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Review Article

Anaesthesia Section

Managing Neuraxial Anaesthesia in Patients undergoing Anticoagulation Therapy: A Narrative Review

PRACHI SIDDHARTH KAMBLE[1], NEETA VERMA[2], AMREESH PAUL[3]

ABSTRACT

Neuraxial anaesthesia in patients taking antithrombotic medications, including anticoagulants and antiplatelet agents, poses considerable challenges due to the increased risk of bleeding complications such as spinal or epidural haematomas. The use of both conventional anticoagulants like warfarin and newer drugs like Direct Oral Anticoagulants (DOACs) has made the decision-making process for anaesthesiologists more complex because these drugs affect the coagulation system differently. This review covers the mechanisms of action of antiplatelet and anticoagulant medications, and their impact on bleeding risk and complications during neuraxial anaesthesia. Preoperative considerations of relevance, e.g., drug cessation timing and coagulation monitoring, also receive mention as key to avoiding patient harm. Bleeding risks are minimised with appropriate drug regimen adjustment, selective anaesthetic practice, and case-by-case assessment of patients, all of which are discussed here. The review examines the influence of antithrombotic therapy on anaesthetic practice. It emphasises the necessity of meticulous planning to weigh the advantages of neuraxial anaesthesia against the risk of poor outcomes. In spite of the availability of some guidelines, there are a lot of controversies in the management of patients on dual or multiple antithrombotic therapy, particularly in light of the recent trend of the usage of novel anticoagulants. The review brings into focus the need for further studies to formulate evidence-based guidelines to perform neuraxial anaesthesia safely in such patients. Ultimately, better comprehension of the coagulation management, testing, and safety profile of such therapies will come to bear as improved outcomes and more precise clinical practice guidelines in this high-risk population.

Keywords: Antithrombotic agents, Antiplatelet drugs, Spinal haematoma, Epidural haematoma, Warfarin

INTRODUCTION

Neuraxial anaesthesia, including spinal as well as epidural techniques, is a cornerstone of regional anaesthesia for a wide variety of surgical and diagnostic procedures. Neuraxial anaesthesia is adequate for pain relief, decreases the demand for systemic analgesics, and ensures improved patient recovery. Neuraxial anaesthesia is especially beneficial in patients with some comorbidities, patients with lower limb surgery, or for prolonged pain relief postoperatively. Though widely used and effective, neuraxial anaesthesia in antiplatelet or anticoagulation therapy patients involves significant risk. Antiplatelet and anticoagulant drugs are widely employed to manage and prevent thromboembolic conditions like Pulmonary Embolism (PE) and Deep Vein Thrombosis (DVT) and the prevention of stroke and Myocardial Infarction (MI) [1].

Drugs such as aspirin, clopidogrel, warfarin, and DOACs affect the coagulation pathway by preventing platelet aggregation or affecting clotting factors. Therefore, they make the patient susceptible to bleeding complications. In neuraxial anaesthesia, this possibility of bleeding may be particularly inconvenient because it can lead to dangerous complications such as epidural or spinal haematoma, with devastating consequences, such as permanent neurological injury or paralysis. The core issue of management of patients requiring neuraxial anaesthesia who are on antithrombotic therapy is weighing the advantage of regional anaesthesia against increased bleeding risk [2].

The risk profile of the patient should be assessed judiciously by the clinician, whether he is on any anticoagulant or antiplatelet medication and if yes, when the last dose was administered, and also the reason for antithrombotic cover. Neuraxial anaesthesia can be administered safely in these patients with judicious perioperative management, namely, what to stop, change, or continue the drug [3]. The objective of this review is to provide a comprehensive summary of the intricacies of administering neuraxial anaesthesia to antiplatelet or anticoagulated patients. Through providing a general impression of these matters, the paper seeks to assist clinicians in making informed, evidence-based decisions when balancing adequate anaesthesia and patient safety in this at-risk population.

