# ΣΤΕΦΑΝΙΑΙΑ ΝΟΣΟΣ ΠΕΡΙΚΑΡΔΙΤΙΔΑ

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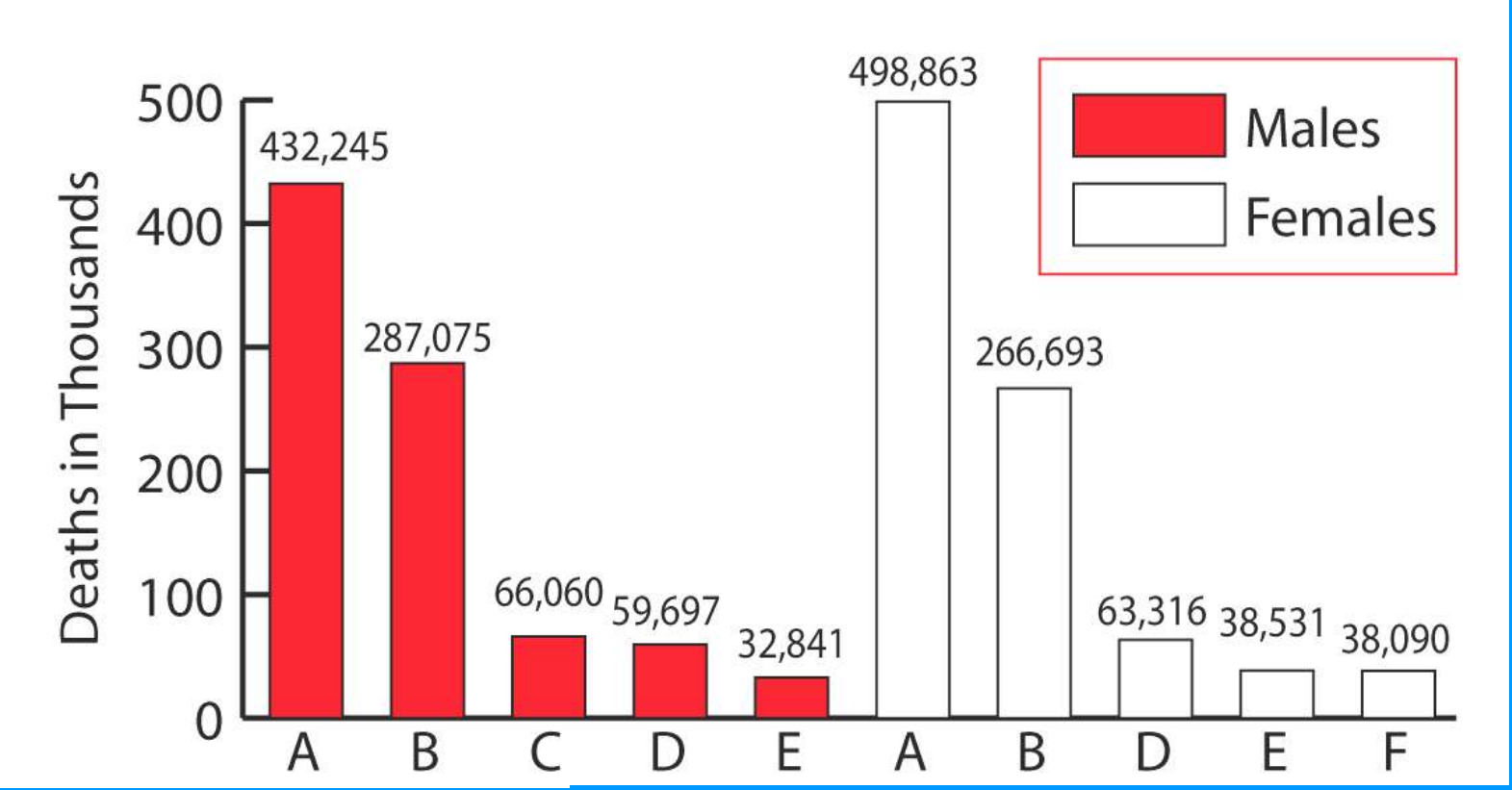
# ΣΤΕΦΑΝΙΑΙΑ ΝΟΣΟΣ

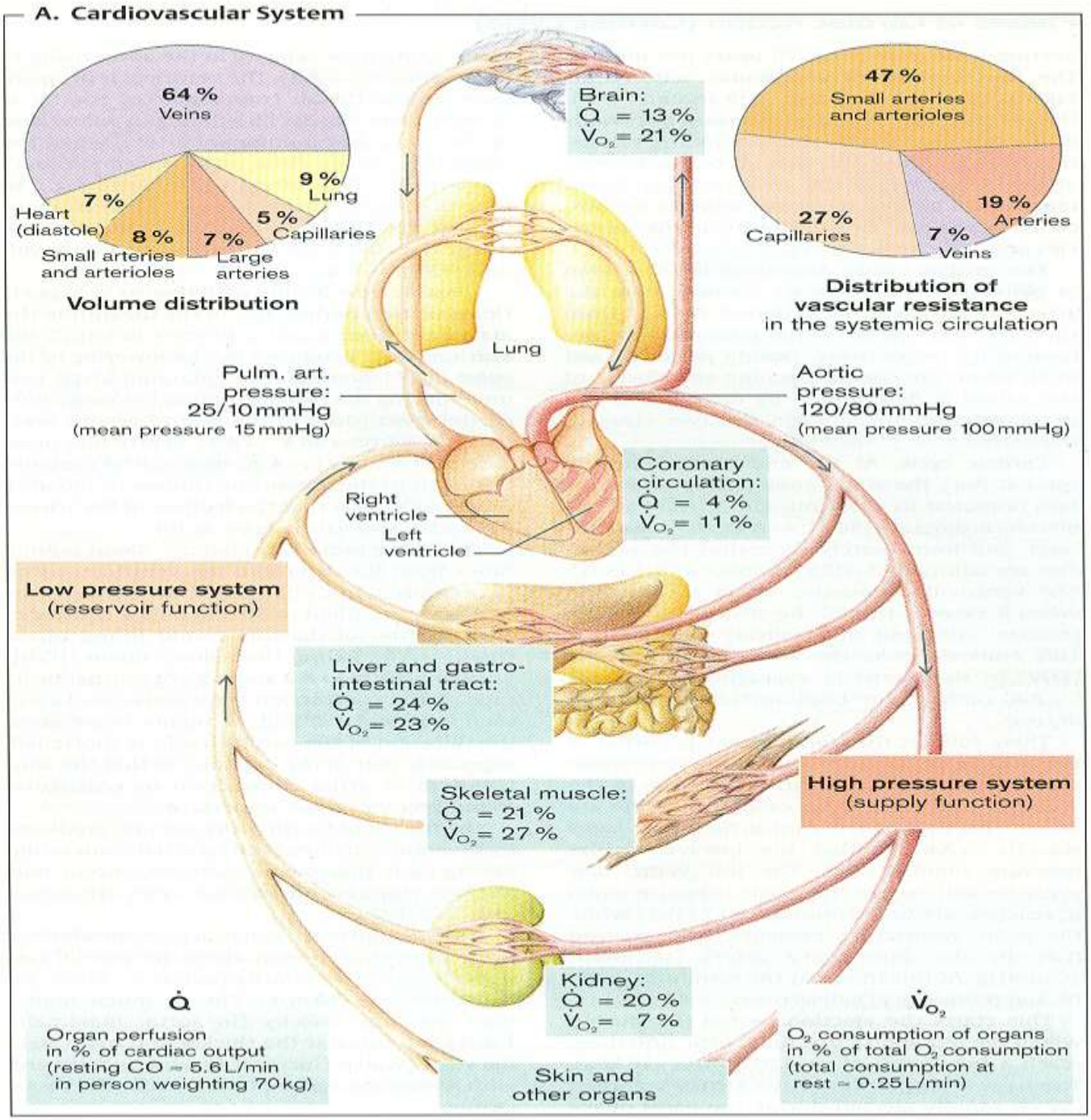
# Leading Causes of Death for All Males and Females

## United States: 2001




A Total CVD
B Cancer
C Accidents
D Chronic Lower Respiratory Diseases
E Diabetes Mellitus
F Alzheimer's Disease

***Source: CDC/NCHS.***


A. Cardiovascular System
64 %
Veins
9 %
Lung
7 %
Heart
(diastole)
5 %
Capillaries
8 %
Small arteries
and arterioles
7 %
Large
arteries
Volume distribution
47 %
Small arteries
and arterioles
19 %
Arteries
27 %
Capillaries
7 %
Veins
Distribution of
vascular resistance
in the systemic circulation
Brain:
$\dot{Q}$ = 13 %
$\dot{V}_{O_2}$ = 21 %
Lung
Pulm. art.
pressure:
25/10 mmHg
(mean pressure 15 mmHg)
Aortic
pressure:
120/80 mmHg
(mean pressure 100 mmHg)
Coronary
circulation:
$\dot{Q}$ = 4 %
$\dot{V}_{O_2}$ = 11 %
Right
ventricle
Left
ventricle
Low pressure system
(reservoir function)
Liver and gastro-
intestinal tract:
$\dot{Q}$ = 24 %
$\dot{V}_{O_2}$ = 23 %
Skeletal muscle:
$\dot{Q}$ = 21 %
$\dot{V}_{O_2}$ = 27 %
High pressure system
(supply function)
Kidney:
$\dot{Q}$ = 20 %
$\dot{V}_{O_2}$ = 7 %
Skin and
other organs
$\dot{Q}$
Organ perfusion
in % of cardiac output
(resting CO = 5.6 L/min
in person weighting 70 kg)
$\dot{V}_{O_2}$
$O_2$ consumption of organs
in % of total $O_2$ consumption
(total consumption at
rest = 0.25 L/min)

# Control Of Cardiac Function

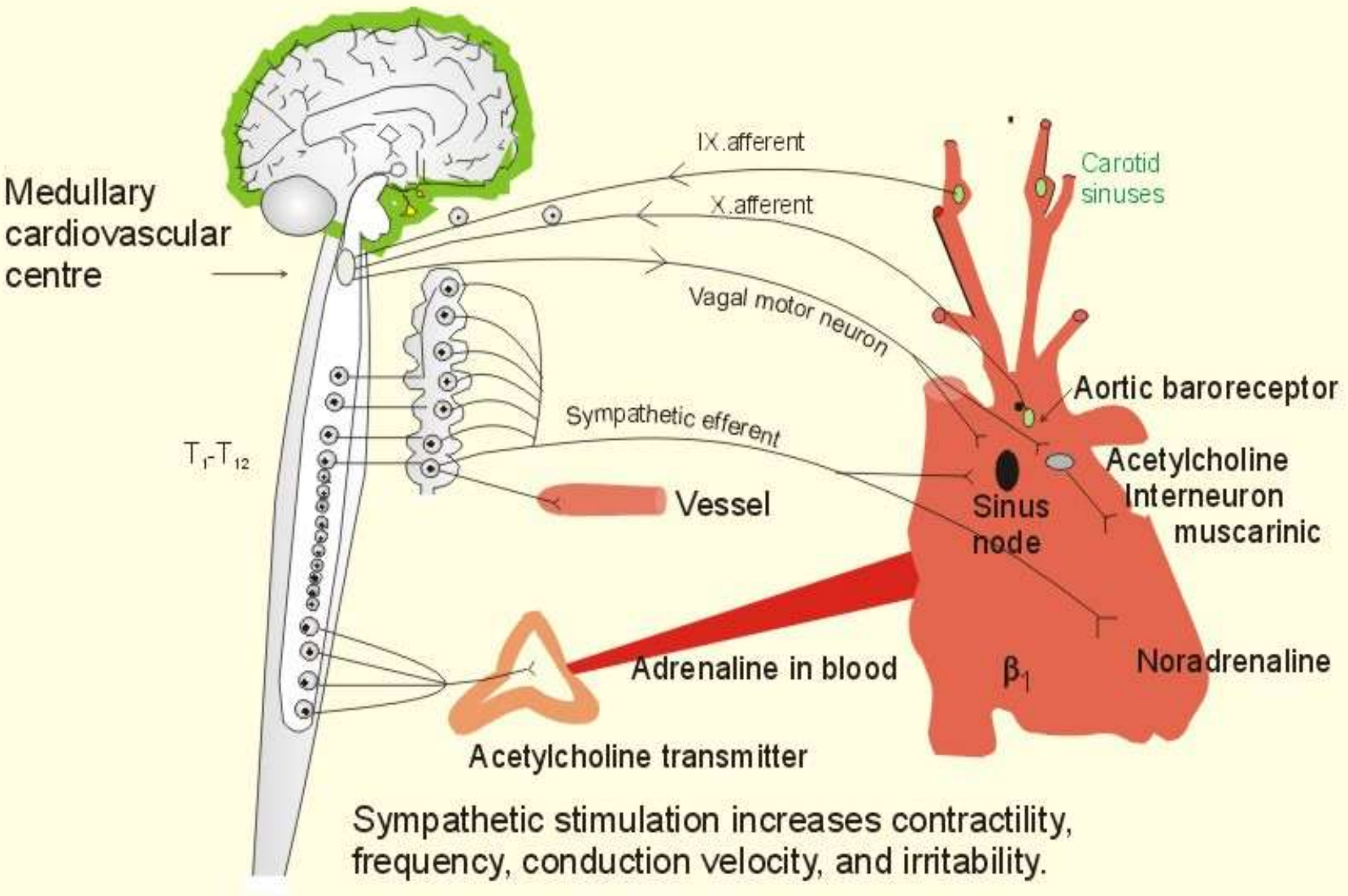
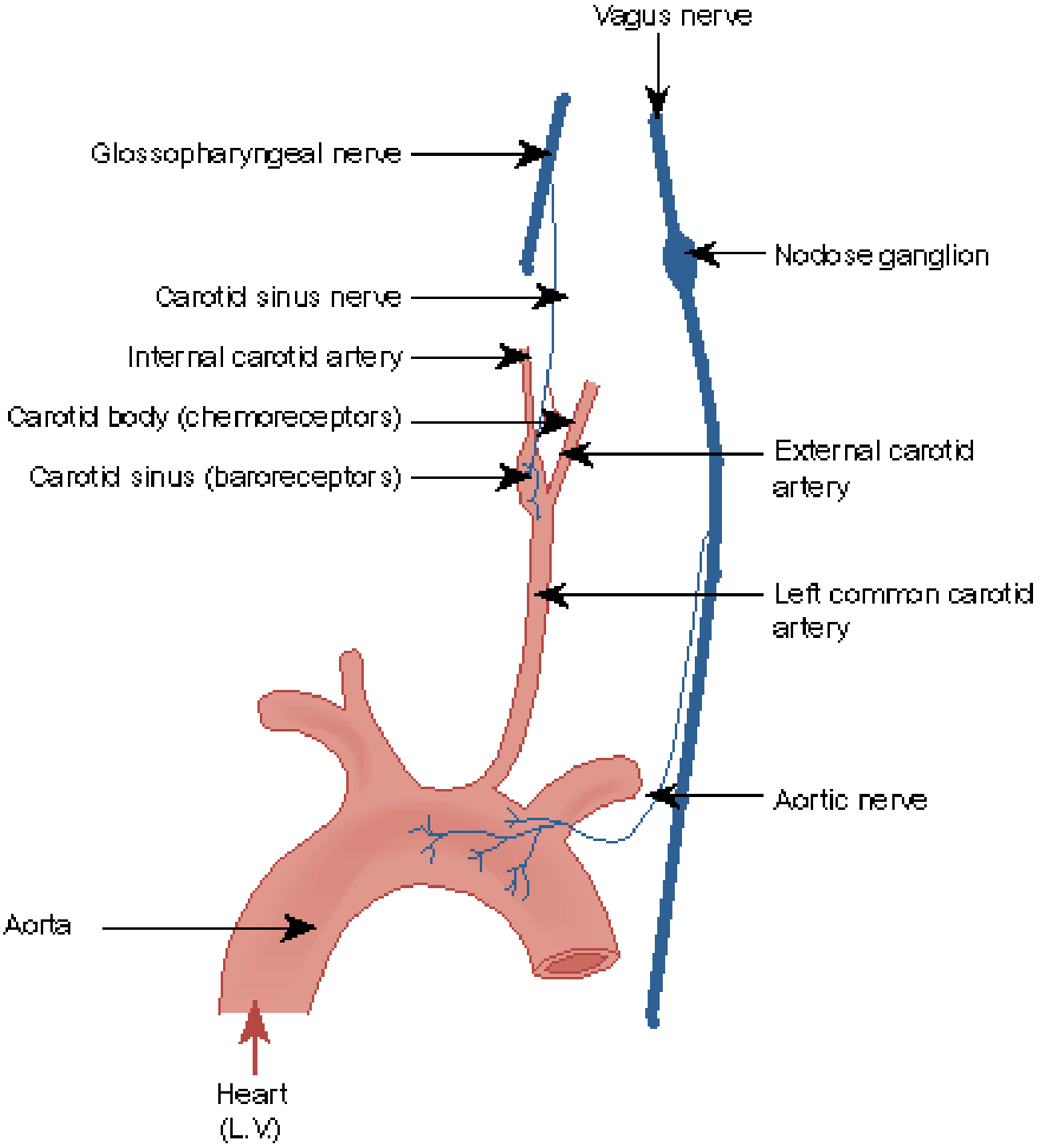



Fig. 11-1

KMc


Vagus nerve
Glossopharyngeal nerve
Nodose ganglion
Carotid sinus nerve
Internal carotid artery
Carotid body (chemoreceptors)
Carotid sinus (baroreceptors)
External carotid artery
Left common carotid artery
Aortic nerve
Aorta
Heart (L.V.)

# One Cardiac Cycle At Rest

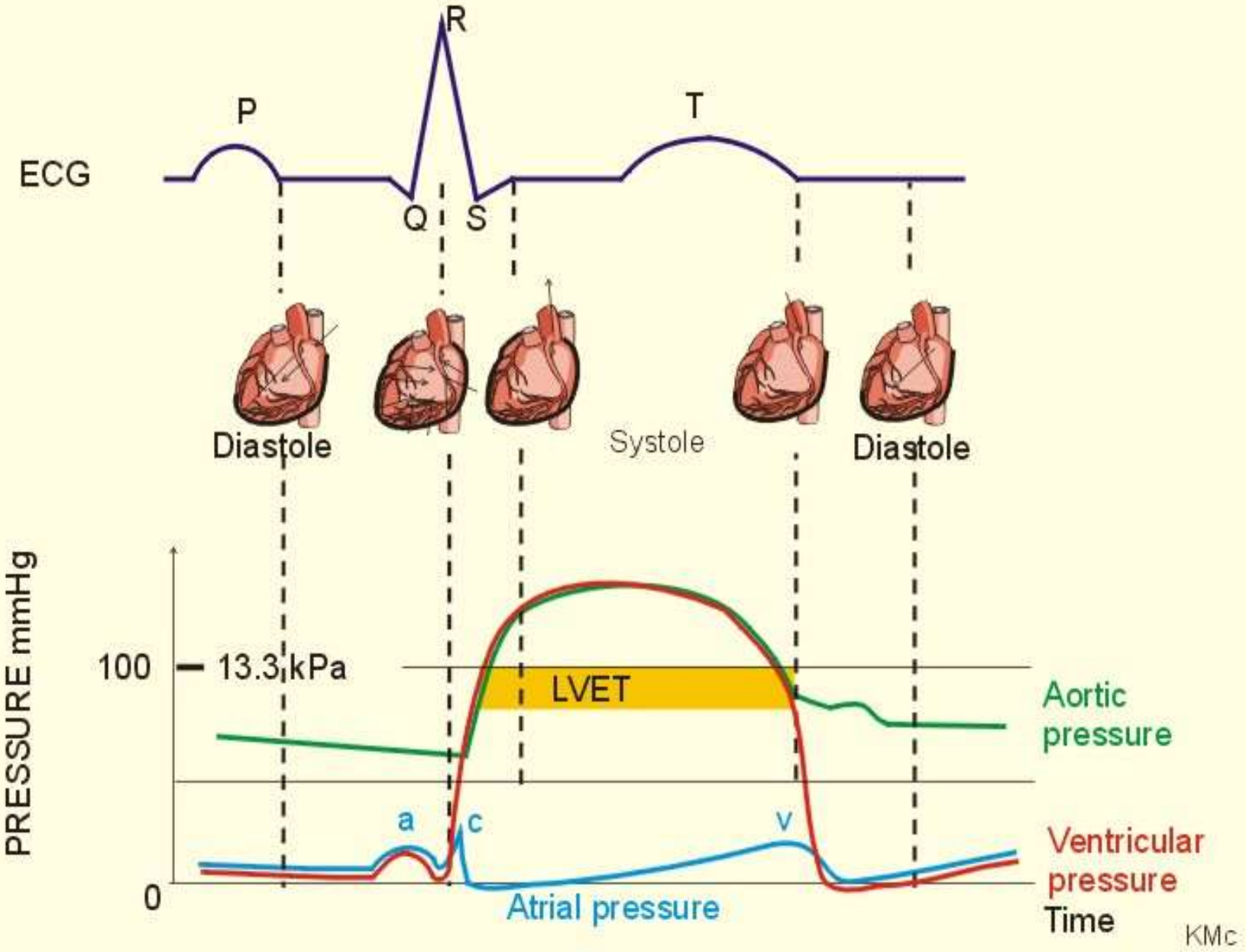
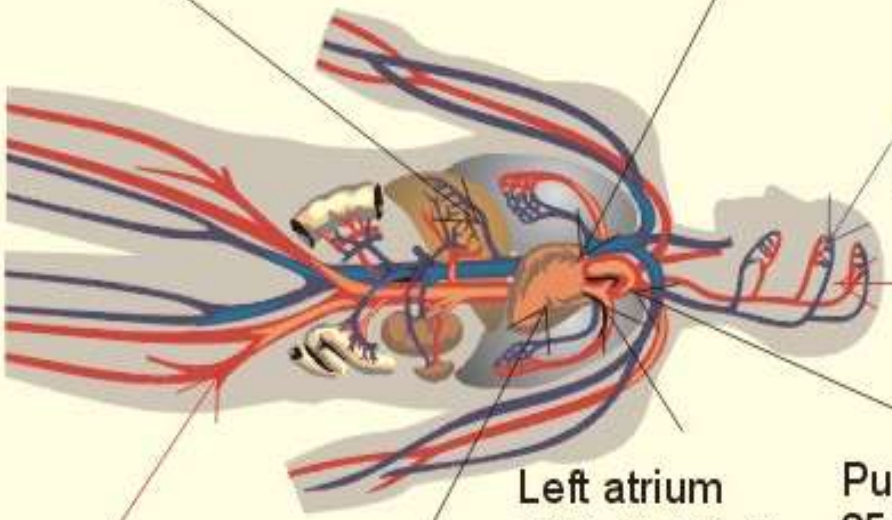



Fig. 10-1

KMc

# Normal Systolic And Diastolic Pressures

Supine person at rest. Pressures in mmHg and (kPa)



Vena portae
10-12 (1.3 - 1.6 )

Right atrium
6/-2 (0.8/-0.3)

Capillaries
20 (2.7)

Veins
10 (2.7)

Arteries
120/80 (16/10.7)

Left atrium
12/1 (1.6/0.1)
Mean: 0.7

Left ventricle:
120/1 (16/0.1)

Pulmonary artery:
25/10 (3.3/1.3)
Mean: 1.7

Right ventricle:
25/-1 (3.3/-0.13)

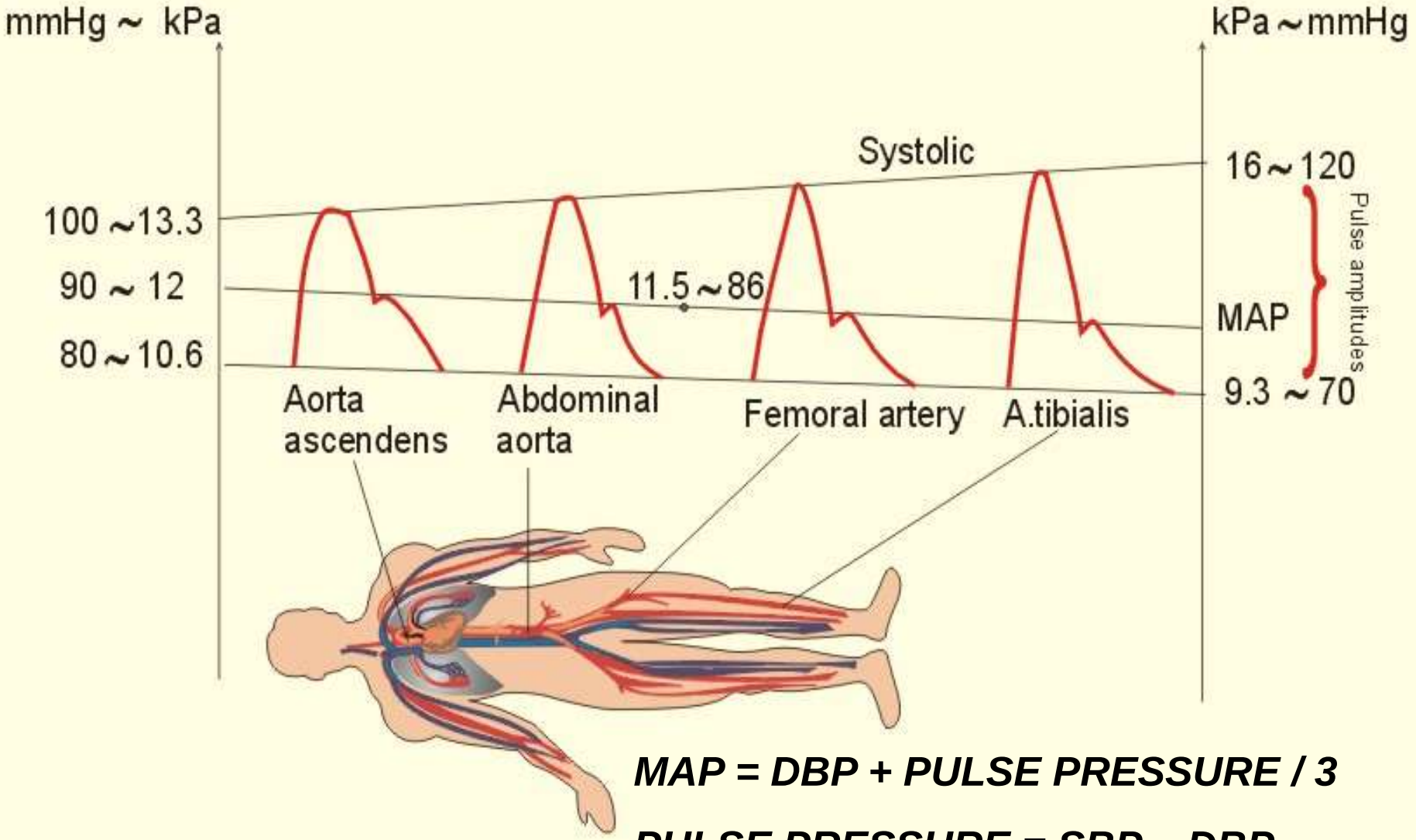
Pulmonary driving pressure: 1.7 - 0.7 = 1 kPa

*Pulmonary vascular resistance (PVR)* = Driving pressure/Q

Fig. 9-4

KMc

# Changes In Arterial Pressures In A Supine Person




***MAP = DBP + PULSE PRESSURE / 3***

***PULSE PRESSURE = SBP – DBP***

Fig. 9-3

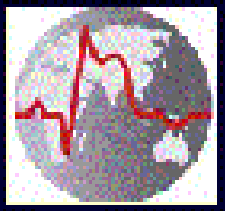
KMc

Coronary heart disease (CHD) is the most common form of heart disease, the leading cause of death for Americans.

About 12.6 million Americans suffer from CHD, which often results in a heart attack.

About 1.1 million Americans suffer a heart attack each year—about 515,000 of these heart attacks are fatal.

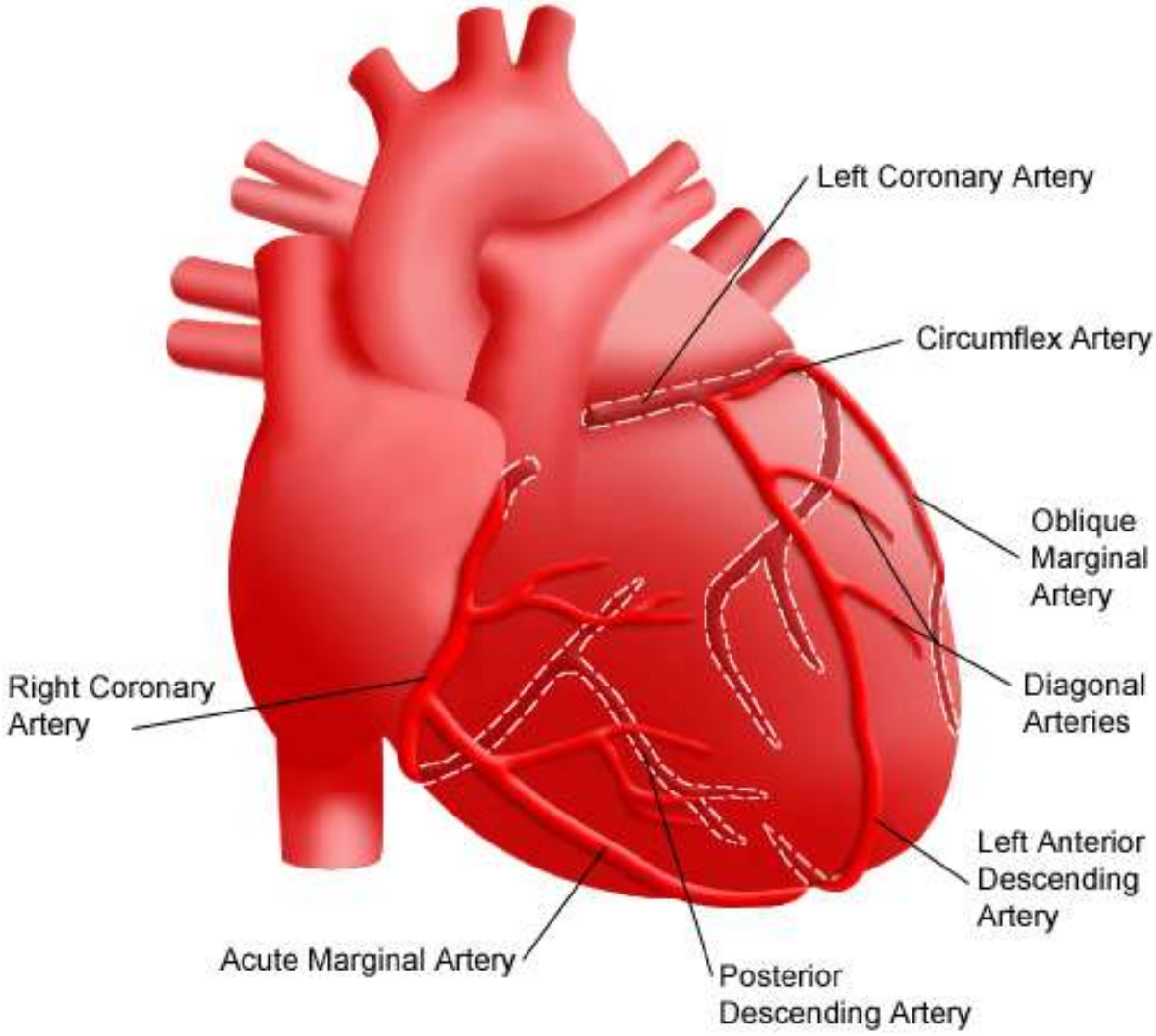
# PROCAM: Ranking of Risk Factors



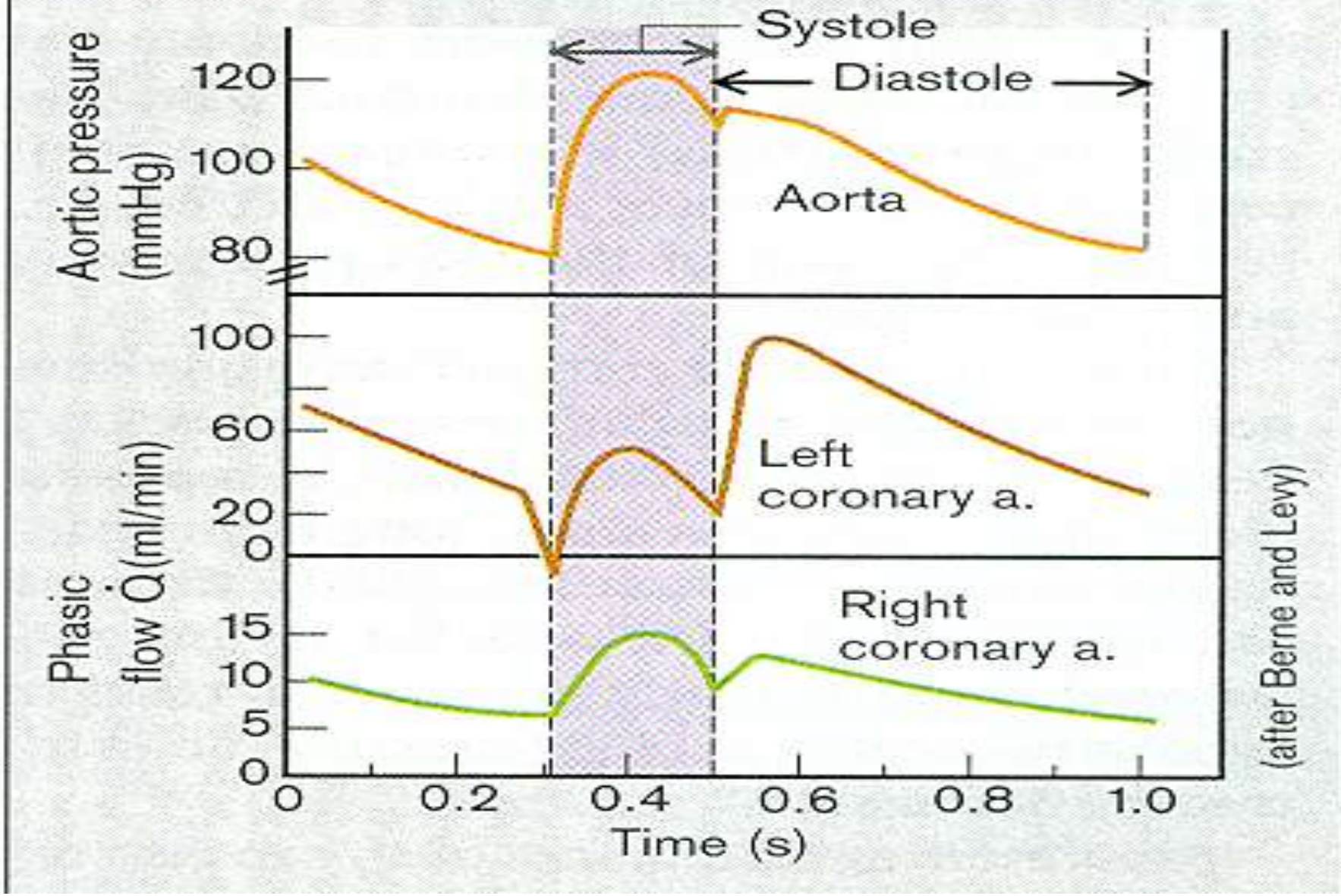
| | R |
|---|---|
| 1. Age | 0.2395 |
| 2. LDL cholesterol | 0.2096 |
| 3. Smoking | 0.1309 |
| 4. HDL cholesterol | -0.1018 |
| 5. Systolic blood pressure | 0.0955 |
| 6. Diabetes | 0.0635 |
| 7. Triglycerides | 0.0625 |
| 8. Family history of MI | 0.0523 |

