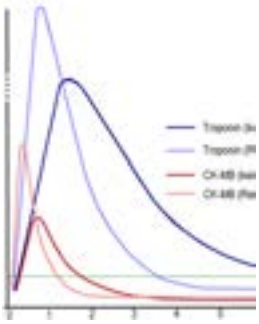

Das akute Koronarsyndrom -was tun?

Stefan Harb
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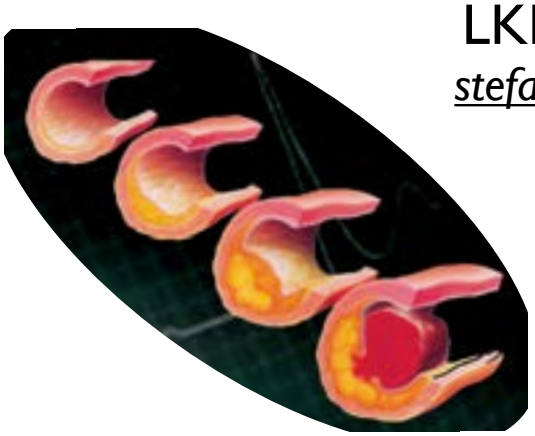


Überblick

Symptom Angina pectoris
DD
Statistiken
ACS-Definition
EKG
STEMI Erstversorgung/Logistik
Lyse/PCI
Troponin
NSTEMI/IAP
Risikoabschätzung
Pathophysiologie Koronararterie
4 Fallbeispiele im Katheterlabor
Rehabilitation/Prävention

William Heberden's classic description of angina pectoris was first presented to the Royal College of Physicians in 1768. It was published in 1772, in the Medical Transactions of the College. Many aspects of his description are true to this day. He describes both typical exertional angina as well as variant angina which eventually affected a patient only when he was in bed and was relieved by sitting up. He also points out the influence of mental stress. Although it is a classic, it is not the first description of angina. A case was described in 1632 in the memoirs of the Earl of Clarendon.

Pectoris Dolor

published in:
Commentaries on the History and Cure of Diseases
By William Heberden, M.D.
London (1802)
reprinted from:
Heberden W. Some account of a disorder of the breast.
Medical Transactions 2, 59-67 (1772) London: Royal College of Physicians.

Besides the asthma, hysteric oppressions the acute darting pains, in pleurisies, and the chronical ones in consumptions, the breast is often the seat of pains, which are distressing, sometimes even from their vehemence, oftener from their duration as they have continued to tease the patient for six for eight, for nine, and for fourteen years. There have been several examples of their returning periodically every night, or alternately with a headache. They have been called gouty, and rheumatic and spasmodic. There has appeared no reason to judge that they proceed from any cause of much importance to health (being attended with no fever,) or that they lead to any dangerous consequences and if the patient were not uneasy with what he feels, he needs never to be so on account of anything which he has to fear.

If these pains should return at night and disturb the sleep, small doses of opium have been found serviceable, and may be used alone, or joined with an opening medicine, with a preparation of antimony, or with the fetid gums. Externally, a small perpetual blister applied to the breast has been successful, and so has an issue made in the thigh. A large cumin plaster has been worn over the seat of the pain with advantage. The volatile, or saponaceous liniment, may be rubbed in over the part affected. Bathing in the sea, or in any cold water, may be used at the same time.

But there is a **disorder** of the breast marked with strong and peculiar symptoms, considerable for the kind of **danger** belonging to it, and **not extremely rare**, which deserves to be mentioned more at length. **The seat of it, and sense of strangling, and anxiety with which it is attended, may make it not improperly be called angina pectoris**.

They who are afflicted with it, are seized while they are **walking**, (more especially if it be **up hill, and soon after eating**) with a **painful and most disagreeable sensation in the breast**, which **seems as if it would extinguish life**, if it were to increase or continue; **but the moment they stand still, all this uneasiness vanishes**.

In all other respects, the patients are, at the beginning of this disorder, perfectly well, and in particular have no shortness of breath, from which it is totally different. The pain is sometimes situated in the upper part, sometimes in the middle, sometimes at the bottom of the **os sterni**, and often more inclined **to the left** than to the right side. It likewise very frequently extends **from the breast to the middle of the left arm**. The pulse is, at least sometimes, not disturbed by this pain, as I have had opportunities of observing by feeling the pulse during the paroxysm. **Males** are most liable to that disease, especially such as have **passed their fiftieth year**.

After it has continued a year or more, it will not cease so instantaneously upon standing still; and **it will come** on not only when the persons are walking, but **when they are lying down**, especially if they lie on their left side, and oblige them to rise up out of their beds. In some inveterate cases it has been brought on by the motion of a horse, or a carriage, and even by swallowing, coughing, going to stool, or speaking, or any disturbance of mind.

> Such is the most usual appearance of this disease; but some varieties may be met with. Some have been seized while they were standing still or sitting; also upon first waking out of sleep: and the pain sometimes reaches to the right arm, as well as to the left, and even down to the hands, but this is uncommon: in a very few instances the arm has at the same time been numbed and swelled. In one of two persons the pain has lasted some hours, or even days; but this has happened when the complaint has been of long standing, and thoroughly rooted in the constitution: once only the very first attack continued the whole night.

I have seen nearly a hundred people under this disorder, of which number there have been three women, and one boy twelve years old. All the rest were men near, or past the fiftieth year of their age.

Persons who have persevered in walking till the pain has returned four or five times, have then sometimes **vomited**.

A man in the sixtieth year of his life began to feel, while he was walking, an uneasy sensation in his **left arm**. He never perceived it while he was traveling in a carriage. After it had continued ten years, it would come upon him two or three times a week at night, while he was in bed, and then he was obliged to sit up for an hour or two before it would abate so much as to suffer him to lie down. In all other respects he was very healthy, and had always been a remarkably strong man. The breast was never affected. This disorder, its seat excepted, perfectly resembled the angina pectoris, gradually increasing in the same manner, and being both excited and relieved by all the same causes. **He died suddenly without a groan** at the age of seventy-five.

The termination of the angina pectoris is remarkable. For, if no accidents intervene, but the disease go on to its height, the patients **all suddenly fall down, and perish almost immediately**. Of which indeed their frequent faintnesses, and sensations as if all the powers of life were failing, afford no obscure intimation.

The angina pectoris, as far as I have been able to investigate, belongs to the class of **spasmodic**, not inflammatory complaints,

For,

In the 1st place, the access and the recess of the fit is sudden.
2dly, There are long intervals of perfect health.
3dly, Wine, and spirituous liquors, and opium afford considerable relief.
4thly, It is increased by disturbance of the mind.
5thly, It continues many years without any other injury to the health.
6thly, In the beginning it is not brought on by riding on horseback, or in a carriage, as is usual in diseases arising from scirrhus or inflammation.
7thly, During the fit the pulse is not quickened.
Lastly, Its attacks are often after the first sleep, which is a circumstance common to many spasmodic disorders.

Yet it is not to be denied that I have met with one or two patients, who have told me they now and then spit up matter and blood, and that it seemed to them to come from the seat of the disease. In another, who fell down dead without any notice, there immediately arose such as offensive smell, as made all who were present judged that some foul abscess had just then broken.

On opening the body of one who died suddenly of this disease, a very skillful anatomist could discover no fault in the heart, in the valves, in the arteries, or neighboring veins, excepting some small rudiments of **ossification in the aorta**. The brain was likewise every where sound. In this person, as it has happened to others who have died by the same disease, the **blood** continued fluid two or three days after death, not dividing itself into crassamentum and serum, but **thick, like cream**. Hence when a vein has been opened a little before death, or perhaps soon after, the blood has continued to ooze out as long as the body remained unburied.

With respect to the **treatment** of this complaint, I have little or nothing to advance: Nor indeed is it to be expected we should have made much progress in the cure of a disease, which has hitherto hardly had a place or a name in medical books. **Quiet and warmth**, and spirituous **liquors**, help restore patients who are nearly exhausted, and to dispel the effects of a fit when it does not soon go off. **Opium** taken at bed-time will prevent the attacks at night. I knew one who set himself a task of sawing wood for half an hour every day, and was nearly cured. In one also the disorder ceased of itself. Bleeding, vomiting, and purging, appear to me to be improper.

William Heberden, 21. Juli 1768:

„Es gibt eine **Störung im Thoraxbereich** - gravierend wegen der ihr eigenen potentiellen **Gefahr** - die durch **heftige und eigentümliche Symptome** gekennzeichnet und **nicht einmal extrem selten** ist.

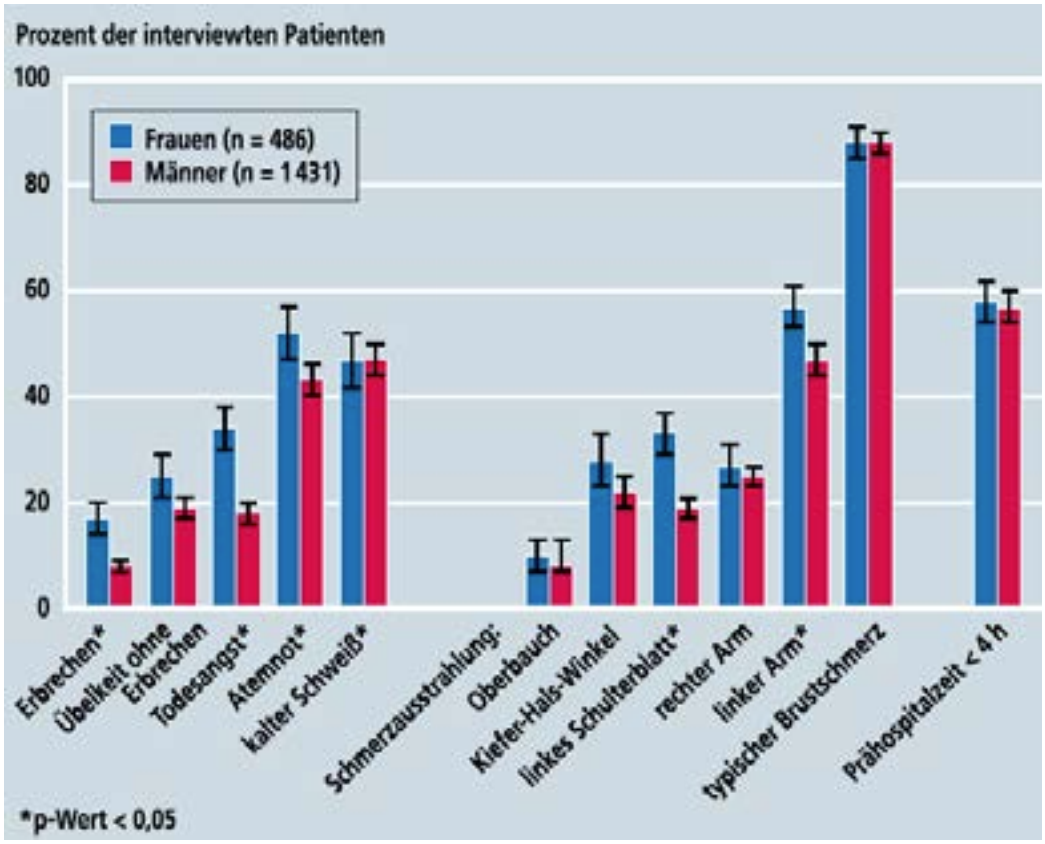
Ihr **Sitz**, das **Gefühl der Strangulation** und die **Angst**, von der dieses **Engegefühl** begleitet wird, rechtfertigen die Bezeichnung **"Angina pectoris"**.

Bei den von ihr Betroffenen kommt es zu einem Anfall beim **Gehen**, und ganz besonders dann, wenn sie **bald nach dem Essen** aufbrechen, wobei die Beschwerden mit einer schmerzhaften und höchst unangenehmen Missempfindung im Brustraum verbunden sind, die das **Leben auszulöschen** scheinen, sofern sie sich steigern oder fortsetzen würden.

In dem Augenblick, in dem die Patienten stehen bleiben, klingen alle diese Symptome ab."

- Kardiovaskuläre Erkrankungen
  - **Akutes Koronarsyndrom, KHK**
  - (Tachykarde) Rhythmusstörungen
  - Perikarditis, Myokarditis
  - Aortendissektion
  - HOCM
  - Aortenstenose
  - Hypertensive Herzkrankheit
- Pulmonale Erkrankungen
  - Lungenembolie
  - Pleuritis, Pneumonie
  - Pneumothorax
- Skeletterkrankungen
  - Rippenfrakturen, Prellungen
  - BWS-Syndrom
  - Tietze-Syndrom
- Gastrointestinale Erkrankungen
  - Ösophagitis/Ösophagusruptur
  - Ulcus
  - Akute Pankreatitis
  - Gallenkolik
- Weitere Erkrankungen
  - Herpes Zoster
  - Tumorerkrankungen des Skeletts/der Thoraxwand

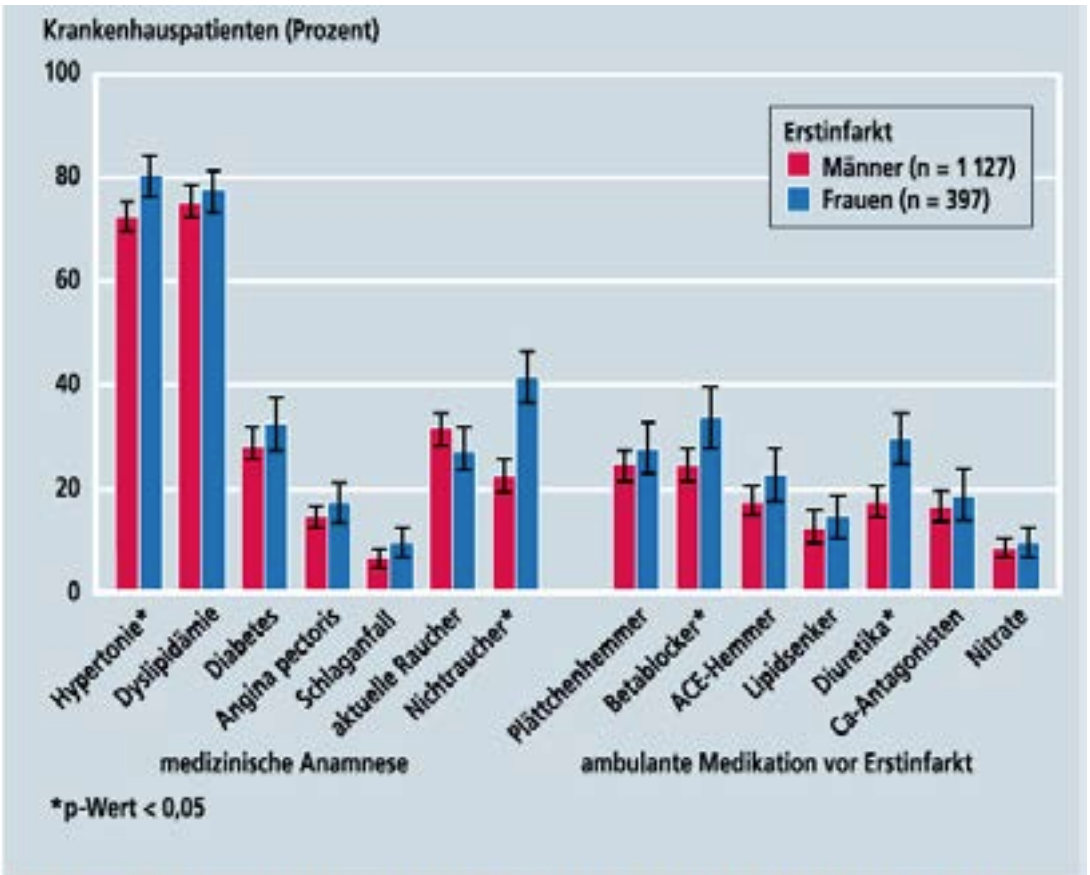
Chest-Pain-Unit!



Akute Symptomatik in Prozent (95-Prozent-Konfidenzintervalle; *p-Wert < 0,05) bei interviewten 25- bis 74-jährigen Patienten mit Erst- oder Reinfarkt nach Geschlecht, altersadjustiert. MONICA/KORA-Herzinfarktregister 2001–2003

Angina pectoris

- schwere anhaltende Schmerzen im Brustkorb
- starkes Engegefühl,heftiger Druck im Brustkorb
- blasse, fahle Gesichtsfarbe, kalter Schweiss (insb. bei Männern)
- Schmerzausstrahlung in Rücken (insb. bei Frauen), Kiefer und Bauch
- plötzliche Übelkeit, häufig mit Erbrechen (insb. bei Frauen)
- Luftnot, flache Atmung
- Schwächeanfall (auch ohne Schmerz), evtl. Bewusstlosigkeit



Medizinische Anamnese und ambulante Medikation vor dem Erstinfarkt in Prozent (95 Prozent-Konfidenzintervalle) bei mindestens 24 Stunden überlebenden Krankenhauspatienten nach Geschlecht, Alter 25–74 Jahre, altersadjustiert. MONICA/KORA-Herzinfarktregister 2001–2003

Risikofaktoren

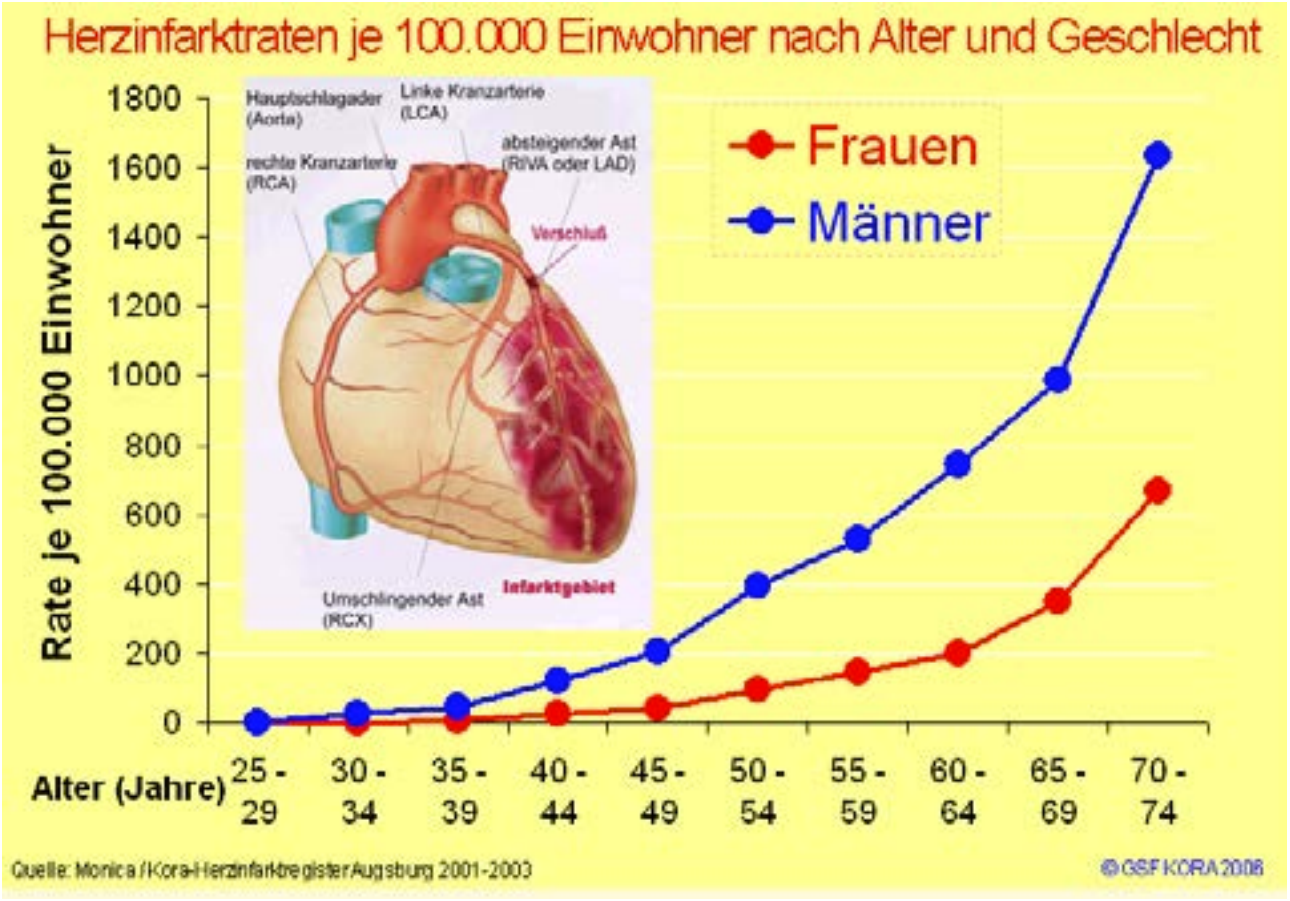
beeinflussbar

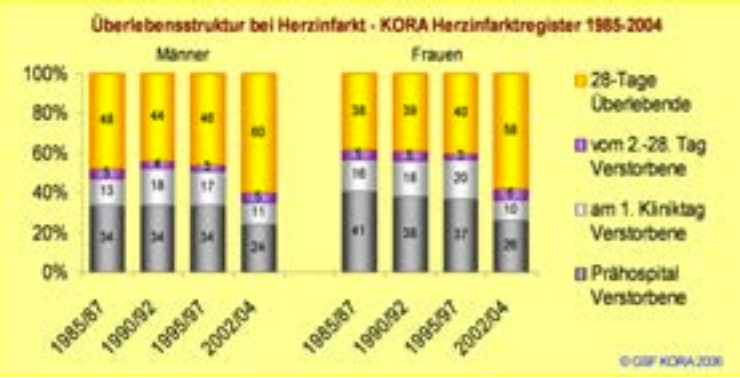
Hypertonie
HDL/LDL/TG
Rauchen
Stress
Bewegungsmangel
Übergewicht
Typ II Diabetes

