Doppler Imaging of Arteriogenic and Venogenic causes of Erectile Dysfunction: A Pictorial Review

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Learning Objectives

To describe the ultrasound findings of arterial and venous causes of erectile dysfunction
Background

Erectile dysfunction is a multifactorial disease which can occur following trauma or in concert with the burden of increasing age. The vascularity and neural sensitivity of the penis also make it vulnerable to systemic disease processes. Erectile dysfunction (ED) in particular can indicate cardiovascular risk [1] and the most common cause of ED is from arteriogenic or venogenic causes [2]. The prevalence of ED is uncertain, but in older males it is estimated to be between 25-30% and 52% [3] [4].

Ultrasound is a commonly used, readily available and minimally invasive modality for investigating the uniquely superficial structure and vascular anatomy of the penis. Its current use is in patients with poor, or no response to, oral medication, or in the post trauma setting [5]. Ultrasound allows assessment of the anatomy of the penis and vascular hemodynamics. Evaluation of pharmacologically induced erection can complete the picture of vascular, traumatic or central neurogenic causes of ED.
Anatomy of the Penis

The erectile tissue lies surrounded by the fibrous tunica albuginea (Fig 1 open arrow). It comprises three columns of trabecular, sinusoidal smooth muscle network: paired corpora cavernosa (Fig. 1 b), enclosing central cavernosus arteries (Fig 1 solid arrow), and the corpus spongiosum (Fig 1 a), enclosing the urethra and distally forming the glans penis. Specialised lacunar spaces formed by these networks are normally readily dilated with arterial blood during erection[6].

Doppler ultrasonography can usefully demonstrate much of the penile vasculature[7]. The internal pudendal artery divides into three branches: the dorsal penile artery, supplying the glans, the cavernosal artery, supplying the corpora cavernosa, and the bulbar artery, supplying bulb and corpus spongiosum. The cavernosal artery divides to supply the paired cavernosa, terminating in helicine arteries that open into the lacunar spaces. Arterial variation, usually due to additional branches, is common[8],[9] and can be surgically important if pudendal blood supply is compromised[10].

When flaccid, emissary veins freely collect lacunar blood, passing beneath the tunica albuginea and joining extratunical veins, eventually forming venous plexuses. Drainage is typically parallel and unconnected between veins of the corpora cavernosa and corpus spongiosum. Venous outflow can be so inconspicuous that it is undetectable on colour Doppler without partial erection[11],[12].

Erection Physiology

When flaccid, corporal smooth muscle is naturally semi-contracted. Local or psychogenic stimulation leads to neurovascular changes that produce tumescence via the "veno-occlusive mechanism"[13].

Sacral parasympathetic release of nitric oxide raises intracellular cyclic GMP (cGMP) concentrations, causing smooth muscle relaxation. Cavernosal and helicine arterioles vasodilate, and compliance increases in the trabecular network, allowing sinusoids to fill rapidly with blood. The non-distensible tunica albuginea limits this engorgement, which eventually compresses and effectively occludes venous outflow. Increasing pressure produces erection at approximately
100mmHg [14], and in normal physiological states the penis increases in rigidity by several hundred millimetres of mercury thereafter.

Detumescence primarily occurs when phosphodiesterase type 5 (PDE5) degrades cGMP, returning smooth muscle to a contracted state and reversing veno-occlusion. This can be targeted with orally available selective PDE5 inhibitors[15], prolonging elevated cGMP and its downstream effects. Another pharmaceutical target is direct modulation of the trabecular network with injectable prostaglandin E1 (PGE1), the only endogenous prostaglandin known to substantially relax human cavernosal tissue[16].

Investigation of Erectile Dysfunction

In our practice, the patient is imaged supine with the penis lying on the anterior abdominal wall. A high frequency probe is used (typically 12MHz linear transducer) and the penis is scanned transversely from the glans to the base.

Initially a global survey is performed to examine for the presence of fibrosis within the corporal bodies or palpable abnormalities such as Peyronie's plaques and their location noted. Occasionally, specific examination of the penile urethra is performed, and distension with a viscous lidocaine gel is used to aid visualisation. Imaging in the longitudinal plane is then performed of each corporal body and the cavernosal artery examined as a baseline.

Prostaglandin E1 is then injected. 10ug is injected into the dorsal 2/3rd of each corpora (for a total dose of 20ug). Doppler analysis of each cavernosal artery is performed every 5 minutes for 30 minutes. Traces are obtained near the base of the penis to minimise the Doppler angle. Peak systolic and end diastolic measurements are obtained. Additionally, the diameter of cavernosal arteries are measured pre and post injection as a 75% -120% increase in the diameter is a good indicator of normal arterial inflow into the cavernosal artery [9]

Normal

Doppler imaging is a reliable measure of investigation of cavernosal arterial blood flow as dynamic infusion cavernosometry and cavernosography (DICC) studies in the past have demonstrated that a systolic flow rate of between 5 and 40 ml/min is required to maintain an erection [17].In our practice a normal study constitutes a PSV greater than 35cm/s and an EDV of less than 5cm/s.
Changes in the spectral wave of the cavernosal arteries reflect the progression of the developing erection. This has been classified into six phases: 0- flaccid, I-III erection onset, IV- venoocclusion, V- rigid erection [18]

In the flaccid penis (stage 0) the spectral wave form is monophasic with minimal diastolic flow (Fig. 2a). As the erection develops (stages I-III), there is an increase in both systolic and diastolic flow (Fig 2b) with the appearance of a dicrotic notch at the end of systole (Fig 2c) and abatement of of diastolic blood flow (Fig 2d). The onset of veno-occlusion (stage 4) is heralded by reversal of diastolic flow (Fig 2e), with loss of reversal of flow and often reduction of the systolic peak at tumescence (Fig 2f).

Arteriogenic Erectile Dysfunction

There is debate about the threshold for diagnosing arteriogenic causes of ED [5], however, in our practice we use a cut off of PSV < 35cm/s, with severe arterial deficiency identified at PSV < 25cm/s (Fig 3).

Following injection, there is an increase in the PSV, however even at 30 minutes post injection, the PSV remains below 35cm/s. Furthermore, there is frequently persistent elevation of the EDV, indicating that the PSV has been insufficient to allow veno-occlusion.

Venogenic Erectile Dysfunction

Venogenic erectile dysfunction is secondary to failure of veno-occlusion due to venous incompetence, or venous leakage. On doppler imaging, this corresponds to an elevated EDV of >5cm/s, in the context of a normal PSV. EDV values remain elevated throughout the procedure, with loss of reversal of flow signifying veno-occlusion (Fig 4).
Fig. 1: a) corpus spongiosum b) corpora cavernosum solid arrow: cavernosal artery open arrow: tunica albuginea

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Fig. 2: Normal stages of erection: a) Stage 0; b) Stage I; c) Stage II; d) Stage III; e) Stage IV; f) Stage V

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Fig. 3: Arteriogenic Erectile Dysfunction. a) 0 minutes post injection; b) 10 minutes post injection; c) 20 minutes post injection; d) 30 minutes post injection

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Fig. 4: Venogenic Erectile Dysfunction. a) 0 minutes post injection; b) 10 minutes post injection; c) 20 minutes post injection; d) 30 minutes post injection

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Conclusion

Erectile dysfunction is a relatively common complaint which has, with the advent of PDE 5 inhibitors, become easier to manage and treat. However, for patients whom have continued erectile dysfunction, doppler ultrasound with PGE1 stimulation is a relatively safe procedure which can help elicit a cause and direct clinician management appropriately.
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