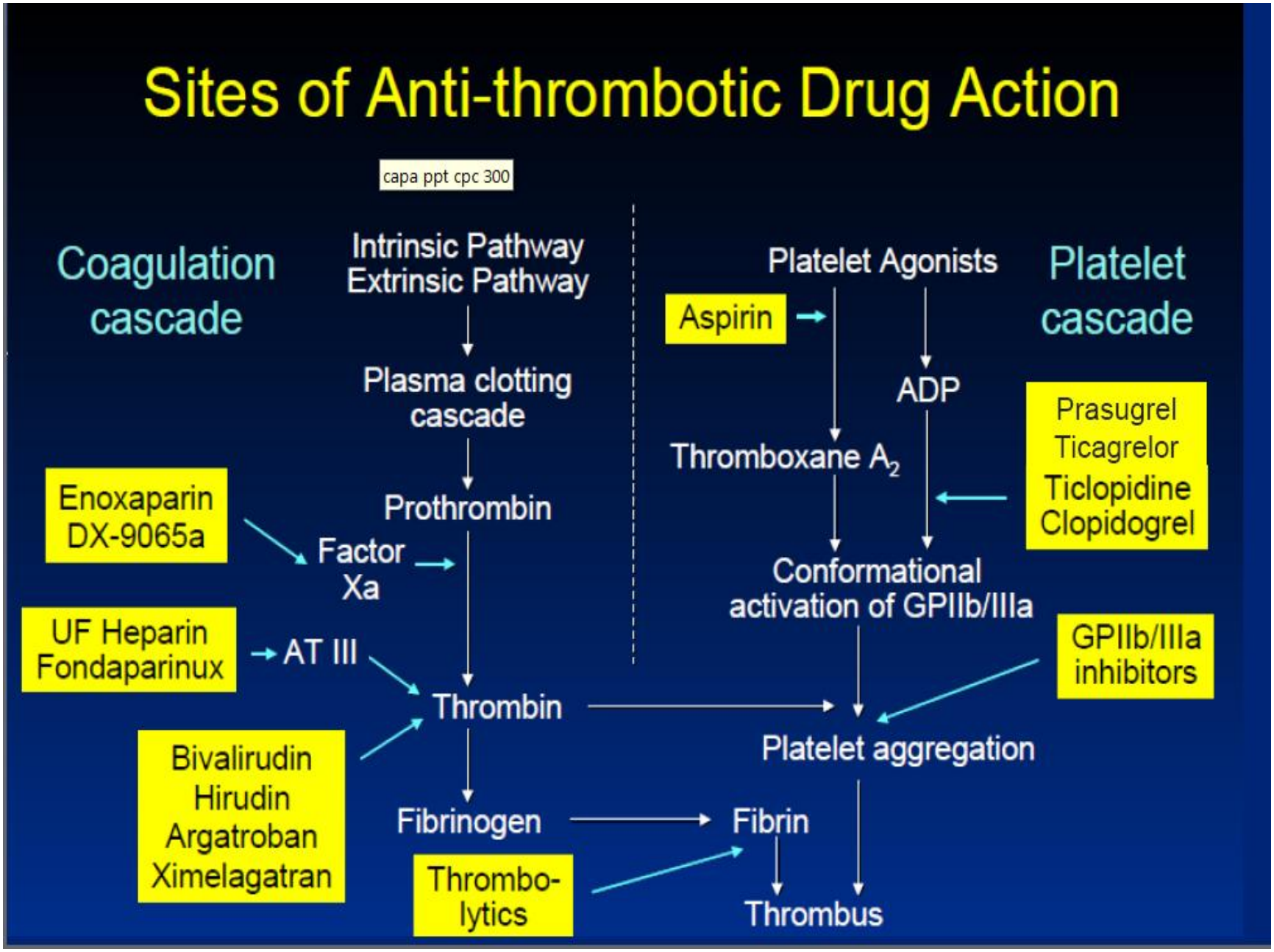
Revisão da terapia com heparinas nas síndromes coronarianas agudas

