

A Subtle Clinical Finding in Free Tissue Transfer: The Blood Hammer Phenomenon

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Sir,

Flap monitoring after free tissue transfer is crucial for reducing the risk of vascular death (Table 1). A common approach for flap monitoring is the use of acoustic Doppler sonography, in which the frequency of ultrasonic waves emitted from a probe changes in response to the underlying flap blood flow. These frequency variations result in sound production, mediating the interpretation of flap perfusion. Doppler sonography allows for an accurate assessment of arterial perfusion. It is less useful for venous assessment due to difficulties with interpretation and frequent absence of the venous signal. Nevertheless, careful attention to the arterial signal may offer subtle clues regarding the venous system. We present one such finding, which, though rarely discussed, is potentially invaluable in flap assessment.

A well-studied principle of hydraulic engineering is the water hammer phenomenon.¹ This effect occurs when the flow of a fluid within a pipe is halted by an abrupt valve closure. The kinetic energy of the upstream fluid reduces to zero, resulting in an increased pressure at the valve, an upstream movement of a primary pressure wave at the speed of sound, and the presence of secondary (“bouncing”) waves until the fluid comes to a rest.² In 1976, Damsa et al³ identified a similar effect during the investigation of cerebral hemodynamics and, by way of analogy, introduced the term “blood hammer phenomenon” to describe a sudden increase in the upstream pressure in a vessel due to obstruction. Subsequently, a blood hammer effect has been identified in both large and small vessels, particularly in the setting of vessel occlusion. Mathematic and computer modeling of this phenomenon (Fig. 1) has facilitated a quantitative study of the effects of partial and complete vessel obstruction on pressure, blood flow velocity, and sound frequency.^{4,5}

In our experience, an often subtle but early indicator of venous compromise is a characteristic change in the arterial Doppler signal that is dampened, muffled, and suggestive of an increased pressure within the system. In contrast to normal triphasic signals, a subtle yet characteristic change may be appreciated during auscultation

Table 1. Common Methods of Free Flap Monitoring

	Method	Description
Noninvasive	Physical examination	Assessment of flap appearance, color, temperature, turgor, capillary refill
	Acoustic Doppler sonography	Frequency of ultrasound waves fluctuates in relation to blood flow velocity, producing an audible signal
	Color duplex ultrasonography	Use of ultrasound to directly visualize vessels
	Laser Doppler flowmetry	Frequency of laser light fluctuates in relation to blood flow velocity, producing velocity measurements
	Near-infrared spectroscopy	Measurement of concentrations of oxygenated and deoxygenated hemoglobin
Invasive	Implantable Doppler Flow coupler	Doppler probe secured directly around an artery or vein through a wire attached to the venous coupler
	Bleeding pin-prick	A needle is used for illicit bleeding from the surface of the flap
	Fluorescent dye perfusion	Evaluation of flap perfusion using an injectable fluorescent dye

of flaps with venous obstruction. When considering physiologic and hemodynamic alterations associated with outflow obstruction, we hypothesize that the blood hammer phenomenon may underlie the arterial signal change audible by Doppler sonography in a venous-compromised flap. Similar to the closure of a pipe valve, venous obstruction results in changes to pressure and blood flow within the flap vasculature. Upon application of a Doppler probe, the frequency of the emitted ultrasound waves responds to these changes, producing an altered signal.

This article is the first to address how a subtle yet characteristic change in arterial signal quality may signify venous obstruction due to the blood hammer phenomenon. However, further analysis is required to conclusively make this link. Additionally, it would be useful to elucidate the sensitivity and specificity of this signal change in detecting venous compromise. The decision to return to the operating room for flap exploration is not always straightforward, and surgeons must use available indicators to determine the flap status. The clinical finding presented herein offers an additional means to potentially identify venous obstruction before the onset of more obvious signs.

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$$\Delta p = v\rho \times [\rho (K + d/E\delta)]^{-1/2}$$

Δp , change in upstream pressure (mm Hg)

v , blood flow velocity (m/s)

ρ , density of blood (kg/m³)

K , compressibility of blood (10⁻¹⁰/Pa)

d , internal vessel diameter (mm)

E , elasticity modulus (10⁶ Pa)

δ , vessel wall thickness (mm)

Fig. 1. The equation to calculate an upstream change in pressure (Δp) at the rapid closure of valves. Applied by Ahlqvist⁵ to calculate the increase in the upstream pressure after an embolic occlusion of the middle cerebral artery, the equation shows the relationship between pressure change (Δp) and the vessel diameter (d), which may be affected by thrombus, embolus, or other obstructive events.

DISCLOSURES

The authors have no financial interest to declare in relation to the content of this article.

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