PERAWATAN LANJUT

- Mobilisasi: duduk, makan, jalan
- Pengawasan komplikasi lain :
  - Tromboemboli : deep vein, pulmo, sistemik,
  - Gagal jantung
  - Perikarditis
  - Aritmia ventrikuler
  - Angina dan iskemi pasca inrfak

Rekomendasi menejemen dan terapi awal NSTEMI

Secara umum obati pasien dgn :
- **Antitrombin (heparin)**
- **Antiplatelet (aspirin)**

Antitrombin (heparin)
plus
Antiplatelet (aspirin)

Ubahlah terapi ini jika pasien berada didalam kreteria resiko tinggi

Kriteria resiko tinggi
- **Depresi ST = 1 mm dan abnormalitas EKG yg menetap**
- **Gejala menetap, iskemia berulang**
- **Fungsi Vki menurun**
- **Gagal jantung kongesti**
- **Pelepasan marker serum : troponin / CKMB**
- **Resiko PJK = 3**
- **Riwayat stenosis koroner = 50%**

Sumber : Medika No. 2, Pebruari 2003, p.114

Rekomendasi menejemen dan terapi awal NSTEMI

Pasien dengan resiko tinggi akan bermanfaat dengan pengobatan :
- Aspirin dan
- LMWH atau dan
- Kombinasi

Penghambat glikoprotein IIb/IIIa dengan UFH

→ Antitrombin (heparin) plus Antiplatelet (aspirin) plus Penghambat glikoprotein IIb/IIIa

Semua pasien tanpa kontraindikasi hrs mendapat

→ Penghambat beta

Pasien yg penderita angina berulang juga hrs mendapat

→ Nitrat

Agen ketiga yg digunakan pd angina refrakter / pasien dgn kontraindikasi penghambat beta

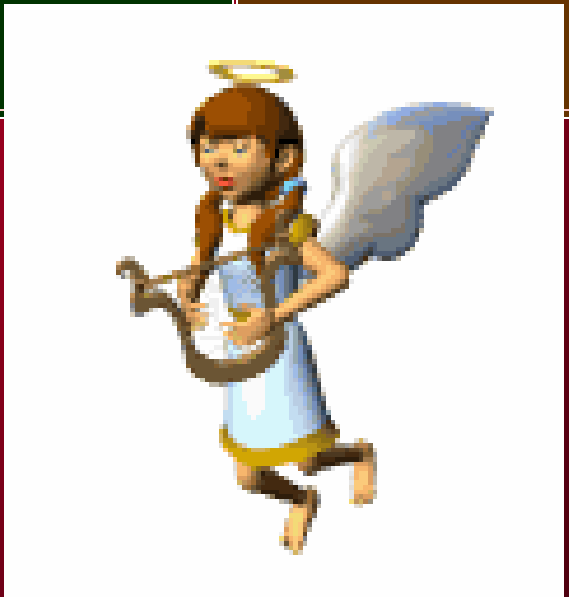
→ Penyekat kanal kalsium

Sumber : Medika No. 2, Pebruari 2003, p:114

Agen-agen fibrinolisis yang sering dipakai

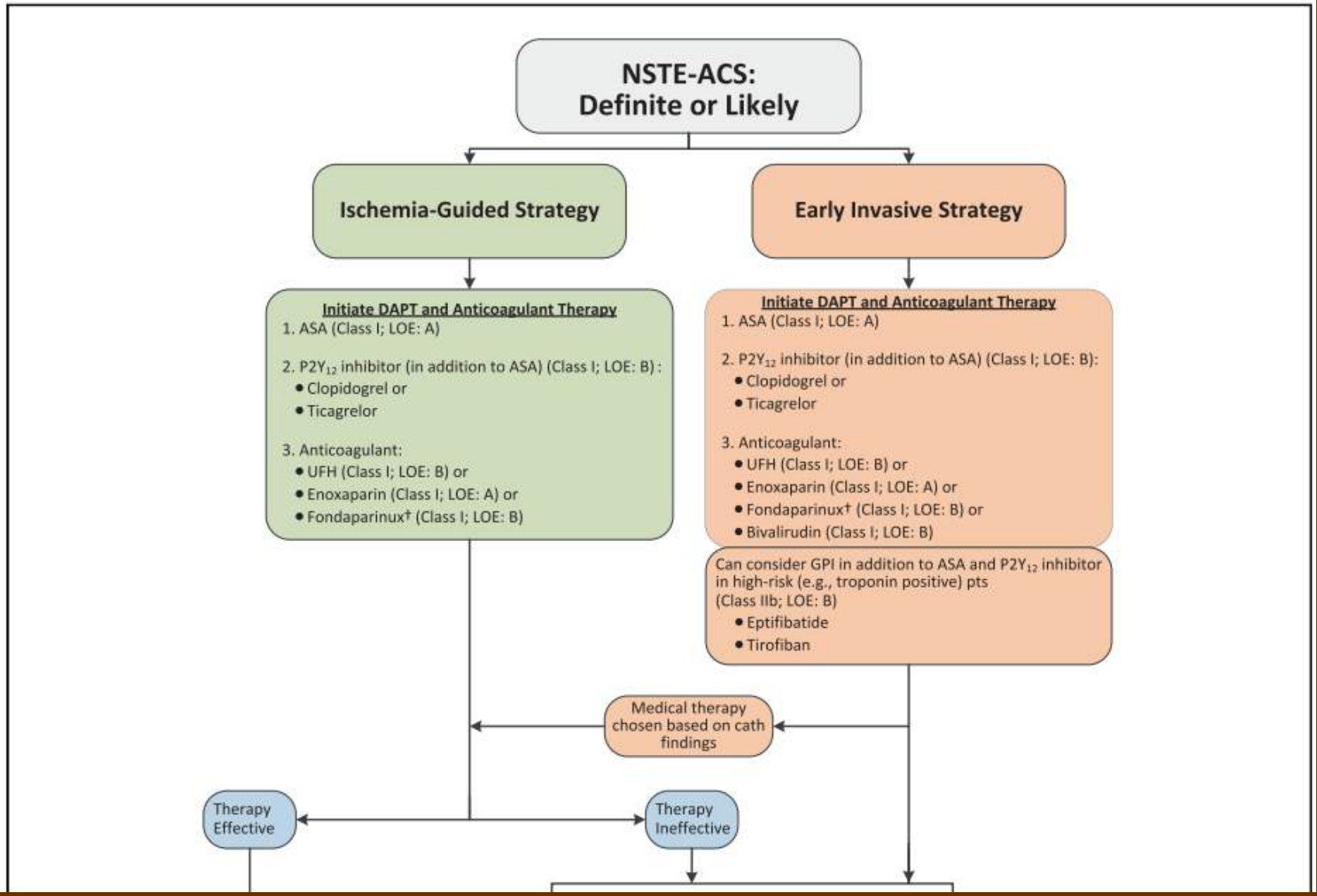
Fibrinolitik	Nama Dagang	Cara Pemberian	Terapi Anjuran
Streptokinase r-TPA	Streptase, Kabinikase Alteplase, Actylase	1,5 juta unit dlm 1 jam 15 mg bolus, 0.75 mg/kg/30 menit pertama (maks 50 mg), 0.5 mg/kg/60 menit kedua (maks 35 mg)	Heparinisasi pasca fibrinolisis, 5000 unit bolus. 1000 unit/jam dgn a-PTT 1,5 - 2 kali kontrol

r-TPA = recombinant tissue plasminogen activator
a-PTT = activated partial thromboplastin time