Reunião HCI Março 2012
José Fábio Fabris Júnior
Renato Sanchez Antonio

Sites of Anti-thrombotic Drug Action

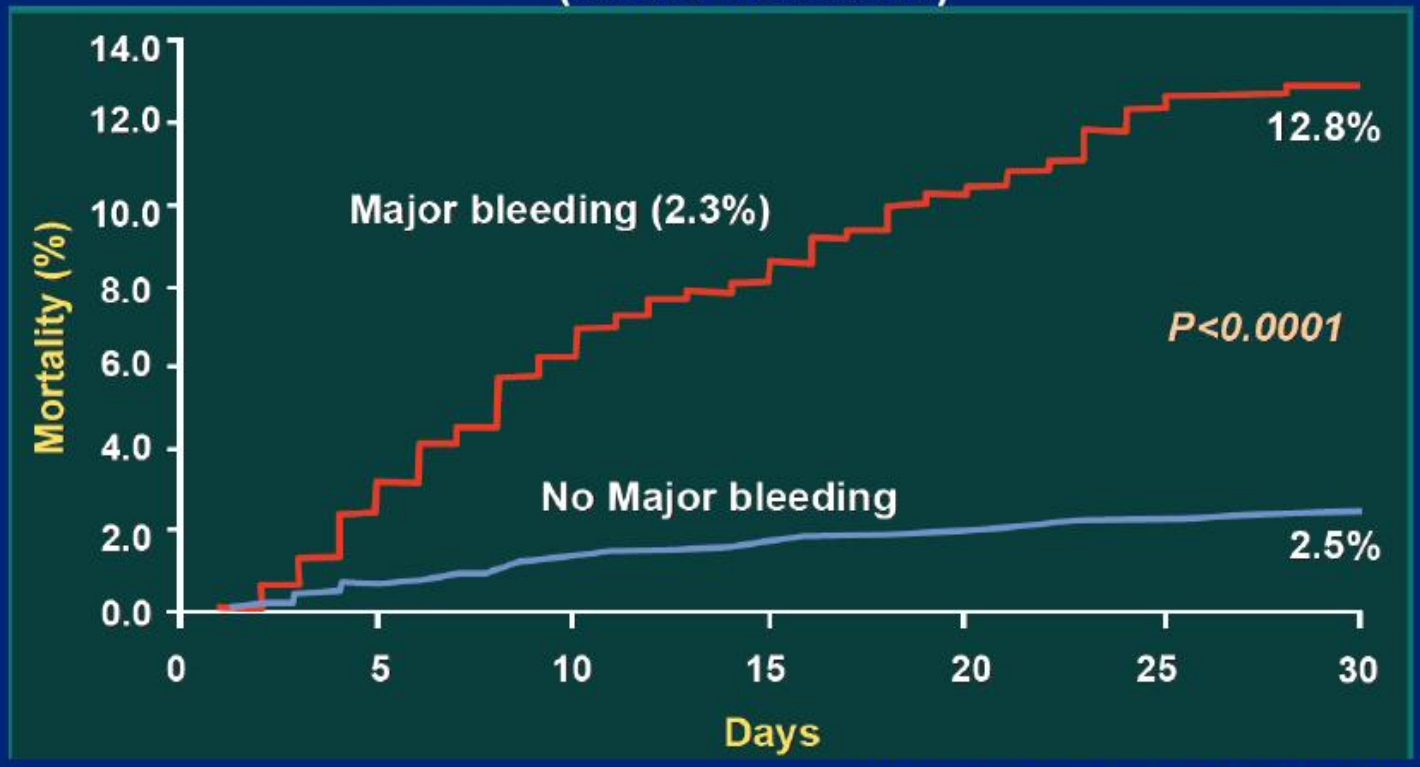


CENTRO DE PESQUISA CARDIOVASCULAR

Terapia Adjuvante

Impacto do Sangramento

Sangramento hospitalar em 34.146 pacientes com SCA (OASIS-1/2 +CURE)



