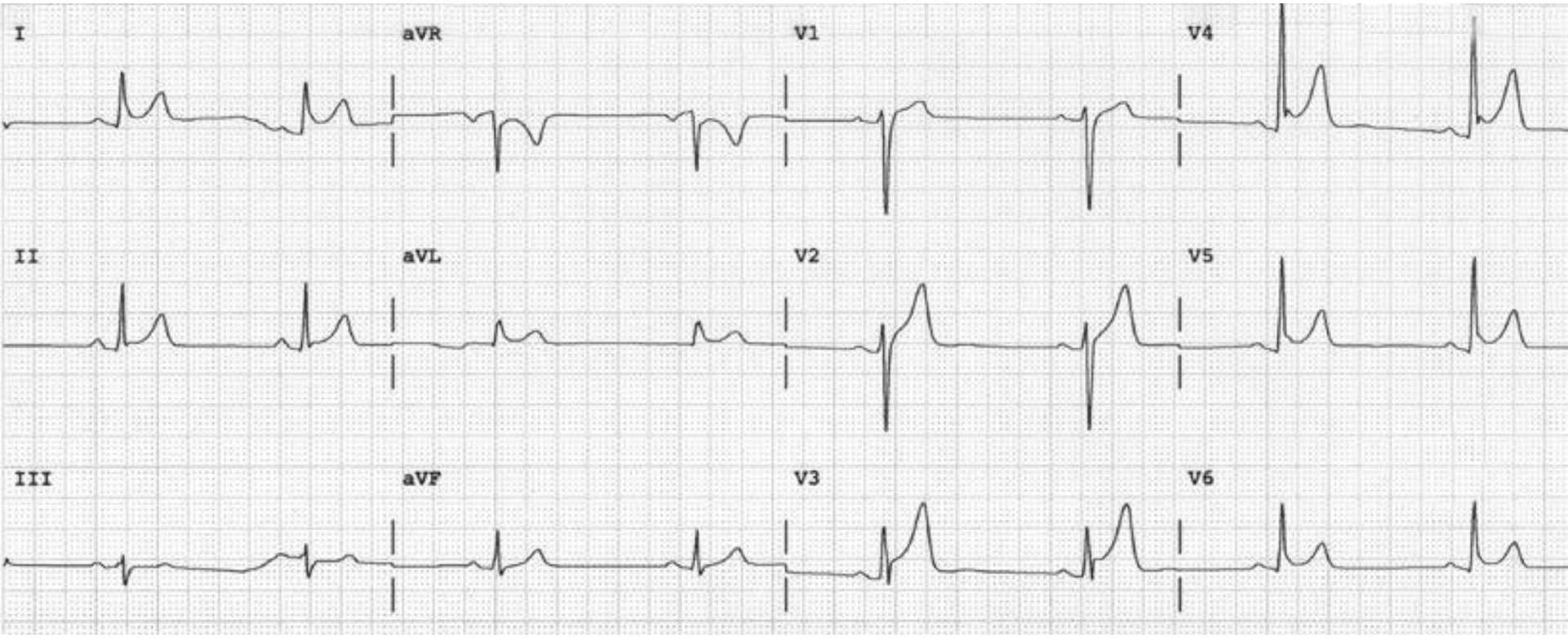


Abnormaal?

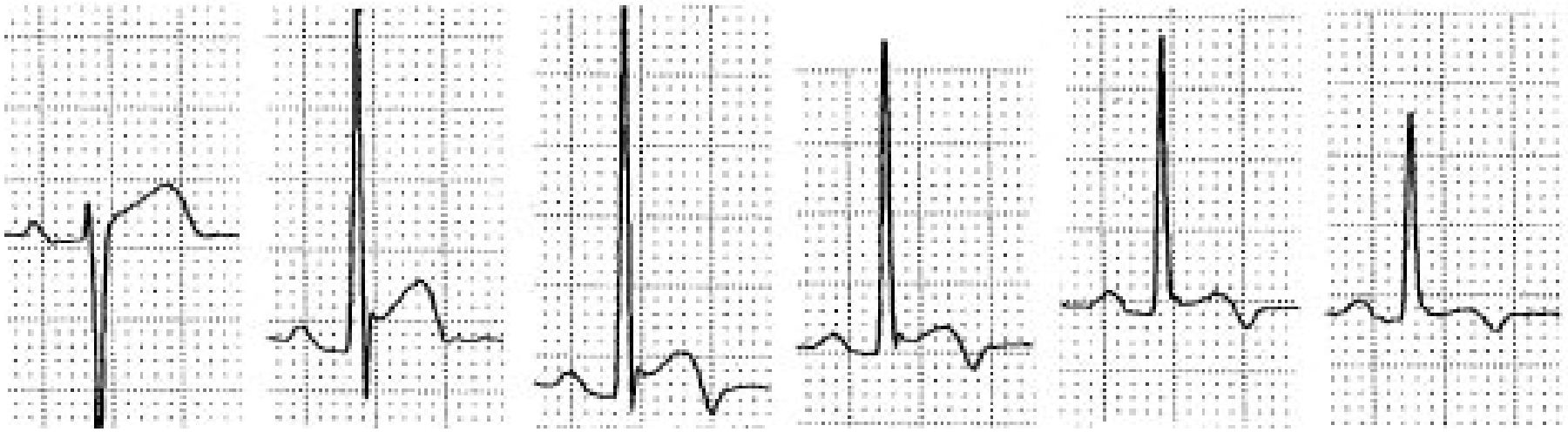


ST elevatie

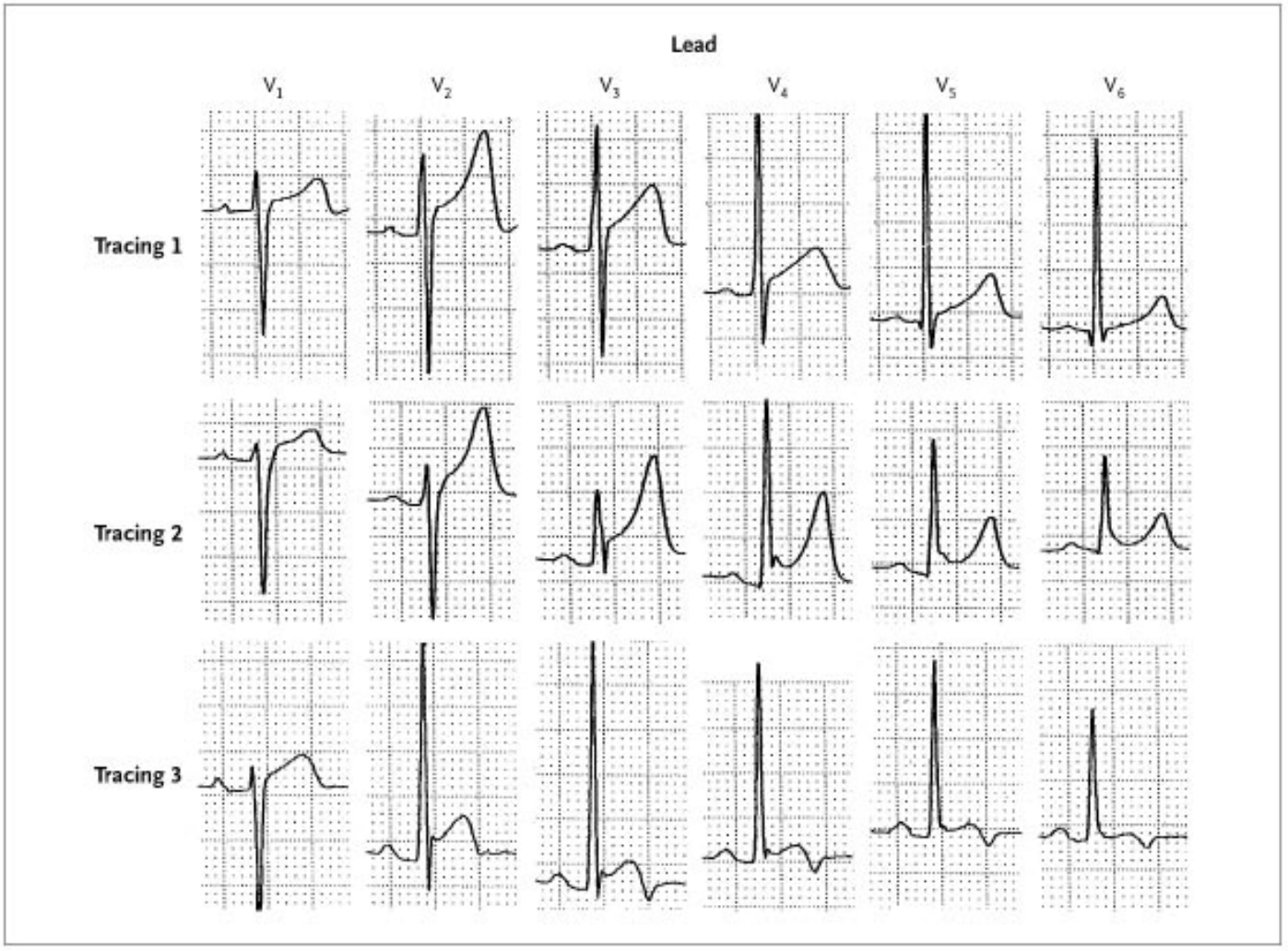
- **Vroege repolarisatie**
  - **Zeer frequente bevinding**
  - **“Smiley”configuratie**
  - **Overigens gezonde asymptomatische jonge volwassene**
  - **Vaak in voorwands afleidingen**
  - **Notching J punt**
  - **Geen Q**
  - **Geen reciproke ST depressie**


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Abnormaal?



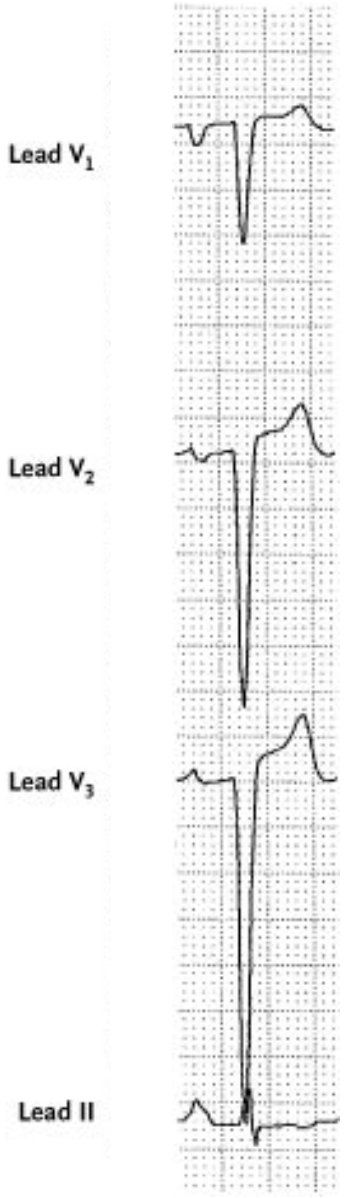
(Varianten van) normaal



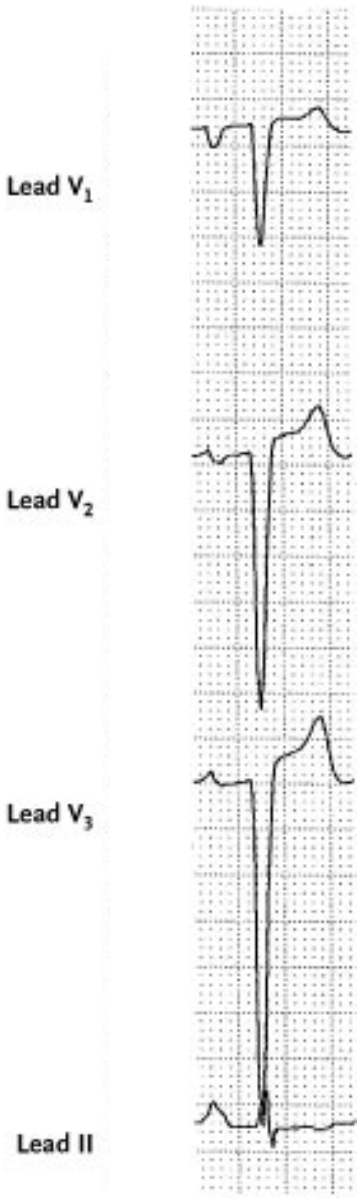
ST segment elevatie

- Differentiaal diagnose?

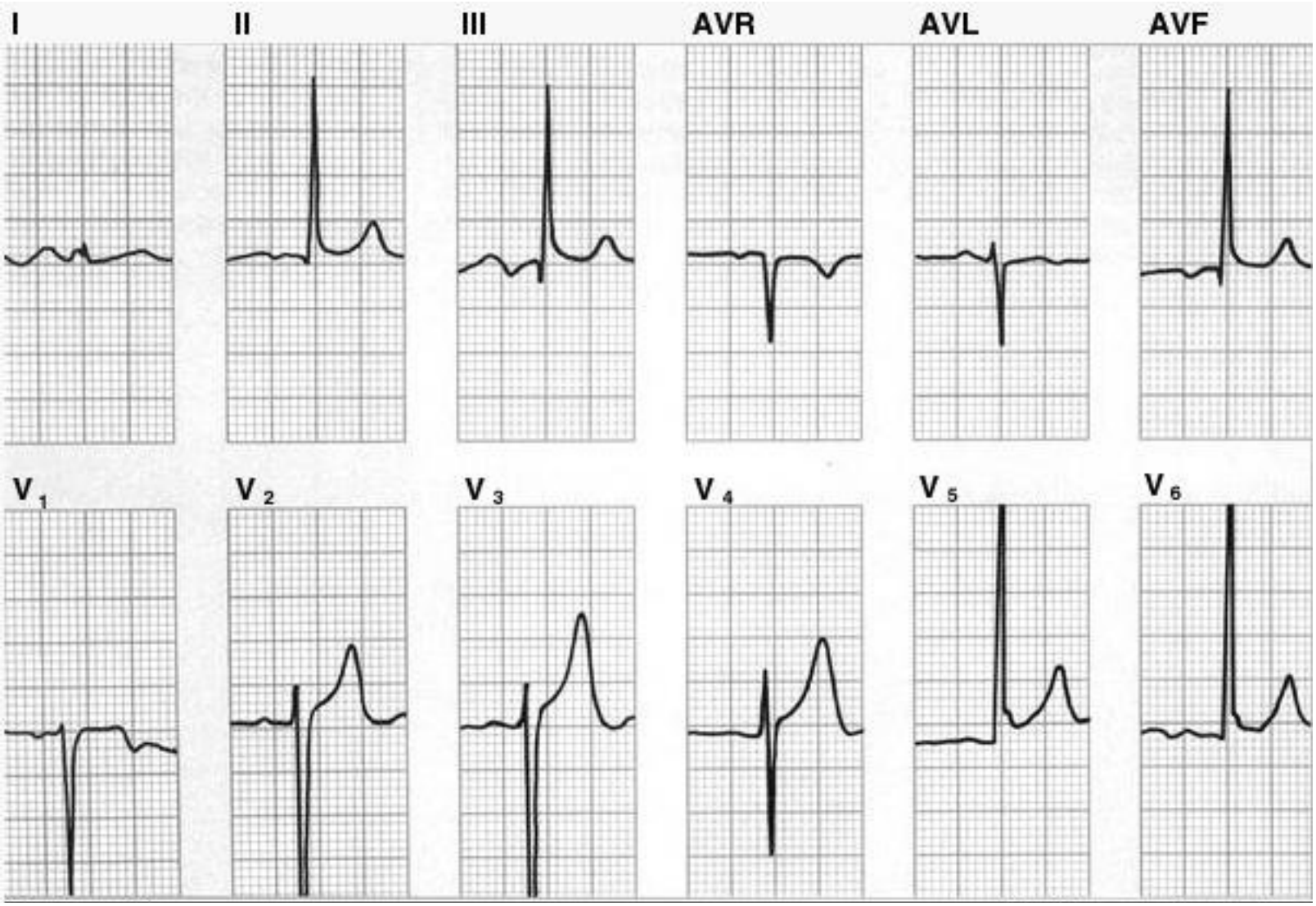
Etiologie ST elevatie?



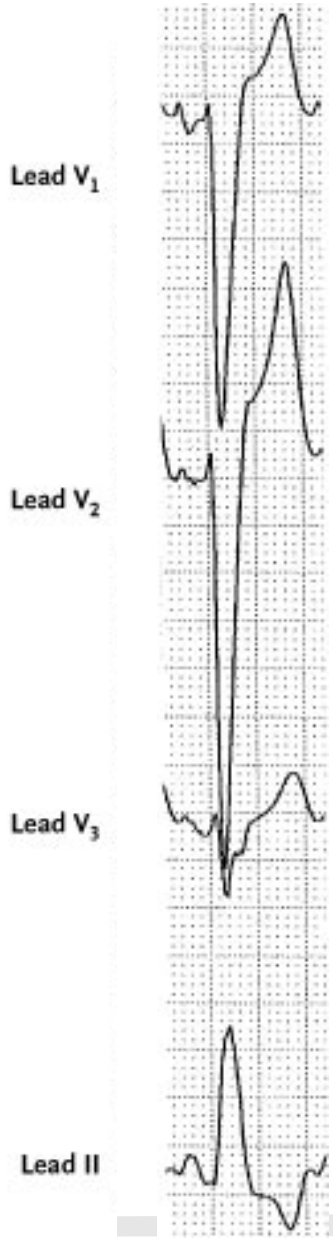
Etiologie ST elevatie



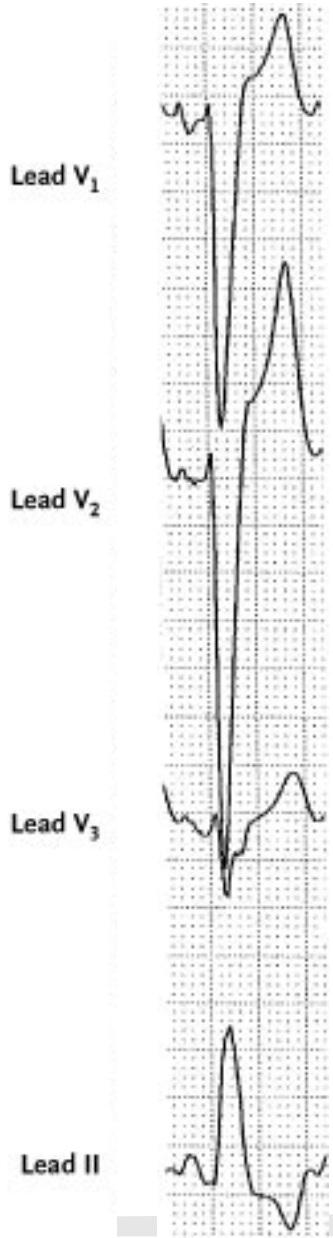
ECG van patient met
Linker Ventrikel Hypertrofie


I
II
III
AVR
AVL
AVF
V1
V2
V3
V4
V5
V6

Etiologie ST elevatie?

