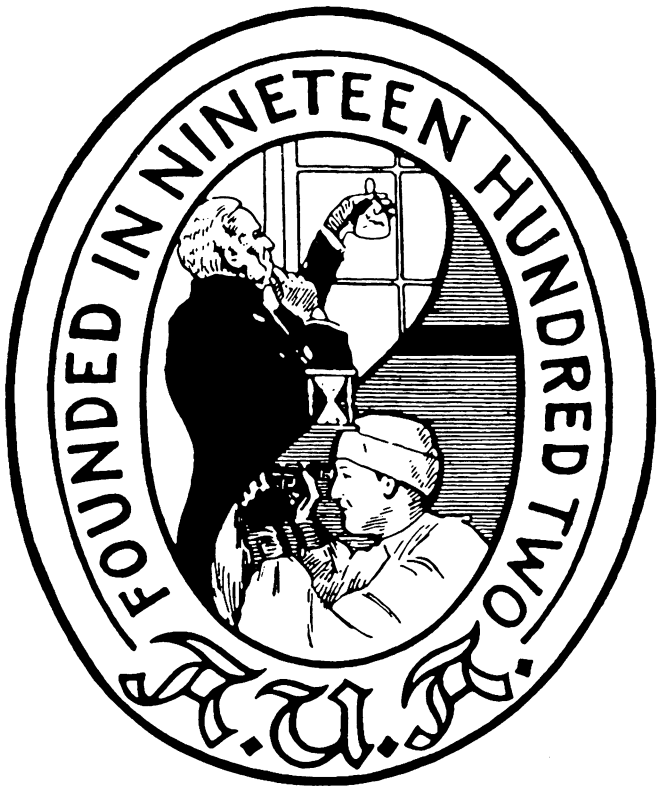
Volume 148 September 1992 Number 3

The Journal of UROLOGY®

Official Journal of the American Urological Association, Inc.
Founded In 1917 By Hugh Hampton Young



Annual Meeting, American Urological Association, Inc., San Antonio, Texas, May 15–20, 1993

The Journal of
UROLOGY®

Volume 148 September 1992 Number 3

CLINICAL UROLOGY

Contents continued on page A9

Contents continued from page A6

Case Reports

Special Communication

Letters to the Editor

Errata

Retraction

INVESTIGATIVE UROLOGY

Contents continued on page A16

Contents continued from page A 9

UROLOGICAL SURVEY

PROPRIETARY NAMES

Many of the words appearing in the JOURNAL OF UROLOGY are proprietary names even though no reference to this fact is made in the text. The appearance of any name without designation as proprietary is, therefore, not to be regarded as a representation by the editorial committee or publisher that it is not the subject of proprietary rights.

GUIDELINES FOR SUPPLEMENTS

The Editors of the Journal will consider requests for and solicit sponsors of Supplements to The Journal of Urology. For those interested the following are the proposed steps for publication of a Supplement.

1) Proposal formulated by sponsors of the Supplement or solicited by the Journal Editorial Staff that would include an indication of the major topics with a limited outline of subtopics, the nature of the articles to be included (review, original papers with or without discussion), identification of a sponsoring group or individual, identification of the type of internal quality control group available for the Journal Editors to work with and an indication of the financial support available.

2) Response by the Editorial Staff with identification of a specific individual to work with the Editorial Committee of the sponsors.

3) Agree to a deadline for submission of the papers, number of papers and so forth.

4) Formulation of and agreement on procedure for initial screening and editorial evaluation of manuscripts and discussions with active participation by the sponsoring group.

5) Submission to the Editorial Staff of the Journal for their evaluation.

6) Interaction between Journal and sponsoring editorial group. Final decision is retained by Journal Editorial Staff.

7) Redaction, page proofing and so forth by Editorial Staff. Articles in supplements would be indexed in the Journal.

8) Publication.

DISCLAIMER

0022-5347/92/1483-0878$03.00/0
THE JOURNAL OF UROLOGY
Vol. 148, 878-879, September 1992
Printed in U.S.A.

A NEW THERAPEUTIC CONCEPT FOR LONG-LASTING IATROGENIC PRIAPISM: A CASE REPORT

H. FREYMANN, M. DJAMILIAN, W. F. THON, C. G. STIEF AND U. JONAS

From the Department of Urology, Medical School Hannover, Hannover, Germany

ABSTRACT

In a therapeutic attempt on an 18-year-old patient with iatrogenic priapism lasting for more than 2 weeks after internal urethrotomy, intracavernous lysis was commenced with 80,000 IU streptokinase per hour. Following a dosage of 300,000 IU streptokinase the lysis was stopped because of severe bleeding from the urethrotomy scar. At 4 weeks after the patient was discharged from the hospital he reported normal erections and intercourse, while single potential analysis of cavernous electrical activity and ultrasound returned to normal. Provided there are no contraindications, intracavernous lysis seems to be an effective treatment for long-lasting priapism induced by intracavernous thrombosis.