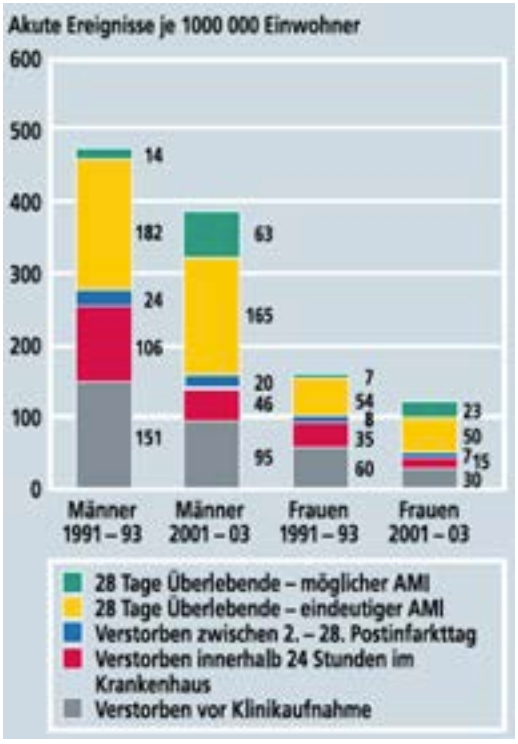
nicht beeinflussbar

Alter
Postmenopause
familiäre Belastung mit
- o KHK<55a
- o Hypertonie
- o Fettstoffwechsel

Typ I Diabetes

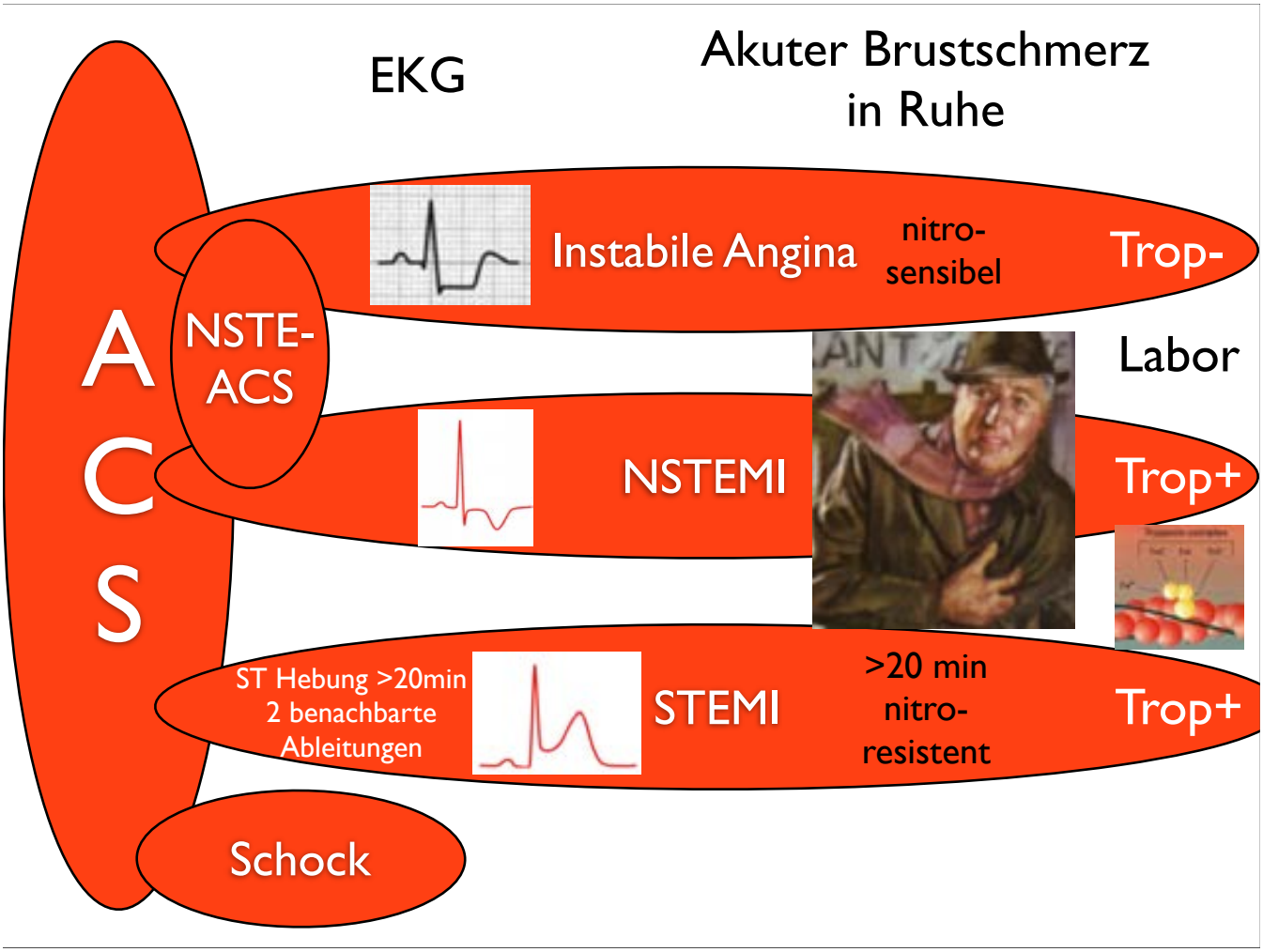




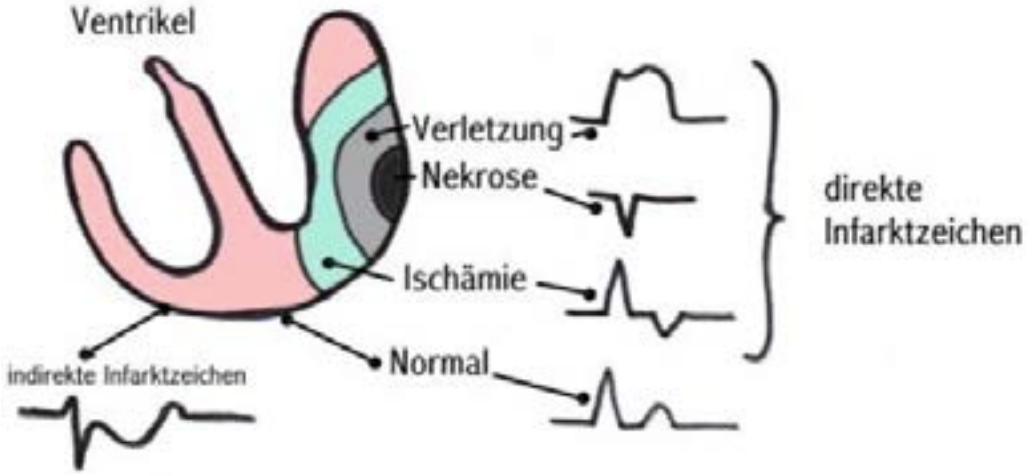


Altersstandardisierte Morbidität an akutem Myokardinfarkt (AMI; tödliche und nichttödliche AMI inklusive koronare Todesfälle) je 100 000 Einwohner nach Geschlecht und Versorgungsstadium; Alter 25–74 Jahre. MONICA/KORA-Herzinfarktregister 1991–1993 und 2001–2003

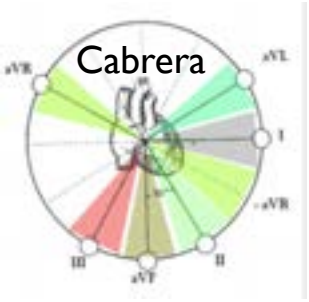
Mehr als 50% erleben den nächsten Tag nicht!

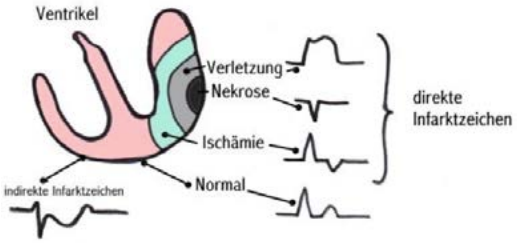


EKG bei Ischämie

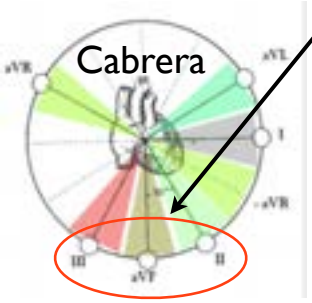


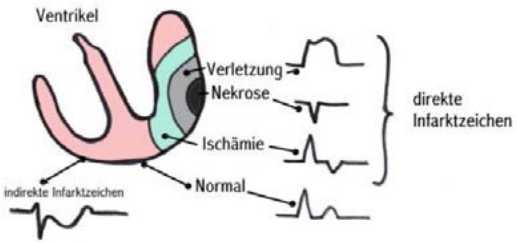
Spiegelbilder



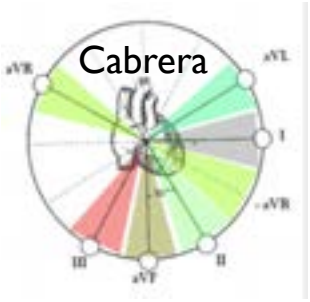


benachbarte Ableitungen
für den inferioren Infarkt

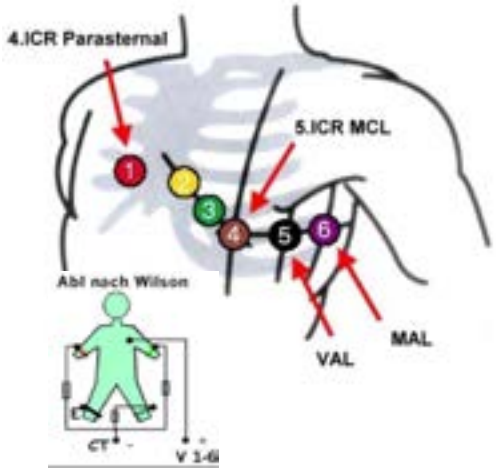


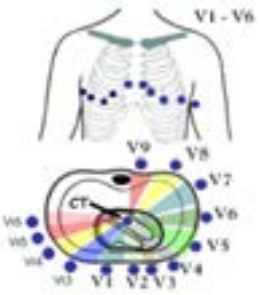


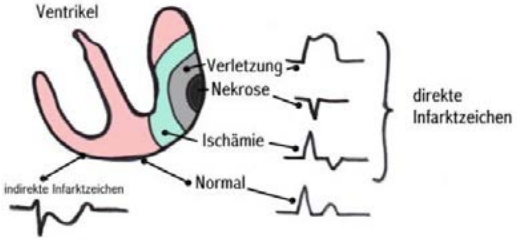

Extremitätenableitungen nach Einthoven
Abl I
Abl II
Abl III

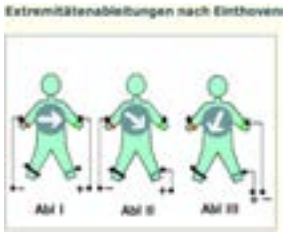

Cabrera
aVR
aVL
I
-aVR
III
aVF
II

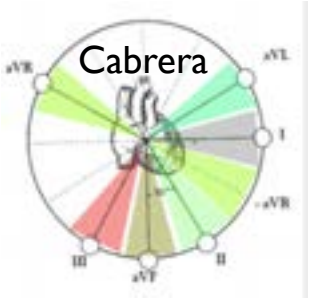

Extremitätenableitungen nach Goldberger
aVR
aVL
aVF


4.ICR Parasternal
5.ICR MCL
1
2
3
4
5
6
Abl nach Wilson
CT
V 1-6
VAL
MAL

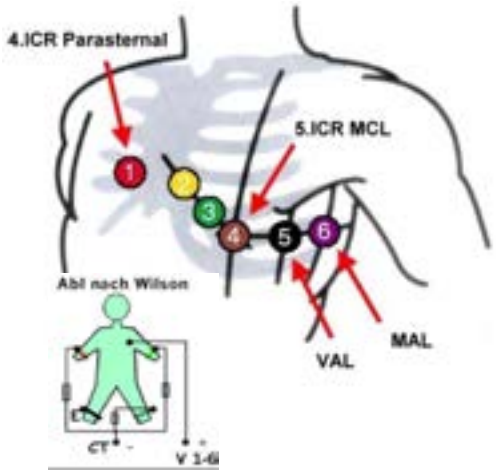

V1 - V6
V9
V8
V7
V6
V5
V4
V1
V2
V3
CT

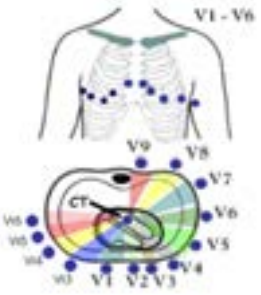

Ventrikel
Verletzung
Nekrose
Ischämie
Normal
indirekte Infarktzeichen
direkte
Infarktzeichen

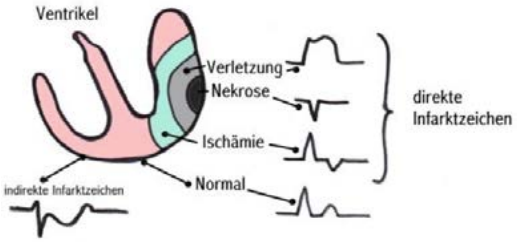

Extremitätenableitungen nach Einthoven
Abl I
Abl II
Abl III


Cabrera
aVR
aVL
I
-aVR
III
aVF
II

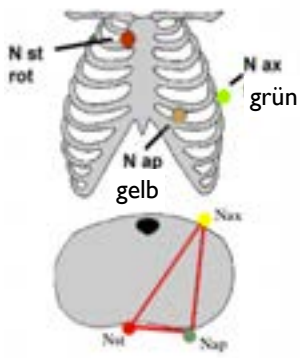

Extremitätenableitungen nach Goldberger
aVR
aVL
aVF

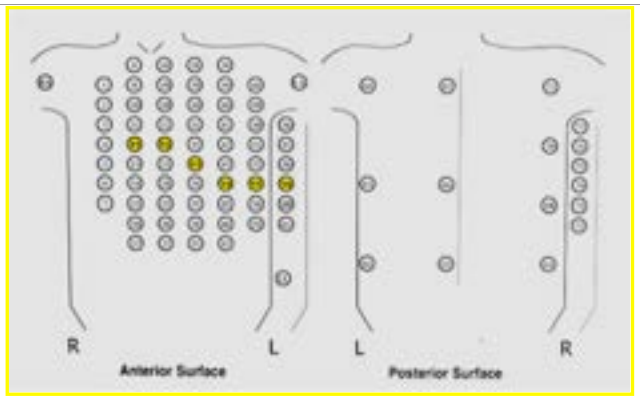

4.ICR Parasternal
5.ICR MCL
1
2
3
4
5
6
Abl nach Wilson
CT
V 1-6
VAL
MAL


V1 - V6
V9
V8
V7
V6
V5
V4
V1
V2
V3
CT


Ventrikel
Verletzung
Nekrose
Ischämie
Normal
indirekte Infarktzeichen
direkte
Infarktzeichen

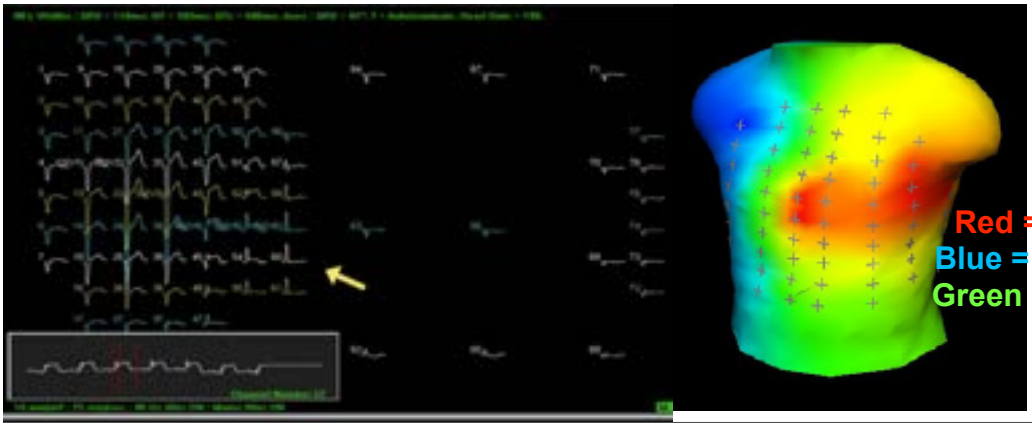
Nehb


N st
rot
N ax
grün
N ap
gelb
Nax
Nst
Nap

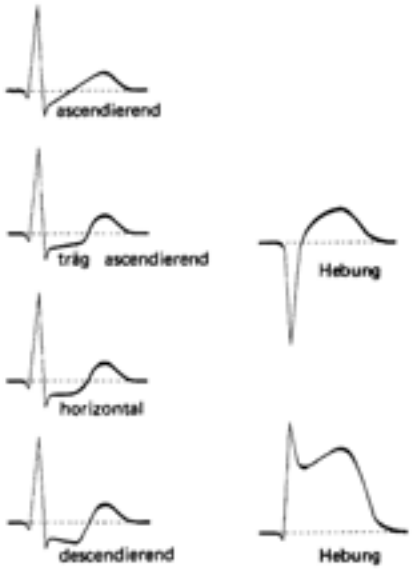


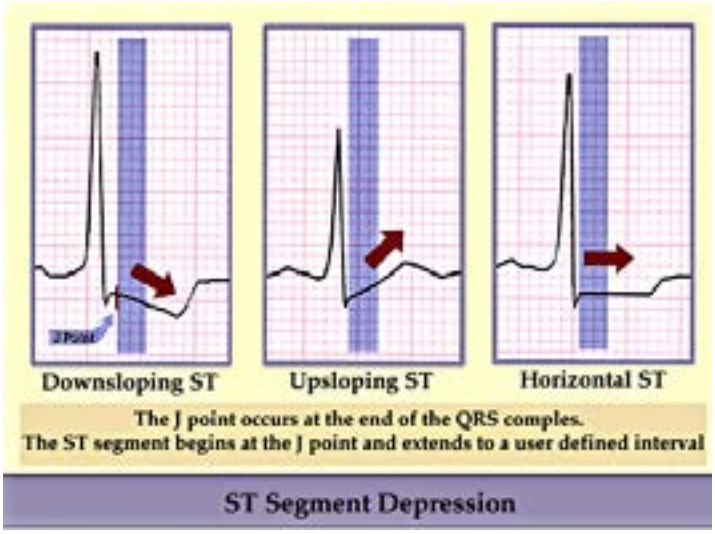
The Optimal Cardiovascular Diagnostic Evaluation
Enabling Faster Treatment of Myocardial Infarction(**OCCULT-MI**) trial

The 80-lead mapping system **detected 27.5% more patients with STEMI** than the standard 12-lead ECG.



ST-Morphologie





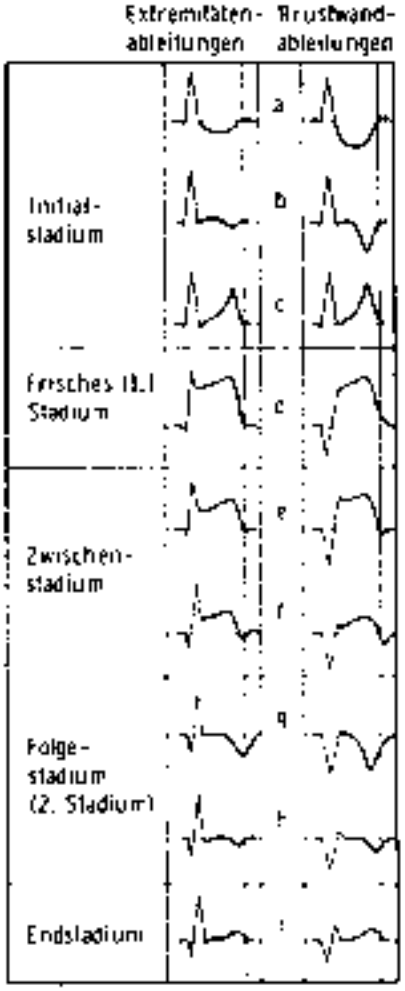
Stadium 0 =
Frühbefund kurz nach dem Infarkt
- T positiv, hoch, breit

Stadium I =
Befund nach einigen Stunden
- Q klein
- R klein
- deutliche ST-Streckenhebung
- T positiv

Stadium II =
Befund nach 1 Tag
- Q groß
- R klein
- ST-Hebung, rückläufig
- T spitz, negativ

Stadium III =
Befund nach mehreren Tagen
- Q groß
- R höher als im Stadium II
- ST-Hebung verschwunden
- T spitz, negativ

Stadium IV =
Spätbefund
- Q noch groß
- R wieder normal groß
- keine ST-Hebung
- keine ST-Senkung
- T wieder positiv



LVHT LSB Peri karditis Hyper-kaliämie VWI VWI RSB Brugada

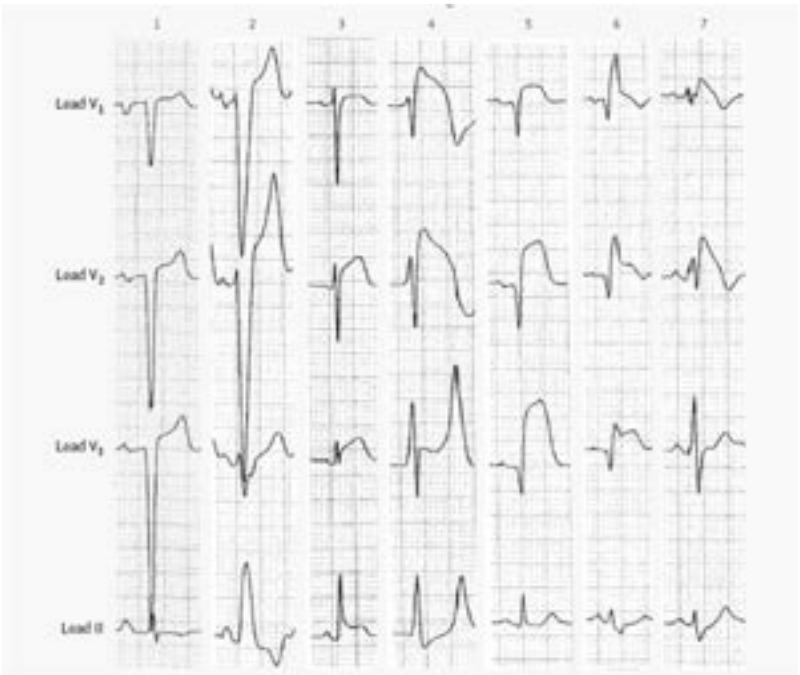


Figure 2. **Electrocardiograms Showing ST-Segment Elevation in Various Conditions.**
Tracing 1 is from a patient with left ventricular hypertrophy, and tracing 2 is from a patient with left bundle branch block. Tracing 3, from a patient with acute pericarditis, is the only tracing with ST-segment elevation in both precordial leads and lead II and PR-segment depression. Tracing 4 shows a pseudoinfarction pattern in a patient with hyperkalemia. The T wave in V_2 is tall, narrow, pointed, and tented. Tracing 5 is from a patient with acute anteroseptal infarction. The distinctive features of tracing 6, from a patient with acute anteroseptal infarction and right bundle-branch block, include the remaining R′ wave and the distinct transition between the downstroke of R′ and the beginning of the ST segment. Tracing 7, from a patient with the Brugada syndrome, shows rSR′ and ST-segment elevation limited to V_1 and V_2. The ST segment begins from the top of the R′ and is downsloping.

