Recent advances in anesthesia For Vascular surgery

Essay
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Abstract

Anesthetic requirements for vascular surgery for aortic, carotid, brain aneurysm and peripheral vascular disease are reviewed. Peculiarities of the surgery which may impinge on anaesthetic management are discussed together with the pre-operative assessment issues of particular relevance to patients with generalized vascular disease.

The detailed Recent advances in anaesthetic management for carotid and aortic endovascular repair is addressed. The lowered peri-operative stress and general morbidity levels which occur with endovascular surgery allow sicker patients with greater risk factors to present for this type of surgery, thus increasing the challenges facing anaesthetists.

Key word: aneathesia

Anta

Cantid

Cerebral aneurysm

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AAA:	Abdominal aortic aneurysm
ACE:	Angiotensin converting enzyme inhibitor
ACE:	Angiotensin converting enzyme
AT:	Anaerobic threshold
AVD:	Atherosclerotic vascular disease
CAB:	Coronary artery bypass graft
CAD:	Coronary artery disease
CEA:	Carotid endarterectomy
CPK:	Creatinine phosphokinase
CVP:	Central venous pressure
DSA:	Digital subtraction analysis
DSE:	Dobutamine stress echocardiography
DTS:	Dipyridamole thallium scan
EEG:	Electroencephalography
Endo AAR:	Endovascular aortic aneurysm repair
FEV_1:	Forced expiratory volume in the 1st second
FVC:	Forced vital capacity
GFR:	Glomerular filteration rate
HDL:	High density lipoprotein
HIT:	Heparin induced thrombocytopenia
INR:	International normalized ratio
JVP:	Jugular venous pressure
LDL:	Low density lipoprotein
LMWH:	Low molecular weight heparin
MAC:	Minimum alveolar concentration
MI:	Myocardial infarction
NSAID:	Nonsteroidal anti-inflammatory drug
PAC:	Pulmonary artery catheter
PAD:	Peripheral arterial disease
PMI:	Perioperative myocardial infarction
PTCA:	Percutaneous transluminal coronary angioplasty
TCD:	Trans-cranial Doppler

Introduction

The patient who presents for major elective vascular surgery is a high-risk surgical candidate, and is typically characterized by a history of atherosclerosis, hypertension, cigarette smoking, coronary artery disease, and diabetes mellitus. A combination of these clinical entities is often present and, depending on the severity of illness and state of control, these morbidities have an important impact on anesthetic practice. Recent studies have indicated that even less extensive procedures such as peripheral vascular surgery can have considerable morbidity and mortality.

Obviously any treatment that can significantly reduce the operative risk would benefit this group of patients.

In the last five years, vascular surgery has undergone a considerable change in emphasis with respect to the breadth of conditions being treated and in the techniques used in therapy.

The evidence base for vascular surgical intervention has broadened considerably, particularly in the fields of carotid interventions and the treatment of abdominal and thoracic aneurysms. This newly gathered evidence base has been used to further define the indications for vascular reconstruction. **(1)**

The emphasis on the development of new techniques for vascular intervention has continued, with the focus on minimally invasive and endovascular therapy. **(2)**

The application of endovascular therapy for the treatment of aortic and carotid disease is still largely confined to specialist centers but these techniques are likely to represent the future of vascular intervention.[3]

The change in direction of traditional vascular surgery has significant implications for anesthetic practice as most of the newer vascular techniques are amenable to loco-regional anesthesia.

The heart is the principle focus for the anesthesiologist in the management of patients undergoing peripheral vascular surgery, emphasizing the fact that myocardial dysfunctions the single most important cause of morbidity following vascular surgery.[4]

Prevention of other organ system (particularly the kidneys and brain) is also crucial. Low serum albumin values and high American society of anesthesiologists (ASA) physical classification are predictors of morbidity and mortality after surgery.[5]

Epidemiologic and pathophysiologic aspects of vascular diseases

Pathophysiology of Atherosclerosis

Atherosclerosis is a generalized inflammatory disorder of the arterial tree with associated endothelial dysfunction [6] .Putative etiologies are endothelial damage caused by hemodynamic shear stress, inflammation from chronic infections, hypercoagulability resulting in thrombosis, and the destructive effects of oxidized low-density lipoproteins (LDLs) that result in intimal damage. Inflammatory and degenerative processes which are characterized by the formation of intimal plaques composed of oxidized lipid accumulation, inflammatory cells, smooth muscle cells, connective tissue fibers, and calcium deposits play an important role in the pathogenesis of atherosclerosis. Later on Disruption of the fibrous cap over a lipid deposit can lead to plaque rupture and ulceration. Vasoactive influence can result in spasm and acute thrombosis.

As a major regulator of vascular homeostasis, the endothelium exerts numerous vasoprotective effects such as vasodilatation, suppression of smooth muscle cell growth, and inhibition of inflammatory responses. Many of these are mediated by nitric oxide, the most potent endogenous vasodilator. In atherosclerosis, the normal homeostatic functions of endothelium are altered, promoting an inflammatory response. In fact, chronic inflammation has been implicated at every stage of atherosclerosis, from initiation to progression and eventually, plaque rupture. Adhesion molecules expressed by inflamed

endothelium recruit leukocytes, including monocytes which then penetrate into the intima, predisposing the vessel wall to lipid accretion and vacuities. Markers of inflammation (e.g., acute phase reactants such as high-sensitivity C- reactive protein [has-CRP]) may be useful in predicting an increased risk of coronary heart disease.

