0022-5347/81/1253-0329$02.00/0
THE JOURNAL OF UROLOGY
Vol. 125, March

THE INTRA-ABDOMINAL TESTES: MICROVASCULAR AUTOTRANSPLANTATION

SHERMAN J. SILBER*

From St. Luke's West Hospital, St. Louis, Missouri

ABSTRACT

Four patients with high intraperitoneal testes underwent autotransplantation to the scrotum by division of the spermatic vessels. On one side a microsurgical reanastomosis was performed. No reanastomosis was performed on the other side. Microsurgical reanastomosis prevented atrophy.

Recently, there has been considerable discussion on how to localize best a non-palpable cryptorchid testis (spermatic venography, EMI scan or spermatic arteriography).[1, 2] Furthermore, when the testis is high and intraperitoneal there has been much debate over proper surgical management.[1, 3–5]

In my experience laparoscopy has been the simplest, safest and most reliable method of localizing high intra-abdominal testes.[6] For surgical management division of the spermatic vessels allows the testicle to be brought into the scrotum. There is no apparent damage in many cases because of collateral blood supply via the deferential artery.[5] However, the risk of testicular atrophy when collateral circulation is inadequate may need re-emphasis.

Recently, several patients were seen with bilateral intra-abdominal testes who underwent simple division of the spermatic vessels on one side at another institution with subsequent testicular atrophy. Spermatic vessel division on the other side was done on these patients with microsurgical reanastomosis to the inferior epigastric vessels.[7] On the side in which revascularization of the divided spermatic vessels was done the testis retained its normal size and texture.

PATIENT SELECTION

Four patients with high intraperitoneal testes were referred specifically because of anticipation that division of the spermatic vessels without reanastomosis might be hazardous in their particular cases. Of these patients 2 (both 5 years old) had undergone previous division of the spermatic vessels on one side only, using the Fowler-Stephens technique to bring the high testes into the scrotum, but this had resulted in complete atrophy. Therefore, these 2 patients were referred to me with only 1 remaining testicle and a history suggesting that simple division of the spermatic vessels without microsurgical reanastomosis might result in the loss of their only remaining testicle. A third patient had undergone no previous attempt at orchiopexy but, since he was 21 years old and had normal postpuberal development as well as a heavy abdominal musculature, it was feared that there would be tension on the vas deferens and deferential artery. The fourth patient was a 5-year-old boy with high testes, who had undergone no previous attempts at orchiopexy.

TESTICLE AUTOTRANSPLANT CASE REPORTS

Case 1. A 5-year-old boy with non-palpable undescended testes was referred from out of state. The patient had undergone bilateral inguinal exploration 1 year earlier and no testes were found in the inguinal canal. An intraperitoneal extension of the incision on each side demonstrated high intraperitoneal testes. The internal spermatic vessels on the right side were clamped and a small incision was made in the testis to check for adequacy of the collateral circulation. Bleeding was observed but was not brisk. Therefore, the Fowler-Stephens test was believed to be equivocal on the right side but the vessels were divided and the testicle then was placed in the scrotum. The Fowler-Stephens test also was equivocal on the left side but the vessels were not divided and the procedure was abandoned. Postoperatively, during the next 3 months, the right testicle atrophied totally. The patient then was referred to me for autotransplantation of the remaining left testicle.

Laparoscopy showed that the left testicle was intraperitoneal and appeared tacked down in the area of the internal ring. The internal spermatic vessels were under tension. The vas deferens was not under tension. The abdomen then was opened through a midline intraperitoneal incision. The spermatic vessels were clamped proximally and a microvascular clamp was placed on the vessels distally. The epigastric vessels were freed up inferior to the area of the previous left groin incision. The spermatic artery was spatulated and then anastomosed to the inferior epigastric artery by the technique described previously (figs. 1 and 2).[6, 8] Ischemia time was 40 minutes. The testicle then was placed in the left scrotal sac with the vas deferens coming just over the pubic symphysis, the shortest possible route. There was no tension on the microvascular anastomosis but there was a fair degree of tension on the vas deferens and the deferential vessels that seemed unavoidable.

