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Spermatic Cord Torsion: Loss of Spermatogenesis Despite Return of Blood Flow[1]

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ABSTRACT

Previous work in animal models has recapitulated and refined the clinical observation that longer periods of testicular torsion are associated within increased damage to the testis. Minimum times of damage-inducing torsion have not been established, however, nor has it been established to what degree blood flow returns to the testis on examination several hours or days after the torsion repair. Adult male rats were subjected to 0.5-h or 1.0-h 720° torsion and examined for effects on testis weight, histology, and daily sperm production (DSP). None of the parameters examined were affected by 0.5-h, but all were affected by 1.0-h torsion. In a subsequent experiment, adult rats were subjected to unilateral, 1-h, 720° torsion, and bilateral testicular blood flow as measured by radiolabeled microsphere distribution was determined during torsion and 4 h, 24 h, 2 days, 7 days, 15 days, and 30 days after repair of torsion. Ipsilateral testicular blood flow was reduced 94% during torsion. Blood flow was not significantly different from control or contralateral values by 4 or 24 h after repair of torsion. In spite of this return of blood flow, testis weights were significantly reduced by half by 7 days after torsion repair and spermatogenesis was clearly disrupted upon histological examination 30 days after torsion repair. Increasing the time of torsion to 2 or 4 h did not inhibit return of blood flow to the testis as measured 48 h after torsion repair. Unless the testis is infarcted, testicular blood flow in the rat model returns to normal after torsion repair. It is speculated that testicular injury occurs at or before initial reperfusion, and experiments are underway to determine specific cell responses to graded periods of torsion. Contralateral testes were not altered by ipsilateral torsion.

INTRODUCTION

Testicular torsion, or more properly, torsion of the spermatic cord, is a medical emergency that must be treated promptly to avoid loss of the ipsilateral testis [1, 2]. Torsion of the spermatic cord compresses the spermatic vessels, reduces or halts testicular blood flow, and induces testicular ischemia. Most salvageable testes have produced symptoms for 6–10 h or less [3–5]; but even in cases in which surgically successful detorsion has occurred within this time period, the ipsilateral testis often becomes permanently disfunctional [2, 6], though it may still be present as a palpable mass in the scrotum. These testes are not "salvaged" in any biologically useful sense.

The reason for this loss of function in potentially viable testes is often unclear, but this may be so because the pathophysiology of testicular torsion, in general, is little understood. For example, specific effects of graded ischemia or Leydig cell or Sertoli cell function are unknown. Whether the testicular injury after torsion is due to hypoxia during torsion or to injury resulting from return blood flow, i.e., reperfusion, subsequent to detorsion is not clear [7]. Even more basically, the degree to which blood flow actually returns after repair of torsion is unknown even though blood flow return should have a major impact on tissue viability and endocrine signalling.

In the clinical setting, the surgeon intraoperatively examines the detorqued testis for visual evidence of reperfusion. Nonreperfusing, infarcted testes are conventionally removed and reperfusing testes are left in place. Thus, the presence or absence of reperfusion is typically noted; but in the clinical setting, the degree to which blood flow returns to the testis after torsion repair has not been evaluated. Whether blood flow is normal upon relief or torsion, or perhaps more importantly, whether blood flow is normal some hours or days after relief or torsion is a question that has been virtually unexamined in humans or, for that matter, in laboratory animal models.

In an earlier study from this laboratory, 720° rotation of the rat testis completely eliminated testicular blood flow during the torsion period [8], but 24 h later, blood flow in the torqued testes had returned to values not significantly different from those of controls. It was the case, however, that testicular blood flow values in the post-torsioned testes were more variable than in control testes. Tzika et al. [9] have also reported highly variable reperfusion of the rat testes 3 h after detorsion. Testicular blood flow at other times after detorsion has not been reported. The present study was undertaken to determine the minimum period of testicular ischemia that would eliminate spermatogenesis in the rat model and to provide first-time evidence of testicular reperfusion minutes, hours, and days after testicular torsion.

MATERIALS AND METHODS

Adult, male, Sprague-Dawley rats (450–550 g) were acquired from university vivarium sources and maintained on a 12L:12D cycle with ad libitum food and water.

