Complications of Percutaneous Coronary Intervention

The Survival Handbook

Alistair Lindsay
Kamal Chitkara
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Preface

One of the most remarkable features of interventional cardiology practice is its low complication rate. Less than 1 % of procedures result in a serious complication overall, and this figure has remained consistently low even as the technical complexity of coronary procedures has evolved over the last few decades.

When complications do occur, however, they may not always be straightforward to diagnose or treat; a coronary intervention procedure can go wrong in the blink of an eye for any number of reasons. In the heat of the moment, the cardiologist may be forced to perform several demanding tasks at once, such as weighing up feedback on the patient's condition, gaining more vascular access, and inserting pieces of equipment – all while trying to assess the pros and cons of possible subsequent courses of action. In such circumstances, a cool head and swift hands are needed to ensure an optimal outcome for the patient, and it is here that even the slightest bit of prior knowledge on how to best deal with a complication can help guide the cardiologist's actions, and ultimately make the difference between life and death.

Although there can be no substitute for experience, the purpose of this book is to provide the knowledge that may help in emergency situations. With increasingly busy clinical schedules, there is now less time than ever before for clinicians to discuss the nuances of interventional techniques, and what may go wrong with them. By drawing together the vast experience of a wide range of interventional cardiology practitioners, this book has been constructed to show what can be done in many of the most common – and some not so common – emergencies in the cardiac catherisation suite. Additionally, it provides practical hints and tips for overcoming many of the most common difficulties found in day-to-day interventional practice, such as difficulties in gaining vascular access. It is intended for anyone that practices or assists cardiac catheterisation techniques, but particularly those starting out on their interventional cardiology careers. For staff who support interventional cardiology procedures, such as nurses, technicians, and radiographers, the description of commonly encountered emergencies and the actions needed to resolve them will also be of value.

Whatever their job description or level of experience, it is our hope that, through the practical solutions outlined in the following pages, the reader will find him- or herself better prepared for future emergencies, and that many lives are saved as a result.

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Part I

Accessing the Heart

Preventing and Treating Vasovagal Reactions

1

Andreas S. Kalogeropoulos and Alistair Lindsay

Abstract

Vasovagal reactions are common in patients undergoing percutaneous coronary intervention under local anaesthetic, and most commonly present with a low heart rate and/or peripheral vasodilatation coupled with some degree of transient cerebral dysfunction, ranging from lightheadedness to a brief loss of consciousness. While normally brief, and often self-limiting, prompt recognition and treatment – and where possible prevention – of vasovagal reactions is an important skill for all interventional cardiologists. Several prophylactic measures such as the avoidance of dehydration, the reduction of pain perception with adequate analgesia and conscious sedation, the utilization of closure devices after removal of femoral arterial sheath and the intra-arterial administration of vasodilators in the radial artery approach, may be of great value.

Keywords

Vasovagal • Anxiety • Hypotension • Bradycardia • Analgesia • Sedation

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Introduction

Coronary angiography (CAG) and percutaneous coronary intervention (PCI) are cornerstones in the process of detecting, quantifying and treating coronary artery disease. Amongst the various complications associated with PCI, vasovagal reactions are relatively common, with a reported incidence of between 3.4 and 13.9 % [1–3]. Therefore, the implementation of a preventive strategy using appropriate preprocedural preparation is highly recommended in all patients, not just those who show signs of anxiety prior to the procedure.

Definition, Pathophysiology, Symptoms

Vasovagal reactions refer to a constellation of clinical symptoms and signs, caused by a neural reflex which results in an inappropriate, usually self-limiting, decrease in blood pressure. They are commonly characterized by low heart rate and/or peripheral vasodilatation and some degree of transient cerebral dysfunction, ranging from lightheadedness to a brief loss of consciousness [4, 5]. However, in some patients, those with an implanted permanent pacing system, a vasovagal phenomenon may be manifested mainly by a decline in systemic blood pressure, with little or no change in heart rate [6]. In general, a vasovagal reaction can manifest in three different ways:

(a) Mixed response (most common) with cardio-inhibitory and vasodepressor components.
(b) Cardio-inhibitory response with low heart rate.
(c) Vasodepressor response with little or no change in heart rate.

A vasovagal reaction usually occurs in response to painful and noxious stimuli, tissue injury, or strong emotional stress (Table 1.1) and is often accompanied by additional symptoms including diaphoresis, nausea, pallor, hyperventilation, and mydriasis.