Convalescence was uneventful. The patient was discharged from the hospital 6 days later. On followup examination 8 months later the testicle was of normal size and consistency with no sign of atrophy. A human chorionic gonadotropin stimulation test showed a normal testosterone response similar to that preoperatively.

Case 2. A 5-year-old child with prune belly syndrome underwent orchiopexy on the left side at another institution with the Fowler-Stephens procedure, dividing the spermatic vessels and allowing the testicle to be supplied by the artery of the vas. The vascular pedicle was divided high and the testis was brought down on a long strip of peritoneum that was attached to the vas and its blood supply. The surgeon noted that there still was some tension on the vas deferens and collateral vasculature supplying the testes but that it finally was anchored satisfactorily in the dartos pouch in the left scrotum.

Postoperatively, the testis was found to be located actually just above the pubic symphysis in the subcutaneous fatty tissue and not truly in the scrotum. There was partial atrophy as well. Therefore, it was decided to perform the orchiopexy on the opposite side using the microvascular technique.

Laparoscopy demonstrated an essentially undisturbed intraperitoneal testicle (fig. 3). Autotransplantation was accomplished by division of the spermatic vessels with reanastomosis to the inferior epigastric vessels. Postoperatively, there was good position of the testes with no atrophy. There was some

Accepted for publication June 6, 1980.
Read at annual meeting of American Urological Association, San Francisco, California, May 18–22, 1980.
* Requests for reprints: 456 N. New Ballas Rd., St. Louis, Missouri 63141.

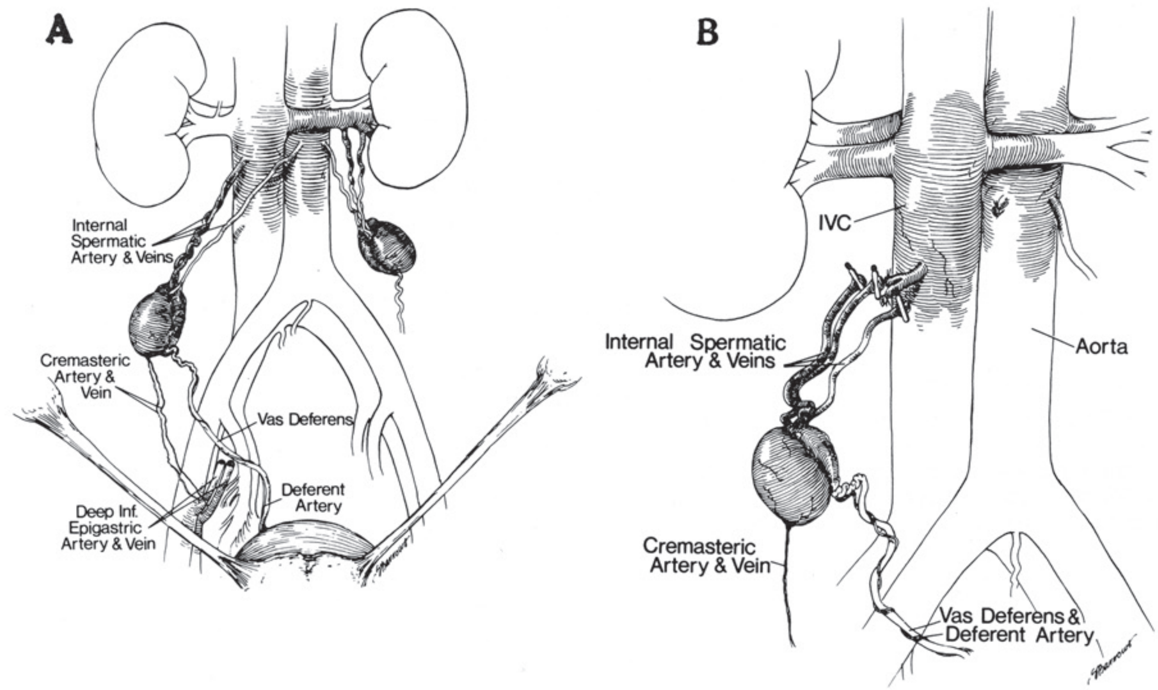

FIG. 1. *A,* anatomic configuration of high intraperitoneal testicle (not "long looped vas") which requires division of spermatic vessels to bring into scrotum. *B,* high division of spermatic vessels. Reprinted with permission.[8]