Eikelboom JW. *Circulation* 2006;114:774

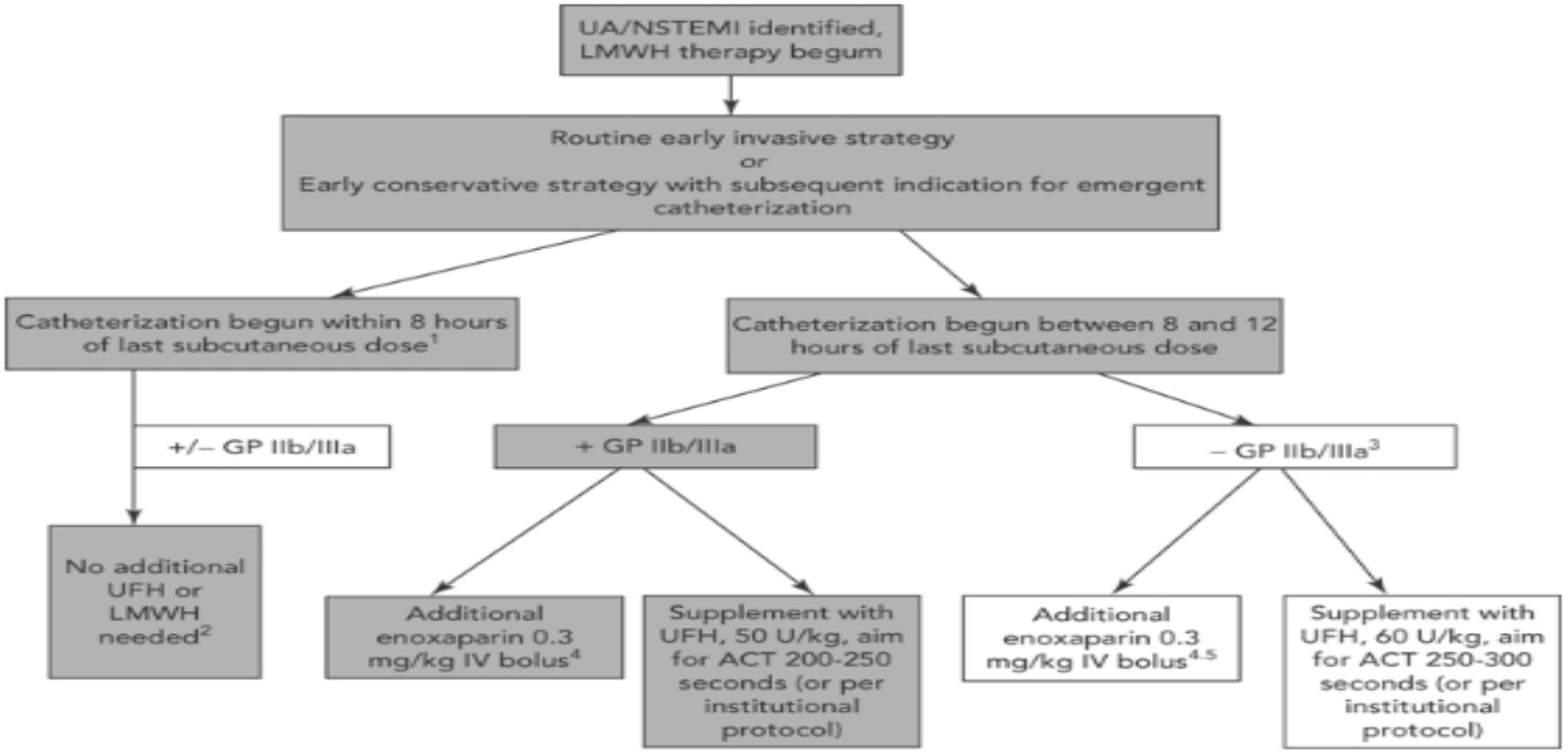


Figure 3.14 Strategies for the transition from medical therapy to procedural anticoagulation in patients receiving low–molecular-weight heparin. UA/NSTEMI, unstable angina/non-ST elevation myocardial infarction; LMWH, low–molecular weight. (From Kereiakes DJ, et al. Low-molecular weight heparin therapy for non-ST-elevation acute coronary syndromes and during percutaneous coronary intervention: an expert consensus. *Am Heart J* 2002;144:621).

Note:

1. For PCI, wait at least 30 to 60 minutes after SC injection, depending on molecular weight of the agent (30 minutes for enoxaparin, 60 minutes for dalteparin).
2. Insufficient data are available to guide heparinization in patients who have received only 1 dose of SC LMWH.
3. Fewer data are available on patients treated with SC enoxaparin and no GP Iib/IIia receptors antagonists undergoing PCI.
4. If the patient has been receiving dalteparin, switch to UFH, as there are no available data on transitioning from medical to interventional therapy when the last SC dose of dalteparin was given 8 to 12 hours before PCI.
5. Consideration can be given to enoxaparin 0.5 mg/kg in those patients not receiving concomitant GP IIb/IIIa receptor antagonist therapy.