KEY WORDS: priapism, streptokinase, penile erection, thrombosis

Currently, priapism is mostly seen after intracavernous injections of vasoactive drugs in the diagnosis and therapy of male erectile dysfunction.[1] Various other reasons for this syndrome are known, including primary priapism (idiopathic or after prolonged sexual excitement) and secondary priapism (hematological diseases, neurological dysfunctions, local disorders and pharmacological agents).[2] Postoperative priapism is a rare but severe complication of various urological and other iatrogenic manipulations.[3] The diagnosis is made by clinical evaluation. Differentiation between high flow and low flow priapism can be made by Doppler sonography. The nervous dysfunction is demonstrated by single potential analysis of cavernous electrical activity with electrical anodes applied to the penis, and registered and analyzed by a neurophysiological measuring instrument.

Early intervention is of prime importance in the treatment of all forms of priapism. Therapeutic trials performed after 24 hours are mostly of limited success because of thrombosis and fibrosis of the corpora cavernosa.[4] The initial therapy administered is aspiration of intracavernous blood and/or intracavernous injection of an α-adrenoreceptor agonist, such as metaraminol (2 to 4 mg.).[5,6] In case of failure, a shunt procedure[7-9] is done, or in the case of arterial high inflow priapism selective temporary embolization of the pudendal arteries with autologous clots may follow.[10] In regard to long-term results, many patients with priapism are permanently impotent. In this case the only therapeutic option is placement of a semirigid or malleable penile prosthesis, which imposes difficulties in the operative management due to cavernous fibrosis.[11]

CASE REPORT

Iatrogenic priapism occurred in an 18-year-old patient after internal urethrotomy for urethral stricture due to catheterization during a cardiac operation 13 years ago. At 2 weeks following urethrotomy the patient was referred to our hospital for priapism, which had begun 14 days previously. At clinical evaluation the penis was still erect but painless. On palpation it was not entirely rigid but the consistency of the corporeal bodies was dramatically increased even though the glans was soft.

Doppler ultrasound of the penile arteries showed strong cavernous artery pulses bilaterally but decreased inflow velocity. Ultrasound showed increased density of the cavernous tissue (fig. 1) and single potential analysis of cavernous electrical activity revealed significantly decreased cavernous electrical activity.[12] Two attempts involving aspiration of the cavernous blood (2 times 200 ml.), which appeared glutinous and cyanotic, followed by intracavernous injection of 0.4 mg. phenylephrine and then 4 mg. metaraminol induced no detumescence.

Since cavernous thrombosis could be assumed by ultrasound, single potential analysis of cavernous electrical activity and clinical evaluation, intracavernous lysis with streptokinase was attempted. Lysis was begun with 80,000 IU per hour streptokinase perfused intracavernously via 2 butterfly cannulas bilaterally under cardiovascular monitoring to attain the maximal dosage of 500,000 IU streptokinase. A dosage of only 300,000 IU streptokinase could be injected due to severe bleeding from the urethrotomy scar. There was a marked decrease in rigidity while under therapy.

Single potential analysis of cavernous electrical activity 7 days later showed improved cavernous activity and ultrasound revealed clear diminution of intracavernous hyperechogenicity (fig. 2). At 4 weeks after he was discharged from the hospital the patient reported normal erections and intercourse. Clinical evaluation revealed the corpora cavernosa to be soft. No deviation or fibrosis was palpable, and single potential analysis of cavernous electrical activity and ultrasound findings returned to normal.

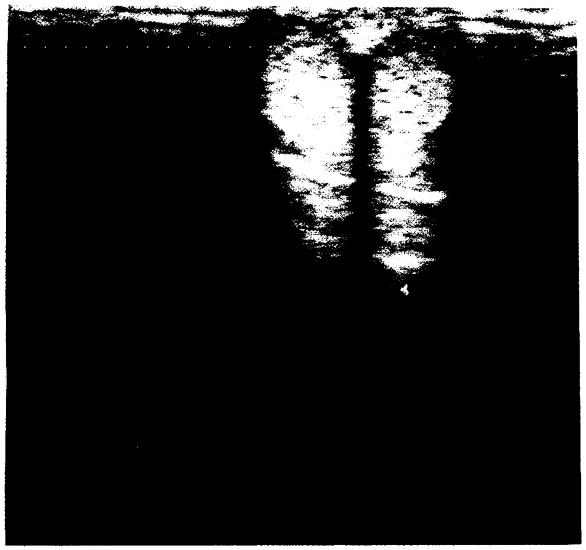


FIG. 1. Ultrasonography of corpora cavernosa with thrombosis and beginning of fibrosis before therapy.

Accepted for publication February 14, 1992.

FIG. 2. Ultrasonography of corpora cavernosa 7 days after intracavernous lysis. Note obvious decrease of intracavernous echo reflections.

