NHS
Greater Glasgow and Clyde

CLINICAL GUIDELINE

Anticoagulants and Antiplatelets for ELECTIVE / NON-EMERGENCY Percutaneous Procedures

A guideline is intended to assist healthcare professionals in the choice of disease-specific treatments.

Clinical judgement should be exercised on the applicability of any guideline, influenced by individual patient characteristics. Clinicians should be mindful of the potential for harmful polypharmacy and increased susceptibility to adverse drug reactions in patients with multiple morbidities or frailty.

If, after discussion with the patient or carer, there are good reasons for not following a guideline, it is good practice to record these and communicate them to others involved in the care of the patient.

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Important Note:

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NHS Greater Glasgow & Clyde
DIAGNOSTIC & INTERVENTIONAL RADIOLOGY DEPARTMENT

*"The Perioperative Management of **Anticoagulants** and **Antiplatelets** for **ELECTIVE / NON-EMERGENCY** Percutaneous Procedures **in Adult Patients** in Diagnostic & Interventional Radiology"*

Revision document: U Pisano, IR Consultant, January 2025.
UPDATE INFORMATION - HOW THIS VERSION CHANGES PRIOR DOCUMENTS:
- *Moderate* risk procedures now grouped with *high* risk (instead of with low risk)
- Implementation of BSIR / BSH guidance in relation to clopidogrel (up to 7 days discontinuation, rather than 5), warfarin discontinuation for 2-3 days for low-risk interventions. Creatinine clearance replaced the eGFR for discontinuation of heparin treatment in renal impairment. Additional section on specific circumstances: end-stage renal failure patients requiring interventions on haemodialysis access (*fistuloplasty, graftoplasty, venous stenting;* after consultation with vascular access IR specialists R Kasthuri and C Stove) and renal cryoablation (D Alcorn). Specification on continuation of antithrombotic therapy for nephrostomy exchanges. Notes about COMPASS trial-related regimens and low-dose anticoagulant continuation when possible.

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Management of *anticoagulants* and *antiplatelets* (occasionally referred collectively as ***antithrombotic*** drugs) in patient undergoing image-guided intervention is complex because of the wide range of procedures and equally wide range of patient demographics and comorbidities. Concomitant increases in the use of short- and long-term anticoagulation, as well as use of antiplatelet agents, further complicates patient management[1].

Initial work-up of patients is determined by the **procedure-associated bleeding risk** (*Table 1*). The coagulation status of patients must be assessed whenever the procedure involves direct entry into the arterial or venous system as an anticipated part of the intervention, or whenever there is a concrete possibility of inadvertent entry into an artery or vein. Essential laboratory data is listed in Table 2: a dedicated bleeding history should also be acquired from the patient before any intervention using a validated questionnaire (e.g., 'HEMSTOP'[2]).

Another equally important factor to bear in mind is the **risk of thromboembolism** in patients undergoing an intervention: this is going to translate in higher likelihood of **stroke** in those suffering from atrial fibrillation[3], possible worsening of an established **deep venous thrombosis (DVT)** or **new thrombosis** in those with hypercoagulable state (e.g., related to cancer[4]). Individuals with mechanical heart valves are at risk of thrombosis if therapeutic anticoagulation is withheld. Because most scoring systems provide long-term or annual thrombotic and thromboembolic risk, it is difficult to predict the probability of postprocedural bleeding and / or thrombosis for each intervention.

These guidelines represent a suggested pathway for the periprocedural management of antiplatelets and anticoagulants in adult patients undergoing image-guided procedures in elective and non-emergency cases. *For complex scenarios (i.e., when a concomitant concerning risk of thromboembolism is presumed to exist) and whenever there is doubt, please contact the Radiology Department for a patient-tailored discussion* (Duty Diagnostic Radiology – Queen Elizabeth University Hospital - QEUH, ext. 8357; Duty Interventional Radiology QEUH, ext. 83644).

BRIEF SYNOPSIS ON HAEMOSTASIS

Haemostasis is a precisely orchestrated process involving platelets, clotting factors, and endothelium that occurs at the site of vascular injury. It culminates with the formation of a blood clot, which serves to prevent or limit the extent of bleeding. Arteriolar vasoconstriction occurs immediately and reduces blood flow to the injured area; this is mediated by reflex neurogenic mechanisms and enhanced by local secretion of factors (e.g., endothelin)[5].