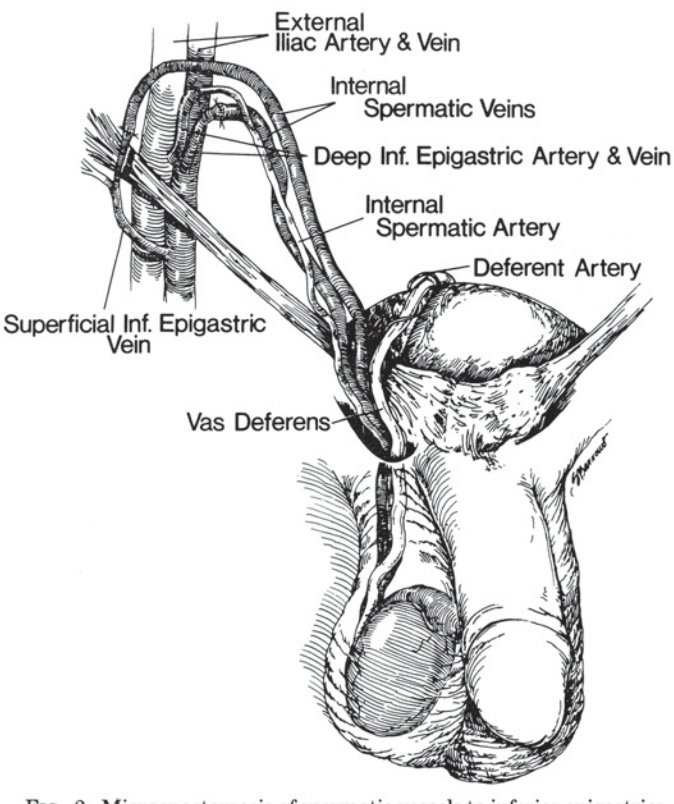

FIG. 2. Microanastomosis of spermatic vessels to inferior epigastrics. Reprinted with permission.[8]

tension on the vas deferens but this did not compromise the blood supply coming from the reanastomosed internal spermatic vessels. Postoperatively, the human chorionic gonadotropin stimulation test demonstrated a normal testosterone response as it did preoperatively.

Case 3. A 6-year-old boy without any palpable testes and a positive human chorionic gonadotropin stimulation test had an EMI scan that revealed the testes to be located high intraperitoneally. The abdomen was opened through a midline intraperitoneal incision and 2 normal-sized testes were located intraperitoneally and rather high up. The Fowler-Stephens maneuver was done on each side and free bleeding was noted. The spermatic vessels then were divided and the testes were mobilized out of the abdomen with no interference with its collateral blood supply through the deferential artery. However, it was noted that there would be tension on the vas deferens on each side if the testes were brought into the scrotal sac.

No reanastomosis of the vessels was done on the left side and the testicle was allowed to remain in the left scrotal sac under some tension, relying on collateral circulation via the deferential artery and vein. However, on the right side the inferior epigastric artery and veins were anastomosed directly to the divided spermatic vessels with microsurgery.

The left testicle had atrophied almost completely 6 months postoperatively and the right testicle was of normal size and consistency. The postoperative human chorionic gonadotropin stimulation test showed a normal testosterone response.

Case 4. A 21-year-old man who was about to be married was concerned that he had never noticed testicles in the scrotal sac. The patient was turned down from the army for this reason and was seeking consultation. He had undergone normal postpuberal development. Testosterone levels averaged 315 ng./100 ml. Follicle-stimulating hormone essentially was at castrate levels (>100 mIU/ml.) and luteinizing hormone was elevated markedly in the range of 45 to 55 mIU/100 ml. (upper limit of normal <30 mIU/ml.). An EMI scan demonstrated 2 normal-sized testicles intraperitoneally at about the level of the pelvic brim. Semen analysis demonstrated azoospermia with a normal semen volume and a normal fructose.