NEJM 349;22

Normvarianten

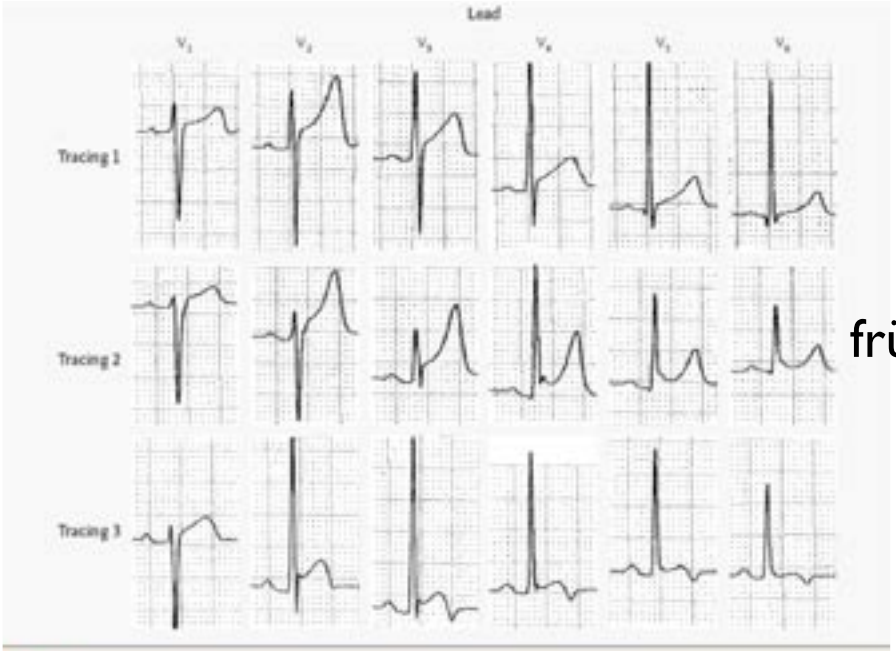


Figure 1. **Electrocardiograms Showing Normal ST-Segment Elevation and Normal Variants.**
Tracing 1 shows normal ST-segment elevation. Approximately 90 percent of healthy young men have ST-segment elevation of 1 to 3 mm in one or more precordial leads. The ST segment is concave. Tracing 2 shows the early-repolarization pattern, with a notch at the J point in V_4. The ST segment is concave, and the T waves are relatively tall. Tracing 3 shows a normal variant that is characterized by terminal T-wave inversion. The QT interval tends to be short, and the ST segment is coved.

normale konkave ST Hebung

frühe Repolarisation

terminale T-inversion

NEJM 349;22

TIMI Risk Score for STEMI

Age ≥ 65 years?	○ **< 65** +0 ○ **65-74** +2 ○ **≥ 75** +3
DM or HTN or Angina?	☐ **Yes** +1
SBP < 100 mmHg?	☐ **Yes** +3
HR > 100 bpm?	☐ **Yes** +2
Killip Class II-IV?	☐ **Yes** +2
Weight < 67 kg (147.7 lbs)?	☐ **Yes** +1
Anterior ST Elevation or LBBB?	☐ **Yes** +1
Time to Treatment > 4 hrs?	☐ **Yes** +1
Score	Click! **points**

http://www.mdcalc.com/timi-risk-score-for-stemi

		30 Tage Mortalität bei akutem MI
Killip I	keine Herzinsuffizienz	3-5%
Killip II	feinblasige RG 3.HT Jugularvenenstauung	6-10%
Killip III	Lungenödem	20-30%
Killip IV	Kardiogener Schock Zyanose Oligurie	>80%

Killip T, Kimball JT. Treatment of myocardial infarction in a coronary care unit: a two year experience of 250 patients. Am J Cardiol 1967; 20: 457-464

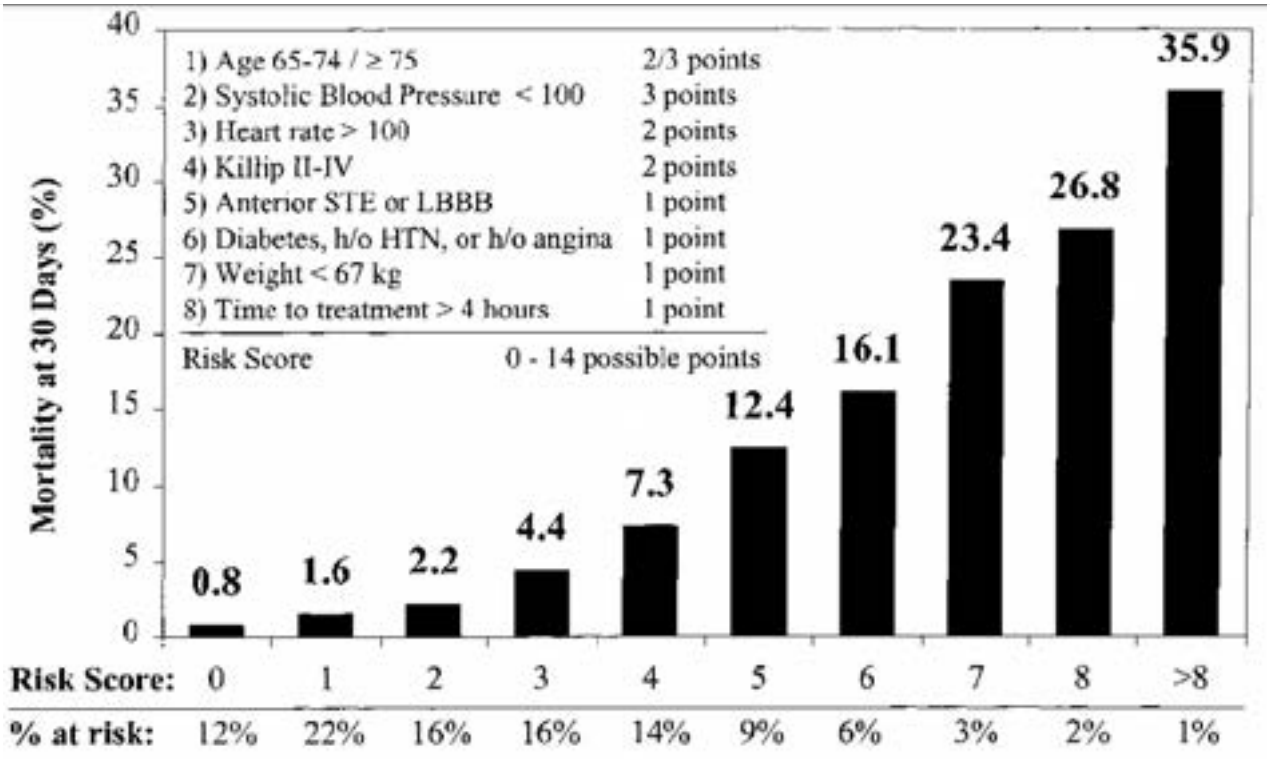


Figure 2. TIMI risk score for STEMI for predicting 30-day mortality. STE indicates ST elevation; h/o, history of.

(*Circulation*. 2000;102:2031.)

Untersuchung

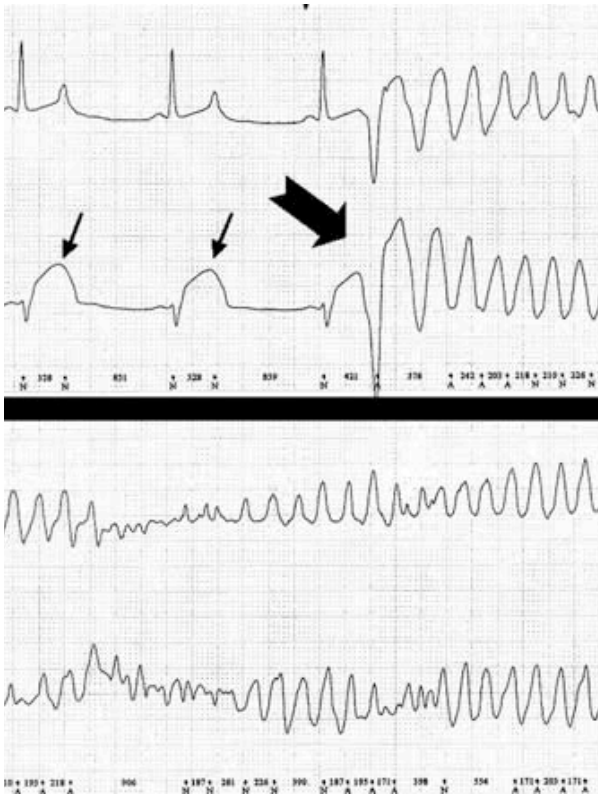
- **Palpation**
  Pulse radial/Carotiden/femoral
  Aortendissektion

  Herzspitzenstoss Perikarderguss
  Ventrikelruptur
- **Auskultation**
  Systolikum Aortenstenose/HOCM
  Mitralinsuffizienz
  Septumdefekt
  4. Herzton

Priorität der Massnahmen

- Was ist zu erwarten?
  z.B. Kammerflimmern
- Was brauche ich dann, um helfen zu können?
  EKG, Defibrillator, Intubation, Beatmung, Katecholaminperfusor
- Wie transportiere ich in welcher Zeit in welches Herzkatheterlabor?
- Ist dieses einsatzbereit?

Ischämie-Arrhythmie

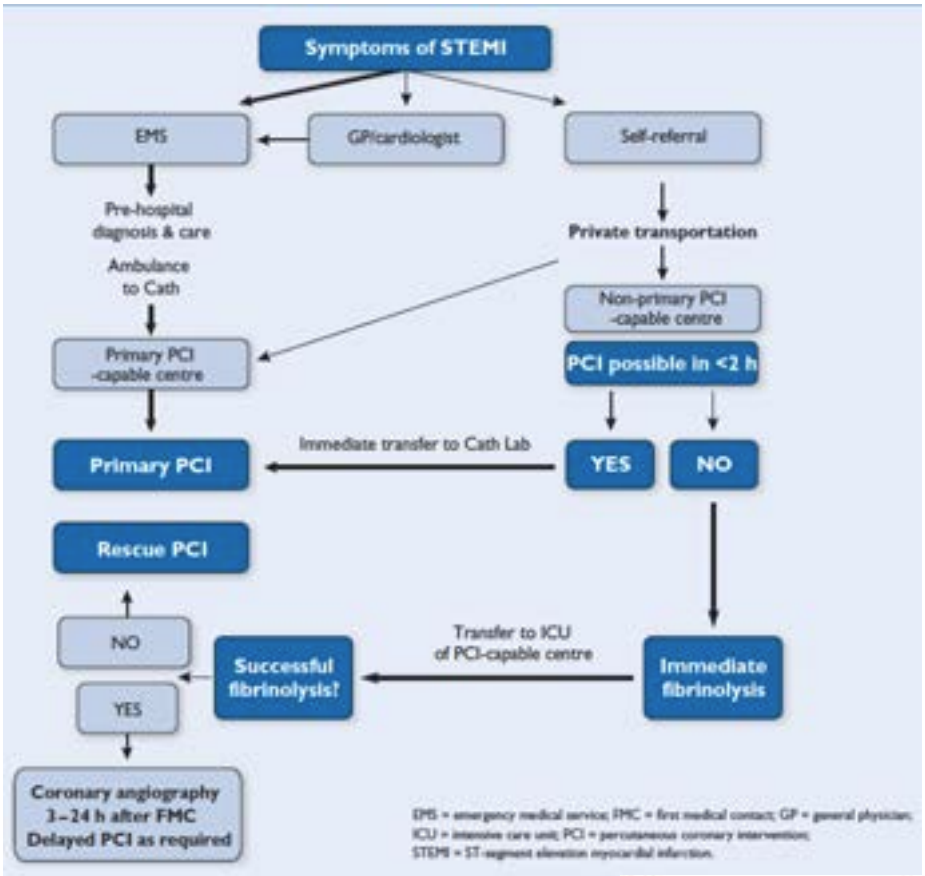


Was tun bei STEMI?
Beruhigen
bequem lagern
Nitrospray (ausser bei Schock)
Sauerstoff
ASS 250 mg
iv Leitung/Analgosedierung Morphin
Morphin/Benzodiazepin
Transport organisieren PCI/Lyse
Betablocker
UFH Bolus 4000E (60mg/kg)i.v.
Clopidogrel 600mg/Prasugrel 60mg/Ticagrelor 180mg
Aufklärung/Anamnese
Volumen/Katecholamine
Defibrillator

ZEIT = MUSKEL

Nach initialer Dysfunktion breitet sich die **Nekrose schnell von** innen nach außen aus (abhängig vom Ausmaß vorhandener Kollateralen) und **erreicht im Allgemeinen nach wenigen Stunden ihr Maximum.**

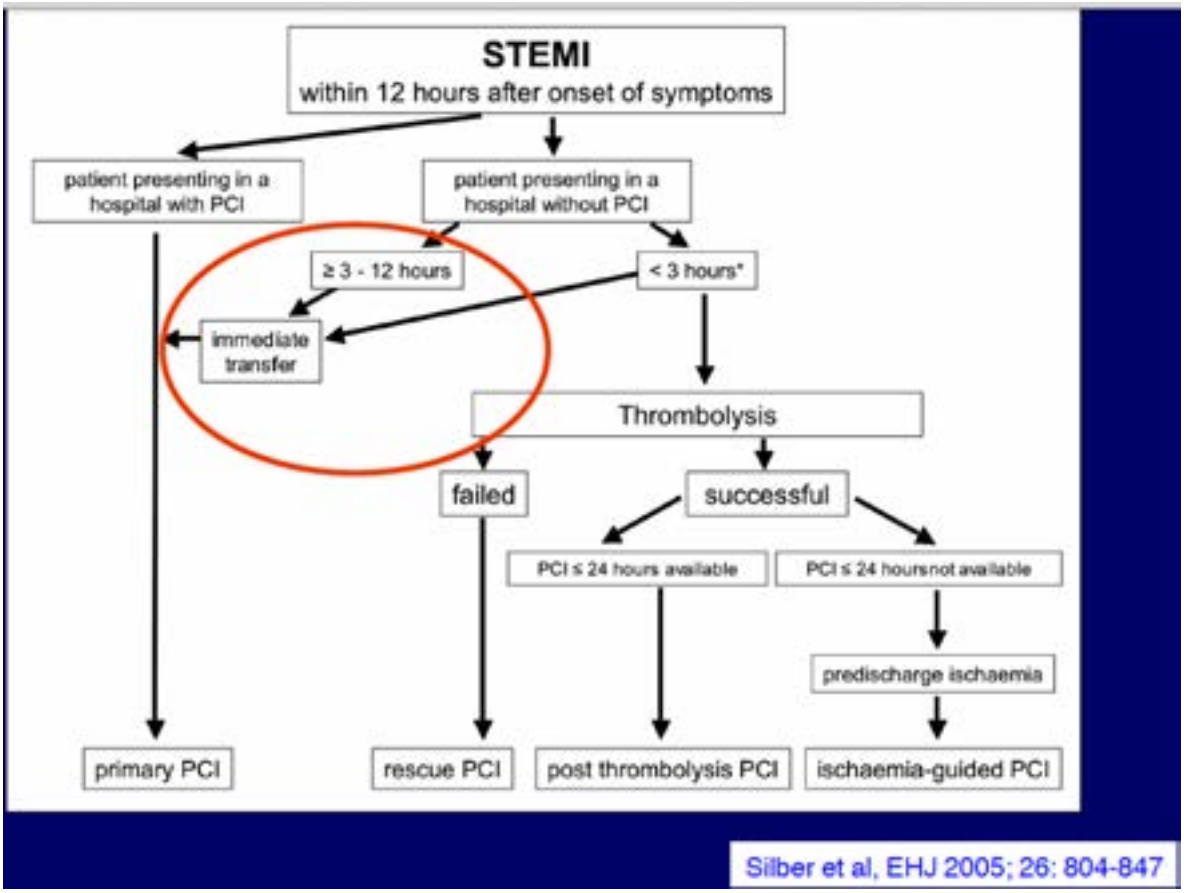
nach: Amtz, *Fibrinolyse News* 2003; 1: 5..



European Heart Journal (2010) **31**, 2501–2555
doi:10.1093/eurheartj/ehq277

ESC/EACTS GUIDELINES

Guidelines on myocardial revascularization



Aufnahmezeiten der steirischen Herzkatheterplätze

Universitätsklinik Graz:
Regeldienstzeit Mo-Fr östlich der Mur,
Nachtdienst Mo, Mi, Do, Sa und jeden 2. So

LKH Graz West:
Regeldienstzeit Mo-Fr westlich der Mur,
Nachtdienst Di, Fr und jeden 2. So

LKH Bruck: Obersteiermark,
Regeldienstzeit Mo-Fr,
Wochenenddienst Fr-So

PPCI vs. thrombolysis - ACC / AHA guidance (2004):

Fibrinolysis is generally preferred if (see Section 6.3.1.6.3.1 of the full-text guidelines):

- *Early presentation (3 hours or less from symptom onset and delay to invasive strategy; see below)*
- *Invasive strategy is not an option*
  - Catheterization laboratory occupied/not available
  - Vascular access difficulties
  - Lack of access to a skilled PCI laboratory†‡
- *Delay to invasive strategy*
  - Prolonged transport
  - (Door-to-Balloon) – (Door-to-Needle) time is greater than 1 hour*§
  - Medical contact–to-balloon or door-to-balloon time is greater than 90 minutes

An invasive strategy is generally preferred if (see Section 6.3.1.6.4.2 of the full-text guidelines):

- *Skilled PCI laboratory available with surgical backup†‡*
  - Medical contact–to-balloon or door-to-balloon time less than 90 minutes
  - (Door-to-Balloon) – (Door-to-Needle) is less than 1 hour*
- *High risk from STEMI*
  - Cardiogenic shock
  - Killip class greater than or equal to 3
- *Contraindications to fibrinolysis, including increased risk of bleeding and ICH*
- *Late presentation*
  - Symptom onset was more than 3 hours ago
- *Diagnosis of STEMI is in doubt*

"If presentation is less than 3 hours and there is no delay to an invasive strategy, there is no preference for either strategy"

…similar ESC Guidance 2005

Lysetherapie
Absolute Kontraindikationen

Zn Hirnblutung
RR > 200/110
OAK
rezente Ulcusblutung
Gravidität

kgKG	Tenecteplase (Metalyse) U
<60	6000
60-70	7000
70-80	8000
80-90	9000
>90	10.000

4.3 Gegenanzeigen

Metalyse darf nicht bei Patienten angewendet werden, die in der Anamnese anaphylaktische (d. h. lebensbedrohliche) Reaktionen gegen einen der Inhaltsstoffe (z. B. Tenecteplase oder einen der Bestandteile) oder Gentamicin (Spurenrückstand aus dem Herstellungsprozess) gezeigt haben. Wird die Behandlung mit Metalyse dennoch als notwendig erachtet, sollte für den Notfall die Ausstattung zur sofortigen Reanimation bereit stehen.