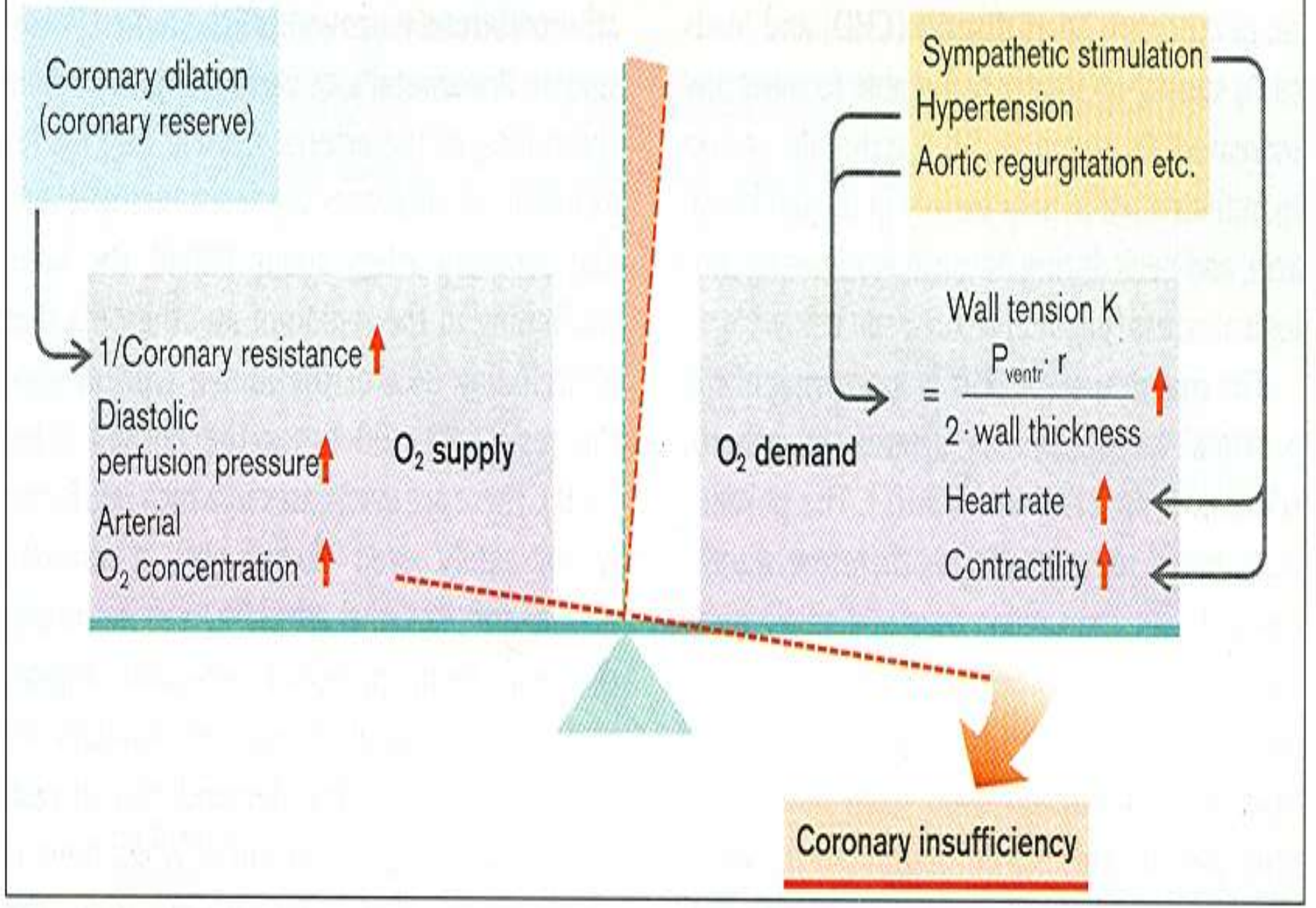
Assmann, Cullen, Schulte; Circulation. 105: 310-315; 2002

# Coronary Arteries of the Heart



# A. Coronary Blood Flow




C. Components of $O_2$ Balance in Myocardium
Coronary dilation
(coronary reserve)
Sympathetic stimulation
Hypertension
Aortic regurgitation etc.
1/Coronary resistance
Diastolic
perfusion pressure
Arterial
$O_2$ concentration
$O_2$ supply
$O_2$ demand
Wall tension K
$= \frac{P_{ventr} \cdot r}{2 \cdot \text{wall thickness}}$
Heart rate
Contractility
Coronary insufficiency

## C. Myocardial Energy Metabolism

**Normal**

Glucose → G-6-P → Pyruvate → Acetyl-CoA → $CO_2$

Little ATP

Pyruvate → NADH

Acetyl-CoA + $O_2$ → ATP

$FADH_2$ → $NAD^+$ + FAD

Lactate (consumption)

Free fatty acids

ATP → Normal myocardial contraction

**Ischemic anoxia (early stage)**

Glucose → G-6-P → Pyruvate → Acetyl-CoA

Glycogen store → G-6-P

Little ATP

NADH + $O_2$ → $NAD^+$ + $CO_2$

$H^+$

Lactate (formation)

Free fatty acids → Intracellular fat deposition

FAD

Little ATP → ATP deficiency

Creatine phosphate store → Cr~P

Little ATP → Cr

Reversible cell damage

Abnormal myocardial contraction

No removal of $H^+$ and Lactate

**Ischemia** (longer than 15–20 min)

Acidosis

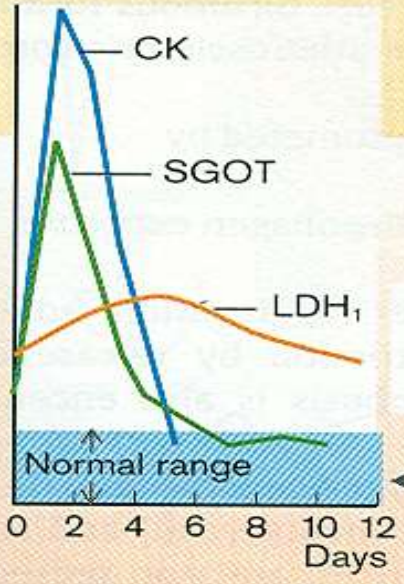
Lactate accumulation

Inhibits glycolysis (and others)

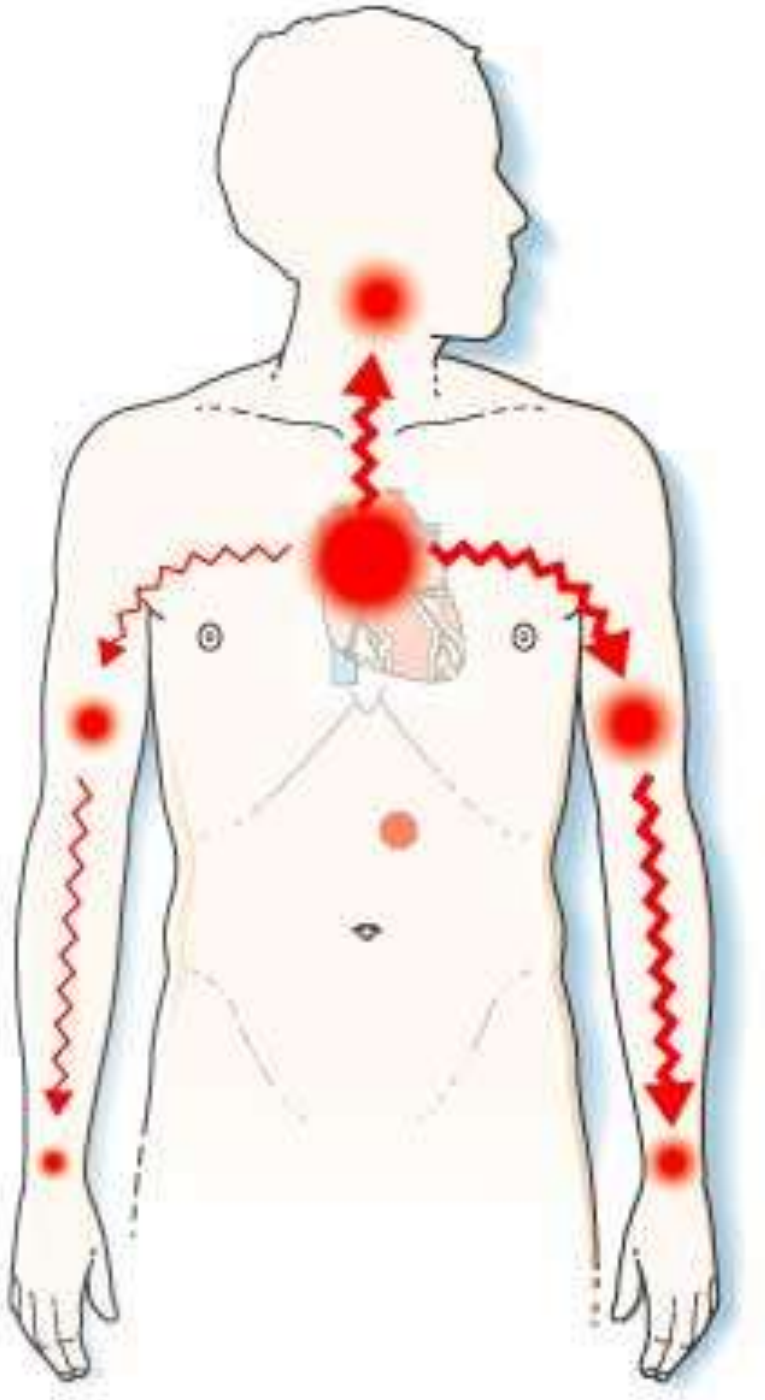
ATP deficiency

Irreversible cell damage → Infarction

**Angina pectoris**



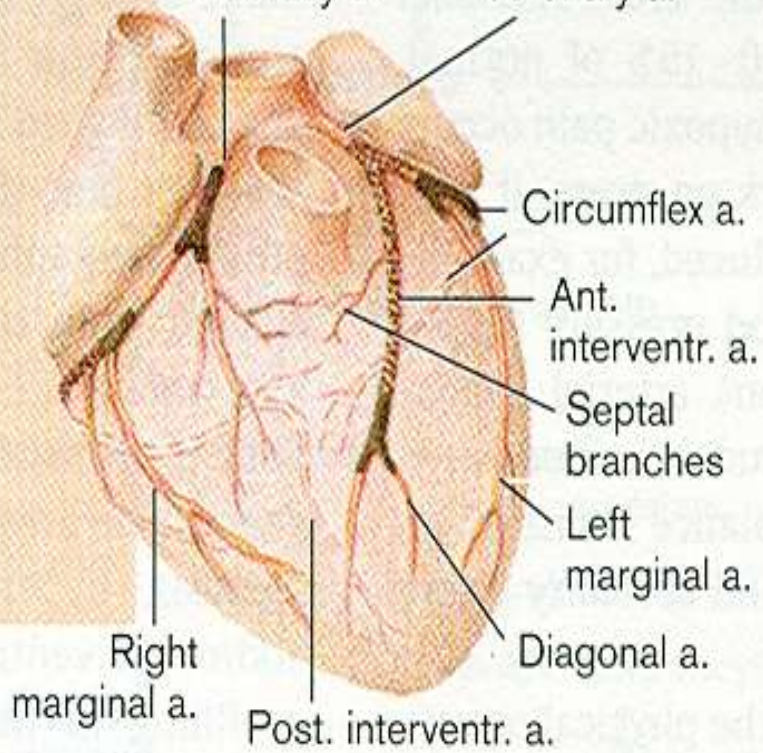

Enzyme released into plasma



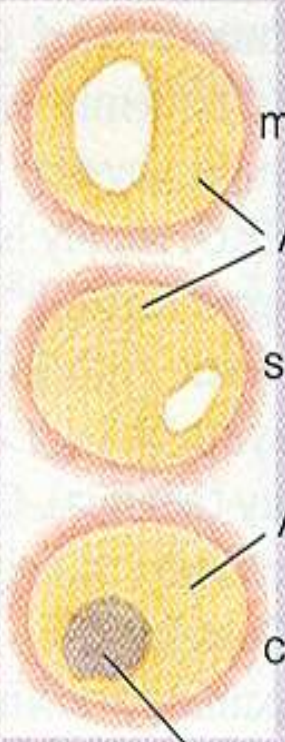
## D. Atherosclerosis of Coronary Arteries

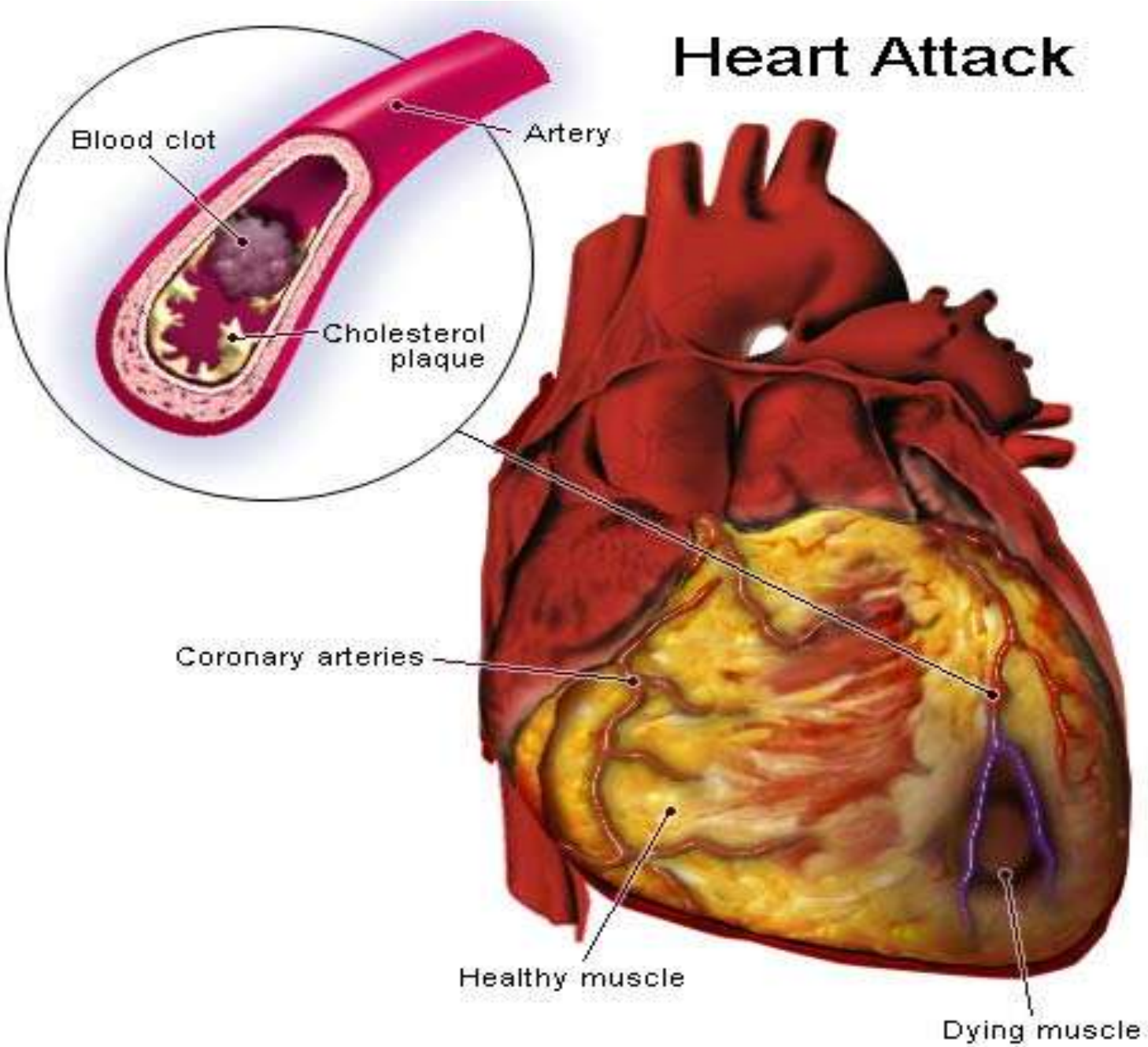
**Risk factors of atherosclerosis**

Smoking
Overweight
Lack of exercise
Food (fat, cholesterol)
Psychological stress
Lipid metabolism
Diabetes mellitus
Hypertension
Age etc.



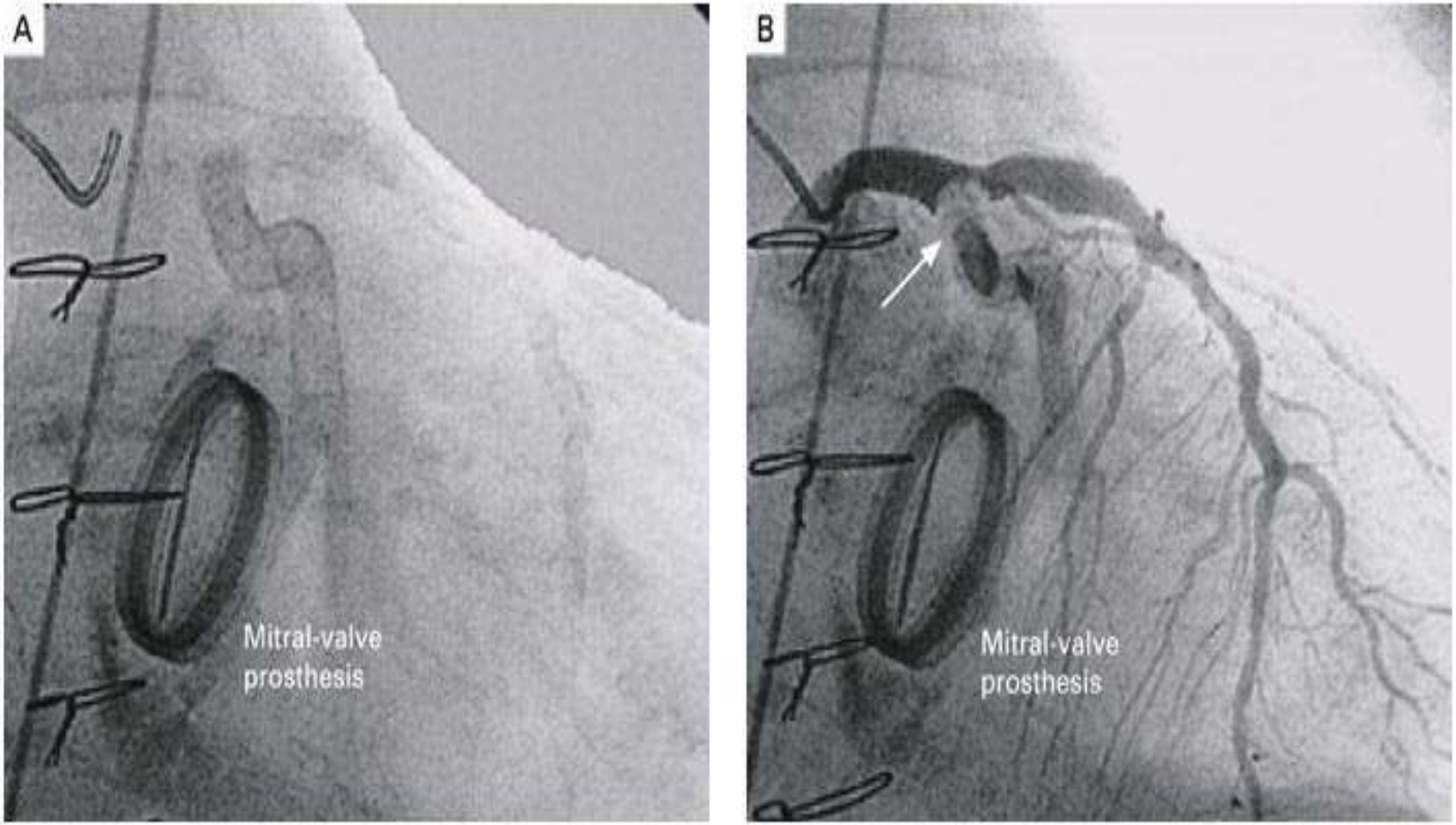

Obstruction of coronary a.

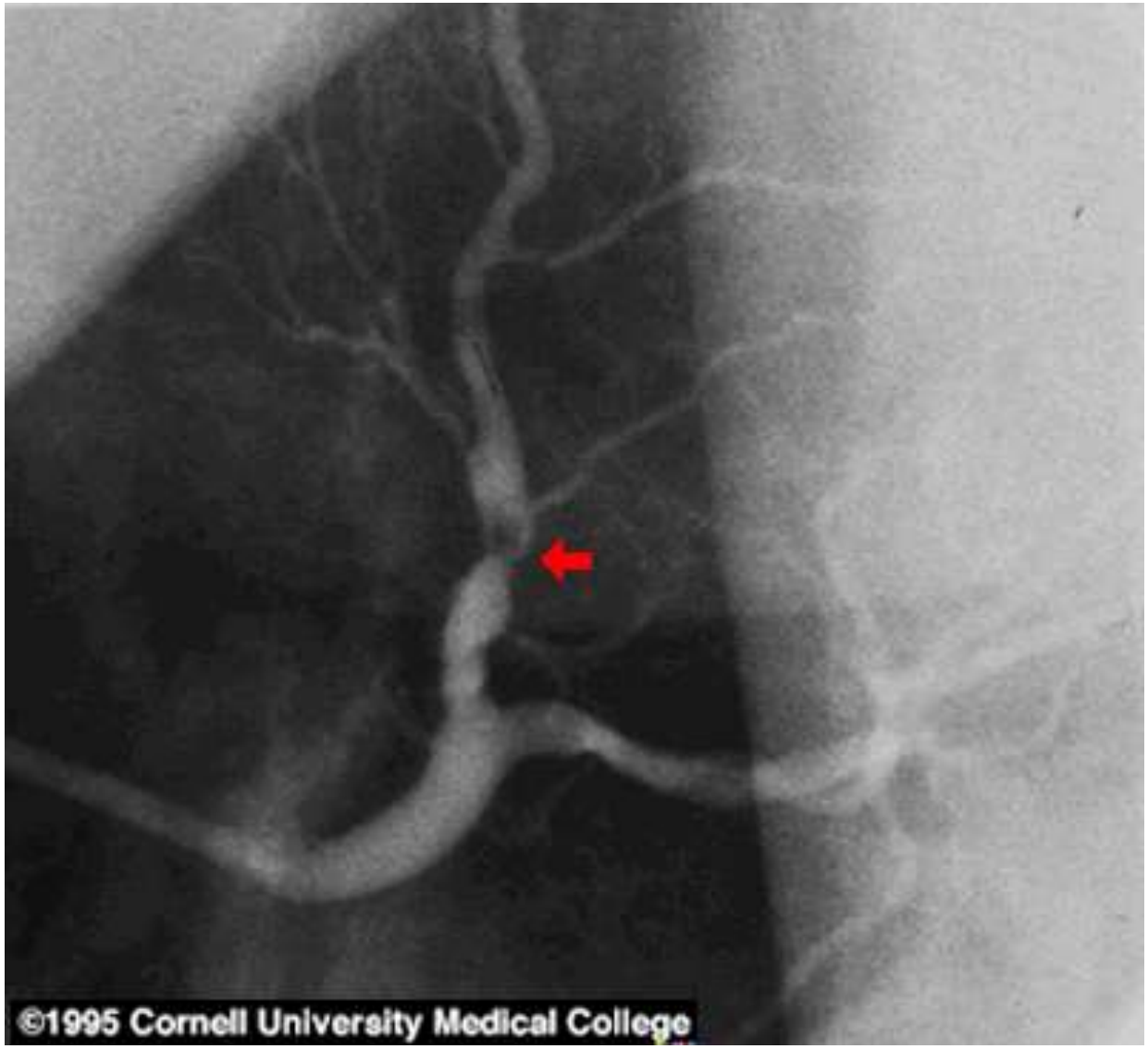



Heart Attack
Blood clot
Artery
Cholesterol plaque
Coronary arteries
Healthy muscle
Dying muscle

Thrombosis of epicardial coronary artery…


…the cause of STEMI


A
Mitral-valve
prosthesis
B
Mitral-valve
prosthesis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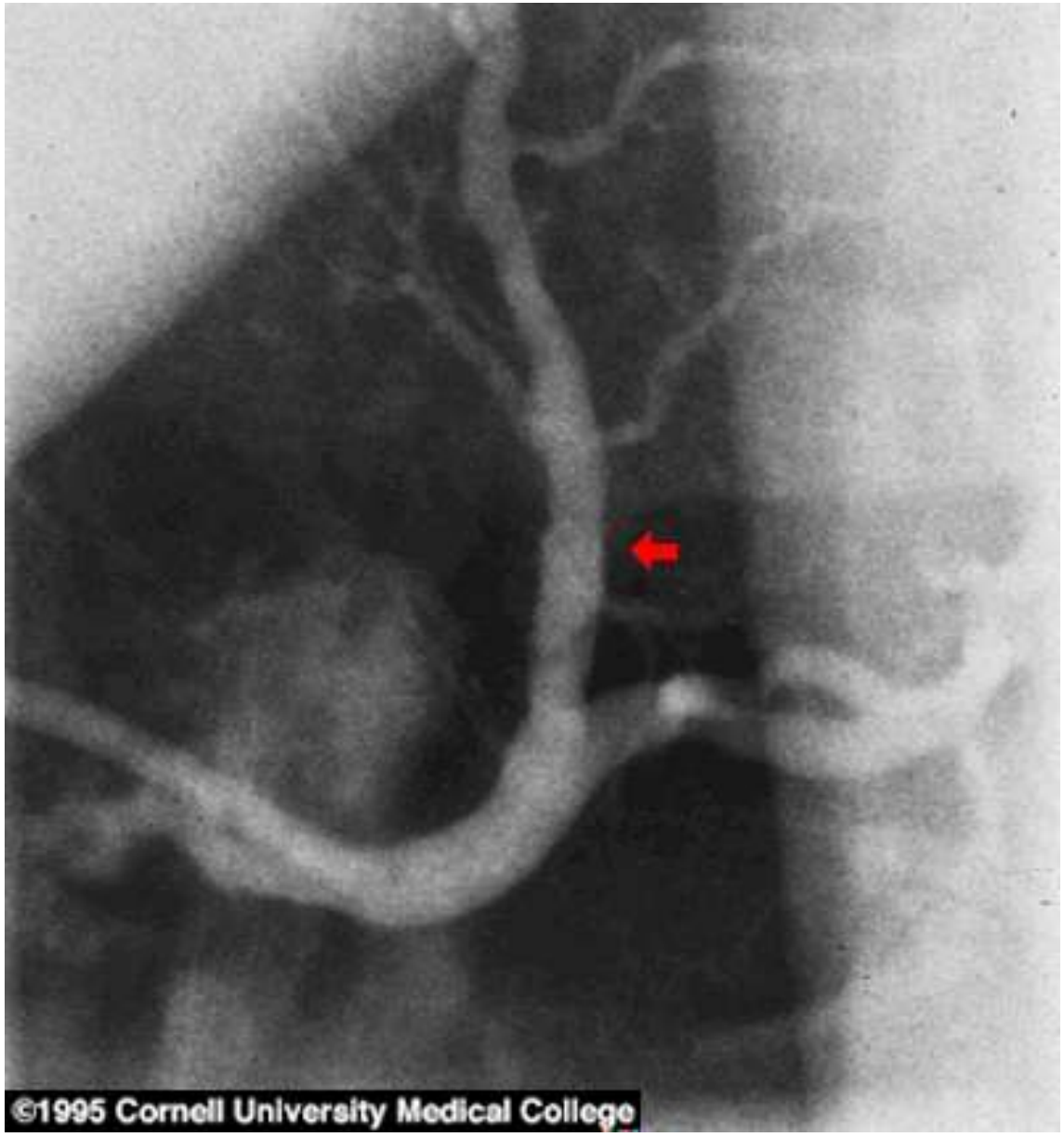
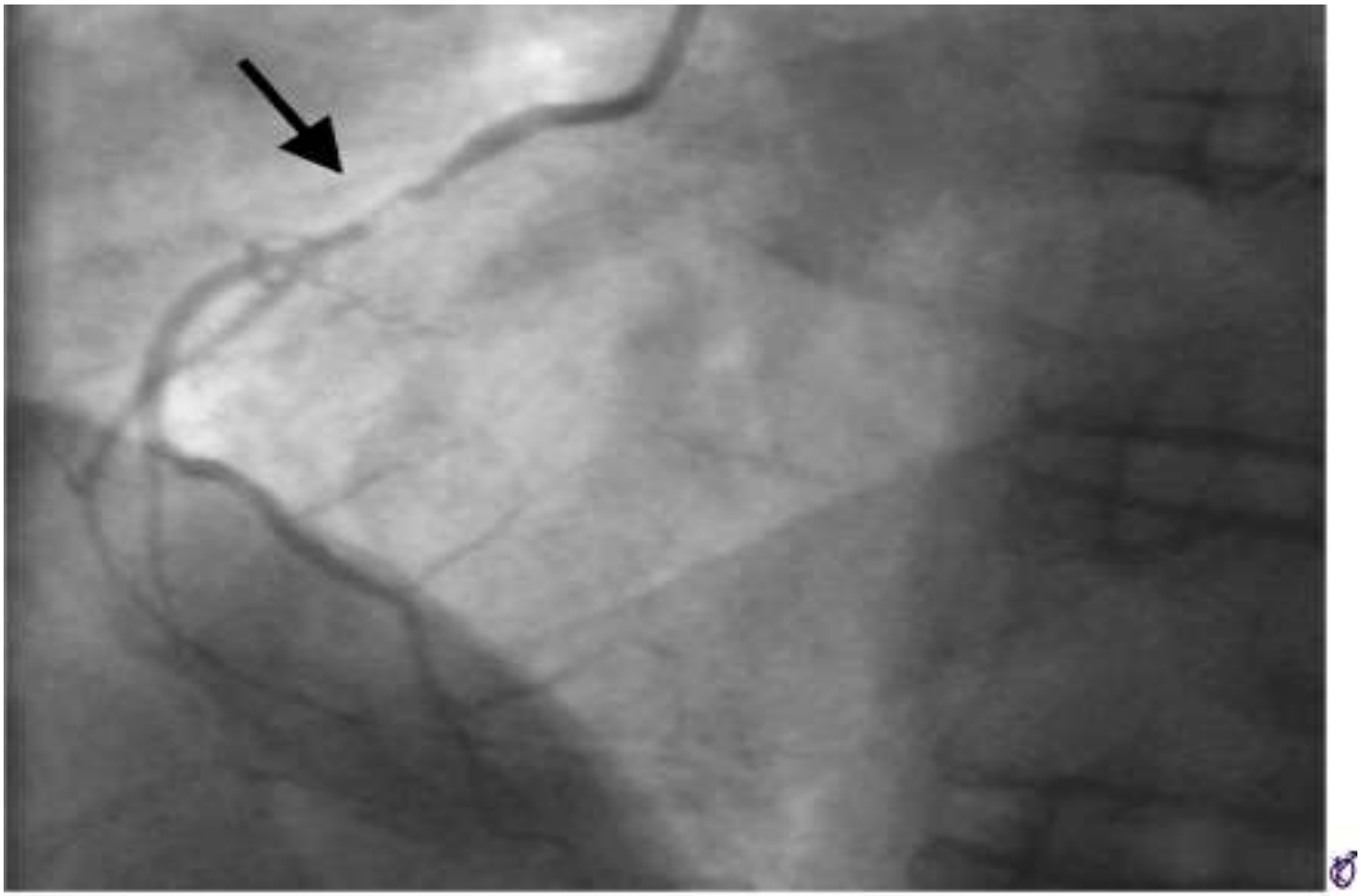
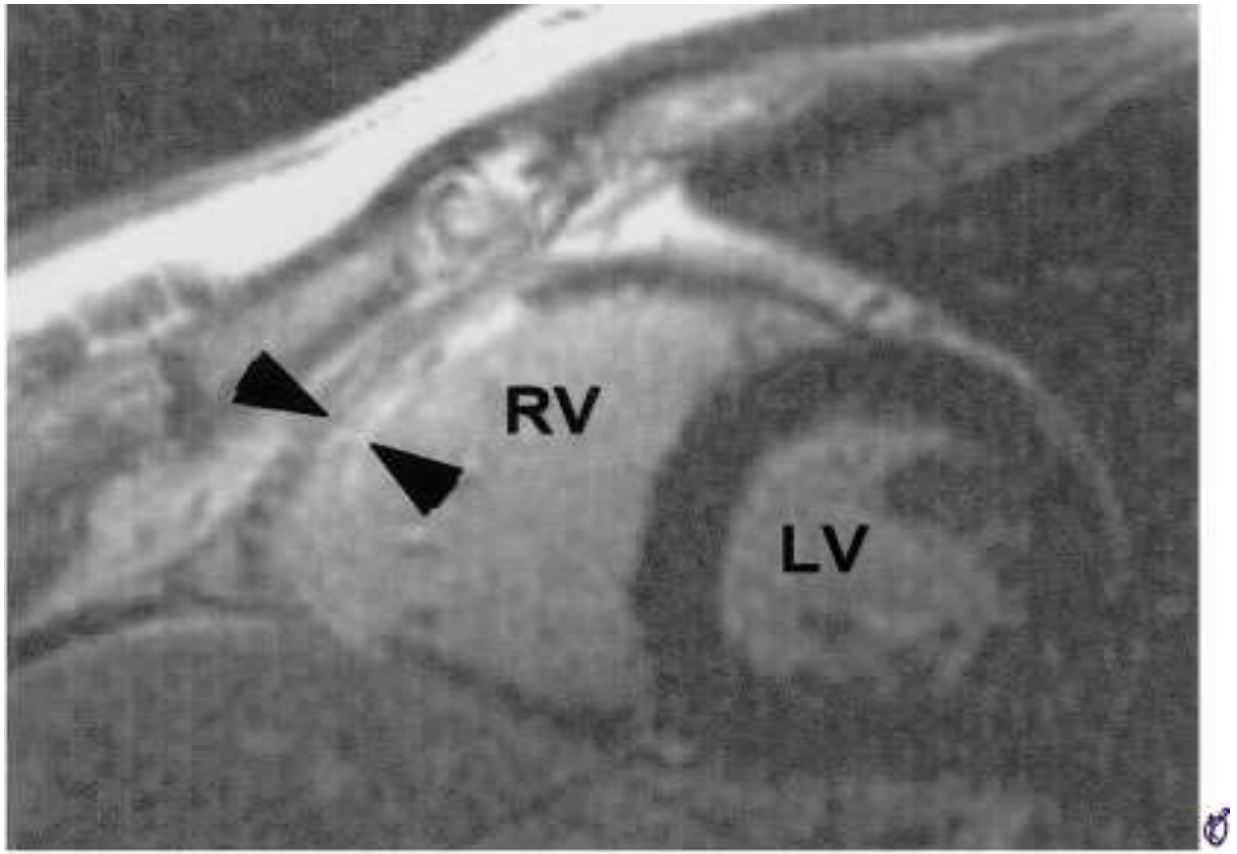