Enhanced vagal tone with simultaneous withdrawal of sympathetic stimulation constitutes the predominant underlying pathophysiologic mechanism [7, 8] and can be mediated by two main pathways: (1) a central pathway, triggered by pain or anxiety; (2) a reflex pathway, via vagal afferent nerves, initiated by the left ventricular chemoreceptors and mechanoreceptors (the so-called Bezold-Jarisch reflex). In the setting of CAG and PCI the first mechanism is more commonly encountered, since the patient often experiences discomfort or emotional stress, is supine at the time of the reaction, and normally does not have any substantial bleeding. However, the Bezold-Jarisch reflex is a common pathophysiologic feature of the autonomic symptoms that accompany other complications that might ensue during coronary artery interventions, such as myocardial infarction (most commonly of the right coronary artery), tamponade and bleeding – conditions that should always be included in the differential diagnosis when a patient develops symptoms and signs of haemodynamic compromise in the catheterization laboratory.

Table 1.1 Triggering stimuli for vasovagal reactions

1. Prolonged standing or upright sitting
2. Standing up quickly
3. Stress
4. Postural orthostatic tachycardia
5. Pain (arterial puncture, venipuncture, lidocaine needle insertion, chest pain)
6. Inferior myocardial infraction (Bezold-Jarisch reflex)
7. Trauma
8. Unpleasant visual stimuli
9. Extreme emotional reaction
10. Lack of sleep
11. Dehydration
12. Urination or defecation
13. Compression of vagal nerve regions or sites of vagal innervation (throat, sinuses, eyes)
14. Drugs that acutely raise blood pressure such as amphetamine

Common Causes of Vasovagal Reactions

During diagnostic heart catheterization or PCI, a vasovagal reaction is usually the result of intractable discomfort and pain or profound emotional stress and anxiety of the patient. Symptoms similar to a vasovagal phenomenon have also been associated with the intracoronary injection of dye contrast agents, which might result in inappropriate arterial vasodilatation and vasodepressor response [9]. The latter was a common adverse reaction after the administration of ionic, hyperosmolar contrast agents and has been significantly diminished with the introduction of iso-osmolar or low-osmolar, non-ionic contrast agents [9]. Other precipitating factors that can render patients susceptible to neuro-cardiogenic syncope are dehydration and prolonged starvation prior to the procedure or even some anti-hypertensive medications such as β-blockers, non-dihydropyridine calcium channel blockers or the combination of these two [2].

Vasovagal events most commonly occur during the administration of local anaesthetic and the subsequent insertion of the arterial sheath, or later during the removal of the arterial sheath and the application of manual pressure. In a previous large retrospective analysis of 2,967 patients who underwent cardiac catheterization, more than 80 % of the vasovagal events occurred during the period when vascular access was being obtained [10]. In another prospective study with 611 participants who underwent a PCI; pain intensity, intervention to the left anterior descending artery, administration of nitrates during sheath removal and lower body mass index (BMI) were the strongest independent predictors for the occurrence of a vasovagal reaction [11].

Generally, vasovagal events are benign and rarely result in major adverse cardiac events; in the aforementioned study of patients who underwent cardiac catheterization and PCI there was no difference in the rate of major adverse cardiac events or acute stent thrombosis at 30 days post procedure compared to patients without

vasovagal episodes [11]. However, patients with critical coronary or valvular disease may undergo irreversible decompensation from vagally mediated hypotension; serious consequences such as asystole or myocardial infarction might occur.

Prevention and Management of Vasovagal Reactions

Prevention and prompt recognition and treatment of vasovagal reactions during percutaneous coronary interventions is pivotal in order to establish a smooth and uneventful procedure and avoid subsequent serious consequences; not least in patients with underlying co-morbidities such as significant valvular stenosis or critical coronary lesions. Advanced equipment design, improved peri-procedural management and increased experience of diagnostic centers and operators are indisputable parameters in the prevention of complications during and following percutaneous coronary artery interventions. In particular, careful identification of high-risk patients and adequate pre-procedural preparation should always be undertaken. Prolonged starvation and dehydration should be avoided and, if it is clinically indicated, treatment with intravenous normal saline is recommended. Prevention of hypovolaemia can be achieved with intravenous (IV) normal saline (more than 500 mL) for 4–6 h prior to the initiation of the procedure and more than 1,000 mL for 4–6 h after the procedure. Generally, younger patients with a low body mass index are more prone to develop vasovagal reactions [10]. Additional attention should be paid in nervous and anxious patients with marked emotional stress; conscious sedation might be indicated in order to minimize stress and diminish discomfort from the stressful stimuli. Prevention of pain at the site of the puncture, as well as during sheath insertion and removal, is essential in order to minimize the risk of developing vasovagal reactions during PCI. In addition, inadequate pain control may adversely affect patients' capacity to co-operate during the procedure and significantly increase the likelihood of other complications such as bleeding and vascular injuries. For both femoral and radial access strategies, arterial cannulation and sheath insertion is often the most painful part of the procedure. Several strategies aimed at minimizing pain perception and preventing vasovagal reactions in both the transfemoral and transradial approaches (Summary of management in Table 1.2) are described below.