MECHANISMS OF ACTION OF ANTIPLATELET AND ANTICOAGULANT DRUGS

Antiplatelet and anticoagulant medications have diverse mechanisms of action in the prevention of thromboembolic occurrences but share the same purpose of inhibiting the formation of blood clots. The medications are essential to employ in patients at high risk of cardiovascular events like stroke, MI, and DVT. The action of drugs should be known, particularly their effect on processes such as neuraxial anaesthesia, when bleeding complications are a significant cause for worry. Antiplatelet medications like clopidogrel and aspirin act mainly against platelet function. The platelets are responsible for causing blood clots by clumping together at the site of injury to the blood vessel. Aspirin inhibits Cyclooxygenase-1 (COX-1), an enzyme utilised in thromboxane A2 production, a platelet aggregation stimulant. Aspirin inhibits platelet clumping by inhibiting this enzyme, therefore inhibiting clot formation in its early stages. Other antiplatelet medications, such as clopidogrel, a thienopyridine, inhibit the Adenosine Diphosphate (ADP) receptors of platelets, preventing platelet activation and clumping. Consequently, antiplatelet therapy is better at the prevention of the formation of blood clots, which is beneficial in the prevention of stroke or MI, but carries a bleeding risk during surgery or invasive interventions, including neuraxial anaesthesia [4].

Anticoagulants have a more distal action in the coagulation cascade, or sequence of events the body takes to create a clot following vascular injury. Classical anticoagulants, like warfarin, accomplish

this through interference with the vitamin K-dependent coagulation factors (II, VII, IX, and X) produced in the liver. By reducing the output of these coagulation factors, warfarin degrades the coagulability of the blood. Warfarin's anticoagulant effect must be monitored by observing the International Normalised Ratio (INR) of the clotting tendency of the blood in order to maintain it within a therapeutic range, since excessive anticoagulation leads to life-threatening bleeding. Newer oral anticoagulants (DOACs) like dabigatran, rivaroxaban, and apixaban inhibit specific coagulation factors directly. Dabigatran blocks thrombin (factor IIa), whereas rivaroxaban and apixaban block factor Xa. All three of these new anticoagulants have the benefit of a fixed anticoagulant effect with no requirement for routine monitoring. However, they remain a source of bleeding risk when neuraxial anaesthesia is used in procedures [5].

Both drug classes decrease the body's capability to develop clots, but the location at which they block their action varies. Antiplatelet drugs act mainly on the earlier stages of clot formation by preventing platelet aggregation, whereas anticoagulants act on the later stages of clotting by preventing a particular clotting factor. This difference in their mechanisms implies that although both categories of therapy present an elevated bleeding risk, the care of patients on these drugs undergoing neuraxial anaesthesia calls for individualised approaches to limit the risk of bleeding complications like epidural or spinal haematomas. Elucidation of these mechanisms is central to the making of informed decisions with regard to drug discontinuation timing, bridging strategies, and the safety of performing regional anaesthesia in such patients [4,5]. [Table/Fig-1] summarises the onset, half-life, and elimination pathways of key antiplatelet and anticoagulant drugs to guide perioperative bleeding risk management during neuraxial anaesthesia [4,5].

Drug	Class	Onset of action	Half-life	Elimination
Aspirin [4]	Antiplatelet (COX-1 inhibitor)	30-60 minutes	Irreversible (platelet lifespan ~7-10 days)	Hepatic metabolism, renal excretion
Clopidogrel [4]	Antiplatelet (ADP receptor inhibitor)	~2 hours (prodrug, requires activation)	~8 hours (irreversible)	Hepatic metabolism (CYP450), renal/fecal excretion
Warfarin [5]	Anticoagulant (Vitamin K antagonist)	36-72 hours	20-60 hours (highly variable)	Hepatic metabolism (CYP2C9), renal excretion of metabolites
Dabigatran [5]	DOAC (Direct thrombin inhibitor)	1-2 hours	12-17 hours	Primarily renal (80%)
Rivaroxaban [5]	DOAC (Factor Xa inhibitor)	2-4 hours	5-9 hours (young), 11-13 hours (elderly)	Hepatic (CYP3A4), renal and faecal
Apixaban [5]	DOAC (Factor Xa inhibitor)	3-4 hours	~12 hours	Hepatic metabolism (CYP3A4), renal and faecal

[Table/Fig-1]: Pharmacokinetics of common antiplatelet and anticoagulant medications relevant to neuraxial anaesthesia [4,5].