Sumber : Medika No. 2, Pebruari 2003, p:115

ACC / AHA 2014 Guidelines Update Untuk UA dan NSTEMI



ACC / AHA 2014 Guidelines Update Untuk UA dan NSTEMI

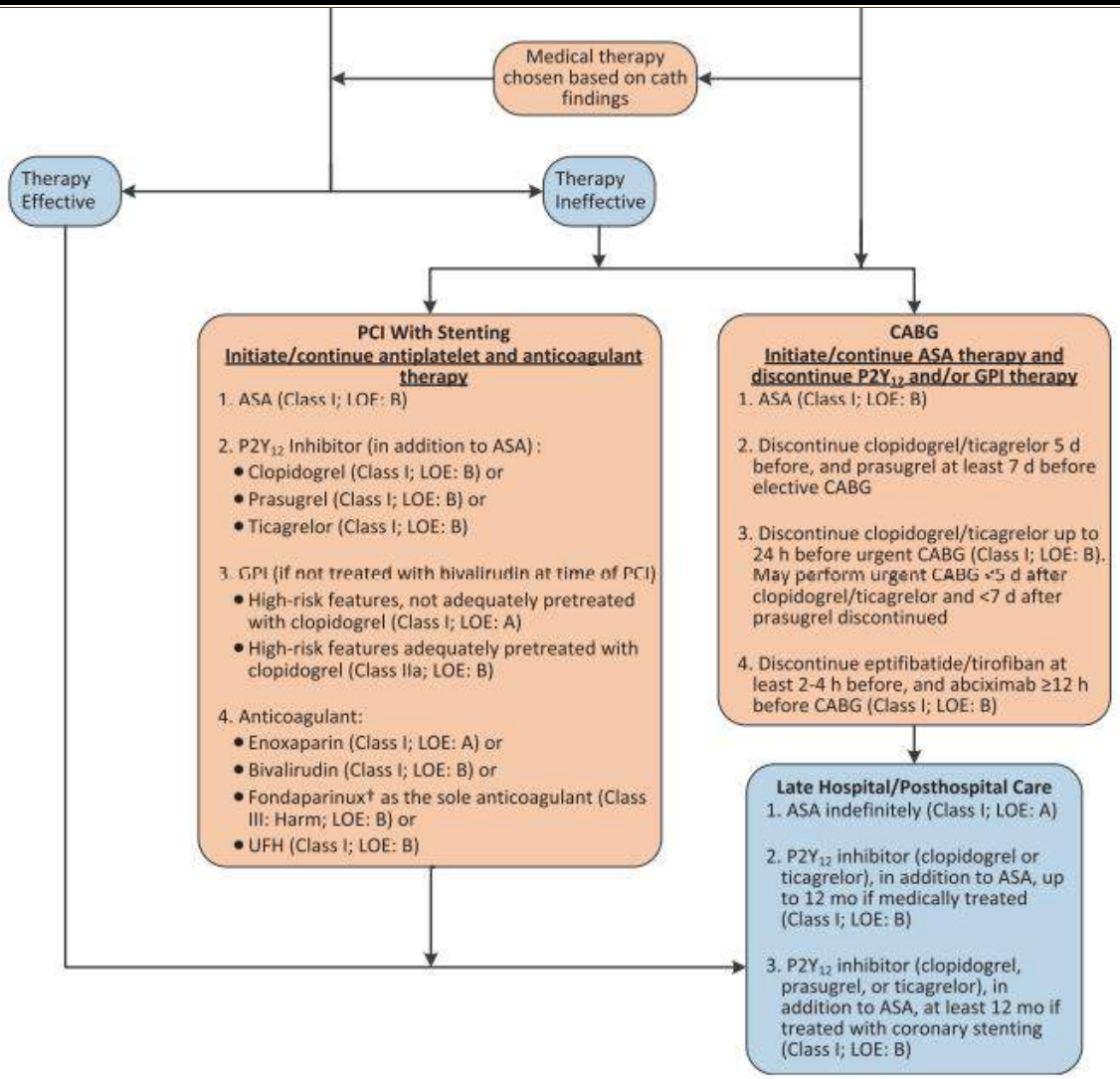
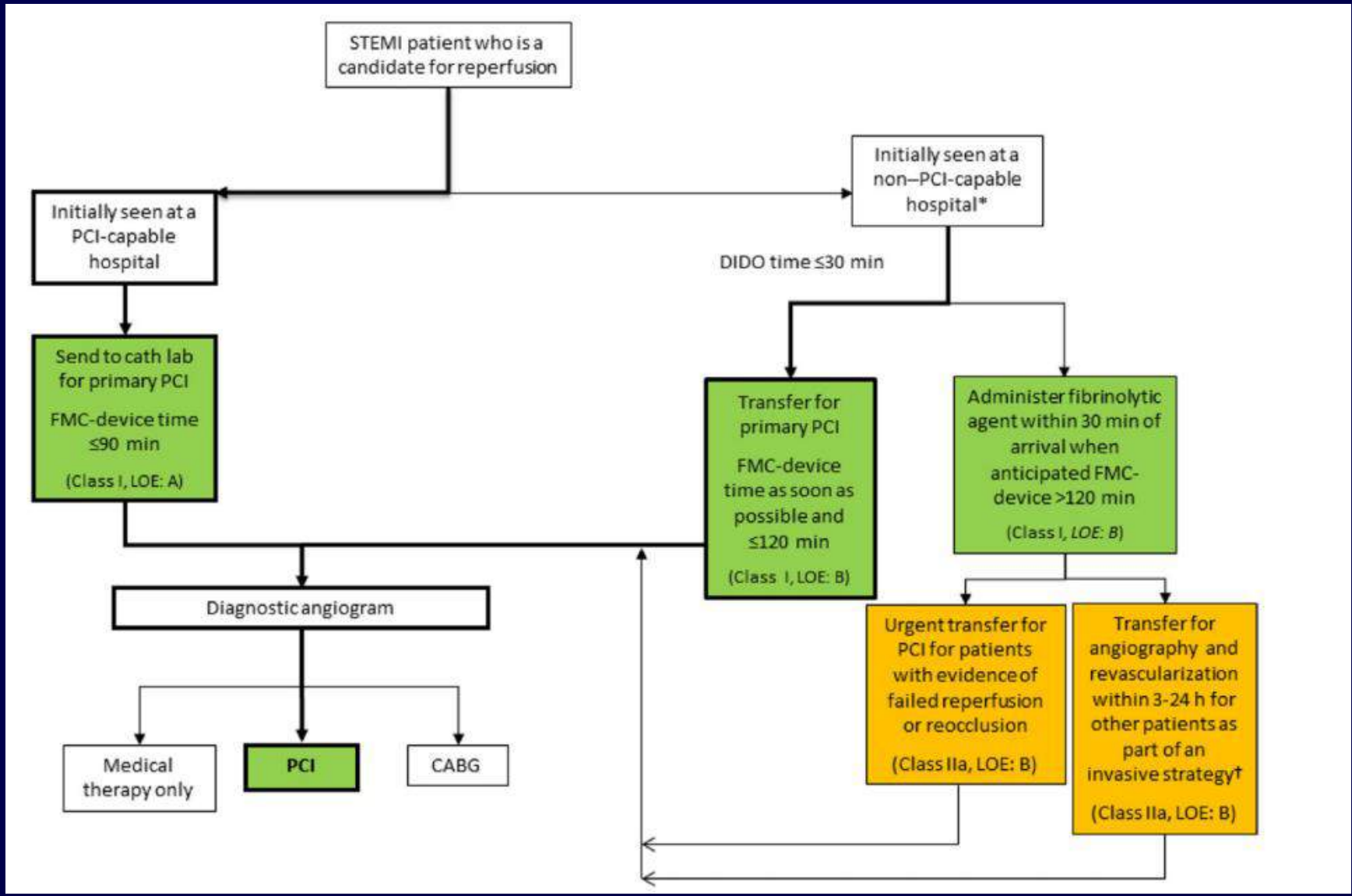