Exposure of the surface under the endothelium allows interaction between platelets and von Willebrand factor (vWF): platelets therefore undergo adherence against the vessel wall, and activation: this consists of a shape change (from small, rounded discs to flat plates with spiky protrusions), as well as the release of secretory granules[5]. Platelet aggregation ('clustering') at this stage represents the *primary haemostasis*.

Secondary haemostasis instead relies on the coagulation cascade: a chain reaction based on enzymatic cleavage of bloodstream proteins, and that ends up with the formation of *thrombin*. Thrombin modifies fibrinogen into insoluble *fibrin*, thus creating a meshwork which covers and stabilises the platelet cluster. The process of clot (*thrombus*) formation is inherently limited by counterregulatory mechanisms (*thrombolysis*); **this is to limit the extension of the clot to the site of the injury[5,6].**

Antiplatelets work by reducing the capacity of platelets to aggregate and initiate clot formation. Their distinction in classes is based on the interaction with different platelet receptors, and a full description is beyond the scope of these guidelines. **Anticoagulants** target instead different components of the coagulation cascade, limiting the stability of the newly formed platelet clusters, and therefore the overall capacity to form a new clot.

ANTIPLATELET DISCONTINUATION AND RESUMPTION DURING IR PROCEDURES

Aspirin (acetylsalicylic acid, ASA), and other non-steroidal anti-inflammatory (NSAIDs) drugs work by inhibiting the *cycloxygenase (COX)* enzymes; therefore, molecules such as thromboxane A_2 (a potent vasoconstrictor, which helps platelet adhesion) are not produced. Different pharmaceuticals target different forms of the COX (the constitutive *COX-1* and inducible *COX-2*), and can be short-acting (**ibuprofen, diclofenac, ketoprofen, indomethacin***:* 2–6 hours half-life), intermediate-acting (**naproxen, sulindac, diflunisal, celecoxib**: 7–15 hours), and long-acting (**meloxicam, nabumetone, piroxicam***:* > 20 hours of half-life). Unlike previous recommendations[7], the latest consensus from interventional radiology societies[1,8,9] is to continue NSAIDs prior to percutaneous interventions.

Cilostazol is an inhibitor of the enzyme phosphodiesterase 3 (PDE3); after reversible blockage of the enzyme, the concentration of cyclic adenosine monophosphate (cAMP) increases, and the capacity of platelets to aggregate is reduced significantly. It is indicated for intermittent claudication in patients without rest pain and no peripheral tissue necrosis[10]. For elective interventions, cilostazol does not need to be interrupted. It can be restarted immediately thereafter.

Dipyridamole is another PDE3 inhibitor, used alone or with aspirin in secondary prevention of ischaemic stroke and transient ischaemic attack; or as an adjunct to oral anticoagulation for prophylaxis of thromboembolism associated with prosthetic heart valves[11]. For dipyridamole alone, no discontinuation is needed. When taken in combination with ASA, we feel prudent to suspend the formulation for 48 hours prior a high bleeding risk intervention.

Clopidogrel, ticagrelor, prasugrel and **cangrelor** are oral antiplatelets sharing a similar thienopyridine structure that inhibit binding of adenosine diphosphate (ADP) to the platelet P2Y12 receptor (therefore *limiting platelet aggregation*)[12].

Clopidogrel is indicated in prevention of atherothrombotic events in acute coronary syndrome (ACS), in high-risk atrial fibrillation patients, in peripheral vessel disease, or in transient ischaemic attacks where contraindications to aspirin exists[13]. Applications to ticagrelor are limited to prevention of acute arterial thrombosis in ACS or prior myocardial infarction[14]. Prasugrel is licensed in ACS settings for patients undergoing percutaneous coronary intervention (PCI)[15]. Cangrelor is a fast-acting, intravenous, direct P2Y12 inhibitor given during PCI to patients who have not received an oral P2Y12 inhibitor, or when this is not a suitable option[16,17].

If the antiplatelet regimen is related to an acute cardiovascular event (and recent coronary stent insertion) rather than primary prevention, the risks of arterial / in-stent thrombosis could outweigh the benefits of the intervention. In these scenarios, it might be in the patient's interest to continue the antiplatelet(s). Case-by-case discussion with the radiology department is essential.