RISKS ASSOCIATED WITH NEURAXIAL ANAESTHESIA IN PATIENTS ON ANTITHROMBOTIC THERAPY

The administration of neuraxial anaesthesia, either epidural or spinal, is not without intrinsic risks, especially if administered in the setting of antithrombotic medication in the form of antiplatelet or anticoagulant therapy. Although these drugs are paramount in preventing thromboembolic issues, they may also be prone to increasing bleeding risk following and during the procedure to a great extent. Among the most dangerous of these complications in this setting is the formation of spinal or epidural haematomas, which, unless actively identified and managed, can cause irreversible neurological injury. Spinal and epidural haematomas result when haemorrhage accumulates surrounding the spinal cord or nerves. The haematoma may compress the spinal cord and cause a spectrum of neurological deficits ranging from mild sensory changes to overt motor weakness or paralysis. Haematoma risk is most often associated with the disruption of normal haemostasis by antithrombotic drugs. These medications decrease the clotting potential of the blood and make it more difficult to close off any resultant minor bleeding associated with the placement of a needle or catheter for neuraxial anaesthesia [6].

Long-term anticoagulation therapy patients, especially warfarin patients, are at risk of significant bleeding because of the drug's action on the coagulation cascade. Likewise, newer anticoagulants such as DOACs pose a challenge because their mechanism of action on factors like thrombin or factor Xa makes them efficient in preventing clotting but also raises the bar in controlling any bleeding episodes. Although these newer drugs can provide a predictable anticoagulant effect, their quick onset and offset are troublesome, particularly in emergencies where reversal of anticoagulation is required. Antiplatelet medications, including aspirin and clopidogrel, also add to bleeding risk, but through a different mechanism. These medications block platelet aggregation, which is necessary for the early phases of clot formation. Without adequate platelet aggregation, even minor trauma during neuraxial anaesthesia may lead to prolonged or excessive bleeding. While the risk of bleeding due to antiplatelet drugs might not be as severe as that with anticoagulants, it still predisposes to haematoma formation, especially in the epidural or spinal spaces where bleeding can readily collect and cause pressure on the spinal cord [6,7].

PREOPERATIVE CONSIDERATIONS

Preoperative factors are most important when making decisions for neuraxial anaesthesia in individuals on antiplatelet or anticoagulant therapy. The key aim is to determine the patient's risk profile so that regional anaesthesia is safer than regional anaesthesia risks involving bleeding complications. Proper assessment and management prior to the procedure should be made in order to prevent possible adverse consequences, like epidural or spinal haematomas. One of the initial steps in the preoperative evaluation is to take a detailed history of the patient, including the type of antithrombotic therapy administered. Determining whether the patient is on antiplatelet medication (e.g., aspirin, clopidogrel) or anticoagulation (e.g., warfarin, DOACs) directs the decision-making process. The time since the patient's most recent dose of medication is especially relevant because it has a direct impact on the risk of bleeding with neuraxial anaesthesia. As an example, anticoagulants such as warfarin or DOACs have guidelines for when to hold the medication temporarily prior to a procedure, usually 24 to 72 hours, depending on the drug and the renal function of the patient. Antiplatelet drugs need to be discontinued for a briefer interval, but their action on platelet function may persist for days or even weeks. In addition to the type and timing of the medication, it is essential to assess the patient's renal function, liver function, and other comorbid conditions, as these factors can influence both the pharmacokinetics of the antithrombotic drugs and the patient's ability to tolerate bleeding. For example, patients with impaired renal function may have prolonged anticoagulant effects from drugs like DOACs, necessitating a more extended discontinuation period. Similarly, liver disease will impact the metabolism of warfarin, so it may be hard to make an accurate prediction of the drug's anticoagulant effect [8,9].