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2011 ACCF/AHA Focused Update of the Guidelines for the Management of Patients With Unstable Angina/ Non –ST-Elevation Myocardial Infarction (Updating the 2007 Guideline) : A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

2011 Writing Group Members, R. Scott Wright, Jeffrey L. Anderson, Cynthia D. Adams, Charles R. Bridges, Donald E. Casey, Jr, Steven M. Ettinger, Francis M. Fesmire, Theodore G. Ganiats, Hani Jneid, A. Michael Lincoff, Eric D. Peterson, George J. Philippides, Pierre Theroux, Nanette K. Wenger and James Patrick Zidar

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- As drogas devem ser denominadas anticoagulantes ao invés de antitrombínicos
- ALERTA INICIAL:
- A) Estratégia anticoagulante ideal ?
- - Incertezas, equivalência de doses, duração da terapia, uso prévio de terapia anticoagulante (efeito residual)
- - Administração de regimes antiplaquetários distintos

- PREOCUPAÇÃO ADICIONAL
- - Os trabalhos baseiam-se no critério atual de não inferioridade (equivalência) entre as terapias (“margem de não inferioridade”)
- - Dificuldade atual de trabalhos controlados por placebo
- - Recomenda-se cautela na avaliação dos resultados de não inferioridade

- Classe I: Estratégias anticoagulantes aceitáveis
- - a preferência por uma estratégia particular não é clara, devendo-se levar em consideração fatores tais como: erros de medicação, anticoagulação dupla, risco de sangramento, familiaridade local (particulamente se ATC planejada), necessidade de reversão do efeito anticoagulante

- Comitê redator desta diretriz julga, com base nos atuais estudos, ser difícil determinar a terapêutica anticoagulante ideal nos pacientes alocados para estratégia invasiva precoce
- Na estratégia não invasiva os resultados são menos confusos e apontariam em termos de preferência a utilização de foundaparinux, enoxaparina e HNF
- A bivalirudina não foi testada na estratégia não invasiva
- A duração de terapia anticoagulante permanece incerta

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association
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2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention : A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions

Writing Committee Members, Glenn N. Levine, Eric R. Bates, James C. Blankenship, Steven R. Bailey, John A. Bittl, Bojan Cercek, Charles E. Chambers, Stephen G. Ellis, Robert A. Guyton, Steven M. Hollenberg, Umesh N. Khot, Richard A. Lange, Laura Mauri, Roxana Mehran, Issam D. Moussa, Debabrata Mukherjee, Brahmajee K. Nallamothu and Henry H. Ting

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Class IIb

1. In patients undergoing elective PCI with stent implantation treated with UFH and adequately pretreated with clopidogrel, it might be reasonable to administer a GP IIb/IIIa inhibitor (abciximab, double-bolus eptifibatide, or high-bolus dose tirofiban).[618,622–624] *(Level of Evidence: B)*

See Online Data Supplement 24 for additional data regarding IV antiplatelet therapy.

In the era before DAPT, trials of adequately dosed GP IIb/IIIa inhibitors in patients undergoing balloon angioplasty and coronary stent implantation demonstrated a reduction in the incidence of composite ischemic events with GP IIb/IIIa treatment, primarily through a reduction of enzymatically defined MI.[613,615,618,620,621] Earlier RCTs of GP IIb/IIIa inhibitors were generally conducted in patients treated with UFH. In some trials, use of GP IIb/IIIa inhibitors are associated with some increased bleeding risk, and trials of these agents have generally excluded patients at high risk of bleeding (e.g. coagulopathy).[584,587–589,613–618,620–626] Thus, recommendations about use of GP IIb/IIIa inhibitors are best construed as applying to those patients not at high risk of bleeding complications. Abciximab, double-bolus eptifibatide (180 mcg/kg bolus followed 10 minutes later by a second 180 mcg/kg bolus), and high-bolus dose tirofiban (25 mcg/kg) all result in a high degree of platelet inhibition,[627–629] have been demonstrated to reduce ischemic complications in patients undergoing PCI,[608,609,613,615,618–621] and appear to lead to comparable angiographic and clinical outcomes.[630,631]

Trials of GP IIb/IIIa inhibitors in the setting of STEMI and primary PCI were conducted in the era before routine stenting and DAPT. The results of these and more recent trials, as well as several meta-analyses, have yielded mixed results.[584–590] Therefore, it is reasonable to administer GP IIb/IIIa inhibitors in patients with STEMI undergoing PCI, although these agents cannot be definitively recommended as routine therapy. These agents might provide more benefit in selective use, such as in patients with large anterior MI and/or large thrombus burden. Trials of precatheterization laboratory (e.g. ambulance or emergency room) administered GP IIb/IIIa inhibitors in patients with STEMI undergoing PCI, with or without fibrinolytic therapy, have generally shown no clinical benefit, and GP IIb/IIIa inhibitor use in this setting may be associated with an increased risk of bleeding.[605–610,612] Studies of intracoronary GP IIb/IIIa inhibitor administration (predominantly using abciximab) consist of several small RCTs, retrospective analyses, retrospective and prospective registries, cohort analyses, and case reports. Although most of these published studies have reported some benefit of intracoronary administration in terms of acute angiographic parameters, infarct size, left ventricle myocardial salvage, and composite clinical endpoints, several other studies have not detected any benefit with intracoronary administration.[589,591–604]