Da eine thrombolytische Therapie das Blutungsrisiko erhöht, darf Metalyse außerdem in folgenden Situationen nicht angewendet werden:

- Schwerwiegende Blutung (akut oder innerhalb der vergangenen 6 Monate)
- Patienten unter oraler Antikoagulanzientherapie (INR >1,3)
- Jede Erkrankung des zentralen Nervensystems (z.B. Neoplasma, Aneurysma, intrakranielle oder intraspinale Operation) in der Anamnese
- Bekannte hämorrhagische Diathese
- Schwere, nicht kontrollierbare Hypertonie
- Große Operation, Biopsie eines parenchymatösen Organs oder schweres Trauma in den letzten zwei Monaten (einschließlich jeglicher mit dem akuten Herzinfarkt zusammenhängender Traumen)
- Kürzlich erlittene Kopf- oder Schädelverletzungen
- Längerandauernde Wiederbelebungsmaßnahmen (>2 Minuten) in den letzten zwei Wochen
- Akute Perikarditis und/oder subakute bakterielle Endokarditis
- Akute Pankreatitis
- Schwere Leberfunktionsstörung einschließlich Leberversagen, Zirrhose, Pfortaderhochdruck (Ösophagusvarizen) und aktiver Hepatitis
- Aktive peptische Ulzera
- Arterielles Aneurysma und bekannte arteriovenöse Missbildungen
- Neoplasma mit erhöhtem Blutungsrisiko
- Hämorrhagischer Schlaganfall oder Schlaganfall unklarer Genese in der Anamnese
- Bekannter ischämischer Schlaganfall oder transiente ischämische Attacke in den vergangenen 6 Monaten
- Demenz

Lyseschema Alteplase (Actilyse)

Herzinfarkt

a) 90 Minuten (= akzeleriertes)-Infusionsschema für Patienten innerhalb von 6 Stunden nach Beginn der Symptome:

	Alteplase Konzentration	
	1 mg/ml	2 mg/ml
	ml	ml
15 mg als intravenöser Bolus	15	7,5
50 mg als intravenöse Infusion in den folgenden 30 Minuten	50	25
gefolgt von einer Infusion von 35 mg über 60 Minuten, bis zur Maximaldosis von 100 mg	35	17,5

Bei Patienten mit einem Körpergewicht unter 65 kg sollte eine gewichtsbezogene Dosierung erfolgen:

	Alteplase Konzentration	
	1 mg/ml	2 mg/ml
	ml	ml
15 mg als intravenöser Bolus	15	7,5
	ml/kg KG	ml/kg KG
0,75 mg/kg Körpergewicht (KG) in den folgenden 30 Minuten (maximal 50 mg)	0,75	0,375
gefolgt von einer Infusion von 0,5 mg/kg Körpergewicht (KG) über 60 Minuten (maximal 35 mg)	0,5	0,25

b) 3 Stunden-Infusionsschema für Patienten innerhalb von 6 bis 12 Stunden nach Beginn der Symptome:

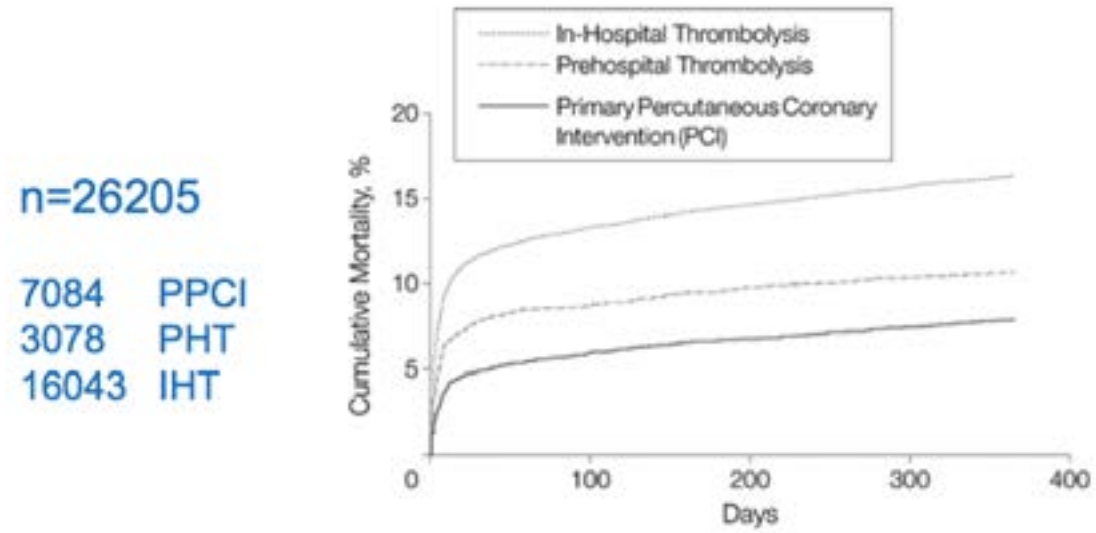
	Alteplase Konzentration	
	1 mg/ml	2 mg/ml
	ml	ml
10 mg als intravenöser Bolus	10	5
50 mg als intravenöse Infusion in der folgenden Stunde	50	25
	ml/30 min	ml/30 min
gefolgt von Infusionen von 10 mg pro 30 Minuten über 2 Stunden, bis zur Maximaldosis von insgesamt 100 mg in 3 Stunden	10	5

Bei Patienten mit einem Körpergewicht unter 65 kg sollte eine Gesamtdosis von 1,5 mg/kg nicht überschritten werden.
Eine Dosis von mehr als 100 mg Alteplase sollte nicht verabreicht werden.

Begleittherapie:

Nach der Gabe von Actilyse sollte eine intravenöse Heparinisierung begonnen (oder wieder aufgenommen) werden, sobald die Werte der aPTT unterhalb des 2fachen des oberen Normalwertes liegen. Die Infusion sollte auf einen aPTT-Wert von 50 – 70 Sekunden (Verlängerung des Ausgangswertes um das 1,5 - 2,5fache) eingestellt werden.

Zur Vermeidung einer Rebound-Thrombose ist anschliessend die aPTT mit Heparin zwischen 50-70 Sek. zu halten!



n=26205

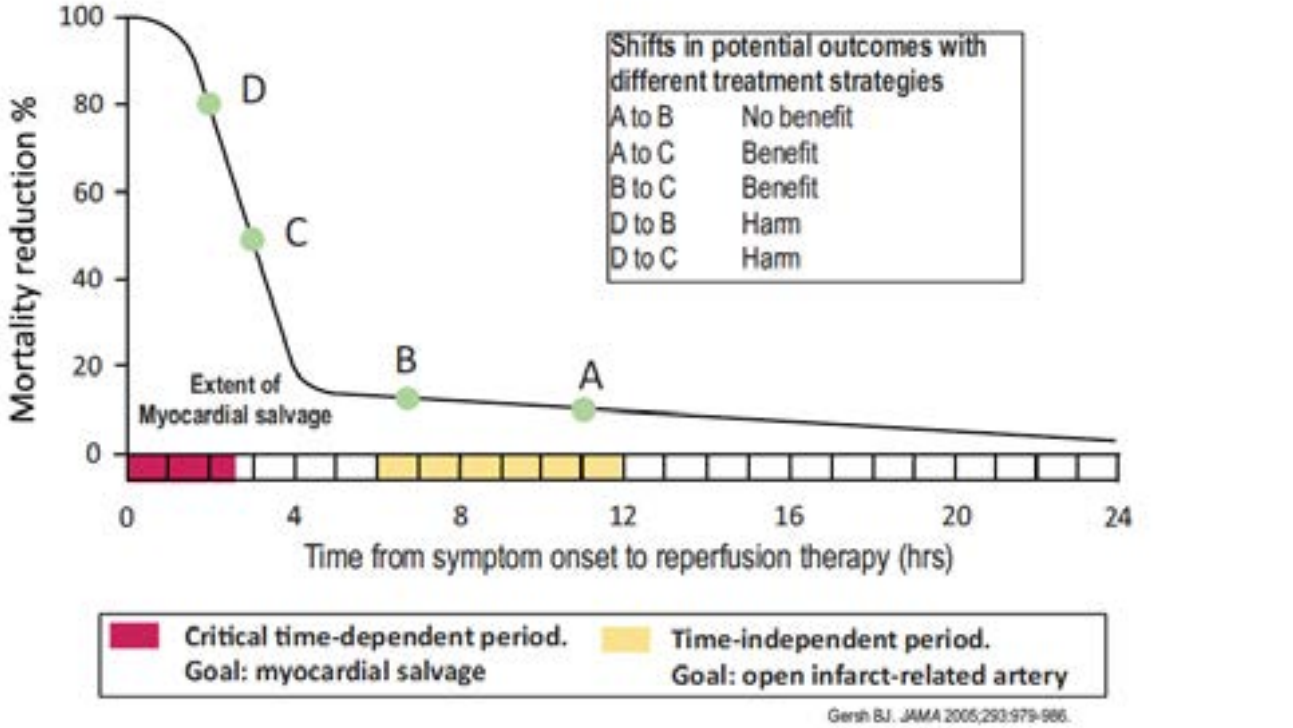
7084 PPCI
3078 PHT
16043 IHT

No. at Risk				
Thrombolysis				
In-Hospital	14260	12322	12100	11931
Prehospital	2736	2491	2460	2442
Primary PCI	6030	5661	5607	5555

Stenestrand, U. et al. JAMA 2006;296:1749-1756.

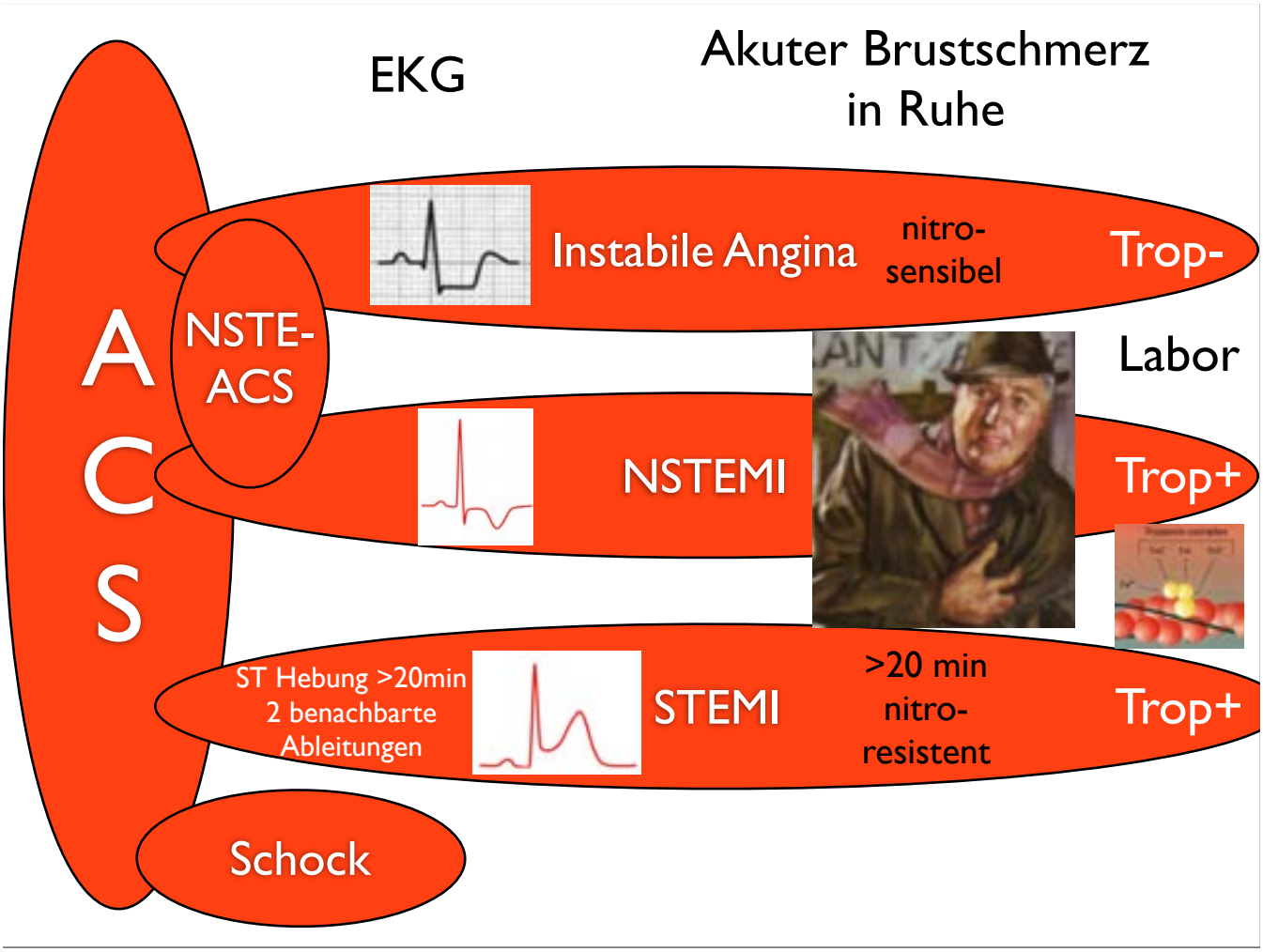
Lyseindikation

STEMI
>20 Minuten anhaltender nitroresistenter Schmerz
>20 Minuten anhaltende ST-Hebung
Schmerzbeginn vor <3 Stunden
Contact to balloon time >2 Stunden
PCI erst in mehr als 90 Minuten möglich
Keine Ausschlusskriterien für Lyse

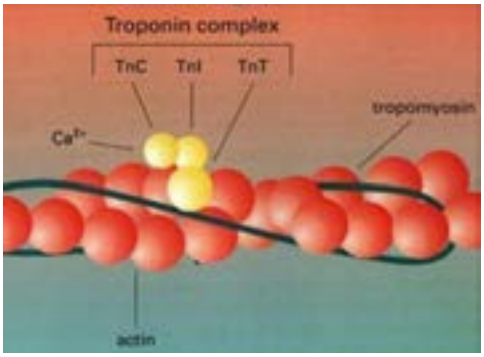


Sensible Schlüsselstellen im Herz und Folgen von Ischämie

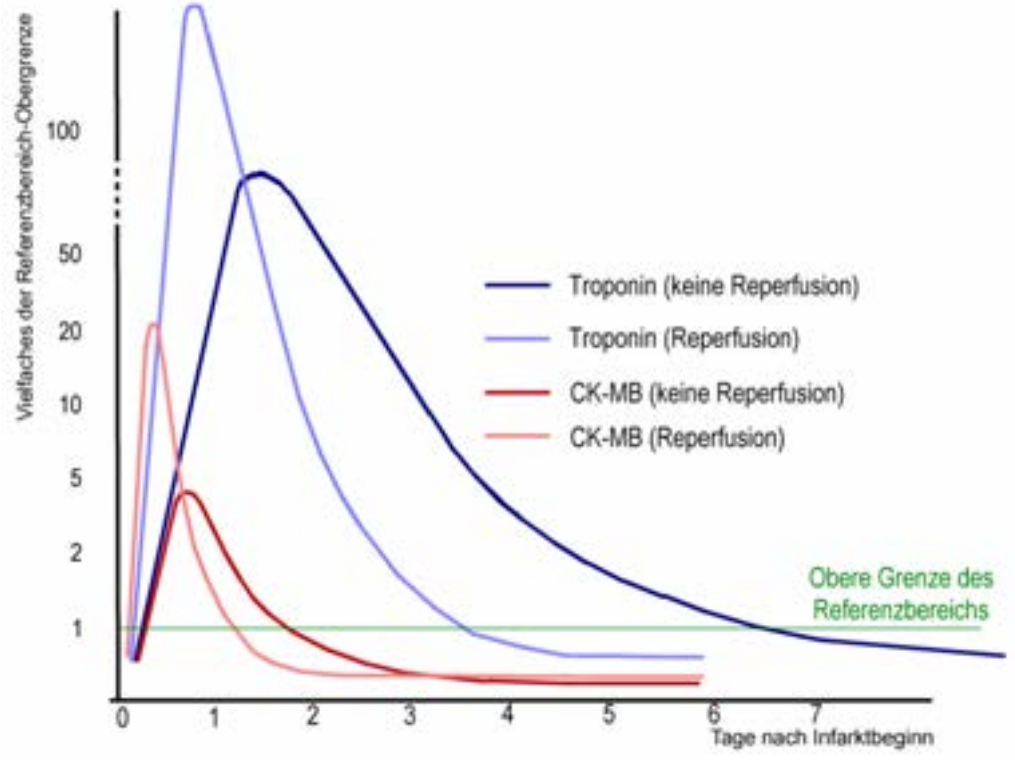
Arrhythmie-Kammerflimmern-Tod
Sinusknotenarterie Asystolie
AV-Knotenarterie AV Block III Asystolie
Reizleitungssystem HisBündel
Schenkelblock AV Block III
Papillarmuskeldysfunktion/-abriss Mitralinsuffizienz
Septumruptur ischämisch
Ventrikelruptur Tamponade Tod



Troponine im Blut sind sehr spezifisch für NSTEMI und STEMI



Man findet sie aber auch bei
Myokarditis, Niereninsuffizienz, akuter Herzinsuffizienz, langdauernden Tachykardien, hypertensiver Krise, Sepsis, Thoraxtrauma, akuter Pulmonalembolie und nach Reanimation!



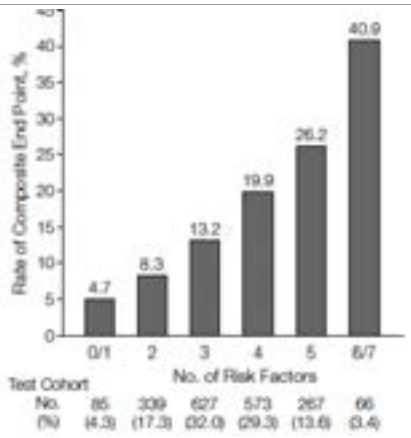
Trop
3xULN

Reperfusionsschaden ?
Cooling 33°vor Reperfusion

abgewandelt von ACC/AHA Practice Guidelines S. E32 IDat

TIMI Risk Score for UA/NSTEMI

Age ≥ 65 years?	Yes +1
≥ 3 Risk Factors for CAD?	Yes +1
Known CAD (stenosis ≥ 50%)?	Yes +1
ASA Use in Past 7d?	Yes +1
Severe angina (≥ 2 episodes w/in 24 hrs)?	Yes +1
ST changes ≥ 0.5mm?	Yes +1
+ Cardiac Marker?	Yes +1
Score	Click! points



Rates of all-cause mortality, myocardial infarction, and severe recurrent ischemia prompting urgent revascularization through 14 days after randomization were calculated for various patient subgroups based on the number of risk factors present in the test cohort (the group in the Thrombolysis in [illegible] TIMI] 118 trial; n=1957) (see [illegible] eased significantly as the TIMI [illegible] .001 by χ^2 for trend).

Latest LDL Guidelines (NCEP 2004 Update):

- "Very" High Risk: Guidelines suggest < 70mg/dl may be a reasonable option, but data may suggest there is little incremental benefit to this extreme but with high associated cost
- High Risk (known CAD, known other atherosclerotic dz, diabetes, etc): < 100 mg/dl
- Moderate Risk (> 1 risk factor): <130 mg/dl
- Lower Risk (0-1 risk factors): <160 mg/dl

Risk Factors:

- Diabetes
- Cigarette smoking
- HTN (BP 140/90 mm Hg or on antihypertensive medication)
- Low HDL cholesterol (< 40 mg/dL)
- Family history of premature CAD (CAD in male first-degree relative, or father less than 55, or female first-degree relative or mother less than 65)

http://www.mdcalc.com/timi-risk-score-for-uanstemi

PCI-Dringlichkeit bei NSTE- ACS

dringlich<2h

nitroresistente oder wiederkehrende AP
mit ST-Senkung>2mm
oder koronaren T
hämodynamisch instabil
Herzinsuffizienz
Kammerflimmern
Kammertachykardie

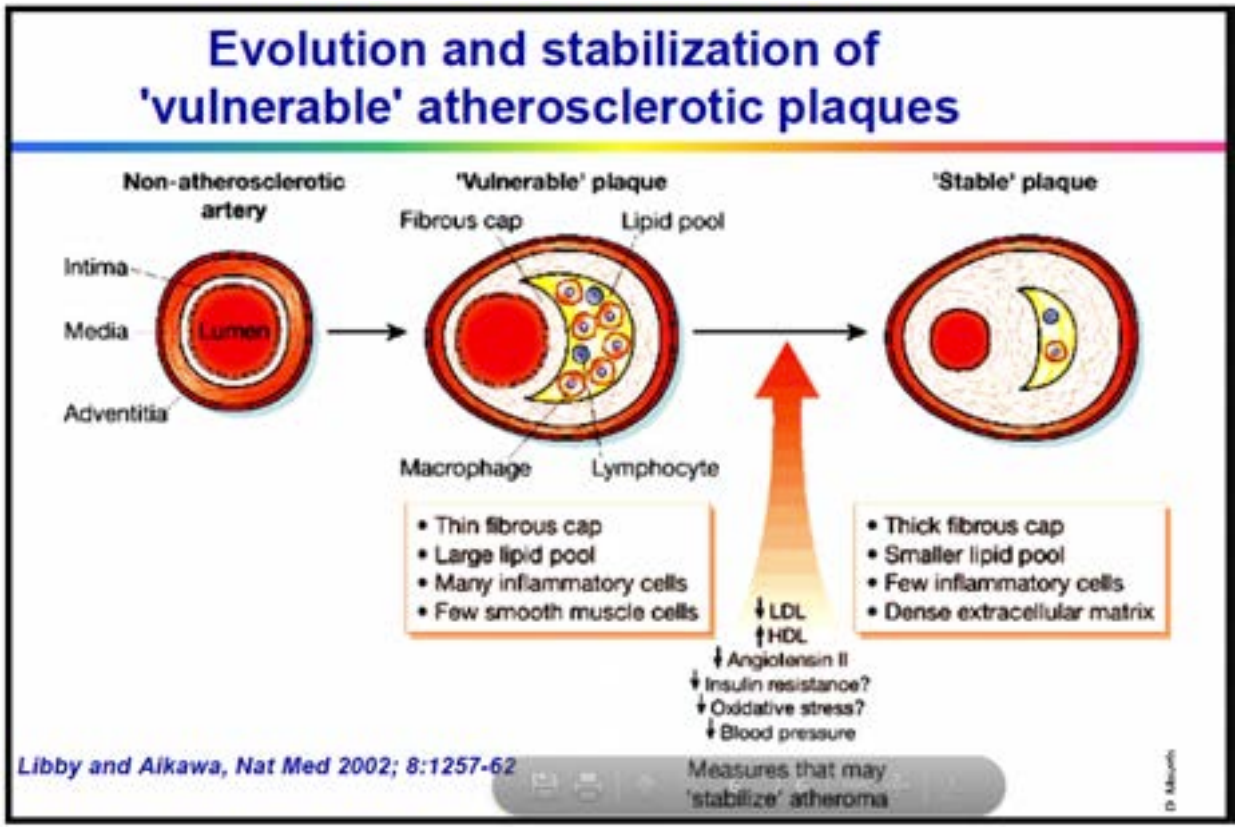
früh<72h

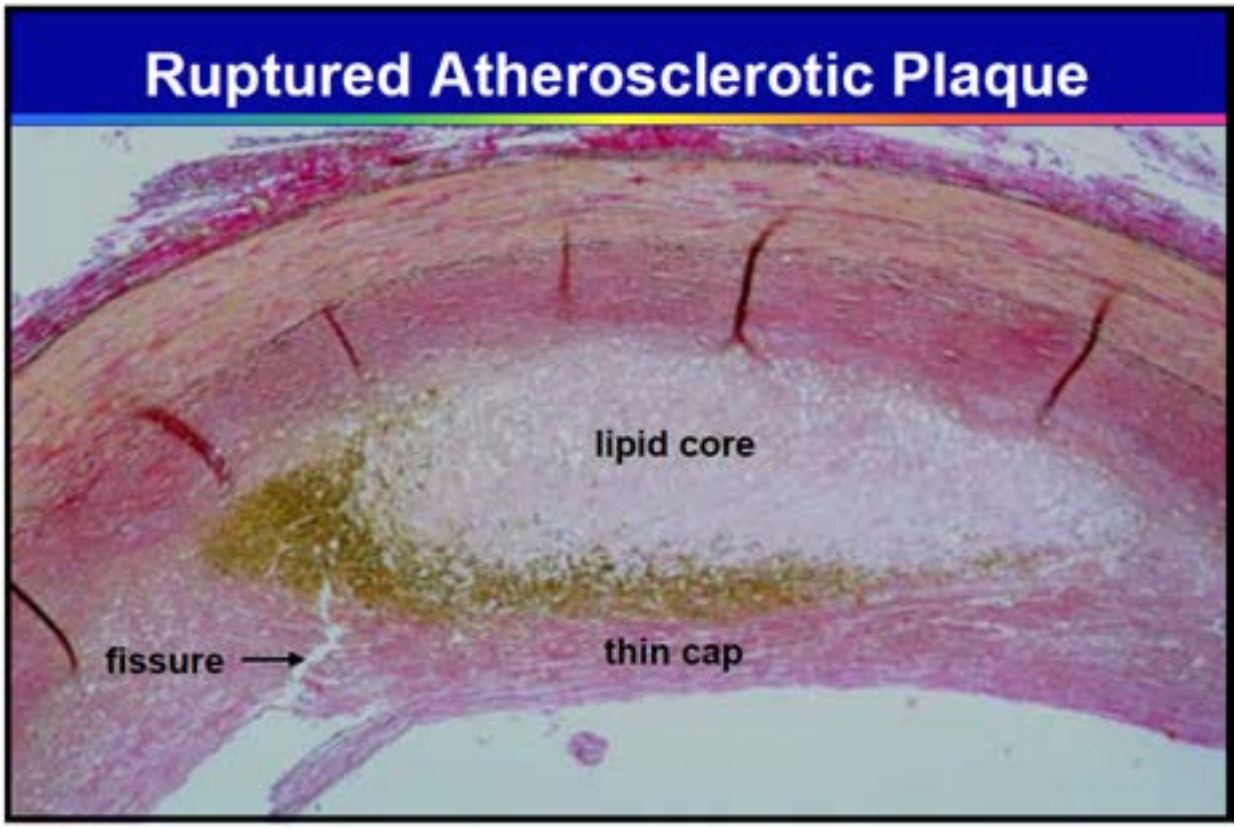
Trop pos
ST/T Dynamik
Diabetes
GFR<60 (hydrieren)
EF<40%
Postinfarkt-AP
Z.n. PCI (6 Mon)
Z.n. CABG
mittleres bis hohes Risiko
GRACE Risk Score