Bridging therapy is another essential element of preoperative preparation. It involves determining whether bridging therapy will be needed for patients who need to continue anticoagulation for high-risk conditions, such as atrial fibrillation or mechanical heart valves. Bridging involves the administration of a short-acting anticoagulant,

such as Low-Molecular-Weight Heparin (LMWH), while the long-acting drug, such as warfarin, is briefly withheld. This approach is designed to prevent thromboembolic events with minimal risk of bleeding during the perioperative interval. Bridging therapy should be carefully planned, and both the timing of the last dose of the long-acting anticoagulant and the start of bridging therapy should be coordinated with anaesthesia planning [10].

IMPACT OF ANTIPLATELET AND ANTICOAGULANT MEDICATIONS ON ANAESTHETIC TECHNIQUE

The administration of antiplatelet and anticoagulant drugs has a considerable bearing on the decision regarding anaesthetic technique, especially in cases where patients are to receive neuraxial anaesthesia. Because these drugs may cause an elevation in the risk of bleeding, they call for adjustments in routine anaesthetic protocols to prevent possible complications like epidural or spinal haematoma, which may result in severe neurological complications. The test is finding the balance between the demand for successful pain relief and the intrinsic dangers of these drugs to the coagulation system. One of the main changes in anaesthetic technique is in the timing and dosage of local anaesthetics. Lower doses of local anaesthetics are a consideration in patients on antithrombotic therapy, as bleeding risk is increased, and a conservative strategy will assist in minimising this. Epidural or spinal anaesthesia tends to demand close observation of local anaesthetic agent spread so that a satisfactory level of anaesthesia can be maintained without transgressing safe levels. Additionally, the administration of some local anaesthetics, those more likely to induce tissue irritation or inflammation, might be prohibited in such patients to minimise bleeding or haematoma risks [11].

Monitoring coagulation instruments becomes more significant in these patients. Point-of-care testing, e.g., platelet function or coagulation parameter (INR, activated partial thromboplastin time, or anti-Xa level for DOACs) measurement, can assist the anaesthesiologist in deciding whether the patient's risk of bleeding is safe for neuraxial anaesthesia. This may help provide more informed judgments regarding whether to continue with the procedure if bridging therapy is required or if other methods of anaesthesia should be used [12].

CLINICAL STRATEGIES TO MINIMISE RISKS

In anticoagulated or antiplatelet therapy patients, the minimisation of bleeding risks from neuraxial anaesthesia is achieved through a mix of preventive strategies to minimise bleeding complications without compromising adequate anaesthesia. The strategies are a multidisciplinary approach, perioperative planning, selection of the patient, and precise monitoring pre-procedure, intra-procedure, and post-procedure. The most important clinical strategy is how to discontinue or adjust antithrombotic medications. The decision regarding continuation or modification of drugs like aspirin, clopidogrel, or anticoagulants like warfarin or DOACs should be based on the patient's individual risk profile. For instance, warfarin may be stopped a few days before the procedure to allow normalisation of INR. DOACs, on the other hand, may have a shorter withholding period due to their rapid clearance. Bridging with LMWH or Unfractionated Heparin (UFH) may be necessary in high-risk patients, such as those with mechanical heart valves or atrial fibrillation, to maintain the patient anticoagulated with low risk of bleeding. Bridging therapy needs to be adequately timetabled to maintain the patient appropriately anticoagulated during the perioperative period, with the added advantage of reducing risks of bleeding due to neuraxial methods [13].