Predisposing factors of atherosclerosis

Predisposing risk factors for atherosclerosis include many aspects of the metabolic syndrome: abdominal obesity, atherogenic dyslipidemia (increased LDL, decreased HDL , increased total cholesterol) , raised blood pressure, insulin resistance, proinflammatory state, and prothrombotic state[(7)] . More recent recommendations for more aggressive treatment of hypertension reflect studies that suggest that this will delay the progression of atherosclerotic disease; beginning at 115/75 mm Hg, cardiovascular disease risk doubles for each increment of 20/10 mm Hg throughout the blood pressure range [(8)].

Morbidities associated with atherosclerosis

Morbidity associated with atherosclerosis arises from plaque enlargement and lumen obstruction (e.g., lower-extremity arterial occlusion with limb ischemia) or plaque ulceration, embolization, and thrombus formation (e.g., transient ischemic attacks in patients with carotid disease). Alternatively, atrophy of the media due to atherosclerotic disease may weaken the arterial wall, producing aneurismal dilatation. The clinical expression of atherosclerosis tends to be focal, with clinical symptoms caused by localized interference with circulation occurring in several critical sites.

Major arterial sites that are particularly prone to the development of advanced atherosclerotic lesions include the coronary arteries, carotid bifurcation, infrarenal abdominal aorta, and iliofemoral vessels.

Natural History of Patients with Vascular Diseases

Throughout the last half of the twentieth century, coronary artery atherosclerosis has been a major focus for basic and clinical investigation. As a result, considerable strides have been made in the development of programs to prevent and treat the clinical manifestations of CAD. Yet atherosclerosis is a systemic disease with important sequelae in many other regional circulations, including those supplying the brain, kidneys, mesentery, and limbs. Elderly patients with symptomatic or even asymptomatic peripheral vascular disease have greatly increased mortality rates, particularly from cardiovascular causes (6- to 15-fold increases).

The prevalence of >25% carotid stenosis in patients older than 65 years of age was 43% in men and 34% in women in one of the Framingham studies. Patients with cerebral atherosclerosis are at increased risk for ischemic stroke. Presence of carotid artery disease also identifies patients at risk for fatal and nonfatal myocardial infarction (MI). Stroke risk is most strongly associated with previous stroke history and greater degree of illness. The risk of stroke is relatively uncommon (0.4% to 0.6% of patients) after noncarotid peripheral vascular surgery, but when it does occur it is associated with longer length of stay in hospital and higher mortality. [(9)]

Patients with renal artery atherosclerosis are at risk for severe and refractory hypertension and renal failure. The principal clinical syndromes associated with aortic atherosclerosis are abdominal aortic

aneurysms (AAAs), aortic dissection, peripheral atheroembolism, penetrating aortic ulcer, and intramural hematoma.

Patients with atherosclerosis affecting the limb (i.e., peripheral arterial disease [PAD]) can develop disabling symptoms of claudication or critical limb ischemia and its associated threat to limb viability. The prevalence of claudication is 2% among older adults, but 10 times as many elderly patients have asymptomatic lower extremity atherosclerosis, which can be detected by comparing blood pressure in the legs with blood pressure in the arms (ankle-brachial index).

Moreover, once disease is apparent in one vascular territory, there is increased risk for adverse events in other territories. For example, patients with PAD have a fourfold greater risk of MI and a twofold to threefold greater risk of stroke than patients without PAD.

AAAs occurs in up to 5% of men older than 65 years of age; most of them are small and require only infrequent follow-up. Data suggest that the risk of rupture is very low for AAAs 4.0 cm in diameter or less but rises exponentially for AAAs greater than 5 cm. AAAs between 4 and 5 cm in diameter should be followed every 6 to 12 months to determine whether they are increasing in size. [(10)]

Aneurismal Disease:

Aneurysms pose an ever-present threat to life because of their unpredictable tendency to rupture or embolize. Mortality from rupture may be as high as 85%, and even patients who receive emergent surgery have mortality rates one-half that. Therefore, early recognition and aggressive surgical management are warranted, even in the absence of symptoms.

Epidemiology and Pathophysiology of Abdominal Aortic Aneurysm

There are approximately 200,000 new AAAs diagnosed annually with approximately 45,000 undergoing surgical repair per year in the United States. A population-based study in Norway in 1994 to 1995 used ultrasound to measure renal and infrarenal aortic diameters; an aneurysm was present in 263 (8.9%) of men and 74 (2.2%) of women ($P < .001$). Risk factors for aneurysm included advanced age, smoking >40 years, hypertension, low serum HDL cholesterol, high level of plasma fibrinogen, and low blood platelet count. This study indicates that risk factors for atherosclerosis are also associated with increased risk for AAA Thoracoabdominal aneurysms also occur in patients with hypertension or other risk factors for atherosclerotic disease.

AAA represents a dilatation of the abdominal aorta generally below the level of the renal arteries. The risk of rupture of the AAA is directly related to the luminal diameter of the aortic aneurysm. The aneurysm can develop an inner lining of mural thrombus, thereby decreasing the effective luminal diameter, but the size of the mural thrombus has not been shown to significantly decrease the risk of rupture. The risk of aortic rupture is only related to the absolute diameter of the aortic aneurysm sac. The risk of rupture increases once the aneurysm is greater than 4.5 to 5 cm in diameter. The size of the aneurysm is the most important predictor of subsequent rupture and mortality.

A prospective study followed 300 consecutive patients (mean age 70 years; 70% men) who presented with AAA (average size, 4.1 cm) and were initially managed no operatively. The diameter of the aneurysm increased by a median of 0.3 cm per year. The 6-year cumulative