Trials of GP IIb/IIIa inhibitors in patients with UA/NSTEMI undergoing PCI demonstrated reduced ischemic outcomes, particularly in those with high-risk features such as positive biomarkers. Most trials were conducted in a prior PCI era and without $P2Y_{12}$ inhibitor pretreatment,[613,615,618,632,633] although several trials have also demonstrated benefit in patients with high-risk features pretreated with clopidogrel.[614,619] In most older studies of stable patients undergoing balloon angioplasty or coronary stenting, treatment with GP IIb/IIIa inhibitors resulted in a reduction of composite ischemic events, primarily enzymatically defined MI.[613–618,620,621,634,635] More contemporary trials of patients pretreated with a thienopyridine have not demonstrated any benefit with GP IIb/IIIa inhibitor therapy in patients with stable symptoms undergoing elective PCI.[619,622–624]

5.7.4. Anticoagulant Therapy

5.7.4.1. Use of Parenteral Anticoagulants During PCI: Recommendation

Class I

1. An anticoagulant should be administered to patients undergoing PCI. *(Level of Evidence: C)*

Anticoagulant therapy prevents thrombus formation at the site of arterial injury, on the coronary guidewire, and in the catheters used for PCI.[8] With rare exceptions,[626] all PCI studies have used some form of anticoagulant. It is the consensus of the writing committee that PCI be performed with the use of some form of anticoagulant therapy. Suggested dosing regimens of parenteral agents used in PCI are given in Table 12. Recommendations for antiplatelet and antithrombin pharmacotherapy in PCI are given in Table 13.

5.7.4.2. UFH: Recommendation

Class I

1. Administration of IV UFH is useful in patients undergoing PCI. *(Level of Evidence: C)*

As the only anticoagulant available for PCI for many years, UFH became the standard of care by default.[8] The dose of UFH for PCI has been based on empiricism and experience from RCTs. Suggested UFH dosing regimens are given in Table 12. When UFH is used during PCI, most cardiologists assess the degree of anticoagulation by measuring the activated clotting time. Although measurements are useful to show that an anti-IIa anticoagulant has been given, the value of the activated clotting time in current practice has been questioned. Although studies in the balloon angioplasty era did demonstrate a relationship between activated clotting time levels and ischemic complications,[653–655] more recent analyses from the coronary stent era have not found a clear relationship between activated clotting time and outcomes.[349,656,657] There may, however, be a modest relation between bleeding and activated clotting time levels.[349,657] In addition, not only are there differences between activated clotting time levels measured by Hemochron and HemoTec devices, but both devices have less than optimal precision.[658] Thus, although traditional target activated clotting time levels are included in this document, the utility of measured activated clotting time levels in current practice should be considered uncertain.

Table 12. Dosing of Parenteral Anticoagulants During PCI

Drug	Patient Has Received Prior Anticoagulant Therapy	Patient Has Not Received Prior Anticoagulant Therapy
UFH	• IV GPI planned: additional UFH as needed (eg, 2000 to 5000 U) to achieve an ACT of 200 to 250 s • No IV GPI planned: additional UFH as needed (eg, 2000 to 5000 U) to achieve an ACT of 250 to 300 s for HemoTec, 300 to 350 s for Hemochron	• IV GPI planned: 50 to 70 U/kg bolus to achieve an ACT of 200 to 250 s • No IV GPI planned: 70 to 100 U/kg bolus to achieve target ACT of 250 to 300 s for HemoTec, 300 to 350 s for Hemochron
Enoxaparin	• For prior treatment with enoxaparin, if the last SC dose was administered 8 to 12 h earlier or if only 1 SC dose of enoxaparin has been administered, an IV dose of 0.3 mg/kg of enoxaparin should be given. • If the last SC dose was administered within the prior 8 h, no additional enoxaparin should be given.	0.5 to 0.75 mg/kg IV bolus
Bivalirudin	For patients who have received UFH, wait 30 min, then give 0.75 mg/kg IV bolus, then 1.75 mg/kg per h IV infusion.	0.75 mg/kg bolus, 1.75 mg/kg per h IV infusion
Fondaparinux	For prior treatment with fondaparinux, administer additional IV treatment with an anticoagulant possessing anti-IIa activity, taking into account whether GPI receptor antagonists have been administered.	N/A
Argatroban	200 mcg/kg IV bolus, then 15 mcg/kg per min IV infusion	350 mcg/kg bolus, then 25 mcg/kg per min IV infusion

ACT indicates activated clotting time; IV, intravenous; GPI, glycoprotein inhibitor; N/A, not applicable; PCI, percutaneous coronary intervention; SC, subcutaneous; and UFH, unfractionated heparin.