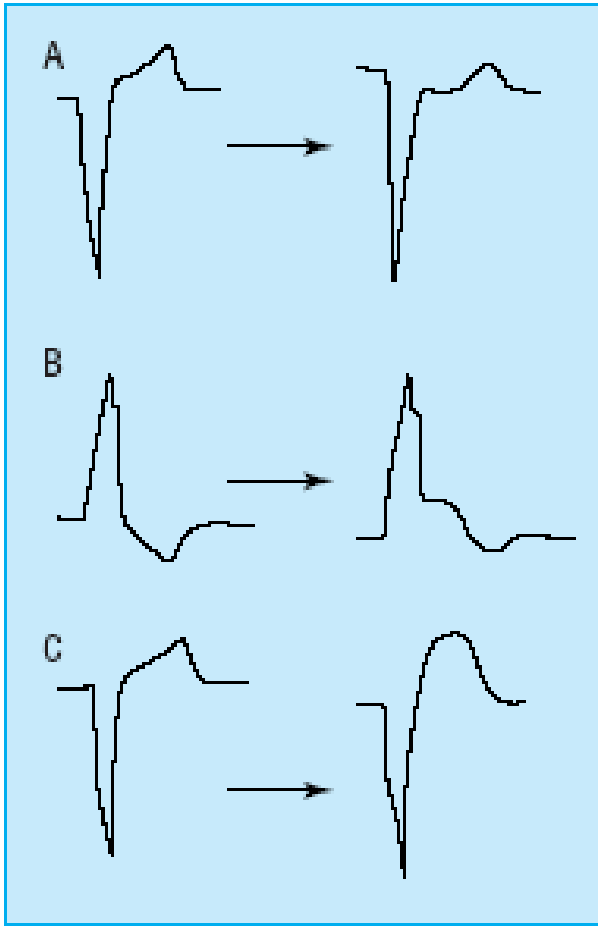


Etiologie ST elevatie



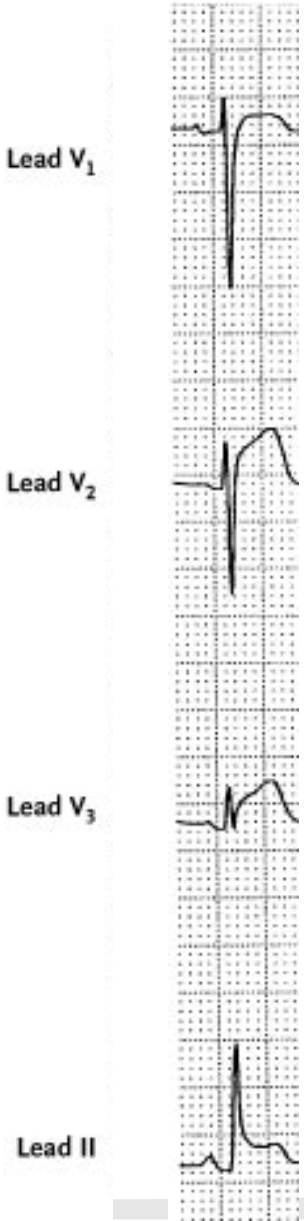
Passend bij LBBB

LBBB
Infarct diagnostiek

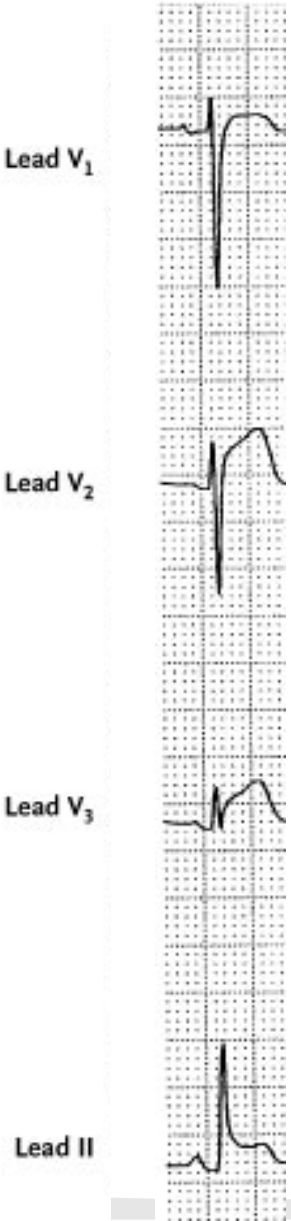


- Tekenen van infact bij LBBB w.b. ST segment
  - Concordantie i.p.v. discordantie
  - Toename van discordantie in V1 (>5 mm)

Etiologie ST elevatie?



Etiologie ST elevatie

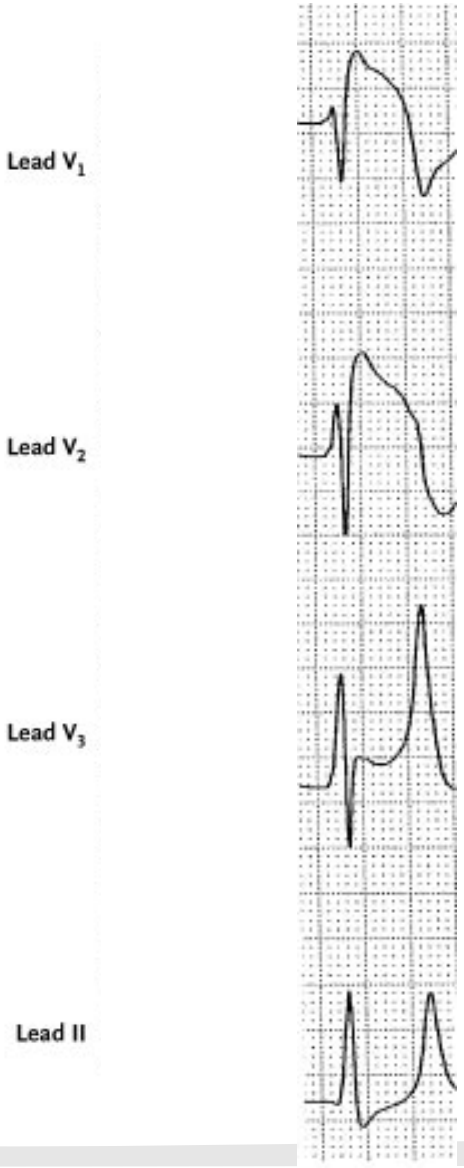


ST elevatie in precordiale afleidingen én in afleiding II.
Let ook op P_T depressie in II

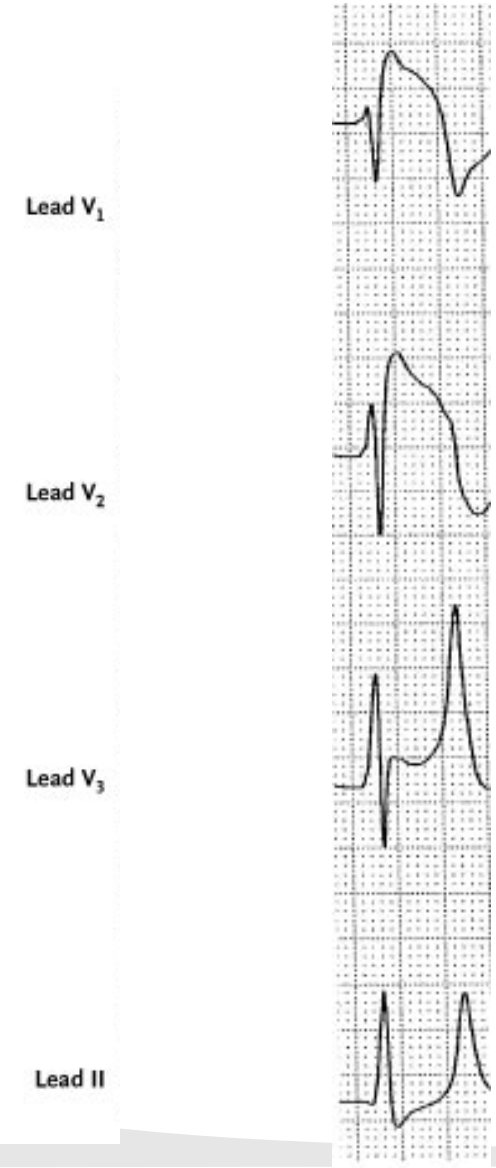
ST elevatie

- **Pericarditis**
  - **Anamnese**
  - **Fysische diagnostiek**
  - **“Alles positief of alles negatief”**
  - **In acute stadium in vrijwel alle afleidingen ST elevatie, behalve eventueel in V1 en AVR en III**
  - **“Smiley”ST – elevatie**
  - **Geen reciproke depressies**
  - **Pt depresie (onderwands afleidingen)**

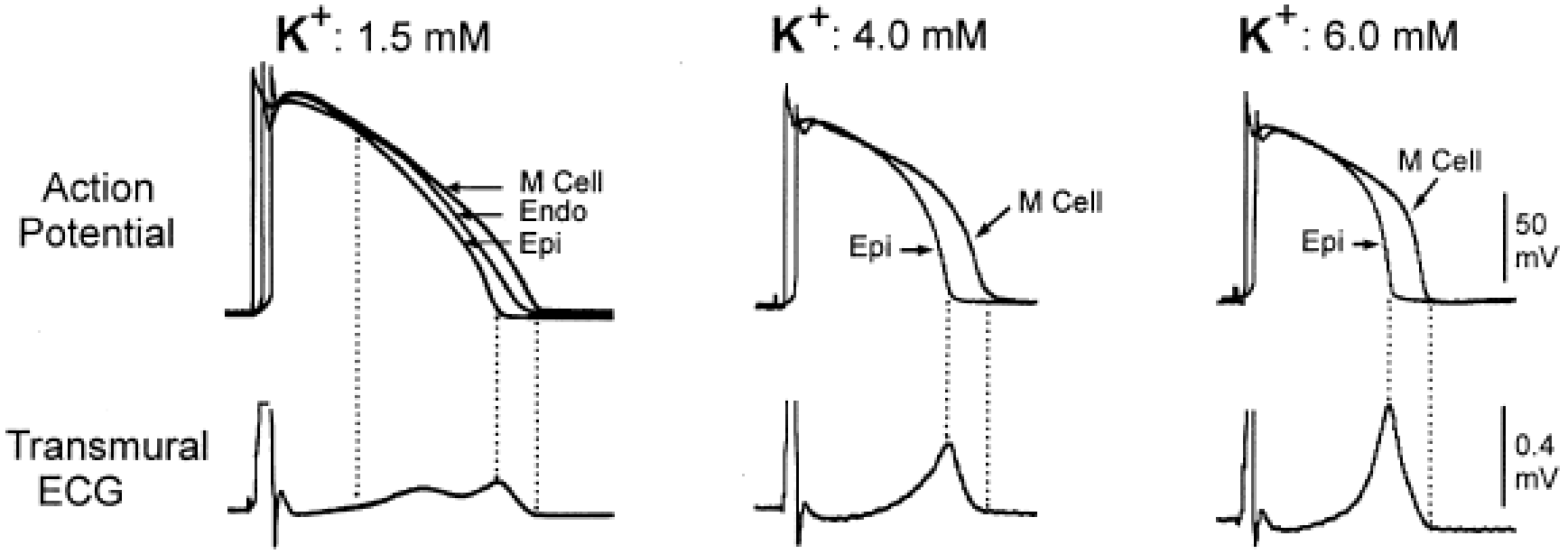
Etiologie ST elevatie?



Etiologie ST elevatie

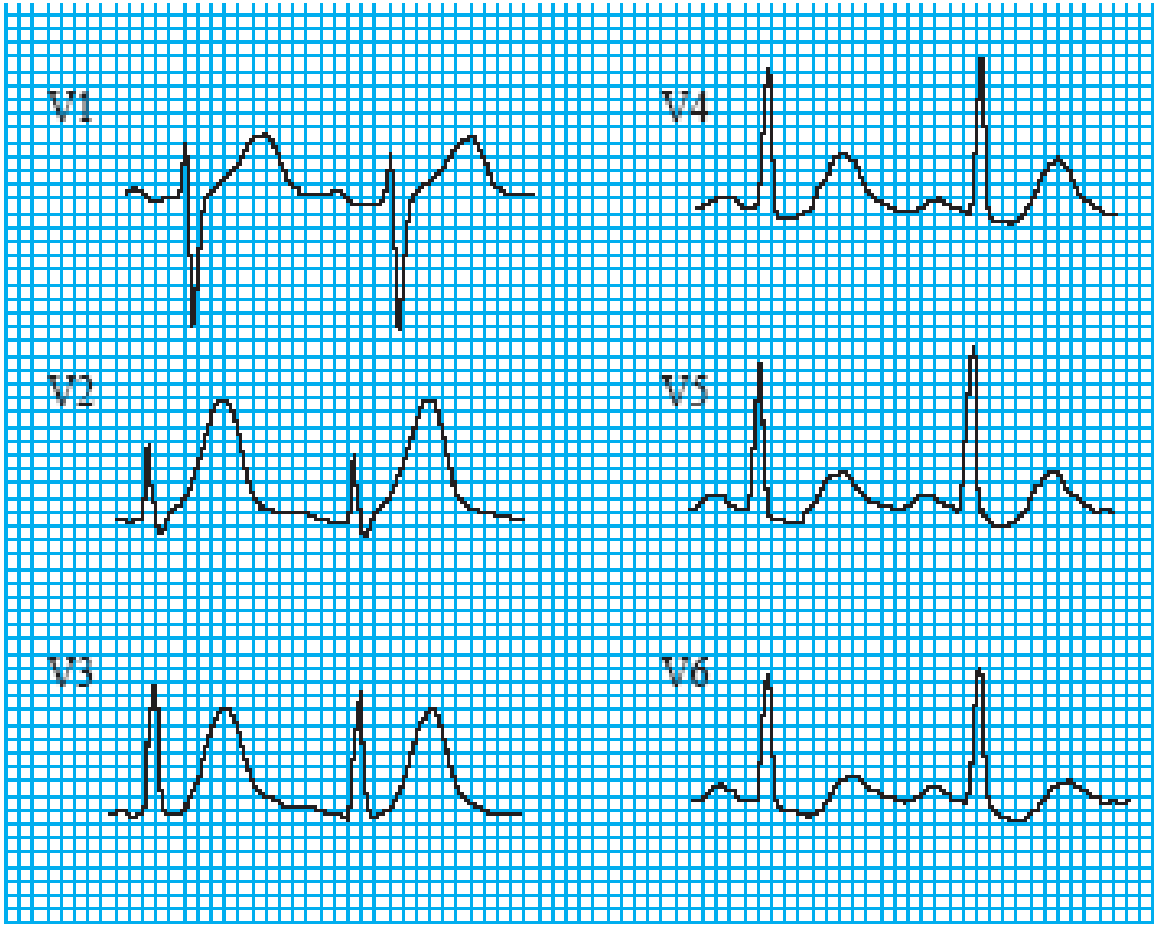


- T top hoog en tentvormig
- QRS verbreed
- P top verdwijnt
- ST elevatie soms zeer uitgesproken heeft echter i.t.t. bij een infarct een downsloping aspect



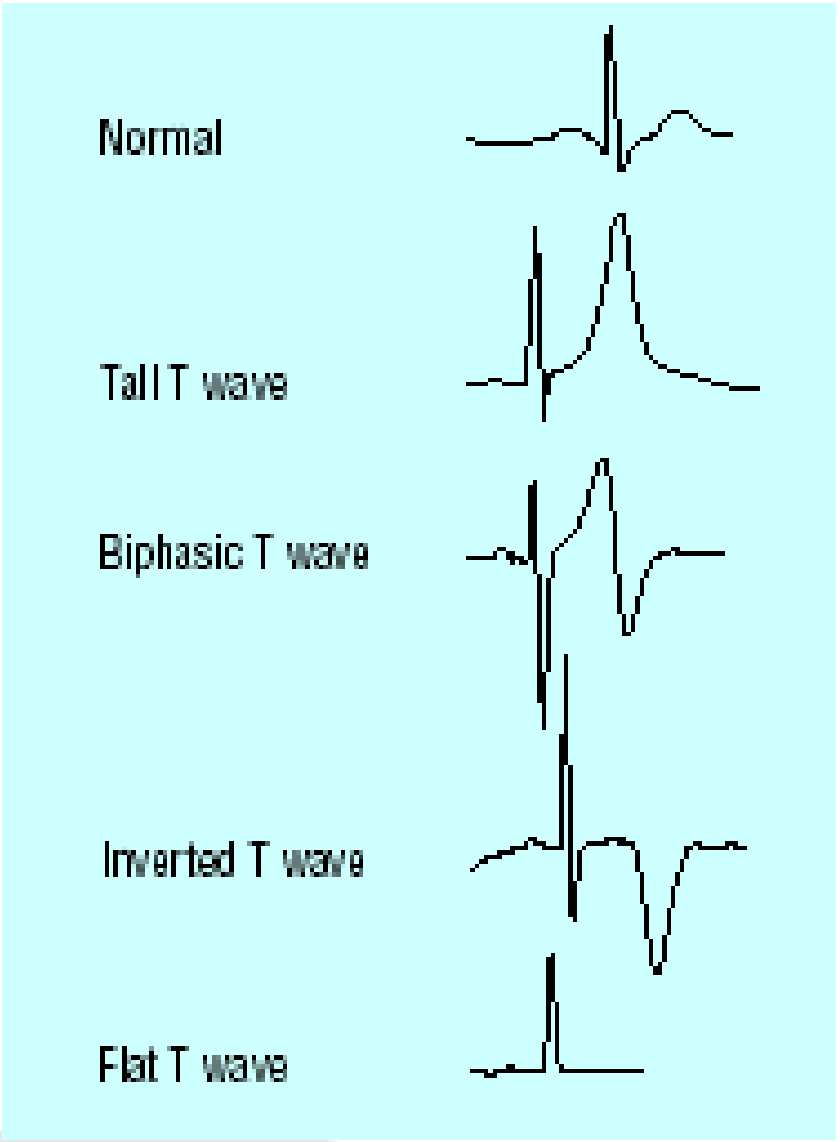
Hyperkaliëmie versnelt de fase 3 repolarisatie

?