©1995 Cornell University Medical College



B



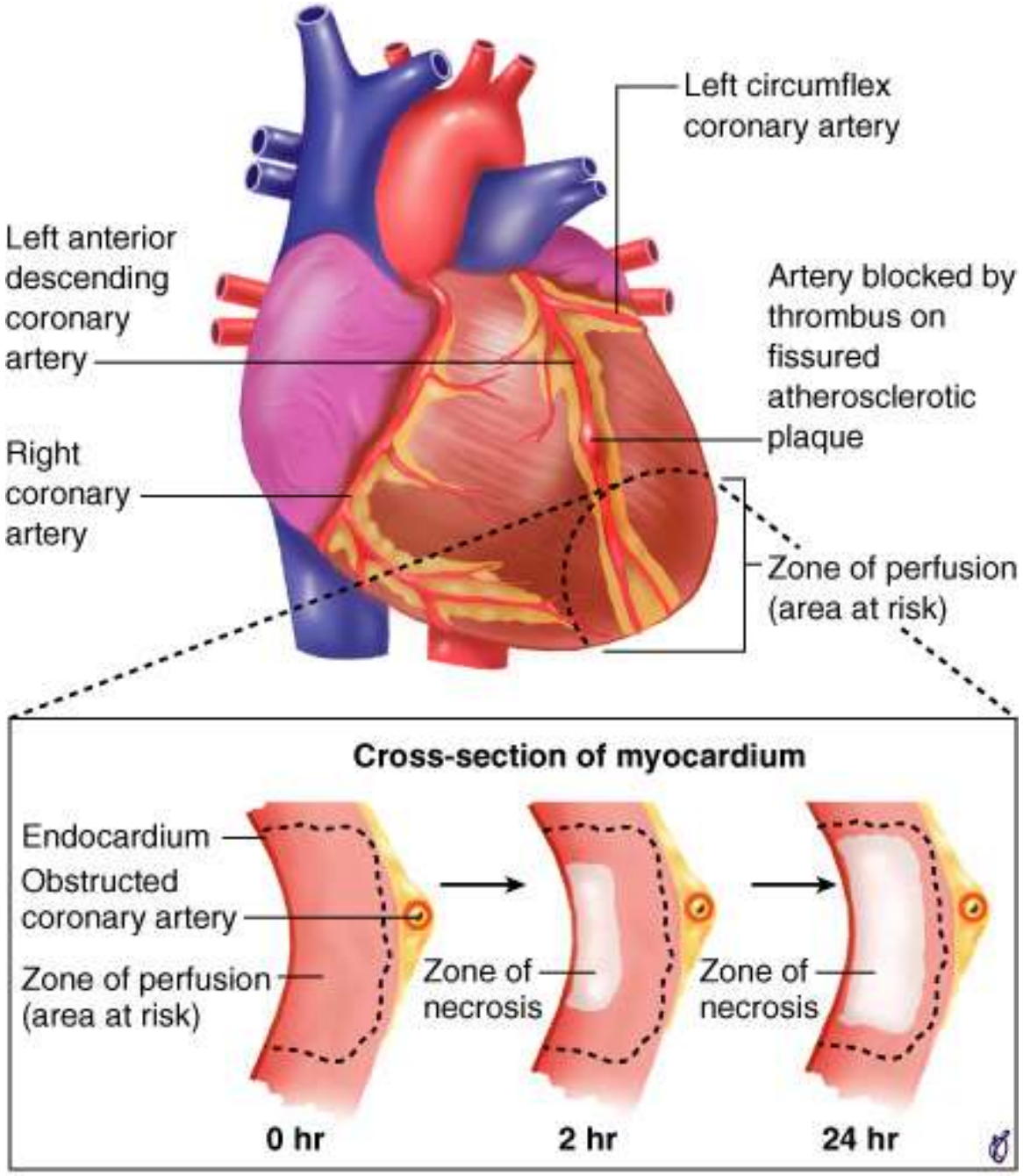
The patient was treated with fibrinolytic therapy and transferred for catheterization. Angiography revealed a tight stenosis of a proximal nondominant right coronary artery (**B,** arrow) without significant disease in the left coronary artery.

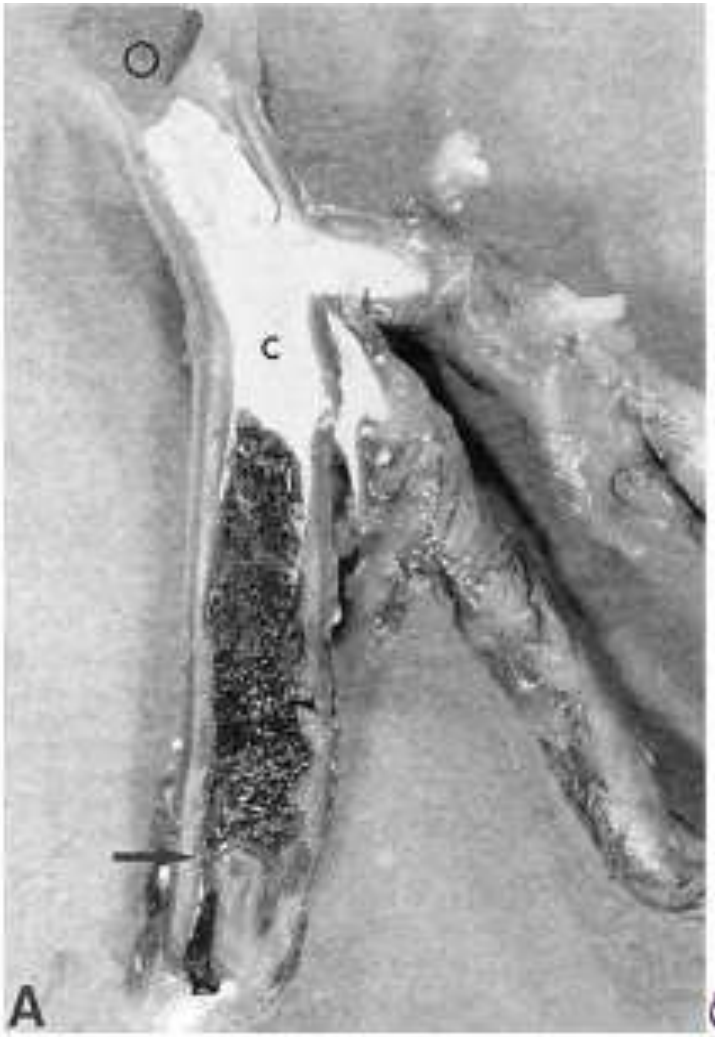



C

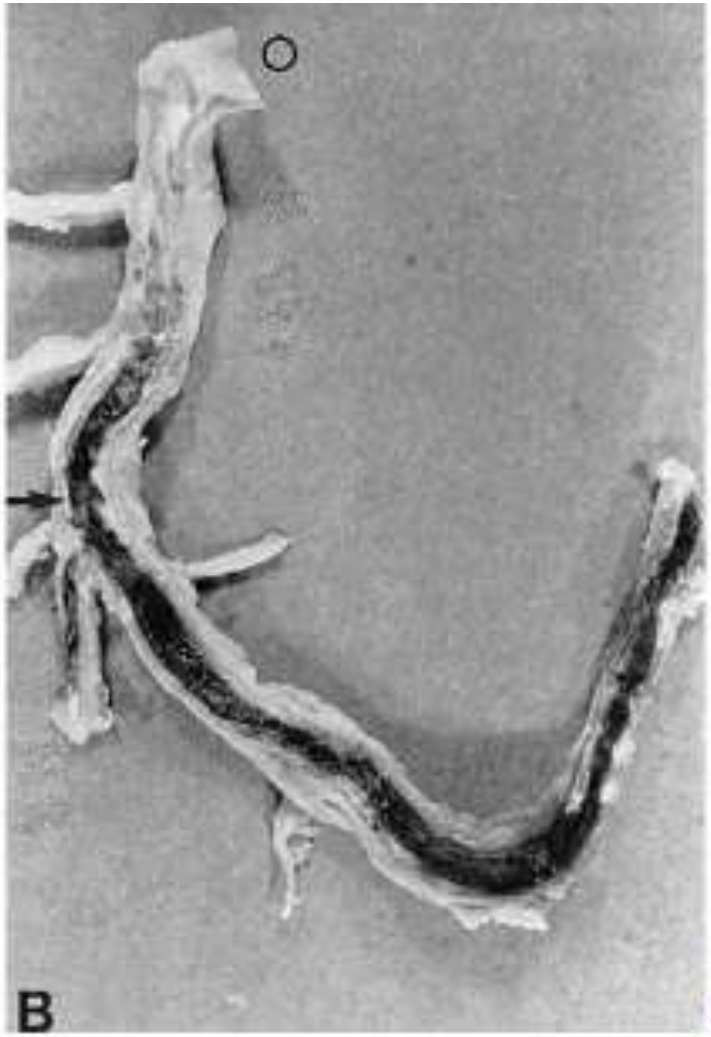


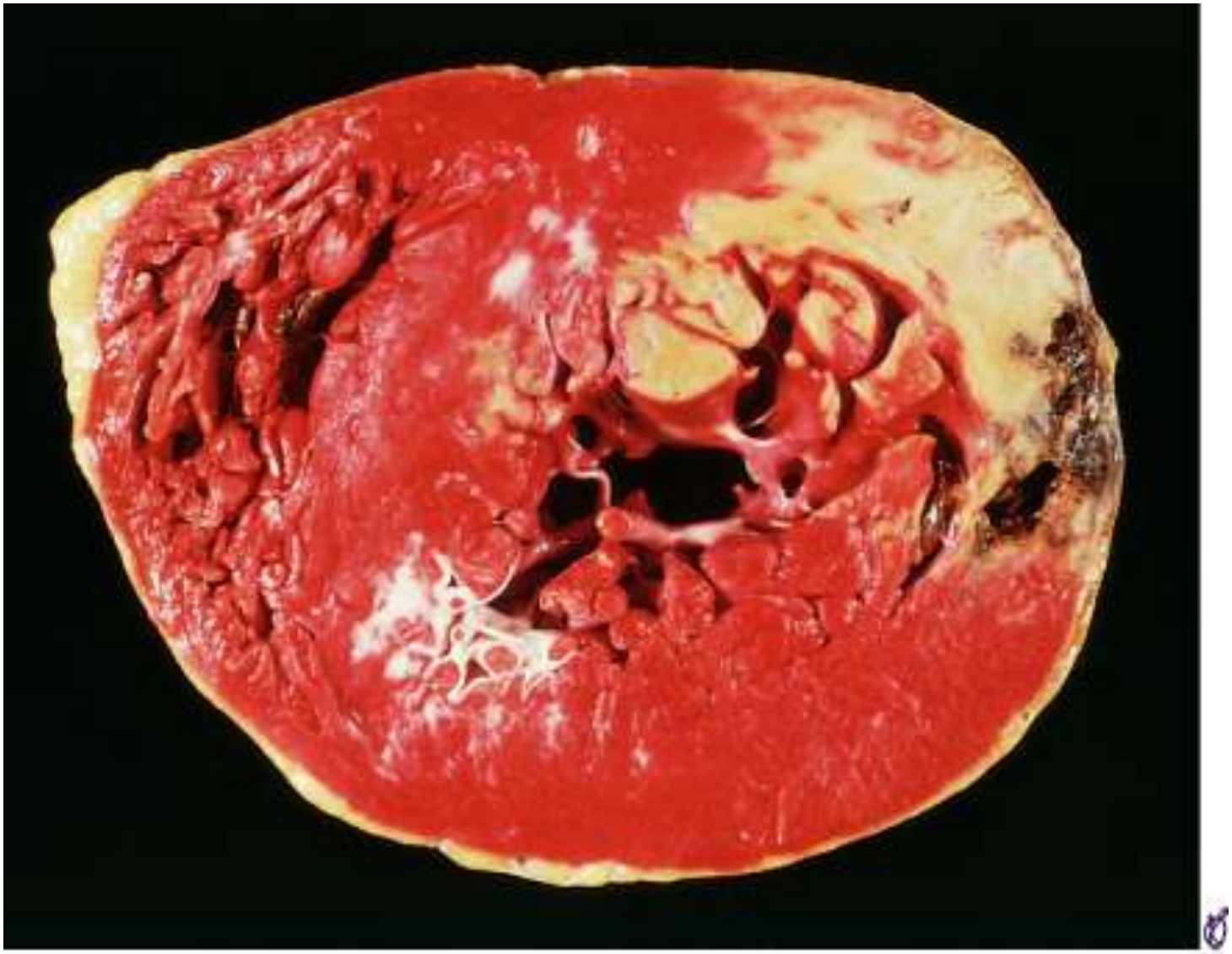
Contrastenhanced cardiac magnetic resonance imaging **(C)** demonstrated delayed hyperenhancement consistent with injury of the right ventricle (RV) with distinct involvement of the right ventricular free wall (arrowhead), sparing the left ventricle (LV) as well as the right ventricular apex. The patient remained hemodynamically stable throughout his hospital course and was discharged home.







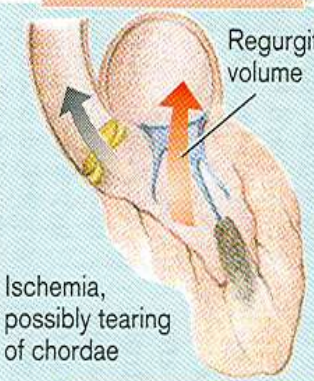


## G. Mechanical Consequences in the Heart after Myocardial Infarction (in Left Ventricle)

Myocardial infarction (left ventricle)

In papillary muscle

1



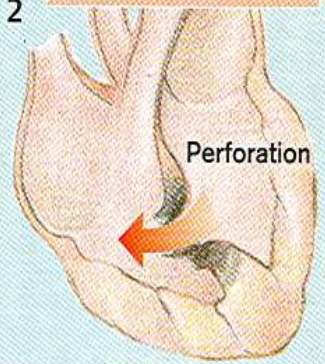

Acute mitral regurgitation

'Forward' stroke volume ↓

Pulmonary congestion

In ventricular septum

2




Left-to-right shunt

Central blood volume ↑

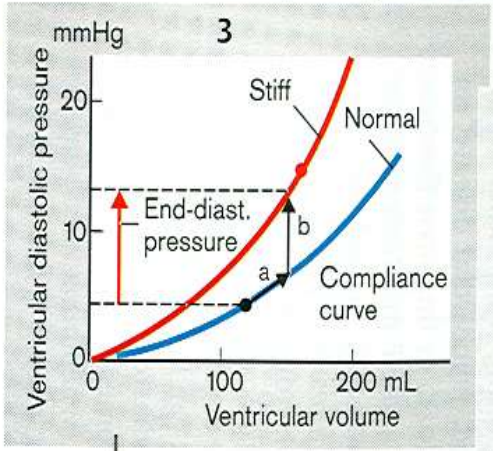
In outer ventricular wall

CO ↓

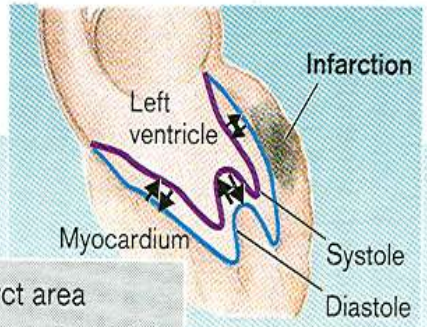
End-diastolic volume ↑

a

b

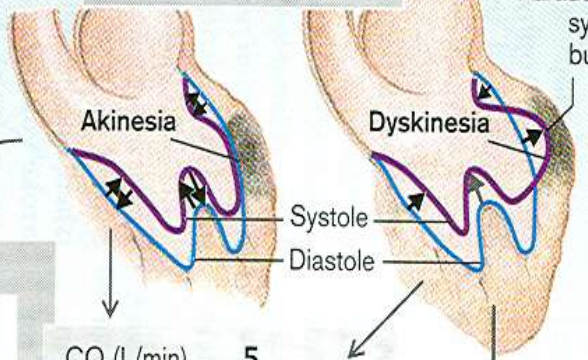


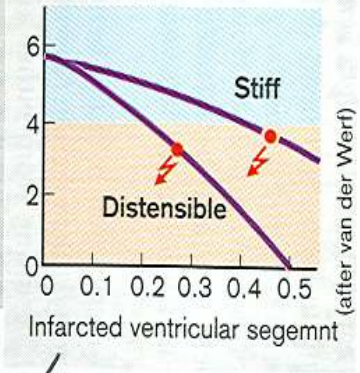

Pulmonary edema

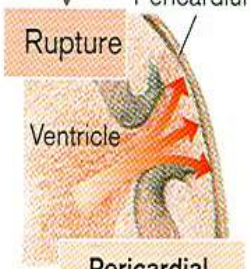



Infarct area

stiff distensible



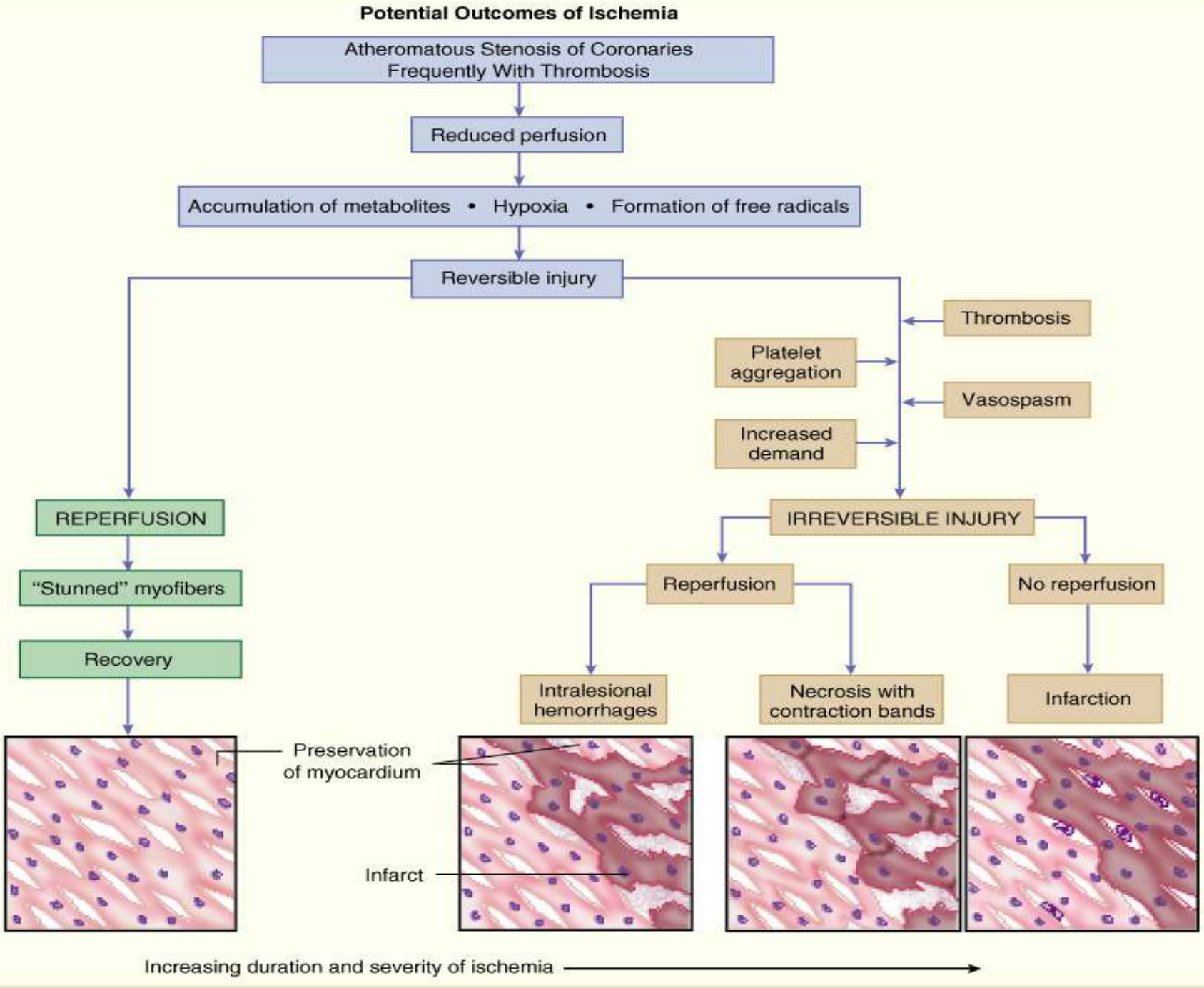




Pericardial tamponade

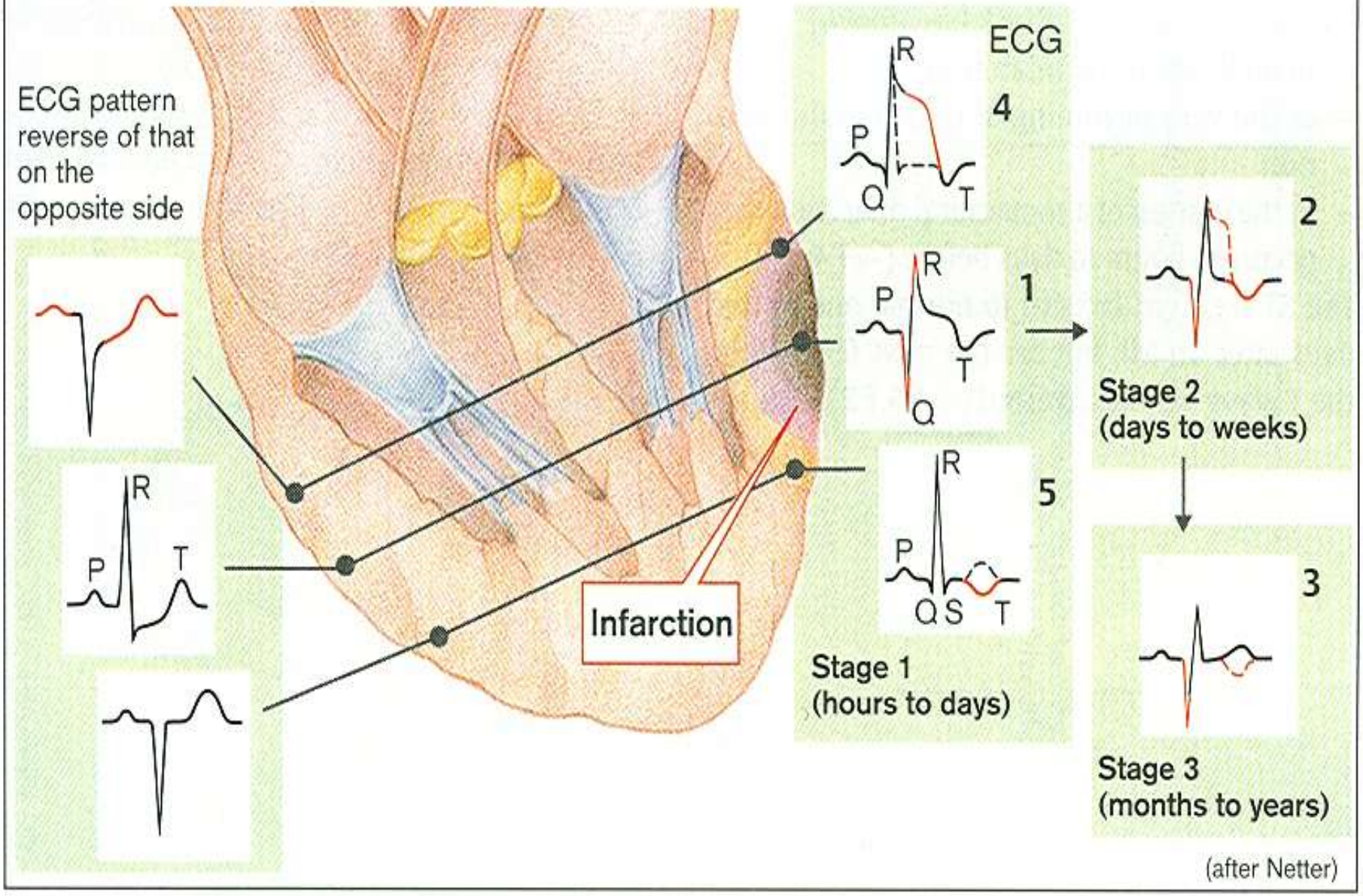
Cardiogenic shock

80 %

Death after infarction

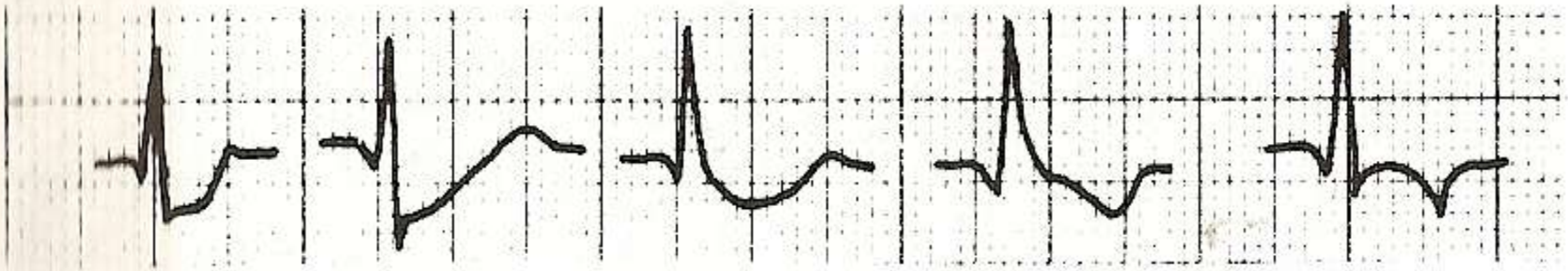


## F. ECG in Coronary Infarction

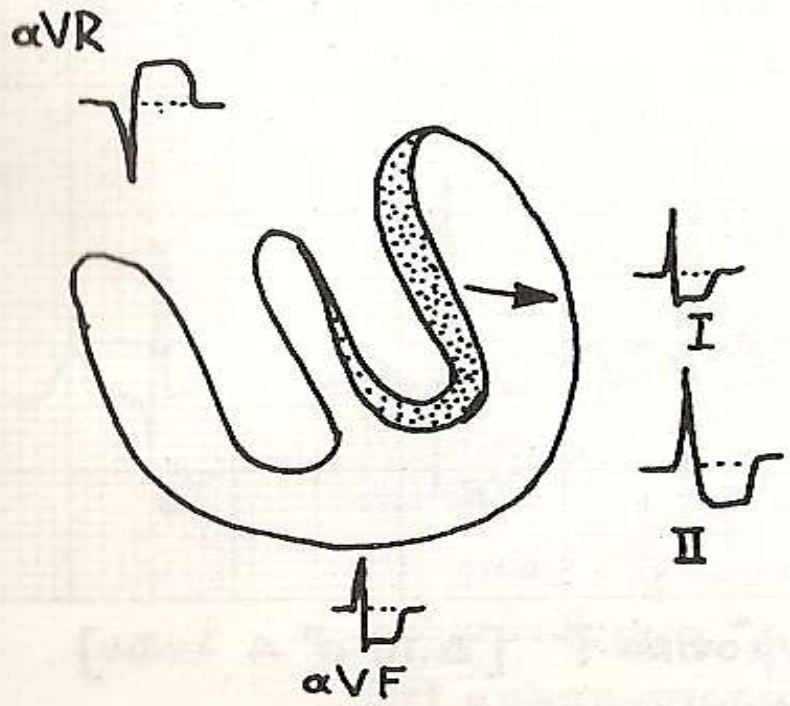


# 1 ΙΣΧΑΙΜΙΑ ΤΟΥ ΜΥΟΚΑΡΔΙΟΥ

Ἡ ἰσχαιμία στό μυοκάρδιο ἐκδηλώνεται μέ μεταβολές στό ST καί Τ καί σπάνια στό U. Οἱ μεταβολές αὐτές μπορεῖ νά ἔχουν ποικίλη μορφολογία, σάν τά ἑπόμενα παραδείγματα :



[Α] Ἡ κατάσπαση τοῦ ST ὀφείλεται στό γεγονός ὅτι συμβαίνει ὑπενδοκάρδιος ἰσχαιμία [μικρά τά ἀγγεῖα - μεγάλη ἡ πίεση - τάση] καί παροδική βλάβη. Ἡ ἀπαγωγή αVR, πού "βλέπει" τό ἰσχαιμικό ἐνδοκάρδιο, θά καταγράφει ἀνάσπαση τοῦ ST, ἐνῶ ὅλες οἱ ἀπέναντι ἀπαγωγές [π.χ. I, II, aVF $V_5$, $V_6$ κλπ] θά καταγράφουν κατάσπαση τοῦ ST.