Because the patient was concerned about possible development of an intra-abdominal testicular malignancy, as well as the possibility of premature loss of endocrine function of the testicles, laparotomy was done. Two normal-sized testes were located at the pelvic brim as predicted by the EMI scan. The intraperitoneal appearance was similar to that of 2 normal ovaries in a young woman (fig. 4). The Fowler-Stephens maneuver was done on each side and bleeding from the cut tunica albuginea of the testicle was present but was equivocal in briskness. Division of the spermatic vessels with microvascular reanastomosis to the inferior epigastric vessels was done on the right side. However, the spermatic vessels were merely divided on the left side and the testicle was brought into the scrotal sac, relying on collateral circulation via the deferential artery and veins, which were preserved properly. There was a great deal of tension on the vas deferens despite the fact that the shortest possible route was used to get the testicle into the scrotum.

The left testicle was atrophied completely 3 months postoperatively and the right testicle was of normal size and consistency (fig. 5). Postoperative hormone values were unchanged from those preoperative. Followup on this patient extended to 6 months without any sign of deterioration of the right testicle. Shortly after the last followup visit the patient was killed in an automobile accident.

ADULT CRYPTORCHID TESTES

A total of 3 men with untreated cryptorchid testes underwent orchiectomy. The 3 patients were 28, 42 and 52 years old, respectively. The 52-year-old man had bilateral cryptorchidism. The other 2 cases were unilateral. The patient with bilateral adult cryptorchid testes had undergone normal puberty with a normal sex life and was admitted to the general surgery service with bilateral inguinal hernias. This patient had been impotent for the last 5 years and expressed no interest in sex. Preoperatively, follicle-stimulating and luteinizing hormones were at castrate levels and the testosterone was consistently <150 ng./100 ml. At the time of bilateral inguinal herniorrhaphy the general surgeon removed both cryptorchid testicles in the inguinal canal. Histology of both testes demonstrated severe disorganization and hyalinization with essentially no seminiferous tubules and few normal appearing Leydig cells. The 2 unilateral cryptorchid testicles had similar findings except that in the 28-year-old patient the Leydig cell changes were minimal.

INADVERTENT DIVISION OF SPERMATIC VESSELS AT TIME OF BILATERAL VARICOCELECTOMY OR BILATERAL INFANT HERNIORRHAPHY

Example 1. A 31-year-old man had been noted several years earlier to have a sperm count of 98,000,000/cc but allegedly the

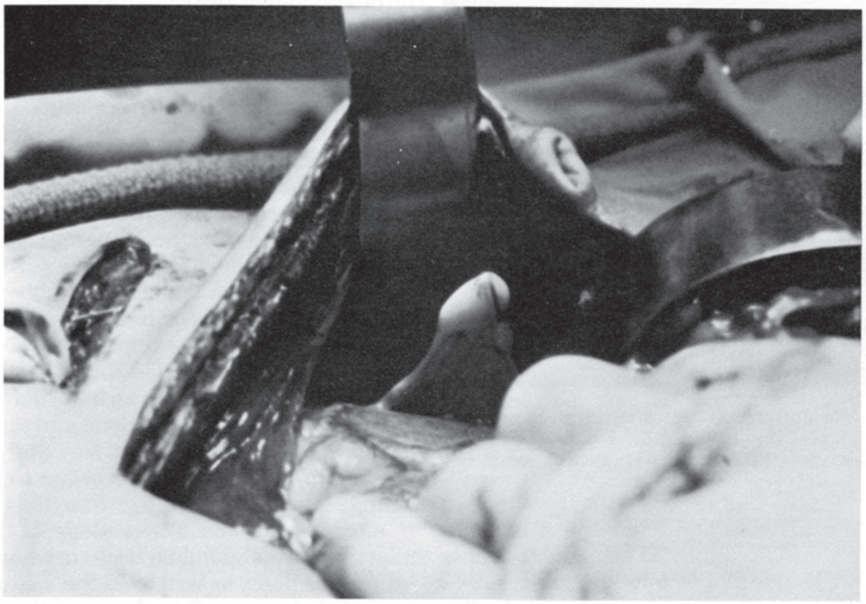

FIG. 3. High intraperitoneal testis in child without prune belly syndrome. Reprinted with permission[8]