Also, patients undergoing an arterial intervention for critical limb ischaemia (CLI) will likely be on antiplatelet/s (aspirin and / or clopidogrel), *which do not need to be interrupted.* After such cases it is recommended to discuss with the referring vascular team regarding the resumption of a thienopyridine

antiplatelet (i.e., clopidogrel): this might preclude the possibility of a spinal anaesthesia, should the endovascular intervention fail, and an amputation become necessary.

For all other interventions, antiplatelet *discontinuation ranging from 2-7 days* is needed *(see table 3);* however, *if the intervention has a low bleeding risk, the operator might choose to continue clopidogrel, ticagrelor or prasugrel: if the patient is on cangrelor, the intervention should be postponed until course completion.*

One of the key steps of platelet aggregation is the activation of a surface protein, *GP IIb/IIIa*, an integrin that binds both vWF and fibrinogen; agents that target GP IIb/IIIa are **tirofiban, eptifibatide,** and **abciximab**; they were developed with the rationale to reduce occurrence of death, myocardial infarction (MI), and need for target vessel revascularization after PCI. Most of the available data relates to abciximab[18] , but recent reviews do not show a significantly increased bleeding risk with the other GP IIb/IIIa antagonists[19]. Tirofiban has shown to reduce cardiovascular events in patients with ACS[19,20] with no increase of bleeding events in the context of coronary artery bypass graft (CABG) after failed revascularisation[21] . Equally, eptifibatide does not appear to confer increased bleeding risk when compared to standard therapy[22] in pragmatic settings. GP IIb/IIIa antagonists feature a short half-life, ranging from 30 mins (abciximab) to 2 – 2.5 hours (tirofiban, eptifibatide)[12] .

It is unlikely that elective/non-emergency percutaneous interventions will be performed on patients under GP IIb/IIIa antagonists. While a discussion with haematology would always be required, a discontinuation for 24 hours is deemed safe; nonetheless, an additional antiplatelet and/or anticoagulant regimen would most certainly be in place for these patients.

Table 3 and Table 4 provide a summary of recommendations with regards to discontinuation and post-operative resumption of antiplatelets.

ANTICOAGULANTS DISCONTINUATION AND RESUMPTION DURING ELECTIVE IR PROCEDURES

During the coagulation cascade, the involved proteins take place to a sequence of reactions featuring an *enzyme* (i.e., an *activated* coagulation factor), a *substrate* (inactive form of another coagulation factor), and a *reaction accelerator*. Such process takes place on a negatively charged phospholipid surface (the platelet membrane)[5]. Calcium is an essential component of some of these steps.

The division into *intrinsic* and *extrinsic* pathway relates to assays carried out in clinical laboratories. The prothrombin time (PT) examines the activity of the extrinsic pathway (factors VII, X, V, II, and fibrinogen); in a nutshell: tissue factor, phospholipids, and calcium are added to plasma, and the time for a fibrin clot to form is recorded. Instead, the partial thromboplastin time (PTT) assay screens the function of the proteins in the intrinsic pathway (factors XII, XI, IX, VIII, X, V, II, and fibrinogen)[5].

Unfractioned heparin (UFH) can be administered intravenously or subcutaneously to achieve anticoagulation via different routes: i.e., boosting the activity of *circulating antithrombin* (which neutralises thrombin), and by *cleaving activated coagulation factors IXa, Xa, XIa, XIIa* and *plasmin*.

Antithrombin is a plasma protein produced by the liver, able to neutralise multiple clotting factors of the intrinsic and extrinsic pathways (with little activity against factor VIIa)[12]. The free circulating form is named *antithrombin III,* according to laboratory criteria developed in the earliest study[6].

UFH is given as initial bolus and titrated according to PTT (therapeutic window usually between 1.5-2.5 times the normal PTT values). ***We suggest holding UFH for 4 hours for low bleeding risk procedures, and 6 hours for moderate / high risk interventions*.** Resumption can be reinstated after one hour, regardless of the bleeding risk[9].

Dalteparin, tinzaparin and **enoxaparin** are **low-molecular-weight heparins (LMWHs)**; they are derived from UFH but, unlike their predecessor, have a small effect on PTT; their mechanism is based on *binding to antithrombin* and *increasing manifold its efficacy*: their half-life is 2–4 times that of UFH (4.5 hours after a single dose; roughly 7 hours after repeated doses). For **prophylactic** LMWH, interruption for 12 and 24 hour is suggested for low and moderate / high bleeding risks, respectively. Resumption after 6

hours is recommended for all procedures, unless specified otherwise[9]. A full 24-hour interruption of **therapeutic** LMWH is suggested for all procedures.

