Penile Veins are the Principal Component in Erectile Rigidity: A Study of Penile Venous Stripping on Defrosted Human Cadavers

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Running title: Penile veins are the chief determinant of erectile rigidity

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The human erectile mechanism is an intricate interplay of hormonal, vascular, neurological, sinusoidal, pharmacological, and psychological factors. However, the relative influence of each respective component remains somewhat unclear, and merits further study. We investigated the role of venous outflow in an attempt to isolate the key determinant of erectile function. Dynamic infusion cavernosometry and cavernosography was conducted on fifteen defrosted human cadavers, both before and after the systematic removal and ligation of erection-related penile veins. Preoperatively, an infusion rate of >28.1 mL/min (from >14.0 - 85.0) was required to induce a rigid erection (defined as intracavernosal pressure [ICP] exceeding 90 mmHg). Following surgery, we were able to obtain the same result at a rate of 7.3 mL/min (from 3.1 - 13.5) across the entire sample. Thus, we witnessed statistically significant postoperative differences (all p ≤ 0.01), consistently remarking increased ICP, lower perfusion volumes, and a general reduction in time taken to attain rigidity. The cavernograms provided further evidence substantiating the critical role played by erection-related veins, while histological samples confirmed the postoperative integrity of the corpora cavernosa. Given that our use of cadavers eliminated the influence of hormonal, arterial, neurological, sinusoidal, pharmacological, and psychological factors, we believe that our study demonstrates that the human erection is fundamentally a mechanical event contingent on venous competence.
Introduction

The anatomy of the penis is well-established (Aboseif et al, 1989; Kirby, 1999; Moscovici et al, 1999; Fuchs et al, 1989; Goldstein et al, 1990; Gray, 1989; Lue, 2007; Putz et al, 2001). In recent studies (Hsu et al, 1992; Hsu et al, 2003; Hsu et al, 2005) we have furthered this knowledge with the discovery of a bilayered tunica albuginea, the human distal ligament, and a much more elaborate venous drainage system.

It is also well documented that erectile function is governed by a number of interdependent components; hormonal, vascular, neurological, sinusoidal, pharmacological, and psychological (Karacan et al, 1983; Wagner et al, 1982; Andersson and Wagner, 1995; Giuliani et al, 1995; Rajfer et al, 1992; Burnett et al, 1992). These factors may be further compounded by the adverse effects of drugs and by chronic systemic diseases (Christian et al, 2010; Kaminetsky, 2008). The landmark 1998 introduction of phosphodiesterase-5 (PDE-5) inhibitors — now firmly established as the frontline therapy of choice for treating erectile dysfunction (ED) (Anonymous, 1998; Ballard et al, 1998; Nachtsheim, 1998; Gresser and Gleiter, 2002) — brought with it further insights into penile smooth muscle function (Dean and Lue, 2005; Saenz et al, 2004; Burnett, 2002; Williams et al, 2005; Andersson, 2001 & 2011; Rodriguez et al, 1998; Mas, 2011; Melman, 2008; Uckert et al, 2004; Toda et al 2005; Cartledge et al, 2001). Accordingly, fibrosis of the cavernosal sinus smooth muscle was identified as a major contributor to ED (Nehra et al, 1996; Gonzalez, 2009; Kovanecz et al, 2009). Although this was a major breakthrough in our understanding of ED, our knowledge of penile anatomy has remained comparatively static (Eardley and Sethia, 2003).

During an episode in the Second World War, a critically injured soldier exhibited
a rigid post-mortem erection following the administration of an intracavernous blood transfusion at 25 mL/min (Newman et al, 1964). This incident implies that the human erection could prove to be, at its core, a mechanical matter. A recent hemodynamic study on fresh cadavers demonstrated that, following the ligation of erection-related veins adjacent to the tunica albuginea, a rigid erection can be elicited at a perfusion rate of 150 mL/min with normal saline (Hsieh et al, 2005). One should bear two caveats in mind, however: the saline perfusion rate was well in excess of the physiological limit, and the cells lining the sinusoids may have plausibly remained physiologically active six hours after the subject’s death. Despite these shortcomings, the study remains testament to the fact that erectile rigidity relies upon the corpora cavernosa functioning as an enclosed environment, and thus adheres to Pascal’s Law (Halliday, 1997): erection results when pressure is distributed equally to all walls of the corpora cavernosa — provided that there is no extraneous leakage (Hsu et al, 2004, Hsu et al 2006). In our study we elected to explore this principle further, hoping to somewhat demystify the erectile process.