FIG. 4. High intraperitoneal testes in 21-year-old man. Reprinted with permission[8]

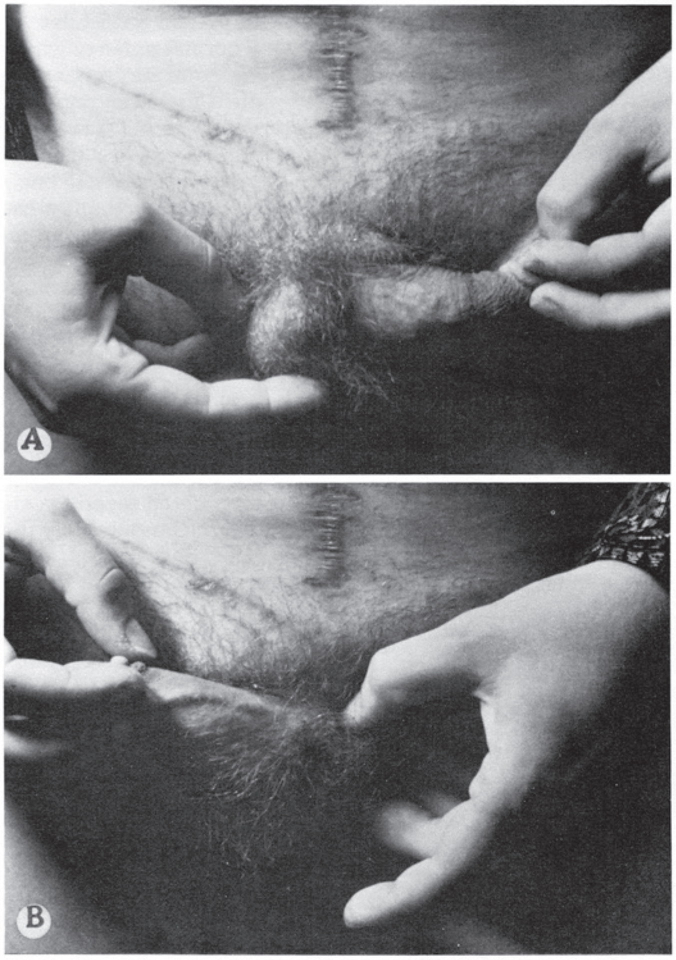

FIG. 5. *A,* right testicle did not atrophy after autotransplantation with revascularization. *B,* left testicle atrophied after division of spermatic vessels without reanastomosis. Reprinted with permission.[8]

motility was poor. He and his wife had been trying to have children for several years unsuccessfully. The patient had a bilateral varicocele and was subjected to bilateral varicocelectomy at another institution. Six months postoperatively the sperm count consistently was <1 million per cc and eventually he became azoospermic. A testicle biopsy done at the time of the bilateral varicocelectomy was normal, showing all stages of spermatogenesis with many mature spermatids. The testicle biopsy performed 6 months postoperatively revealed severe maturation arrest and hypospermatogenesis. Follicle-stimulating and luteinizing hormones, and testosterone were in the normal range preoperatively. Follicle-stimulating and luteinizing hormones were elevated 6 months postoperatively. Follicle-stimulating hormone was in the range of 40 to 50 mIU/ml. (upper limit of normal 17 mIU/ml.) and luteinizing hormone was >35 mIU/ml. (upper limit of normal 29 mIU/ml.). Testosterone remained in the range of 300 to 400 ng./100 ml. It appeared that this patient underwent a severe deterioration of spermatogenesis as well as some loss of Leydig cell function as a result of inadvertent ligation of the spermatic artery bilaterally.

Example 2. A 28-year-old man who had sperm counts ranging between 15,000,000 and 25,000,000/ml. was evaluated for infertility at another institution. The patient had had a completely atrophic left testicle since birth. He was said to have a right varicocele and underwent right varicocelectomy at that institution. Postoperatively, the patient became azoospermic. Serum testosterone decreased to <100 ng./100 ml. and the follicle-stimulating and luteinizing hormones increased to castrate levels. The testicle underwent severe atrophy.