A common situation is represented by a patient with renal impairment, requiring a percutaneous procedure (e.g., an urgent nephrostomy) while exposed to prophylactic LMWH. With a creatinine clearance (CrCl) <30ml/min based on Cockroft – Gault equation*, the clearance of LMWH is impaired although this should not cause an overtly increased haemorrhagic risk[23–25]. If possible, a full 24 hours and 48 hours of suspension should be achieved prior to low- and moderate / high-risk operations in patients with impaired renal function under LMWH.

The oral anticoagulants licensed for use in the UK are vitamin K antagonists (**warfarin**, acenocoumarol and phenindione) and direct oral anticoagulants (**apixaban**, **dabigatran etexilate**, **edoxaban** and **rivaroxaban**[23,26]). Warfarin is usually the drug of choice among the first group: it antagonizes the biosynthesis and activation of the vitamin K–dependent extrinsic pathway clotting factors (II, VII, IX, X), and protein C and S in the liver[9].

Apart from factors like *dose, pharmacogenetics, comorbidities, drug therapy* and *diet*, the half-life of warfarin (37 hours) is also dependent on the quantity of circulating clotting factors, the most important of which is factor II (i.e., *prothrombin*: estimated half-life 96 h)[9]. Effect of warfarin is measured by PT, usually expressed as international normalised ratio (INR; to standardise PT results among different laboratories) and the therapeutic window is narrow, between 2-3 for commonest indications (prophylaxis of embolization in rheumatic heart disease / atrial fibrillation; prophylaxis after heart valve; prophylaxis and treatment of venous thromboembolism / pulmonary embolism)[27].

We recommend withholding warfarin for at least 5 days prior to moderate / high-bleeding risk procedures[8,9,28], and for 2-3 days for low-risk cases[8]. If this cannot be achieved due to underlying clinical need and risk of thromboembolism, bridging with intravenous UFH *should be initiated by the referring team.* Interruption and resumption of UFH follow the principles written above. Warfarin can be recommenced after 12 hours after low-risk procedures, and after full 24 hours from moderate / high risk interventions.

Dabigatran etexilate works by *direct inhibition of both free and clot-bound thrombin (factor IIa)*, thus decreasing the conversion of fibrinogen into fibrin. It is not routinely monitored. The half-life is around 12-17 hours but varies greatly in elderly patients with possible renal impairment. Bearing this in mind, we suggest suspension for 48 hours for low- and moderate risk procedures, and 72 hours for high bleeding risk operations[9]; up to four days might be required in individuals with poor GFR[8]. Resumption is suggested after 24 (low risk) and 48 hours (moderate / high risk procedures). *Idarucizumab* is licensed for the rapid reversal of dabigatran in life-threatening or uncontrolled bleeding, or for emergency surgery or urgent procedures[29].

Argatroban monohydrate, like dabigatran, is a direct anti-thrombin inhibitor *but is currently only licensed for intravenous use as an antithrombotic in individuals who developed a type II heparin-induced thrombocytopenia (HIT)*[30]. The BSIR-BSH consensus suggests withholding it for 2-4 hours for low-risk, and for 4 hours for high risks procedures[8]. However, given the related complexity, patients on argatroban require a dedicated discussion with haematology before undergoing any percutaneous procedure, in particular arterial interventions[9].

Bivariludin shares the same mechanism of action but only approved for intravenous use in patients with acute coronary syndrome, preceding or undergoing percutaneous coronary intervention (PCI) in addition to aspirin and clopidogrel[31]. We advise to wait 4 hours prior to performing any procedure. Resumption can take place after one hour.

Rivaroxaban and **apixaban** cause a *reversible inhibition of factor Xa*[12]. They are both indicated in the prophylaxis of venous thromboembolism (VTE) following knee- or hip replacement surgery; in the treatment or recurrence prevention of pulmonary embolism (PE) or deep vein thrombosis (DVT)[32,33]. Both can be used in high-risk atrial fibrillation patients for prophylaxis of stroke / systemic embolism while rivaroxaban can be employed in secondary prevention following acute coronary syndrome[33]. Low dose (i.e., 2.5mg BD) of rivaroxaban have been approved for primary or secondary prevention of atherothrombotic events in combination with an antiplatelet[34,35]: the operator might choose not to stop the rivaroxaban for low bleeding risk procedures, at his/her discretion.