Most cardiologists remove femoral sheaths when the activated clotting time falls to <150 to 180 seconds or when the activated partial thromboplastin time falls to <50 seconds. Full-dose anticoagulation is no longer used after successful PCI procedures. Almost all large clinical trials have enrolled patients who underwent transfemoral PCI, but recent small studies assessing the transradial approach have used similar doses of UFH[659] and similar activated clotting time target levels.[660]

5.7.4.3. Enoxaparin: Recommendations

Class I

1. An additional dose of 0.3 mg/kg IV enoxaparin should be administered at the time of PCI to patients who have received fewer than 2 therapeutic subcutaneous doses (eg, 1 mg/kg) or received the last subcutaneous enoxaparin dose 8 to 12 hours before PCI.[649,661–664] *(Level of Evidence: B)*

Class IIb

1. Performance of PCI with enoxaparin may be reasonable in patients either treated with "upstream" subcutaneous enoxaparin for UA/NSTEMI or who have not received prior antithrombin therapy and are administered IV enoxaparin at the time of PCI.[646–658] *(Level of Evidence: B)*

Class III: HARM

1. UFH should not be given to patients already receiving therapeutic subcutaneous enoxaparin.[649,665] *(Level of Evidence: B)*

Trials of enoxaparin relevant to PCI include both studies in which patients with UA/NSTEMI were started on upstream subcutaneous enoxaparin therapy that was continued up to the time of PCI and trials in which patients who had received no prior antithrombin therapy were treated with IV enoxaparin at the time of PCI.[646–650,661–663,666] In the SYNERGY (Superior Yield of the New strategy of Enoxaparin, Revascularization and Glycoprotein IIb/IIIa Inhibitors) trial, there was an increased incidence of bleeding in those treated with upstream enoxaparin, later attributed at least in part to the fact that some patients being treated with enoxaparin were also administered UFH at the time of PCI (so-called "stacking").[649,663] Almost all patients undergoing elective PCI who are administered enoxaparin (0.5 mg/kg IV) will have a peak anti-Xa level >0.5 IU/mL.[647] Most clinical studies have used a regimen of 0.5 to 0.75 mg IV.[667] Several studies have used this regimen in elective patients and those with STEMI.[646] Patients who have received multiple doses of subcutaneously administered enoxaparin who undergo PCI within 8 hours of the last subcutaneous dose generally have adequate degrees of anticoagulation to undergo PCI, but the degree of anticoagulation may diminish in the 8- to 12-hour period after the last subcutaneous dose. In such patients, as well as in patients who have received only 1 subcutaneous dose of enoxaparin, the addition of enoxaparin (0.3 mg/kg IV) at the time of PCI provides an additional degree of anticoagulation and has become standard practice.[649,661–664] Patients who undergo PCI >12 hours after the last subcutaneous dose are usually treated with full-dose de novo anticoagulation using an established regimen (eg, full-dose UFH or bivalirudin).

5.7.4.4. Bivalirudin and Argatroban: Recommendations

Class I

1. For patients undergoing PCI, bivalirudin is useful as an anticoagulant with or without prior treatment with UFH.[625,637–645] *(Level of Evidence: B)*

Table 13. Recommendations for Antiplatelet and Antithrombin Pharmacotherapy at the Time of PCI

