Spasm in Arterial Grafts in Coronary Artery Bypass Grafting Surgery

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Spasm of arterial grafts in coronary artery bypass grafting surgery is still a clinical problem, and refractory spasm can occasionally be lethal. Perioperative spasm in bypass grafts and coronary arteries has been reported in 0.43% of all coronary artery bypass grafting surgery, but this may be an underestimate. Spasm can develop not only in the internal mammary artery but more frequently in the right gastroepiploic and radial artery. The mechanism of spasm can involve many pathways, particularly those involving regulation of the intracellular calcium concentration. Endothelial dysfunction also plays a role in spasm. Depending on the clinical scenario, the possibility of spasm during and after coronary artery bypass grafting should be confirmed by angiography. If present, immediate intraluminal injection of vasodilators is often effective, although other procedures such as an intraaortic balloon pump or extracorporeal membrane oxygenation may also become necessary to salvage the patient. Prevention of spasm involves many considerations, and the principles are discussed in this review article.

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A search for original articles focusing on coronary revascularization published between 1950 and 2014 was performed in the Medline and PubMed databases. The search terms used were "arterial grafting antispastic," "coronary artery bypass grafting," and "spasm coronary arterial grafting," alone and in combination. All articles identified were English-language, full-text papers. We also searched the reference lists of identified articles for further relevant papers.

Coronary artery bypass grafting (CABG) surgery remains the standard treatment for patients with multivessel coronary artery disease. During the past decade, percutaneous coronary intervention has been increasingly used for revascularization in patients with one- and two-vessel disease (especially if not involving the proximal left anterior descending coronary artery), and for some simple three-vessel disease cases [1].

Using autologous arteries rather than veins as bypass grafts provides superior long-term outcomes [2, 3]. In particular, use of the left internal mammary artery (IMA) to graft the diseased left anterior descending coronary artery has become the standard method for almost all CABG surgery [3], although the long-term patency of the IMA also depends on the severity of the native artery stenosis and the quality of the distal vascular bed, as well as pedicled versus free graft, for example.

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During the last decade the potential additional survival advantage of a second IMA graft has been increasingly recognized [3]. In addition, other arteries such as the radial artery (RA) [4, 5], and less commonly the right gastroepiploic artery (GEA) [6–8] and the inferior epigastric artery (IEA) [9, 10], have been used as bypass grafts. However, with the exception of the IMA, other arterial grafts have a greater muscular component in the vessel wall and a consequent tendency to develop vasospasm during surgery. Therefore, antispastic therapy remains an important issue in CABG and particularly in the setting of multiple arterial grafting.

Studies of the pathophysiology of spasm and the development of antispastic pharmacologic and nonpharmacologic methods have paralleled the use of arterial grafts in CABG [11–31]. However, because of the complexity of the antispastic drugs and other methods involved, no single technique for antispastic management has been uniformly accepted or adopted. In this review, we discuss the evidence base for the existence and incidence of vasospasm in CABG surgery using arterial grafts, its pathophysiologic mechanisms, its clinical manifestations and management, and the principles of its prevention. Because of their complexity, pharmacologic antispastic methods and mechanisms of vasodilator agents will be discussed in detail in a separate review.

Material and Methods

Clinical Classification of Arterial Grafts

To better understand the differing biologic behavior of arterial grafts, a clinical classification may be useful for

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Abbreviations and Acronyms

CABG = coronary artery bypass grafting
ECMO = extracorporeal membrane oxygenation
GEA = gastroepiploic artery
IABP = intraaortic balloon pump
IEA = inferior epigastric artery
IMA = internal mammary artery
RA = radial artery

the practicing surgeon. Based on experimental studies of their vasoreactivity combined with anatomic, physiologic, and embryologic considerations, a functional classification for arterial grafts has been proposed that suggests there are three types of arterial grafts: type I, somatic arteries; type II, splanchnic arteries; and type III, limb arteries [19, 32].

Type I arteries, such as the IMA, have better endothelial function and release more nitric oxide and other vasorelaxing factors [32–34]; type II arteries, such as GEA, and type III arteries, such as the RA, have higher pharmacologic reactivity to vasoconstrictors and require more active pharmacologic interventions. In particular, type III arteries have even greater contractility [35] and less endothelial nitric oxide synthase expression [36] than type II arteries. This classification helps to explain why the IMA has the best long-term patency.