V1
V4
V2
V5
V3
V6

Hyperacute T-top

T - top



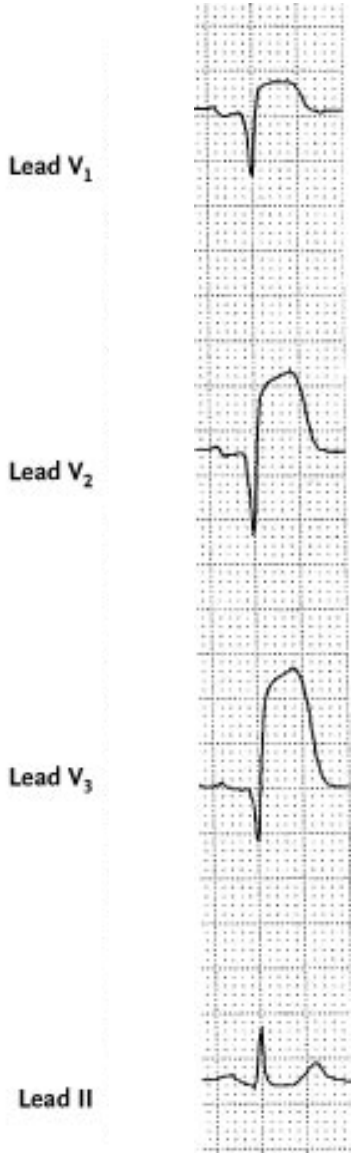
Normale T-top

- > 1/8 van de R-top
- < 2/3 van de R-top
- hoogte < 10 mm
- mag negatief zijn in
  III, AVR, V1
  (en V2 als de T top in V1
  ook neg. is)

Normale T-top

- **> 1/8 van de R-top**
- **< 2/3 van de R-top**
- **hoogte < 10 mm**
- **mag negatief zijn in III, AVR, V1 (en V2 als de T top in V1 ook neg. is)**

Etiologie ST elevatie?

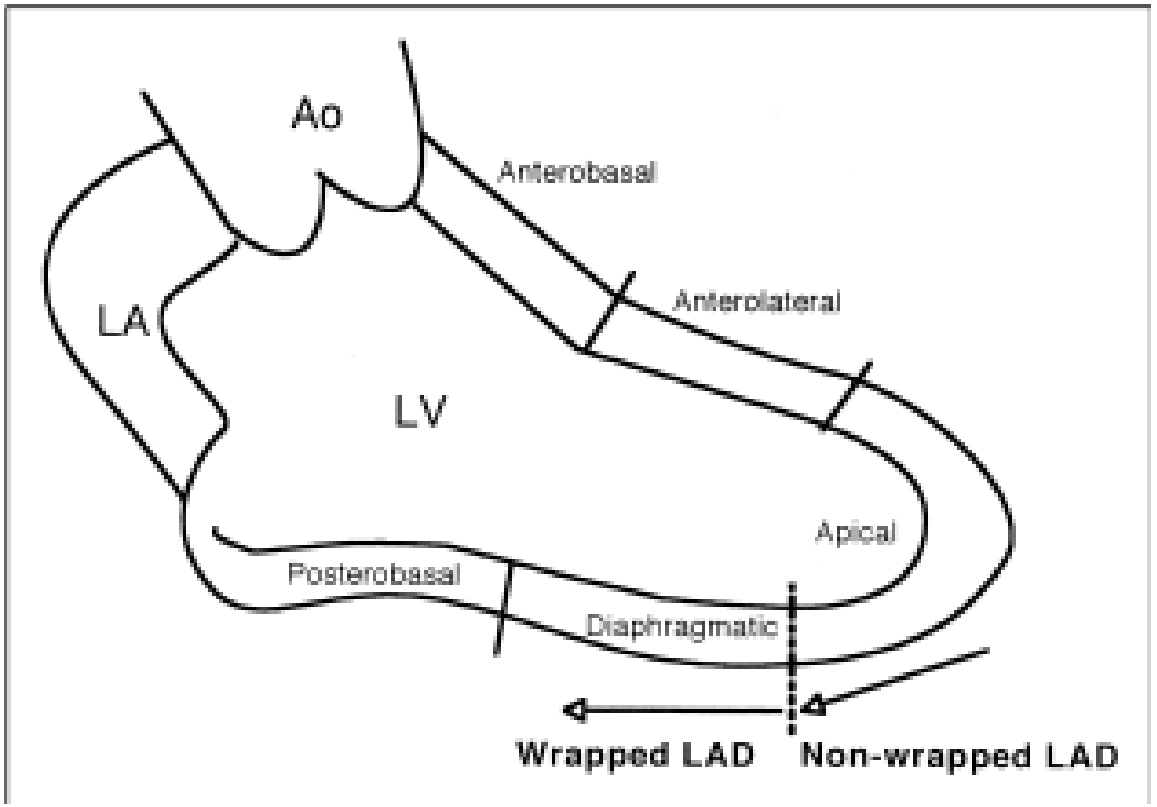


Relation of ST-Segment Changes in Inferior Leads During Anterior Wall Acute Myocardial Infarction to Length and Occlusion Site of the Left AnteriorDescending Coronary Artery

- 159 patients with anterior wall acute myocardial infarction (AMI).
- Patients with 1-vessel LAD artery lesions were divided based on ST-segment changes in the inferior leads, into:
  - ST depression group (n = 40),
  - ST elevation group (n = 25)
  - no-ST-change group (n = 94)
- They investigated the relation between each group and the
  - infarct-related lesion and
  - the presence of a wrapped LAD artery

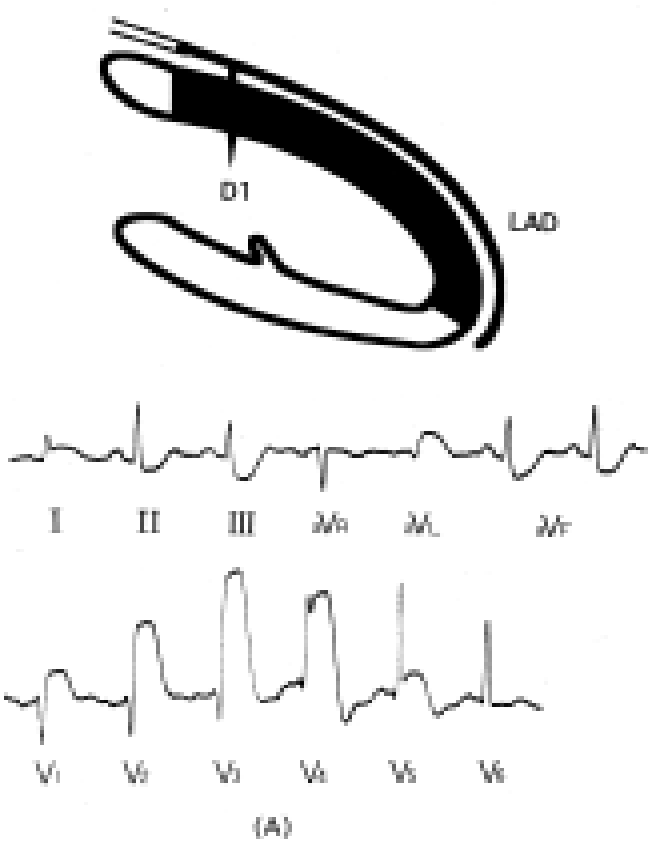
Sasaki et al., Am J Cardiol 2001;87:1340–1345)

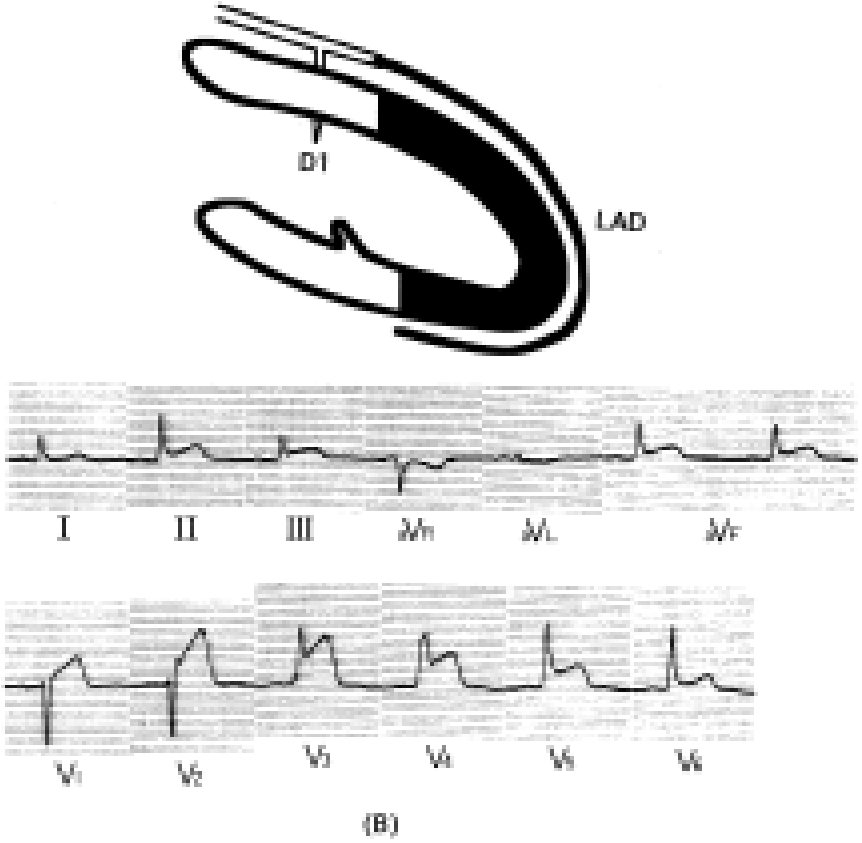
Reciproke ST depressie



AJC 2001;87: 1340-5

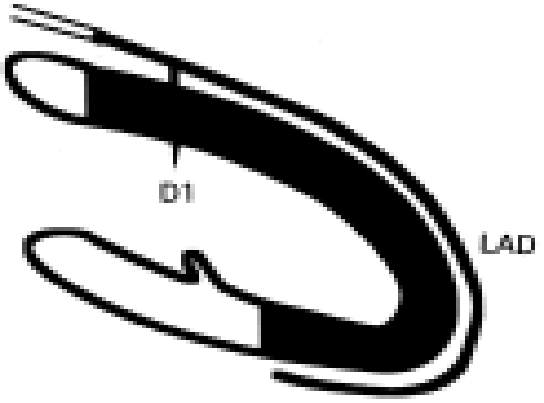
Reciproke ST depressie

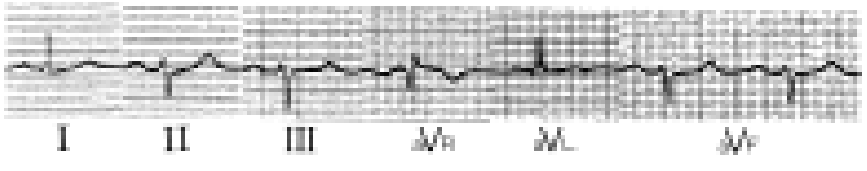


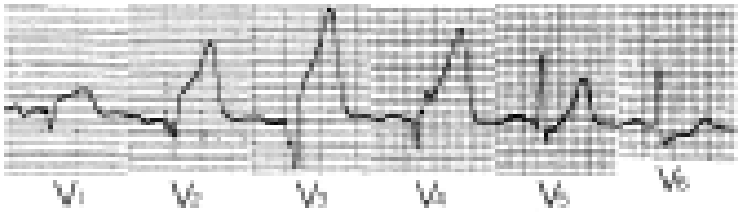


AJC 2001;87: 1340-5

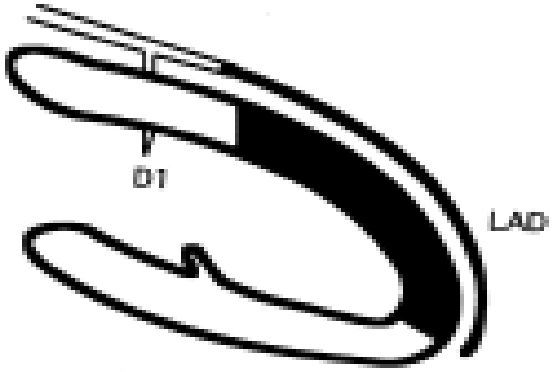
Reciproke ST depressie

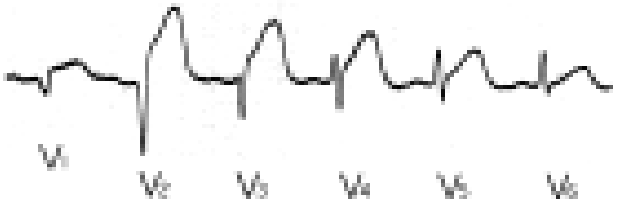






(A)

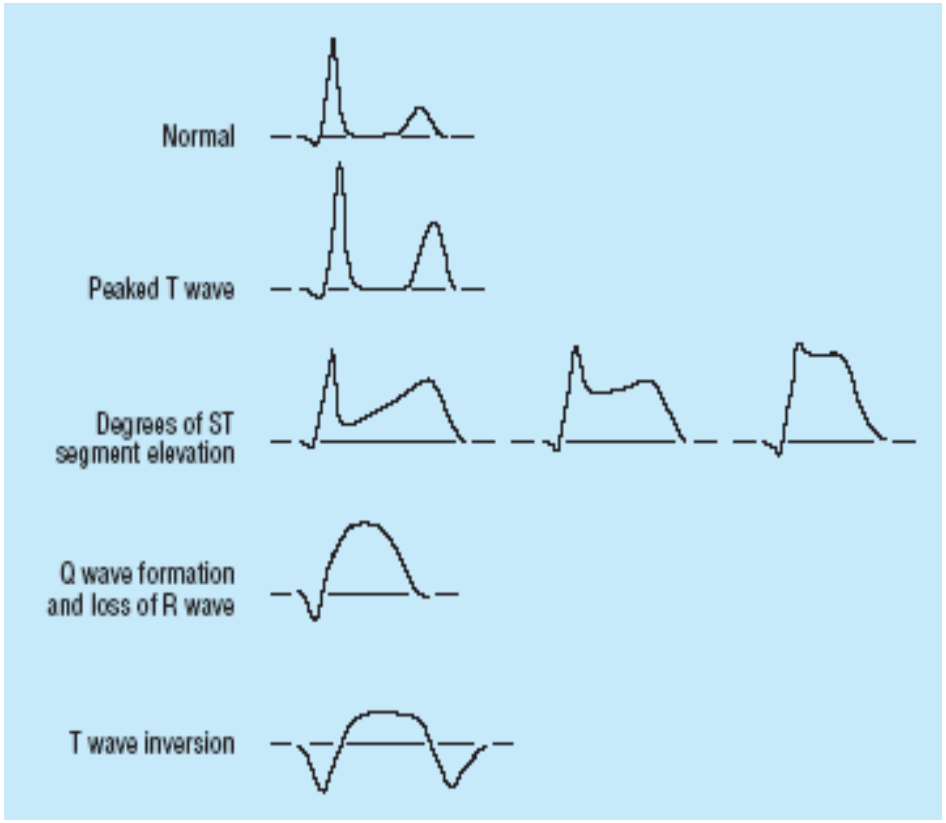




(B)

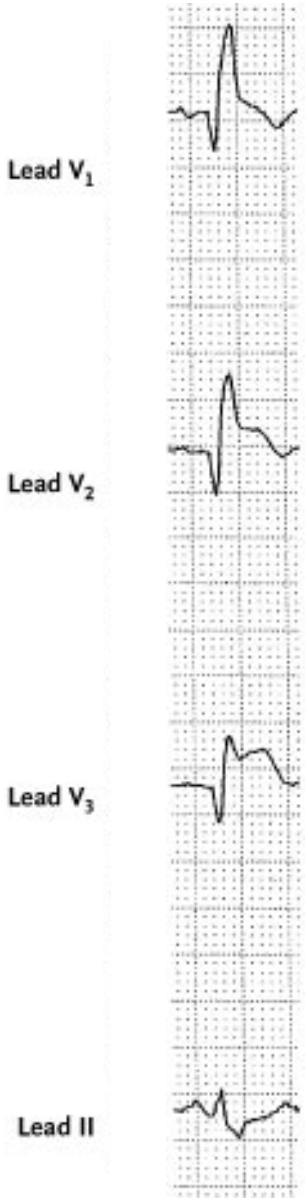
AJC 2001;87: 1340-5

Volgorde ECG veranderingen na begin klachten

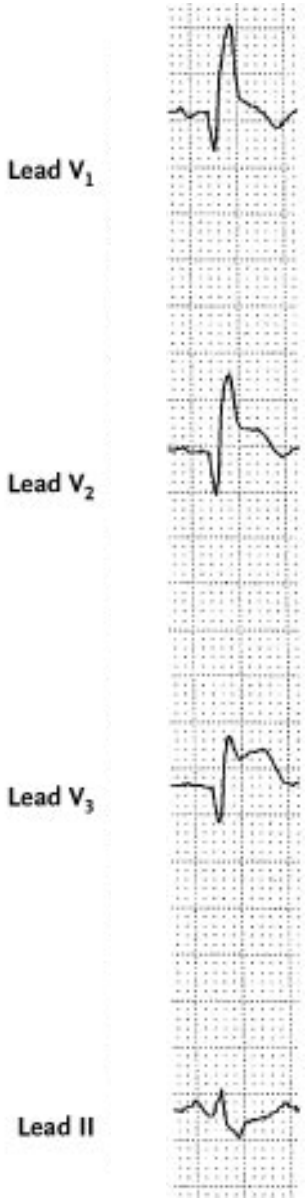


- Hyperacute T-top na 5 -30 min
- ST-T hoek verstrijkt en “t-top wordt breed”
- ST-elevatie ontstaat en verandert van vorm (concaaf → convex)
- Q soms < 1 a 2 uur, meestal > 12 uur en soms na 24 uur (kan na verloop van tijd verdwijnen)
- ST elevatie verdwijnt bij OWI < 2 weken, bij VWI soms nog iets later (persisteert bij aneurysma)
- T-top inversie kan maanden of permanent aanwezig blijven

Etiologie ST elevatie?