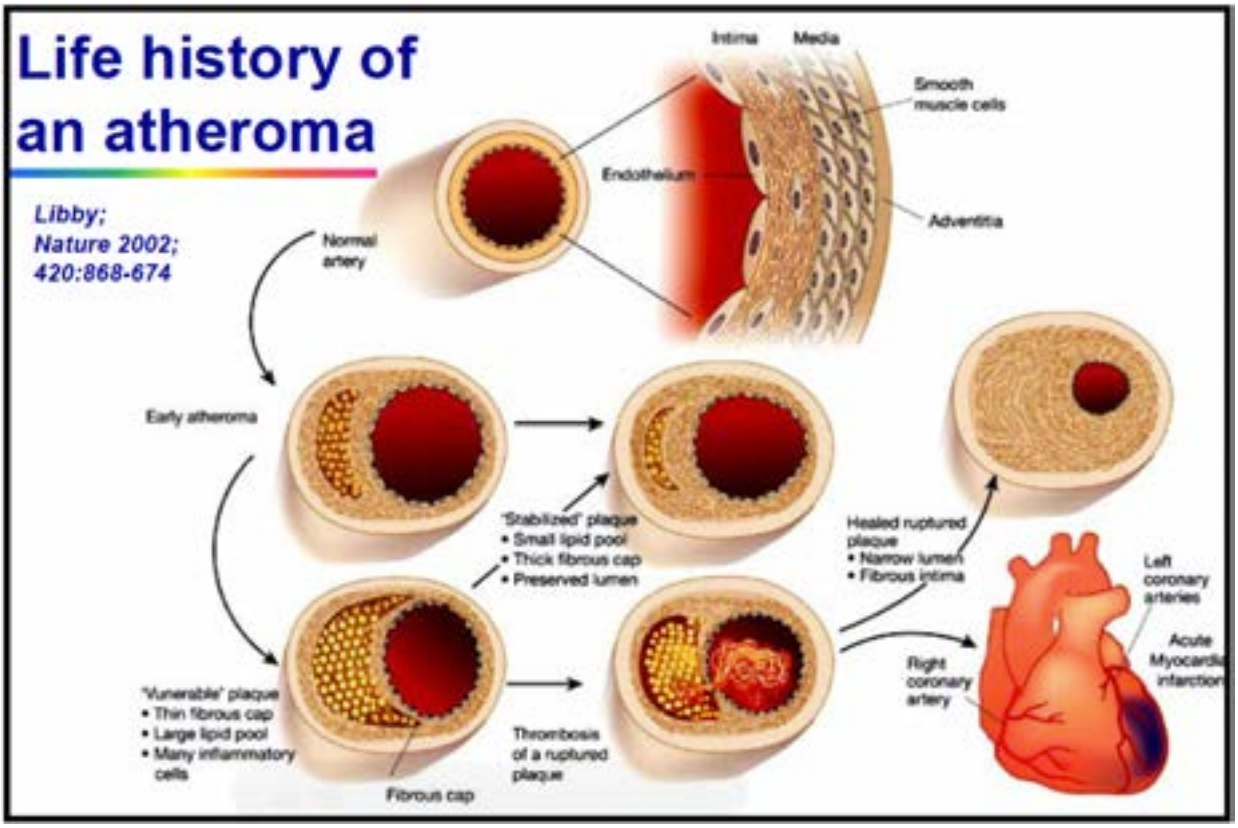
www.outcomes-umassmed.org/grace/

elektiv

Trop neg/EKG normal
Aufnahme und 6-12h
keine AP mehr


Evolution and stabilization of 'vulnerable' atherosclerotic plaques
Non-atherosclerotic artery
'Vulnerable' plaque
'Stable' plaque
Intima
Media
Adventitia
Lumen
Fibrous cap
Lipid pool
Macrophage
Lymphocyte
• Thin fibrous cap
• Large lipid pool
• Many inflammatory cells
• Few smooth muscle cells
• Thick fibrous cap
• Smaller lipid pool
• Few inflammatory cells
• Dense extracellular matrix
LDL
HDL
Angiotensin II
Insulin resistance?
Oxidative stress?
Blood pressure
Measures that may 'stabilize' atheroma
Libby and Aikawa, Nat Med 2002; 8:1257-62


Ruptured Atherosclerotic Plaque
lipid core
fissure
thin cap



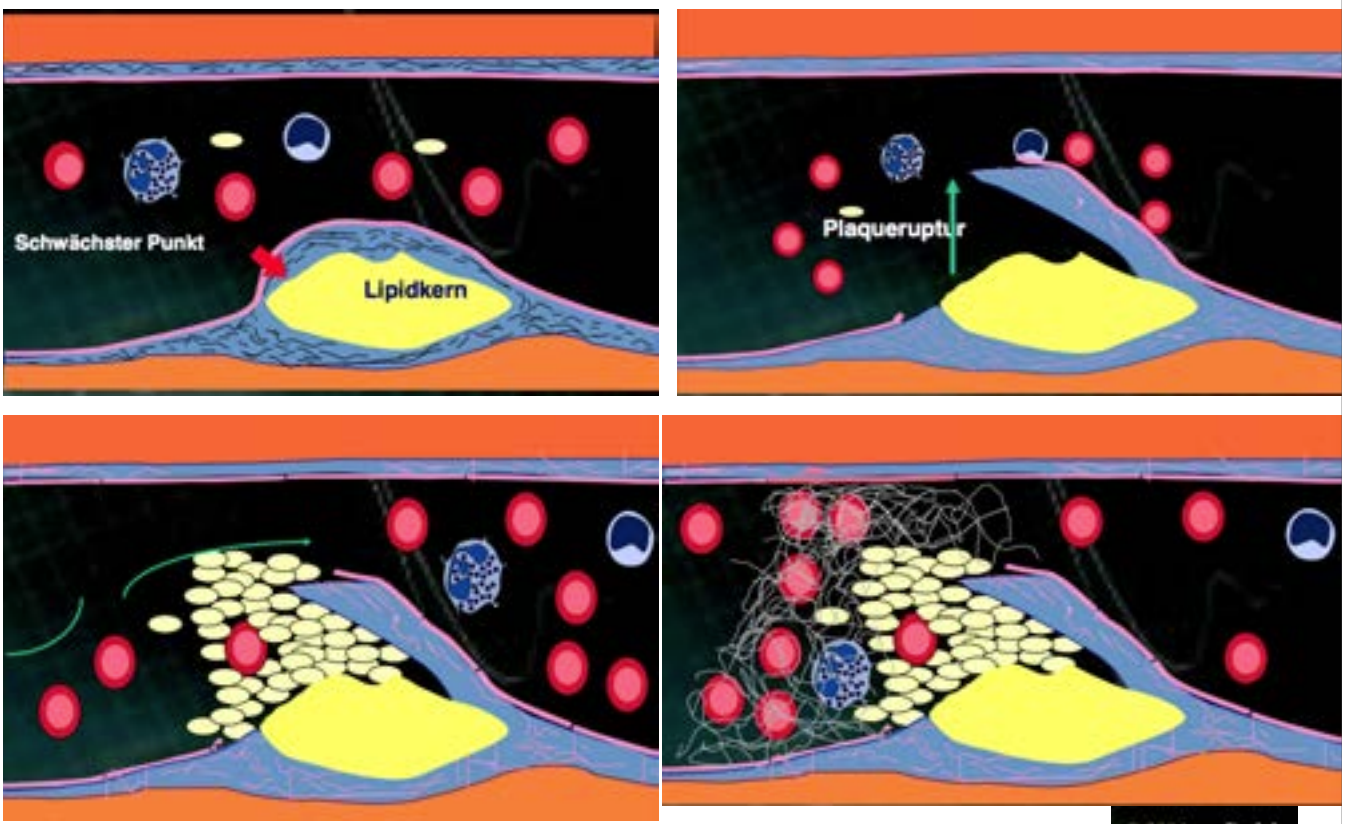


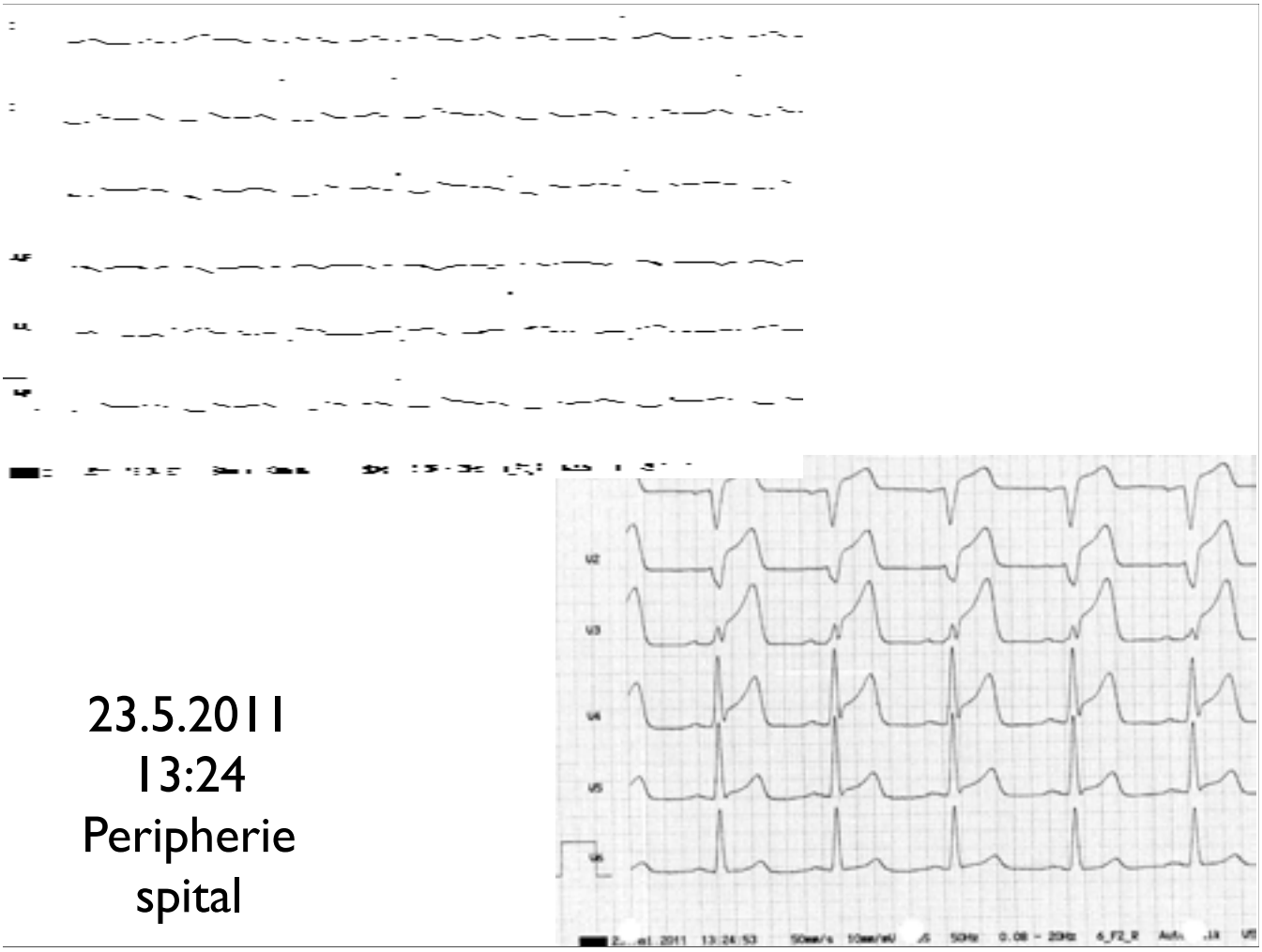
Illustration von:

4 Fallberichte

- Was hätte ich getan?
  als Patient
  als Angehöriger
  als Hausarzt
  als Notarzt
  als Turnusarzt im Peripheriespital
- Fragen sind willkommen!

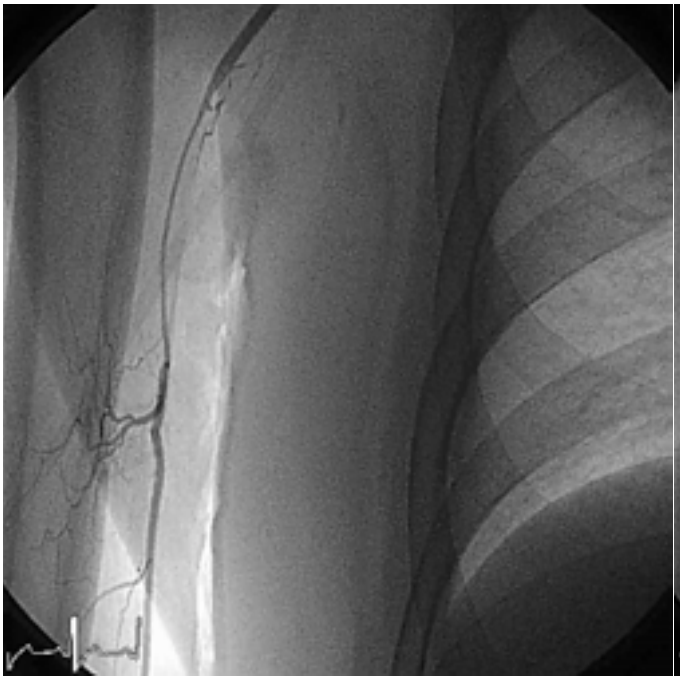
Fall 1

Pat. männlich, 49 a
Vor einer Woche erstmals Belastungs-AP
bei hohen Blutdruckwerten
heute seit 13 Uhr massive Ruheangina anhaltend
Risiko:
art. Hypertonie, Nikotinabusus, Hyperlipidämie, Stress
bekannte geringe Niereninsuffizienz

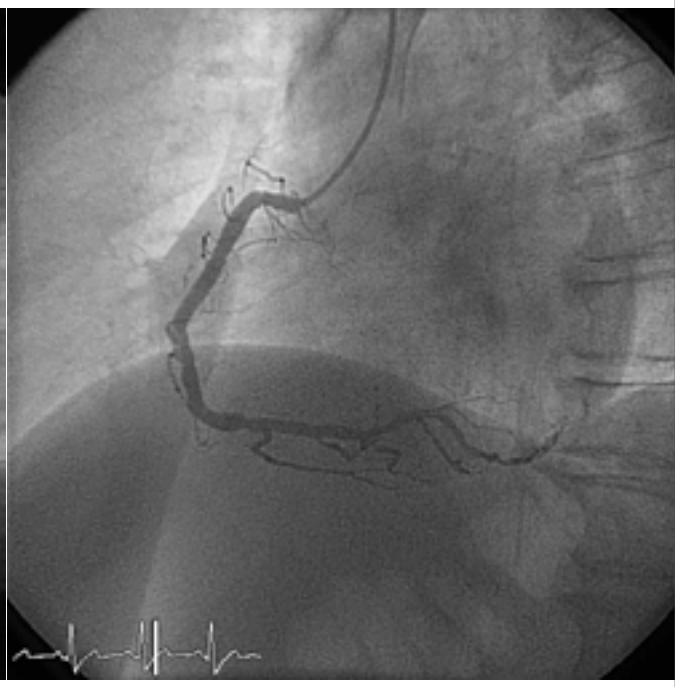


23.5.2011
13:24
Peripherie
spital

Spastische A. rad. dext.