The half-life of rivaroxaban approximates 5-9 hours, but it is extended in elderly patients[12]; it is roughly 12 hours for apixaban[9]. *Andexanet alpha* (recombinant form of Xa) exists as possible reversal agent for both drugs, in case of uncontrolled bleeding[36] .

* https://handbook.gqcmedicines.org.uk/guidelines/infections/creatinine-clearance-equation/
* https://scottish.sharepoint.com/sites/GGC-ClinicalInfo/SitePages/Medicine%20Calculators.aspx

Suspension for 24 hours is advised for low-risk procedures, and for 48 to 72 hours for moderate / high-risk interventions[36,37]. Unless specified otherwise, resumption can restart within 24 hours (low-risk) and within 48 hours after moderate / high-risk procedures[8,9]. **Edoxaban**[38] is also a more recently developed factor Xa reversible inhibitor, and it shares similar pharmacokinetic and pharmacodynamic characteristics[39]. Given its half-life of 10-14 hours, *discontinuation and resumption should follow the same recommendations for apixaban.*

Fondaparinux is a synthetic pentasaccharide approved for VTE prophylaxis after major general or orthopaedic surgery, for treatment of PE/DVT, unstable angina, and ST- and non-ST-elevation myocardial infarction[40].

Comparably to UFH and LMWHs, it increases efficacy of antithrombin III, but its main advantage is the sensibly reduced chance to provoke a heparin-induced thrombocytopenia (HIT). It does not affect PT nor aPTT. Its half-life ranges from 17-21 hours.

It is recommended to discontinue fondaparinux for 48 for low-risk intervention, and 72 hours for moderate / high-risk interventions[9]. Resumption can take place after 6 hours from any operation, unless specified otherwise[8].

Table 5 and 6 contains the recommendations for anticoagulants discontinuation and resumption following non-emergency percutaneous procedures.

NOTES ON SPECIFIC CIRCUMSTANCES

ANTICOAGULANTS DISCONTINUATION IN INDIVIDUALS WITH END-STAGE RENAL FAILURE (ESRF) REQUIRING INTERVENTIONS ON ARTERIOVENOUS DIALYSIS ACCESS

ESRF patients with arteriovenous fistulae and grafts can be expected to be under antithrombotics to prevent thrombosis of their fistula or graft in between their dialysis sessions. They will most likely also receive high doses of intravenous heparin at the time of their dialysis sessions.

IR procedures in these patients are undertaken for surveillance but also for treatment of a dysfunctional access (due to peripheral or central stenoses, or to insert / revise a dialysis catheter, or for complex venous recanalisations).

On many occasions, *the periprocedural risk of access thrombosis will outweigh the risk of intraprocedural bleeding*: this particularly applies to invasive surveillance in arteriovenous grafts (where low-calibre sheaths are usually sited in the graft). ***Anticoagulation*** *and* ***antiplatelet drug therapy*** *should not be stopped in these patients,* if possible.

On the contrary, interventions associated with **moderate bleeding risk in ESRF patients** (e.g., tunnelled dialysis catheter insertion, or underdeveloped arteriovenous fistulae requiring endovascular maturation intervention; see Table 1) would require discontinuation of anticoagulants; however, *it would be beneficial to continue the antiplatelet regimen if there is one in place, at operator discretion*.

RENAL CRYOABLATION

In the NHS GGC, selected patients with a diagnosis of renal cell carcinoma (RCC) will be offered an image-guided percutaneous intervention with the aim to destroy the renal malignancy using a cryoablation (CRA) technique (based on Argon-mediated multi-probe freezing effect), after urology MDT consensus. CRA has proven to be not inferior to radiofrequency ablation (RFA) in the treatment of RCC[41,42], and less likely to cause renal function impairment than conventional partial nephrectomy[43,44]. Roughly 100-150 cases are performed per year: the experience of this centre is that the cauterization and haemostasis achieved during tumour ablation via CRA are less effective when compared to RFA or microwave thermal ablation (MWA).

In the literature, the rates of haemorrhage following CRA differ: Lyttrup reported an overall bleeding rate of 14%[45], higher than other series[46,47]. Rodriguez states that only two patients in his cohort of 117 RCCs had symptomatic postoperative perinephric haematomas (both patients were on clopidogrel,

which was discontinued for 5 days and restarted *'after a few days'* from CRA)[48]. In a smaller case series, the rate of clinically significant haematomas requiring transfusions was 4% (2/51)[49].