Materials and Methods

Following its approval by the institutional review board of China Medical University, we carried out our study over the course of a thirty-one-month period beginning in May 2009. We operated on fifteen male cadavers that had been frozen within ten hours of death and preserved for three months without formalin fixation. These were thawed within a controlled defrost environment over three days. We excluded two cadavers due to excessive postmortem changes and employed a final pair for equipment set-up and
calibration.

In order to facilitate venous access, all cadavers underwent an initial circumferential incision. This was followed by a dorsal median longitudinal incision (Figure 1), starting at the retrocoronal sulcus and extending along the penile shaft to reach the upper margin of the symphysis pubis. Two #19 scalp needles were inserted in the corpora cavernosa at the 3 and 9 o’clock mid-shaft positions and affixed in place with 4-0 silk sutures. One needle was connected to an infusion pump and subsequently used to inject a 10% colloid solution into the corpora cavernosa. The other was used to monitor the intracavernosal pressure (ICP) via a standard IV set connected to a negative feedback pressure monitoring system. We then performed baseline infusion cavernosometry at a rate of 35 mL/min. Measures of the ICP, the time required to reach an ICP >100mmHg, the perfusion volume, and the maintenance flow at an ICP of 90 mmHg were all recorded. A solution of 60 mL Iohexol in a 60 mL syringe was switched to the infusion line for cavernosograms as required.

During the operation, the DDV (Figure 2) was stripped and ligated distally with 5-0 nylon to the level of the retrocoronal sulcus, and laterally as close as possible to the junction between the corpus spongiosum and corpora cavernosa. It was necessary to use two 8.5 × 1.6 cm right-angle retractors to access vessels close to the infrapubic angle (located at depths of roughly 7cm).

The CVs were then subjected to stripping and ligation, followed subsequently by the PAVs. Finally, the penile artery was ligated proximal to the penile hilum. Infusion cavernosometry (Figure 3) was performed at each major stage. Accordingly, five sets of cavernosometric data were generated for analysis.
Samples from cadavers ten and eleven were removed during biopsy and stained with hematoxylin and Masson trichrome for histological examination. Data analysis was performed using the Wilcoxon signed-ranks test, with a \( p \) value of < 0.05 defined as representing a significant difference.

**Results**

In order to offset any potential issues of unreliability relating to the early calibration of our equipment, we elected to exclude the cavernosometric data obtained from the initial two cadavers. Additionally, we disqualified another pair from the study due to postmortem changes beyond our control. Demographic data from the eleven cadavers used in this study are summarized in Table 1. Infusion rates ranged from 14.0 to 85.0 mL/min in Table 2. At each incremental stage of the venous stripping procedure, we recorded statistically significant differences in ICP (124.87 vs. 233.19 vs. 323.93 mmHg \( p = 0.009 \)), time to base pressure (219" [3'39"] vs. 181" [3'1"] vs. 171" [2'51"] s \( p = 0.009 \)), and injection volumes (110 vs. 80 vs. 70 mL \( p = 0.009 \)).

Preoperatively, an infusion rate of 35 mL/min proved adequate in eliciting a rigid erection in a majority of cadavers (from \( >14 - 85 \) mL/min, \( p = 0.009 \)). Following the ligation of all three venous systems — DDV, CVs, and PAVs — there was a marked reduction in the rate required to maintain an ICP of 90mmHg (\( >21.9 \) vs. 8.3, \( p = 0.009 \)).

We noted that one particular subject, who had committed suicide by hanging, proved unusually responsive during cavernosometry (Figure 4). Regrettably, the pressure-monitoring line became disconnected from two cadavers (nos. 5 and 6) when the ICP rose to 702.59 mmHg and 557.48 mmHg, respectively.
The cavernosogram of cadaver ten (Figures 5A, 5B, and 5C) shows a fully rigid erection; the ICP having risen smoothly to 150mmHg (Figure 5). This is a contrast with that of cadaver eleven, which exhibited greater postmortem degradation (Figures 5D, 5E, and 5F). This disparity was further accentuated by the condition of their sinusoids, with those of cadaver ten (Figures 6A, 6B, 6C, and 6D) exhibiting markedly fewer hyaline changes than those of cadaver eleven (Figures 6E, 6F, 6G, and 6H).