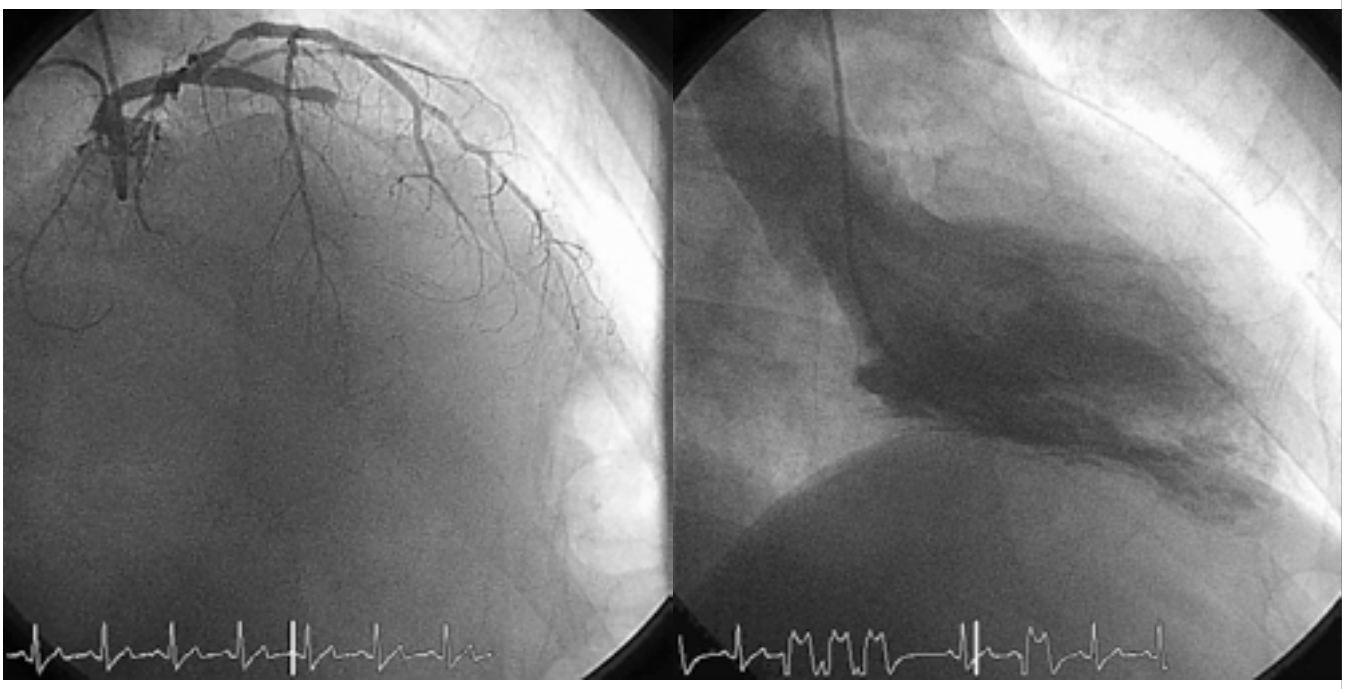


Diffus sklerosierte RCA

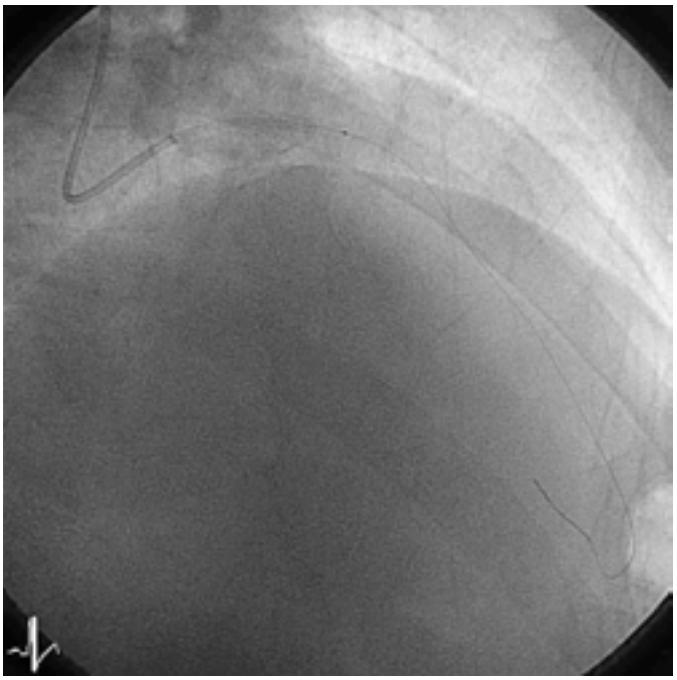


LAD verschlossen
R. int. 50%

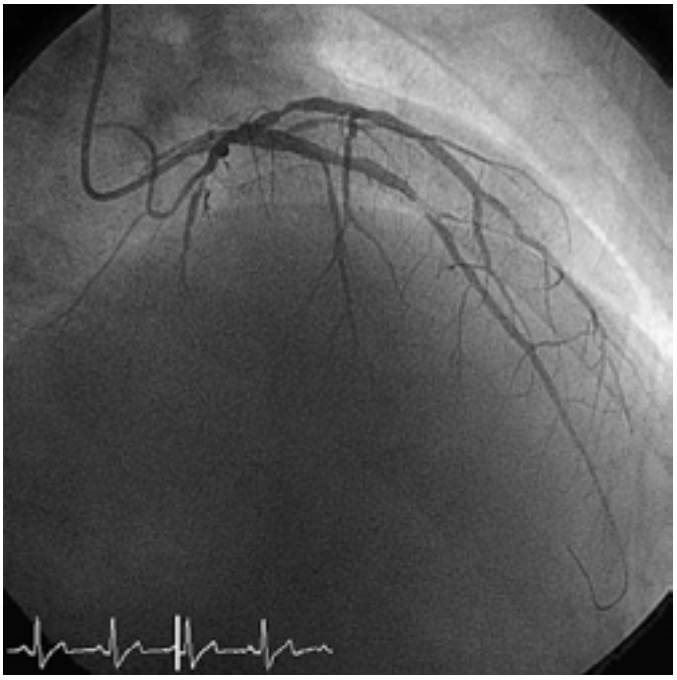
Vorderwand a-/dyskinetisch



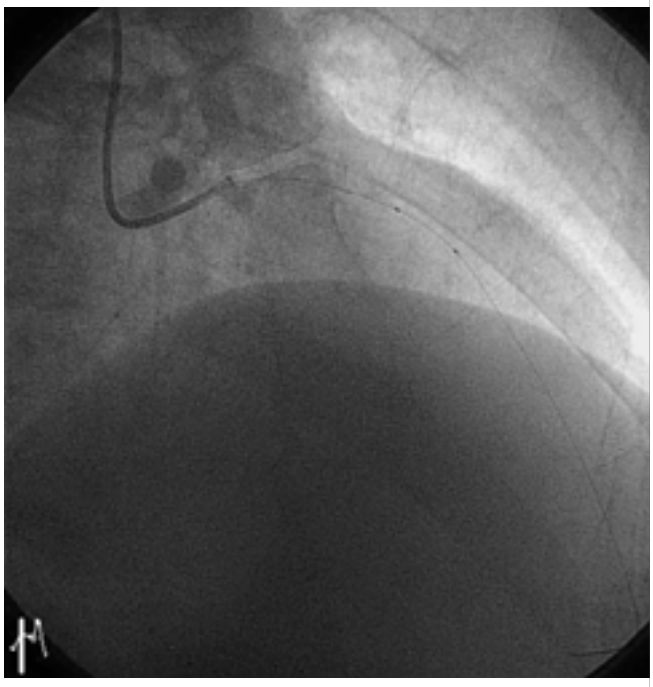
Thrombusabsaugung



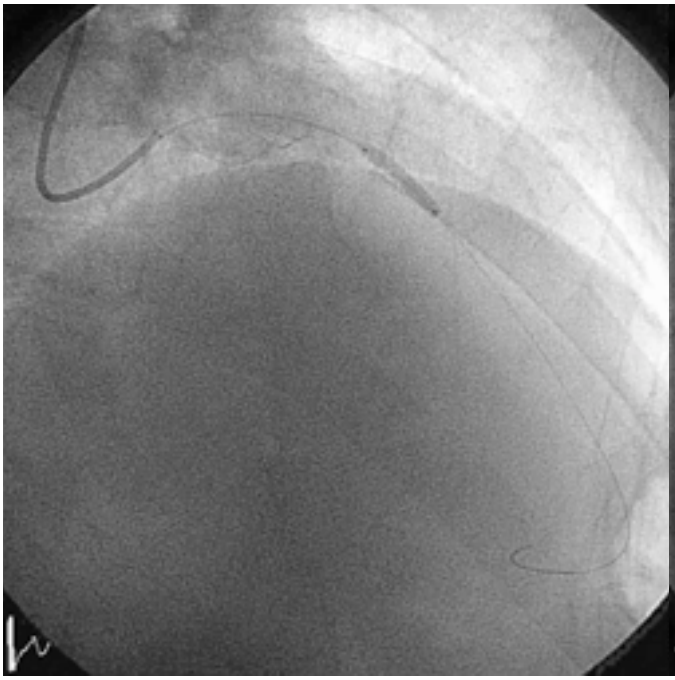
LAD offen



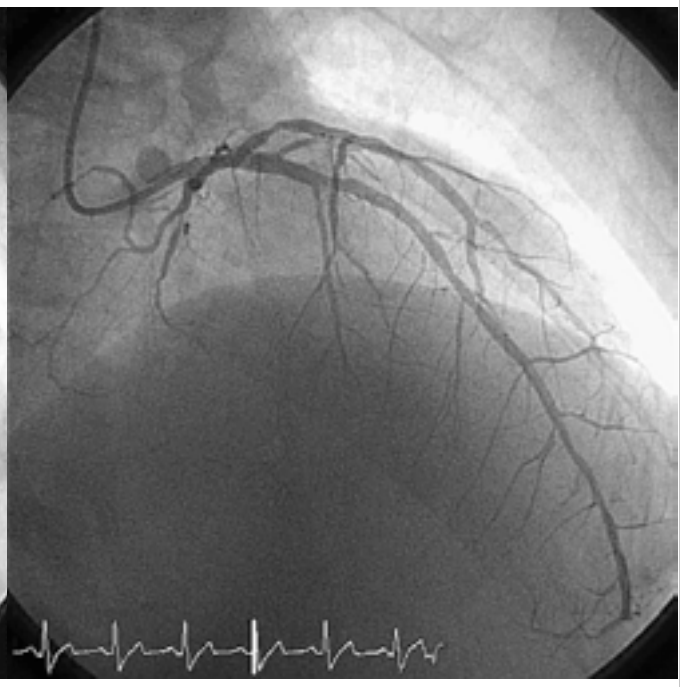
Infusionsballon Abciximab



DE-Stent

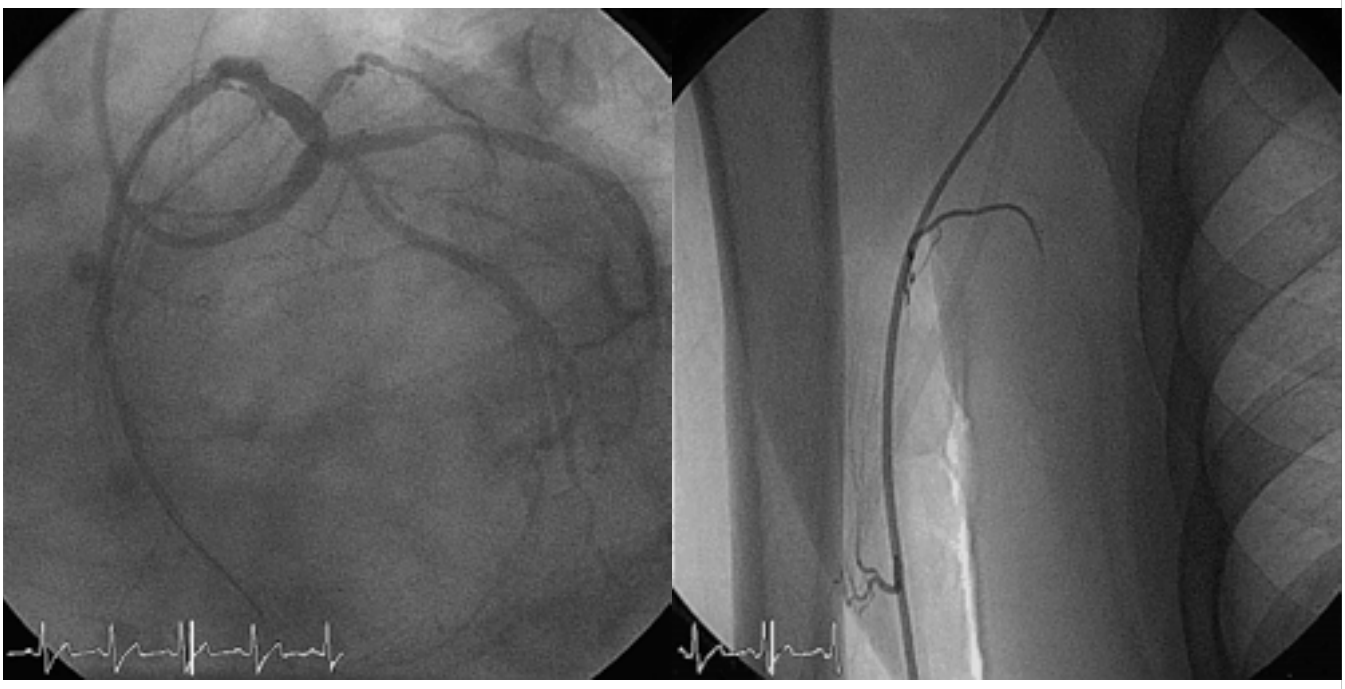


TIMI 3 Fluss

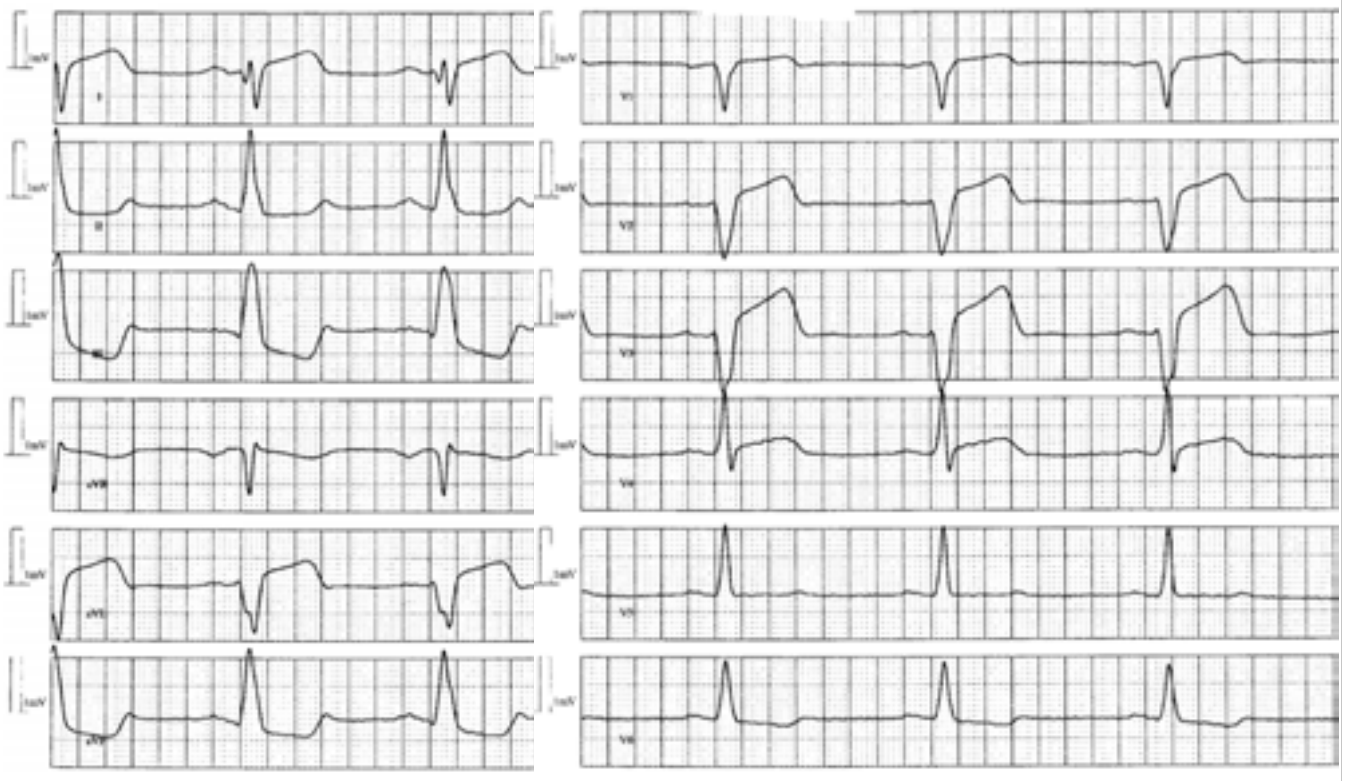


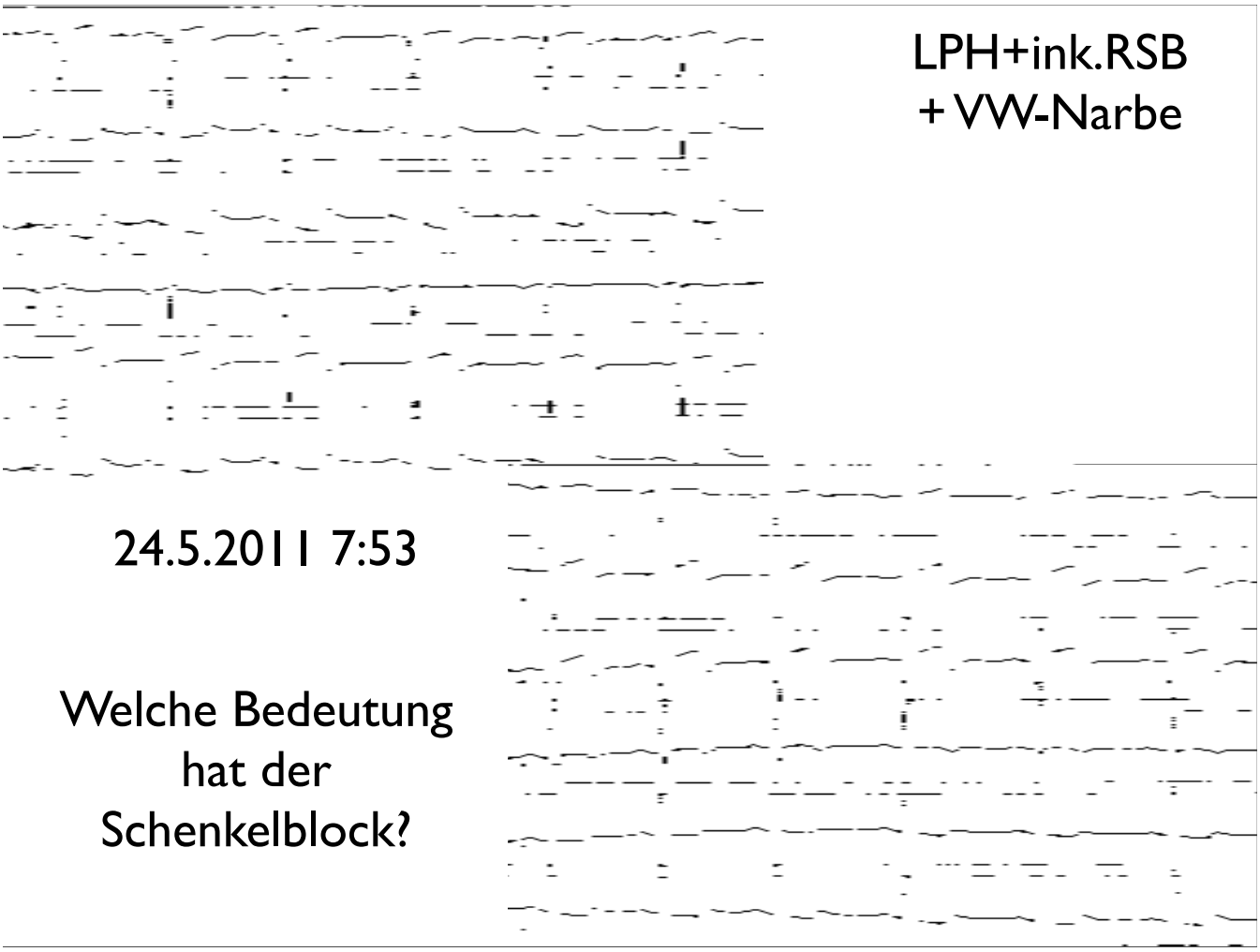
R. int. 50%

A rad. entspannt



15:34





LPH+ink.RSB
+VW-Narbe

24.5.2011 7:53

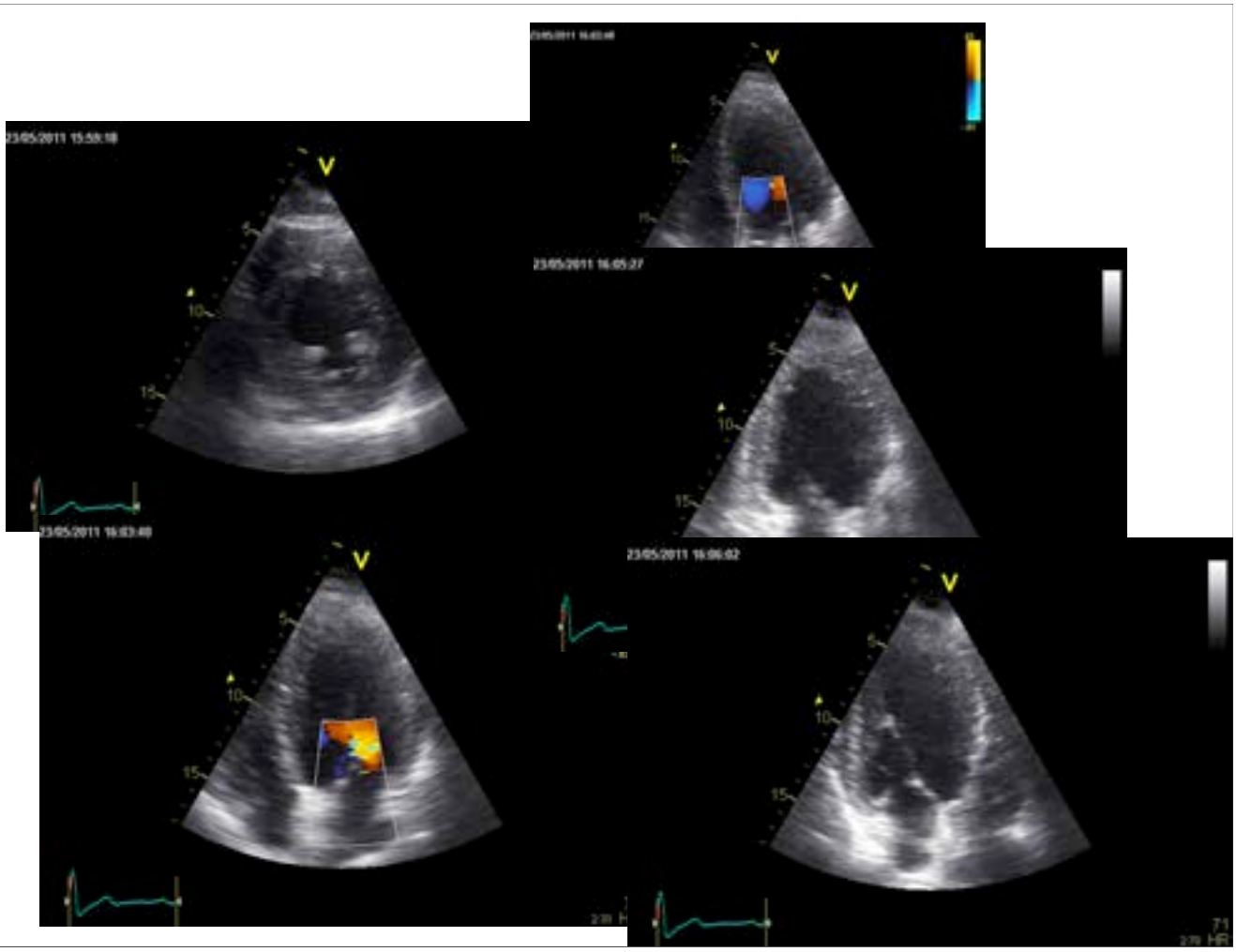
Welche Bedeutung hat der Schenkelblock?