In NHS GGC there is no current consensus regarding an optimal discontinuation interval for antithrombotics before CRA. Many RCC patients will be complex and might require a longer discontinuation of antiplatelets and anticoagulants, *which will be determined at the time of their assessment in the IR clinic.* Resumption should also be decided by the responsible IR consultant on a case-by-case basis, depending on intraprocedural findings and risk of thrombosis in the recovery phase.

NEPHROSTOMY EXCHANGES

The routine exchange of an indwelling nephrostomy catheter (performed at NHS GGC every three months, to mitigate the chance of infection and blockage) **does not normally require suspension** of antithrombotic medications. This is also generally the case of retrograde nephrostomy tube exchange via an ileal conduit.

TABLE 1. Classification of different vascular and non-vascular percutaneous interventions based on the bleeding risk.

Low Bleeding Risk	**Moderate** Bleeding Risk	**High** Bleeding Risk
	NON-VASCULAR INTERVENTIONS	
Superficial Biopsy / Drainage (extra-thoracic and extra-abdominal)	Intra-abdominal and retroperitoneal biopsy or drainage ***(excluding liver or spleen)***	Hepatic, or splenic, or any other renal parenchymal biopsy or drainage.
Drainage catheter replacement (e.g., nephrostomy exchange†)	Lung biopsy	Biliary intervention (**new tract)**
Oesophageal / Colonic stenting	Pleural drainage catheter insertion	*Complex* thermal ablation procedure*
Joint aspiration / injection	Decompressive nephrostomy in **hydronephrotic kidney**	Nephrostomy tube placement (defunctioning, non-dilated system)
	Simple thermal ablation procedure*	Lumbar puncture, myelography, epidural injection
	Percutaneous cholecystostomy tube (original placement and exchanges)	Spinal biopsy
	Gastrostomy tube placement (original placement and exchanges)	
	Biliary tube exchange	
	Vertebroplasty, kyphoplasty	
	Retrograde or antegrade ureteric stenting **(old tract)**	
	VASCULAR INTERVENTIONS	
Arteriovenous fistula / graft intervention (*excluding central veins*)**	Arterial intervention with access size up to 7 Fr	TIPS, BRTO
Venography	Venous intervention (plasty / stenting, *including central veins*)	Complex / bilateral iliac angioplasty / stenting
Central line removal	Trans-arterial bland and chemoembolization	Visceral artery angioplasty / stenting
IVC filter placement	Uterine fibroid embolization	EVAR, FEVAR, TEVAR
PICC placement	Prostate artery embolization	Pulmonary arteriovenous malformation embolisation
Varicocele embolization	Tunnelled central venous catheter	*Complex* IVC filter removal***
	Subcutaneous port device placement	
	Trans-jugular liver biopsy	
	Conventional IVC filter removal	

†*Unless specified otherwise, conventional nephrostomy tube replacements do not require the suspension of antithrombotics.*

Complex *tumour ablation procedures imply the treatment of a lesion in a location near major vessels, or when a large amount of hepatic or non-hepatic parenchyma must be traversed to access the lesion. Renal cryoablations might require longer discontinuation of antithrombotics.*

***Patients with **end-stage renal failure** have **high thrombotic risk** of their renovascular access. Consider continuing **both** anticoagulants and antiplatelets for low bleeding risk procedures; **stopping anticoagulants** but **continuing antiplatelets** for moderate / high bleeding risk procedures.*

*****Complex** venous filter removal includes cases where retrieval is known to be challenging on account of adverse anatomical / radiological factors or a previous failed attempt.