Example 3. A 26-year-old man was referred for azoospermia. The patient had a history of having undergone bilateral inguinal herniorrhaphy when he was 2 years old. His parents recount that the right testicle atrophied and disappeared within 3 months of the herniorrhaphy but the left testicle showed no change and developed normally. The patient underwent left inguinal exploration and testicle biopsy at this institution. He was found to have had a complete resection of the vas deferens, deferential artery and internal spermatic vessels. The left testicle still was viable only because of collateral circulation at the level of the external inguinal ring. Testicle biopsy demonstrated maturation arrest and hyalinization of many seminiferous tubules.

DISCUSSION

The controversies that encircle the problem of the intra-abdominal testes fall into 2 major categories: 1) does it make any sense to try to treat surgically these intraperitoneal testes and 2) if there is a rationale for treating these cases what is the safest operative approach?

The danger of unrecognized cancer in the patient with an intra-abdominal testicle already has been dealt with in detail and certainly provides 1 motive for placing these testicles in the scrotum or, possibly, removing them.[9, 10] Furthermore, the intra-abdominal testis is subject easily to the possibility of torsion that could present as a severe acute abdomen.[11]

Previously, it had been assumed generally that the cryptorchid testicle suffers only a loss of spermatogenic function; hormonal function supposedly was unaltered. However, recent studies have demonstrated that the abdominal environment also affects the endocrine function of the testis and will result in premature loss of testosterone production. Cryptorchid patients demonstrate impaired intratesticular androgen production and patients with bilateral cryptorchidism will have an elevated luteinizing hormone level.[12–14] Experimental studies in the rat have demonstrated that when normal testes are placed intra-abdominally there is an immediate and dramatic elevation in follicle-stimulating hormone corresponding to a rapid deterioration of spermatogenesis. However, during a longer interval the luteinizing hormone also begins to increase gradually, indicating loss of endocrinologic function of the cryptorchid testes in adulthood.[15] The observations in the 1 adult patient with bilateral intra-abdominal testes in whom testicle autotransplantation was done, as well as in the 3 adults upon whom orchiectomy was done, confirm the mounting evidence that the intra-abdominal environment is detrimental not only to spermatogenesis but also to hormone production of the testes. It simply requires longer for this aspect of testicular function to deteriorate.

The major unanswered question is whether transplantation of these testes to the scrotum will enhance the ultimate development of fertility when these children grow up. There is overwhelming experimental evidence that making a testicle cryptorchid diminishes spermatogenesis and then replacing that cryptorchid testicle in the proper scrotal environment allows spermatogenesis to recover.[16-20]

More dramatic are the few but well documented case reports of azoospermic adults (16 to 25 years old) with bilateral cryptorchidism who regained normal spermatogenesis and reasonable sperm counts after orchiopexy. These cases are unusual but demonstrate dramatically the potential for fertility in cryptorchid patients.[21, 22] In these dramatic cases follicle-stimulating hormone was elevated and, yet, this did not prevent recovery of spermatogenesis.

The second question in this controversy is what is the safest method for transferring the high intraperitoneal testis into the scrotum. The concept of simply dividing the internal spermatic vessels is not new. Bevan first recommended this approach in 1903.[23] He and Moschcowitz[24] reported good results. However, others reported uniformly poor results with the Bevan operation.[25–27]