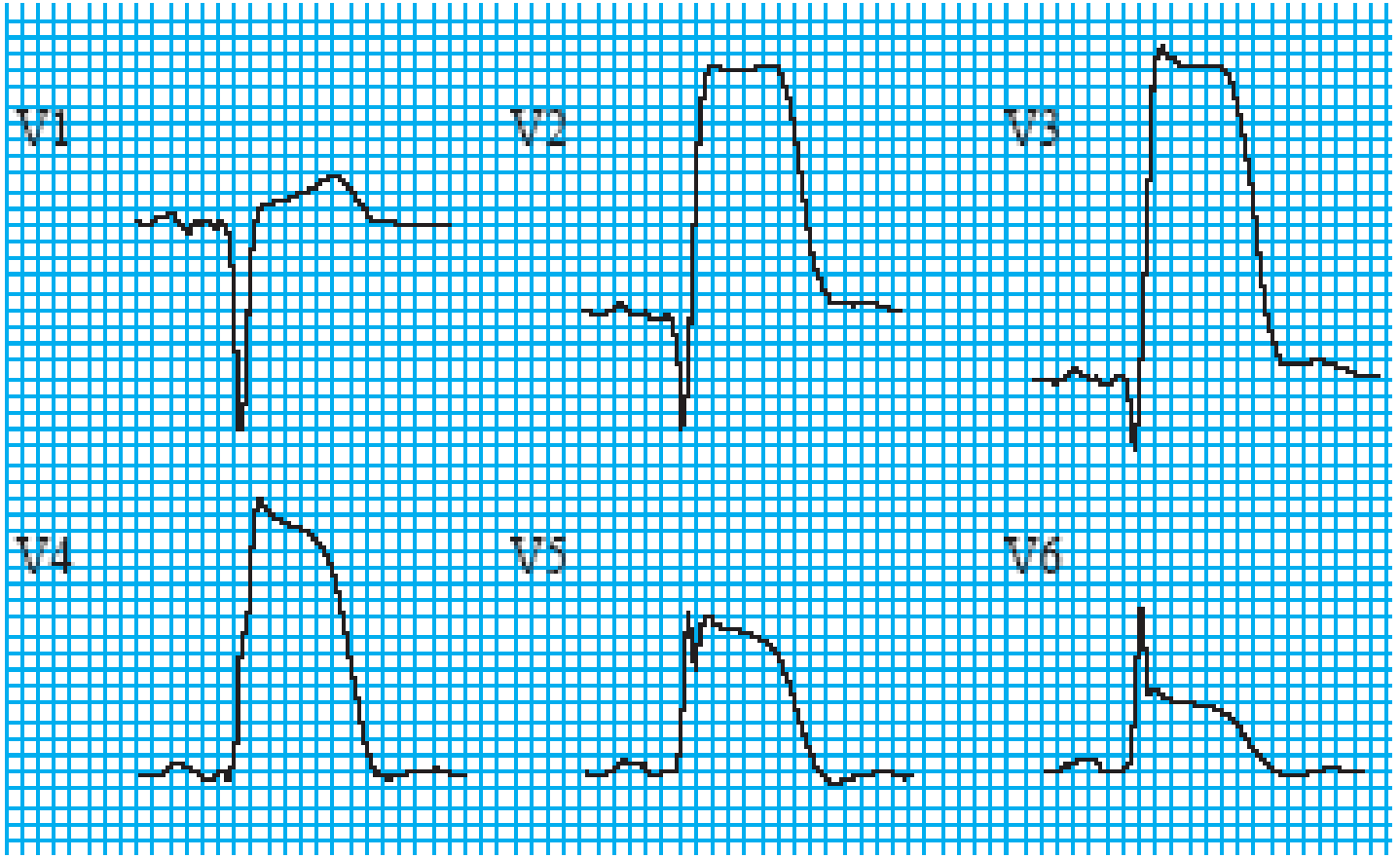


Etiologie ST elevatie

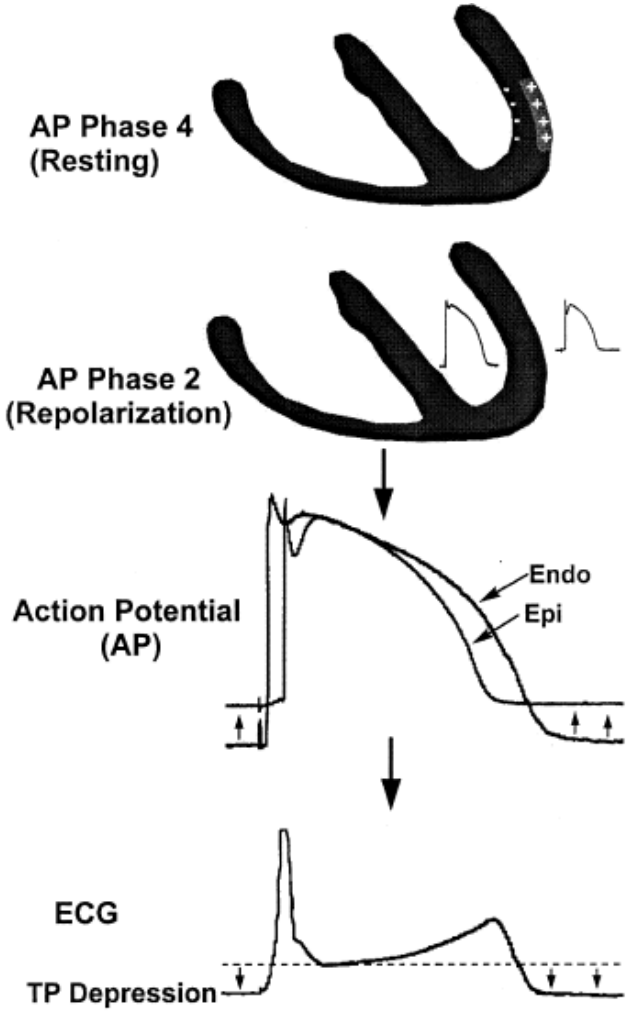


Anteroseptaal infarct en RBBB

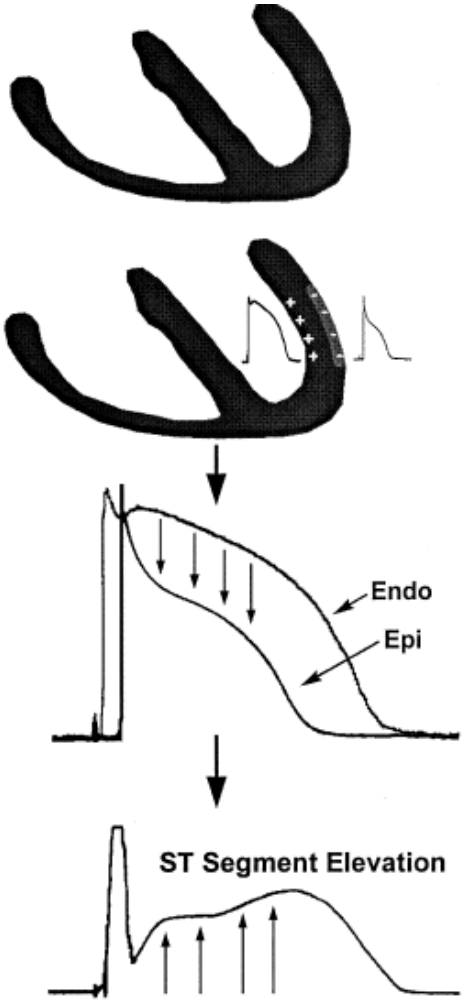
Fusie QRS, ST en T-top tot “giant R wave” of “tombstone”



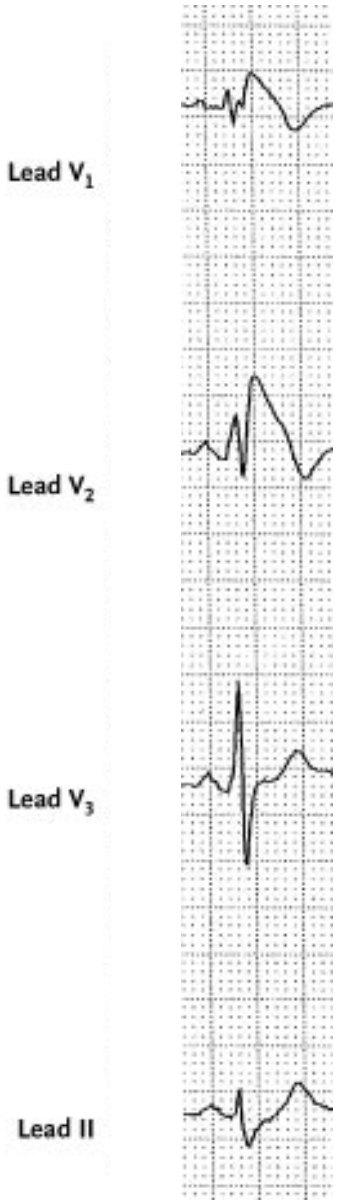
“Current of injury” theorie



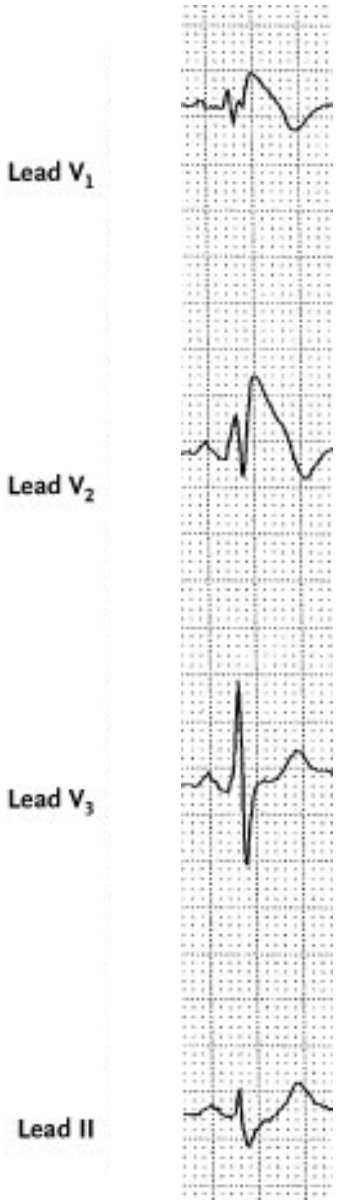
“Loss of AP dome or plateau amplitude” theorie



Etiologie ST elevatie?

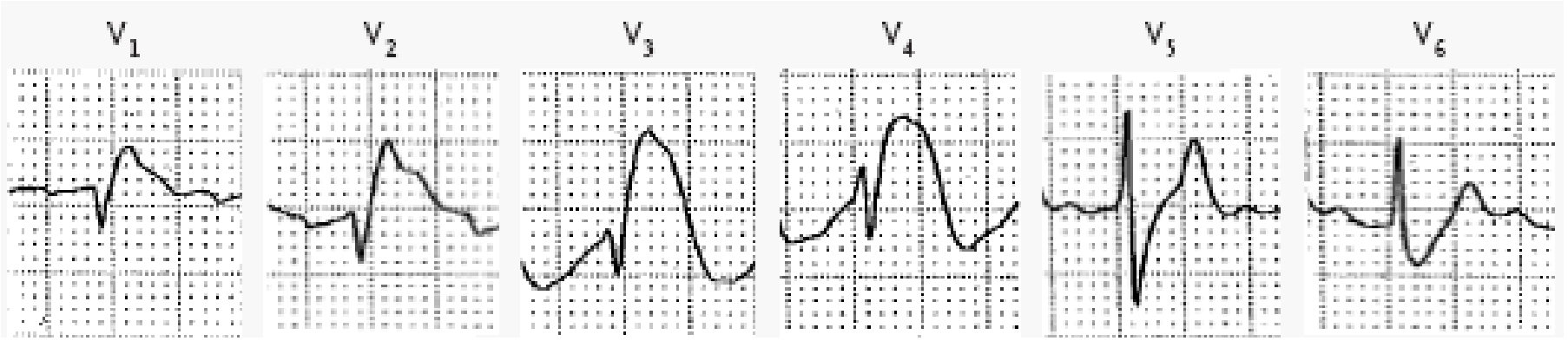


Etiologie ST elevatie

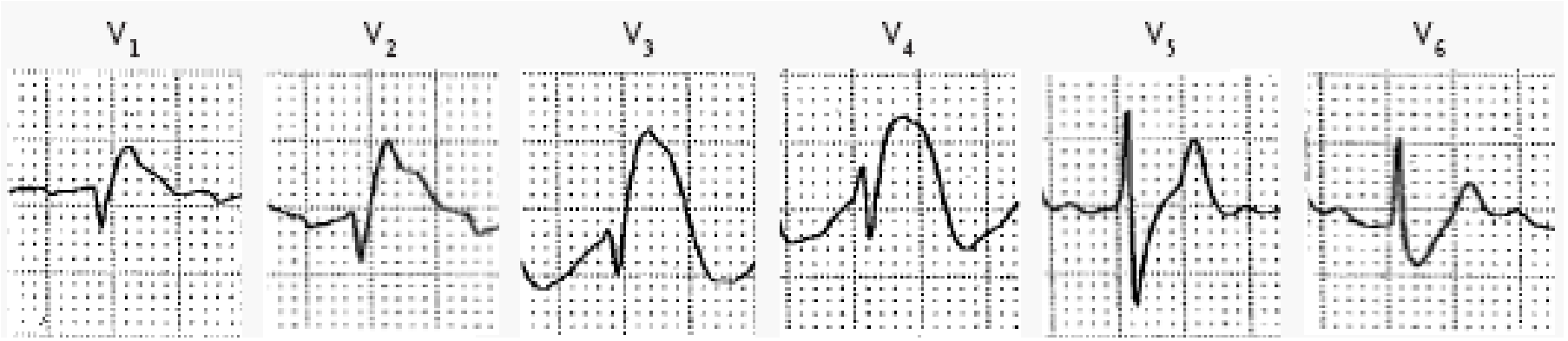


Brugada syndroom

Etiologie ST elevatie?

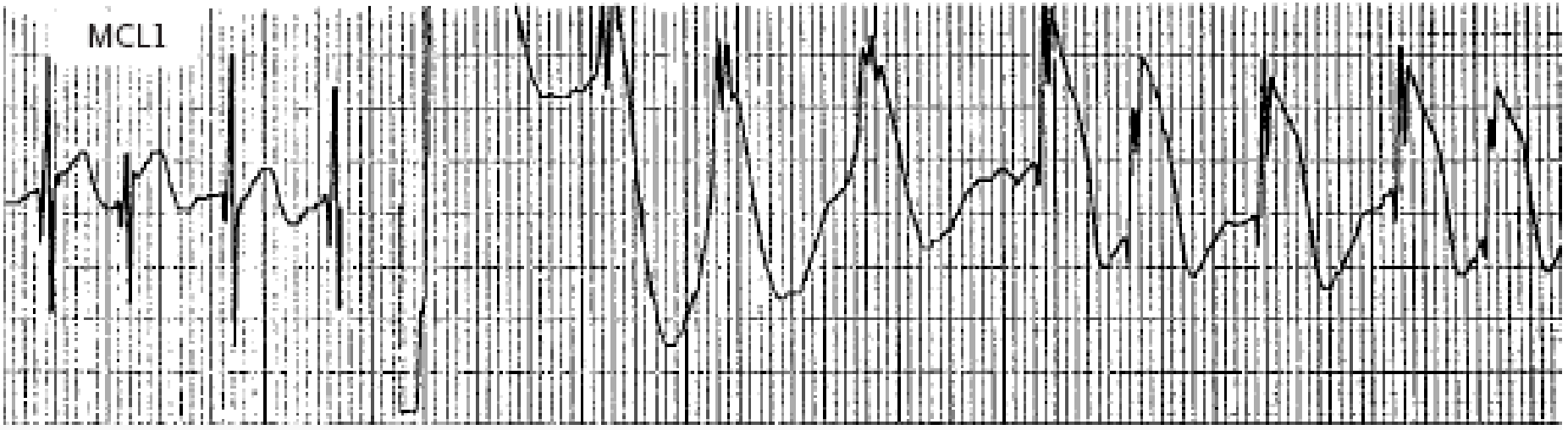


Etiologie ST elevatie

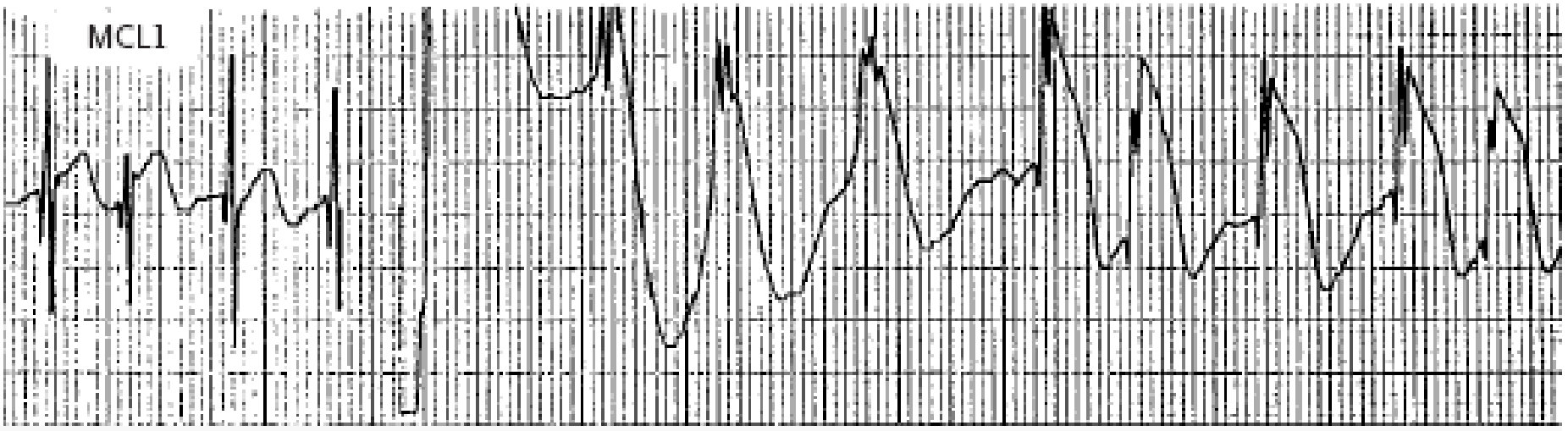


Massale longembolus

Etiologie ST elevatie?