The method of giving neuraxial anaesthesia itself can also be modified. For those on anticoagulants or antiplatelet medication, the placement of an epidural or spinal needle needs to be done with excellent care. There has to be a careful, atraumatic technique so that any puncture or vessel injury is avoided to the utmost extent, as even minimal trauma to the epidural or intraspinal blood vessels may produce severe bleeding with altered coagulation. The anaesthesiologist could also employ small-gauge needles to minimise the chances of vascular trauma and achieve optimum haemostasis intra and postoperatively. Along with the adjustment of technique, the timing of neuraxial anaesthesia in relation to the patient's previous dose of antithrombotic drugs is an important issue. For anticoagulated patients such as those on warfarin, clinicians have to ensure the procedure is scheduled appropriately, so that the effect of the anticoagulant has resolved sufficiently, usually waiting 24-72 hours after the last dose, depending on the drug and the patient's individuality. With DOACs, the more rapid half-life of the drug usually provides for more reliable clearance, and anaesthesia timing may frequently be delayed accordingly. Antiplatelet therapy with drugs like aspirin or clopidogrel will have a more variable effect on the platelets, lasting often for days, so that the bleeding risk is elevated [14].

Postoperative monitoring for any evidence of bleeding or neurological injury is critical. This involves monitoring for signs of haematoma or complications such as weakness, sensory deficits, and bowel or bladder impairment, which represent new-onset back pain, all of which may suggest an underlying hematoma or complication. Frequent neurologic examinations and prompt imaging, if indicated, can recognise problems early on. In addition, for those patients at greatest risk of bleeding complications, it can also be helpful to defer postoperative anticoagulation until the bleeding risk has been thoroughly assessed [15].

TIMING OF NEURAXIAL ANAESTHESIA IN RELATION TO ANTITHROMBOTIC THERAPY

Timing of neuraxial anaesthesia relative to antithrombotic therapy is a key determinant of reducing the risks of bleeding whilst maintaining effective pain relief. Both anticoagulants and antiplatelet agents affect the coagulation cascade, and the effects last for different durations of time. As such, the timing of neuraxial anaesthesia in patients who are on such medications needs proper consideration of the pharmacodynamics of the drugs as well as the patient's risk profile. For patients on oral anticoagulants such as warfarin, the timing of neuraxial anaesthesia is usually determined by the patient's INR, which is a measure of the blood's clotting ability. Warfarin, which inhibits vitamin K-dependent coagulation factors, can extend the INR, making it more likely to bleed. Neuraxial anaesthesia should ideally be done when the INR is in a safe range, usually less than 1.5. In surgery patients, hemiparesis is often prevented by preoperatively withholding warfarin for several days to give enough time to normalise the INR. The duration of warfarin withholding varies with the clinical condition of the patient, and most guidelines suggest withholding it for around 4-5 days prior to a neuraxial block. In patients who are at increased risk of thromboembolic phenomena, bridging anticoagulation with UFH or LMWH can be used during the time of discontinuation of warfarin to continue anticoagulation [4,11].

In the case of DOACs like dabigatran, rivaroxaban, and apixaban, the timing is less complex because their half-lives are shorter. Neuraxial anaesthesia can be carried out after adequate clearance of the drug from the patient's system, usually between 24 to 48 hours following the last dose, depending on the drug and renal function of the patient. Renal function may affect the clearance time, especially in older patients or patients with pre-existing renal impairment. Where there is doubt about the timing of clearance of a DOAC, point-of-care tests like anti-Xa assays (for rivaroxaban and apixaban) or thrombin time (for dabigatran) can be helpful to determine the effect of anticoagulation and the best timing for neuraxial anaesthesia. In patients on antiplatelet therapy like aspirin or clopidogrel, timing is usually less well-defined but still critical to the control of bleeding

risk. The action of aspirin on platelet function may persist for several days, and neuraxial anaesthesia is usually deferred until platelet aggregation has normalised. Discontinuation in most patients on aspirin is advised at least five to seven days before the procedure. However, this may be adjusted based on the patient's underlying condition and the nature of the surgical procedure. Clopidogrel, which is a blocker of platelet ADP receptors, also needs at least 7 to 10 days of discontinuation due to its long duration of action on platelet aggregation. The timing for the cessation of antiplatelet therapy largely relies on the situation at hand; for example, in high-risk patients for cardiovascular events, for instance, with recent coronary stent placement, delaying neuraxial anaesthesia is not possible, and other methods might be planned [4,8].