It may be interesting to note that, for three subjects, arterial ligation prompted a consistent elevation in ICP (39.42 vs. 102.08 mmHg, 38.73 vs. 89.74, and 26.29 vs. 71.73). However, it would be difficult to draw any kind of definitive conclusion from such a limited sample.

Discussion

It was our aim to combat previous criticisms of this type of study by employing cadavers that had been frozen for three months. However, while this would guarantee the inactivity of penile cavernosal smooth muscle cells, the potential rupturing of capillaries and smaller venules due to volume expansion remained a concern. Ultimately, this did not prove to be a problem, as the hemodynamic responses of the defrosted cadavers proved similar to those of their fresh counterparts (Hsieh et al, 2005). We might then confidently infer that the vascular sinusoidal smooth muscle cell system was not compromised by the freezing process, which supports the proposal that the sinusoids are dual venous systems within the corpora cavernosa (Banyan et al, 1989). We believe that our study is novel and will contribute to existing research concerning the hemodynamic response of the human penis (Reiss et al. 1982; Mark et al, 1999; Prieto, 2008; Ghalayini, 2004; Komori et al,
In place of the non-viscous saline solution we injected at 150 mL/min during our previous study, we injected a 10% colloid solution (possessing the same viscosity as blood) at a lesser rate of 35 mL/min. This arterial perfusion rate, far lower than would be typical of a naturally-occurring erection, was chosen to address certain criticisms leveled at the abnormally high rate used in our previous study. Unfortunately, the ICP monitoring equipment uncoupled unexpectedly from cadavers five and six due to excessively high pressures. Although these incidents precluded further recording, inferential comparison could still be made from the data accrued prior to the disconnection of the pressure lines. The tolerance varied between monitoring lines, with initial detachment occurring at respective pressures of 702.59 (cadaver no. 5) and 557.58 (cadaver no. 6) mmHg, and recurrence at 321.90 and 467.67 mmHg following reconnection. As we encountered no resultant signs of corporal rupturing, this would appear to be consistent with a study on Caucasian cadavers which reported that the corpora cavernosa could withstand pressures of up to 1370 mmHg (Bitsch et al, 1990). Of course, it remains a matter of conjecture whether the corpora cavernosa would have tolerated pressures approaching this theoretical upper-limit had the tubes not disconnected.

While performing cavernosometry on the fifth cadaver — a body exhibiting severe hyper-pigmentation as a result of death by hanging — we were surprised to observe an unusually rapid rise in ICP (226.73 mmHg in 85 seconds). A volume of 50 mL at the standard infusion rate of 35 mL/min was sufficient to generate this extremely high pressure level. Moreover, the maintenance flow necessary for sustaining this subject’s ICP at 90 mmHg proved strikingly low at 4.7 mL/min, and was reduced further by
venous removal and ligation (settling finally at 3.3 mL/min). This phenomenon is hard to reconcile with our existing understanding of the erectile mechanism, and would justify further scientific study.

Although we acknowledge that a retrospective chart review of each cadaver does not necessarily provide an accurate sexual history, we found that it constituted a useful guideline. As cadaver one’s record indicated potency, we decided to proceed with the standard infusion rate (35 mL/min), which proved adequate. We later discovered that this conservative rate also sufficed for cadavers with a history of impotence, inducing rigid erections in cadavers six, seven, and eight, who had suffered ED for 3, 4, and 5 years, respectively. Although the accuracy of each cadaver’s history may be questionable we found that irrespective of potency history, we ultimately found that venous ligation and removal dramatically improved erection quality with the inference that arterial inflow is less of a factor in erectile rigidity than originally thought. It is also worth remarking that, although the subjects died from different causes, our study benefited from the fact that half of the cadavers died from cancer and the other half did not. This effectively balanced out the risk of a chronic disease process influencing the results.