FIGURE 3 Algorithm for Management of Patients With Definite or Likely NSTE-ACS*

ACC/AHA 2013 Guidelines of Management UPDATE for STEMI



OBAT-OBAT ANTIPLATELET

Thromboxane A_2 inhibitor
- Acetylsalicylic acid (ASA)

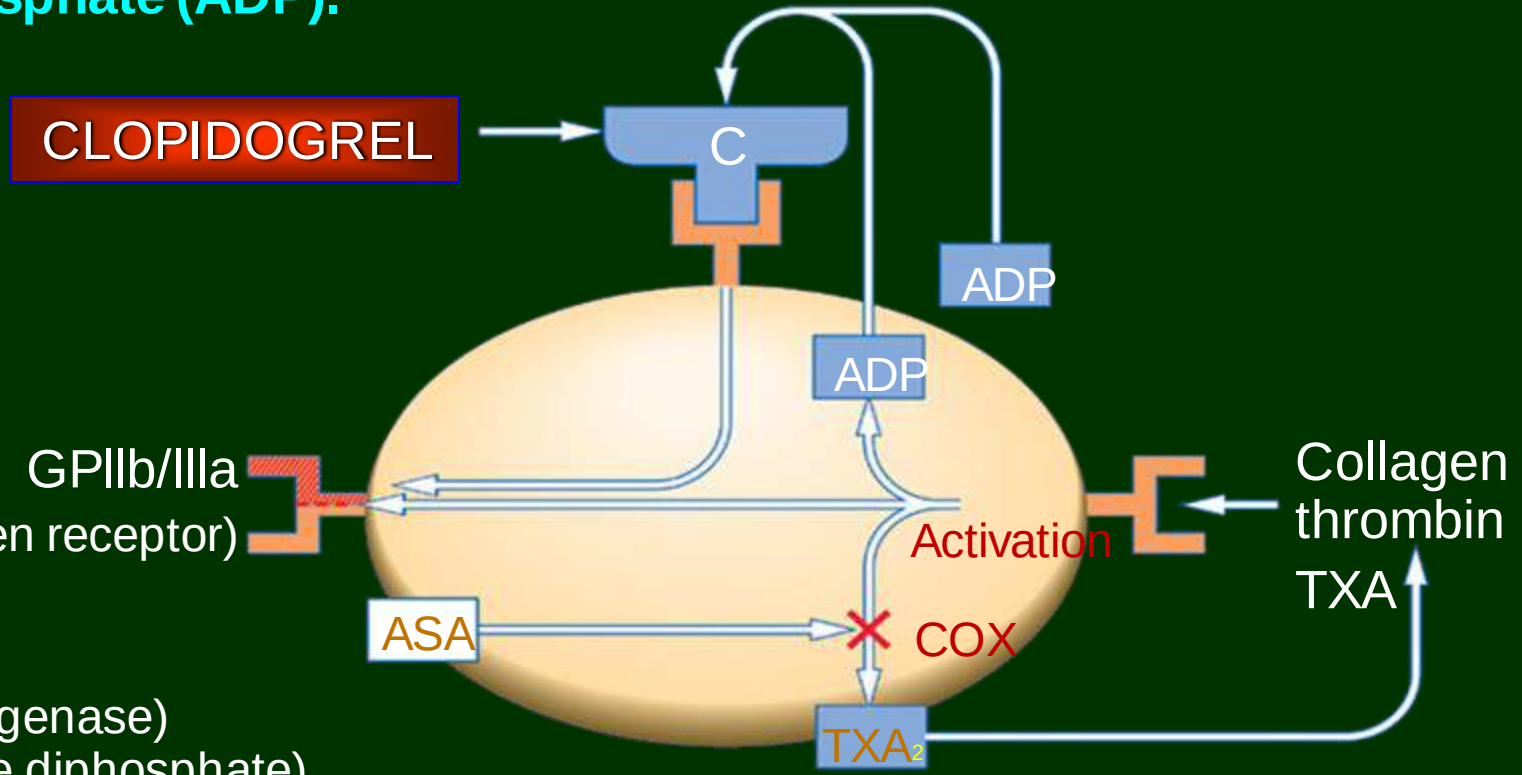
Phosphodiesterase inhibitor
- Dipyridamole

Glycoprotein (GP) Iib/IIIa blockers
- Parenteral: abciximab, eptifibatide, tirofiban

ADP-receptor antagonist
- Clopidogrel (plavix)
- Ticlopidine (ticlid, agulan, ticuring)

ASPIRIN

- Irreversibly inhibits cyclooxygenase and prevents synthesis of thromboxane A_2.
- Effect lasts for the lifetime of the platelet (about 7-10 days).
- Does not prevent aggregation caused by thrombin.
- Only partially inhibits aggregation induced by adenosine diphosphate (ADP).