Femoral Approach

Adequate conscious sedation with the combination of an opioid such as 2.5–5 mg of morphine or 25 mcg of fentanyl IV, with a benzodiazepine such as 1 mg of midazolam or 2.5 mg diazepam IV (reduced dose should be given in elderly patients), is generally recommended in high-risk patients (young, low BMI, anxious, low pain threshold). Identification of the optimal puncture site is crucial in order to: (a) minimize the number of punctures required to cannulate the common femoral artery; (b) facilitate the insertion of the arterial sheath; (c) establish a smooth and uneventful procedure (see Chap. 2, "Difficulty Gaining Femoral Access" for avoiding and

Table 1.2 Measures to prevent a vasovagal reaction, summary

General measures
1. Avoid prolonged starvation and dehydration. If indicated, administration of normal saline 0.9 % IV is recommended (>500 ml for 4–6 h prior and >1,000 mL an if necessary further 0.9 % saline for 4–6 hrs after the procedure). NB - caution must be taken in patients with severe valvular disease or ventricular dysfunction
2. Sedation and analgesia in anxious patients (1–2 mg of midazolam or 2.5–5 mg of diazepam and 2.5 mg of morphine or 25 mcg of fentanyl IV, reduced dose is indicated in elderly patients)
3. In high risk patients e.g. severe AS consider omitting beta-blockers or calcium channel blockers (non-dihydropyridines)
Femoral approach
1. Prior to lidocaine needle puncture consider conscious sedation if the patient is anxious and nervous
2. Achieve good local anaesthesia with lidocaine
3. Adapt an appropriate femoral artery puncturing technique to minimize the patient's discomfort. (Consider defining anatomic landmarks with fluoroscopy or using an ultrasound guided approach in difficult cases to avoid repeated attempts)
4. For haemostasis angioseal closure device is preferred over manual compression unless is contraindicated. If manual compression is chosen additional sedation with analgesia can be administrated as indicated
Radial approach
1. Main target is to avoid radial artery spasm
2. Moderate procedural sedation and analgesia (1 mg of midazolam or 2.5 mg of diazepam and 2.5 mg of morphine or 25 mcg of fentanyl IV, reduced doses in elderly patients)
3. Hydrophilic coated sheaths and catheters
4. Intra-arterial administration of vasodilators, verapamil 2.5 mg or 100–200 mcg of nitrates
5. Use diagnostic catheters specially designed for the engage of both the left and the right system to minimize the need for catheters exchange (TIGER or JACKY catheters, *TERUMO interventional systems*)

managing complications of femoral access). Adequate local anaesthetic should always be given, starting with a dermal bleb with a thin 25-gauge needle to anaesthetize the superficial skin. A 22-gauge needle is then used to anesthetize the deeper tissue layers, starting with the deepest point and working backwards, toward the skin. Usually 10–20 mL of local anaesthetic are required in order to achieve adequate local anaesthesia around the site of the common femoral artery. In cases where larger 6–8 F arterial sheaths are utilized, a nick and tunnel approach can be implemented to minimize tissue resistance and discomfort during sheath insertion. Usually, a 2–3 mm nick is made parallel to the skin crease at the site of the local anaesthesia with a scalpel blade. The nick is then enlarged and deepened with the use of the tip of a small curved forceps.

After optimal local anaesthesia, femoral arterial access is obtained with the use of 18-gauge needle, employing the modified Seldinger technique (see Chap. 2). The femoral artery should be palpated with the index and middle fingers and the needle should be held with the index finger and thumb, with the needle tip bevel facing

upwards. The skin is entered at a 30–40° angulation to ensure that the artery is cannulated approximately 2 cm superior to the skin entry site. More vertical angulation might result in difficulty advancing the sheath and guide wires and can also promote sheath kinking.

Minimizing pain and discomfort during the arterial sheath removal process is fundamental for the prevention of vasovagal reactions. In particular, a prospective (although not randomized) trial that enrolled patients who underwent PCI investigated the role of intravenous sedation and additional local anaesthesia in the prevention of vasovagal reaction after sheath removal and manual controlled compression. The routine use of intravenous fentanyl and midazolam, prior to sheath removal, lead to a significant reduction in pain perception and a trend for lower incidence of vasovagal episodes [11]. In contrast, the administration of local anaesthetic prior to sheath removal did not diminish pain perception and vasovagal events during and after arterial sheath removal. Furthermore, another prospective analysis demonstrated that the use of an angioseal closure device, instead of controlled manual compression, was associated with less pain and faster patient mobilization [1].