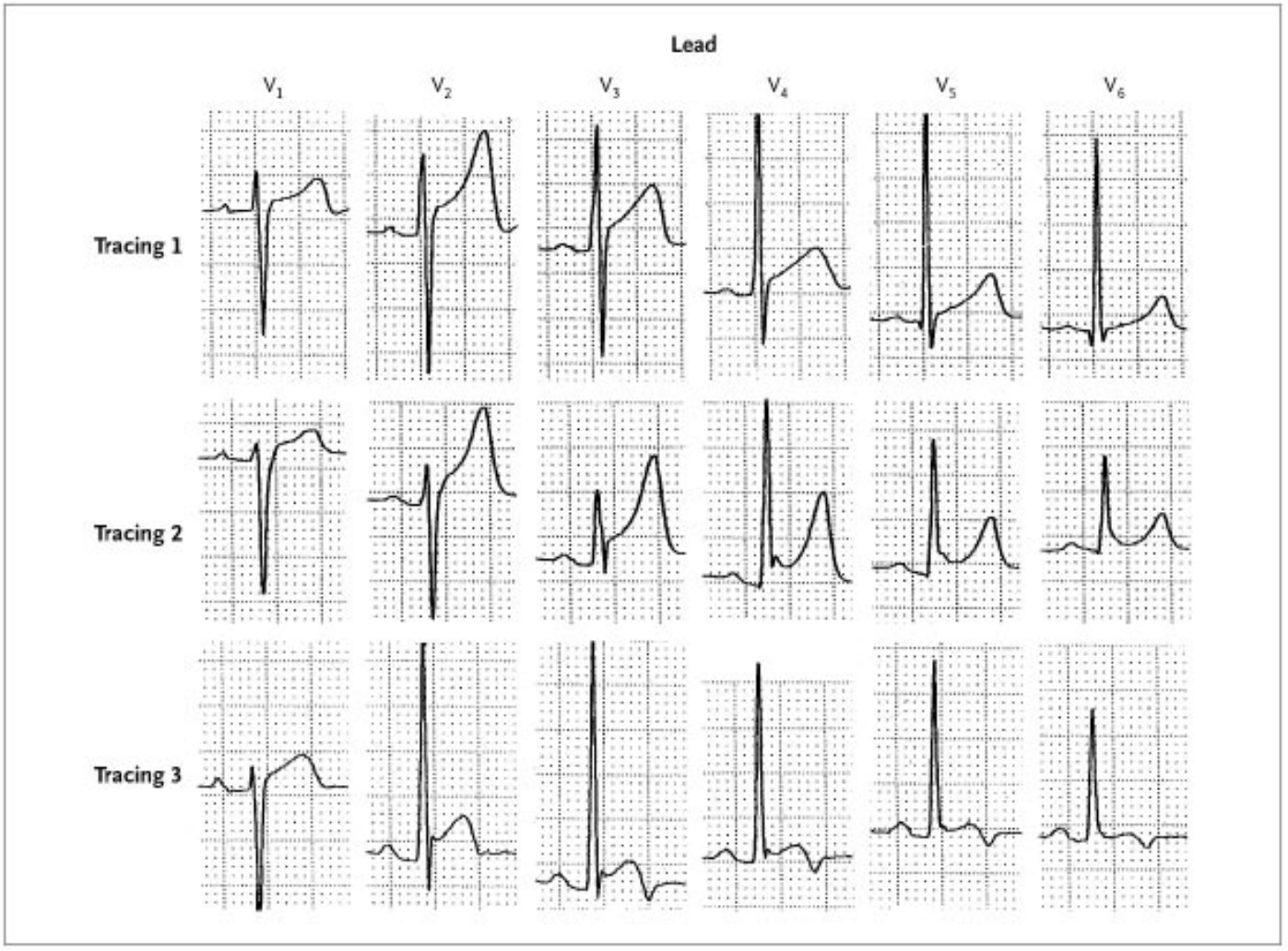


Etiologie ST elevatie



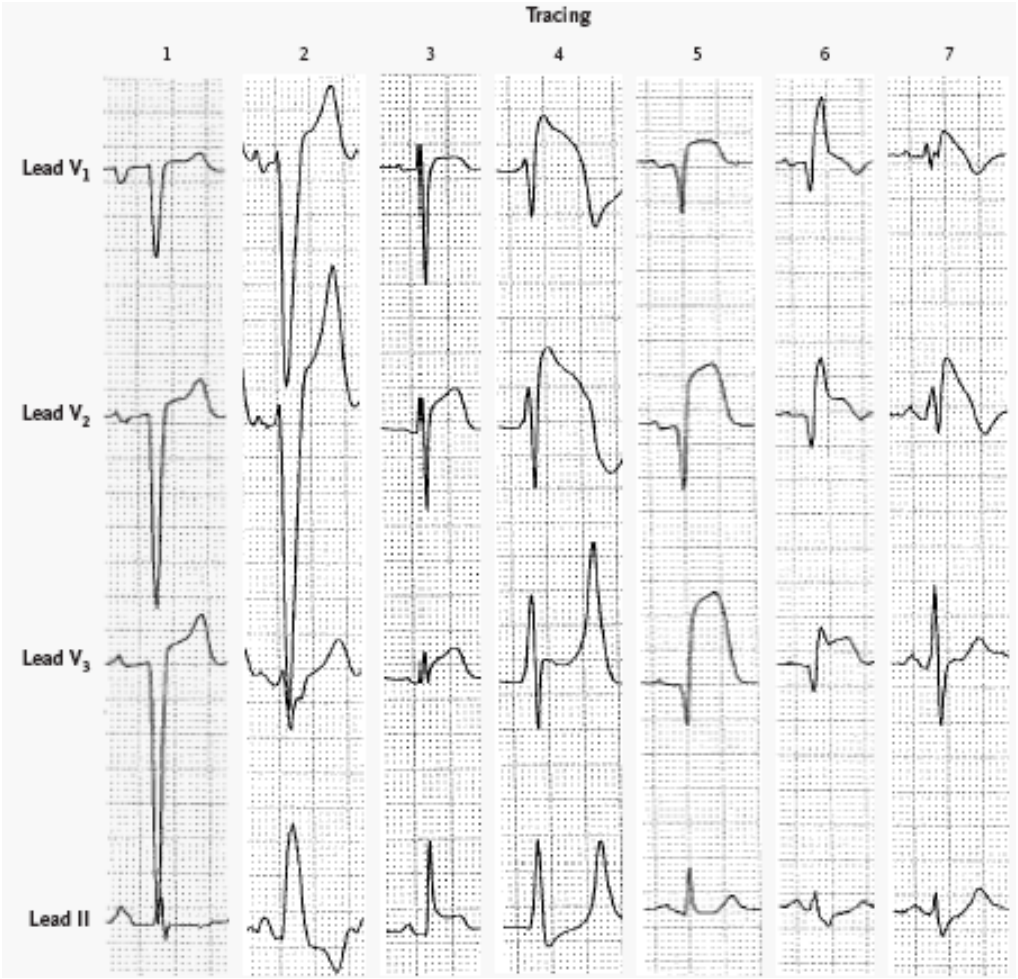
Passagère ST elevatie direct na elektrische cardioversie

(Varianten van) normaal



ST elevaties bij verschillende aandoeningen

Altijd interpreteren met anamnese!!!



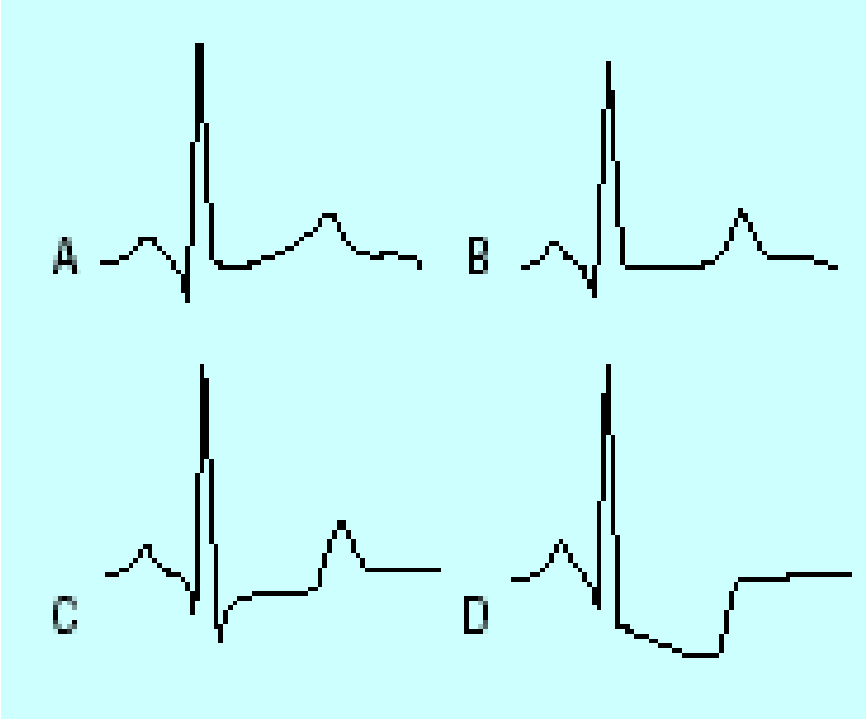
ST segment elevatie

Condition	Features
Normal (so-called male pattern)	Seen in approximately 90 percent of healthy young men; therefore, normal Elevation of 1–3 mm Most marked in V_2 Concave
Early repolarization	Most marked in V_4, with notching at J point Tall, upright T waves Reciprocal ST depression in aVR, not in aVL, when limb leads are involved
ST elevation of normal variant	Seen in V_3 through V_5 with inverted T waves Short QT, high QRS voltage
Left ventricular hypertrophy	Concave Other features of left ventricular hypertrophy
Left bundle-branch block	Concave ST-segment deviation discordant from the QRS
Acute pericarditis	Diffuse ST-segment elevation Reciprocal ST-segment depression in aVR, not in aVL Elevation seldom >5 mm PR-segment depression

Condition	Features
Hyperkalemia	Other features of hyperkalemia present: Widened QRS and tall, peaked, tented T waves Low-amplitude or absent P waves ST segment usually downsloping
Brugada syndrome	rSR' in V_1 and V_2 ST-segment elevation in V_1 and V_2, typically downsloping
Pulmonary embolism	Changes simulating myocardial infarction seen often in both inferior and antero-septal leads
Cardioversion	Striking ST-segment elevation, often >10 mm, but lasting only a minute or two immediately after direct-current shock
Prinzmetal's angina	Same as ST-segment elevation in infarction, but transient
Acute myocardial infarction	ST segment with a plateau or shoulder or upsloping Reciprocal behavior between aVL and III

N Engl J Med 2003;349:2128-35.

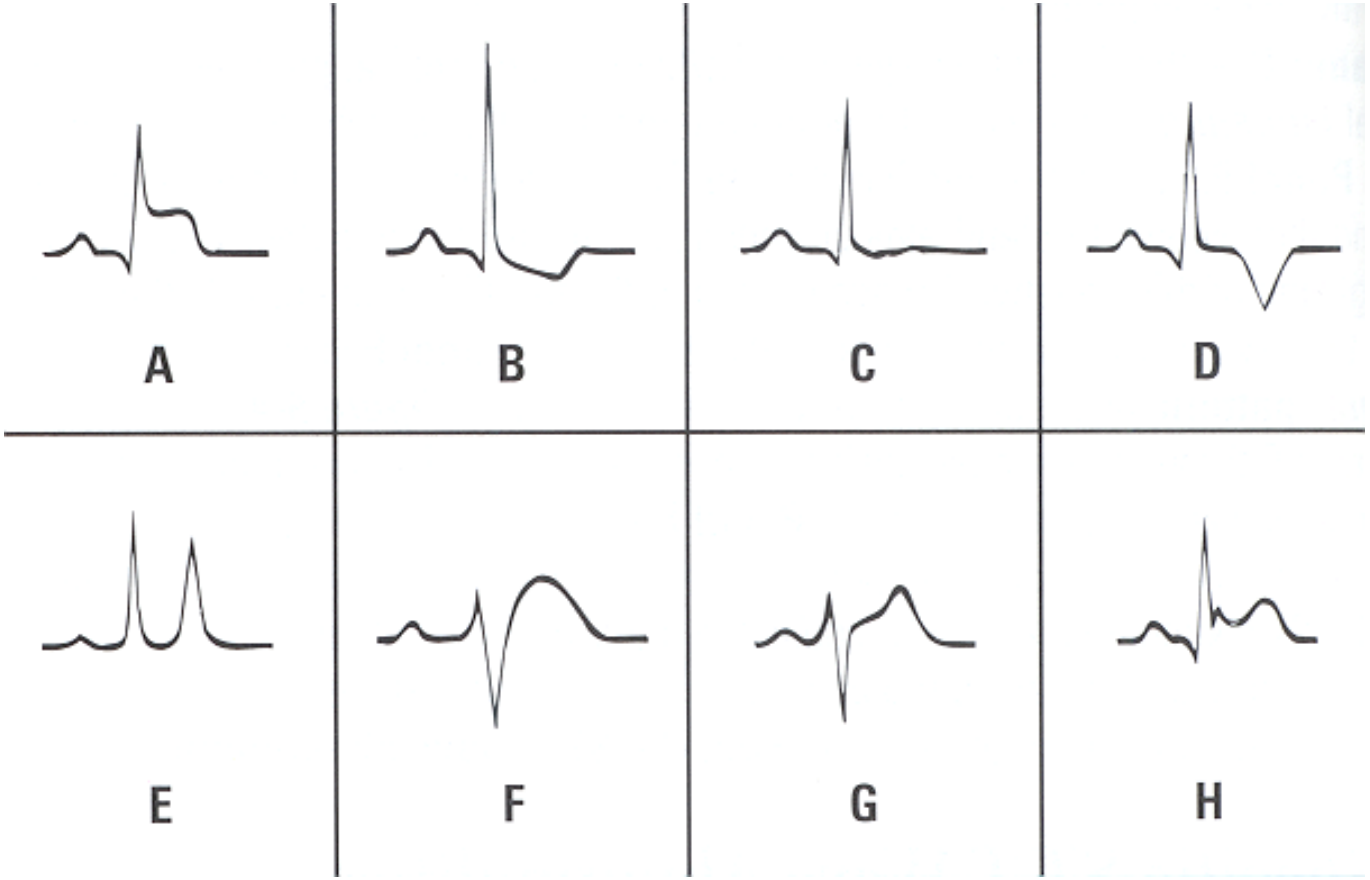
ST - segment



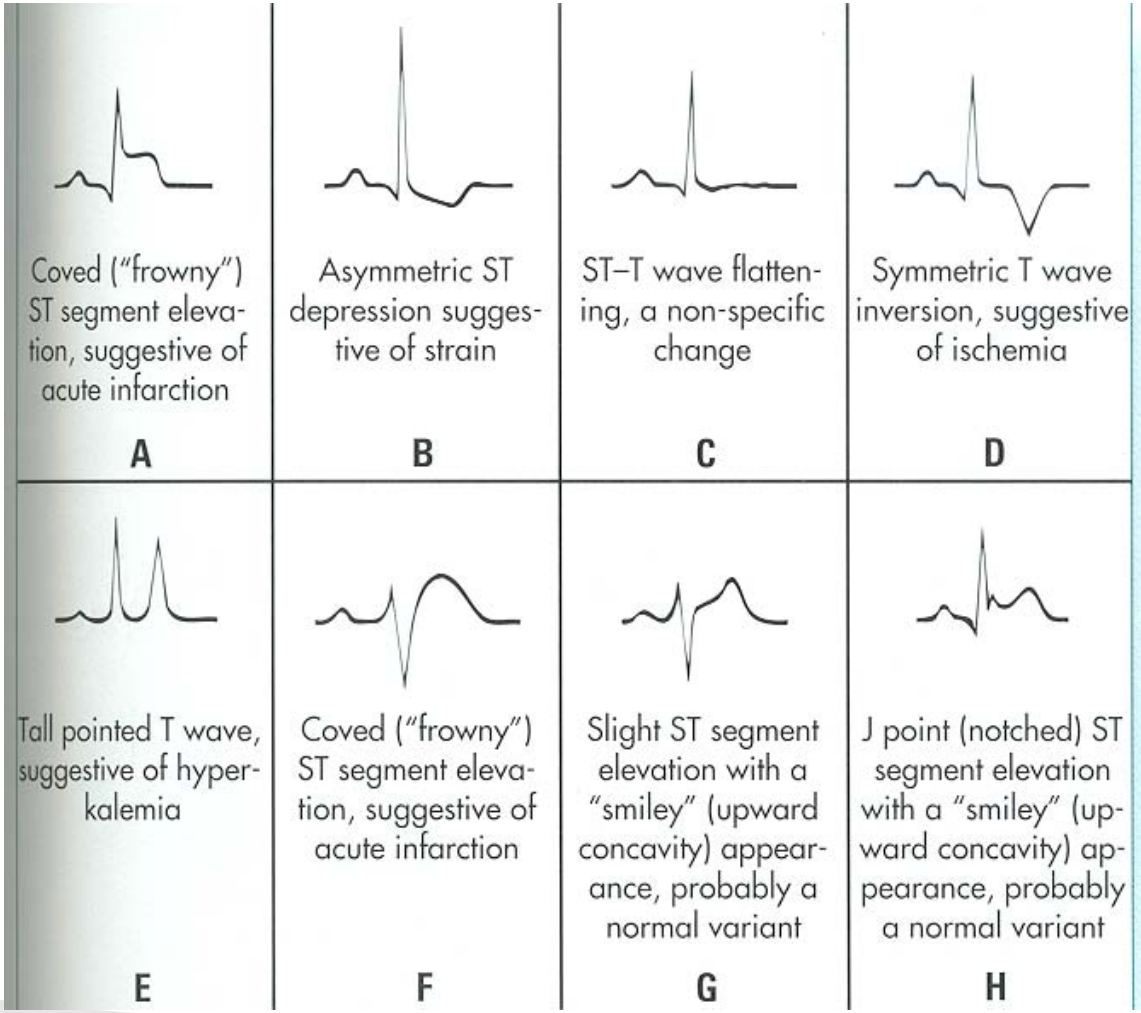
Normale ST- segment

- ≤ 1 mm ST shift onder of boven iso-elektrische lijn
- Geldt niet zo sterk voor V1 en V2, waar het ST segment versmelt met QRS complex en T-top