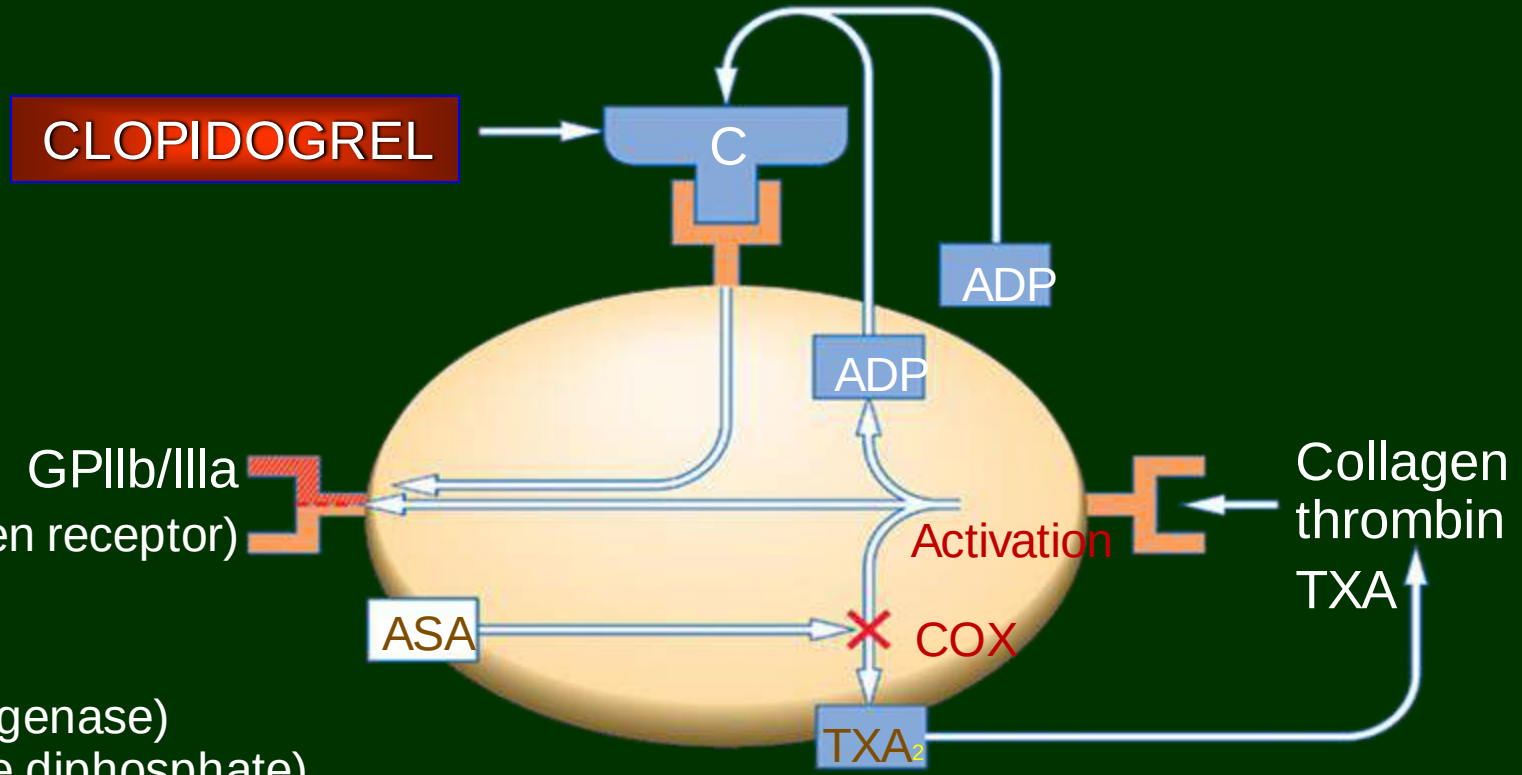


COX (cyclo-oxygenase)
ADP (adenosine diphosphate)
TXA_2 (thromboxane A_2)

Jarvis B, Simpson K. Drugs 2000; 60: 347–77.

CLOPIDOGREL

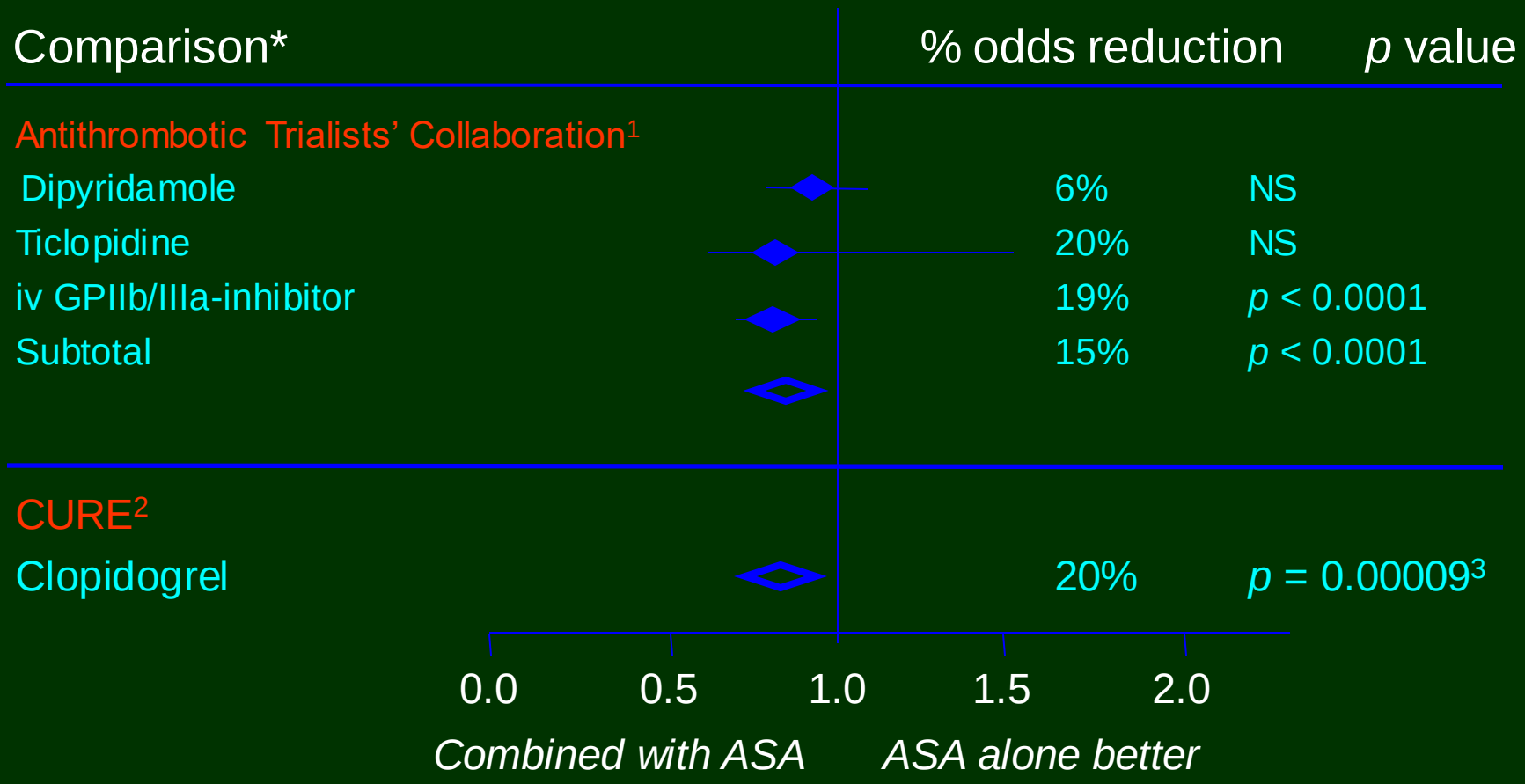
- Platelet adenosine diphosphate (ADP) receptor antagonist.
- Reduced ADP binding inhibits platelet aggregation.
- Also inhibits aggregation stimulated by thrombin, platelet activating factor, and collagen.
- Platelet effects lasts for the lifetime of the platelet (~7-10 days).