In 1963 Fowler and Stephens first demonstrated that by dividing the internal spermatic vessels high up and not dissecting their attachment to the cord it was possible to preserve collateral circulation via the deferential artery.[5] The spermatic vessels could be clamped first and the testicle biopsied to determine the adequacy of collateral blood flow. Even in their hands almost half of the patients underwent some degree of atrophy and a third of them underwent rather severe testicular atrophy. Furthermore, the original diagrams of Fowler and Stephens demonstrate that their technique was most valuable in the so-called "long looped vas" whereby the testis actually is located at the level of the internal inguinal ring and the vas deferens loops down in the canal toward the external ring and then comes back to the testis at the internal ring. By avoiding dissection in the inguinal canal the collateral circulation can be preserved. Most of the cases that Fowler and Stephens described did not involve the severely high intra-abdominal testes, whereby with the division of spermatic vessels there still would be potential tension on the vas deferens after the testicle is brought into the scrotum. Others have resorted to use of the Fowler-Stephens procedure for the high intraperitoneal testes and they have reported satisfaction with this technique but variable instances of atrophy have occurred with all groups using this approach.[1, 3, 4, 28]

To my knowledge 4 groups have used revascularization of the divided spermatic vessels in these cases.[7] Silber and Kelly reported the first such case in 1976.[7] Romas and associates[29] and others[30, 31] reported the second and subsequent series. The debates about whether or not revascularization with the microsurgical technique is necessary have been caused partly by lumping the "long looped vas" (which really is a high inguinal testis) in with the truly intraperitoneal intra-abdominal testicles. The Fowler-Stephens technique of dividing the vessels is almost uniformly successful in the case of the high inguinal "long looped vas" testicle. It is with the high intraperitoneal testicle that atrophy is encountered.

In the 4 cases presented herein referral to this center was based upon reasonable suspicion ahead of time that these patients would suffer testicular atrophy if no revascularization were accomplished. In all cases there was some degree of tension on the vas deferens with proper scrotal placement. This tension could have compromised collateral blood supply afforded by the deferential artery. In such cases if revascularization was not done some degree of testicular atrophy was highly likely. However, when revascularization was done no atrophy, functional or anatomic, was observed.

In 1949 Harrison demonstrated in fresh autopsy dissections that the sum of the diameters of the deferential and cremasteric arteries was equal to that of the testicular artery in only a third of the cases, indicating that adequate functional collateral to the testis is by no means universal.[32, 33] Furthermore, observations in patients who have undergone spermatic artery ligation at the time of varicocelectomy or inadvertently at the time of childhood inguinal herniorrhaphy have demonstrated to us that collateral circulation to the testes is by no means secure and, in some cases, it will be inadequate to nourish the testes after ligation of the spermatic vessels.