CHALLENGES AND CONTROVERSIES

Management of neuraxial anaesthesia in antithrombotic medication-taking patients is beset by challenges and controversies that have given rise to heated debates among physicians. Although the progress in anaesthesia techniques and pharmacology has opened up possibilities for better outcomes in patients, the dangers posed by bleeding complications in such patients are a source of concern. The inherent uncertainty regarding the precise timing of drug discontinuation, the effect of newer anticoagulants, and the risk of complications like spinal or epidural haematomas add to the complexity of the decision-making process [16].

One of the main issues is how to decide on the ideal timing of discontinuing or modifying antithrombotic therapy before neuraxial anaesthesia. Although guidelines do exist, these are frequently broad and do not consider the considerable variation in patient-specific factors influencing drug pharmacokinetics, e.g., renal impairment, age, and comorbid conditions [3,14,17]. The inconsistency of the clearance of drugs like the DOACs, with their varying half-lives, complicates attempts at a single, uniform solution. Specifically, for DOAC-treated patients, the issue of drug discontinuation is complicated by the absence of standardised protocols and the need for physicians to use clinical judgment and patient-specific characteristics, often in the setting of unavailable rapid, reliable tests to quantify drug levels accurately. This absence of explicit, normative guidelines has caused heterogeneity in practice and some procedural delays as teams wait for the best time to perform anaesthesia based on coagulation status [18].

Although more evidence exists that neuraxial anaesthesia is safe in patients on antithrombotic treatment, there remains substantial concern regarding haematomas of the spinal or epidural variety, which will cause irreversible neurological impairment. The inability to accurately predict an individual patient's bleeding risk, particularly with chronic or combined antithrombotic therapy, makes the process of decision-making more challenging. For example, patients taking anticoagulants and antiplatelet agents might be at even greater risk, yet few established protocols for the treatment of such high-risk patients exist. The apparent safety of neuraxial anaesthesia in patients on single-agent therapy, e.g., aspirin or warfarin, is in contrast to the uncertainty regarding its safety in patients on dual or multiple antithrombotic therapies. This creates a dilemma for the clinician, who has to balance the advantages of regional anaesthesia against the danger of potentially disastrous complications [6].

Another controversial topic is the growing use of more recent anticoagulants, i.e., apixaban, rivaroxaban, and dabigatran. Even though the drugs have advantages, like fixed dosing and consistent anticoagulation, they are relatively new, and much remains unknown regarding their interaction with neuraxial anaesthesia. Even though they have a quicker onset and recovery compared to usual warfarin therapy, variable effects in certain groups, such as patients with renal insufficiency or elderly patients, make it difficult to determine rigid guidelines regarding when neuraxial anaesthesia may be employed safely. In addition, the lack of an established reversal agent for some of these newer agents- most significantly dabigatran- adds yet another complicating element to the process of making decisions about its use in patients receiving neuraxial anaesthesia. Difficulty in management has generated debate among anaesthesiologists concerning whether these drugs are as potentially dangerous as warfarin when it comes to bleeding complications or if they pose a less but separate risk [11,19].

Additionally, there is controversy surrounding the need and utility of regular monitoring of coagulation parameters in antithrombotic therapy patients. Specifically, point-of-care testing strategies to measure platelet function or coagulation levels in patients on newer anticoagulants are controversial. A few clinicians propose routine preoperative testing of coagulation status as part of a systematic preoperative evaluation. In contrast, others opine that these tests do not contribute substantial value, especially with no known thresholds for safe neuraxial anaesthesia in patients on newer medications. The lack of universally recognised monitoring guidelines indicates that clinicians need to use their subjective judgment to decide whether or not to perform regional anaesthesia, adding another layer of controversy to clinical practice [20].