COX (cyclo-oxygenase)
ADP (adenosine diphosphate)
TXA_2 (thromboxane A_2)

Jarvis B, Simpson K. Drugs 2000; 60: 347–77.

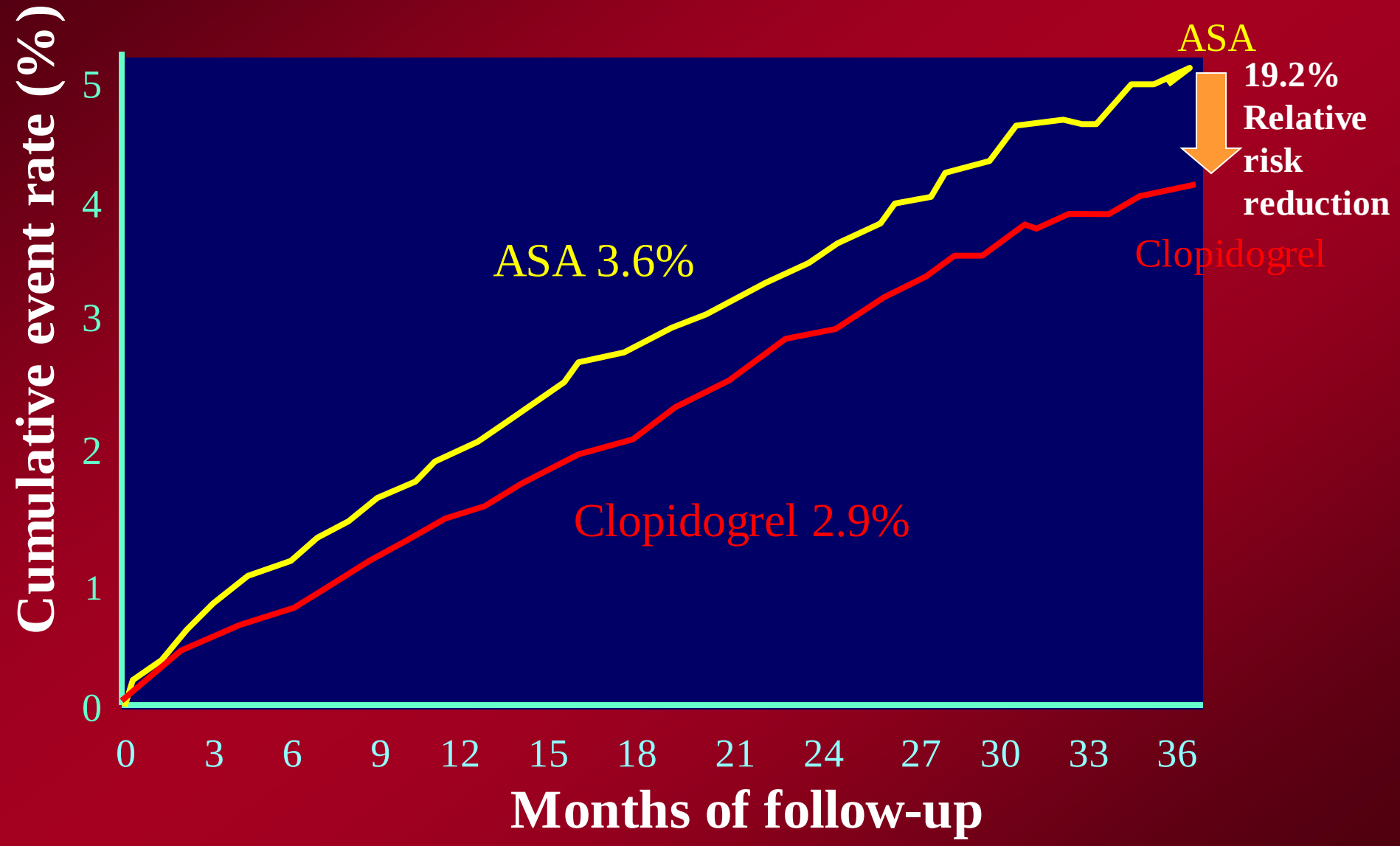
Effects of different antiplatelet agents combined with aspirin vs. aspirin alone