ST -T segment

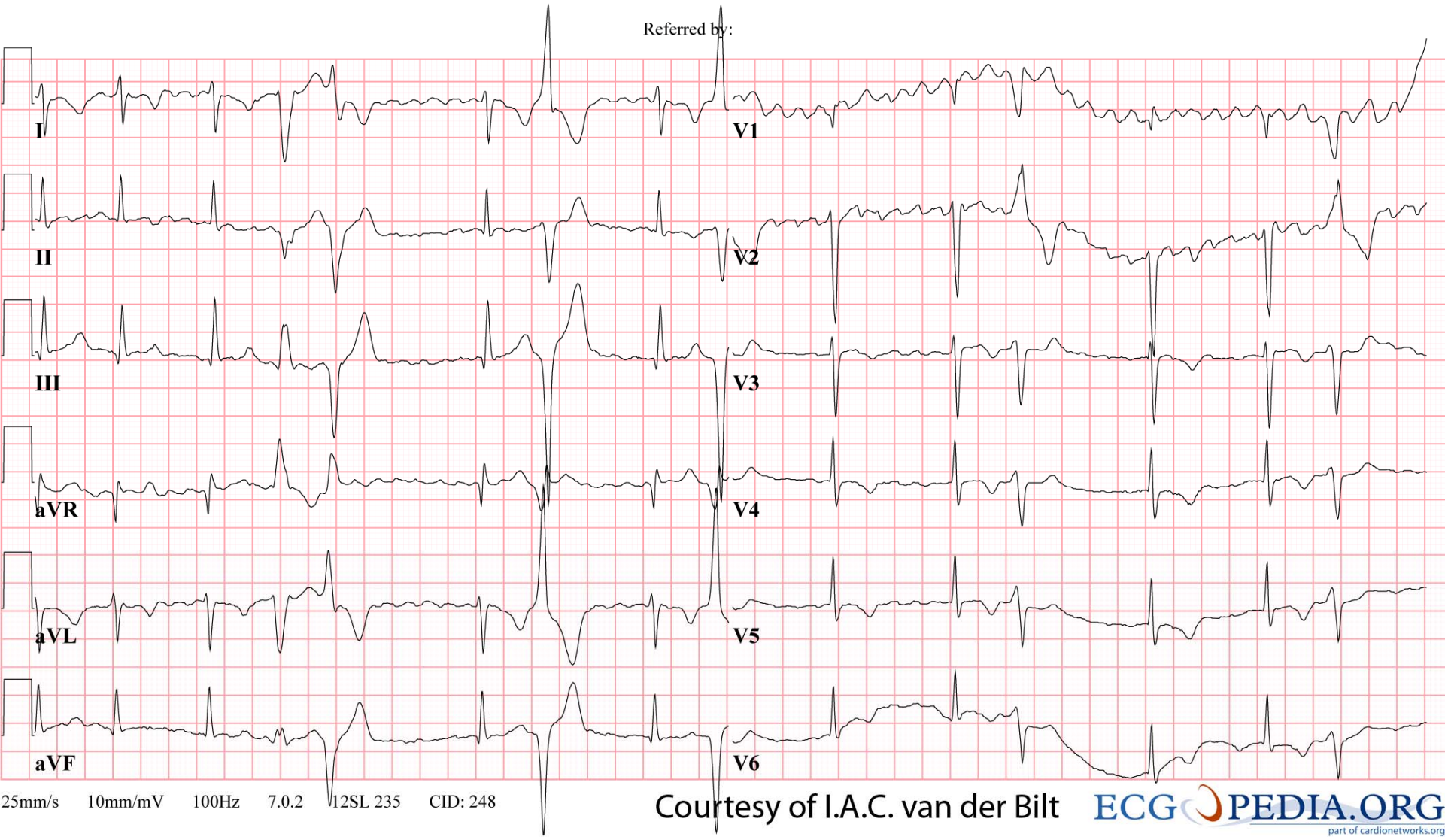


ST -T segment


Coved ("frowny") ST segment elevation, suggestive of acute infarction
A
Asymmetric ST depression suggestive of strain
B
ST–T wave flattening, a non-specific change
C
Symmetric T wave inversion, suggestive of ischemia
D
Tall pointed T wave, suggestive of hyperkalemia
E
Coved ("frowny") ST segment elevation, suggestive of acute infarction
F
Slight ST segment elevation with a "smiley" (upward concavity) appearance, probably a normal variant
G
J point (notched) ST segment elevation with a "smiley" (upward concavity) appearance, probably a normal variant
H

Niet-specifieke ST-T veranderingen

- Ischemie
- LVH
- Cardiomyopathie
- MVP
- Medicatie (digitalis, anti-arrhytmica)
- Elektrolyt stoornissen (hypo-K; hypomagnesiemie)
- CZS pathologie (CVA, bloeding)
- Hyperventilatie .... Etc. Etc.



Courtesy of I.A.C. van der Bilt

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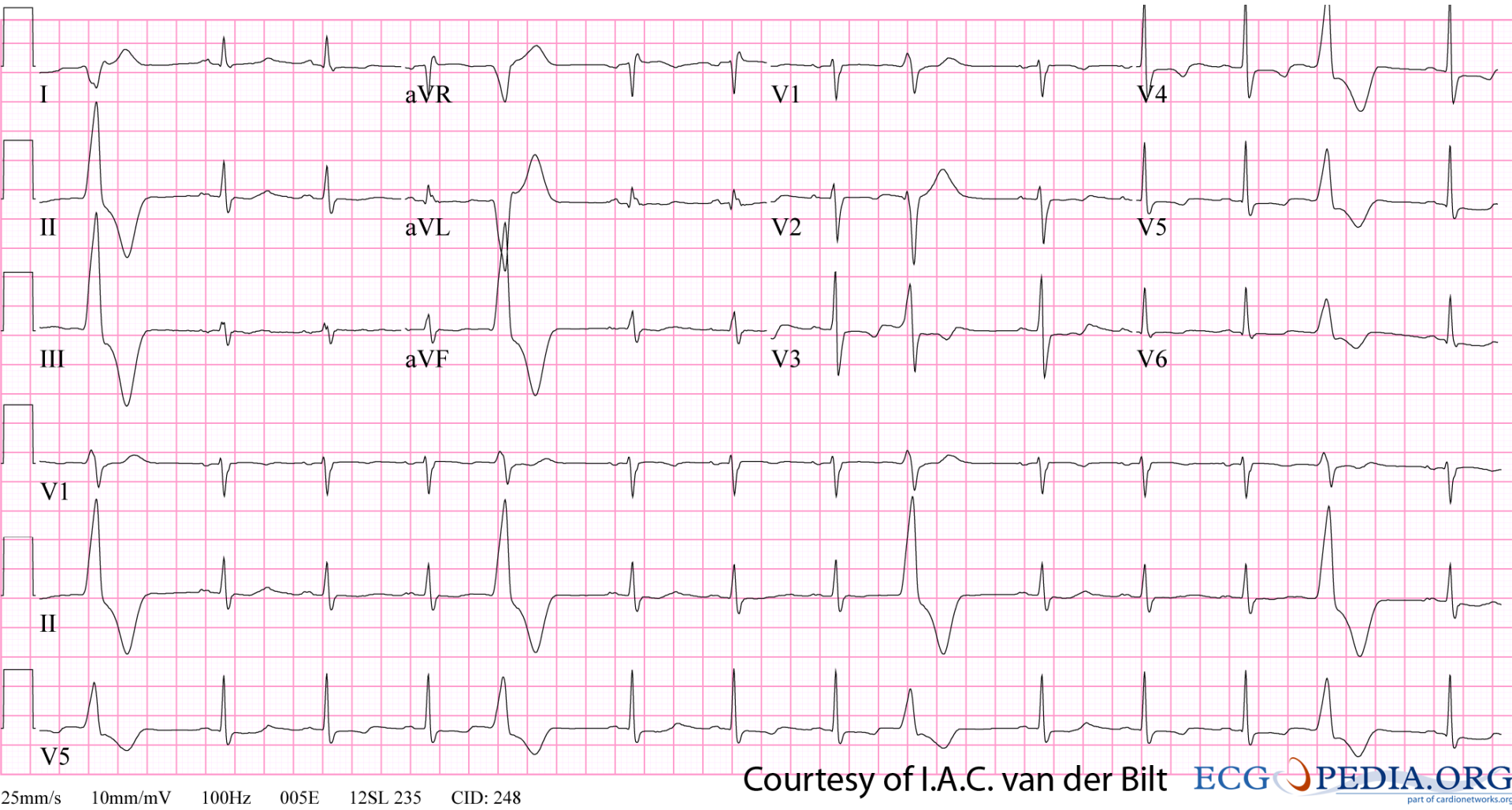
part of cardionetworks.org



Courtesy of I.A.C. van der Bilt

ECG PEDIA.ORG
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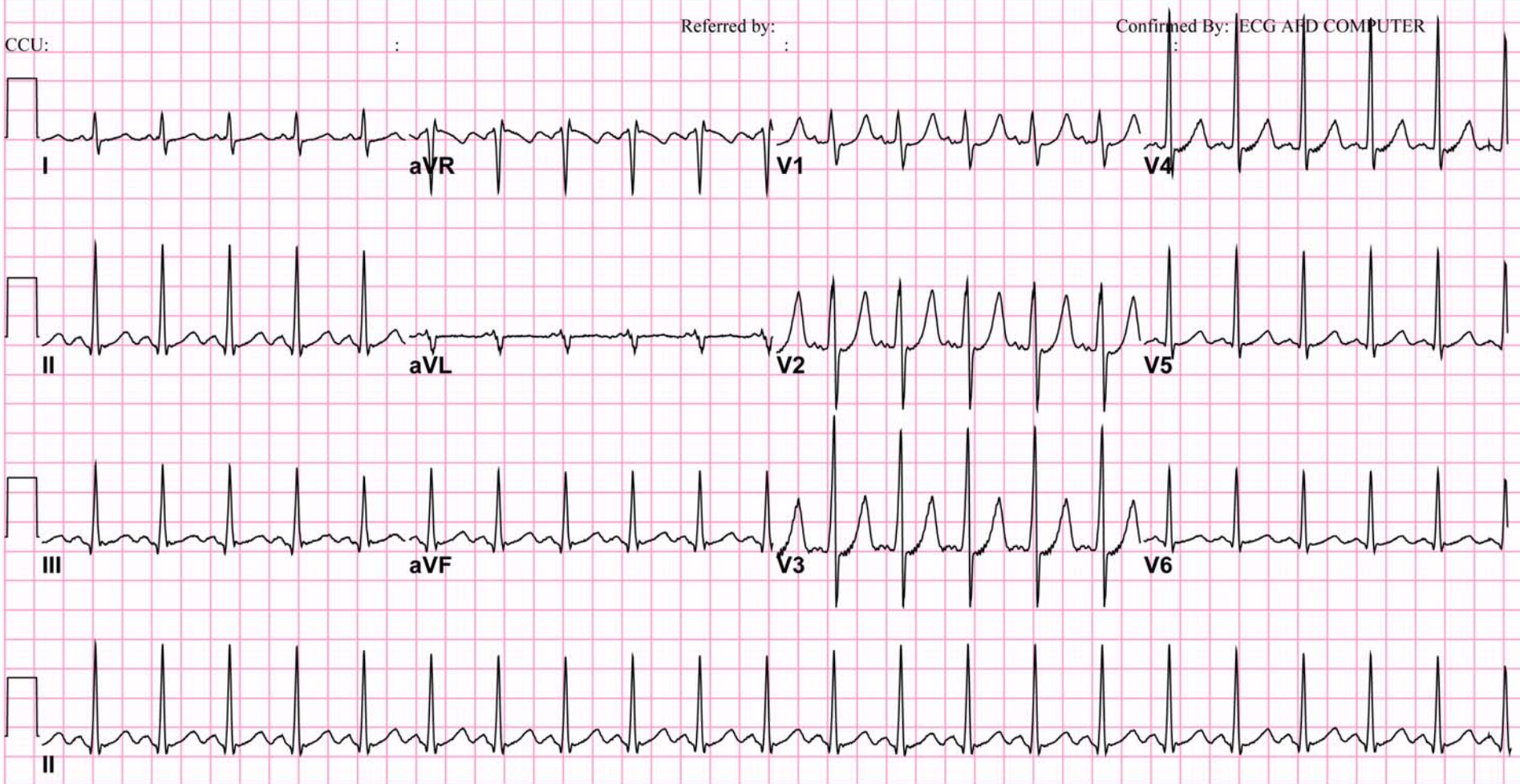
Technician:

CCU: :

Referred by: :

Confirmed By: ECG AED COMPUTER :



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EHBO: :

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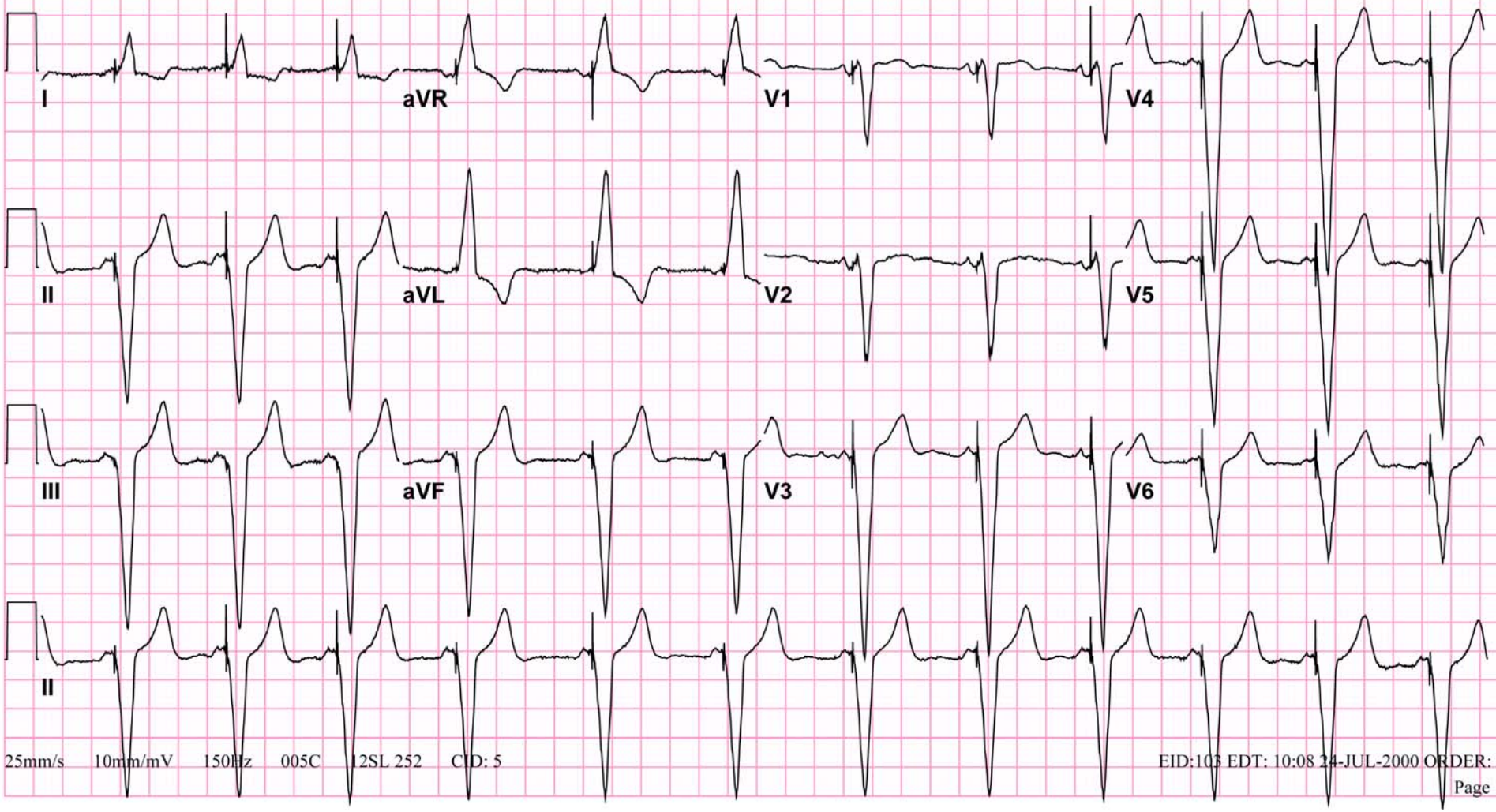