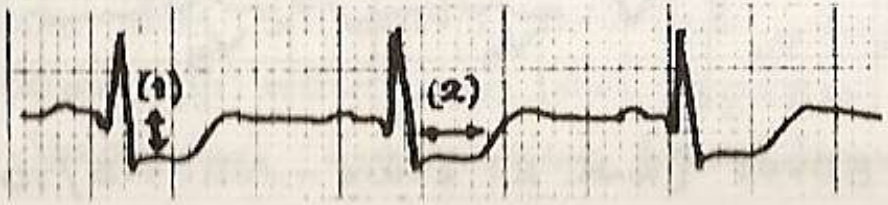



Αὐτή ἡ κατάσπαση τοῦ ST γιά νά εἶναι ἰσχαιμική θά πρέπει νά ἐκπληρώνει τούς ἑξῆς τρεῖς ὅρους:

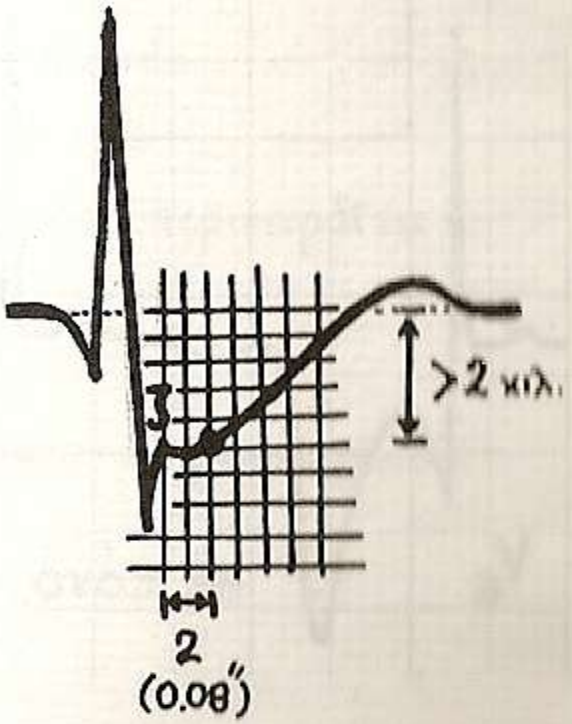
(1) νά εἶναι περισσότερο ἀπό 1 χιλ. (ἢ 2 χιλ.) κάτω τῆς ἰσοηλεκτρικῆς.

(2) νά ἔχει εὖρος [διάρκεια πτώσεως] μεγαλύτερη ἀπό 0.08 sec.

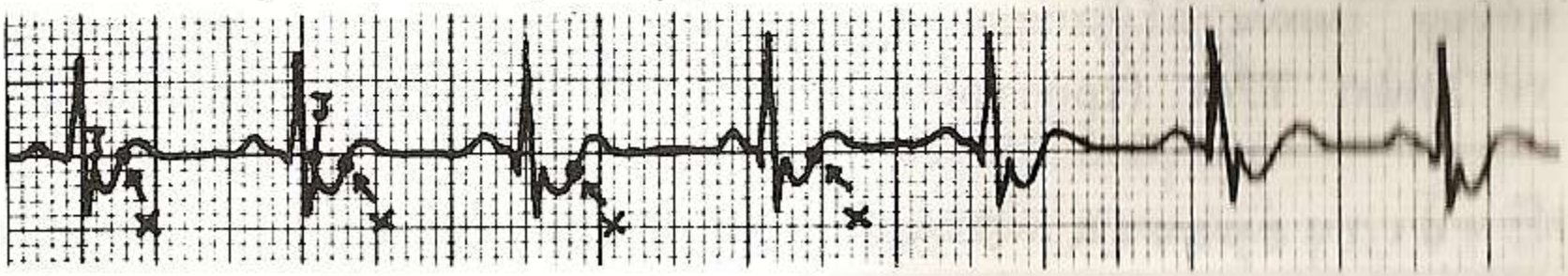
(3) νά παρατηρεῖται σέ 2 ἢ 3 συστολές πού βρίσκονται στήν ἴδια γραμμή [ἐάν ἡ μιά συστολή βρίσκεται χαμηλά ἢ ψηλά ἀλλοιώνει τό ST).



[Β] Η μορφολογία αὐτή εἶναι ἰσχαιμική, ὅταν ἰσχύει ὁ μνημοτεχνικός κανόνας: "2 x 2", ὅταν δηλαδή τό σημεῖο τοῦ ST πού βρίσκεται 2 τεταρτημόρια (0.08") μετά τό J (πού σημαίνει τό τέλος τῆς κοιλιακῆς ἐκπολώσεως), εἶναι ταυτόχρονα καί περισσότερο ἀπό 2 χιλ. κάτω τῆς ἰσοηλεκτρικῆς γραμμῆς.

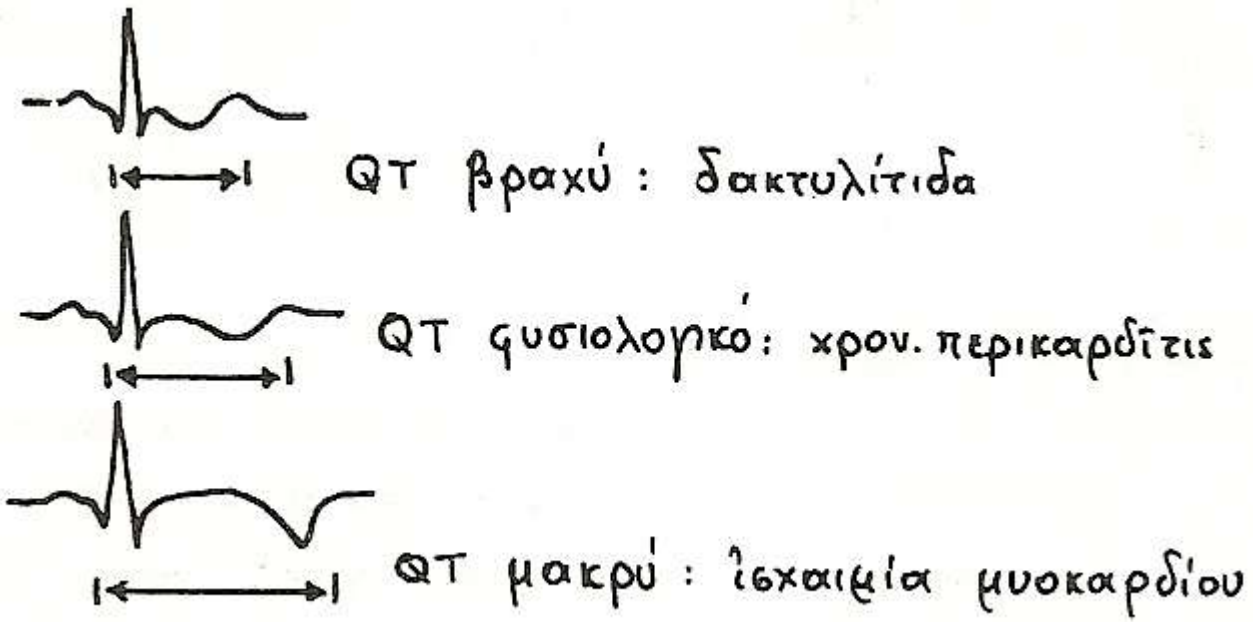



Στό παρακάτω παράδειγμα δέν ὑπάρχει μορφολογία ἰσχαιμίας,

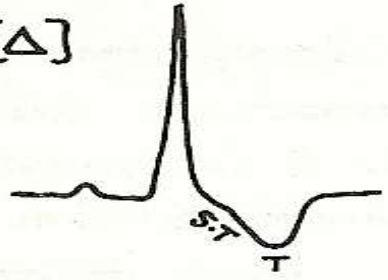



ἀφοῦ τό σημεῖο x, πού βρίσκεται 2 τεταρτημόρια (0.08") μετά τό J, δέν εἶναι 2 χιλ. κάτω τῆς ἰσοηλεκτρικῆς γραμμῆς.

[Γ] Αὐτή ἡ κυπελοειδής διαμόρφωση τοῦ ST παρατηρεῖται κυρίως στή λήψη δακτυλίτιδας. Μπορεῖ ὅμως νά εἶναι καί ἰσχαιμική. Ὑπάρχει ἕνα σημεῖο πού μπορεῖ νά μᾶς βοηθήσει στή διαφορική διάγνωση: Εἶναι τό QT διάστημα πού στή δακτυλίτιδα εἶναι βραχύ, ἐνῶ στήν ἰσχαιμία εἶναι παρατεταμένο.

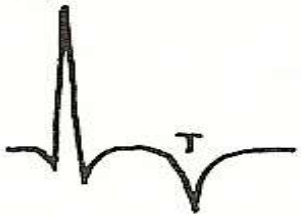


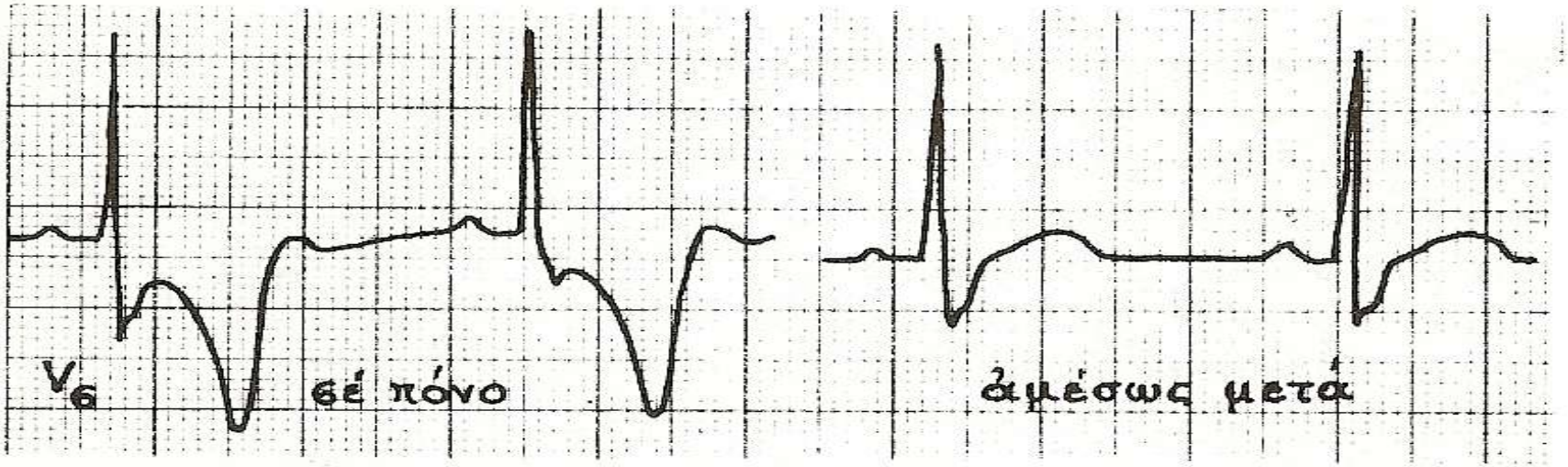
[Δ]




Ἡ μορφολογία αὐτή παρατηρεῖται σέ ὑπερτροφίες (strain), ἀποκλεισμούς σκελῶν κλπ., ἀλλά μπορεῖ νά εἶναι καί ἰσχαιμική.

[Ε] Τά ὀξύαιχμα καί συμμετρικά Τ (ἀρνητικά ἤ καί θετικά) μπορεῖ νά εἶναι ἰσχαιμικά· ἐάν ὅμως παραμείνουν γιά μέρες καί αὐξηθοῦν τά ἔνζυμα (CPK, SGOT, LDH), ὑποδηλώνουν ὑπενδοκάρδιο ἔμφραγμα.




Παράδειγμα ἰσχαιμίας τῶν στεφανιαίων σέ ἀσθενῆ πού εἶχε καί R.BBB (δηλ. S στή $V_6$). Κατά τή διάρκεια τοῦ πόνου ἔκανε ὀξύαιχμα, συμμετρικά, ἀρνητικά Τ καί μετά τή λήψη TNT (τρινιτρίνης) ἐπανῆλθε στό φυσιολογικό.

## (6) Η ΑΞΙΑ ΤΟΥ ΗΚΓ ΣΤΗΝ ΙΣΧΑΙΜΙΑ :

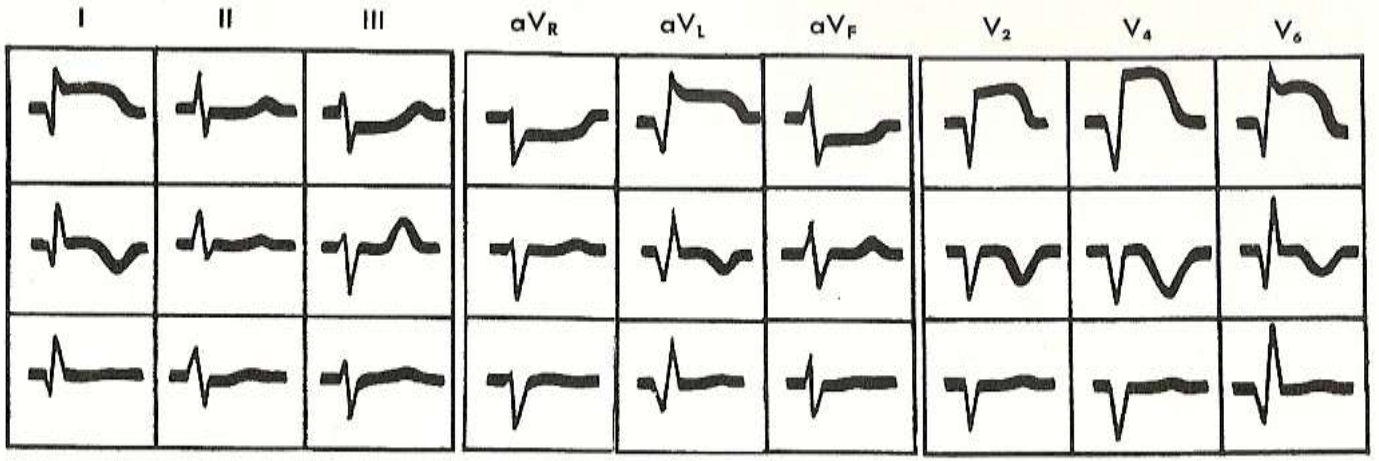
- Τό ΗΚΓ στήν πρεμία είναι ἀποκαλυπτικό ἰσχαιμίας τοῦ μυοκαρδίου μόνο στά 50-60% τῶν ἰσχαιμικῶν ἀρρώστων. Τό ΗΚΓ στήν κόπωση εἶναι ἀποκαλυπτικό στά 80-85%. Τό ὑπόλοιπο 15-20% ἀποκαλύπτεται μόνο μέ στεφανιαιογραφία.

- Ποτέ δέν μιλᾶμε στόν ἄρρωστο γιά ἰσχαιμία μόνο μέ τά ΗΚΓ εὑρήματα, χωρίς συνύπαρξη στηθάγχης.

- Γενικά μποροῦμε νά κάνουμε τήν πιό κάτω κατάταξη, ἀνάλογα μέ τά ΗΚΓραφικά εὑρήματα καί τήν κλινική εἰκόνα :

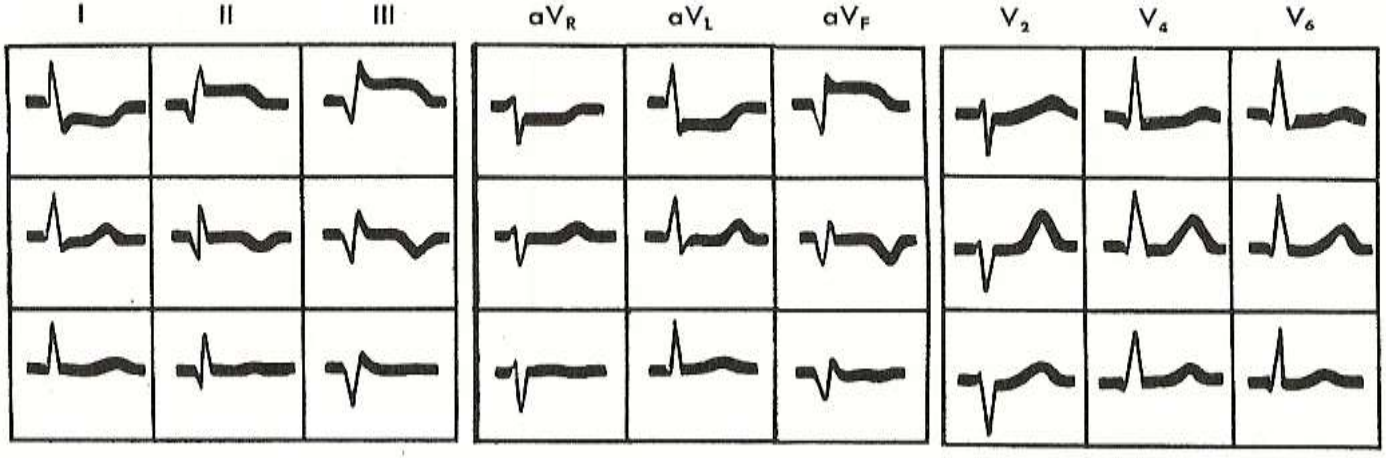
| ΗΚΓ | ΣΤΗΘΑΓΧΗ | ΙΣΧΑΙΜΙΚΗ ΚΑΡΔΙΟΠΑΘΕΙΑ |
|---|---|---|
| [+] | [−] | ἀπίθανη |
| [−] | [+] | πιθανή |
| [+] | [+] | βεβαία |

## ΣΕΙΡΑ ΤΩΝ ΗΚΓ ΑΛΛΟΙΩΣΕΩΝ ΣΤΟ ΕΜΦΡΑΓΜΑ ΤΟΥ ΠΡΟΣΘΙΟΥ ΤΟΙΧΩΜΑΤΟΣ

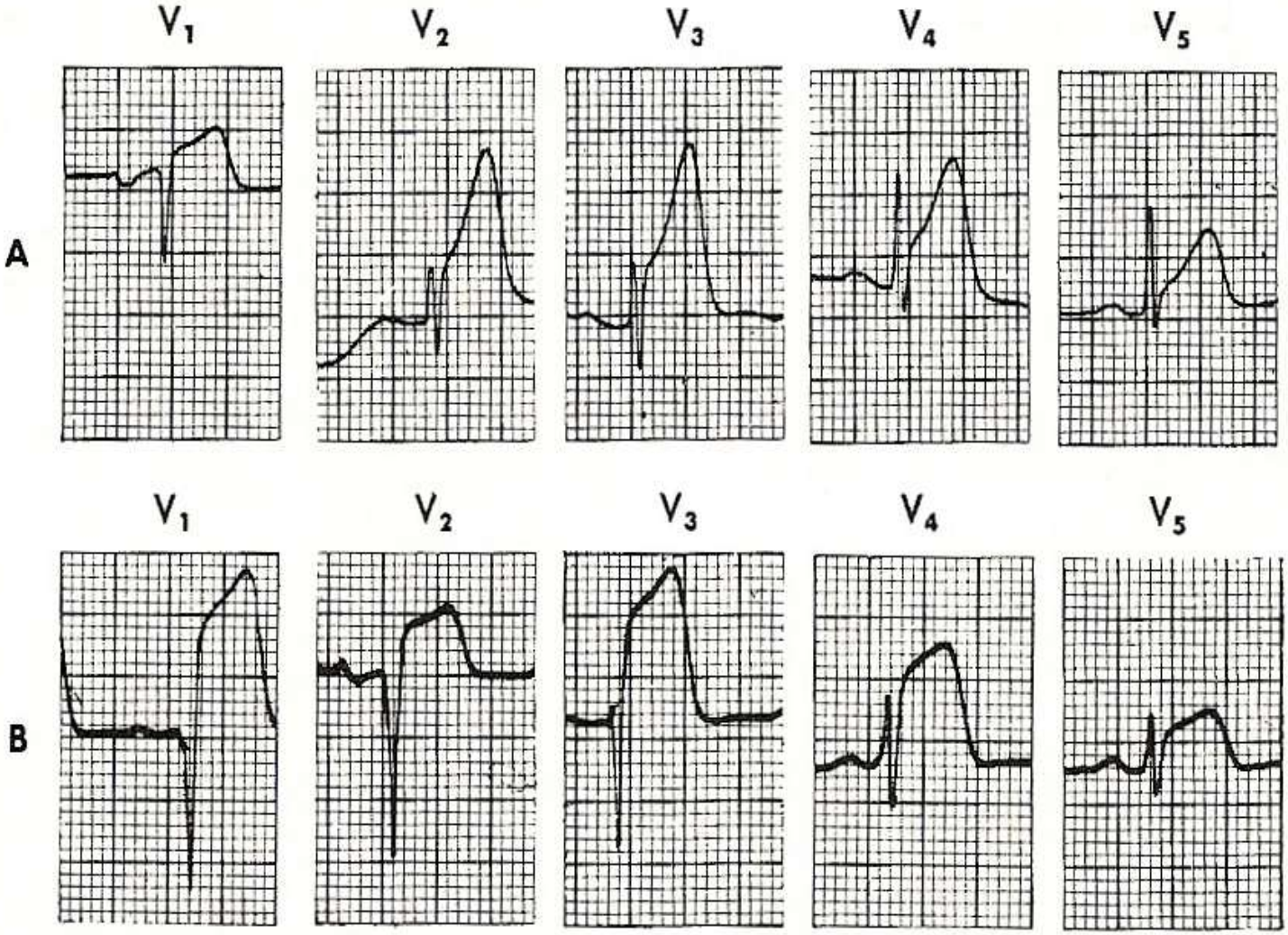



**Εικ. 8-4.** Διαδοχικές ἀλλοιώσεις τοῦ QRS καί τοῦ ST-T στό ἔμφραγμα τοῦ προσθίου τοιχώματος. Παρατηροῦμε τίς ἀντικατοπτρικές ἀλλοιώσεις τοῦ ST-T στίς κατώτερες ἀπαγωγές (II, III καί aVF).

## ΣΕΙΡΑ ΤΩΝ ΗΚΓ ΑΛΛΟΙΩΣΕΩΝ ΣΤΟ ΕΜΦΡΑΓΜΑ ΤΟΥ ΚΑΤΩΤΕΡΟΥ ΤΟΙΧΩΜΑΤΟΣ

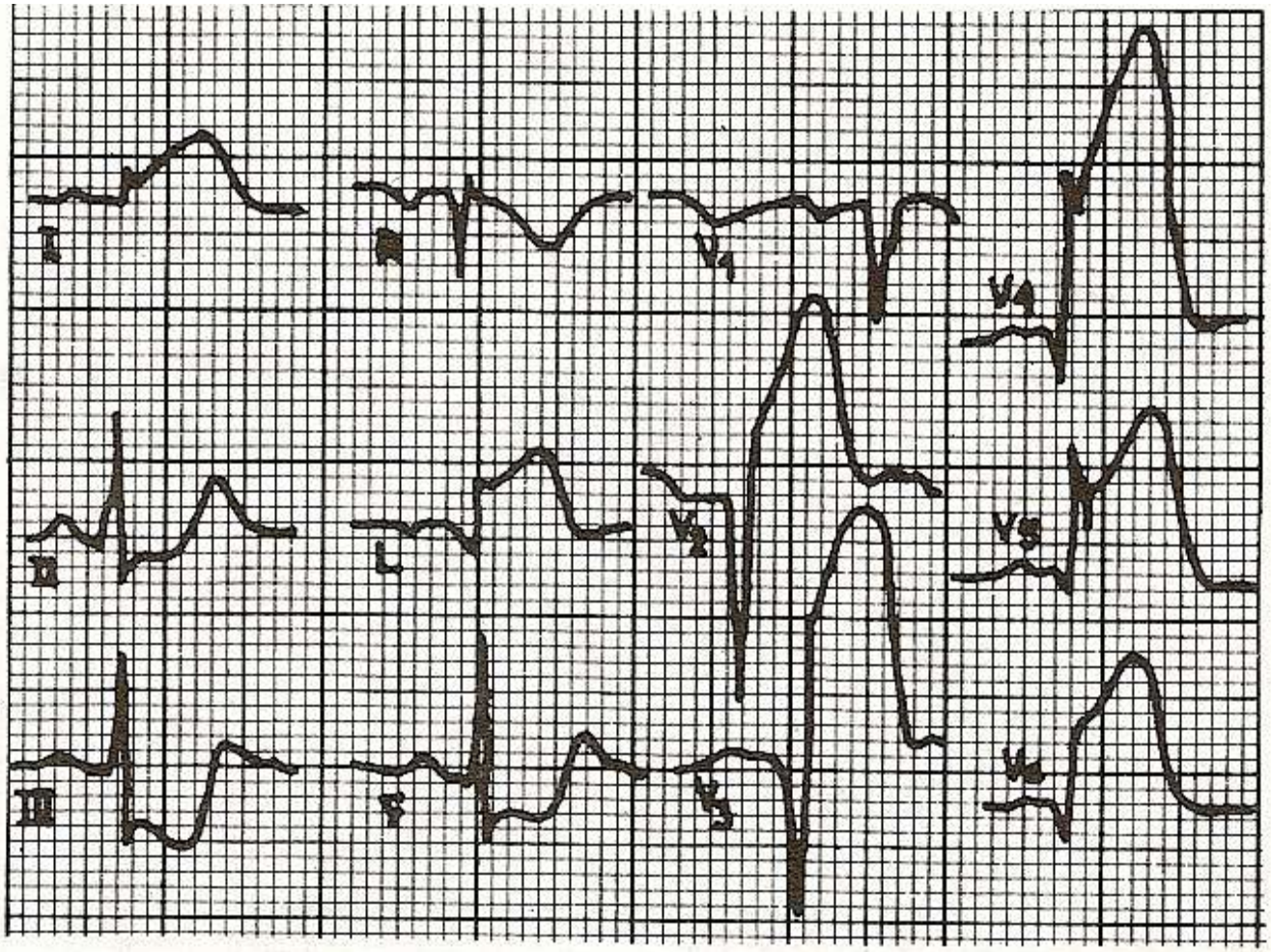


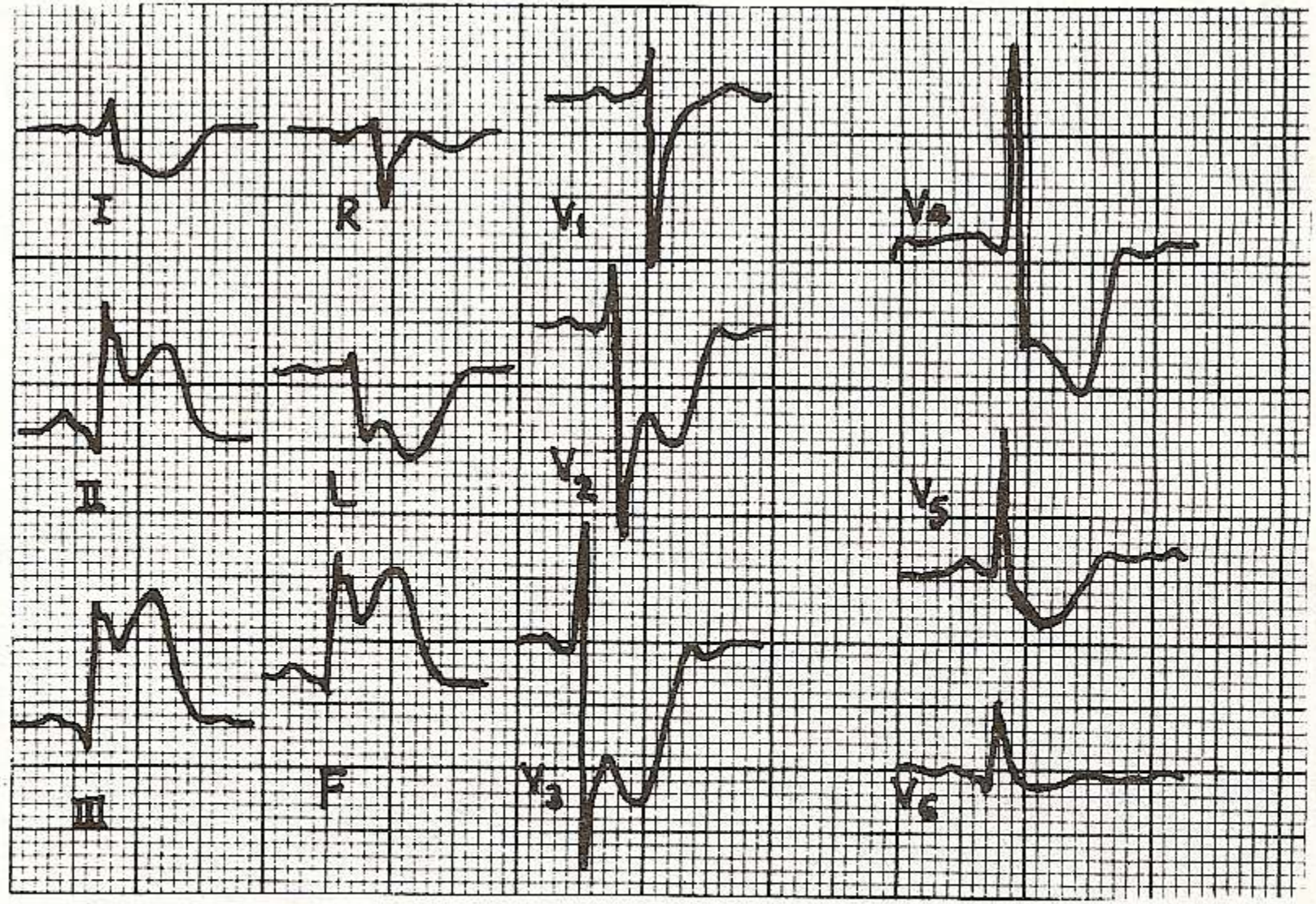

**Εικ. 8-5.** Διαδοχικές ἀλλοιώσεις τοῦ QRS καί τοῦ ST-T στό ἔμφραγμα τοῦ κατωτέρου τοιχώματος. Παρατηροῦμε τίς ἀντικατοπτρικές ἀλλοιώσεις τοῦ ST-T στίς πρόσθιες ἀπαγωγές.



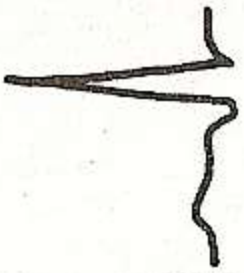
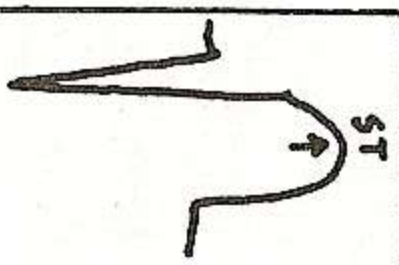
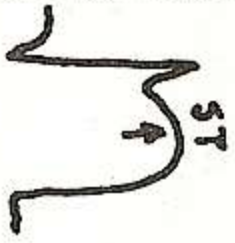
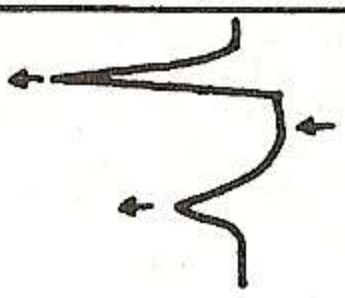
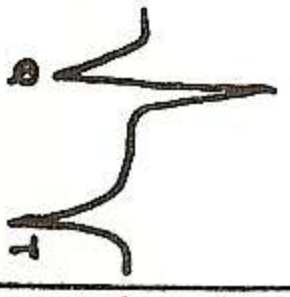

**Εικ. 8-7.** Προκάρδιες ἀπαγωγές ἀρρώστου μέ ὀξύ ἔμφραγμα τοῦ προσθίου τοιχώματος. Σημειῶστε τά ψηλά, θετικά (ὑπεροξέα) Τ, πού παρατηροῦνται στίς ἀπαγωγές $V_2$ ὡς $V_5$ στήν πρωιμώτερη φάση τοῦ ἐμφράγματος, **Α.** Ἡ καταγραφή ΗΚΓ, μερικές ὧρες ἀργότερα, **Β,** ἐμφανίζει ἔκδηλη ἀνύψωση τοῦ τμήματος S-T σέ μερικές ἀπαγωγές (εἰκόνα ρεύματος βλάβης) μέ παθολογικά ἐπάρματα Q στίς $V_1$ καί $V_2$.

Οξύ πρόσθιο
έκτεταμένο
έμφραγμα.
[Στις απαγω-
γές II, III, aVF
υπάρχει εικό-
να κατόπτρου].