We then experimented with lower induction flows and arterial ligation in order to further scrutinize the significance of the arteries in the erectile process. Our attempts with lower infusion rates did not prove successful in inducing rigid erections, although any conclusions we might draw from this are limited by the small sample size. Within our study, the artery served no supply function and could theoretically have been a source of leakage. Arterial ligation was performed on cadavers 2, 3, and 4 in order to verify and demonstrate the vessel’s relatively negligible contribution to outflow.
We realize that, to the casual observer, our results might seem to constitute a challenge to the current consensus on venous surgery for erectile dysfunction, implying that venous removal should function as a straight-forward cure. It is therefore important to emphasize that our chief observation is simply the following: a rigid erection is a hemodynamic phenomenon to which Pascal’s law could be applied. Medical literature already attests to the predominance of venous incompetence as a contributor to erectile dysfunction (Rajfer et al, 1988; Melman and Gingell, 1999), even attributing it to be the most significant factor in those with ED originally thought to be caused by arterial insufficiency (Elhanbly et al 2004). While this study provides further insight into the significance of venous drainage, we must, however, proceed with caution: it is apparent that additional research is warranted.

**Conclusion**

Although we encountered a number of theoretical and practical challenges, our work with non-formalin-fixed cadavers allowed us to negate the hormonal, arterial, neurological, sinusoidal, pharmacological, and psychological factors that otherwise influence the erectile process. Following the removal and ligation of erection-related veins (the DDV, CVs, and PAVs), erectile rigidity becomes a straightforward matter of Pascal’s Principle of uniform pressure distribution in an enclosed space (Hsu et al, 2006; Hsu et al, 2007; Hsieh et al, 2010). We believe that our study indicates that the human erection is fundamentally a mechanical event contingent on venous competence.

**Acknowledgements:**
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15


Williams & Wilkins, 2001:222-239.


**Legends:**

**Figure 1:** Schematic illustration and photographs of a representative cadaver in this hemodynamic study. **A.** Schematic illustration of the surgical approach depicting the placement of scalp needles in the corpora cavernosa. A circumferential incision was made followed by a dorsal median longitudinal incision reaching the upper margin of the symphysis pubis. Two #19 scalp needles were inserted and firmly fixed in place with 4-0 silk sutures at the 3 and 9 o’clock positions. One needle was connected to an infusion pump, while the other was used to monitor the intra-cavernous pressure. **B.** Photograph illustrating the preparation of the penis (as depicted in Panel A) prior to infusion cavernosometry. **C.** Photograph showing a rigid erection with an ICP of 165.00 mmHg, induced with 10% colloid solution at an infusion rate of 35 mL/min.

**Figure 2:** Lateral aspect of the template used as a reference during venous surgery. Note the positions of the ligatures following the stripping procedure. The second, third, and fourth infusion cavernosometric procedures were performed immediately after the stripping of the deep dorsal vein (DDV), cavernosal veins (CVs), and para-arterial veins (PAVs), respectively. The sites of these ligatures should be as close to the tunica
Although minor accidents involving the arterial, nervous, and lymphatic tissues would be unlikely to damage the experiment, any rupture of the tunica albuginea would ruin the watertight milieu for applying Pascal’s law.

**Figure 3:** Cavernosometric data from cadaver no. 6 in Table 2. The upper tracing charts the intra-cavernous pressure (ICP) in mmHg, while the lower tracing displays the infusion flow of a 10% colloid solution at 35 mL/min. 

A. Calibration of the equipment. B. The first infusion cavernosometry (performed prior to the stripping of the deep dorsal vein [DDV]). The ICP reached 179 mmHg, representing an increment of 148.16 (179.00 - 40.84) mmHg over 185 s. C. Infusion cavernosometry performed after the removal and ligation of the DDV. The ICP reached 363.82 (385.42 - 21.59) mmHg in 147 s. D. Infusion cavernosometry performed following the ligation and removal of the cavernosal veins (CVs). The ICP rose to 558.00 (582.52 - 24.52) mmHg in 107 s. E. The fourth and final infusion cavernosometry (performed after the segmental ligation of the para-arterial veins [PAVs]). The ICP rose to 494.82 mmHg, at which point a rupture in the pressure monitoring system prevented further recording.