DISCUSSION

After transurethral manipulations, a blood stasis or low flow priapism can occur by nervous dysfunction caused by anesthesia, urethral injury or spinal cord disorders while the causal mechanism is not clear. This may be assumed as a nervous dysfunction of smooth muscle fibers in the corpora cavernosa. It is not clear if the transformation of cavernous smooth muscle cells to fibroblast-like cells is the cause or a consequence of the priapism.[13] The nervous dysfunction can be proved by single potential analysis of cavernous electrical activity. Blood stasis leads to an elevation of carbon dioxide and an increase in blood viscosity, with consequent edema of the cavernous tissue. Thrombosis and subsequent destruction of the cavernous ultrastructures were observed.[14] In case of complete fibrosis of the corpora cavernosa and consequent impotence, a penile prosthesis seems to be the only available therapy.

A decrease in the blood viscosity and lysis of commencing thrombosis may interrupt the vicious circle. In long-lasting priapism with commencement of fibrosis streptokinase may dissolve the thrombosis and lead to increased blood flow. Streptokinase is a protein from β-hemolyzing streptococci, which begins the human fibrinolysis by activating plasminogen to plasmin.[15] Therefore, old wounds can start bleeding, so that in priapism therapy following transurethral manipulations catheterization should be done.

Patients with long-term, low flow priapism and intracavernous thrombosis should undergo therapy after diagnostic procedures (Doppler sonography, single potential analysis of cavernous electrical activity, aspiration and analysis of intracavernous blood) and exclusion of contraindications (hypertonus, myocardial infarcts more than 12 hours previously, hemostatic diseases, recent operations until day 6 or severe gastrointestinal diseases). A local lysis can be done using a high dosage of fibrinolytic agent (500,000 IU streptokinase), perfused slowly with 2 butterfly cannulas intracavernously. Control of the partial thromboplastin time is inevitable during therapy. Side effects include bleeding, allergic reactions, increase of temperature and nausea. In case of severe bleeding streptokinase infusion should be stopped and a proteinase inhibitor, synthetic antifibrinolytic agents or substitution of coagulation factors should be given.[16] Intracavernous lysis seems to be an effective treatment for long-lasting priapism induced by intracavernous thrombosis. However, more experience in this field is required.

REFERENCES

1. Porst, H. and van Ahlen, H.: Pharmakon-induzierte Priapismen—ein Erfahrungsbericht über 101 Fälle. Urologe A, **28:** 84, 1989.
2. Wasmer, J. M., Carrion, H. M., Mekras, G. and Politano, V. A.: Evaluation and treatment of priapism. J. Urol., **125:** 204, 1981.
3. Hauri, D.: Klinik und Pathophysiologie des Priapismus. Urologe A, **20:** 346, 1981.
4. Marx, F. J.: Zur Therapie des Priapismus. Urologe A, **20:** 353, 1981.
5. Brindley, G. S.: New treatment for priapism. Lancet, **2:** 220, 1984.
6. Stief, C. G., Gilbert, P., Wetterhauer, U., Bähren, W., Thon, W. and Altwein, J. E.: Metaraminol—ein Antidot bei SKAT-bedingter prolongierter Erektion. Urologe A, **25:** 164, 1986.
7. Winter, C. C.: Priapism cured by creation of fistulas between glans penis and corpora cavernosa. J. Urol., **119:** 227, 1978.
8. Quackles, R.: Cure of a patient suffering from priapism by cavernospongiosa anastomosis. Acta Urol. Belg., **32:** 5, 1964.
9. Grayhack, J. T., McCullough, W., O'Conor, V. J., Jr. and Trippel, O.: Venous bypass to control priapism. Invest. Urol., **1:** 509, 1964.
10. Witt, M. A., Goldstein, I., Saenz de Tejada, I., Greenfield, A. and Krane, R. J.: Traumatic laceration of intracavernosal arteries: the pathophysiology of nonischemic, high flow, arterial priapism. J. Urol., **143:** 129, 1990.
11. Bertram, R. A., Carson, C. C., III and Webster, G. D.: Implantation of penile prostheses in patients impotent after priapism. Urology, **26:** 325, 1985.
12. Stief, C. G., Djamilian, M., Schaebsdau, F., Truss, M. C., Schlick, R. W., Abicht, J. H., Allhoff, E. P. and Jonas, U.: Single potential analysis of cavernous electric activity—a possible diagnosis of autonomic impotence? World. J. Urol., **8:** 75, 1990.
13. Spycher, M. A. and Hauri, D.: The ultrastructure of the erectile tissue in priapism. J. Urol., **135:** 142, 1986.
14. Hauri, D., Spycher, M. and Brühlmann, W.: Erection and priapism: a new physiopathological concept. Urol. Int., **38:** 138, 1983.
15. Barthels, M. and Poliwoda, H.: Gerinnungsanalysen. New York: Georg Thieme Verlag, p. 17, 1987.
16. Behring Internal Information for STREPTASE 250000 (R). Behringwerke, 1988.