Penile Doppler sonography in the investigation of erectile dysfunction

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Although there are many causes of erectile dysfunction, the physical aetiologies can be divided into arterial inflow problems, abnormalities of the venous occlusion mechanism and structural penile abnormalities. This review will briefly describe the anatomy and physiology of penile erection, the technique itself and illustrate the main abnormalities that may be encountered.

Analysis of the cavernosal artery flow dynamics using penile Doppler sonography (PDS) was first investigated by Lue et al in 1985. Its role has subsequently evolved, particularly since the introduction of oral PDE5 inhibitors, such as Sildenafil. A successful trial of these oral agents in effect confirms an adequate arterial inflow and a satisfactory venous occlusion mechanism, thereby precluding further investigation. As such, PDS is reserved for those patients with limited or equivocal response or those with complex problems that may require corrective surgery, such as a young patient with pelvic/perineal trauma.

In order to understand a penile Doppler study, a brief review of normal anatomy and erectile physiology is necessary. When viewed with standard transverse grey scale ultrasound the paired hypoechoic corpora cavernosa are visible, surrounded by the thick echogenic tunica albuginea (figure 1). The ventrally-located single corpora spongiosum (which is surrounded by a thinner layer of tunica albuginea) is seen to surround the penile urethra. The corpora cavernosa constitute multiple smooth muscle and endothelial-lined sinusoids which, with onset of erection, distend with blood and are capable of considerable volume expansion. Although penile arterial anatomy is variable, the penis is supplied by branches of the internal pudendal artery (itself a branch of the internal iliac artery). The penile artery (having given off the bulbular artery) divides into the dorsal artery and the cavernosal arteries. As their name suggests, the cavernosal arteries run centrally within the length of the corpora cavernosa and are easily identified by their parallel echogenic walls. These arteries supply the cavernosa via numerous helicine arteries and supply the sinusoids via arterioles. Communication of blood across the midline between cavernosa is permitted by perforations in the intercavernous septum. Emisary veins that pierce the tunica albuginea drain into the deep dorsal vein via the spongiosal, circumflex and cavernosal veins.

The resting smooth muscle tone of the cavernosal arterioles and sinusoids in the flaccid penis is high, with resultant low volume inflow and outflow. Erection begins with neurochemically driven relaxation of smooth muscles of sinusoids and cavernosal arterioles. This results in significantly increased cavernosal artery flow. This increased inflow engorges the cavernosal sinusoids, which distend and cause penile tumescence. The distending sinusoids will eventually compress the exiting venules and emissary veins, thereby restricting venous outflow. The resultant situation of high arterial inflow with restricted venous outflow produces a rapid increase in intracorporal pressure and penile erection.

A PDS study involves intrapenile injection of a pharma-costimulant with assessment of the cavernosal arteries and their temporal velocity and spectral waveform evolution. Although individual techniques vary, the fundamental principle is to ensure regular assessment of the waveforms, until predetermined haemodynamic parameters are met. In order to reduce catecholamine-induced increase in vascular tone and minimise anxiety, the procedure should be performed in a quiet environment. Informed consent should include a 1-2% risk of inducing priapism. The patient lies supine and 10-20mcg of PGE-1 is injected into one of the corpora cavernosa. Bilateral injection is unnecessary as diffusion occurs freely between the cavernosa. In order to reduce the chance of priapism, those who are treatment naive or if psychogenic impotence is felt likely, should be started with 5mcg, with further doses as required.

Once tumescence starts, the penis is assessed with standard grey scale ultrasound for non-vascular abnormalities such as Peyronie’s plaques, areas of fibrosis and tunica albuginea defects. Care should be taken not to misinterpret the tiny echogenic foci of injected air (a result of the cavernosal injection) as pathology. The authors’ preference is to scan the penis dorsally, transversally in the early stages and oblique-longitudinal upon tumescence. By angling the probe 20-30° cephalad, the artery in the root of the cavernosa can be studied. As flow in this region is towards the probe, the artery can be insonated at a Doppler angle of 0°. Angle correction should be corrected to the direction of the artery and kept less than 60°. Velocity measurements are most accurate and reproducible at the base of the penis. At the start of the study, the cavernosal arteries are typically difficult to visualise but become demonstrable after 1-3 minutes. At this point, image capture and measurement should be repeated every five minutes until maximal peak systolic velocity (PSV) and minimal diastolic velocity have been reached (sometimes up to 25 minutes post-injection). Both cavernosal arteries should be assessed and the quality of the erection recorded. The waveform progression is sequentially assessed, the first few typically showing elevating velocities as a consequence of the smooth muscle relaxation. From this point the PSV is followed. The study is complete once minimal diastolic velocity is reached. In addition to sequential waveform analysis, the entire length of the cavernosal arteries should be visualised to identify potential anomalies in the vascular supply.