*In combination with ASA vs ASA alone

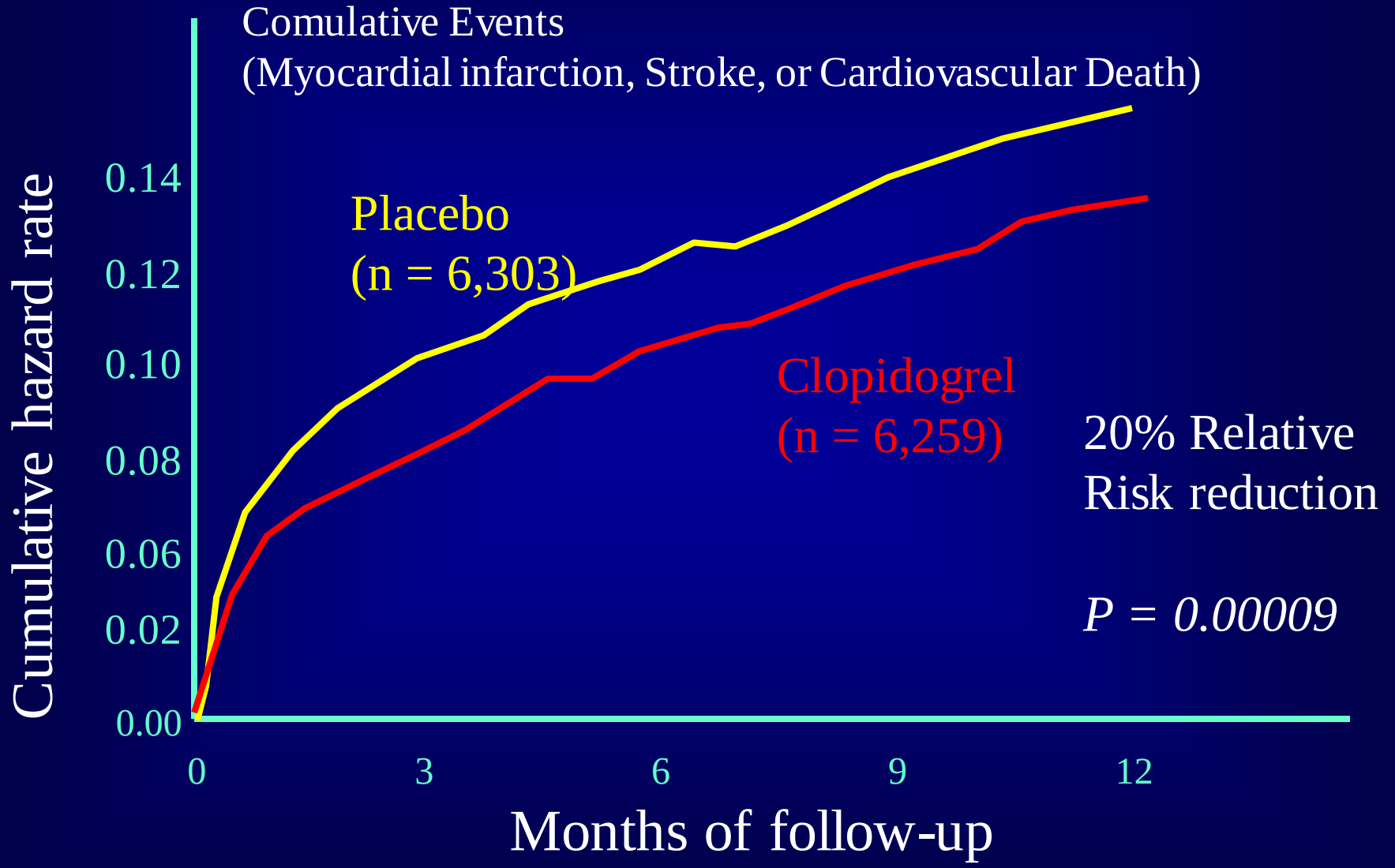
1. Antithrombotic Trialists' Collaboration. *BMJ* 2002; 324: 71–86. 2. The CURE Trial Investigators.
N Engl J Med 2001; 342: 494–502. 3. Data on file, 2002: p73 internal CSR-EFC 3307.

CAPRIE : Benefit of Clopidogrel over ASA in the Reduction of Myocardial Infarction

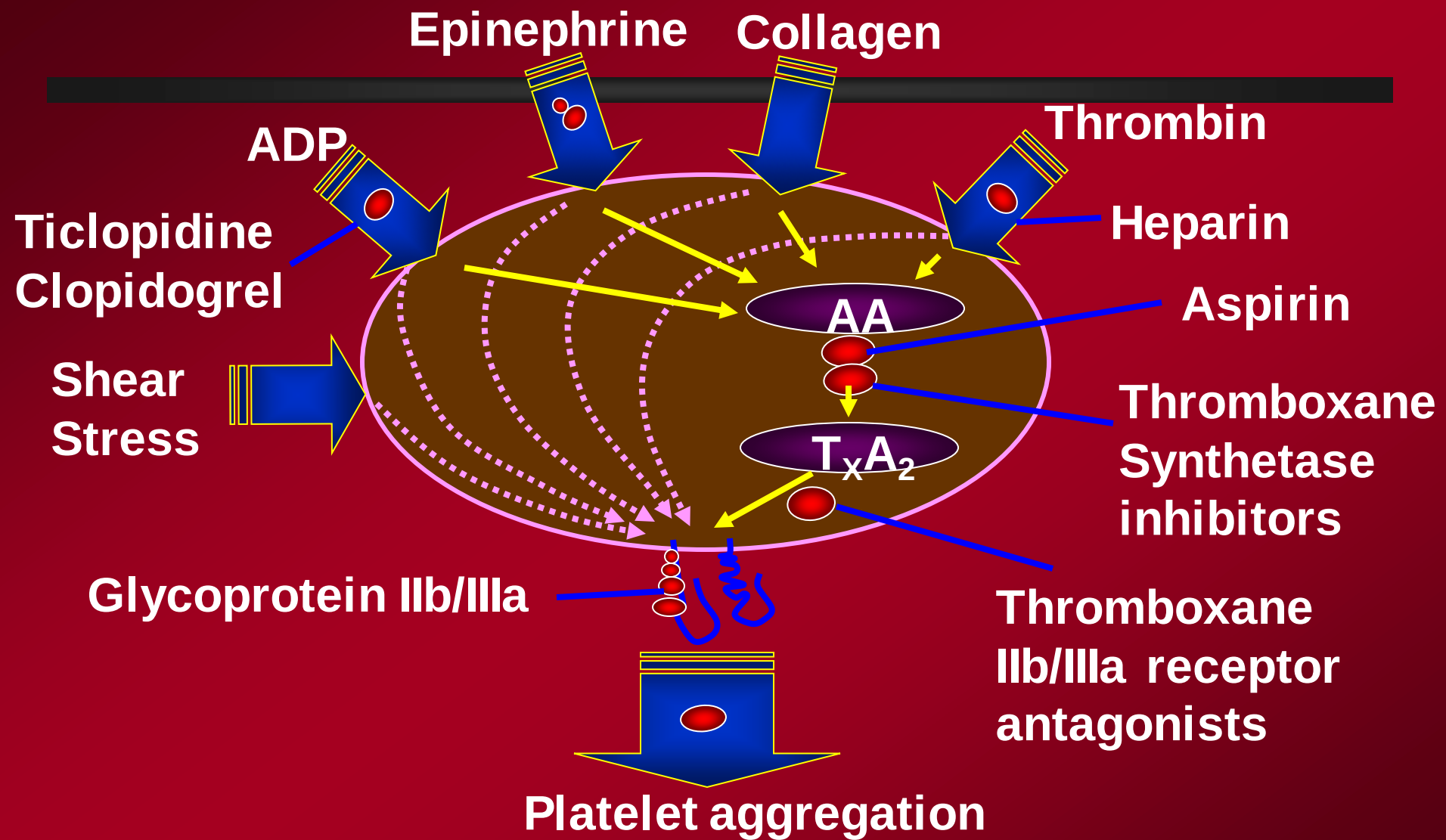


Gent M. Circulation 1997: 96 (suppl 8) : I-467

CURE : Benefits Clopidogrelc



The CURE Trial Investigators. N Engl J Med 2001; 345-502, 2 data on file 2002, p73 internal CSR-EFC 3307