REFERENCES

1. Levitt, S. B., Kogan, S. J., Engel, R. M., Weiss, R. M., Martin, D. C. and Ehrlich, R. M.: The impalpable testis: a rational approach to management. J. Urol., **120:** 515, 1978.
2. Weiss, R. M., Glickman, M. G. and Lytton, B.: Clinical implications of gonadal venography in the management of the non-palpable undescended testis. J. Urol., **121:** 745, 1979.
3. Gibbons, M. D., Cromie, W. J. and Duckett, J. W., Jr.: Management of the abdominal undescended testicle. J. Urol., **122:** 76, 1979.
4. Clatworthy, H. W., Jr., Hollabaugh, R. S. and Grosfeld, J. L.: The "long looped vas" orchidopexy for the high undescended testis. Amer. Surg., **38:** 69, 1972.
5. Fowler, R. and Stephens, F. D.: The role of testicular vascular anatomy in the salvage of high undescended testes. In: Congenital Malformations of the Rectum, Anus, and Genital Urinary Tract. Edited by R. Webster. London: E. & S. Livingston, Ltd., pp. 306–320, 1963.
6. Silber, S. J. and Cohen, R.: Laparoscopy for cryptorchidism. J. Urol., **124:** 928, 1980.
7. Silber, S. J. and Kelly, J.: Successful autotransplantation of an intra-abdominal testis to the scrotum by microvascular technique. J. Urol., **115:** 452, 1976.
8. Silber, S. J.: Microsurgery. Baltimore: The Williams & Wilkins Co., pp. 259–280, 1979.
9. Martin, D. C. and Menck, H. R.: The undescended testis: management after puberty. J. Urol., **114:** 77, 1975.
10. Campbell, H. E.: Incidence of malignant growth of the undescended testicle: a critical and statistical study. Arch. Surg., **44:** 353, 1942.
11. Riegler, H. C.: Torsion of the intra-abdominal testis: an unusual problem in diagnosis of the acute surgical abdomen. Surg. Clin. N. Amer., **52:** 371, 1972.
12. Bramble, F. J., Houghton, L. E., O'Shea, A. and Jacobs, H. S.: Proceedings of the Society for Endocrinology. Endocrine evaluation of treated cryptorchidism: results of a 10 year follow-up. J. Endocr., **64:** 56P, 1975.
13. Atkinson, P. M., Epstein, M. T. and Rippon, A. E.: Plasma gonadotropins and androgens in the surgically treated cryptorchid patient. J. Ped. Surg., **10:** 27, 1975.
14. Epstein, M. T., Atkinson, P. M. and Rippon, A. E.: Proceedings: plasma gonadotropins and androgens in surgically treated cryptorchid patients. J. Endocr., **65:** 11, 1975.
15. Altwein, J. E. and Gittes, R. F.: Effect of cryptorchidism and castration on FSH and LH levels in the adult rat. Invest. Urol., **10:** 167, 1972.
16. Nelson, W. O.: Mammalian spermatogenesis: effect of experimental cryptorchidism in the rat and non-descent of the testis in man. Rec. Prog. Hormone Res., **6:** 29, 1951.
17. Sohval, A. R.: Testicular dysgenesis as an etiologic factor in cryptorchidism. J. Urol., **72:** 693, 1954.
18. Mengel, W., Hienz, H. A., Sippe, W. G., II and Hecker, W. Ch.: Studies on cryptorchidism: a comparison of histologic findings in the germinative epithelium before and after the second year of life. J. Ped. Surg., **9:** 445, 1974.
19. Kiesewetter, W. B., Shull, W. R. and Fetterman, G. H.: Histologic changes in the testis following the anatomically successful orchidopexy. J. Ped. Surg., **4:** 59, 1969.
20. Hadziselimovic, F., Herzog, B. and Seguchi, H.: Surgical correction of cryptorchidism at 2 years: electron microscopic and morphometric investigations. J. Ped. Surg., **10:** 19, 1975.
21. Britton, B. J.: Spermatogenesis following bilateral orchidopexy in adult life. Brit. J. Urol., **47:** 464, 1975.
22. Comhaire, F., Derom, F. and Vermeulen, L.: The recovery of spermatogenesis in an azoospermic patient after operation for bilateral undescended testes at the age of 25 years. Int. J. Androl., **1:** 117, 1978.
23. Bevan, A. D.: The surgical treatment of undescended testicle: a further contribution. J.A.M.A., **41:** 718, 1903.
24. Moschcowitz, A. V.: The anatomy and treatment of undescended testes: with special reference to the Bevan operation. Ann. Surg., **52:** 821, 1910.
25. Mixter, C. G.: Undescended testicle: operative treatment and end-results. Surg., Gynec. & Obst., **39:** 275, 1924.
26. Wangensteen, O. H.: Undescended testes; experimental and clinical study. Arch. Surg., **14:** 663, 1927.
27. MacCollum, D. W.: Clinical study of spermatogenesis of undescended testicles. Arch. Surg., **31:** 290, 1935.
28. Brendler, H. and Wulfsohn, M. A.: Surgical treatment of the high undescended testis. Surg., Gynec. & Obst., **124:** 605, 1967.
29. Romas, N. A., Janecka, I. and Krisiloff, M.: Role of microsurgery in orchiopexy. Urology, **12:** 670, 1978.
30. McMahon, R. A., O'Brien, M. C. and Cussen, L. J.: The use of microsurgery in the treatment of the undescended testis. J. Ped. Surg., **11:** 521, 1976.
31. Wacksman, J., Dinner, M. and Straffon, R. A.: Testicular autotransplant in prune belly disease. Read at annual meeting of the American Urological Association, Washington, D. C., May 21–25, 1978.
32. Harrison, R. G.: The distribution of the vasal and cremasteric arteries to the testis and their functional importance. J. Anat., **83:** 267, 1949.
33. Silber, S. J.: Transplantation of human testis for anorchia. Fertil. Steril., **30:** 181, 1978.