Ethics are also at stake in the challenges to the management of neuraxial anaesthesia in this group. Withholding or withholding and restarting antithrombotic medication involves a sensitive balance between risk of bleeding and risk of thromboembolic event, for example, stroke or DVT. In a few high-risk scenarios, withholding or delaying antithrombotic treatment may make the patient more vulnerable to clot formation than the intervention. This presents an ethical dilemma to medical professionals, who must balance the immediate and long-term risk to the patient. In addition, patient autonomy plays a critical role, with patients needing to be adequately informed of the risks and benefits of neuraxial anaesthesia if they are taking antithrombotic medication [6,11].

FUTURE DIRECTIONS IN RESEARCH

Future studies in neuraxial anaesthesia of antithrombotic-treated patients need to address a few critical areas for improving patient safety and optimising clinical practice. Initial evidence-based standardised guidelines on neuraxial timing in patients who are on standard and newer drugs like DOACs are an immediate requirement. Studies on pharmacokinetics and drug interactions of these medications and anaesthesia administration could provide definite protocols. Examining the role of coagulation monitoring devices, such as point-of-care platelet function testing and certain drug levels, would assist in directing anaesthesia decisions and enhance the predictability of bleeding risk. More research on the safety of neuraxial anaesthesia in patients receiving dual or multiple antithrombotic therapies is required since the risks in these patients are not yet well understood. Studies on the management of bridging anticoagulation therapy may help further elucidate how to balance the risk of bleeding against the requirement for continued anticoagulation in the perioperative setting. These studies are necessary to optimise patient outcomes and establish concise guidelines for anaesthesiologists treating such complicated cases [17,21].

CONCLUSION(S)

The management of neuraxial anaesthesia in patients on antithrombotic medication should balance very carefully the benefits and risks of such medication. Anticoagulant and antiplatelet medications complicate decision-making since they increase the risk of bleeding and possible neurological damage, including epidural or spinal hematoma. While there are recommendations to prevent these complications, an even more personalised, patient-centred strategy based on individual pharmacodynamics, comorbidities, and operative needs is still indicated. The drugs to be used, timing of drug withdrawal, proper coagulation monitoring,

and changes in anaesthetic technique are critical steps to prevent complications. However, there are controversies and challenges, especially in the management of patients on new anticoagulants or dual antithrombotic therapy. In spite of such concerns, research into the pharmacology of antithrombotic medications, coagulation tests, and safety of long-term neuraxial anaesthesia in these patients is critical to the establishment of better, evidence-based guidelines. As the health care environment continues to change with new therapies and technology, ongoing refinement of knowledge regarding the safe delivery of neuraxial anaesthesia to patients on antithrombotic therapy is essential. A team-based, multidisciplinary approach, judicious patient evaluation, and conservative clinical decision-making will continue to be the foundation for optimising patient outcomes and safety. Further research will eventually provide more definitive recommendations, and clinicians will be able to optimise care in this challenging group of patients.