Clinical Incidence of Spasm

EXISTENCE AND PREVALENCE OF SPASM OF ARTERIAL GRAFTS. *Internal Mammary Artery Spasm: History and Current Prevalence.* The first report of early postoperative spasm as a clinical entity was in the left IMA graft in 1987 [37]. Subsequently it was reported that spasm could be a localized phenomenon or a diffuse process, referred to as a "string sign" [38]. Clinically, spasm of the IMA could cause hypoperfusion, typically occurring 30 to 40 minutes after discontinuation of cardiopulmonary bypass but also seen subsequently in the intensive care unit [39], when it may also be confused with coronary artery spasm. Spasm of the IMA has also been documented years after CABG [40].

These clinical reports promoted intense physiologic and pharmacologic studies on the biologic characteristics of the IMA to understand the nature of spasm. In an early study, one of us (G.W.H.) demonstrated that contractility of the distal section of the IMA is inversely correlated to its diameter; that is, the smaller the diameter, the greater the tendency for spasm to develop. This strongly suggests that the distal end of the IMA should not be harvested, thereby helping to prevent graft spasm [41] and also preserving the blood supply of the xiphoid process, reducing the risk of wound problems. In any event it is almost never necessary to use the distal IMA, particularly in the era of composite arterial grafting.

Nevertheless, since the 1980s, a large body of literature has continued to report the existence of spasm in the IMA, even in current practice [42–49], suggesting its ongoing clinical importance in CABG surgery. In the perioperative period, development of spasm is often sudden and angiography may characteristically show diffuse arterial spasm (strong contraction with significant narrowing of the lumen) of IMA or RA grafts as well as the native coronary arteries [42, 43, 48].

The exact prevalence of spasm of arterial grafts, particularly when it is mild, is unknown as it may be asymptomatic. However, when spasm is severe and does not respond to standard pharmacologic management, it can be lethal [42–46, 48]. A recent report indicated that among 5,762 patients who underwent isolated CABG, 7 patients (0.12%) experienced refractory spasm of coronary arteries and grafted conduits, including the left IMA, and an additional 18 patients (0.31%) experienced perioperative vasospasm of a single coronary artery or of a grafted conduit [48]. This suggests that the confirmed perioperative vasospasm in coronary arteries or grafts is approximately 0.43% (25 of 5,762 patients) in all CABG surgery. It is possible, however, that the incidence of vasospasm could be underestimated because there may be some patients whose graft spasm was responsive to the early use of vasodilator agents and therefore not counted as spasm.

Spasm of Other Arterial Grafts

A large body of literature has demonstrated that the RA and GEA are particularly prone to spasm. Indeed, revival of the use of RA during CABG was, at least in part, the result of the use of successful antispastic management [4]. However, patency and functionality of RA grafts are also related to other factors such as the severity of the native target artery stenosis, and that the nature of being a free graft may imply some degree of external wall ischemia, potentially leading to intimal hyperplasia after grafting.

Because it is more widespread, spasm of the RA has been reported more frequently by cardiac surgeons [50], physicians [51], and cardiologists during catheterization [52]. Gabe and colleagues [29] reported an unusual case of CABG, with vasospasm of a grafted RA leading to postoperative ventricular fibrillation. The RA graft spasm was demonstrated by angiography and was successfully resolved by intravenous nitroglycerin administration. However, focal RA graft stenosis may be difficult to differentiate from spasm and can be treated by percutaneous intervention [53].

Spasm of the GEA is also well recognized and can occur during or soon after harvest of these vasoreactive grafts [54–56] or even months or years postoperatively, as demonstrated by angiography [57].

There are fewer reports on IEA spasm. This is probably related to the fact that, first, the IEA is a type I artery that may be less spastic [19, 32] and, second, use of the IEA is not as frequent as for IMA, RA, or GEA grafts. However, occasionally, the IEA graft has been reported to have spasm at 5-year angiographic follow-up [58].

Possible Mechanisms of Spasm

Vascular spasm reflects a complicated physiological status within blood vessels and while its precise mechanism