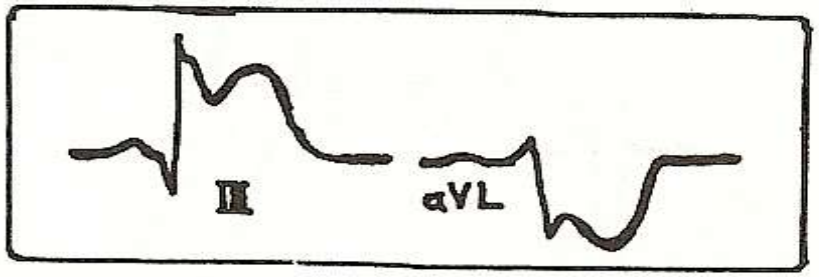



Ὀξύ ἔμφραγμα τοῦ κατωτέρου τοιχώματος [ἀνάσπαση ST στίς II, III καί αVF], μέ εἰκόνα κατόπτρου [κατάσπαση ST στίς I, αVL, $V_2$, $V_3$, $V_4$ καί $V_5$].

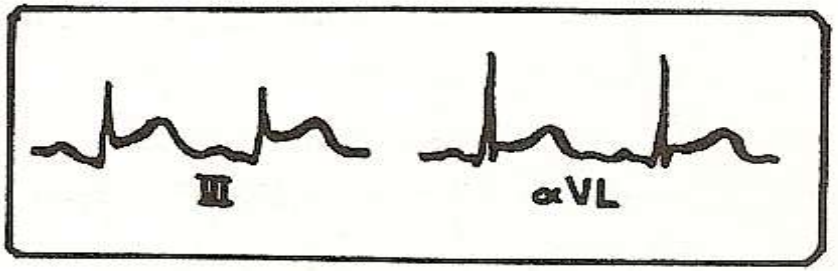
| Δεξιές (π.χ. $V_4$) | Αριστερές (π.χ. I) | ΑΠΑΓΩΓΕΣ |
| --- | --- | --- |
|  |  | ΦΥΣΙΟΛΟΓΙΚΟ ΗΚΓ |
|   |   | ΥΠΕΡΟΞΥ ΕΜΦΡΑΓΜΑ [λίγων ὡρῶν] |
|  |  | ΟΞΥ ΕΜΦΡΑΓΜΑ [πολλῶν ὡρῶν] |
|   |   | ΥΠΟΞΥ ΕΜΦΡΑΓΜΑ [ἡμερῶν ἢ ἑβδομάδων] |
|   |   | ΧΡΟΝΙΟ [ἡμνῶν ἢ ἐτῶν] |

## [VI] ΠΟΥ ΠΑΡΑΤΗΡΕΙΤΑΙ ΑΝΑΣΠΑΣΗ ΤΟΥ ST

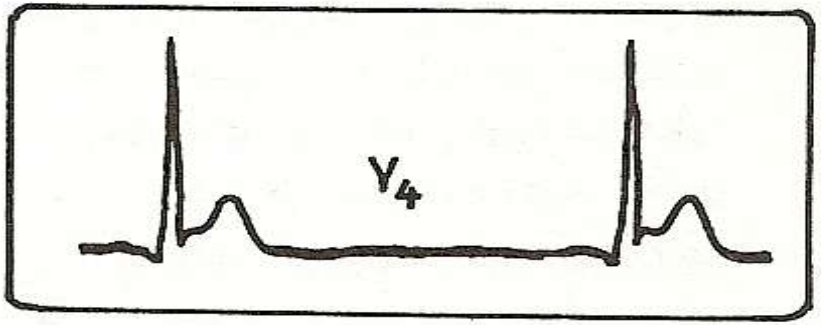
[α] Στό *ἔμφραγμα*
μέ τό κυρτό ἄνω
καί "ἐλιόνα κατόπτρου„.




[β] Στήν *περικαρδίτιδα*
μέ τό κοίλο ἄνω
καί χωρίς "ἐλιόνα κατόπτρου„.




[γ] Στήν *παρασυμπαθητικοτονία*
ὅπου γίνεται πρώϊμη
ἐπαναπόλωση.

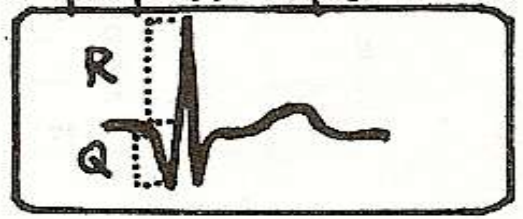



[δ] Στούς *νέγρους*

[β] Παθολογικά

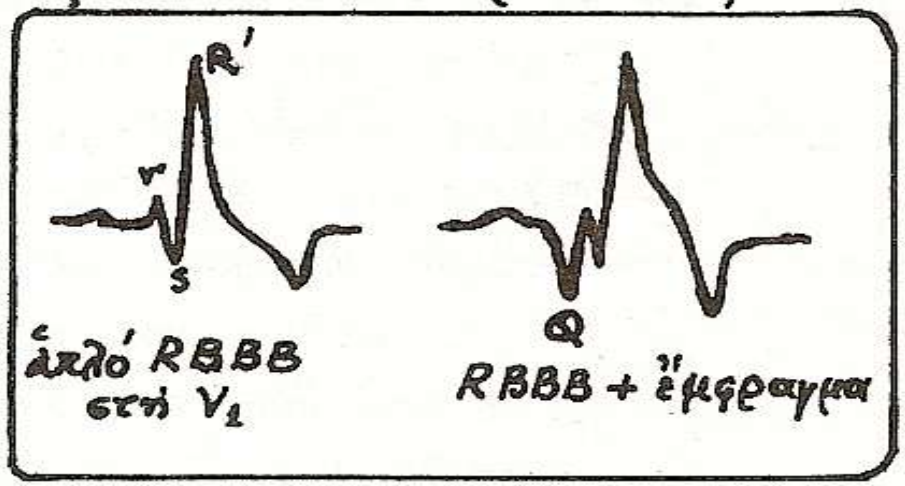
Τά Q εἶναι παθολογικά στίς ἑξῆς περιπτώσεις:

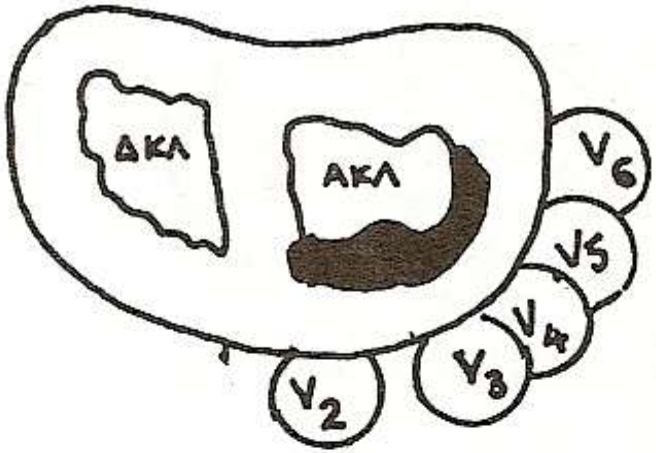
① ἐάν ἔχουν εὖρος $\geqslant$ 0,04 sec καί βάθος $\geqslant$ 4 mm ἤ ἐάν τά Q ἔχουν βάθος ἴσο ἤ μεγαλύτερο ἀπό τό 1/4 τοῦ ὕψους τοῦ R, δηλ. ἐάν ἡ σχέση Q πρός R εἶναι 1 πρός 4 $\left[\frac{Q}{R} = \frac{1}{4}\right]$.

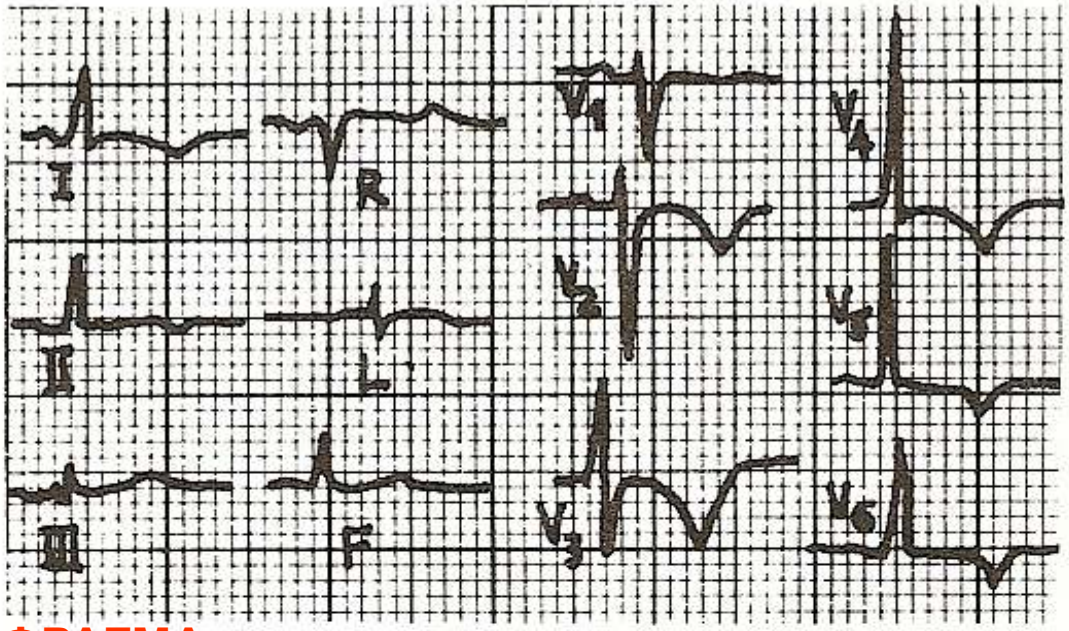



② στόν ἀποκλεισμό τοῦ δεξιοῦ σκέλους (R.B.B.B.) τά Q πάντα ἀξιολογοῦνται καί ἡ παρουσία τους σημαίνει ἔμφραγμα τοῦ μυοκαρδίου.

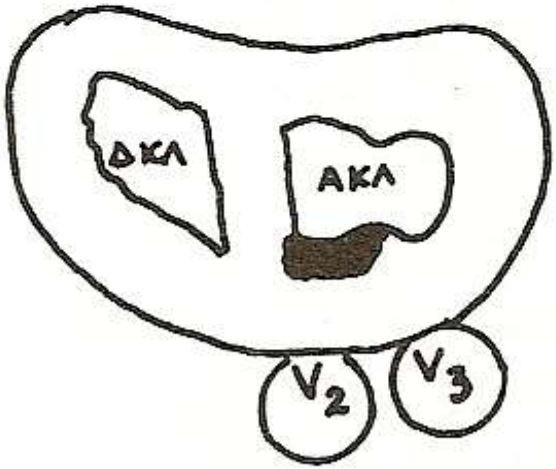
③ στόν ἀποκλεισμό τοῦ ἀριστεροῦ σκέλους (L.B.B.B.) τά Q δέν ἀξιολογοῦνται, παρά μόνον στίς ἀπαγωγές $V_5$ καί $V_6$, ὅπου σημαίνουν ἔμφραγμα τοῦ μυοκαρδίου. [Αὐτό τό σημεῖο ἀναπτύσσεται περισσότερο στό κεφάλαιο τῶν σκελικῶν ἀποκλεισμῶν].

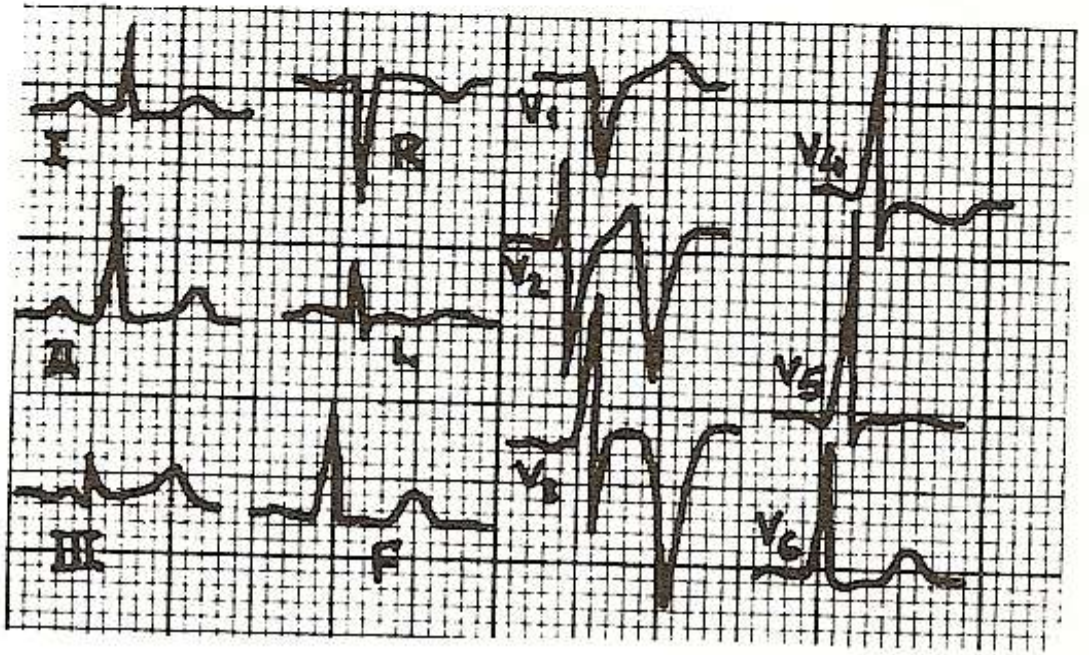


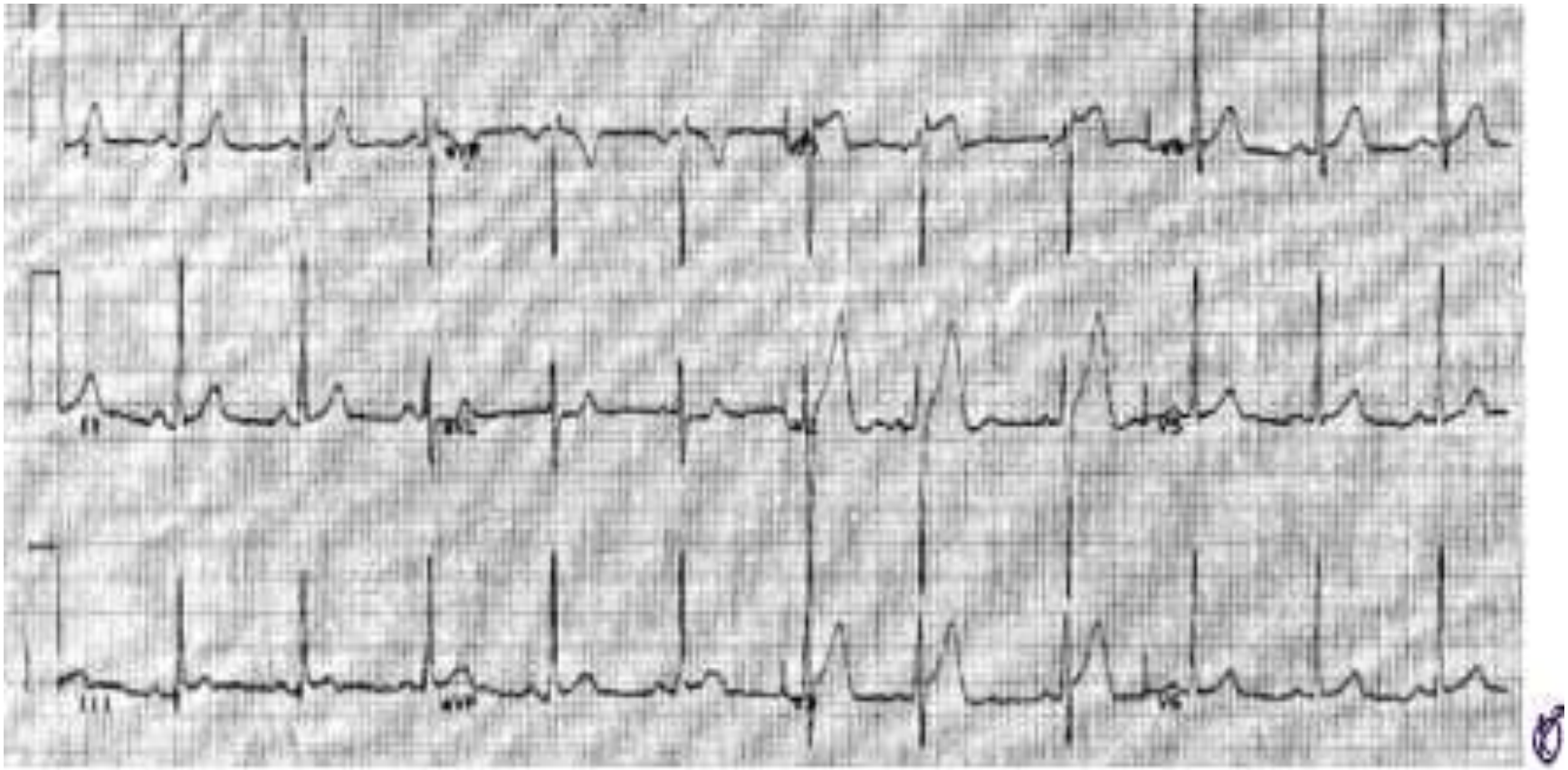



## ΥΠΕΝΔΟΚΑΡΔΙΟ (NSTEMI) ΕΜΦΡΑΓΜΑ



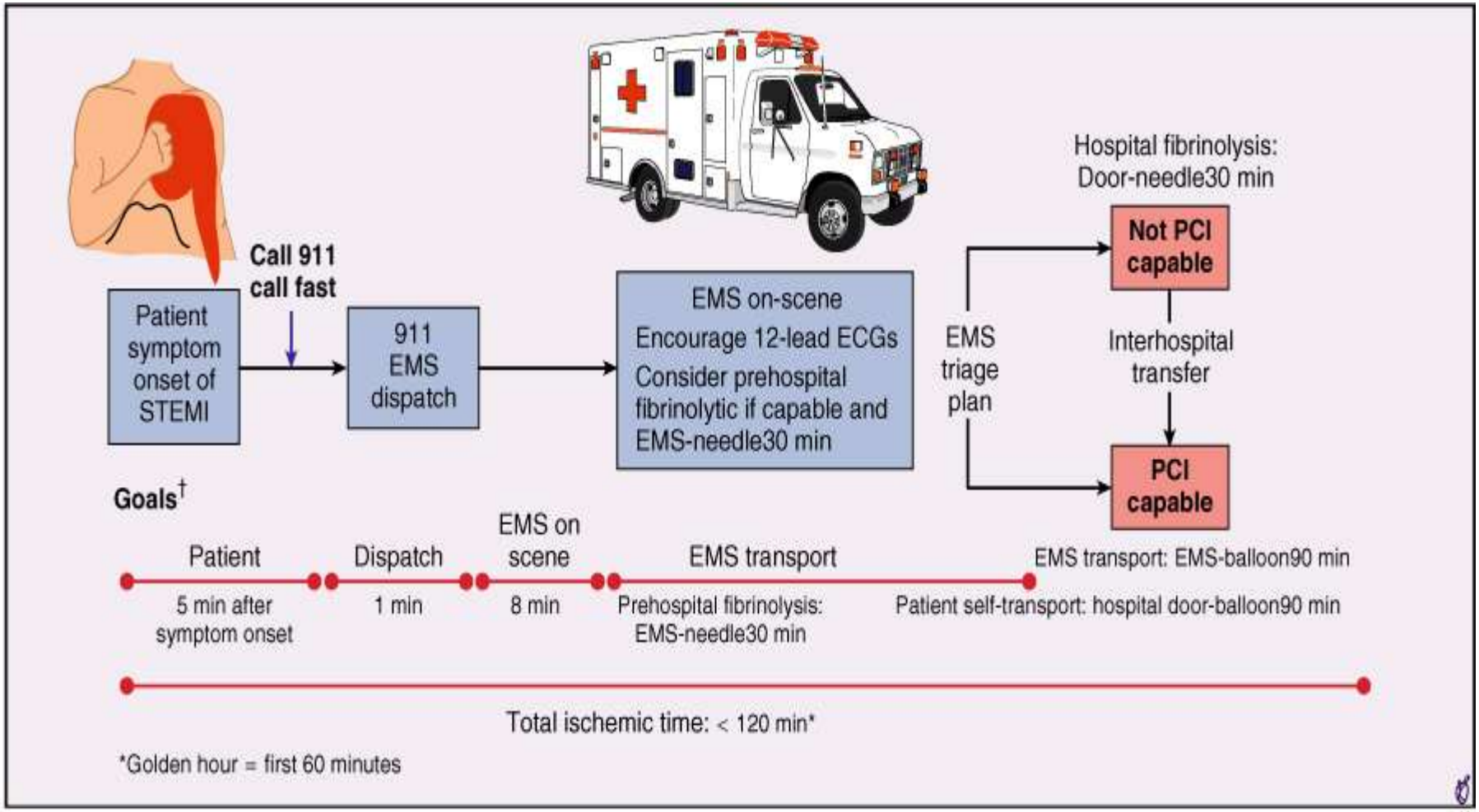





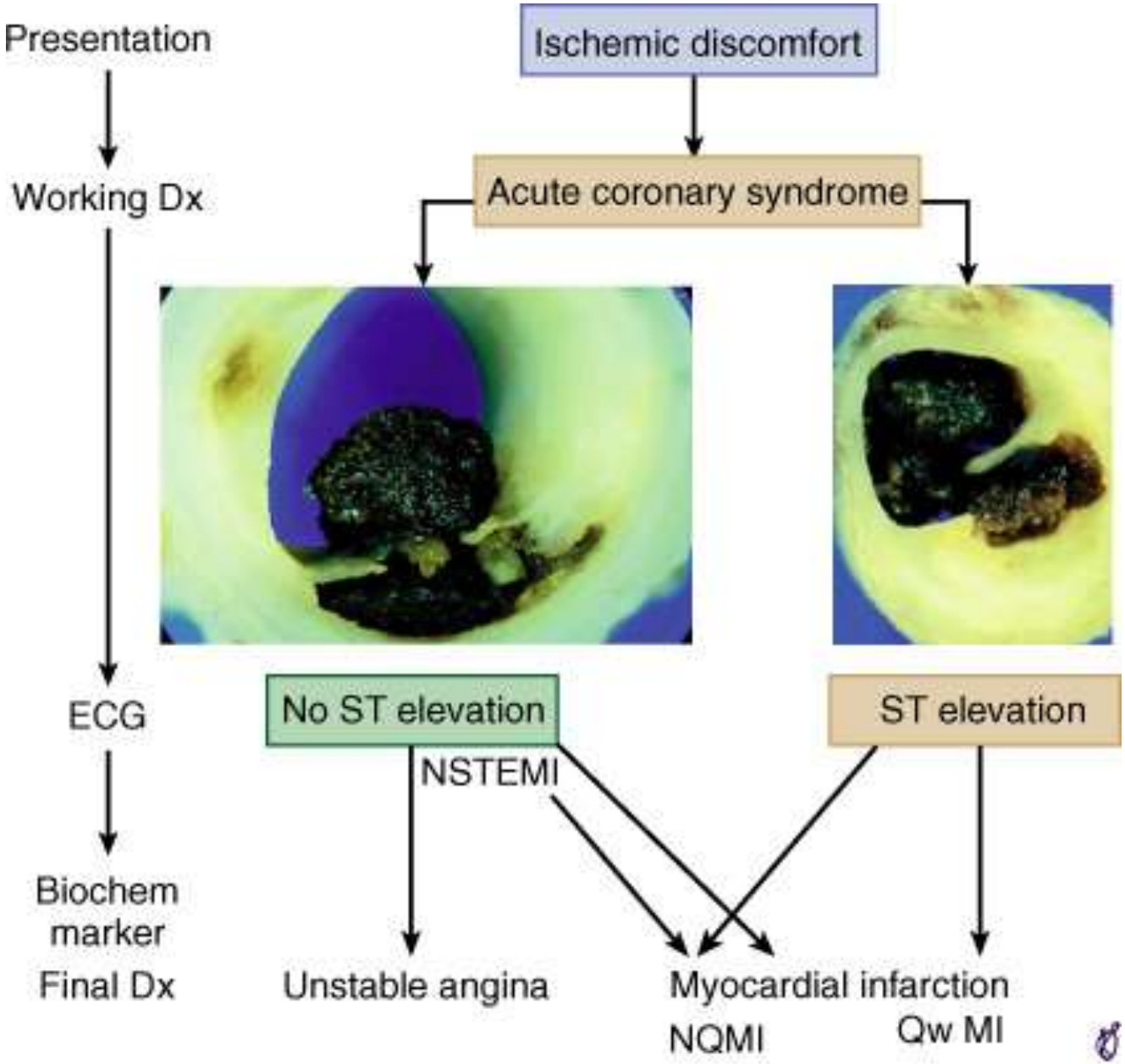
A

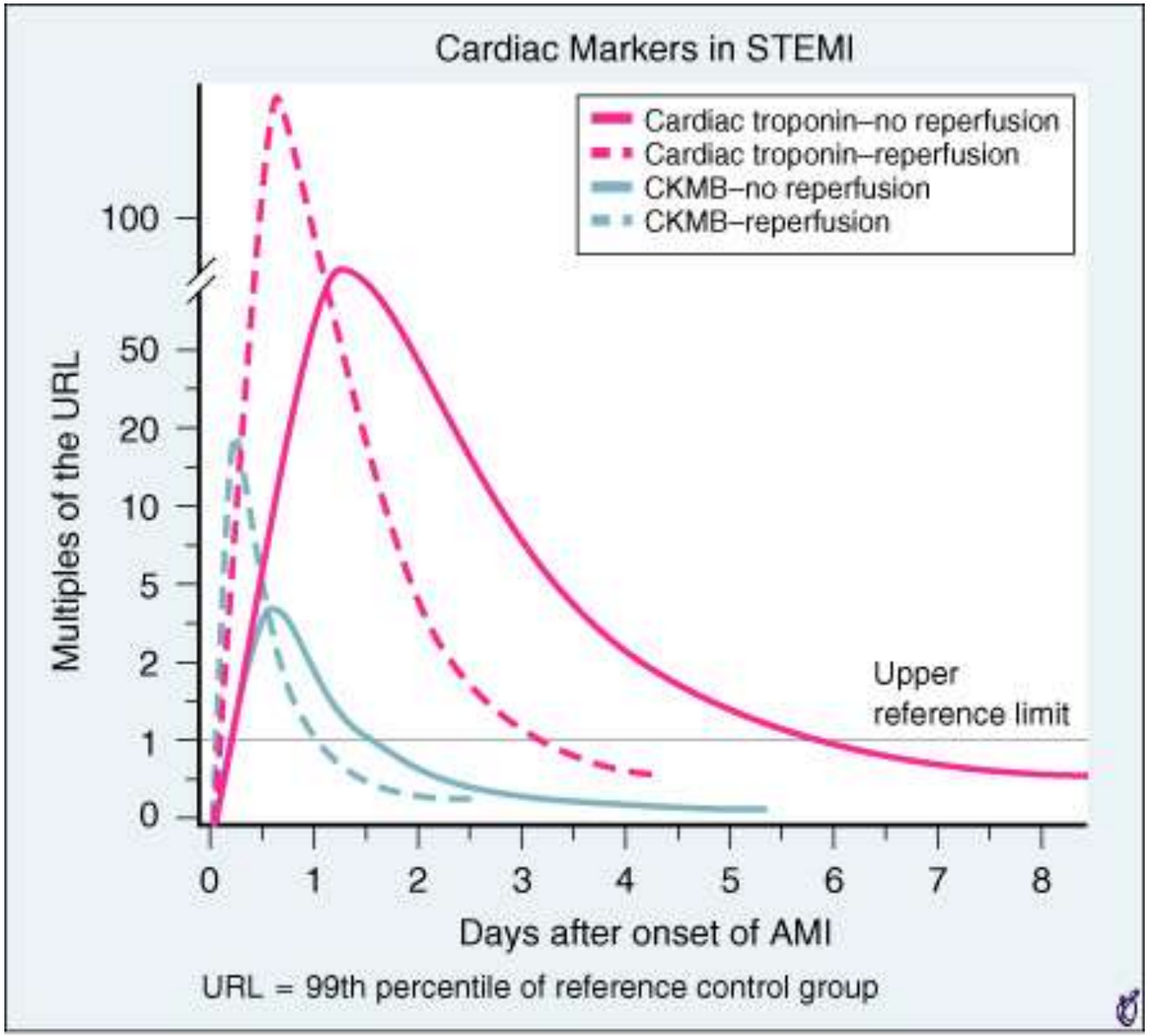


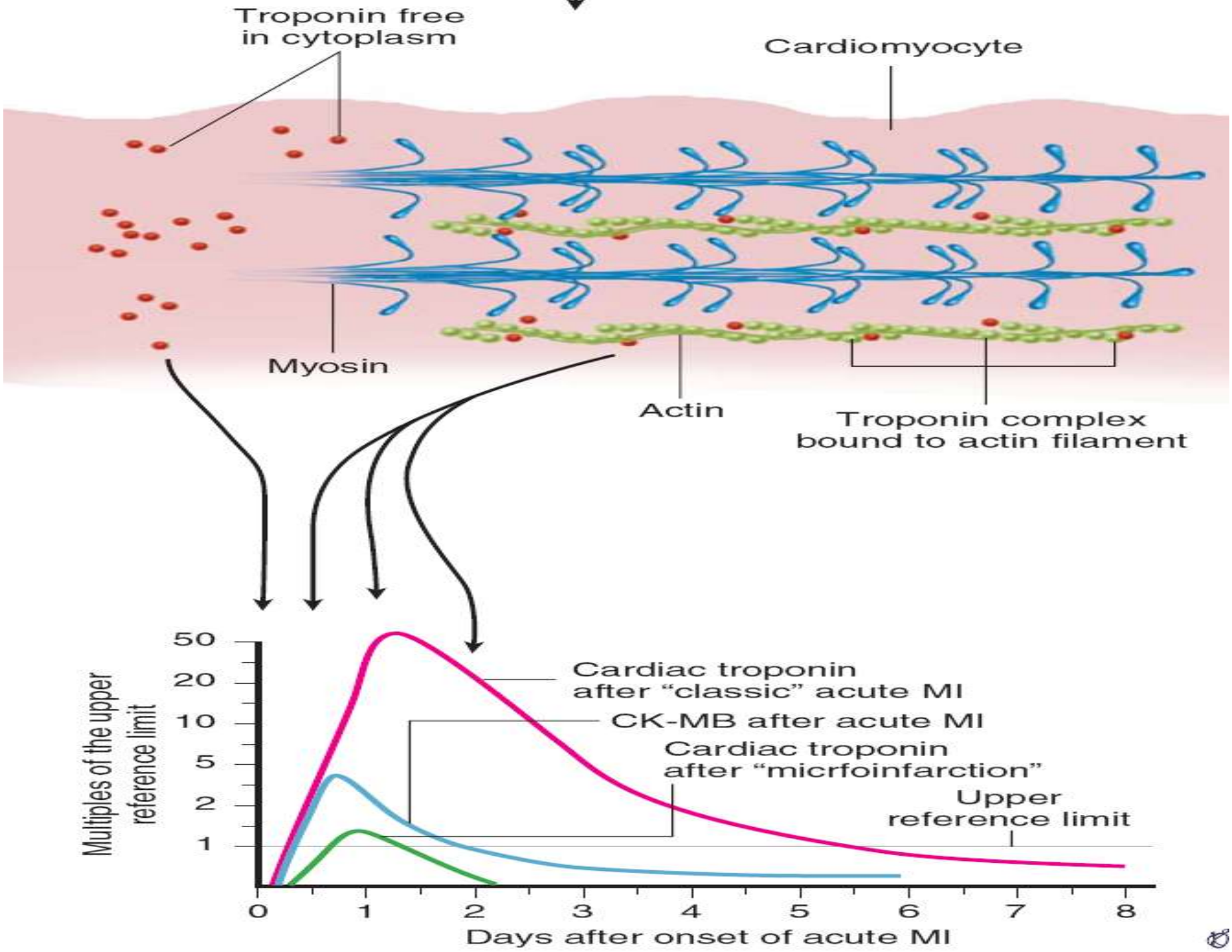
A 47-year-old man with no prior history of cardiac disease presented to an outside hospital describing “ an awesome feeling that just sat in my chest” associated with bilateral arm weakness. The initial electrocardiogram **(A)** revealed STsegment elevation in the right precordial leads and to a lesser extent in the inferior leads.