TABLE 2. Parameters for low, moderate and high bleeding risk procedures

	Low Bleeding Risk	**Moderate** Bleeding Risk	**High** Bleeding Risk
LAB TESTING BEFORE PROCEDURE			
INR	< 2.0	consider correction if > 1.5	consider correction if > 1.5
aPTT	< 35 seconds	consider correction if > 35 sec	consider correction if > 30 sec
Platelet	> 50 x 10^9 / L	consider transfusion if < 50 x 10^9 / L	consider transfusion if < 50 x 10^9 / L
Hb	> 80 g / L	consider transfusion if < 80 g / L	consider transfusion if < 80 g / L

TABLE 3. Recommendations for discontinuation antiplatelets prior to non-emergency percutaneous procedures

AGENT	CLASS	LOW BLEEDING RISK	MODERATE / HIGH BLEEDING RISK
ASA	COX1 inhibitor	not needed	not needed
ASA + Dipyridamole	COX1 and phosophodiesterase inhibitor	not needed	2 days
NSAIDS	COX1-COX2 inhibitors	not needed	not needed
Cilostazole	Phosophodiesterase inhibitor	not needed	not needed
ARTERIAL PERIPHERAL INTERVENTIONS in CLI			
Clopidogrel	ADP receptor antagonist	not needed	not needed
OTHER INTERVENTIONS			
Clopidogrel	ADP receptor antagonist	2 days*	**5-7 days**
Prasugrel	ADP receptor antagonist	5 days*	7 days`
Ticagrelor	ADP receptor antagonist	5 days*	7 days
Tirofiban	GP IIb / IIIa inhibitor	-	24 hours
Eptifibatide	GP IIb / IIIa inhibitor	-	24 hours
Abciximab	GP IIb / IIIa inhibitor	-	24 hours
Cangrelor	ADP receptor antagonist	*Defer procedure*	*Defer procedure*

** If the procedure has low bleeding risk, the operator might choose not to suspend the antiplatelet*

TABLE 4. Suggestions for resumption of antiplatelets after non-emergency procedures

	RESUMPTION	
AGENT	**LOW BLEEDING RISK**	**HIGH – MODERATE BLEEDING RISK**
ASA	immediate	immediate
ASA + DIPYRIDAMOLE	immediate	immediate
CILOSTAZOLE	immediate	immediate
ARTERIAL INTERVENTIONS in CLI		
CLOPIDOGREL	discuss with vascular surgery team prior to commencing / restarting	
OTHER INTERVENTIONS		
CLOPIDOGREL	immediate	immediate
PRASUGREL	immediate	24 hours
TICAGRELOR	immediate	24 hours
TIROFIBAN	-	-
EPTIFIBATIDE	-	-
ABCIXIMAB	-	-

TABLE 5. Recommendations for discontinuation anticoagulants prior to non-emergency percutaneous procedures

		DISCONTINUATION	
AGENT	**CLASS**	**LOW BLEEDING RISK**	**MODERATE – HIGH BLEEDING RISK**
WARFARIN	*Vitamin K inhibitor*	2-3 days	5 days
UFH	*Antithrombin III activation, cleavage of activated clotting factors*	4 hours	6 hours
LMWH	*Antithrombin III activation*	12 hours	24 hours
		THERAPEUTIC REGIMEN	
LMWH	*Antithrombin III activation*	24 hours	24 hours
		RENAL IMPAIRMENT (<30ml/min)	
LMWH	*Antithrombin III activation*	24 hours	48 hours
DABIGATRAN	*Thrombin (IIa) inhibition*	48 hours	72 hours
RIVAROXABAN	*Factor Xa inhibition*	24 hours*	48 hours
APIXABAN	*Factor Xa inhibition*	24 hours	72 hours
EDOXABAN	*Factor Xa inhibition*	24 hours	72 hours
FONDAPARINUX	*Antithrombin III activation*	48 hours	72 hours
BIVALIRUDIN	*Thrombin (IIa) inhibition*	4 hours	4 hours
ARGATROBAN	*Thrombin (IIa) inhibition*	*Discuss with Haematology*	*Discuss with Haematology*

*Rivaroxaban used for prevention of major cardiovascular events (2.5mg BD) in atherosclerotic disease in patients undergoing low-bleeding risk procedures might be continued, at the operator discretion.

TABLE 6. Suggestions for resumption of anticoagulants after non-emergency procedures

AGENT	LOW BLEEDING RISK	MODERATE - HIGH BLEEDING RISK
UFH	1 hour	1 hour
BIVALIRUDIN	1 hour	1 hour
FONDAPARINUX	6 hours	6 hours
LMWH	6 hours	6 hours
WARFARIN	12 hours	24 hours
DABIGATRAN	24 hours	48 hours
RIVAROXABAN	24 hours	48 hours
APIXABAN	24 hours	48 hours
EDOXABAN	24 hours	48 hours
ARGATROBAN	*Discuss with Haematology*	*Discuss with Haematology*

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