Table 1. Guidelines of the American College of Cardiology and the American Heart Association for Temporary or Permanent Implantation of Pacemakers in Patients with Acute Myocardial Infarction.*

Class†	Indications for Temporary Pacing	Indications for Permanent Pacing
I	Asystole Symptomatic bradycardia (including sinus bradycardia or Mobitz type I block with hypotension) Bilateral BBB (alternating BBB or right BBB alternating with LAFB or LPFB) Bifascicular block that is new or of indeterminate age (right BBB with LAFB or LPFB or left BBB) with a prolonged PR interval Mobitz type II second-degree AV block	Persistent second-degree AV block in the His–Purkinje system, with bilateral BBB or third-degree AV block within or below the His–Purkinje system after myocardial infarction Transient advanced (second- or third-degree) infranodal AV block and associated BBB‡ Persistent and symptomatic second- or third-degree AV block
IIa	Right BBB and LAFB or LPFB that is new or of indeterminate age Right BBB with a prolonged PR interval Left BBB that is new or of indeterminate age Recurring sinus pauses not responsive to atropine	None
IIb	Bifascicular block of indeterminate age Isolated right BBB that is new or of indeterminate age	Persistent second- or third-degree AV block at the level of the AV node
III	Prolonged PR interval Type 1 second-degree AV block with normal hemodynamics Accelerated idioventricular rhythm BBB or fascicular block known to exist before acute myocardial infarction	Transient AV conduction disturbances in the absence of intraventricular conduction defects Transient, isolated AV block in the presence of isolated LAFB Acquired LAFB in the absence of AV block Persistent first-degree AV block in the presence of BBB that is old or of indeterminate age

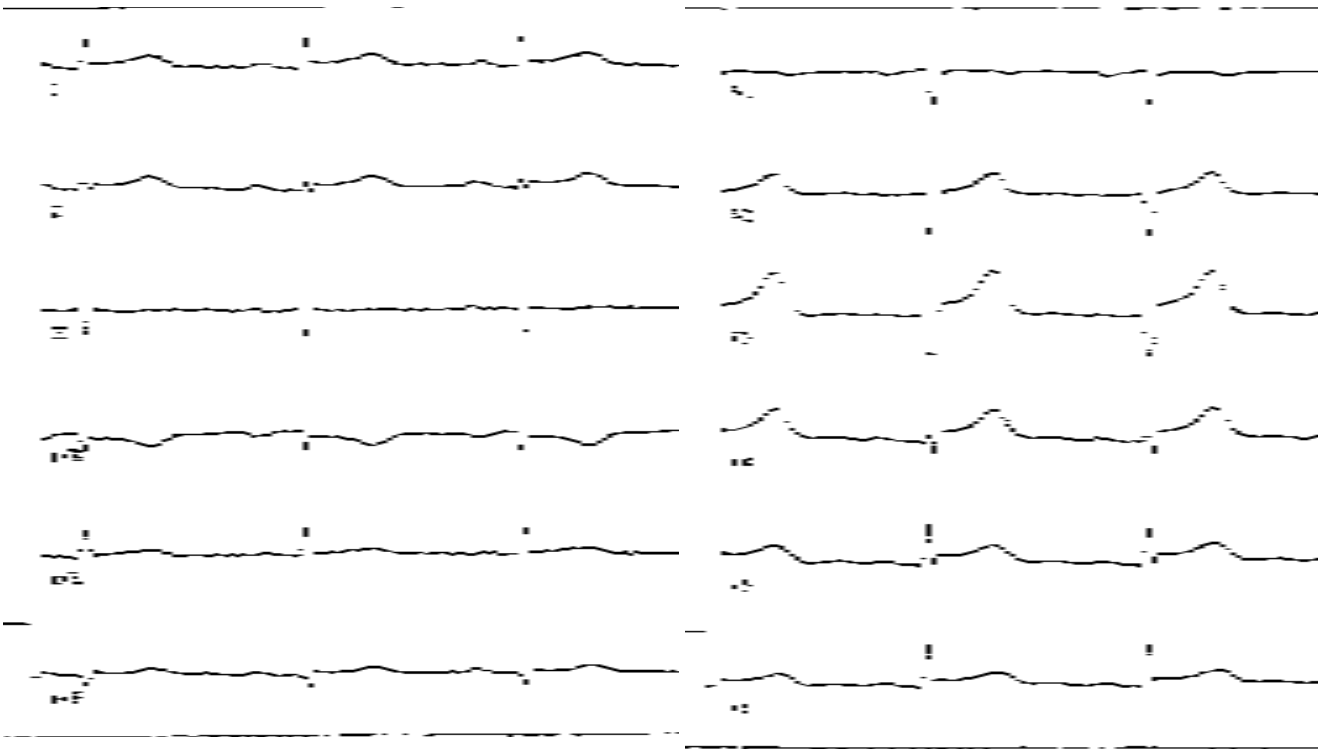
* The information is adapted from Ryan et al.[26] and Gregoratos et al.[27] AV denotes atrioventricular, BBB bundle-branch block, LAFB left anterior fascicular block, and LPFB left posterior fascicular block.
† Class designations refer to the level of evidence supporting the effectiveness of the procedure or treatment, where class I indicates that the evidence is very strong and class III that it is absent or that the procedure is not useful and may be harmful.
‡ An electrophysiological study may be useful to determine the site of the block.

N Engl J Med 2003;348:933-40.

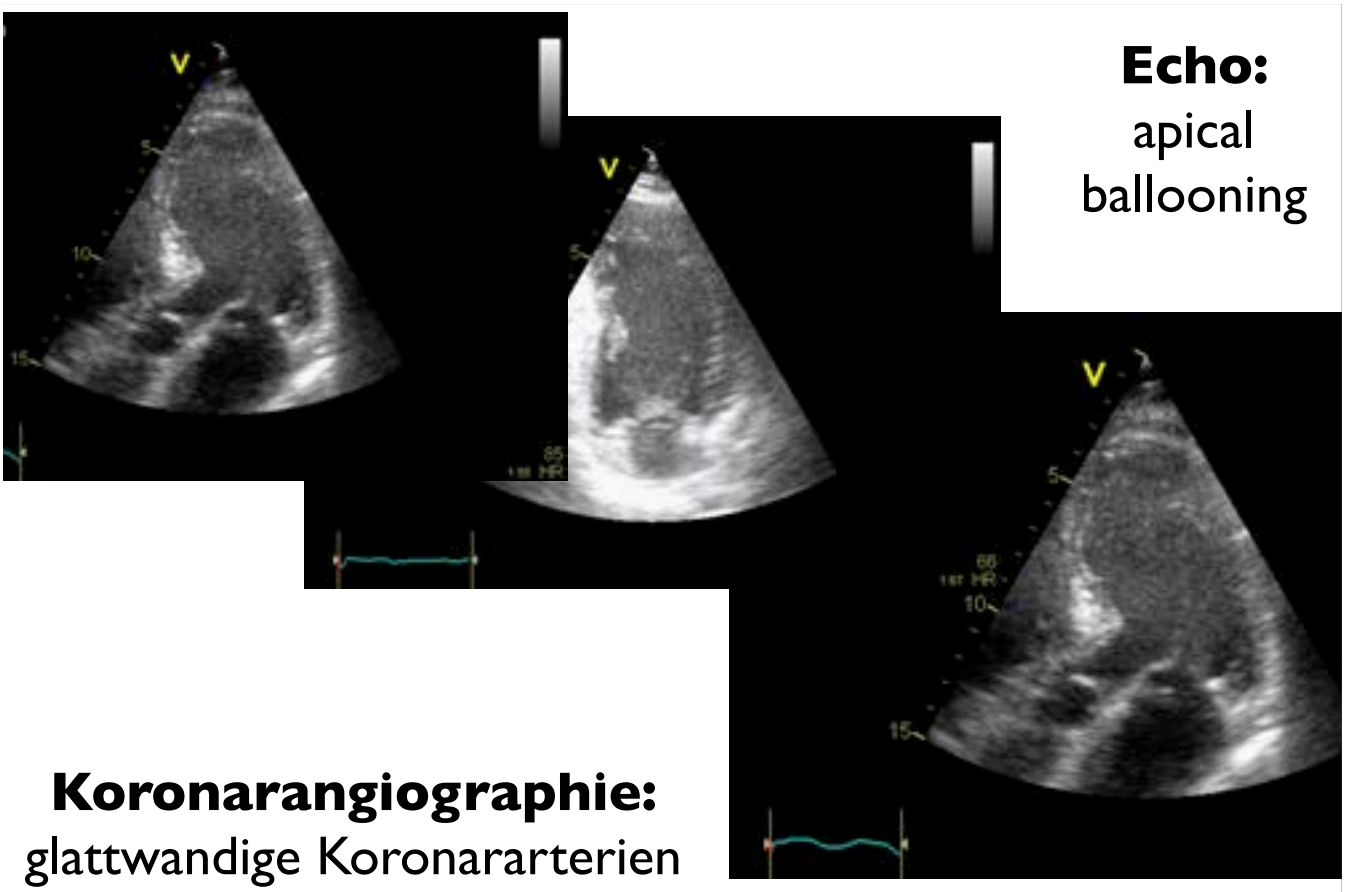


Fall 2

Heiliger Abend
Pat weibl., 63 a
seit 18 Uhr heftiger retrosternaler Schmerz
Suizidgedanken, vor einigen Tagen Tod des Ehemannes
anamn. seit Jahren supraventrikuläre Tachykardien
bek. Struma, art. Hypertonie
Therapie mit Inderal 2x40mg



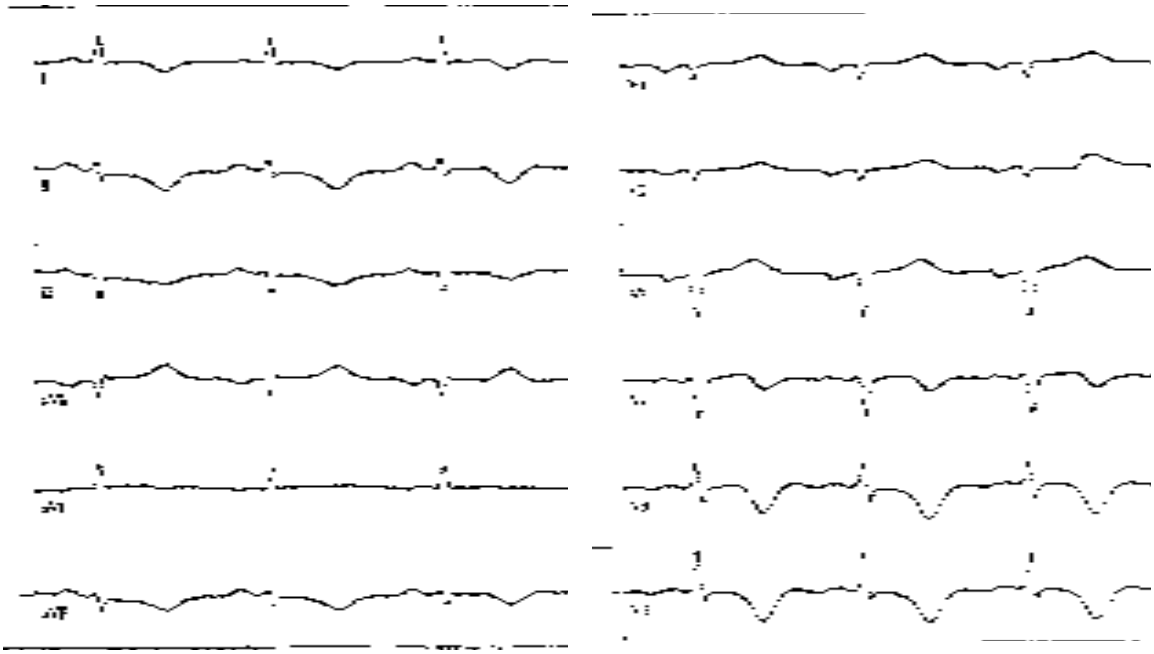
24.12.2004 22:56



Echo:
apical
ballooning

Koronarangiographie:
glattwandige Koronararterien
keine Stenose

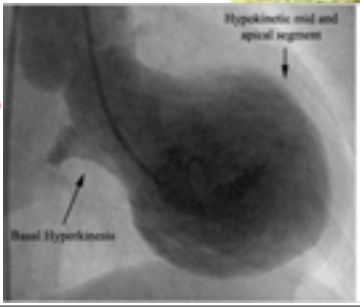
Einige Tage später heftige nächtliche Angina pectoris



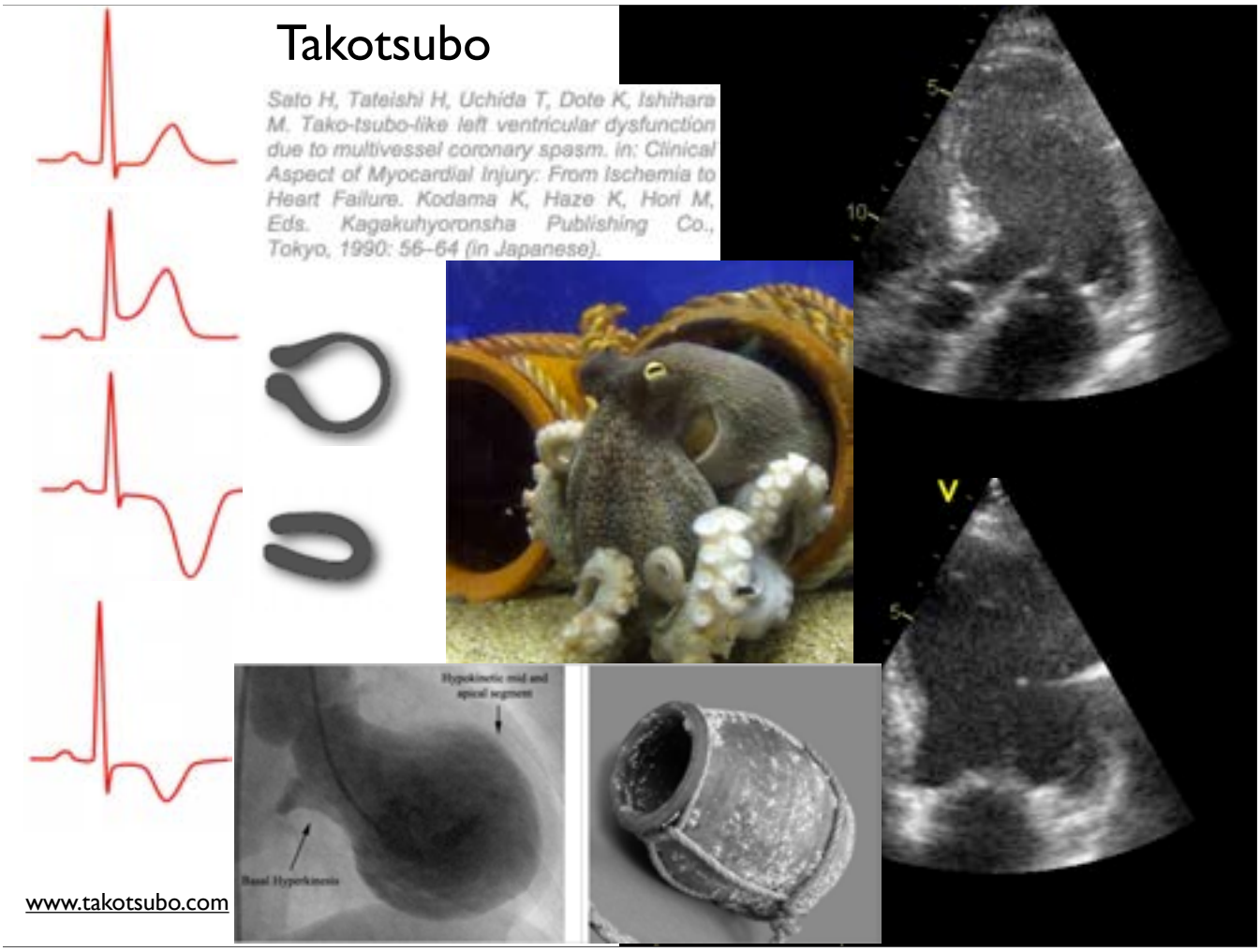
27.12.2004 2:26

Takotsubo

Sato H, Tateishi H, Uchida T, Dote K, Ishihara M. Tako-tsubo-like left ventricular dysfunction due to multivessel coronary spasm. in: Clinical Aspect of Myocardial Injury: From Ischemia to Heart Failure. Kodama K, Haze K, Hori M, Eds. Kagakuhyoronsha Publishing Co., Tokyo, 1990: 56–64 (in Japanese).



www.takotsubo.com



Fall 3

Pat. weibl. , 75a
am 31.5.2011 seit 11 Uhr
heftige Ruheangina
Keine kardialen Risikofaktoren
ASS-Dauertherapie

EKG beim Hausarzt, 11:30

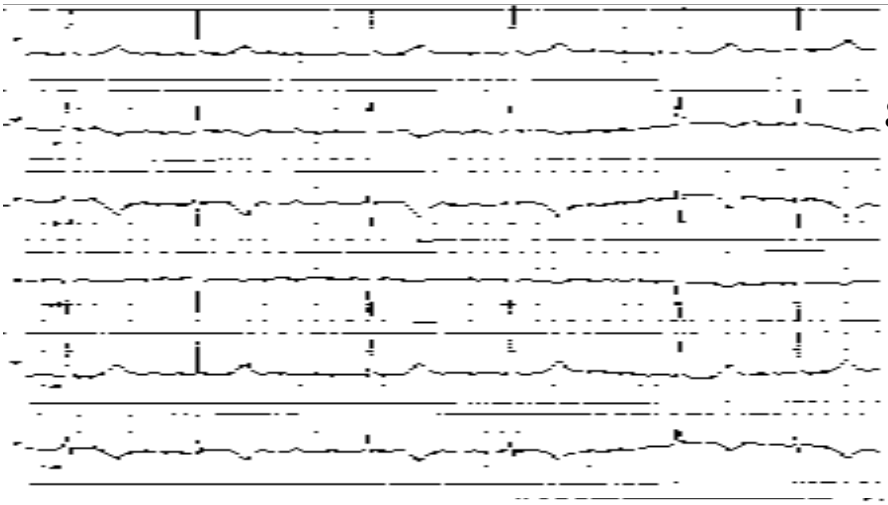
I	V1
II	V2
III	V3
aVR	V4
aVL	V5
aVF	V6

RCA glattwandig

LCA anguliert, glattwandig, CX verschlossen

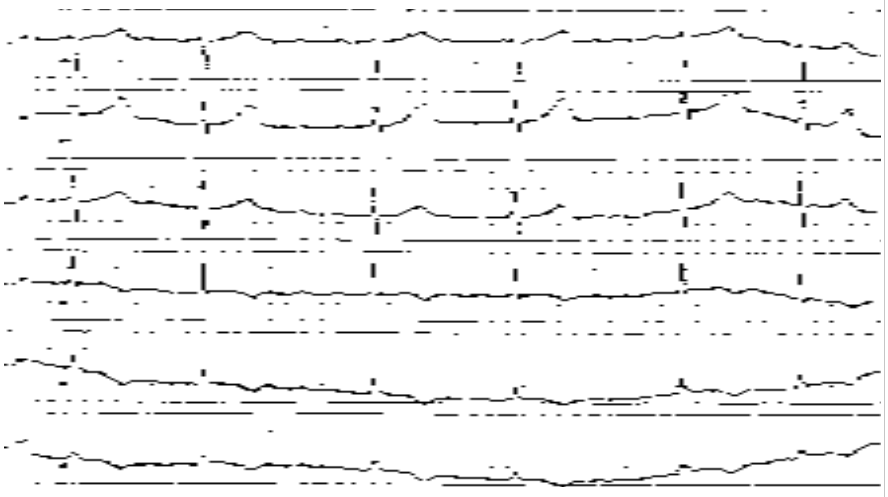
Thrombusabsaugung

Glattwandige CX offen, ohne Stent!



Echo:
gute LVF, LVHT, LVEDP hoch
Hypokinesie posterolateral
Aortenklappensklerose
riesiger linker Vorhof

EKG-Ko
1.6.2011 7:25

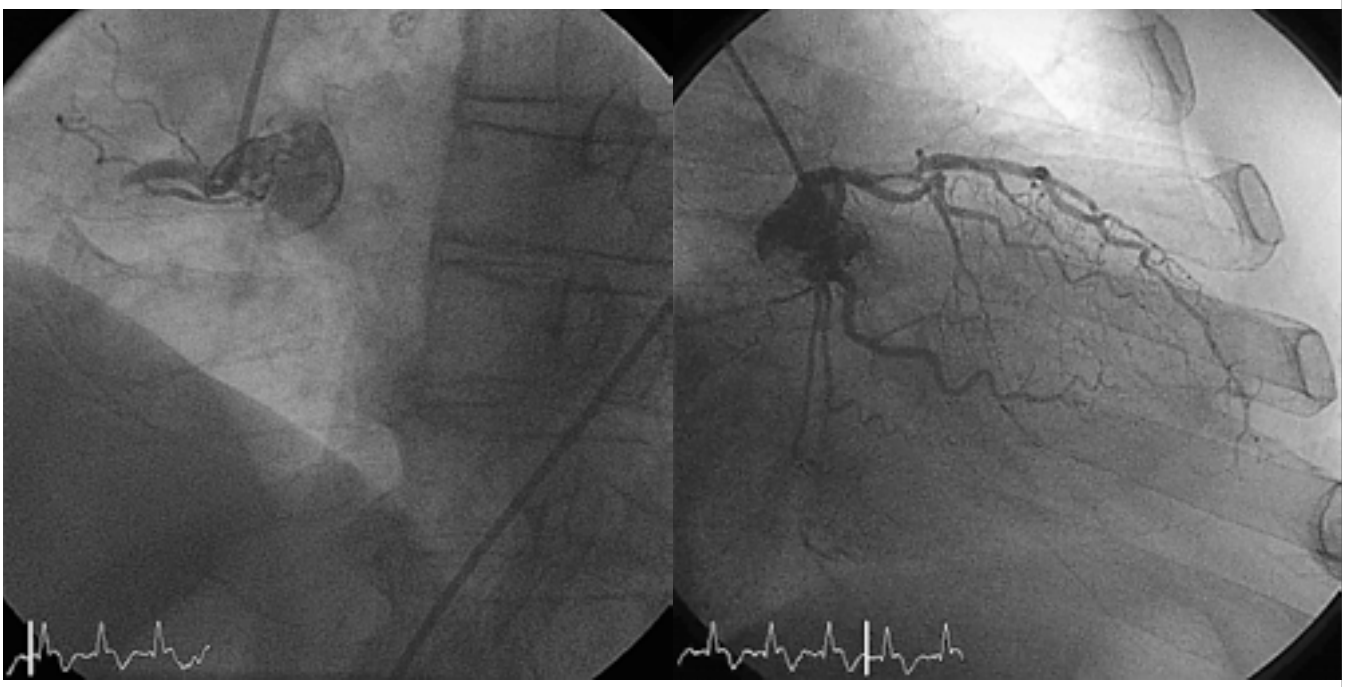


Fall 4

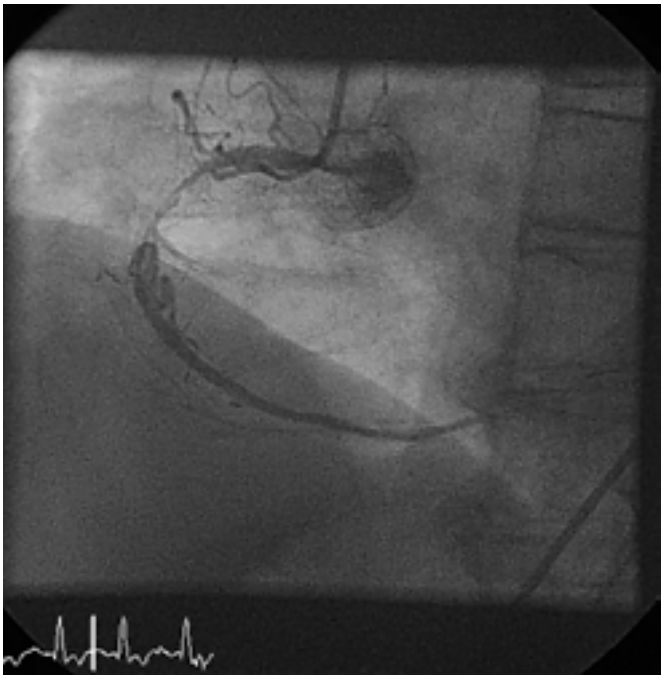
Männlich, 79a
Stubenbergsee
Schmerzbeginn 17.5. 2011 mittags
Pat. geht schlafen
16 Uhr: Hubschrauber