Epinephrine
Collagen
ADP
Thrombin
Ticlopidine
Clopidogrel
Heparin
AA
Aspirin
Shear
Stress
Thromboxane
Synthetase
inhibitors
T_xA_2
Glycoprotein IIb/IIIa
Thromboxane
IIb/IIIa receptor
antagonists
Platelet aggregation

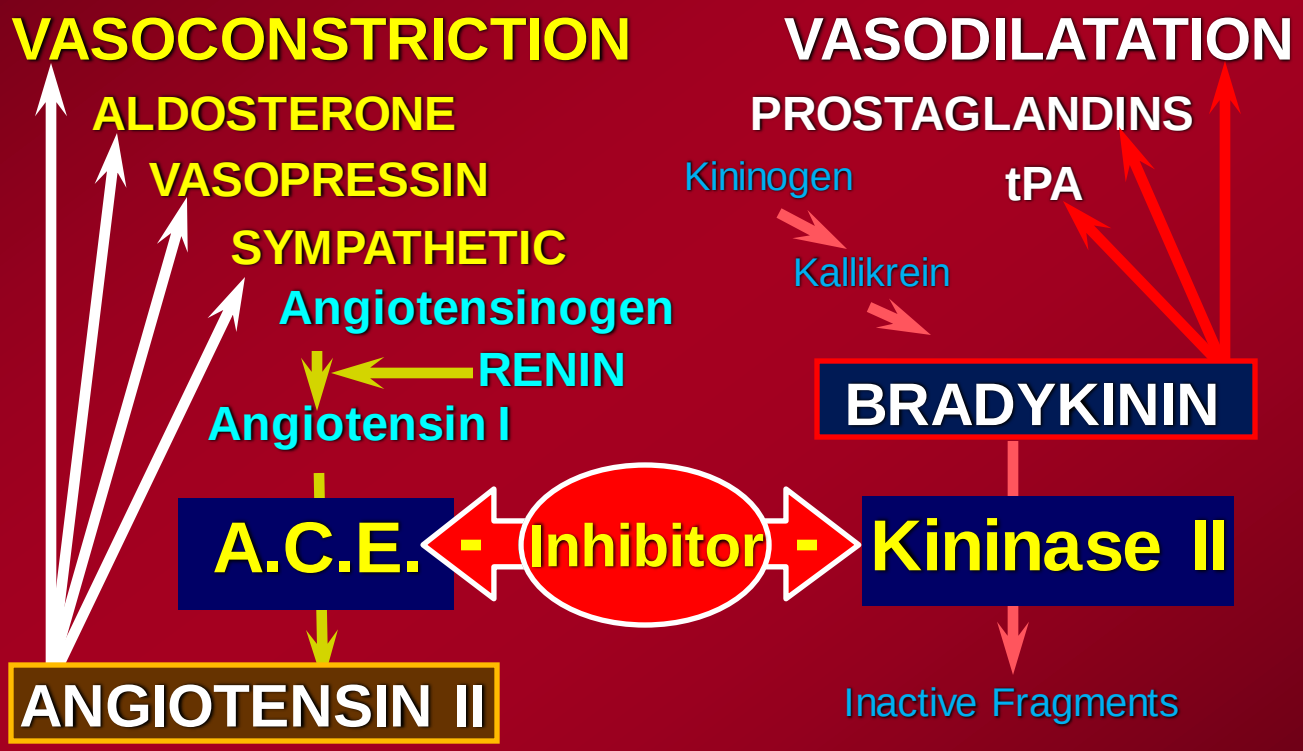
Potential Therapy for Endothelial Dysfunction

- Lipid Lowering Therapy
- ACE inhibitors
- Estrogen
- Antioxidant vitamins
- L-arginine
- Exercise
- Stop smoking
- Tight control of diabetes mellitus
- Folic acid for hyperhomocysteinemia
- Low fat diet

(Lawrence GS, Cardiology update and interventional cardiology)

(Lawrence GS, Cardiology update and interventional cardiology)

ACE Inhibitors
Mechanism of Action



Dosages of ACE-Inhibitors after Myocardial Infarction

- Lisinopril 2.5 to 5 mg/d to start; titrate to10 mg/d or higher as tolerated
- Captopril 6.25 to 12.5 mg 3 times/d to start; titrate to 25 to 50 mg 3 times/d as tolerated
- Ramipril 2.5 mg twice daily to start; titrate to5 mg twice daily as tolerated
- Trandolapril test dose 0.5 mg; titrate up to 4 mg daily as tolerated
- Enalapril 2.5 mg twice daily to start; titrate to 10 to 20 mg twice daily

ACCF/AHA Guideline for The Management of ST-Elevation Myocardial Infarction, 2013
ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation, 2012

Adverse Effect of ACE-Inhibitors Usage

ACE inhibitor side effects

(CAPTOPRIL)

C	Cough
A	Angioneurotic oedema
P	Proteinuria
T	Taste disturbance/ Teratogenic in 1st trimester
O	Other (fatigue, headache)
P	Potassium increased
R	Renal impairment
I	Itch
L	Low BP (1st dose)

Contraindication to ACE-Inhibitors Use during Acute Myocardial Infarction

- Hypotension,
- Shock
- Bilateral renal artery stenosis
- History of worsening of renal function with ACE inhibitor/ARB exposure
- Renal failure
- Drug allergy

ACCF/AHA Guideline for The Management of ST-Elevation Myocardial Infarction, 2013
ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation, 2012

LIPIDS

- Lipids-lowering agents
  - Statins
  - Ezetimibe
  - Proprotein convertase subtilisin/kexin type 9
  - Fibrates

Cosentino F, Grant PJ, Aboyans V, Bailey CJ, Ceriello A, Delgado V, et al. 2019 ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD. Eur Heart J. 2020;41(2):255–323.

PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY (PTCA)