REFERENCES

[1] Agarwala R, Morrison B. Neuraxial anaesthesia and its role in enhanced recovery after surgery: A narrative review. Dig Med Res. 2022;5:20-20.
[2] Douketis JD, Syed S, Li N, Narouze S, Radwi M, Duncan J, et al. A physician survey of perioperative neuraxial anesthesia management in patients on a direct oral anticoagulant. Res Pract Thromb Haemost. 2021;5(1):159-67.
[3] Kietaibl S, Ferrandis R, Godier A, Llau J, Lobo C, Macfarlane AJ, et al. Regional anaesthesia in patients on antithrombotic drugs: Joint ESAIC/ESRA guidelines. Eur J Anaesthesiol. 2022;39(2):100-32.
[4] Eikelboom JW, Hirsh J, Spencer FA, Baglin TP, Weitz JI. Antiplatelet drugs. Chest. 2012;141(2):e89S-e119S.
[5] Alquwaizani M, Buckley L, Adams C, Fanikos J. Anticoagulants: A review of the pharmacology, dosing, and complications. Curr Emerg Hosp Med Rep. 2013;1(2):83-97.
[6] Allen DJ, Chae-Kim SH, Trousdale DM. Risks and complications of neuraxial anesthesia and the use of anticoagulation in the surgical patient. Bayl Univ Med Cent Proc. 2002;15(4):369-73.
[7] Cormier S, Siegal DM. A framework for managing severe DOAC-related bleeding: Recognize, reverse, resume, reduce. Thromb Update. 2024;15:100165.
[8] Ghasemi MA, Ghadimi E, Shamabadi A, Mortazavi SJ. The perioperative management of antiplatelet and anticoagulant drugs in hip fractures: Do the surgery as early as possible. Arch Bone Jt Surg. 2022;10(6):490-500.
[9] Zambouri A. Preoperative evaluation and preparation for anesthesia and surgery. Hippokratia. 2007;11(1):13-21.
[10] Volovár Š, Tancošová R, Rokyta R. Bridging anticoagulation therapy. Cor Vasa. 2018;60(4):e400-e406.
[11] Ashken T, West S. Regional anaesthesia in patients at risk of bleeding. BJA Educ. 2021;21(3):84-94.
[12] Schäfer ST, Otto AC, Acevedo AC, Görlinger K, Massberg S, Kammerer T, et al. Point-of-care detection and differentiation of anticoagulant therapy - development of thromboelastometry-guided decision-making support algorithms. Thromb J. 2021;19(1):63.
[13] Rossini R, Musumeci G, Visconti LO, Bramucci E, Castiglioni B, De Servi S, et al. Perioperative management of antiplatelet therapy in patients with coronary stents undergoing cardiac and non-cardiac surgery: A consensus document from Italian cardiological, surgical and anaesthesiological societies. EuroIntervention. 2014;10(1):38-46.
[14] Horlocker TT, Wedel DJ, Rowlingson JC, Enneking FK, Kopp SL, Benzon HT, et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition). Reg Anesth Pain Med. 2010;35(1):64-101.
[15] Pozza DH, Tavares I, Cruz CD, Fonseca S. Spinal cord injury and complications related to neuraxial anaesthesia procedures: A systematic review. Int J Mol Sci. 2023;24(5):4665.
[16] Gogarten W, Vandermeulen E, Van Aken H, Kozek S, Llau JV, Samama CM. Regional anaesthesia and antithrombotic agents: Recommendations of the European Society of Anaesthesiology: Eur J Anaesthesiol. 2010;27(12):999-1015.
[17] Moster M, Bolliger D. Perioperative guidelines on antiplatelet and anticoagulant agents: 2022 Update. Curr Anesthesiol Rep. 2022;12(2):286-96.
[18] Douketis JD, Spyropoulos AC, Spencer FA, Mayr M, Jaffer AK, Eckman MH, et al. Perioperative management of antithrombotic therapy. Chest. 2012;141(2):e326S-e350S.
[19] Paul C, Baby M, Anthraper AR, K K. NOACs: An emerging class of oral anticoagulants-A review article. Future J Pharm Sci. 2020;6(1):95.
[20] McRae HL, Militello L, Refaai MA. Updates in anticoagulation therapy monitoring. Biomedicines. 2021;9(3):262.
[21] Amaraneni A, Chippa V, Goldin J, et al. Anticoagulation Safety. [Updated 2024 Oct 6]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.

PARTICULARS OF CONTRIBUTORS:
1. Junior Resident, Department of Anaesthesia, Jawaharlal Nehru Medical College, DMIHER, Sawangi, Wardha, Maharashtra, India.
2. Professor, Department of Anaesthesia, Jawaharlal Nehru Medical College, DMIHER, Sawangi, Wardha, Maharashtra, India.
3. Senior Resident, Department of Anaesthesia, Jawaharlal Nehru Medical College, DMIHER, Sawangi, Wardha, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Prachi Siddharth Kamble,
Junior Resident, Department of Anaesthesia, Jawaharlal Nehru Medical College, Sawangi Meghe, Wardha-442107, Maharashtra, India.
E-mail: kambleprachi022@gmail.com

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