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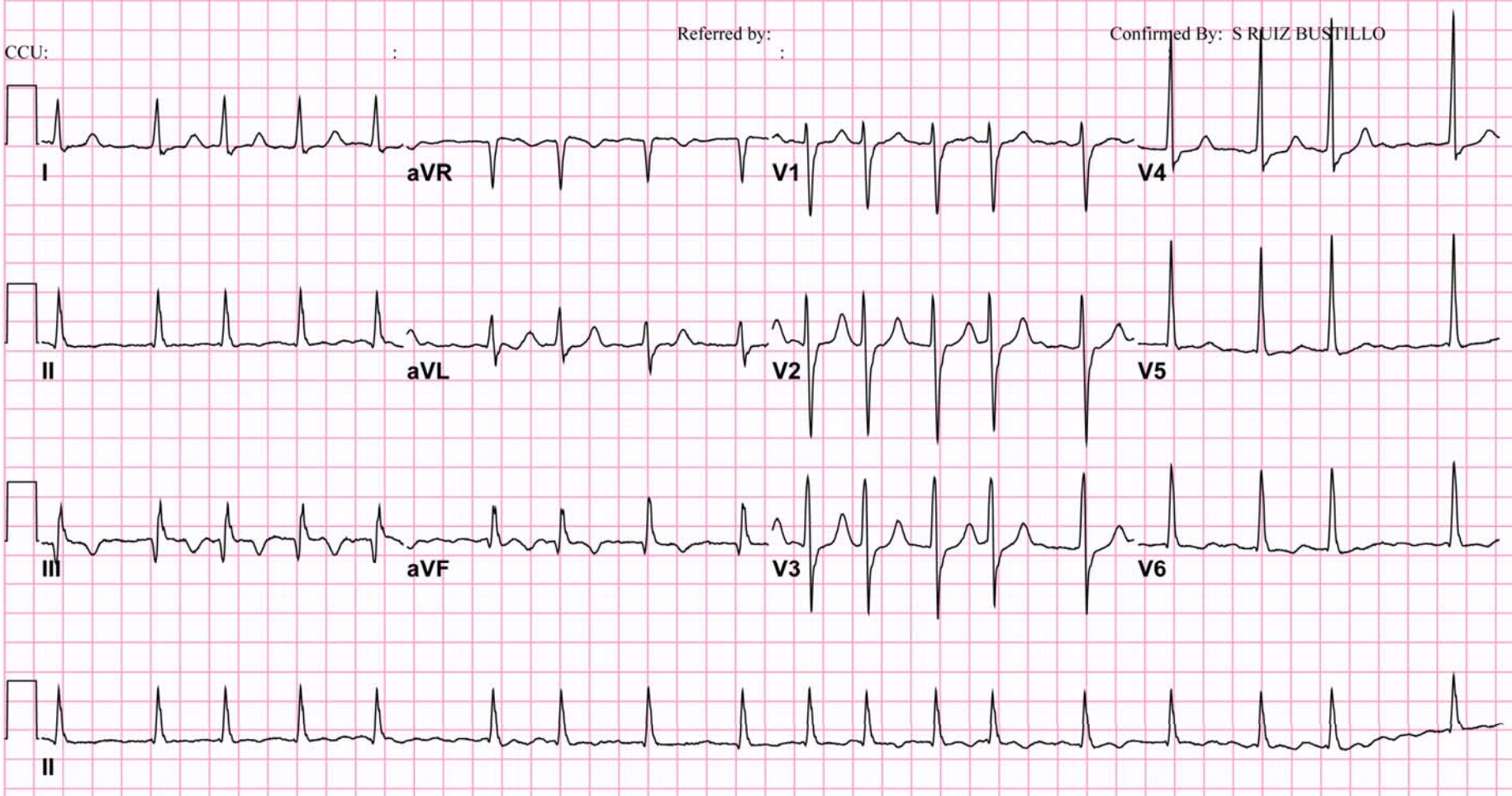

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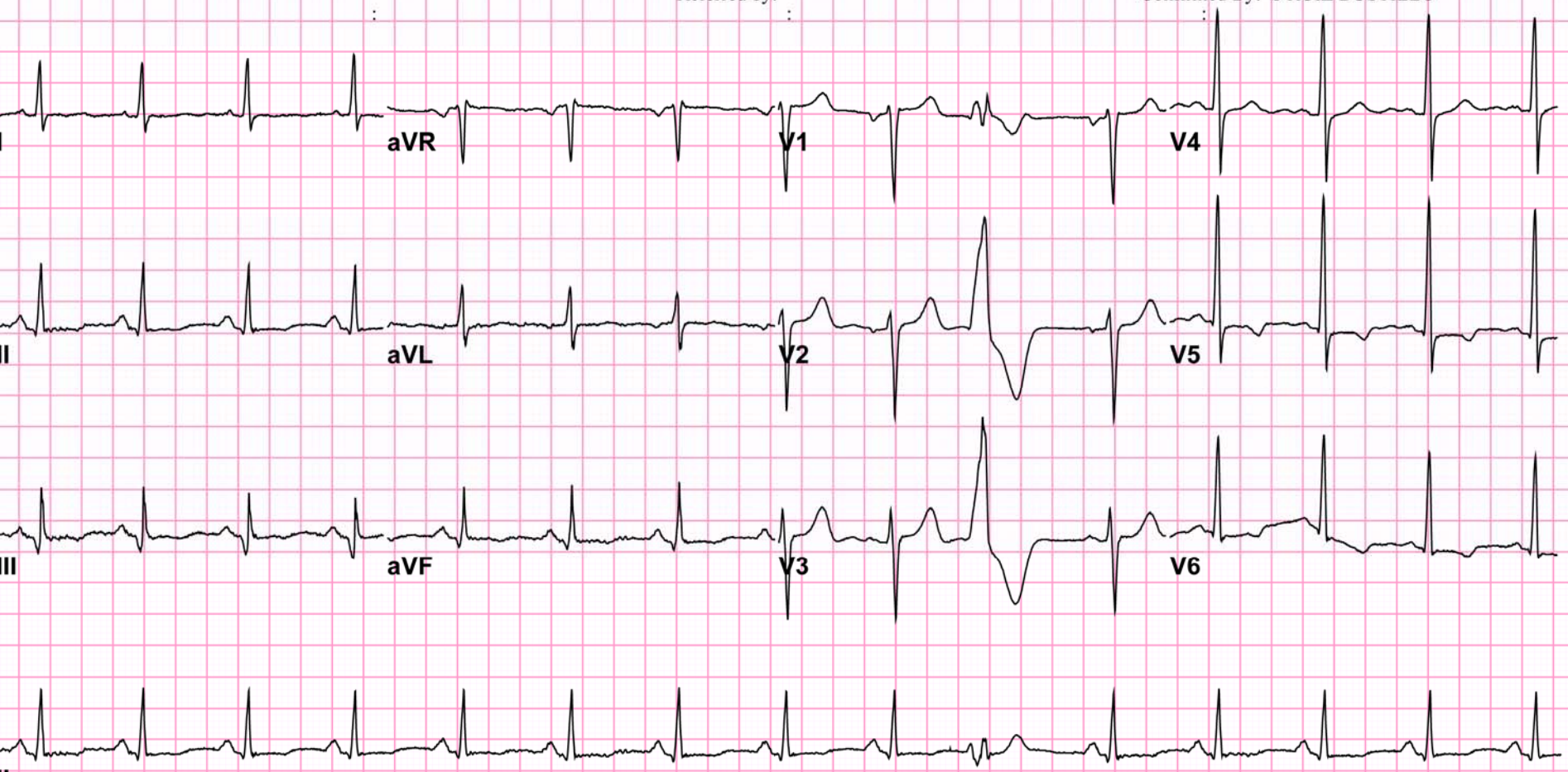

n/s 10mm/mV 150Hz 005C 12SL 254 CID: 7

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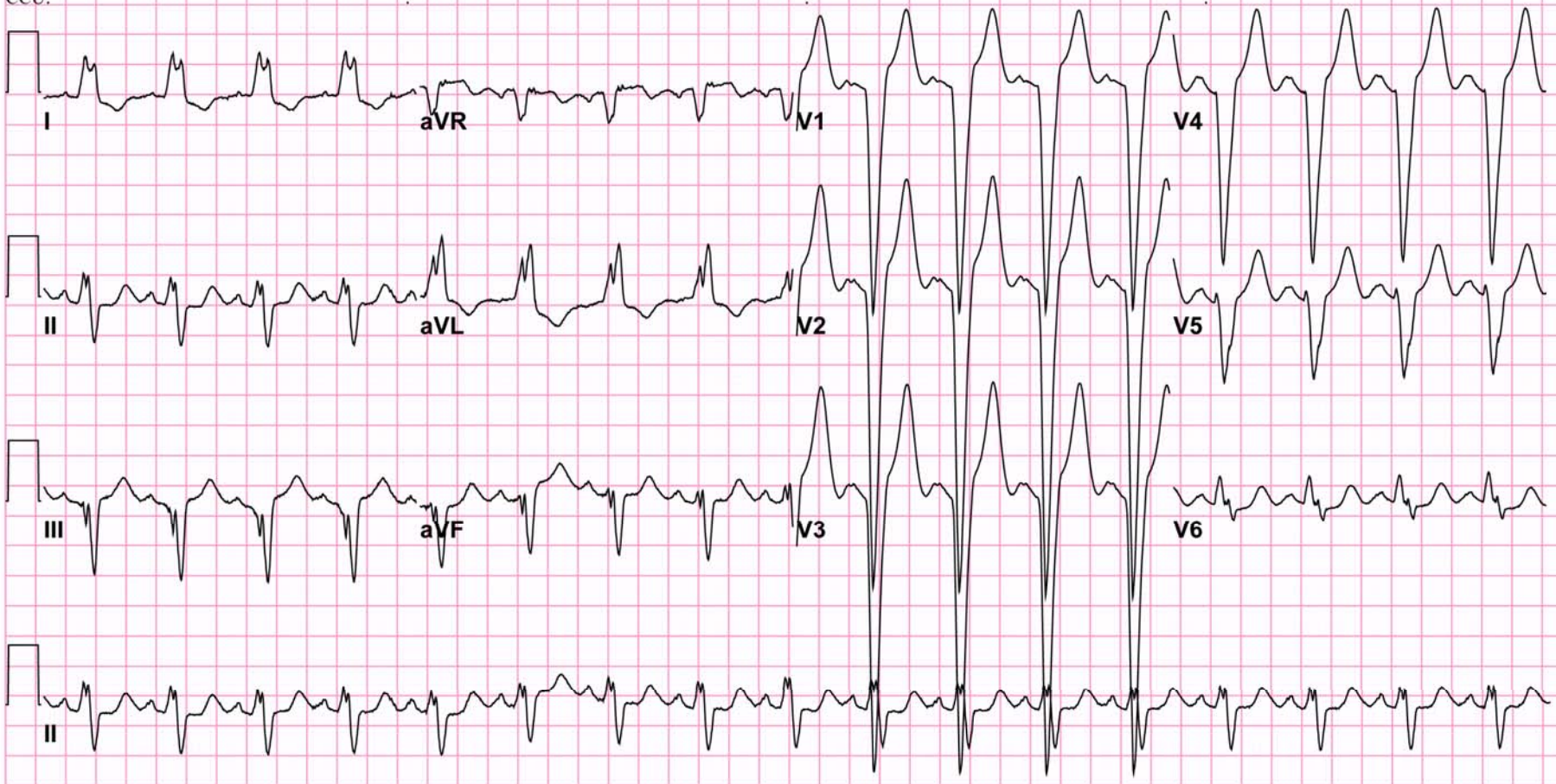

25mm/s 10mm/mV 150Hz 005C 12SL 252 CID: 4

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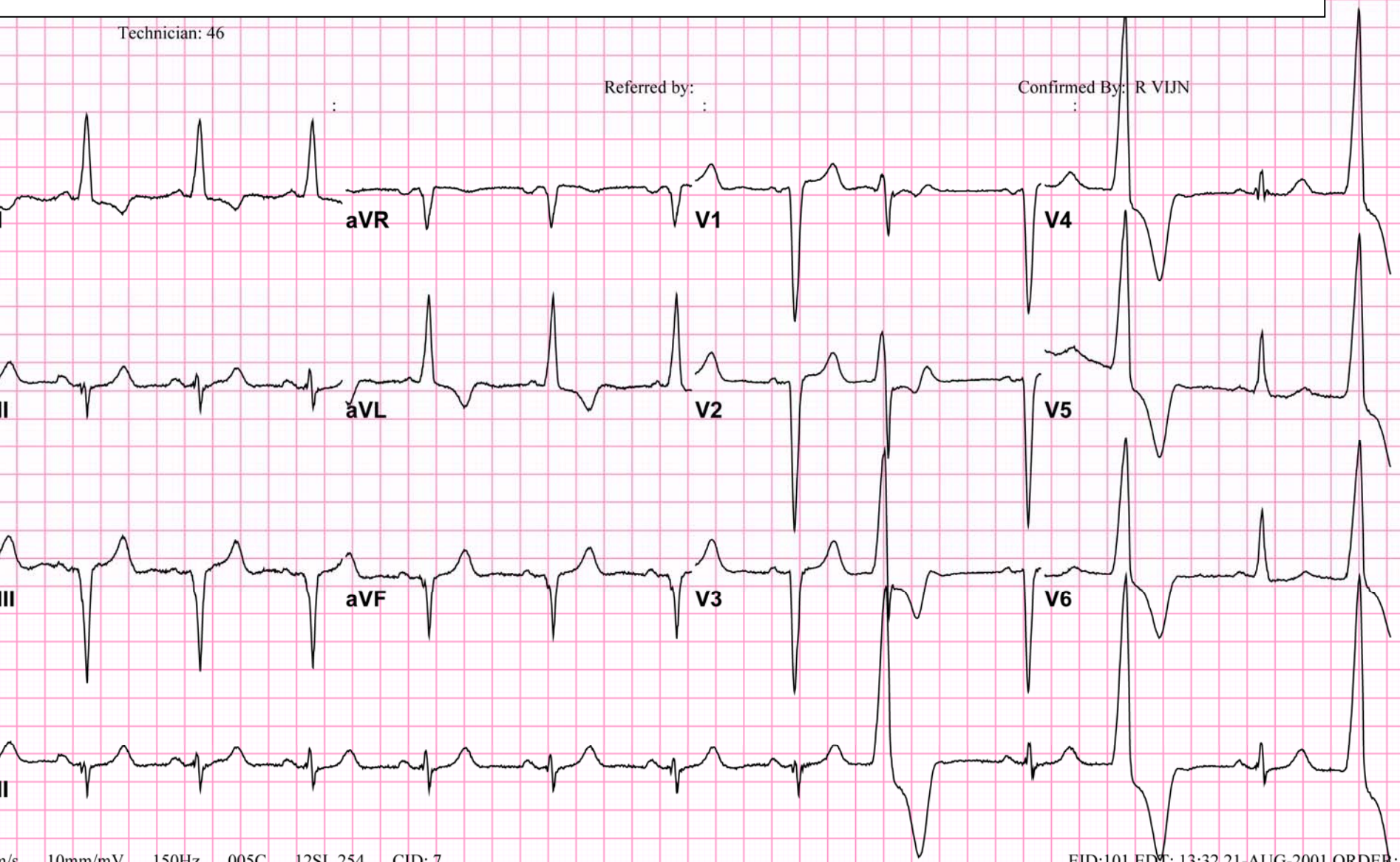

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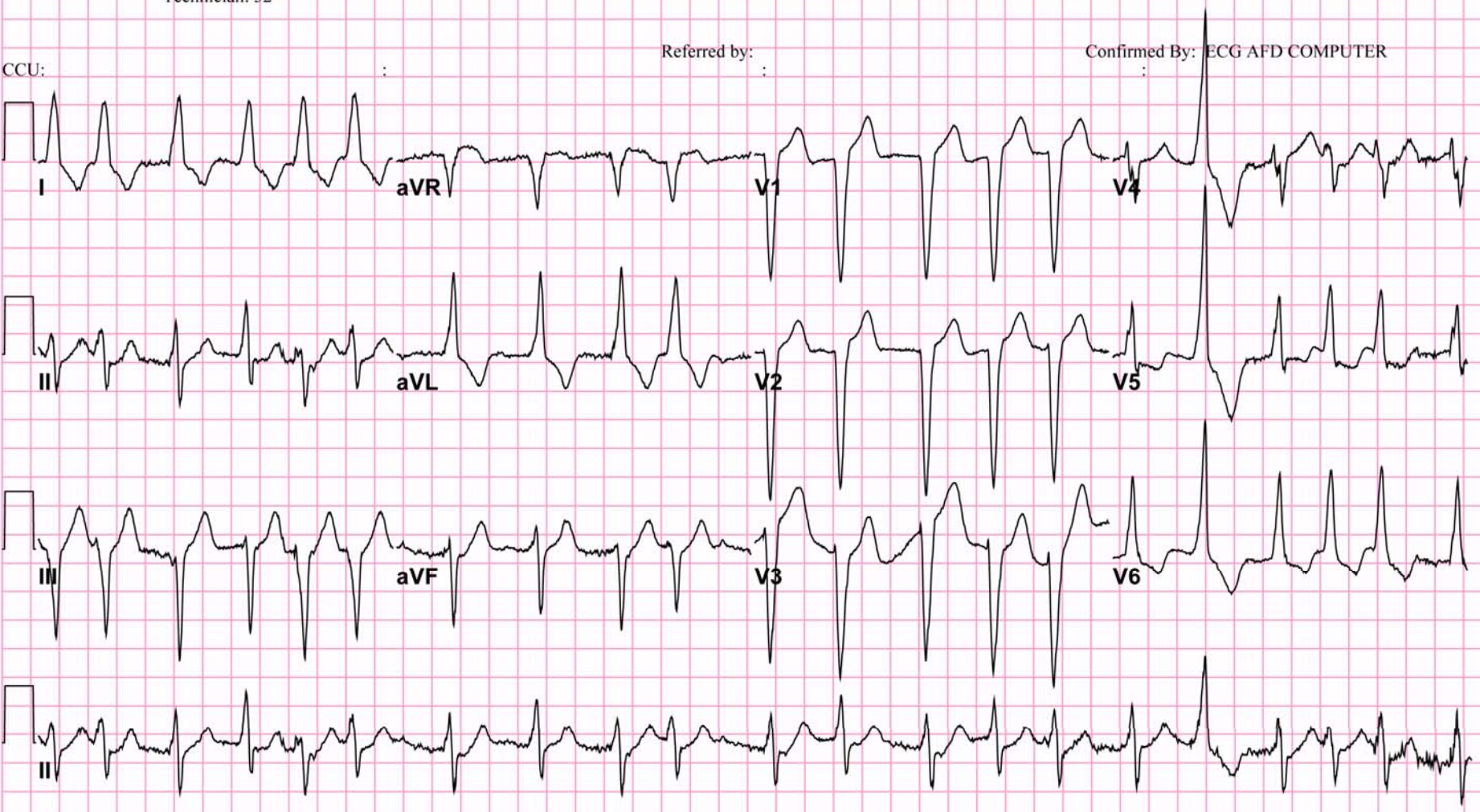