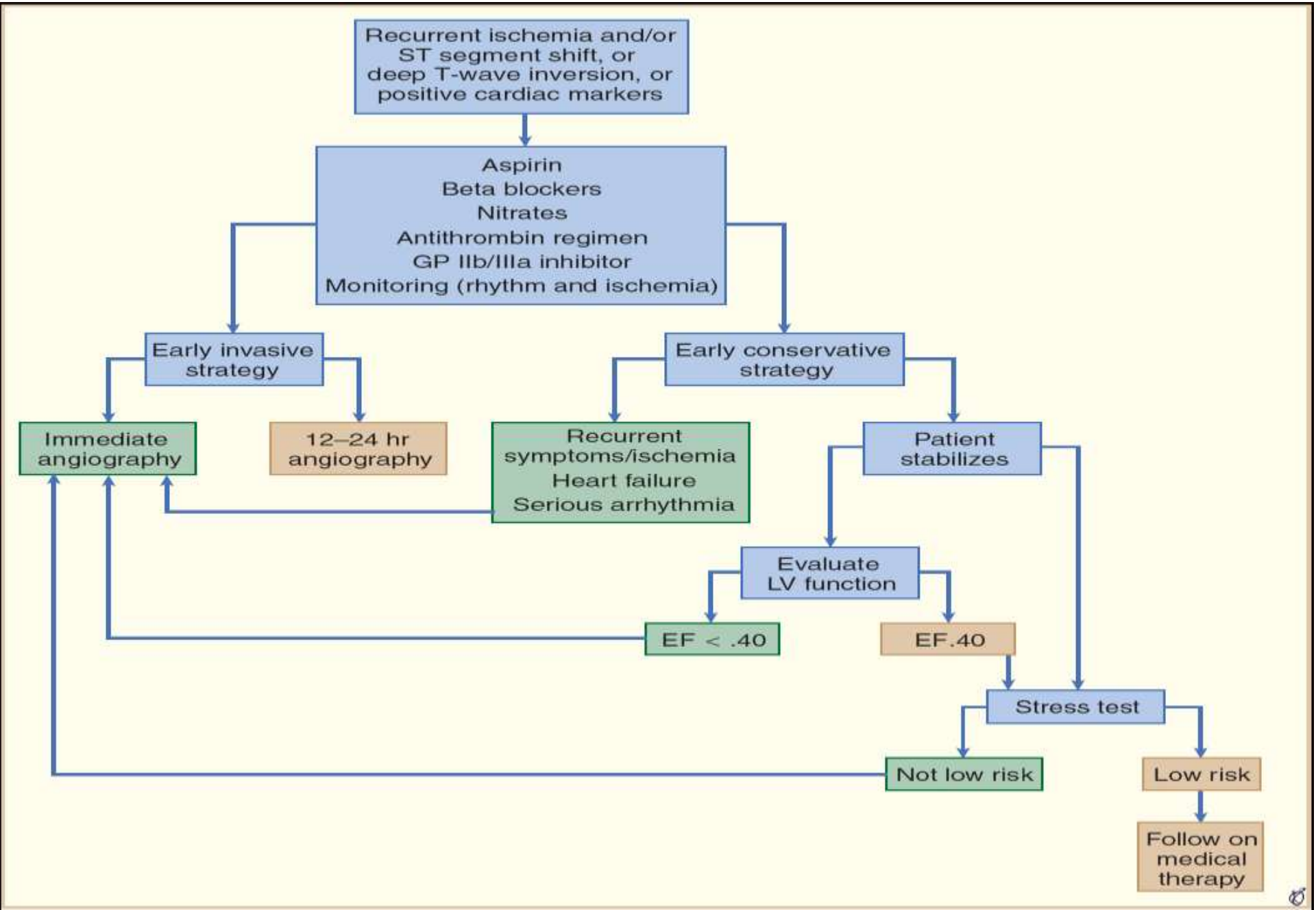

A

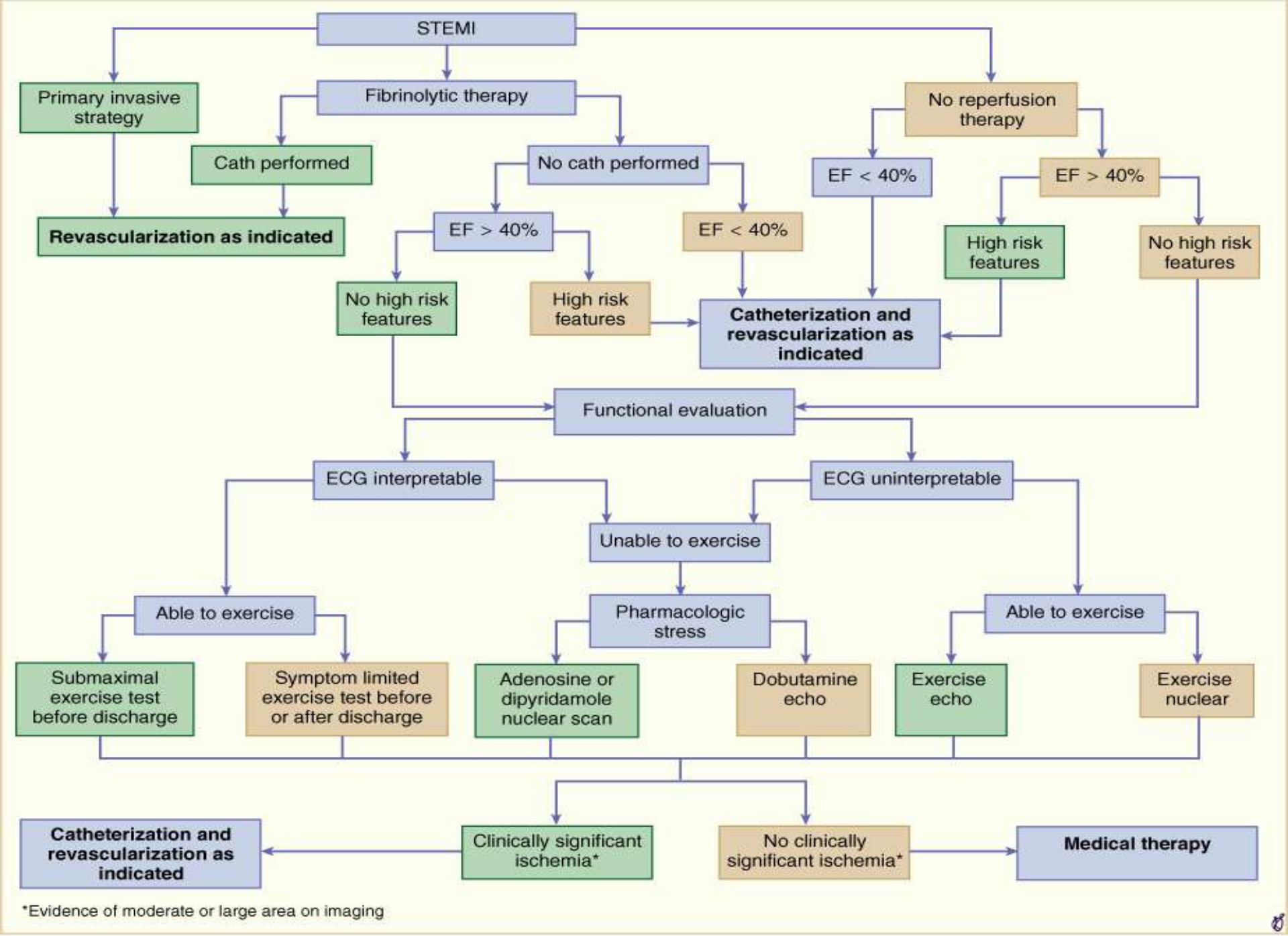





Troponin free in cytoplasm
Cardiomyocyte
Myosin
Actin
Troponin complex bound to actin filament
Multiples of the upper reference limit
50
20
10
5
2
1
Cardiac troponin after "classic" acute MI
CK-MB after acute MI
Cardiac troponin after "micrfoinfarction"
Upper reference limit
0
1
2
3
4
5
6
7
8
Days after onset of acute MI

# Management of patients with UA/NSTEMI





| TABLE 47–5 | Sample Admitting Orders for the STEMI patient |
|---|---|

1. Condition: Serious
2. IV: NS on $D_5W$ to keep vein open. Start a second IV if IV medication is being given. This may be a saline lock.
3. Vital signs: every 1.5 hours until stable, then every 4 hours and as needed. Notify physician if HR is less than 60 beats/min or greater than 100 beats/min, BP is less than 100 mm Hg systolic or greater than 150 mm Hg systolic, respiratory rate is less than 8 or greater than 22.
4. Monitor: Continuous ECG monitoring for dysrhythmia and ST segment deviation
5. Diet: NPO except for sips of water until stable. Then start 2 gm sodium/day, low saturated fat (less than 7% of total calories/day), low cholesterol (less than 200 mg/day) diet, such as Total Lifestyle Change (TLC) diet
6. Activity: Bedside commode and light activity when stable
7. Oxygen: Continuous oximetry monitoring. Nasal cannula at 2 liters/min when stable for 6 hr, reassess for oxygen need (i.e., $O_2$ saturation of less than 90%) and consider discontinuing oxygen.

## TABLE 47–5 Sample Admitting Orders for the STEMI patient

8. Medications:
    a. Nitroglycerin (NTG)
        1. Use sublingual NTG 0.4 mg every 5 min as needed for chest discomfort.
        2. Intravenous NTG for CHF, hypertension, or persistent ischemia.
    b. ASA
        1. If ASA not given in the emergency department (ED), chew nonenteric-coated ASA* 162-325 mg.
        2. If ASA has been given, start daily maintenance of 75-162 mg daily; may use enteric coated for gastrointestinal protection.
    c. Beta blocker
        1. If not given in the ED, assess for contraindications, i.e., bradycardia and hypotension; continue daily assessment to ascertain eligibility for beta blocker.
        2. If given in the ED, continue daily dose and optimize as dictated by heart rate and blood pressure.

**TABLE 47–5 Sample Admitting Orders for the STEMI patient**

8. Medications:

d. ACE inhibitor
   1. Start ACE inhibitor orally in patients with pulmonary congestion or LVEF less than 40 percent if the following are absent: hypotension (SBP less than 100 mm Hg or less than 30 mm Hg below baseline) or known contraindications to this class of medications.

e. Angiotensin receptor blocker (ARB)
   1. Start ARB orally in patients who are intolerant of ACE inhibitors and with either clinical or radiological signs of heart failure or LVEF less than 40 percent.

f. Pain medications
   1. IV morphine sulfate 2-4 mg with increments of 2-8 mg IV at 5- to 15-min intervals as needed to control pain.

g. Anxiolytics (based on a nursing assessment)

h. Daily stool softener

## TABLE 47–2 Assessment of Reperfusion Options for STEMI Patients

**Step 1:** Assess time and risk.

- Time since onset of symptoms
- Risk of STEMI
- Risk of fibrinolysis
- Time required for transport to a skilled PCI lab

**Step 2:** Determine if fibrinolysis or invasive strategy is preferred.

- *If presentation is less than 3 hr and there is no delay to an invasive strategy, there is no preference for either strategy.*

## TABLE 47–2 Assessment of Reperfusion Options for STEMI Patients

**Fibrinolysis is generally preferred if:**

- Early presentation (≤ 3hr from symptom onset and delay to invasive strategy) (see below)
- Invasive strategy is not an option
  - Catheterization lab occupied or not available
  - Vascular access difficulties
  - Lack of access to a skilled PCI lab[†‡]
- Delay to invasive strategy
  - Prolonged transport
  - (Door-to-Balloon)–(Door-to-Needle) more than 1 hr[*§]
  - Medical contact-to-balloon or door-to-balloon more than 90 min

## TABLE 47–2 Assessment of Reperfusion Options for STEMI Patients

**An invasive strategy is generally preferred if:**

- Skilled PCI lab available with surgical backup
  - Skilled PCI lab is available, defined by:[†‡]
  - Medical contact-to-balloon or door-to-balloon less than 90 min
  - (Door-to-Balloon)–(Door-to-Needle) less than 1 hr*
- High risk from STEMI
  - Cardiogenic shock
  - Killip class ≥ 3
- Contraindications to fibrinolysis including increased risk of bleeding and ICH
- Late presentation
  - Symptom onset was more than 3 hr ago
- Diagnosis of STEMI is in doubt

## TABLE 47–3 Contraindications and Cautions for Fibrinolytic Use in STEMI*

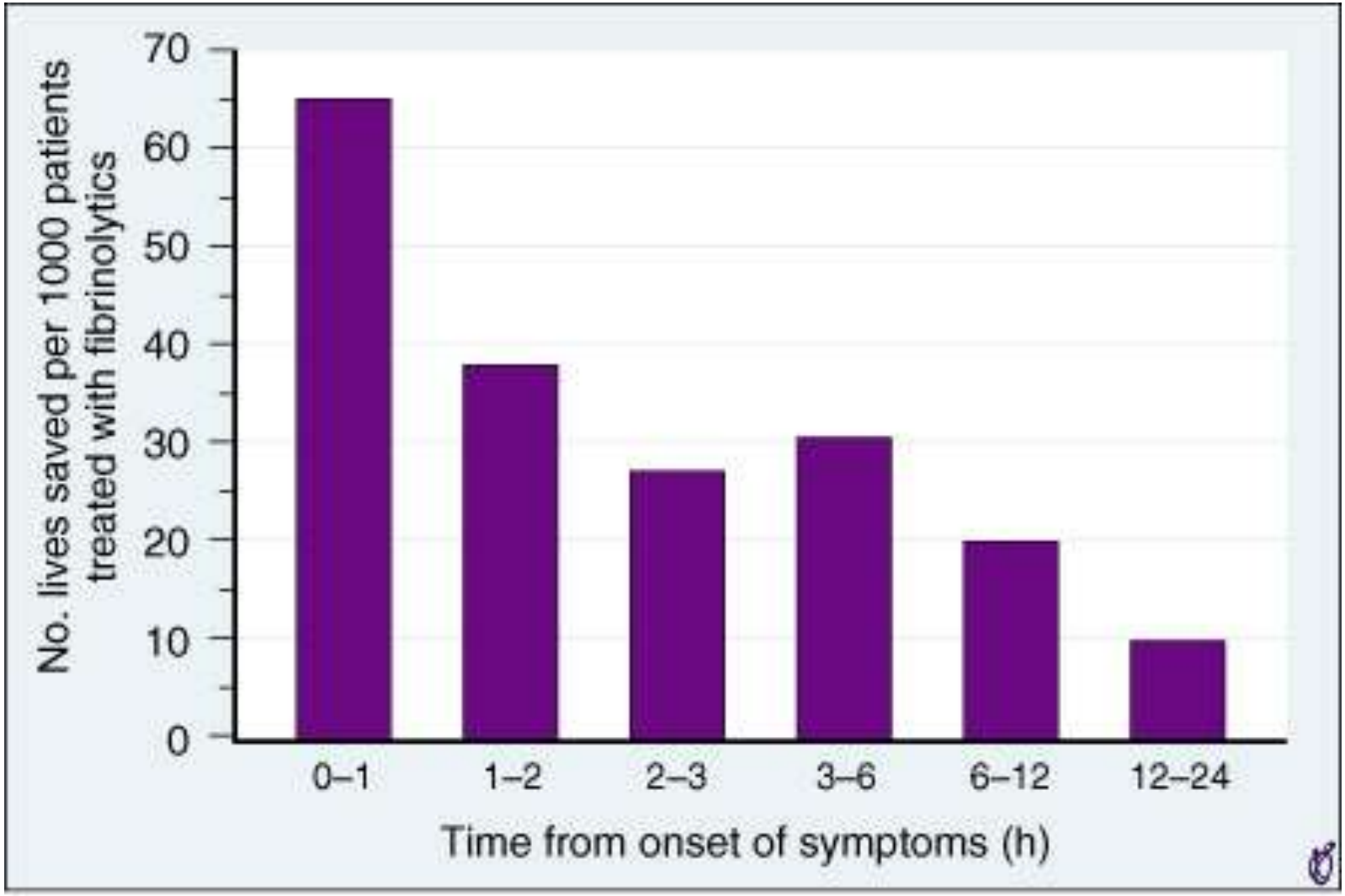
**Absolute contraindications**

- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (e.g., arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head or facial trauma within 3 months

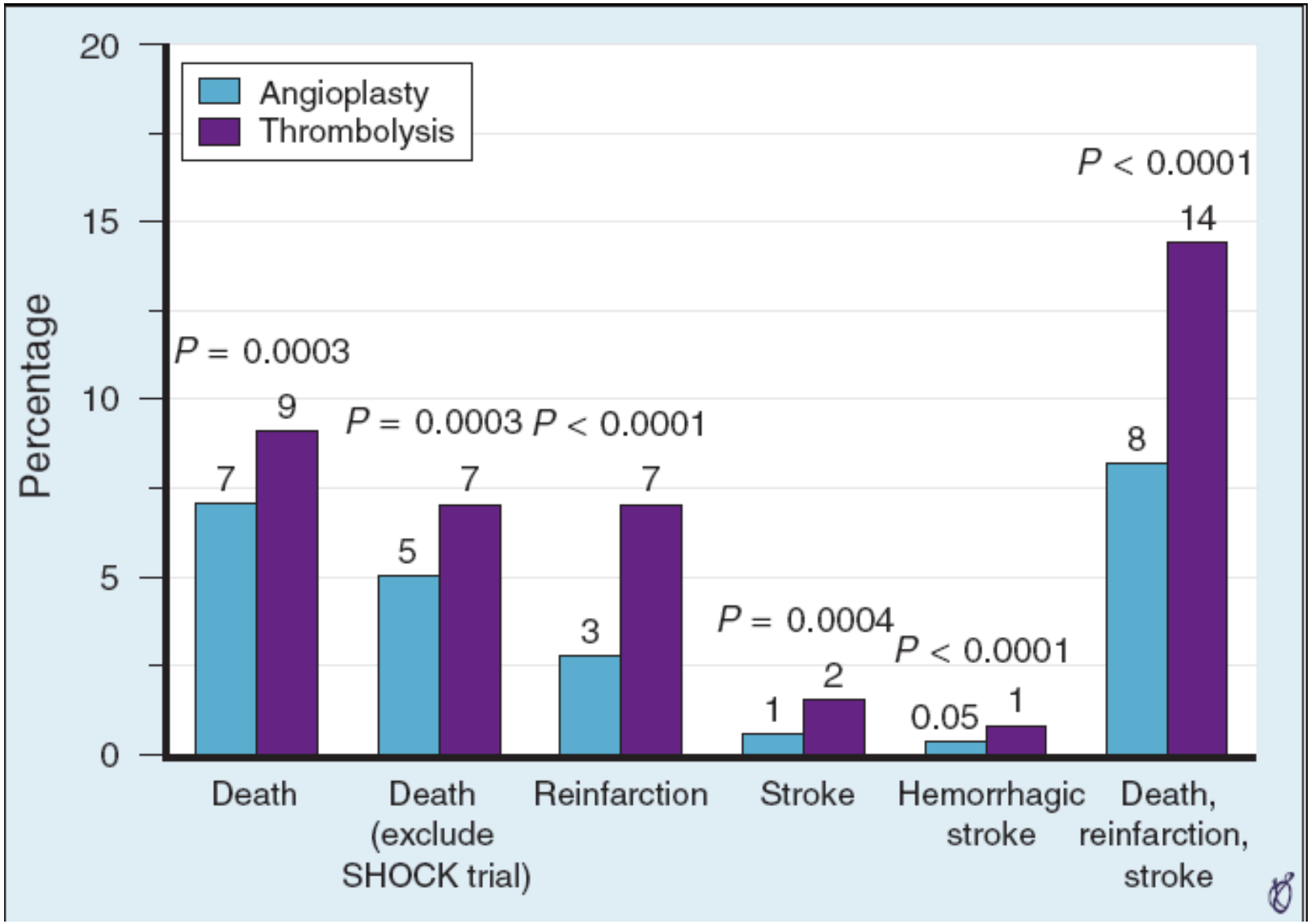
## TABLE 47–3 Contraindications and Cautions for Fibrinolytic Use in STEMI*

**Relative contraindications**

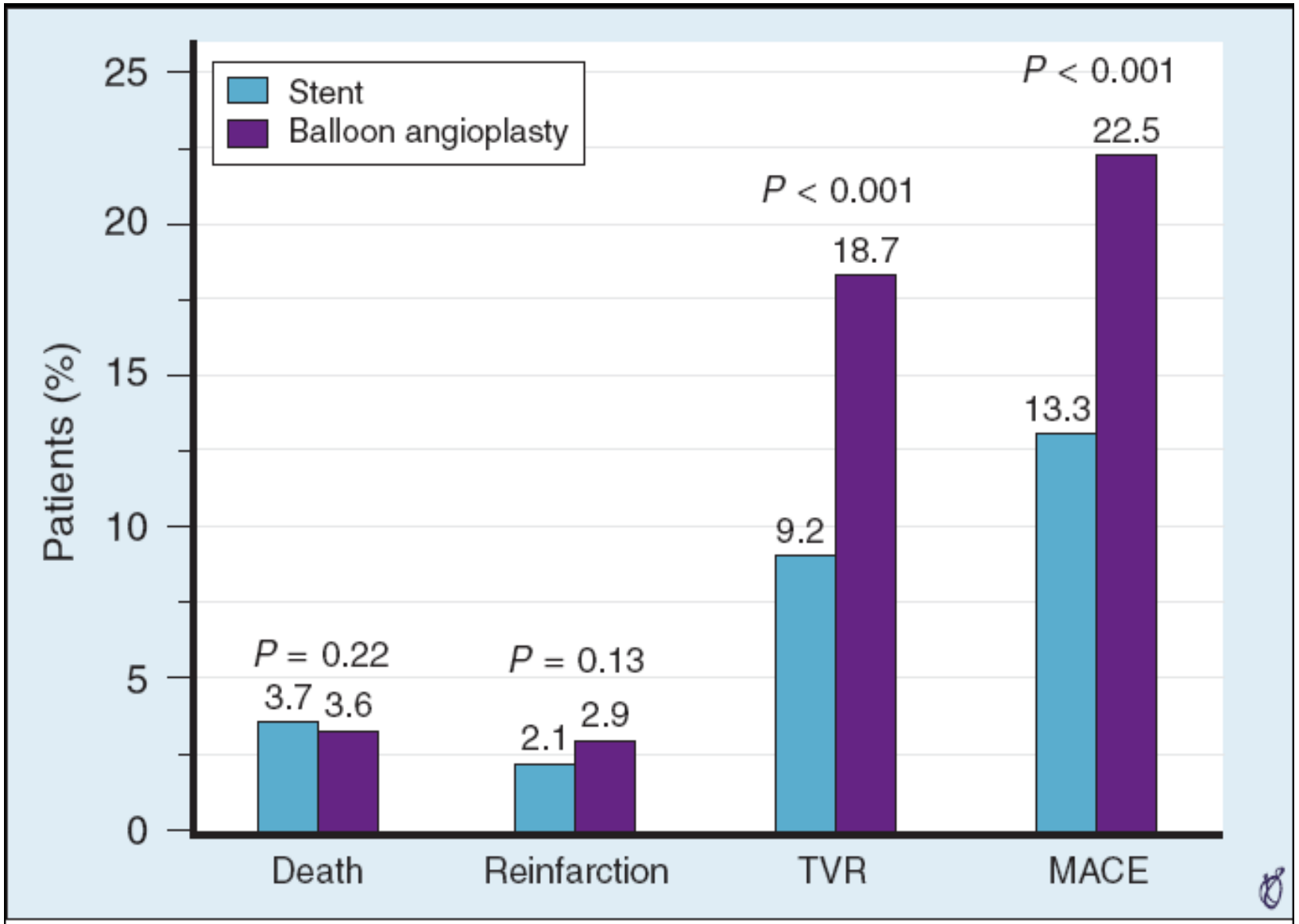
- History of chronic severe poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP greater than 180 or DBP greater than 110 Hg)†
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (more than 10 min) CPR or major surgery (less than 3 wk)
- Recent (within 2-4 weeks) internal bleeding
- Noncompressible vascular punctures
- For streptokinase/anistreplase: prior exposure (more than 5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding



# Short-term clinical outcomes of patients in 23 randomized trials of primary angioplasty versus thrombolysis



Results of meta-analysis comparing primary stenting with primary balloon angioplasty. MACE = major adverse cardiac events including death, reinfarction, and target vessel revascularization (TVR)



## TABLE 47–12 Cardiac Arrhythmias and Their Management During Acute Myocardial Infarction

| Category | Arrhythmia | Objective of Treatment | Therapeutic Options |
|---|---|---|---|
| 1. Electrical instability | Ventricular premature beats | Correction of electrolyte deficits and increased sympathetic tone | Potassium and magnesium solutions, beta blocker |
| | Ventricular tachycardia | Prophylaxis against ventricular fibrillation, restoration of hemodynamic stability | Antiarrhythmic agents; cardioversion/ defibrillation |
| | Ventricular fibrillation | Urgent reversion to sinus rhythm | Defibrillation; bretylium tosylate |
| | Accelerated idioventricular rhythm | Observation unless hemodynamic function is compromised | Increase sinus rate (atropine, atrial pacing); antiarrhythmic agents |
| | Nonparoxysmal atrioventricular junctional tachycardia | Search for precipitating causes (e.g., digitalis intoxication); suppress arrhythmia only if hemodynamic function is compromised | Atrial overdrive pacing; antiarrhythmic agents; cardioversion relatively contraindicated if digitalis intoxication present |

## TABLE 47–12 Cardiac Arrhythmias and Their Management During Acute Myocardial Infarction

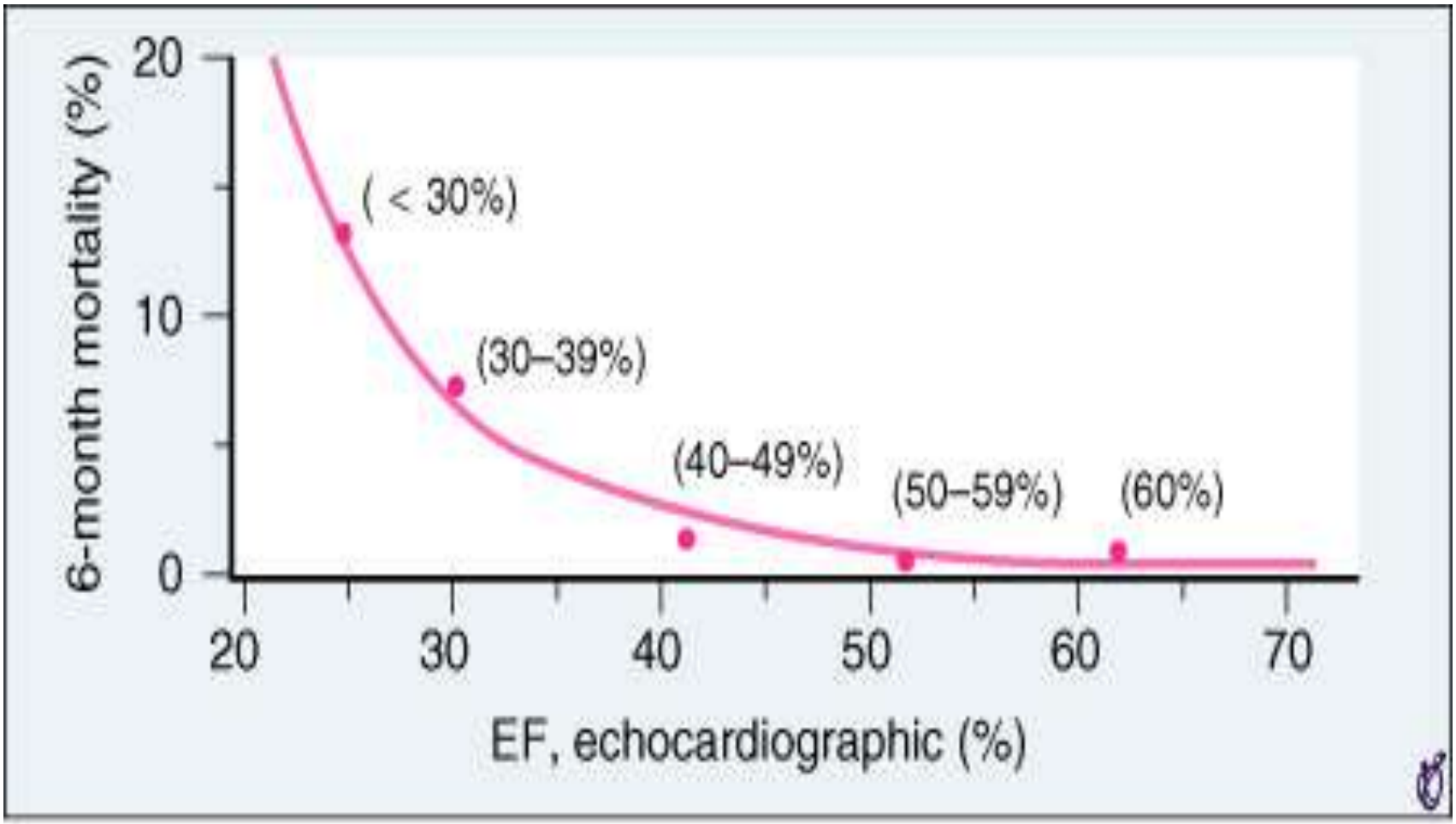
| Category | Arrhythmia | Objective of Treatment | Therapeutic Options |
|---|---|---|---|
| 2. Pump failure/ excessive sympathetic stimulation | Sinus tachycardia | Reduce heart rate to diminish myocardial oxygen demands | Antipyretics; analgesics; consider beta blocker unless congestive heart failure present; treat latter if present with anticongestive measures (diuretics, afterload reduction) |
| | Atrial fibrillation and/or atrial flutter | Reduce ventricular rate; restore sinus rhythm | Verapamil, digitalis glycosides; anticongestive measures (diuretics, afterload reduction); cardioversion; rapid atrial pacing (for atrial flutter) |
| | Paroxysmal supraventricular tachycardia | Reduce ventricular rate; restore sinus rhythm | Vagal maneuvers; verapamil, cardiac glycosides, beta-adrenergic blockers; cardioversion; rapid atrial pacing |
| 3. Bradyarrhythmias and conduction disturbances | Sinus bradycardia | Acceleration of heart rate only if hemodynamic function is compromised | Atropine; atrial pacing |
| | Junctional escape rhythm | Acceleration of sinus rate only if loss of atrial "kick" causes hemodynamic compromise | Atropine; atrial pacing |
| | Atrioventricular block and intraventricular block | | Insertion of pacemaker |

## TABLE 47-9 Hemodynamic Patterns for Common Clinical Conditions

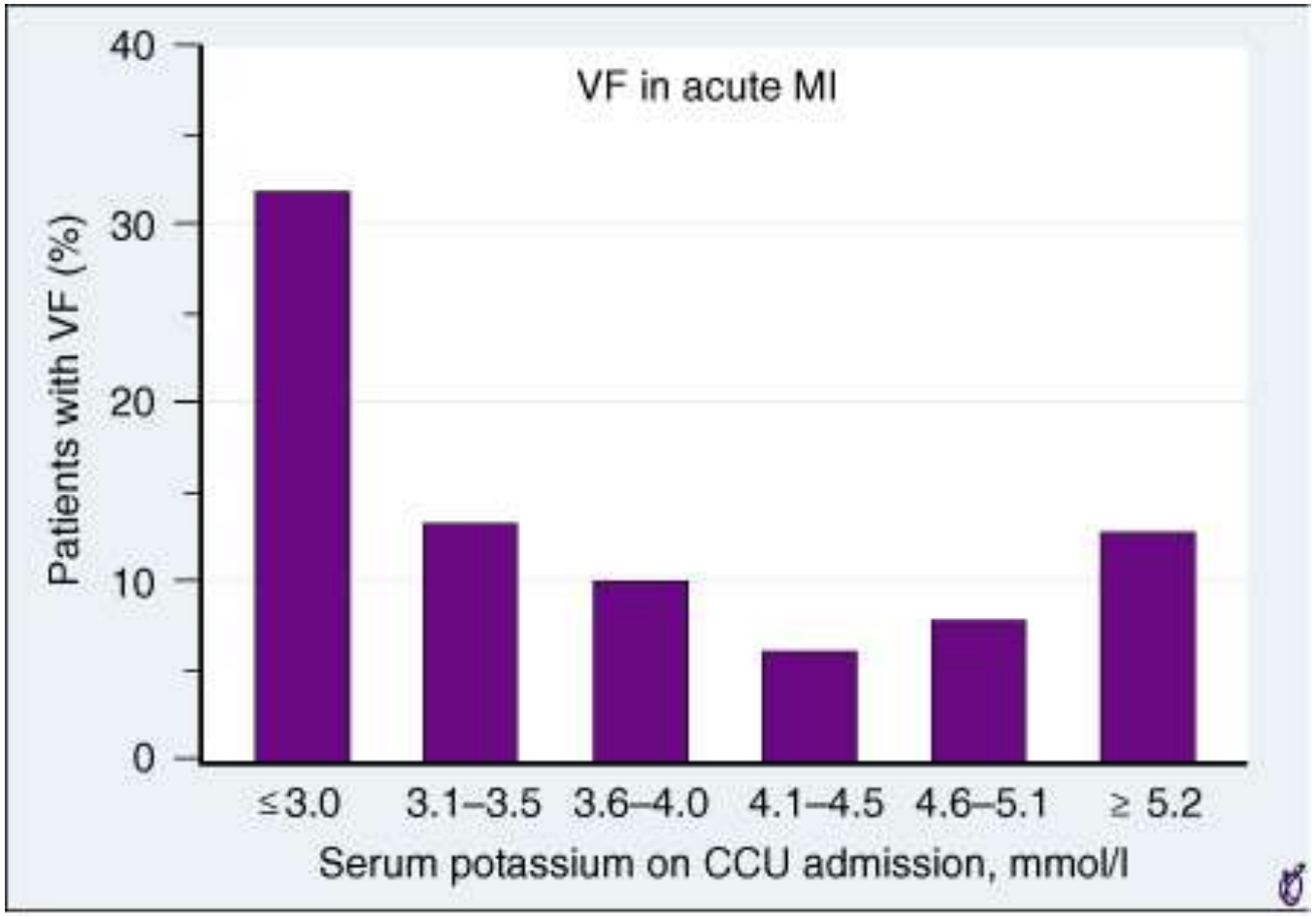
| Cardiac Condition | Chamber Pressure (mm Hg) | | | | |
|---|---|---|---|---|---|
| | *RA* | *RV* | *PA* | *PCW* | *CI* |
| Normal | 0-6 | 25/0-6 | 25/0-12 | 6-12 | ≥2.5 |
| AMI without LVF | 0-6 | 25/0-6 | 30/12-18 | ≤18 | ≥2.5 |
| AMI with LVF | 0-6 | 30-40/0-6 | 30-40/18-25 | >18 | >2.0 |
| Biventricular failure | >6 | 50-60/>6 | 50-60/25 | 18-25 | >2.0 |
| RVMI | 12-20 | 30/12-20 | 30/12 | ≤12 | <2.0 |
| Cardiac tamponade | 12-16 | 25/12-16 | 25/12-16 | 12-16 | <2.0 |
| Pulmonary embolism | 12-20 | 50-60/12-20 | 50-60/12 | <12 | <2.0 |

AMI = acute myocardial infarction; CI = cardiac index; LVF = left ventricular failure; PA = pulmonary artery; PCW = pulmonary capillary wedge; RA = right atrium; RV = right ventricle; RVMI = right ventricular myocardial infarction.