Indikasi klinis : - IMA periode 4 - 6 jam
- AP unstable
- Stenosis rekurens

Angiografik : - 1 vessel, multivessel
- Oklusi total

Komplikasi : okulasi, diseksi koroner, aritmia, restenosis, IMA

Cara : Balloon catheter

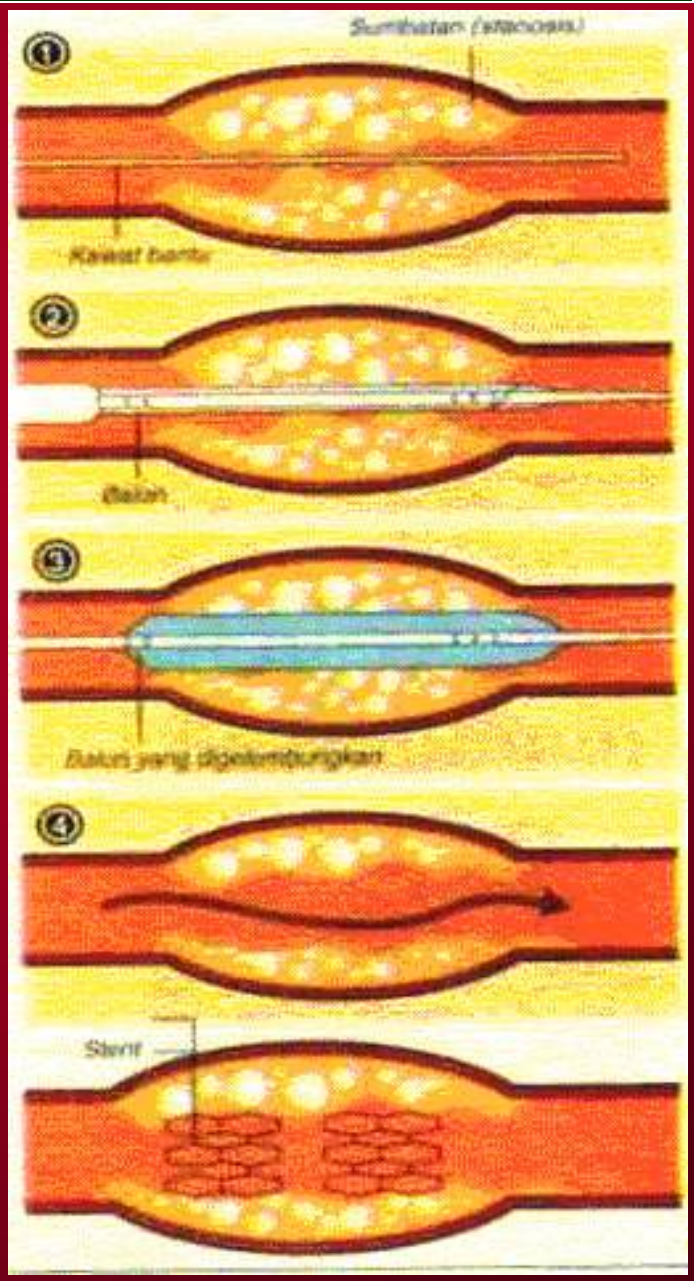
Angioplasti : pemulihan pembuluh darah yg tersebumbat dgn menggunakan “balon” yg bisa dipompa untuk membuka penyumbat / pembuluh darah koroner yg menyempit.

Kawat bantu yg dimasukkan ke dalam pembuluh yg tersumbat

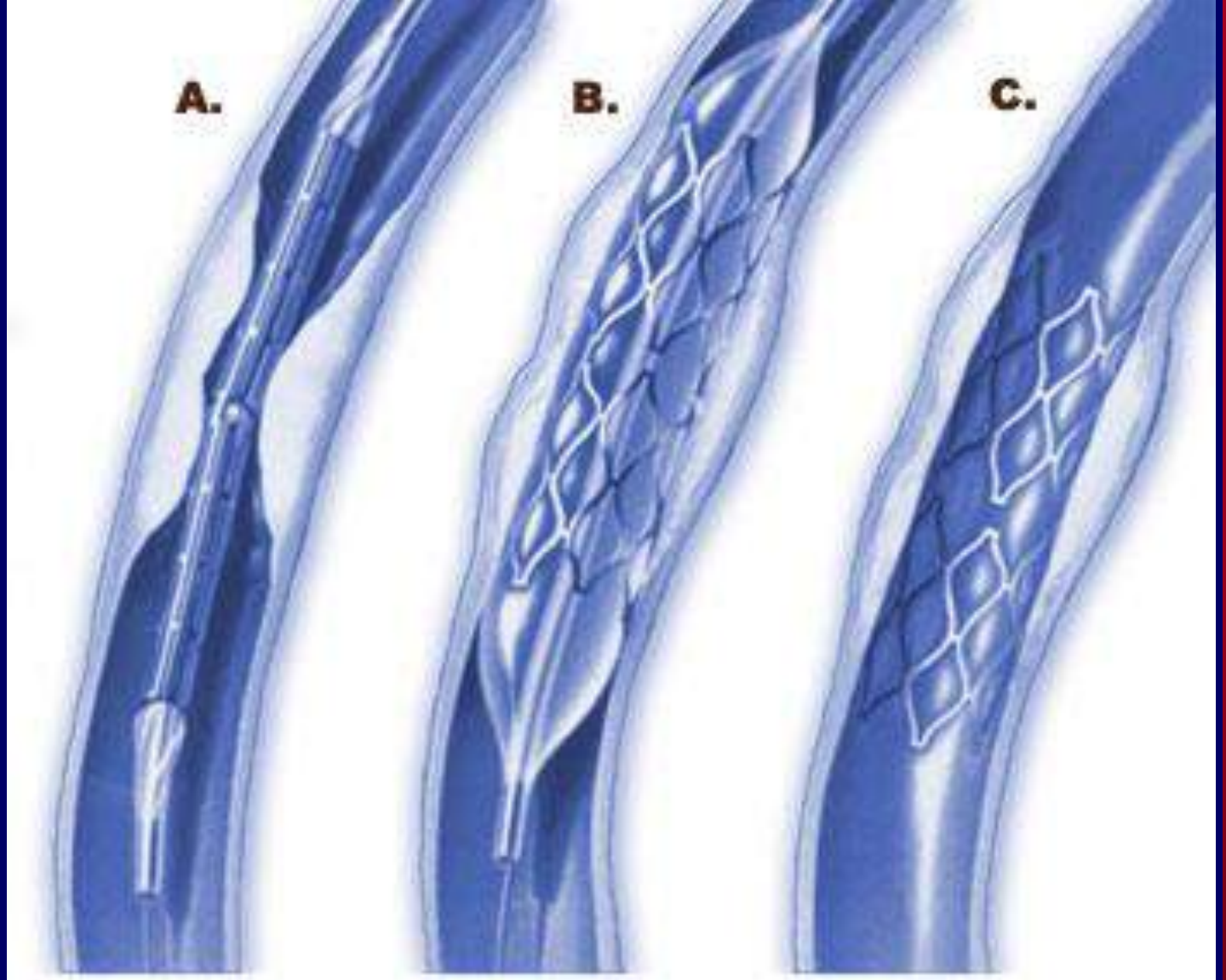
Pipa balon dimasukkan ke dalam sepanjang kawat bantu

Balon ditiup untuk membuka penyumbat

Setelah prosedur tersebut, pembuluh tidak tersumbuat



Untuk mencegah terjadinya pemyumbatan ulang, dipasang stent yaitu semacam cincin yang dipasang secara permanen sebagai penyangga.
Stent bisa dilapisi obat untuk menghambat pertumbuhan sel-sel sehingga tidak terjadi penyempitan



A. Ballon catheter with stent mounted.

B. Stent expanded via balloon.

C. Stent implanted in artery.