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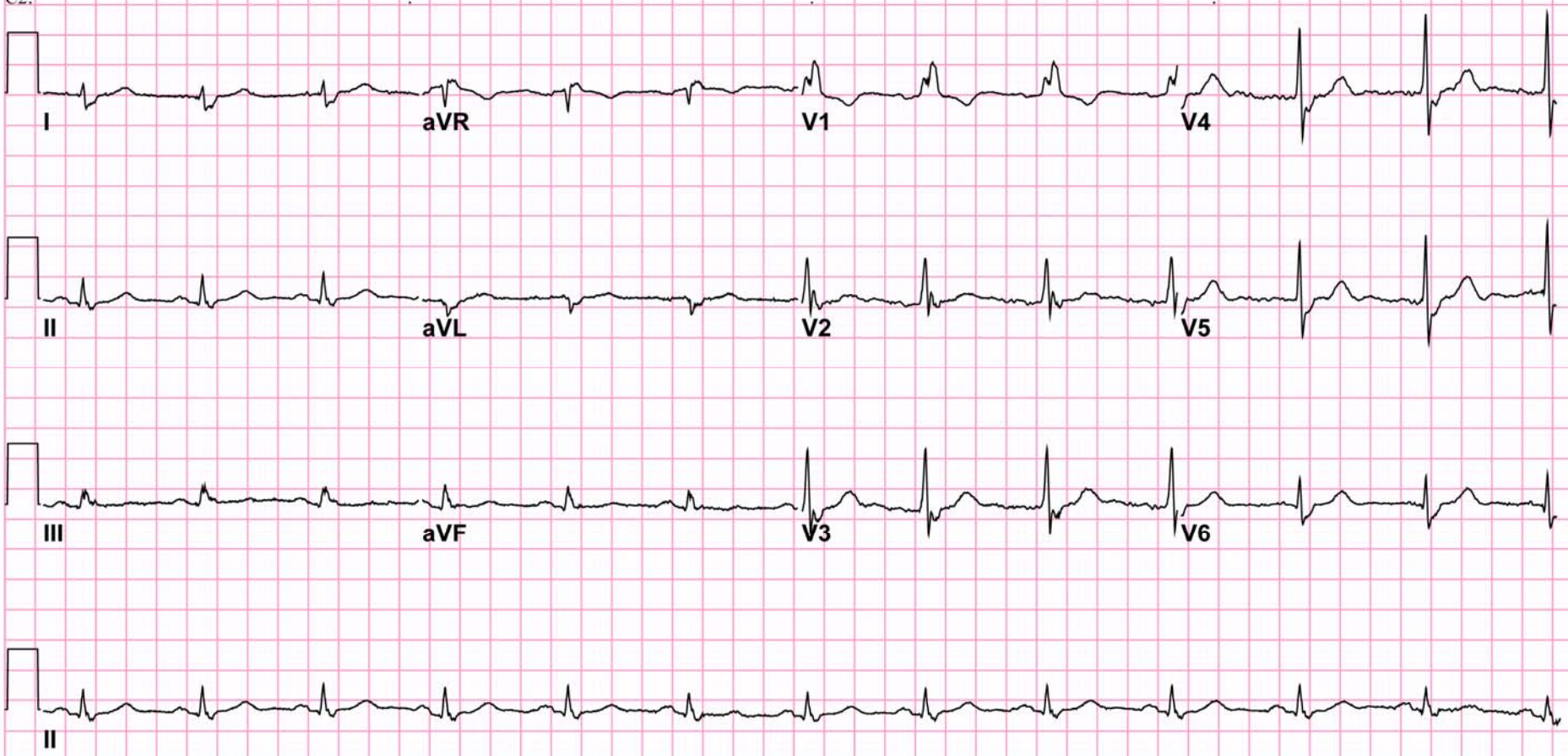
C2:



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Technician: 56

EHBO:

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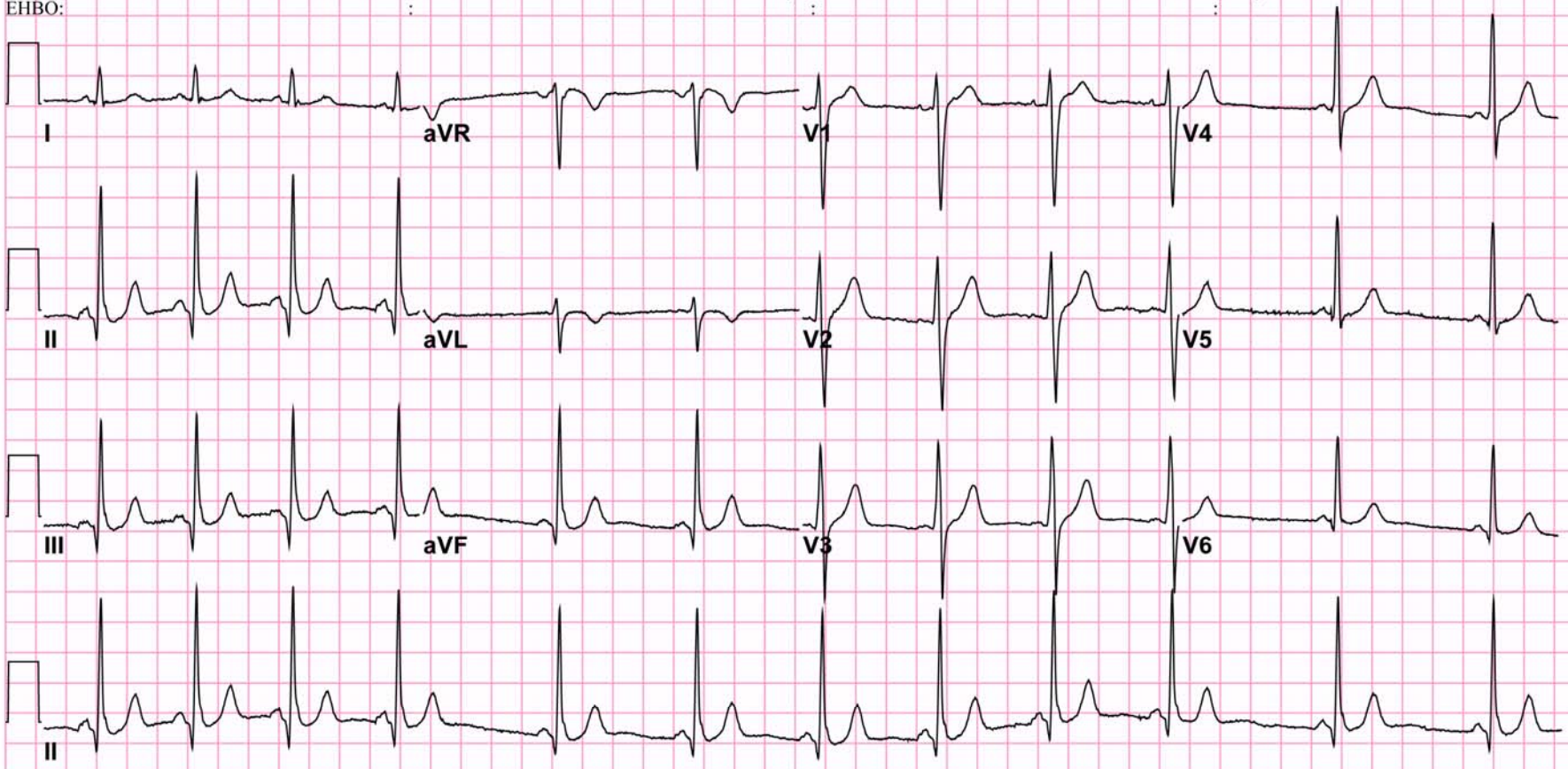

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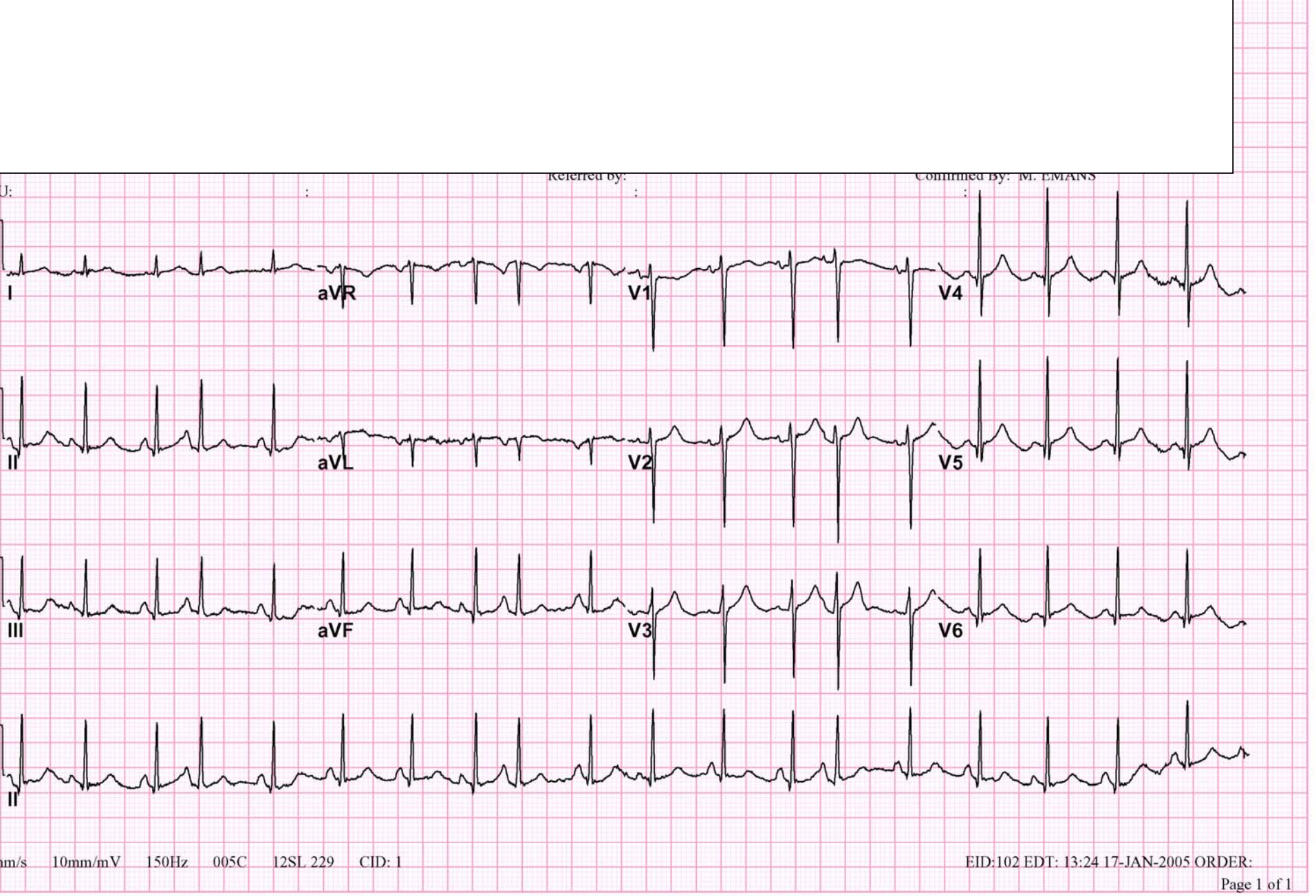
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Confirmed By: M. EMANS
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
10mm/mV
150Hz
005C
12SL 229
CID: 1
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Page 1 of 1

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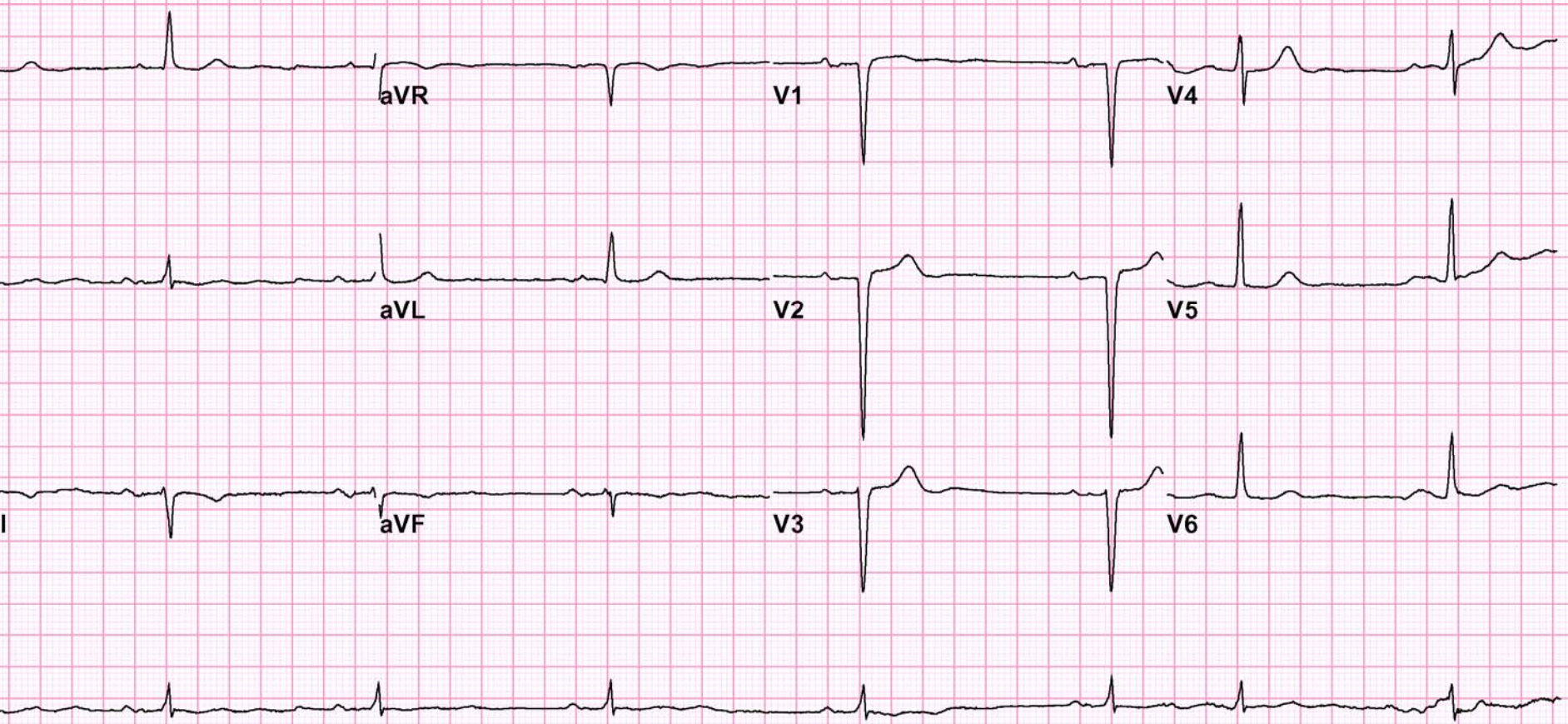

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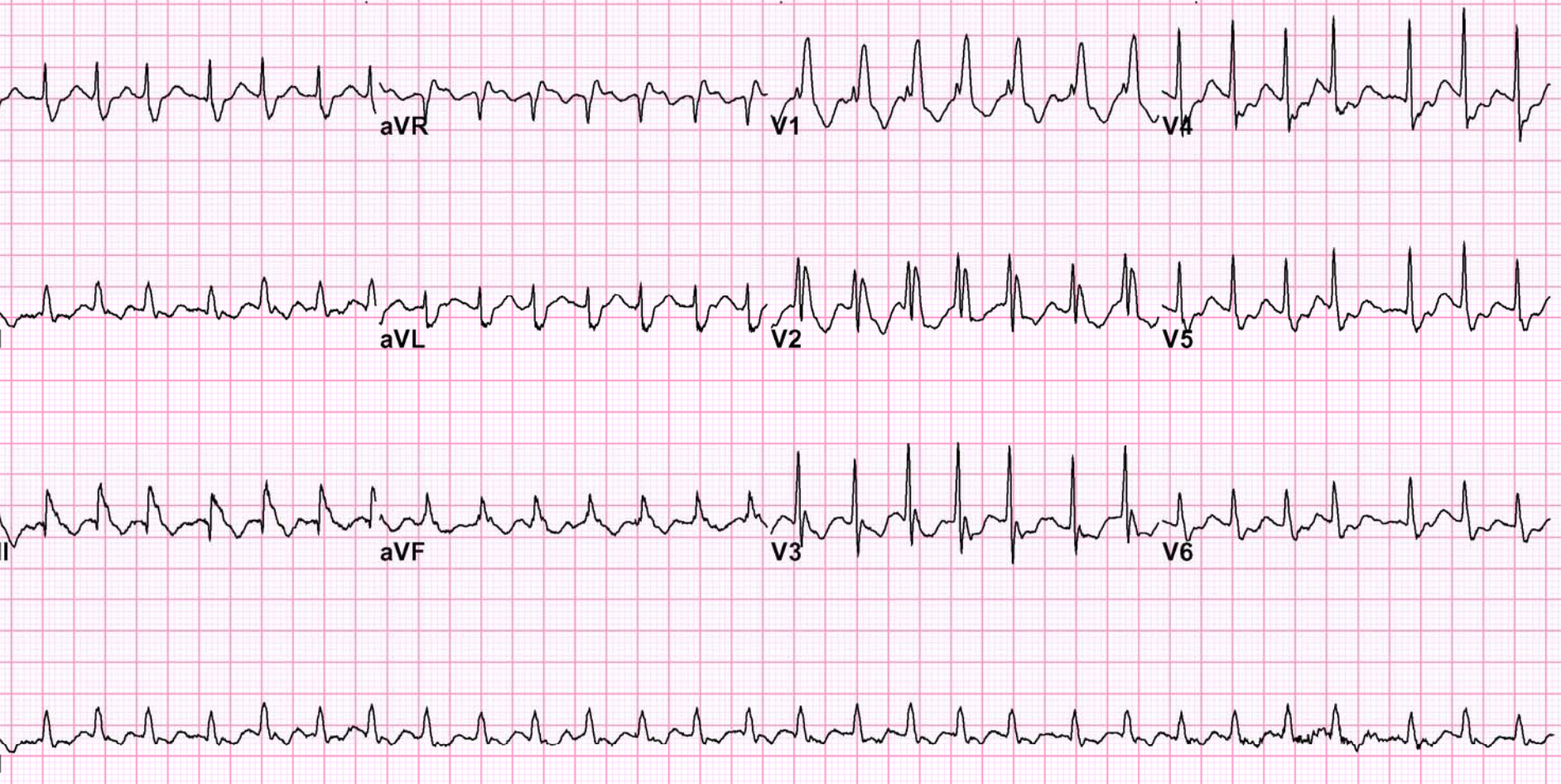

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