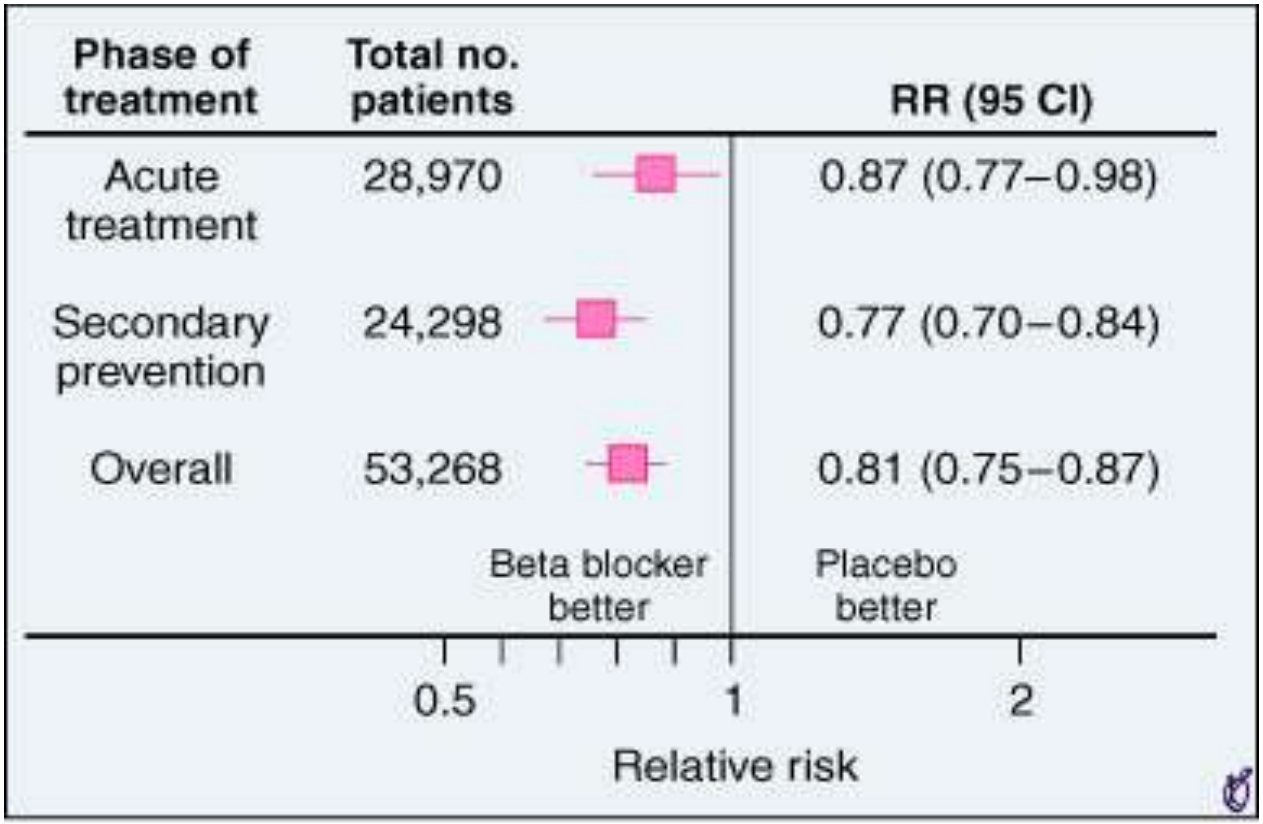
# Impact of left ventricular function on survival following myocardial infarction



# Hypokalemia and ventricular fibrillation in acute myocardial infarction



# Effect of beta blockers on mortality rate in patients with myocardial infarction.

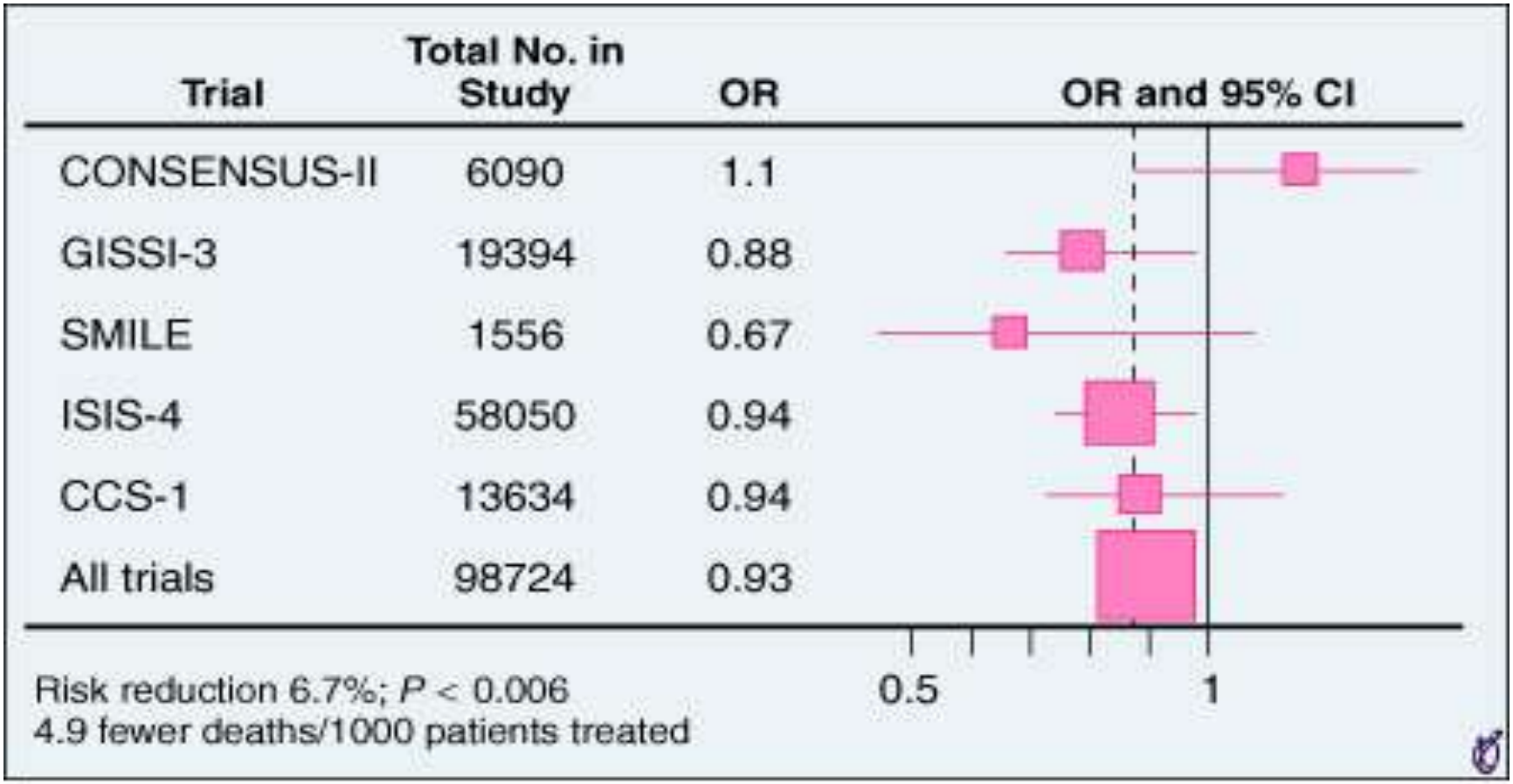




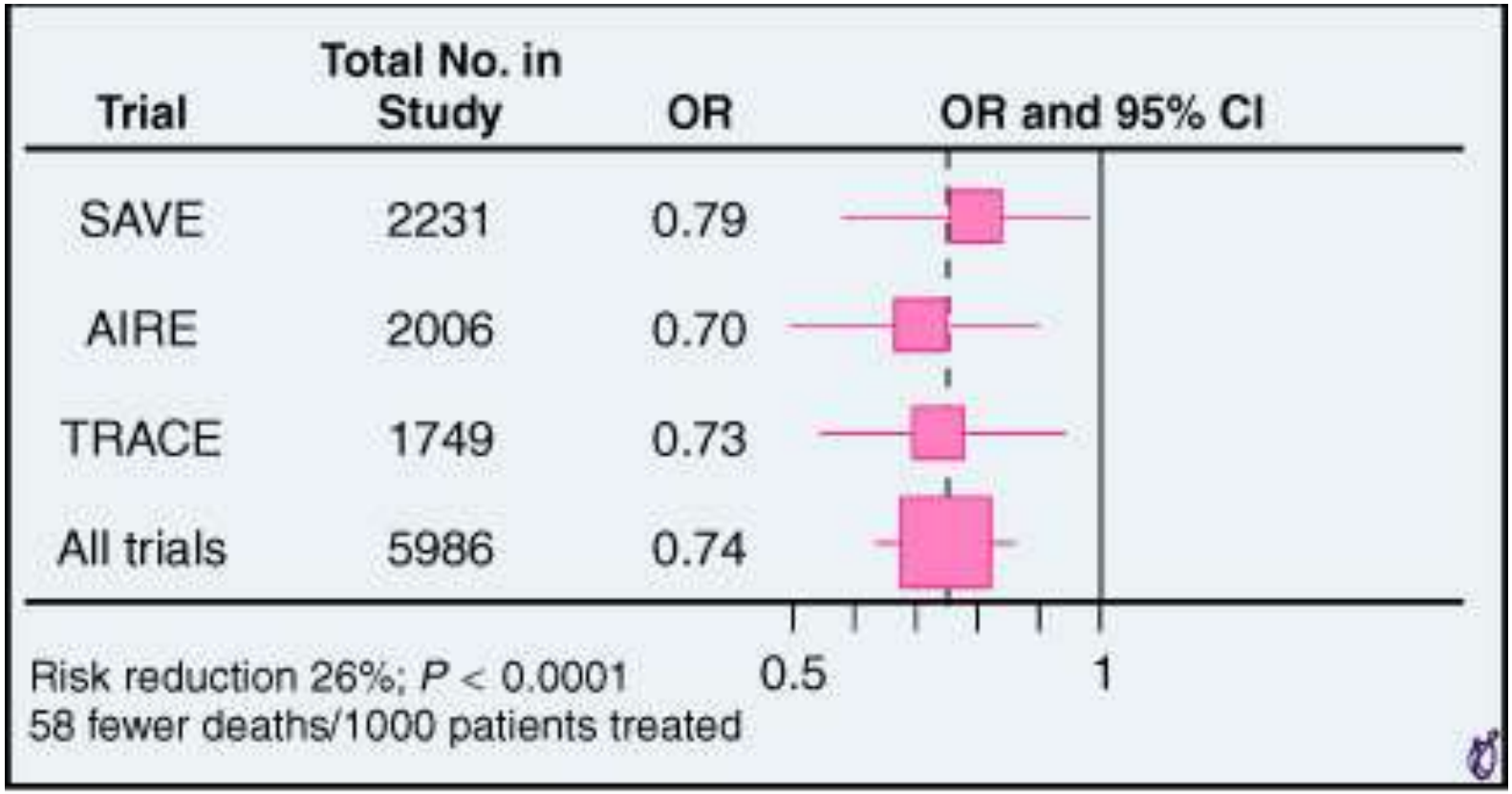

**The relative risk of mortality is reduced with beta blockers both during the acute phase of treatment and when prescribed as secondary prevention after acute myocardial infarction.**

| TABLE 47–6 | Contraindications to Beta-Adrenoceptor Blocker Therapy in Acute Myocardial Infarction |
|---|---|
| Heart rate < 60 beats/min | |
| Systolic arterial pressure < 100 mm Hg | |
| Moderate or severe left ventricular failure | |
| Signs of peripheral hypoperfusion | |
| PR interval > 0.24 second | |
| Second- or third-degree atrioventricular block | |
| Severe chronic obstructive pulmonary disease | |
| History of asthma | |
| Severe peripheral vascular disease | |
| Insulin-dependent diabetes mellitus | |

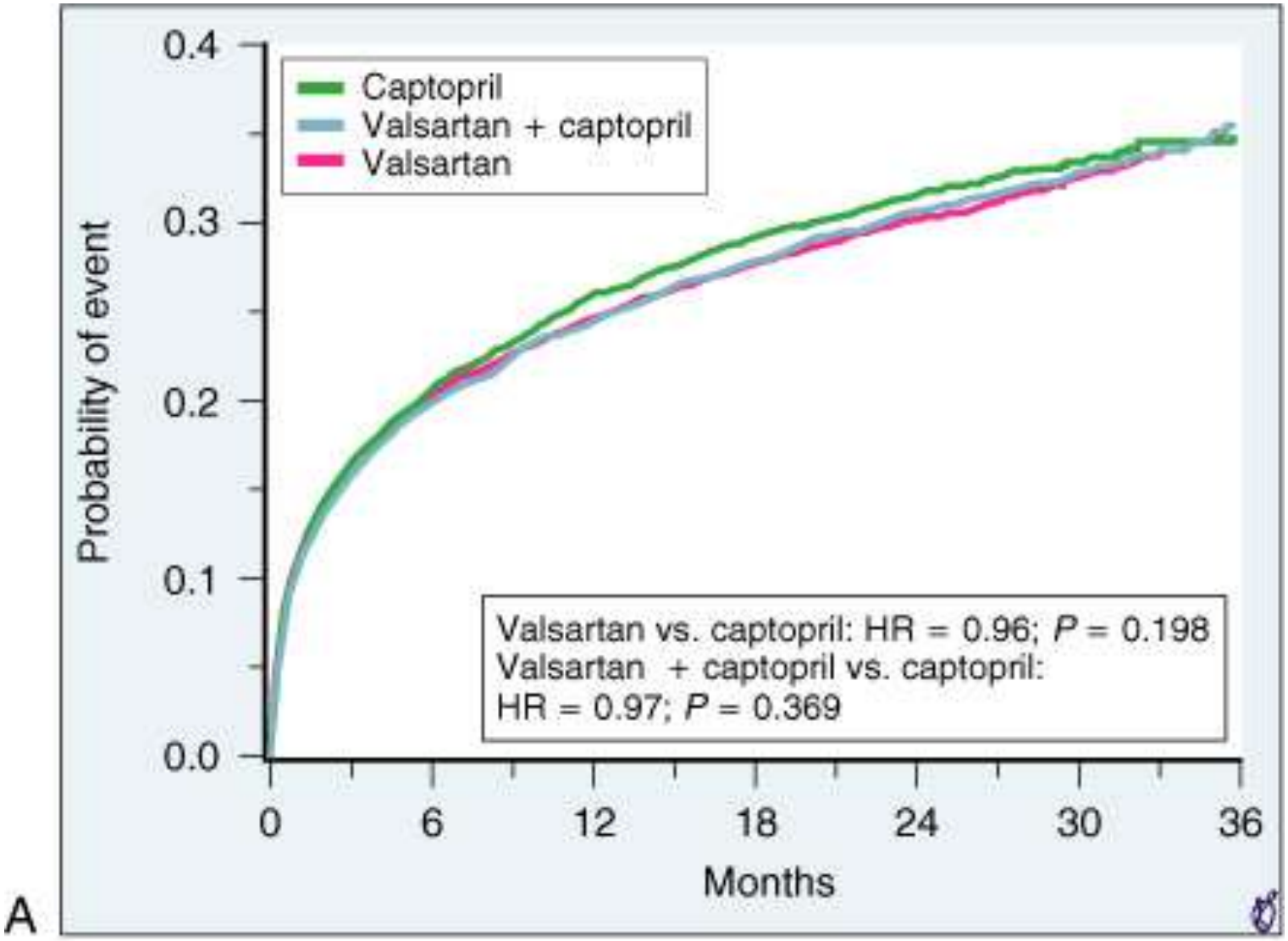
# Effects of angiotensin-converting enzyme inhibitors on mortality after myocardial infarction: results from the short-term trials.



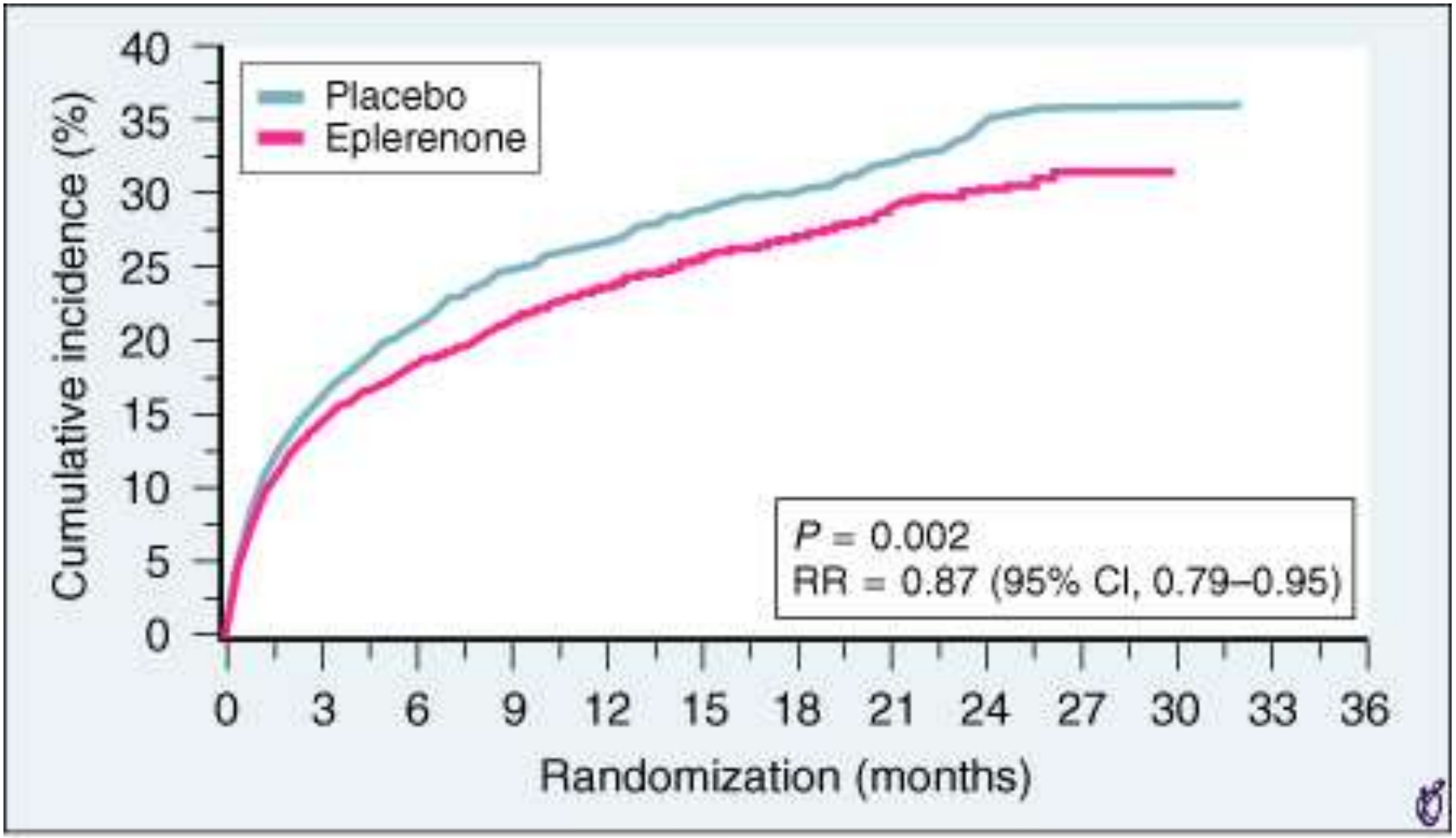
# Effect of angiotensin-converting enzyme inhibitors on mortality after myocardial infarction: results from the long-term trials.



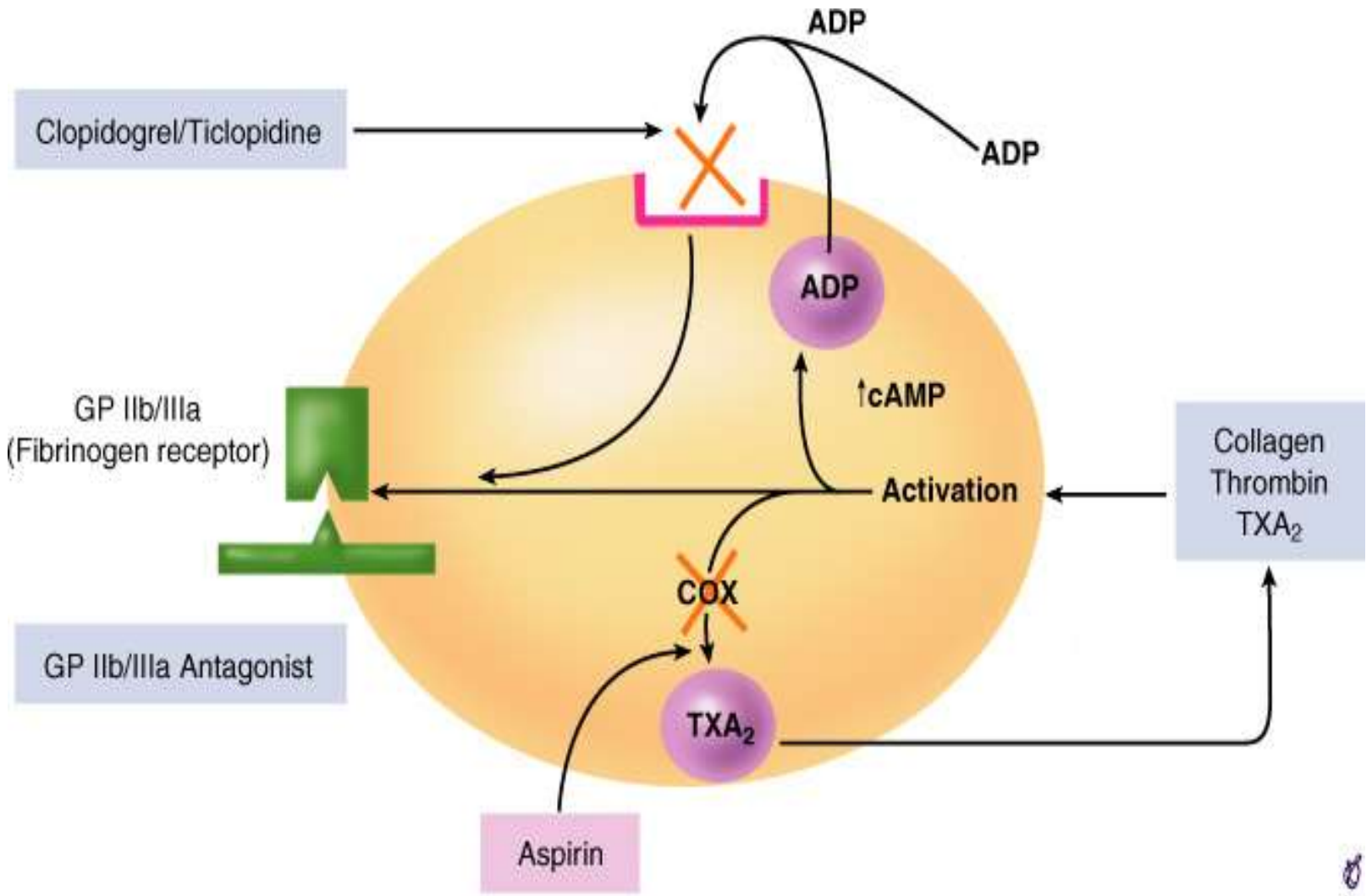
Effects of an angiotensin-converting enzyme inhibitor (captopril), angiotensin receptor blocker (valsartan), or the combination after myocardial infarction. The Kaplan-Meier estimates of mortality.



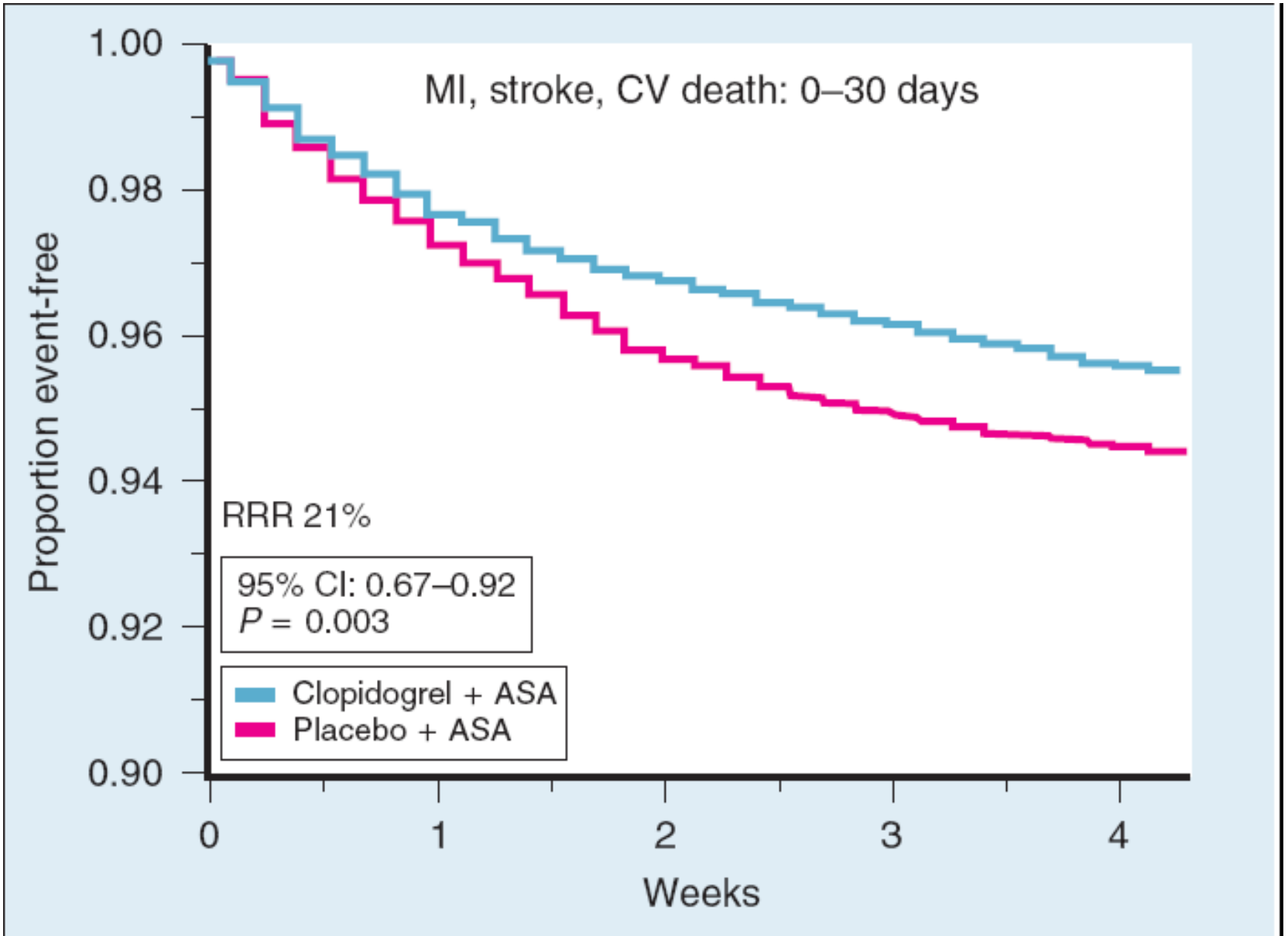
**Effect of a selective aldosterone receptor blocker (eplerenone) after myocardial infarction**. The Kaplan-Meier estimates of the rate of death from cardiovascular causes or hospitalization for cardiovascular events in the **EPHESUS trial** are depicted.



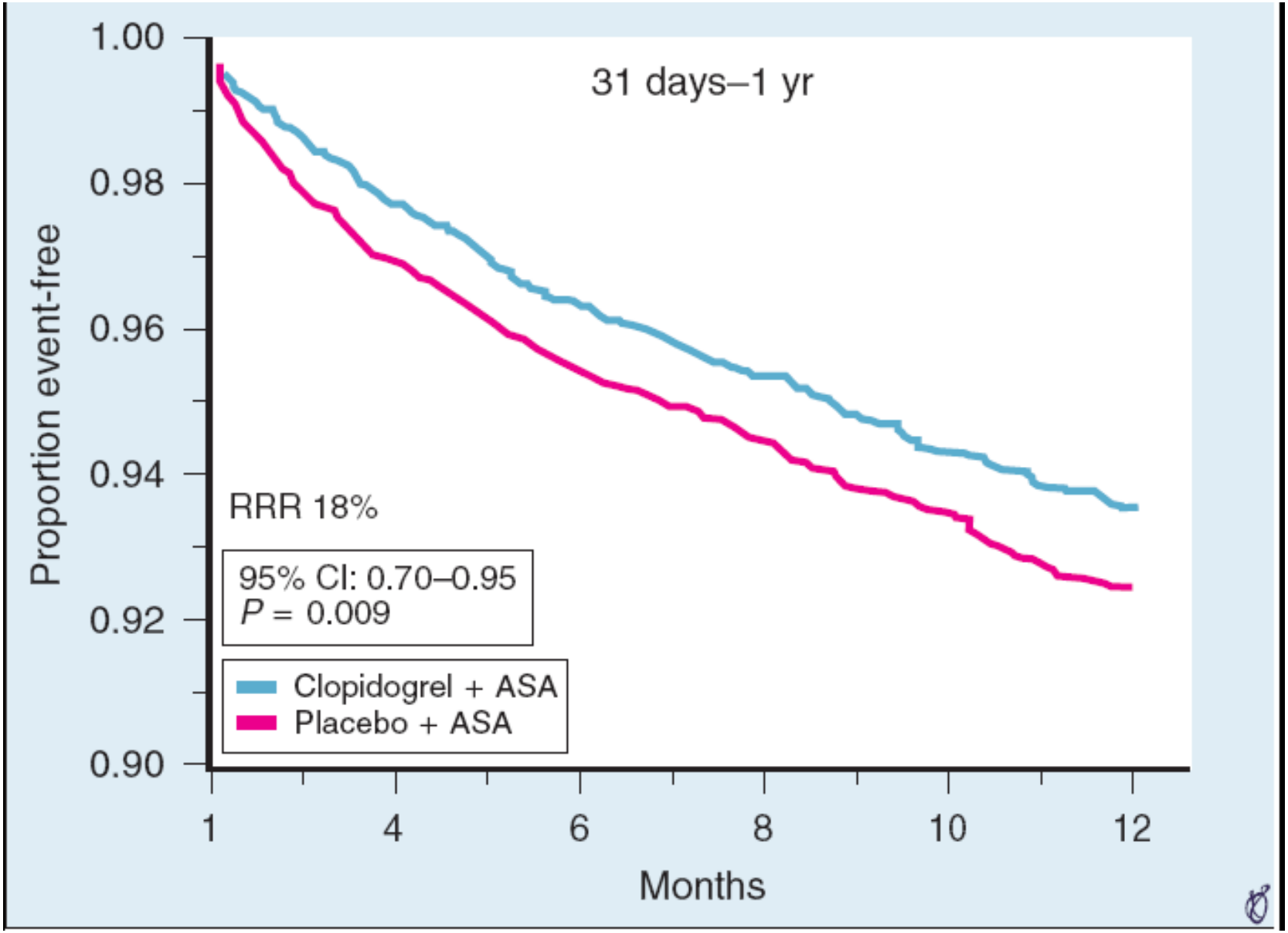
**Mechanisms of action of antiplatelet therapies.**
**ADP=adenosine diphosphate; cAMP=cyclic adenosine monophosphate; COX=cyclooxygenase; GP=glycoprotein; TXA2=thromboxane A2**



# Benefit of the addition of clopidogrel to aspirin compared with placebo and aspirin during the first 30 days



# Benefit of the addition of clopidogrel to aspirin compared with placebo and aspirin between 31 days and 1 year



# ΠΕΡΙΚΑΡΔΙΤΙΔΑ

# PERICARDIUM

The pericardium is composed of two distinct layers.

