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ABSTRACT

To refine the diagnostic method for opacification of aberrant venous drainage in venogenic impotence, an experimental study was done in eight monkeys. In all monkeys, cavernosography after induction of erection by saline perfusion showed significant drainage via the cavernous veins. However, when cavernosography was performed after neurostimulation or papaverine injection, no cavernous drainage was visualized, even when the intracavernous pressure had been significantly lowered by creation of an artificial cavernous leak.

Because erection can result from saline perfusion only when the volume perfused exceeds the venous outflow capability, cavernosography during saline-induced erection will always demonstrate the entire venous system and, thus, is of no diagnostic value. Pharmacocavernosography imitates the physiologic venous occlusive mechanism and should therefore be used to identify the abnormally draining veins in venogenic erectile dysfunction. (J. Urol., 140: 1564-1566, 1988)

The first attempts at surgical treatment of erectile dysfunction were described in the early 20th century. In 1902, Wooten suggested ligation of the dorsal vein of the penis to treat "atonic impotence," with insufficient rigidity for intercourse. In 1936, Lowsley and Bray described a procedure to reduce the dorsal penile venous outflow by tightening the suspensory ligament. However, because of these early authors' lack of knowledge of the physiology of erection and the absence of objective diagnostic criteria, these reports retain only anecdotal meaningfulness.

The significance of venous restriction for erection was observed first by Newman et al. in 1964. Virag introduced the technique of inducing erection by saline perfusion in 1978. He called the quantification of venous outflow and the opacification of the abnormally draining veins in artificial erection "dynamic cavernosography." In 1986, Lue suggested that this method of diagnosing venogenic impotence be done after intracavernous injection of vasoactive drugs.

The diagnosis of cavernous vein leakage is aided by cavernosometry. When the suspicion is confirmed, cavernosography should be done to identify the abnormally draining veins. We undertook the present study to determine whether dynamic or pharmacologic cavernosography will give more useful results. Monkeys were used for this radiographic study because the histologic characteristics of the simian penis and the sequence of simian erection are similar to the human.

MATERIALS AND METHODS

In eight pigtail monkeys, weighing 5.5 to 14.5 kg., a 21-gauge needle was inserted into the distal right corpus cavernosum and connected to a Harvard perfusion pump (Mod. 903). Proximal to this needle, a 19-gauge needle connected to a stop cock was inserted to create a cavernous leak. A 21-gauge needle was inserted into the right corpus cavernosum and connected to a Statham transducer (Mod. P 23 AC) for pressure recording (Grass polygraph Mod. 7). In a previous surgical procedure, a cuff electrode (Avery Lab) had been placed around the cavernous nerve, with the receiver placed subcutaneously in the lower left abdominal quadrant. Cavernous nerve stimulation (2-5 V, 20 Hz) was used to establish the intracavernous pressure level of a full erection (fig. 1).

Our study was divided into three parts. First, saline alone was perfused to raise the intracavernous pressure to the level established by cavernous nerve stimulation, and undiluted contrast medium (diatrizoate meglumine 28 percent) was injected slowly to prevent a pressure rise of more than 20 cm. H2O. Thirty seconds later, an x-ray was taken to visualize the corpus cavernosum and its venous drainage. Five minutes later, erection was again induced by saline perfusion. The stop-cock was then opened to initiate cavernous leakage and the contrast medium was injected after the intracavernous pressure had dropped and plateaued. An x-ray was taken after 30 seconds. Five minutes later, erection was reestablished by saline perfusion alone and the stop-cock was opened again. The perfusion rate was increased to raise the intracavernous pressure to the level of cavernous-nerve-stimulated erection. Contrast medium was injected, while the pressure was continuously maintained, and an x-ray was taken after 30 seconds.