	COR	LOE	References	Relevant Caveats/Comments
Oral antiplatelet agents				
Aspirin	I	B	301–304,560–563	N/A
$P2Y_{12}$ inhibitors	I	A	564–566	• A loading dose of a $P2Y_{12}$ inhibitor should be given to patients undergoing PCI with stenting.
• Clopidogrel	I	B	564–566	• 600-mg loading dose now recommended.
• Prasugrel	I	B	567	• Contraindicated in patients with prior TIA/CVA: *Class III: Harm; LOE: B.* • Generally not recommended in patients >75 y of age (Section 5.7.2). • Consideration of using a lower maintenance dose in patients weighing <60 kg suggested by FDA (Section 5.7.2).
• Ticagrelor	I	B	568	• Issues of patient compliance may be especially important.
GP IIb/IIIa inhibitors (abciximab, double-bolus eptifibatide, high-bolus dose tirofiban)				
• No clopidogrel pretreatment	STEMI: IIa	A	564–570	• UA/NSTEMI recommendation applies to those with high-risk features. • GPI use in STEMI may be most appropriate in those with large anterior MI and/or large thrombus burden. • IC abciximab administration in STEMI: *Class IIb; LOE: B.* • Precatheterization laboratory GPI administration in STEMI: *Class III: No Benefit; LOE: B.* • Recommendations apply to those not at high risk for bleeding complications.
	UA/NSTEMI: I	A	613–618	
	SIHD: IIa	B	619–621	
• Clopidogrel pretreatment	STEMI: IIa	C	564–570	
	UA/NSTEMI: IIa	B	616,619	
	SIHD: IIb	B	619,622–624	
Antithrombin agents				
UFH	I	C	N/A	• Dosing based on whether or not GPI was administered.
Bivalirudin	I	B	625,637–645	• Lower bleeding rates associated with bivalirudin are mitigated when used concomitantly with a GPI.
Enoxaparin	IIb	B	646–650	• Recommendations apply to administration of IV enoxaparin at the time of PCI for those who have not received prior antithrombin therapy or who have received "upstream" SC enoxaparin therapy for UA/NSTEMI. • An additional dose of 0.3 mg/kg IV enoxaparin should be administered at the time of PCI to patients who have received <2 therapeutic SC doses (eg, 1 mg/kg) or received the last SC enoxaparin dose 8 to 12 h before PCI: *Class I; LOE: B.* • Patients treated with SC enoxaparin within 12 h of PCI should not receive additional treatment with UFH during PCI ("stacking"): *Class III: Harm; LOE: B.*
Anti-Xa inhibitors				
Fondaparinux	III: Harm	C	651,652	• PCI should not be performed with fondaparinux as the sole antithrombin agent in patients treated with upstream fondaparinux. An additional anticoagulant with anti-IIa activity should be administered.

ACT indicates activated clotting time; COR, class of recommendation; CVA, cerebrovascular accident; FDA, US Food and Drug Administration; GP, glycoprotein; GPI, glycoprotein IIb/IIIa inhibitor; IC, intracoronary; IV, intravenous; LOE, level of evidence; MI, myocardial infarction; N/A, not applicable; PCI, percutaneous coronary intervention; SC, subcutaneous; SIHD, stable ischemic heart disease; STEMI, ST-elevation myocardial infarction; TIA, transient ischemic attack; UA/NSTEMI, unstable angina/non–ST-elevation myocardial infarction; and UFH, unfractionated heparin.

2. For patients with heparin-induced thrombocytopenia, it is recommended that bivalirudin or argatroban be used to replace UFH.[668,669] *(Level of Evidence: B)*

Bivalirudin is being increasingly used in clinical practice[670] as evidence emerges from clinical trials across the spectrum of CAD.[631–644] In individual trials and meta-analyses, the use of bivalirudin has been associated with reduced bleeding compared with UFH plus a GP IIb/IIIa inhibitor, although concerns about ischemic events have emerged in individual studies.[625,637–645] Longer-term follow-up of the major bivalirudin trials, however, suggests that small or nominal increases in ischemic events have not translated into long-term consequences and that treatment at or before the time of PCI with clopidogrel may mitigate any increased early ischemic risk.[637–645] Thus, a treatment strategy of bivalirudin compared with heparin (or enoxaparin) plus GP IIb/IIIa inhibitor appears to lower the risk of bleeding complications. The lower bleeding rates associated with bivalirudin (compared with UFH plus a GP IIb/IIIa inhibitor) are mitigated when used concomitantly with a GP IIb/IIIa inhibitor.[639] A strategy of use of provisional GP IIb/IIIa inhibitor in patients treated with bivalirudin is widely accepted.[639,643,644]

In patients with heparin-induced thrombocytopenia,[671,672] a direct-thrombin inhibitor (argatroban) has been approved as an alternative parenteral anticoagulant to be used during PCI.[665] The use of bivalirudin for heparin-induced thrombocytopenia has been reported as well.[669]

5.7.4.5. Fondaparinux: Recommendation

Class III: HARM

1. Fondaparinux should not be used as the sole anticoagulant to support PCI. An additional anticoagulant

with anti-IIa activity should be administered because of the risk of catheter thrombosis.[651,652] *(Level of Evidence: C)*

Fondaparinux, a pentasaochariде, is an indirect factor Xa inhibitor but has no effect on thrombin. On the basis of reports of catheter thrombosis when fondaparinux is used alone during primary PCI,[651,652] the writing committee recommends that an anticoagulant with anti-IIa activity be used in patients undergoing PCI.[651,652] One study suggested that clinical outcomes were better when fondaparinux was replaced during PCI by a standard dose of UFH (85 U/kg, 60 U/kg with GP IIb/IIIa inhibitors) rather than by a low dose (50 U/kg).[653]

5.7.5. No-Reflow Pharmacological Therapies: Recommendation

Class IIa

1. Administration of an intracoronary vasodilator (adenosine, calcium channel blocker, or nitroprusside) is reasonable to treat PCI-related no-reflow that occurs during primary or elective PCI.[674–689] *(Level of Evidence: B)*

See Online Data Supplement 25 for additional data regarding no-reflow therapies.