A normal study (figure 2)

As described earlier, prior to stimulation the cavernosal arteries are difficult to appreciate. Following injection and prior to sinusoidal engorgement, the cavernosal sinusoids represent a low resistance bed allowing forward flow throughout the cardiac cycle with a high diastolic velocity. The PSV should continue to increase. As the sinusoids engorge and restrict venous outflow, the system becomes high resistance and forward flow is achieved only during the high-pressure systolic portion of the cardiac cycle/spectral waveform. Consequential changes in the spectral waveform and measurements can be identified, such as a diacrotic notch. As the intracavernosal pressure continues to rise, the shortened duration of systolic flow causes a narrowing of the systolic waveform. Diastolic flow should be reduced to close to zero. When intracavernosal pressure exceeds diastolic pressure, end-diastolic flow reversal is demonstrated. With maximal penile rigidity, the intracavernosal pressure may approach that of systolic pressure with a reduction in systolic velocity.
Although threshold values for normal systolic and diastolic velocities are debated, the authors use a PSV greater than 35 cm/sec and an EDV being either negative or near zero.

**Arterial insufficiency (figure 3)**

An exact threshold PSV value for definitively diagnosing arterial insufficiency as the cause of erectile dysfunction varies, with values between 25 and 35 cm/sec suggested by authors.1,3,5 By correlating measurements of PSV with selective angiographic findings, Benson et al concluded that a PSV less than 25 cm/sec was associated with a strong likelihood of severe arterial disease.14

**Venous incompetence (figure 4)**

In the normal subject, as the engorging sinusoids impede venous outflow, there is an elevation in intracavernosal pressure with consequent reduction in diastolic flow and end diastolic flow reversal. Elevated end diastolic flow values are therefore suggestive of venous incompetence (also called ‘venous leakage’). Again, exact threshold figures are debated, with some authors suggesting an EDV greater than 5 cm/sec to be highly suggestive.15,17 Others use 7 cm/sec as a diagnostic cut-off.19

It is worth mentioning that the specificity of EDV in diagnosing venous incompetence is low when associated with co-existing arterial insufficiency. In this situation, the more invasive dynamic infusion cavernosometry and cavernosography (DICC) is recommended due to its ability to quantify outflow resistance independent of arterial inflow.20 This is particularly true if surgery is contemplated.20

**Peyronie’s Disease (figure 5)**

First described in 1743 by the physician of France’s King Louis XVI, Peyronie’s disease is a relatively common benign connective tissue disease characterised by fibrous thickening of the tunica albuginea. On grey scale imaging, these plaques of fibrosis appear as echogenic irregular thickenings of the tunica often with associated calcification. Their identification may allow patient selection for lichtrofips.21

There is a strong association between erectile dysfunction and Peyronie’s.22,23 Although likely multifactorial, many have suggested vascular abnormalities to be the main aetiology.24-26 Identification of the underlying cause is important for planning future management.

**Priapism**

Priapism is a persistent penile erection that continues beyond, or is unrelated to, sexual stimulation.27 The more common ‘low-flow’ (ischaemic) priapism is a consequence of sinusoidal thrombosis and veno-occlusion and results in a painful, rigid erection. PDS demonstrates an absence of cavernosal flow with a high resistance, low velocity trace from the artery. Prompt treatment is necessary to prevent irreversible ischaemia and sinusoidal fibrosis.28 ‘High-flow’ (non-ischaemic) priapism is classically a consequence of penile/perineal trauma with the development of an arterio-cavernous fistula. The penis is classically only partly rigid and pain free. PDS can accurately visualise the fistulas, with sensitivity approaching 100%.29 The PSV is typically elevated with high diastolic flow and often prominence of the draining veins.30 Diagnostic distinction is essential as the treatment of the two conditions differs, with the low-flow group requiring prompt aspiration and the high-flow type requiring transcatheter embolisation.

**References**

**FIGURE 1**
Normal grey-scale images of the penis.
A. Transverse. Note the echogenic tunica albuginea (long arrow) surrounding the paired corpora cavernosa and single corpus spongiosum.
B. Longitudinal. Note the parallel echogenic walls of the cavernosal artery (long closed arrow). A gradient of cavernosal distension can be observed, with those sinusoids closest to the artery (short closed arrow) distending first and thereby having a more hypoechoic appearance.

**FIGURE 2**
A normal penile Doppler study.
A. A few minutes after injection, as tumescence begins; note the elevating systolic and diastolic velocities
B. After approximately 10 minutes post-stimulation, with increasing intracavernosal pressure there is a reduction in diastolic flow.
C. PSV has peaked above 50cm/sec, indicating normal arterial inflow. There is end-diastolic flow reversal, indicating an intact venous occlusion mechanism. Later in the same study a reduction in systolic velocity may occur (not shown).

**FIGURE 3**
Arterial insufficiency
A–D. Images taken at approximately five minute intervals after an injection of full dose of Caverject. Diastolic flow is positive, indicating pharmacological response, but the PSV is <10cm/sec throughout the study indicating severe arterial insufficiency. No significant penile erection was obtained.

**FIGURE 4**
A. Thick echogenic calcified Peyronie’s plaque (open arrow). Note the posterior acoustic shadowing. Distortion of the underlying cavernosal arteries may be observed.
B. Significant cavernosal fibrosis (closed arrows) as a sequelae of low-flow priapism. Note the echogenic, distorted cavernosa.