This same protocol (x-rays after contrast medium perfusion in full erection, in erection with cavernous leakage, and in erection with leakage and increased saline perfusion to maintain intracavernous pressure) was repeated after erection induced by cavernous nerve stimulation and performed a third time after injection of 10 mg. papaverine. A 25-minute interval was allowed between each experiment.
RESULTS
The mean intracavernous pressure of the flaccid monkey penis was 34.3 cm. H$_2$O (24 to 42 cm. H$_2$O). In six of the eight monkeys, cavernous nerve stimulation induced full erection, with a mean intracavernous pressure rise of 114 cm. H$_2$O (95 to 140 cm. H$_2$O) above baseline; in the remaining two, the mean pressure increase was only 42 cm. H$_2$O. Creation of the venous leak resulted in a mean pressure drop of 67 cm. H$_2$O (41 to 80 cm. H$_2$O). In each individual monkey, the pressure drop was similar, regardless of the mode of induction of erection (saline perfusion, neurostimulation or papaverine).

In the first experiment, a mean saline perfusion rate of 26.7 ml./min. (15.3 to 34.4 ml./min.) was necessary to raise the intracavernous pressure to a level identical with that of the neurostimulation-induced erection established in the six monkeys. In the two monkeys in which cavernous nerve stimulation did not induce full erection, an intracavernous pressure level of 150 cm. H$_2$O was obtained by saline perfusion. In all eight monkeys, this mean intracavernous pressure of 148 cm. H$_2$O produced only medium penile tumescence. X-ray films in all eight showed a huge amount of contrast medium running via the cavernous veins, regardless of when the film was obtained (at full erection pressure, at reduced intracavernous pressure with opened stop-cock, or after reestablishment of full erection pressure by increased perfusion) (fig. 2).

After a pause of 25 minutes, we then induced erection by cavernous nerve stimulation. The same level of intracavernous pressure was reached in the six responders as with the previous neurostimulation. In contrast to the saline perfusion experiment, this pressure level, when provoked by neurostimulation, was concurrent with full erection. No cavernous veins were visible in any of the x-ray films, regardless of when they were obtained (fig. 3). However, in the two monkeys with an incomplete response to cavernous nerve stimulation, the cavernous veins were visualized.

DISCUSSION
As in man, in the monkey the corpora cavernosa function as one unit. Unlike man's, the monkey's corpora are drained only by the cavernous veins. The monkey has no emissary veins of the penile shaft draining into the deep dorsal or superficial veins of the penis.

Our cavernosographic results show that, when erection is caused by saline perfusion alone, the venous channels are not occluded as various authors have assumed. Rather, the draining capability of the cavernous bodies has merely been surpassed, as evidenced by the massive outflow from the cavernous veins to the pudendal veins on the x-ray images. In normal men, however, dynamic cavernosography shows only slight cavernosal drainage or none. We think that this is due to the much larger volume of the human penis compared with the simian penis. During dynamic cavernosography in patients, the x-rays are usually taken relatively soon after contrast medium injection. At that time, cavernous veins are not visualized because the contrast medium is filling the cavernous bodies and nonopaque saline runs via the cavernous veins. In our experience, dynamic cavernosography in patients without venous leakage will show penile vein opacification after some minutes.
When full erection was induced in these studies by cavernous nerve stimulation or papaverine, no cavernous vein visualization was apparent, even after two minutes. To prevent a rise in the intracavernous pressure of more than 20 cm. H₂O, perfusion rates of contrast medium were held to less than 0.5 ml./min., compared with the mean flow rate of 26.7 ml./min. necessary to induce full erection by saline perfusion. This reflects a dramatically decreased cavernous outflow after neurostimulation or papaverine, which caused the contrast medium to be heavily diluted and prevented visualization. This greatly decreased cavernous outflow is also found in man after papaverine injection. A comparison of dynamic cavernosometry with pharmacocavernosometry also shows a decrease of the maintenance flow of about a factor of 5 to 6.\textsuperscript{10,11}

When cavernous vein leakage was initiated during full erection induced by neurostimulation or papaverine, opacification was still not apparent, even when the opening of the stop-cock resulted in a significant intracavernous pressure drop and loss of tumescence. The non-opacification of the cavernous veins as a result of closed venous channels cannot be explained after this pressure drop merely by the passive squeezing of the emissary veins within the tunica albuginea\textsuperscript{12} or by a passive compression of venous lacunae between the cavernous sinusoids and the tunica albuginea.\textsuperscript{13} Our findings suggest an active venous occlusion mechanism which further histologic and electron microscopic studies are needed to identify.

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