No-reflow is a broad term used to describe 2 distinct entities. The first is "interventional no-reflow" attributed to vasospasm and downstream embolization of debris dislodged during PCI, usually in the setting of atherectomy, thrombus, or degenerated SVGs. The second entity is suboptimal reperfusion of an infarct artery, attributed to endothelial injury in addition to embolization and vasospasm. Angiographic no-reflow is the most obvious sequela of the same pathophysiology that produces abnormal TIMI frame counts and TIMI blush scores, so these measures are often used interchangeably. The principal clinical sequela of no-reflow is myonecrosis. Efforts to prevent no-reflow overlap with strategies to reduce MI size and prevent periprocedural MI.

In the setting of MI, several drugs have been shown to reduce the incidence of no-reflow. Evidence for a beneficial effect on no-reflow exists for abciximab, adenosine, nicorandil, and nitroprusside.[674,675,680,682,683,685,687,688,690] However, their adoption into clinical practice has depended on their effect on hard clinical endpoints such as infarct size and mortality. These benefits, and consequentially the use of these agents, have been limited.

For interventional no-reflow, several therapies have proven effective after no-reflow has started. These include adenosine, calcium channel blockers, and nitroprusside.[676,678,679,681,684,686,689,691] There are fewer data to support the use of epinephrine.[692] No-reflow after rotational atherectomy was less common with nicorandil compared with verapamil infusions in 3 studies,[693–695] and an infusion of nicorandil/adenosine during rotational atherectomy prevented no-reflow in 98% of patients.[677] Trials of pre-PCI intracoronary verapamil, nicardipine, and adenosine have reported them to be safe but have not demonstrated reductions in post-PCI no-reflow.[696–698] Mechanical devices to prevent interventional and myocardial infarct reperfusion no-reflow are also covered in Section 5.5.5.

5.8. PCI in Specific Anatomic Situations

5.8.1. CTOs: Recommendation

Class IIa

1. PCI of a CTO in patients with appropriate clinical indications and suitable anatomy is reasonable when performed by operators with appropriate expertise.[699–703] *(Level of Evidence: B)*

See Online Data Supplements 26 to 28 for additional data regarding CTOs.

Approximately one third of patients with suspected CAD who undergo coronary angiography have ≥1 CTO (defined as occlusion of a duration >3 months).[704] Although stress-induced ischemia can be elicited in the majority of patients with CTO despite the presence of collaterals,[704,707] only 8% to 15% of these patients undergo PCI.[708,709] The disparity between the frequency of CTOs and percutaneous treatment underscores not only the technical and procedural complexities of this lesion subtype but also the clinical uncertainties regarding which patients benefit from CTO revascularization. Studies suggest that patients who undergo successful, rather than failed, recanalization of CTOs fare better in terms of symptom status and need for CABG,[690] as well as LV function.[710] However, the impact of successful CTO recanalization on long-term survival remains unsettled.[701,711,712] The decision to try PCI for a CTO (versus continued medical therapy or surgical revascularization) requires an individualized risk-benefit analysis encompassing clinical, angiographic, and technical considerations. Consultation with a cardiothoracic surgeon and use of the Heart Team approach in cases of CTO in which a large territory is subtended and/or multivessel CAD is present are frequently done.

From a technical perspective, successful recanalization of CTOs has steadily increased over the years because of adoption of dedicated wires, novel techniques, and increased operator experience.[702] In patients who undergo successful CTO recanalization, use of DES significantly reduces the need for repeated target-vessel revascularization, compared with BMS and balloon angioplasty, without compromising safety.[703,713–719]

5.8.2. SVGs: Recommendations

Class I

1. EPDs should be used during SVG PCI when technically feasible.[533–535] *(Level of Evidence: B)*

Class III: NO BENEFIT

1. Platelet GP IIb/IIIa inhibitors are not beneficial as adjunctive therapy during SVG PCI.[212,571,720,721] *(Level of Evidence: B)*

Class III: HARM

1. PCI is not recommended for chronic SVG occlusions.[722–724] *(Level of Evidence: C)*