Radial Approach

Transradial arterial access for performing CAG and PCI is now commonplace. Various clinical trials have demonstrated that the implementation of this specific approach in daily practice has several advantages over the femoral route and can diminish vascular complications and patients' discomfort, as well as lessen the duration of hospitalization [12–15]. However, radial spasm can precipitate vasovagal reactions, as can painful sheath insertion and removal [16]. Adequate local anaesthesia and optimal site selection for the arterial puncture are essential in order to minimize the number of attempts to successfully cannulate the radial artery, diminish patient's discomfort and eventually reduce the likelihood of radial spasm occurrence.

Using a 25-gauge needle, local anaesthetic is injected to anaesthetize the superficial skin by creating a small dermal bleb; most operators choose to do this once the arm is prepared and draped, although some prefer to do this earlier in order to ensure the local anaesthetic has time to act. The amount of anaesthetic that is given should be enough to achieve adequate local anaesthesia but not excessive, in order to avoid diminishing the radial pulse and disrupting the puncture process. Furthermore, pre-treatment with a combination of an anxiolytic and an opioid analgesic can significantly reduce the incidence of radial spasm. The implementation of this strategy could be very effective in a special group of patients such as those with excess anxiety and nervousness, with a low pain threshold or those at high risk of developing radial spasm such as females, smokers, patients with small BMI and shorter stature [17]. After adequate anaesthesia is achieved, a nick and tunnel approach is usually applied to minimize tissue resistance and discomfort during sheath insertion. Usually a 2–3 mm nick is made 1–2 cm cranial to the bony prominence of the distal radius with a scalpel blade. The nick is then enlarged and

deepened with the use of the tip of a small curved forceps. The micro-puncture needle is used at a 30- to 45-° angulation and slowly advanced until a small amount of blood pulsates out of the needle. After fixing the position of the needle, a 0.018-in. guidewire is carefully introduced into the artery with a gentle twirling motion. The utilization of hydrophilic-coated arterial sheaths is preferable compared to uncoated ones, as the former have been consistently associated with significantly reduced rates of radial spasm and decreased pain perception by the patient [18–21]. In contrast, there is controversy regarding the effectiveness of longer sheaths with contradictory results as far as the prevention of radial artery spasm is concerned [19]. Furthermore, aiming to use a smaller number and size of catheters might also contribute to a reduction in the occurrence of radial spasm; for more discussion on this topic, please refer to Chap. 4, "Preventing and Treating Radial Spasm."

The intra-arterial administration of vasodilating agents is fundamental to prevent radial artery spasm. Various intra-arterial vasodilating cocktails are used depending on the catheterization laboratory protocol. The combination of 2.5–5 mg of verapamil and 100–200 μg of nitrates is a common regimen. The combination of these agents has been found to reduce the incidence of radial artery spasm and patient's discomfort up to 14 % and 20 %, respectively [22]. In the two largest randomized trials, SPASM 1 and SPASM 2, the combination of verapamil 2.5 mg with molsidonine 1 mg administered intra-arterially reduced the incidence of radial artery vasoconstriction by 17.3 % [23].

Treatment of Vasovagal Reaction

In patients with marked hypotension and bradycardia, prompt treatment with IV administration of atropine 0.6–1.2 mg within 2 min is used as first line treatment, with simultaneous administration of bolus IV saline or colloids for volume expansion (usually repeated bolus doses of 250 mL). Even in cases with an isolated vasodepressor response, atropine can be markedly effective in reversing haemodynamic compromise and stabilizing blood pressure [24, 25]. In patients without peripheral intravenous access, intra-aortic administration of atropine is possible. In patients with refractory bradycardia associated with haemodynamic compromise, temporary transvenous pacing may occasionally be required. In those that do not respond to initial pharmacologic therapy, further treatment with adjunctive inotropic agents might be necessary, and attempts made to look for any more serious complications that may be causing prolonged hypotension and bradycardia.

Conclusion

Vasovagal reactions are common adverse reactions during CAG and PCI. They are usually benign but they can lead to serious haemodynamic compromise in patients with critical coronary artery disease and/or severe aortic stenosis. Thus, risk stratification and good preparation of patients are essential in order to prevent their occurrence. Patients at higher risk are typically young, low BMI, female, short stature individuals or those on beta-blockers, calcium channel