**Figure 4:** Cavernosometric data from cadaver no. 5 in Table 2. The upper tracing charts the intra-cavernous pressure (ICP) in mmHg, while the lower tracing shows the infusion flow of a 10% colloid solution at 35 mL/min. 

A. The first infusion cavernosometry (performed prior to the stripping of the deep dorsal vein [DDV]). ICP reached 179 mmHg, amounting to an increment of 226.73 (220.83 - [-5.90]) mmHg over 85 s. Note that the ICP returned to the baseline with marked difficulty. B. Infusion cavernosometry
following removal and ligation of the DDV. The increase in ICP was 531.37 (535.98 - 4.61) mmHg in 80 s. C. The third infusion cavernosometry (performed following the stripping and ligation of the cavernosal veins [CVs]). The ICP increment was 702.60 (708.60 - 6.00) mmHg over 70 s. D. The fourth infusion cavernosometry (following the segmental ligation of the para-arterial veins [PAVs]). An ICP of 360.90 (that is, an increment of 321.89) mmHg was achieved, at which point the pressure monitoring equipment uncoupled itself. The corpora cavernosa were palpated (see arrow) in an attempt to ascertain the cause of the detachment.

Figure 5: Comparison of cavernosograms. A. Film of cadaver ten prior to the removal of erection-related veins. The corpora cavernosa (white asterisk) were filled with a contrast medium. B. Further injection of the contrast solution induced an ICP of 70 mmHg. Note the sinusoids of the corpora cavernosa (white asterisk), which exhibit significant distension. C. Following the removal of erection-related veins, an ICP of 150mmHg was generated, resulting in full distension of the corpora cavernosa (black asterisk). Panels D, E, and F were obtained from cadaver eleven and rendered for comparison with those of no. 10. Note that, as the ICP failed to rise beyond 65mmHg, the penis never assumed an upright position.

Figure 6: Histological evidence. A. Tissue specimen collected from cadaver ten. The tunica albuginea (bottom) is intact, along with the sinusoids (curved arrow), in which the spindle-shaped smooth muscle cells (red color) are intertwined with green-colored collagen. (Reduced from ×40, Masson trichrome stain). B. Magnification of the sinusoid
(curved arrow). (Reduced from ×100, Masson trichrome stain). C. Further magnification (black asterisk). (Reduced from ×200, Masson trichrome stain). D. A clear image of the sinusoidal walls. (Reduced from ×400, Masson trichrome stain). Panels E, F, G and H were obtained from cadaver eleven and show hyaline changes consistent with advanced postmortem degradation.

Tables

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Table 2 - Cavernosometry of 11 defrosted male human cadavers in this study

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<th>Erection Flow rate(^a) (mL/min)</th>
<th>ICP (Intracavernosal Pressure)(^b) (mmHg), Time (min), and Volume (mLs)</th>
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\(a\) Flow rate required to maintain ICP at 90mmHg
\(b\) Intracavernosal Pressure
\(c\) DDV = Dorsal Denervation
\(d\) CV = Cavernosal
\(e\) PAV = Penile Animation

Note: "x" indicates data not available.
The Wilcoxon signed ranks test was used for analysis with significance level at $p < 0.05$.

- **a** The flow rate, in mL per minute, necessary for keeping the ICP at 90mmHg, with a comparison between baseline and end postoperative maintenance rates.
- **b** The ICP (intra-cavernous pressure) increment, which was recorded either maximally or until the recording line became disconnected as a consequence of excessive pressure.
- **c** The DDV, CVs and PAVs are abbreviations for the deep dorsal vein, cavernosal veins and para-arterial veins, respectively.
- **d** A stands for arterial ligation; D stands for “disconnection” of a pressure monitoring line.
- **e** The infusion rate had been escalated from 35.0, 50.0, 70.0, 80.0 mL/min (3 min for each) and then 85.0 mL/min, which eventually proved to be the reliable rate.
- **f** NA is an abbreviation for not applicable.
- **g** The inferential statistics were made at rupture points for subjects 5 and 6 to their corresponding parameters in the preceding column.
- **h** The inferential statistics were calculated among the middle 7 cadavers at the rupture points in cadavers 5 and 6 to their corresponding parameters in the preceding column, thus cadavers 1, 9, 10 and 11 were excluded and the last two were rendered for comparison.

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