The fibrous parietal pericardium provides a protective sac around the heart to prevent sudden cardiac dilation and to minimize bulk cardiac motion.

The inner, visceral pericardium is intimately related to the surface of the heart.

These two layers are normally separated by 10 to 50 mL of clear fluid, an ultrafiltrate of plasma that is produced by the visceral pericardium and functions as a lubricant to minimize frictional forces between the heart and the pericardium.

In health, the intrapericardial pressure is slightly negative.

# ACUTE PERICARDITIS

The most common clinical pathologic process involving the pericardium is acute pericarditis.

Although multiple causes are possible the most common are viral infection and unknown (idiopathic).

Classically, this disorder is characterized by chest pain, pericardial friction rub, diffuse electrocardiographic changes, and pericardial effusion, although sometimes neither electrocardiographic changes nor a pericardial effusion is present.

The clinical syndrome is often relatively brief (days to weeks) in duration and uncomplicated, although vigilance for progression to tamponade is always prudent.

# ETIOLOGY OF ACUTE PERICARDITIS

## INFECTIOUS PERICARDITIS

Viral (coxsackieviruses A and B, echovirus, mumps, adenovirus, Epstein-Barr, human immunodeficiency virus, influenza)

*Mycobacterium tuberculosis*

Bacterial *(Pneumococcus, Streptococcus, Staphylococcus, Legionella)*

Fungal (histoplasmosis, coccidioidomycosis, candidiasis, blastomycosis)

Other (syphilis, parasites, Q fever)

# ETIOLOGY OF ACUTE PERICARDITIS

## NONINFECTIOUS PERICARDITIS

Idiopathic
Neoplasm
Metastatic (lung cancer, breast cancer, melanoma, lymphoma)
Primary (mesothelioma)
Renal failure
Trauma
Irradiation (especially for breast cancer, Hodgkin's disease)
Myocardial infarction
Hypothyroidism
Aortic dissection with hemopericardium
Chylopericardium (thoracic duct injury)
Post pericardiotomy
Chest wall injury or trauma
Pneumonia

# ETIOLOGY OF ACUTE PERICARDITIS

## HYPERSENSITIVITY PERICARDITIS

Collagen vascular disease
(systemic lupus erythematosus, rheumatoid arthritis, scleroderma, acute rheumatic fever, Sjögren's syndrome, Reiter's syndrome, ankylosing spondylitis)

Drug induced
(procainamide, hydralazine, isoniazid; smallpox vaccine)

Post myocardial infarction (Dressler's syndrome)

Familial Mediterranean fever

# CLINICAL MANIFESTATIONS

Chest pain of acute infectious (viral) pericarditis typically develops in young adults (18 to 30 years) 1 to 2 weeks after a “viral illness.” The symptoms are sudden and severe in onset, characteristically with retrosternal or left precordial pain and referral to the back and trapezius ridge.

Pain may be *preceded* by low-grade fever (in contrast to myocardial infarction, in which the pain precedes the fever). Although radiation to the arms in a manner similar to myocardial ischemia also may occur, it is less common.

The pain is often pleuritic (e.g., accentuated by inspiration or coughing) and may be aggravated (supine or left lateral decubitus posture) or relieved (upright posture) by changes in posture.

# PHYSICAL EXAMINATION

The physical examination in patients with acute pericarditis is most notable for a pericardial friction rub.

Although classically described as triphasic, with systolic and early (passive ventricular filling) and late (atrial systole) diastolic components, more commonly a biphasic (systole and diastole) or a monophasic rub may be heard.

The rub may be transient and positional, often best appreciated in the supine or left lateral decubitus posture.

Low-grade fever, resting tachycardia, and atrial ectopy are common, but atrial fibrillation is unusual.

# ΟΞΕΙΑ ΠΕΡΙΚΑΡΔΙΤΙΔΑ

Ἠλεκτροκαρδιογραφικά χαραυτηριστικά :

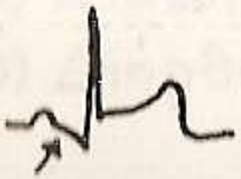
(1) Ἡ ἀνύψωση τοῦ ST ἔχει τό κυρτό πρός τά πάνω.

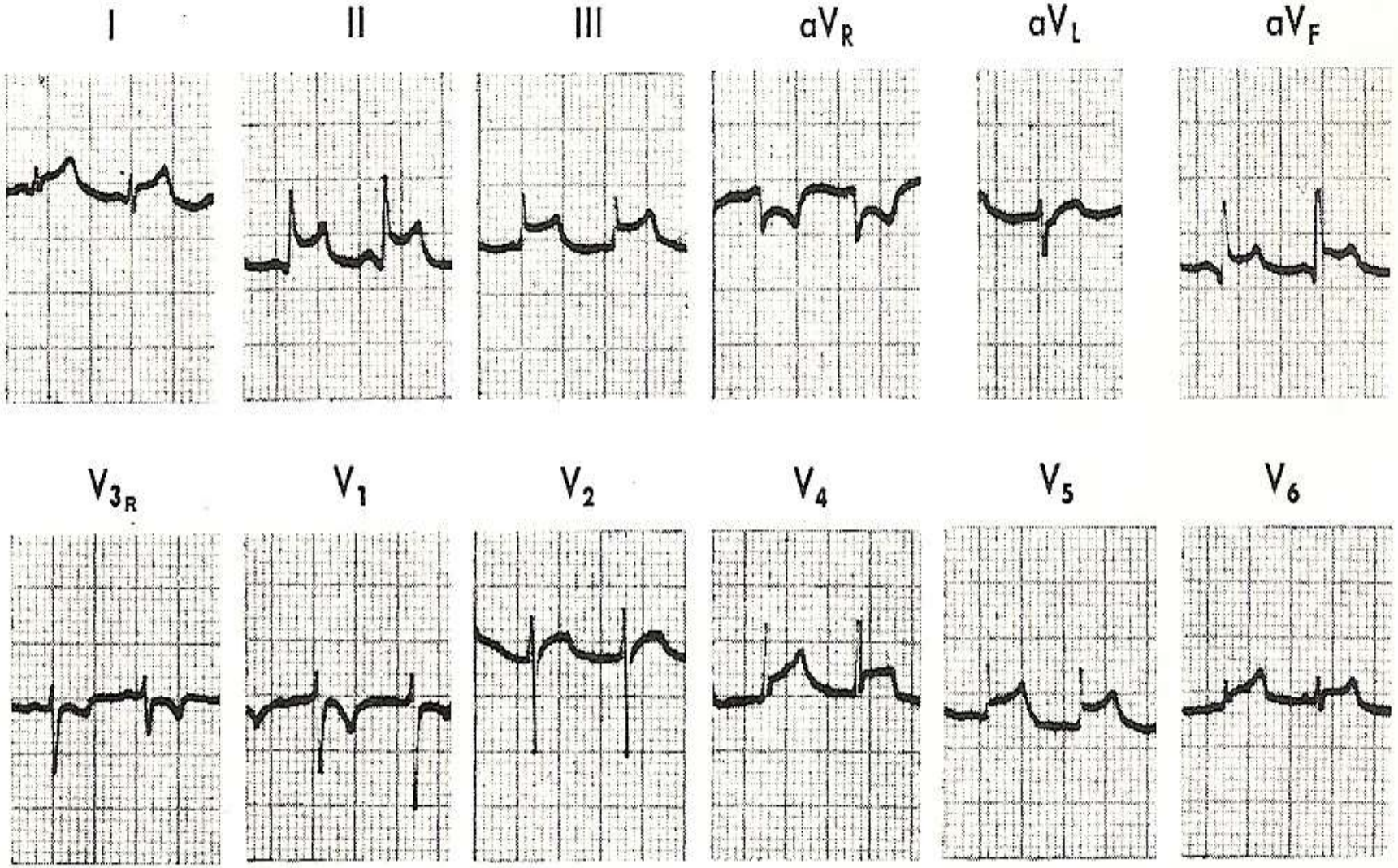
(2) Παρατηρεῖται σέ πολλές ἀπαγωγές - ὄχι σέ μιά συγκεκριμένη περιοχή.

(3) Ἑπομένως δέν ὑπάρχει καί "εἰκόνα κατόπτρου" (δ.δ. ἀπό ἔμφραγμα).

(4) Τό T εἶναι θετικό (στή χρονία φάση ἀναστρέφεται).

(5) Κατάσπαση τοῦ PQ δυνατόν νά ὑπάρχει.

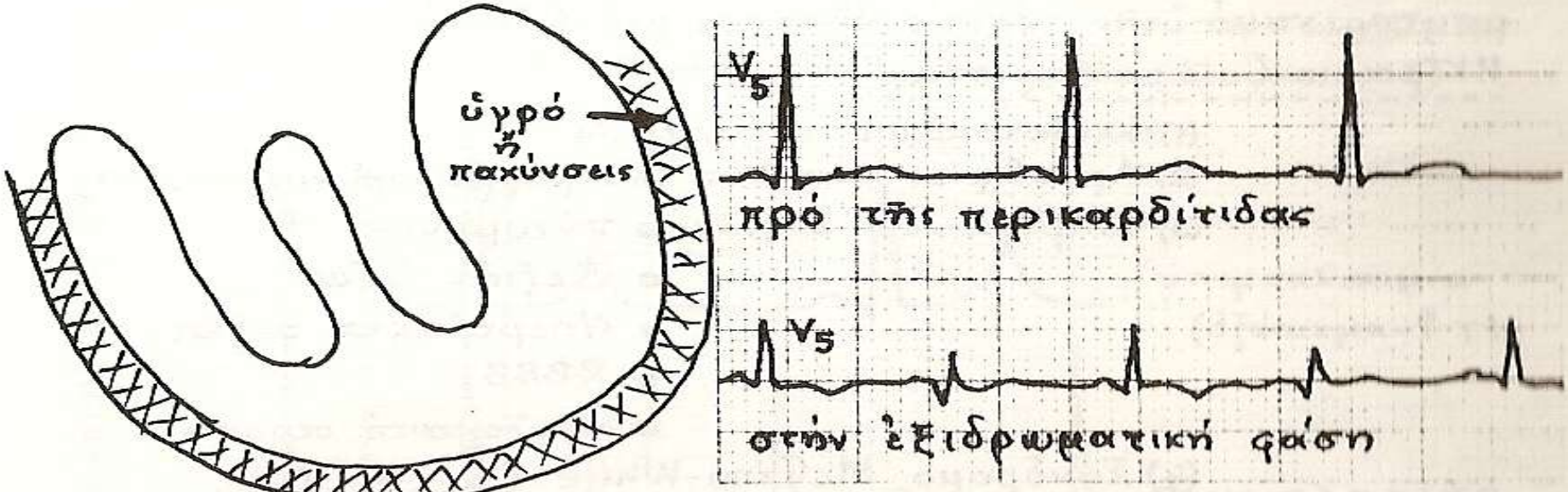





**Εικ. 10-7.** Παρατηροῦνται διάσπαρτες ἀνυψώσεις τοῦ S-T στίς ἀπαγωγές I, II, III, aVF καί $V_4$ ὡς $V_6$. Στήν ἀπαγωγή aVR παρατηρεῖται ἀντίστροφη πτώση τοῦ S-T.

# ΕΞΙΔΡΩΜΑΤΙΚΗ ΠΕΡΙΚΑΡΔΙΤΙΔΑ

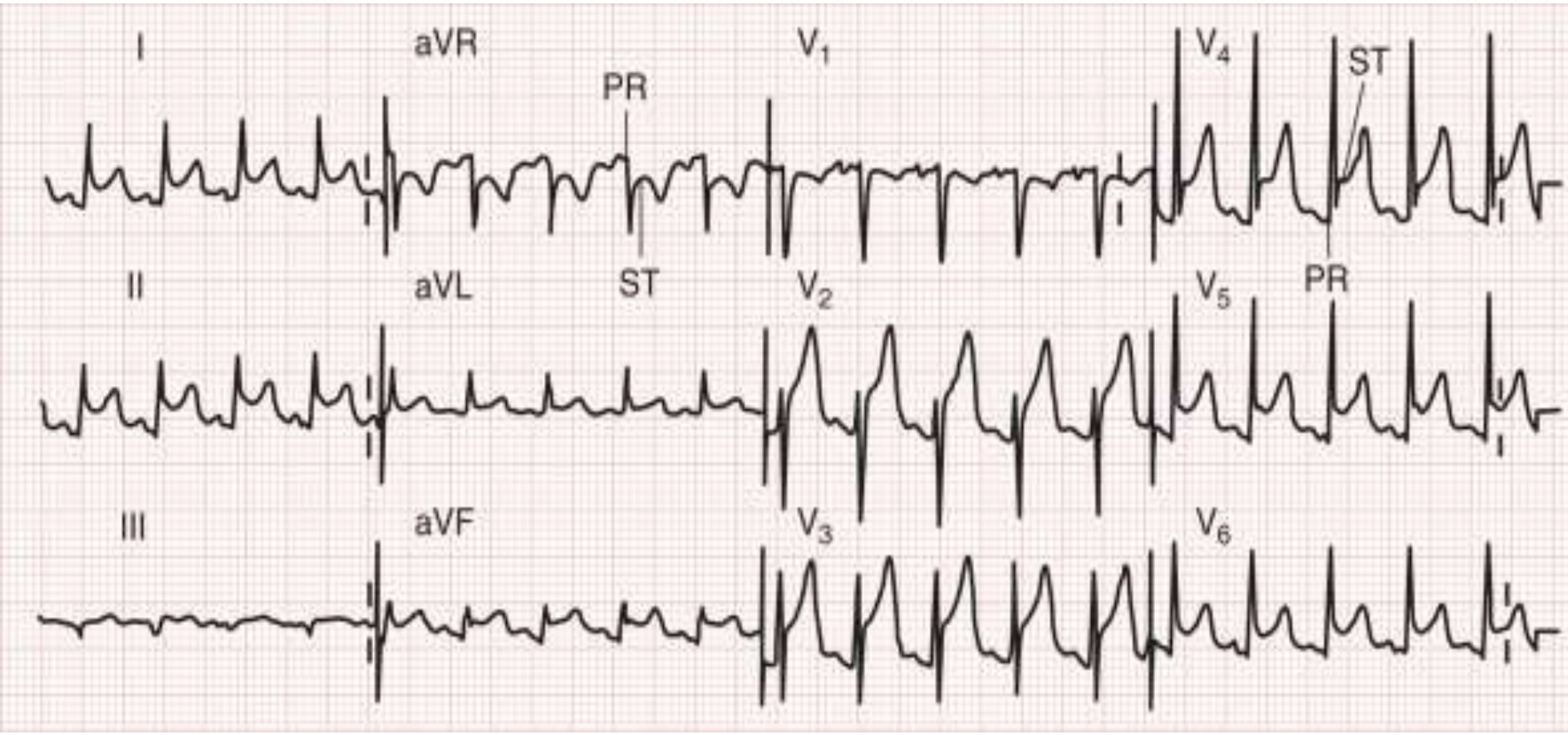
Μετά τήν ὀξεία φλεγμονή συλλέγεται ὑγρό ἐντός τοῦ περικαρδίου ἤ ἀναπτύσσονται παχύνσεις, πού μειώνουν τό ὕψος τῶν δυναμικῶν. Ἡ καρδιά τώρα "κολυμπάει" μέσα στό ὑγρό καί ἀνά δεύτερη συστολή δυνατόν νά ἀλλάζει ἠλεκτρικό ἄξονα τοῦ QRS.

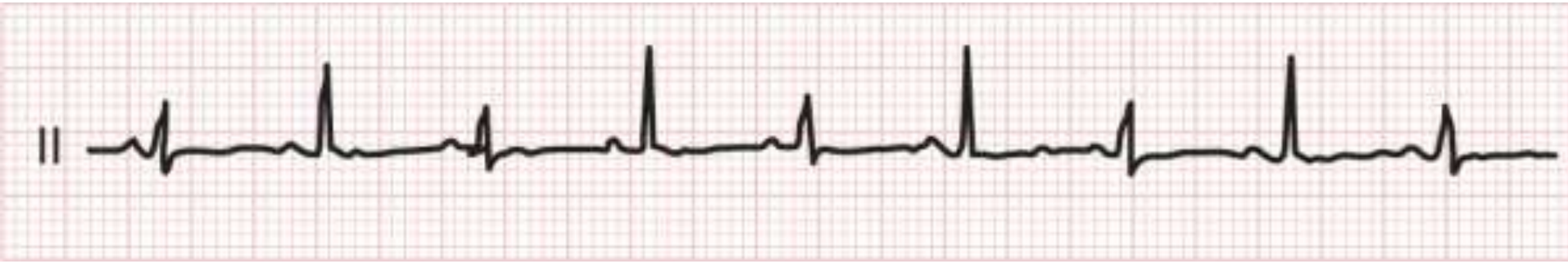



ΗΚΓραφικά χαρακτηριστικά:

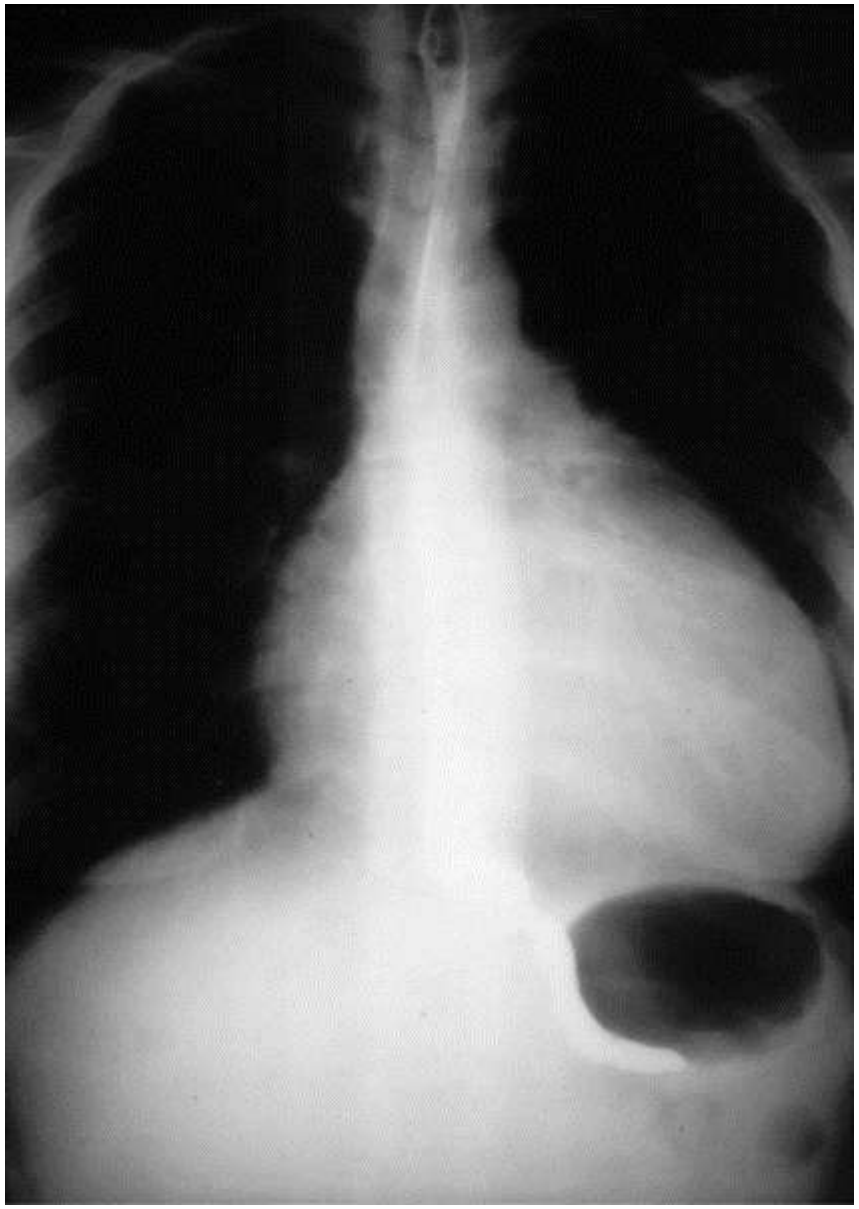
(1) χαμηλά δυναμικά (λόγω ὑγροῦ)
(2) ἐπιπεδωμένα ἤ ἀρνητικά Τ
(3) ἠλεκτρική ἐναλλαγή

Παρόμοια εἰκόνα δίνει καί ὁ ὑποθυρεοειδισμός.


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


II

# Large pericardial effusion



## Καρδιακός Επιπωματισμός

- ↓ Αρτηριακής πίεσης
- Διατεταμένες φλέβες τραχήλου
- Βύθειοι καρδιακοί τόνοι
- Παράδοξος σφυγμός

## Συμπιεστική Περικαρδίτιδα

- Σημείο Kussmaul

| Characteristic | Cardiac Tamponade | Constrictive Pericarditis |
|---|---|---|
| **CLINICAL** | | |
| Pulsus paradoxus | + | +/− |
| Kussmaul's sign | − | + |
| **ELECTROCARDIOGRAPHY** | | |
| Low voltage | + | + |
| Abnormal P waves | − | + |
| Electrical alternans | + | − |
| **CHEST RADIOGRAPHY** | | |
| Cardiomegaly | + | − |
| Pericardial calcification | − | + |
| **ECHOCARDIOGRAPHY** | | |
| Pericardial effusion | + | − |
| Pericardial thickening | − | + |

# TREATMENT OF PERICARDITIS

In the absence of significant pericardial effusion, treatment that is directed primarily at relieving the patient's symptoms can be successful in 85% or so of cases on an outpatient basis.

Among nonsteroidal anti-inflammatory drugs, indomethacin (25 to 50 mg three times daily) is commonly prescribed, but ibuprofen (300 to 800 mg three or four times a day) or aspirin (325 to 650 mg three times daily) also may be used.

Glucocorticoids (prednisone, 20 to 60 mg/day) may be useful for resistant situations.

Anti-inflammatory drugs should be continued at a constant high dose until the patient is afebrile and asymptomatic for 5 to 7 days, followed by a gradual taper during the next several weeks.

For patients with a first episode of viral or idiopathic pericarditis, colchicine (0.6 to 1.2 mg/day for 3 to 12 months) reduces the recurrence rate from about 32% to about 11%.

# TREATMENT OF PERICARDITIS

Viral and idiopathic pericarditis usually is self-limited, but a quarter of patients may have recurrent pericarditis. For this group, prolonged treatment with nonsteroidal anti-inflammatory drugs (e.g., ibuprofen, 300 to 600 mg three times a day) plus colchicine (0.6 mg twice daily, declining to once daily after a year) should be considered.

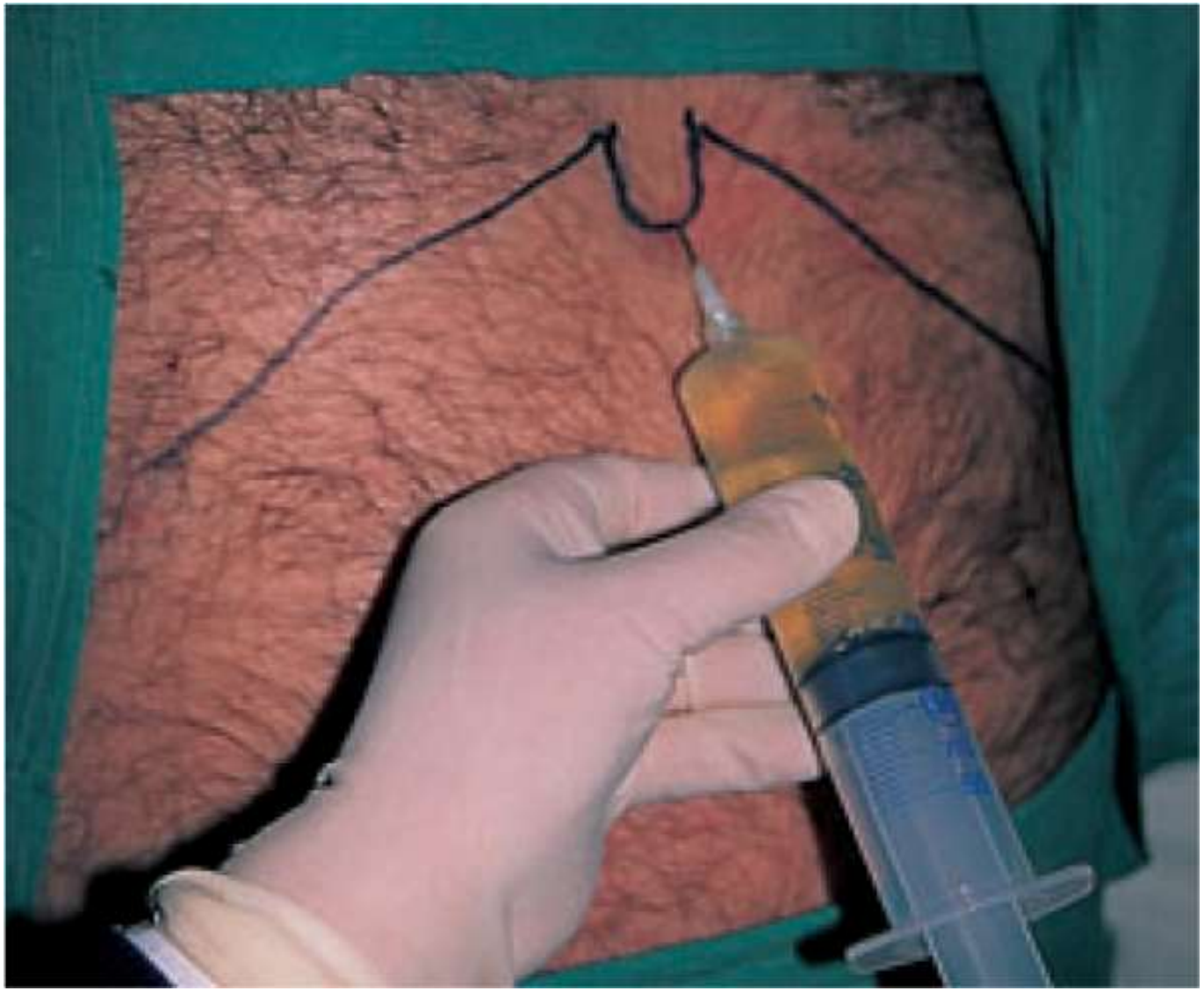
For the 10 to 14% of patients who are intolerant of colchicine and have recurrent episodes despite high-dose nonsteroidal anti-inflammatory drugs (e.g., indomethacin, 50 mg three times a day, or ibuprofen, 800 mg four times a day), oral steroids (e.g., prednisone, 60 mg with a 2- to 4-week taper) and pericardiectomy should be considered.

Patients with recurrent pericarditis are at increased risk for progression to constrictive pericarditis.

# TREATMENT OF PERICARDITIS

When tamponade is suggested clinically and confirmed on echocardiography, acute management includes maintenance of systolic blood pressure with volume resuscitation. In dire circumstances, immediate pericardiocentesis may be life-saving.

The evaluation of chronic pericarditis should exclude the possibility of tuberculosis; a tuberculin skin test, chest radiograph, and (when highly suspicious) analysis of gastric aspirates should be performed. Pericardial biopsy is more commonly diagnostic of tuberculous pericarditis than is pericardial fluid staining or culture. Aggressive drug treatment is indicated.



**Aspiration of pericardial fluid is indicated in cardiac tamponade or to obtain fluid for diagnostic purposes. A wide-bore needle is inserted in the epigastrium below the xiphoid process and advanced in the direction of the medial third of the right clavicle.**

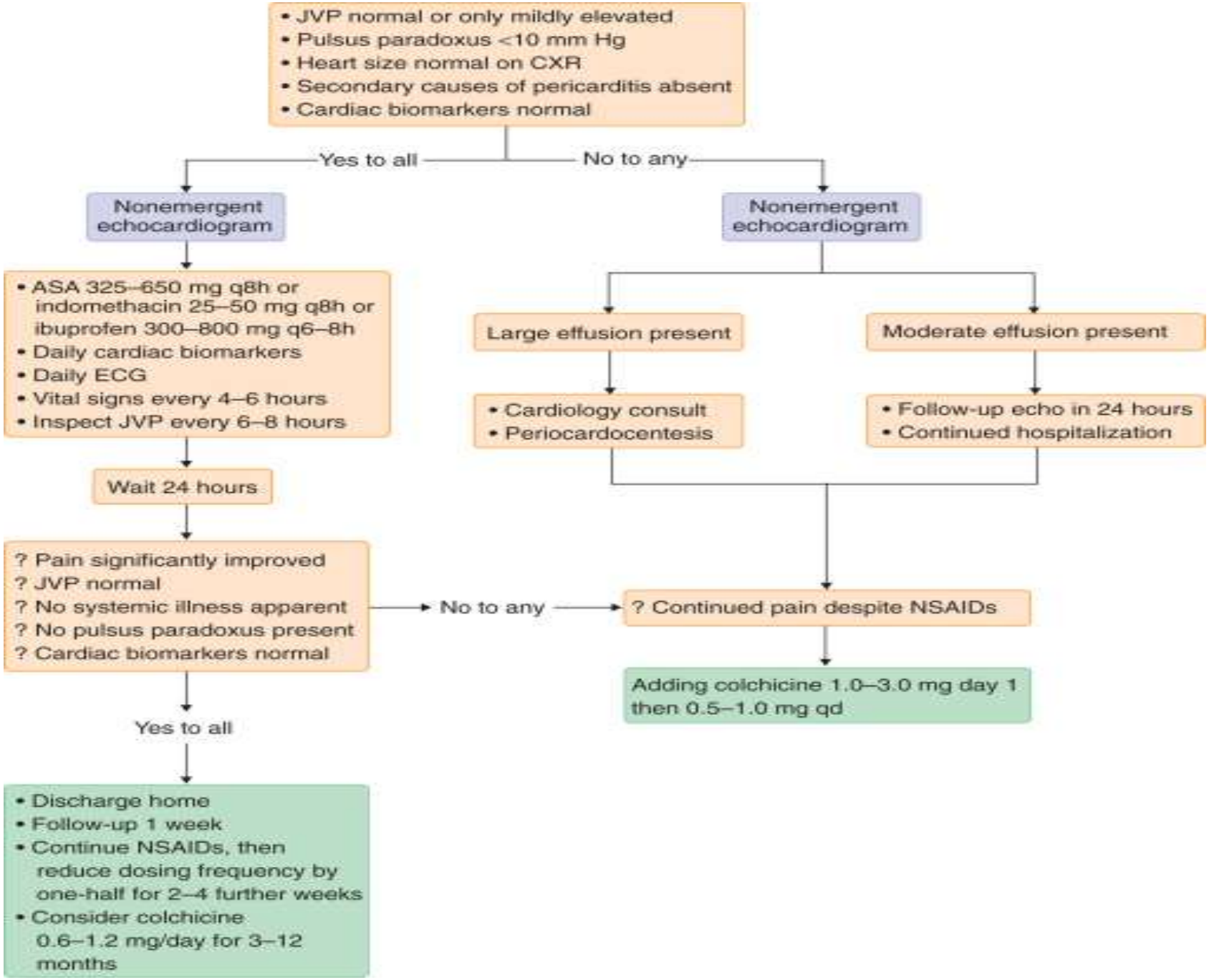
# TREATMENT OF PERICARDITIS

Hypothyroidism-myxedema is another common cause of large pericardial effusions, especially in the elderly. Measurement of TSH is diagnostic. The effusion and coexistent cardiomyopathy respond to hormone replacement, but sometimes slowly during several months.

Uremic pericardial effusions also are common and often respond to initiation of or more intensive dialysis.

Treatment of chronic or recurrent idiopathic effusions is similar to the treatment of recurrent pericarditis.

If medical therapy is unsuccessful, creation of a pericardial window is indicated.


• JVP normal or only mildly elevated
• Pulsus paradoxus <10 mm Hg
• Heart size normal on CXR
• Secondary causes of pericarditis absent
• Cardiac biomarkers normal
Yes to all
No to any
Nonemergent echocardiogram
Nonemergent echocardiogram
• ASA 325–650 mg q8h or indomethacin 25–50 mg q8h or ibuprofen 300–800 mg q6–8h
• Daily cardiac biomarkers
• Daily ECG
• Vital signs every 4–6 hours
• Inspect JVP every 6–8 hours
Large effusion present
Moderate effusion present
• Cardiology consult
• Periocardocentesis
• Follow-up echo in 24 hours
• Continued hospitalization
Wait 24 hours
? Pain significantly improved
? JVP normal
? No systemic illness apparent
? No pulsus paradoxus present
? Cardiac biomarkers normal
No to any
? Continued pain despite NSAIDs
Adding colchicine 1.0–3.0 mg day 1 then 0.5–1.0 mg qd
Yes to all
• Discharge home
• Follow-up 1 week
• Continue NSAIDs, then reduce dosing frequency by one-half for 2–4 further weeks
• Consider colchicine 0.6–1.2 mg/day for 3–12 months

ΕΥΧΑΡΙΣΤΩ