Risiko:
NIDDM, art. Hypertonie, Zn Nikotin vor 30a

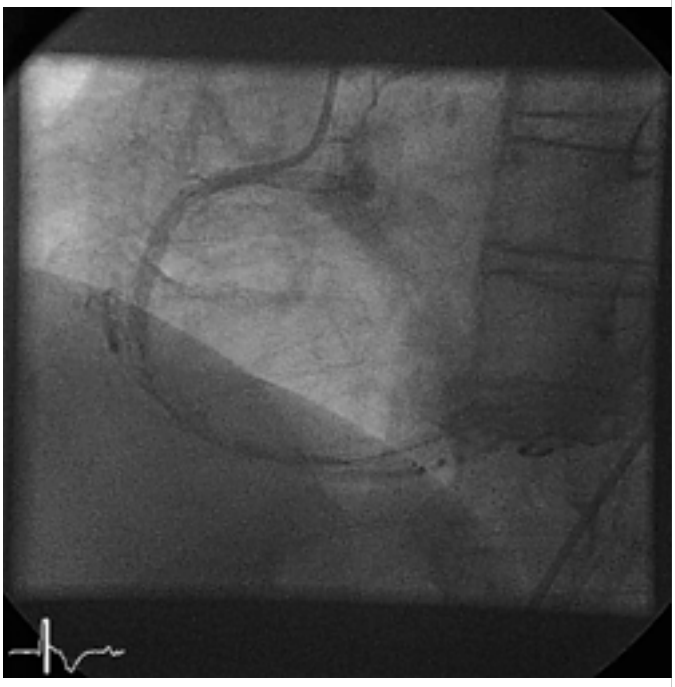
Dominante RCA zu LAD 90%/CX 90%



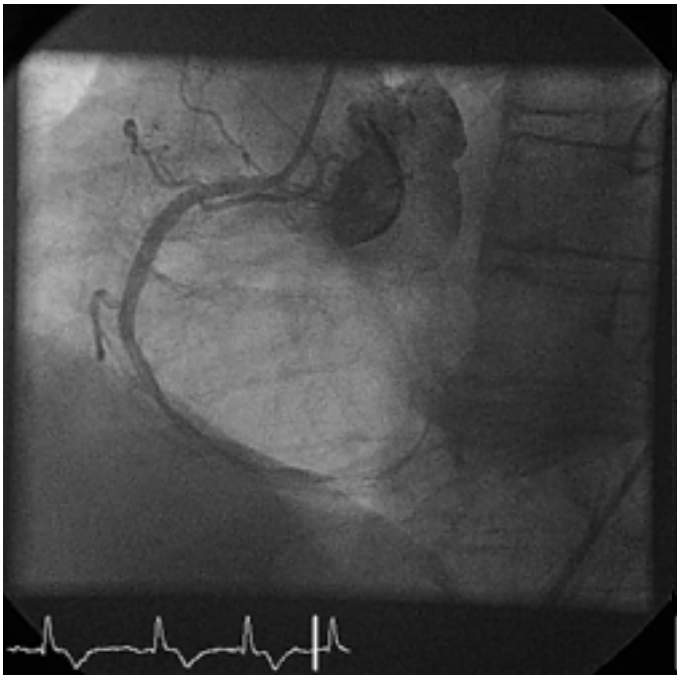
Drahtrekanalisation



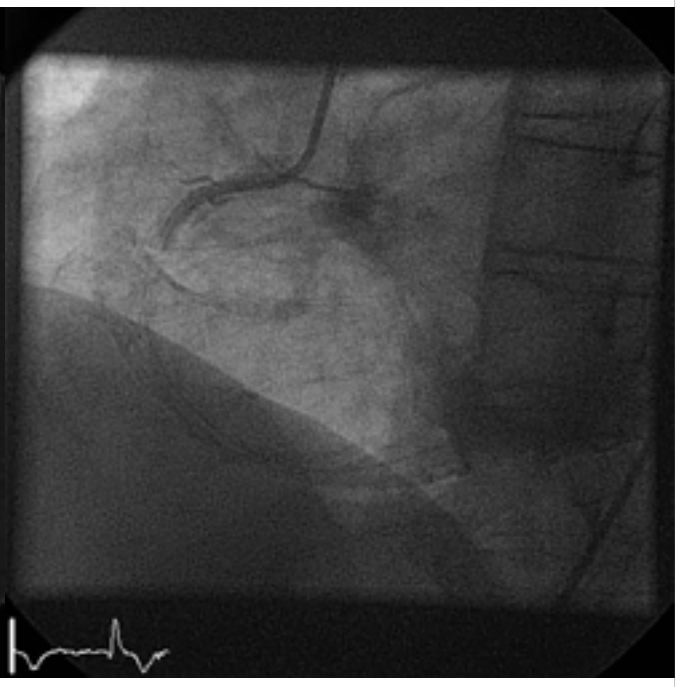
Stent



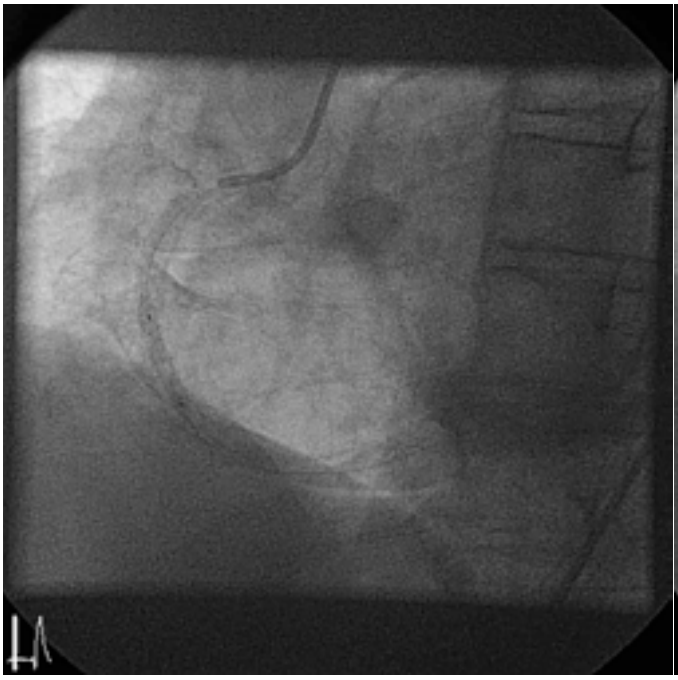
Fluss distal schlecht



RCA wieder zu

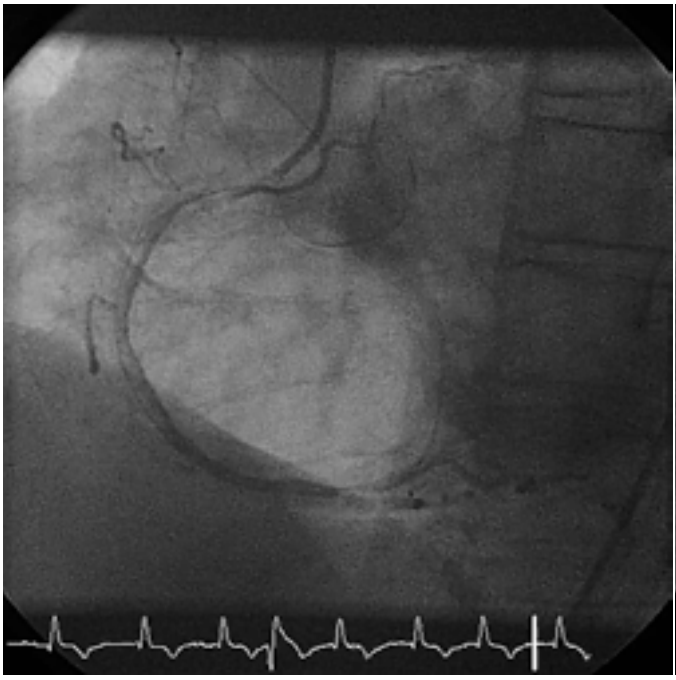


2. Stent

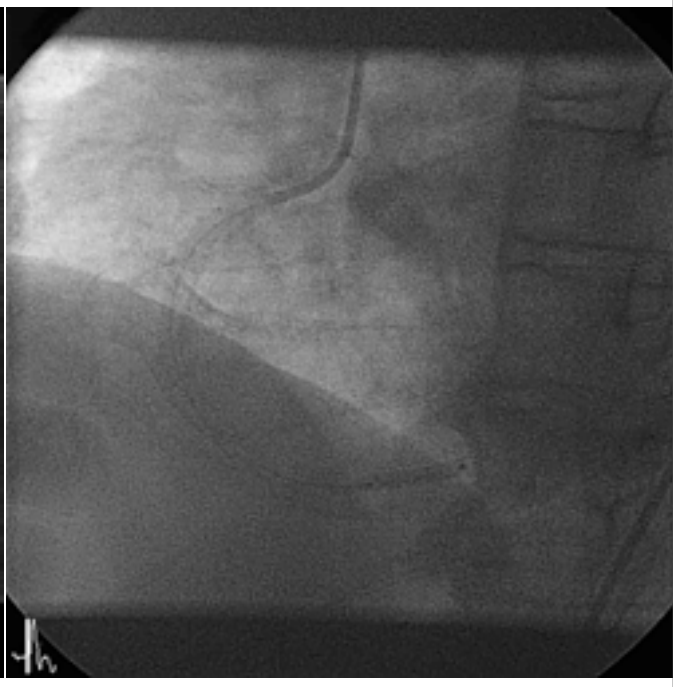


Thrombusabsaugung

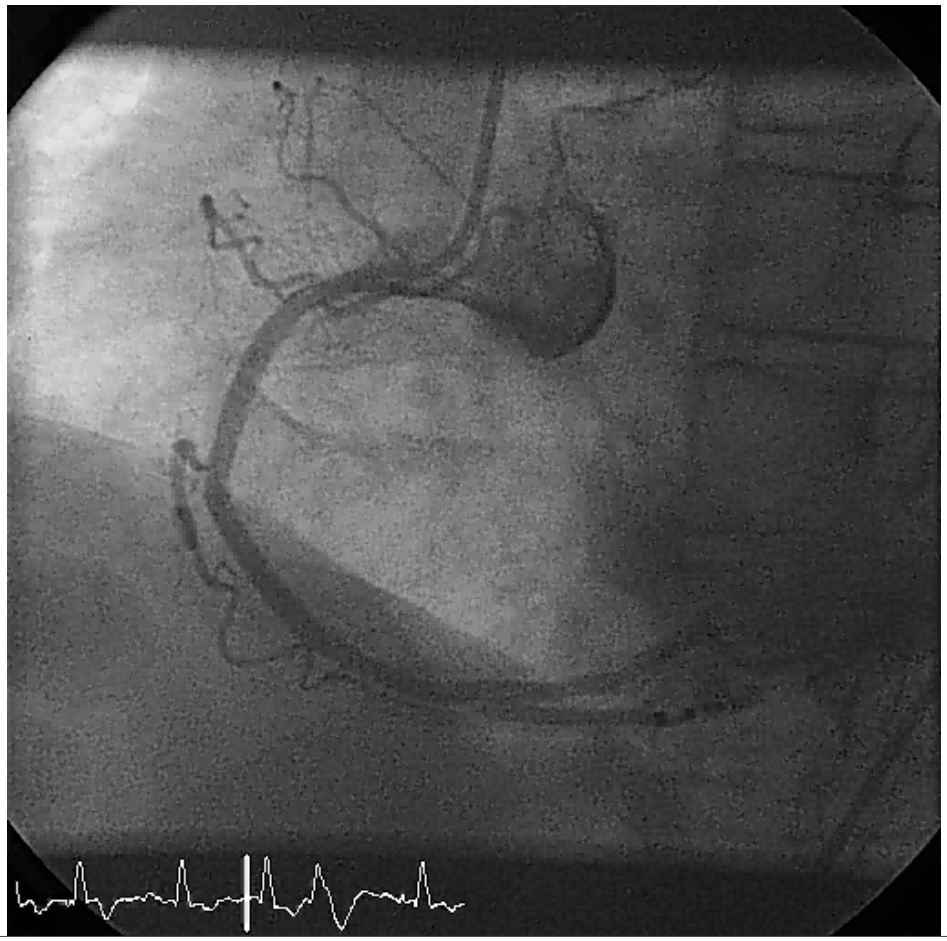
distale Stenose



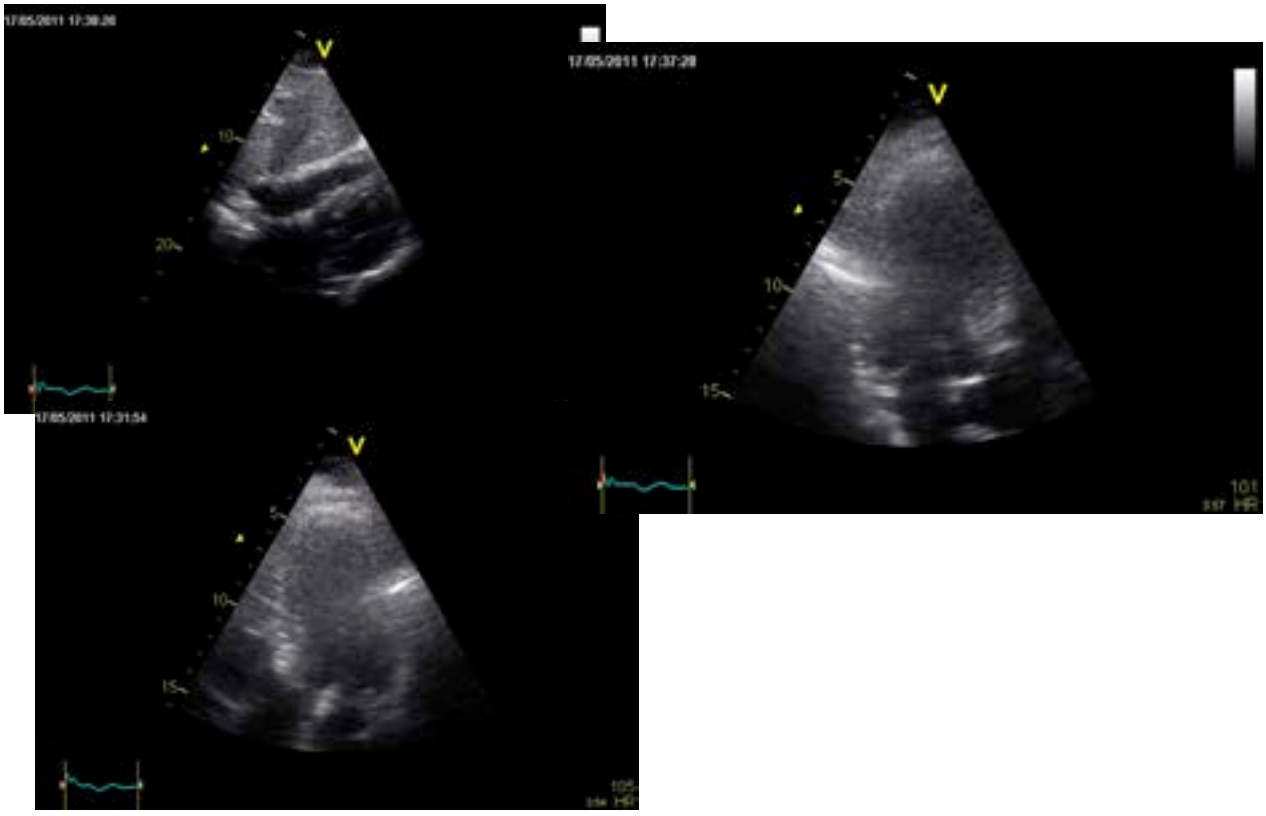
3.Stent



Dominante RCA rekanalisiert

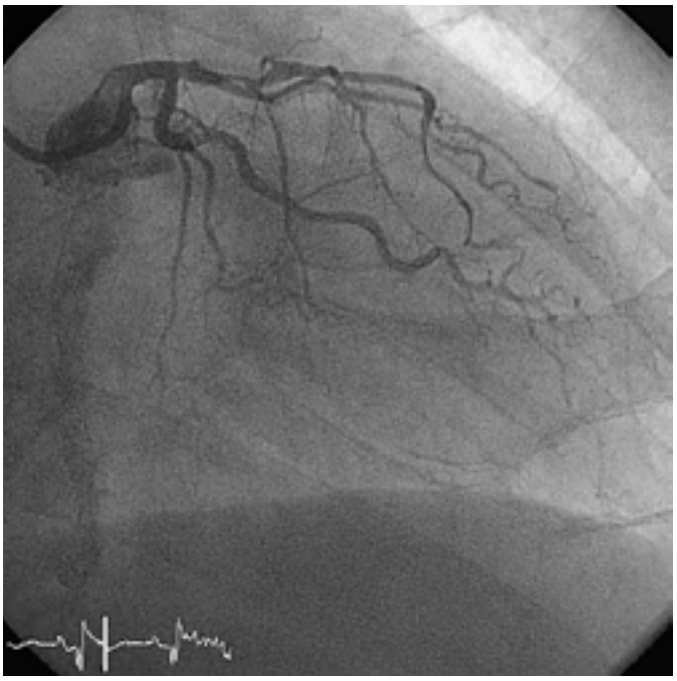


Echo nach PCI



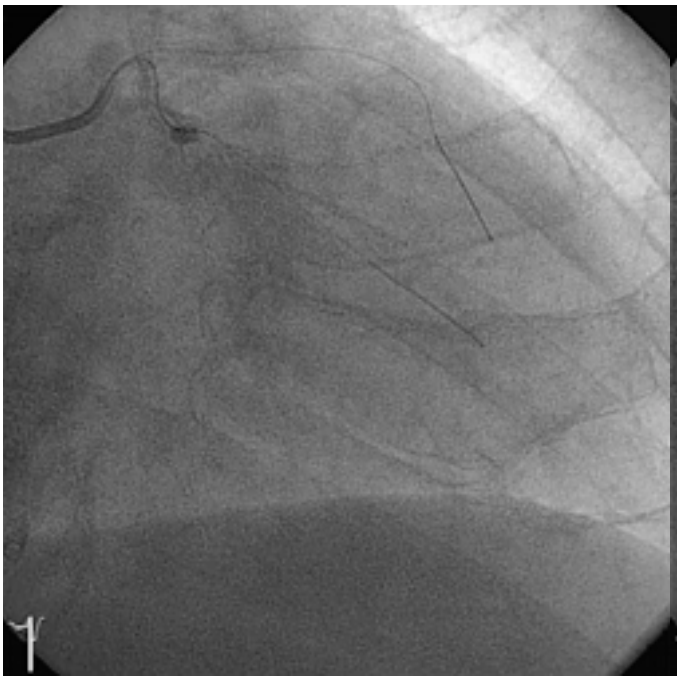
Einige Tage später ...

LAD und CX 90%

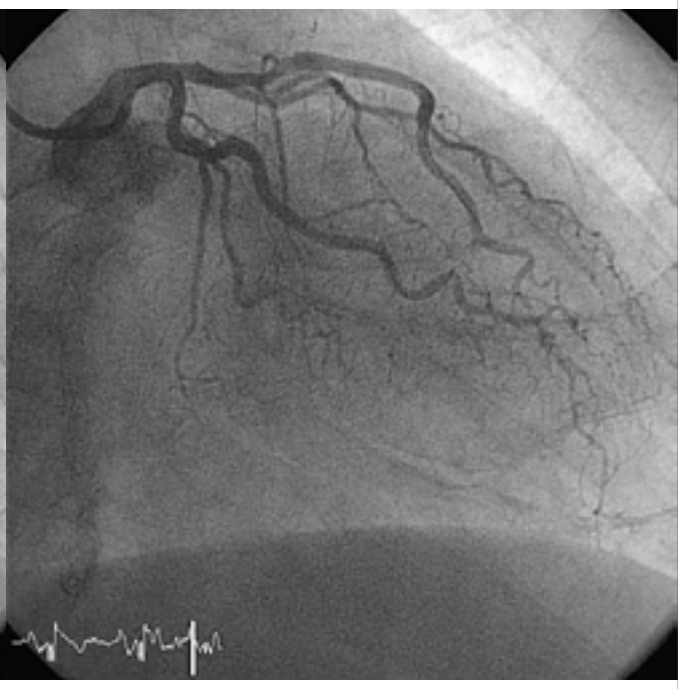


LAD Stent

CX Stent



LAD und CX frei



26.5.2011

Rehabilitation/Prävention

Lebensstil
Bewegung
Ernährung
Entspannung

Ausdauertraining

bewirkt EF-Zunahme (Haykowsky, JACC 2007)
verbessert Koronarperfusion (Gielen S, Circ 2001)
reduziert Rehospitalisierungsrate (Piepoli M, BJM 2004)

William Heberden
über einen Patienten,
der an Angina pectoris litt,
am 21. Juli 1768:

Ich habe einen kennengelernt, der es sich zur Aufgabe machte,
jeden Tag eine halbe Stunde lang Holz zu sägen,
er wurde beinahe geheilt.

Danke
für Ihre
Aufmerksamkeit!