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Effect of Duration of Torsion on Spermatogenesis

Rats were divided into five groups (n = 6 or 7 each): controls and those to be subjected to either 0.5-h or 1.0-h unilateral testicular torsion and examined either 2 days or 30 days later. Animals were anesthetized with urethane (1 mg/g BW), and testicular torsion was induced as previously described [8]. Briefly, the testis (left or right alternatingly) was gently retracted through a low laparotomy incision. The testis was freed from the epididymis, except at the level of the testicular hilus where the efferent ducts and the testicular vessels and nerves leave the testis. The testis was turned 720° along its longitudinal axis and on the spermatic pedicle. The rotational position was noted, and the testis was replaced in the scrotum. The incision was closed and the animal was maintained under anesthesia until 0.5 or 1.0 h later, when the incision was reopened and the testis was again retracted through the laparotomy incision. The testis was observed in order to ensure that it had retained the proper torsion throughout the torsion period; then the torsion was repaired by counter-rotation and by re-suturing of the testicular gubernacular stump to the scrotal gubernacular stump. During the operation, testes were observed for return of blood flow; then testes were replaced into the scrotum, the surgeon making sure that inguinal passage was unobstructed.

Two or thirty days later, ipsilateral and contralateral testes were exposed through a scrotal incision and examined for appearance of blood flow through testicular surface vessels, both grossly and under the operating microscope. Testes were extirpated, weighed, and subjected to determination of daily sperm production (DSP; [10]). Briefly, ipsilateral and contralateral testes were decapsulated and the seminiferous tubules were weighed. Seminiferous tubules were homogenized in 50 ml 0.154 M NaCl + 0.5% (v/v) Triton X-100 and 2% sodium azide. The concentration of condensed sperm nuclei in the resultant solution was determined and DSP (sperm $\times$ 10^6/g/day) was calculated for each testis [10].

In three additional animals/group, contralateral and ipsilateral testes were perfused-fixed with Karnovsky's fixative, embedded in plastic (historesin; LKB Products, Bromma, Sweden), sectioned, and stained with Toluidine blue for observation under the light microscope.

Within ipsilateral or contralateral sides, but between groups, data were examined by analysis of variance followed by Tukey's range test ($p < 0.05$). Within groups, ipsilateral and contralateral data were compared by Student's t-test ($p < 0.05$).

Effect of Duration of Torsion on Blood Flow Return

Rats were divided into nine groups (n = 5 each): control animals; those animals that had been subjected to 1- or 2-h 720° torsion and were examined either 4 h or 1, 2, 7, 15, or 30 days after torsion repair; and those animals that had been subjected to 1-h or 4-h 720° torsion and were examined 48 h after torsion repair. At the designated times, testicular blood flow was determined in each rat by the radiolabeled microsphere distribution technique [11] previously used in our laboratory [12, 13]. Briefly, ^{85}Sr-labeled microspheres (14 mCi/g; New England Nuclear, Boston, MA) were suspended in 0.154 M NaCl containing 0.5% Tween-80 and 10% dextran. Following urethane anesthesia, a PE-50 cannula was inserted into the left ventricle via the right carotid artery. The femoral artery was also cannulated with PE-50 tubing. A model P-20 intravenous pressure transducer (Gould-Stathem Instruments, Hato Rey, PR) and a model 321 dual-channel recorder (Sanborn Co., Palo Alto, CA) were used to monitor heart rate and blood pressure and to determine when the tip of the ventricular cannula was in place. A 0.1-ml solution (0.2–2.0 μCi, depending on specific activity) was injected into the left ventricle over a 10-sec time period. Beginning simultaneously, blood was withdrawn continuously from the femoral cannula over 70 sec. Animals were killed with an intracardiac injection of saturated KCl and the left and right testes and reference organs (liver, prostate, kidney) were obtained. Tissues and blood samples were weighed and the radioactivity within each sample was determined in a gamma counter (model 1280 Ultragamma; LKB, Uppsala, Sweden). Blood flow to each organ was calculated on the basis of the distribution of radioactivity to each organ and was expressed as ml/min/100 g tissue [11, 12] or as ml/min/testis.

Within ipsilateral or contralateral testes, but between times after torsion, data from animals subjected to 1-h unilateral torsion were examined by analysis of variance followed by Tukey's range test ($p < 0.05$). Within times after torsion, but between ipsilateral and contralateral testes, data were analyzed by Student's t-test ($p < 0.05$).

RESULTS

Qualitative Results: Appearance of Intraoperative Blood Flow and Testis Histology

Experimental testicular torsion applied in the manner described typically caused an ischemic "dusky" appearance to the testis within 5 min of torsion. After 0.5- and 1.0-h, 720° torsion, the ipsilateral testis vasculature was typically dark red-to-purple in appearance. Upon relief of torsion, testes appeared to reperfuse with the color of the testicular vasculature returning toward normal within 5 min.

Two days after 0.5-h torsion, all ipsilateral testes appeared slightly more hyperemic than unoperated testes. Thirty days after 0.5-h torsion, this qualitative effect was still present in half of the previously torqued testes, but the remaining ipsilateral testes and all contralateral testes appeared the same as controls.

Two days after 1.0-h torsion, all ipsilateral testes appeared clearly hyperemic, and slight hyperemia was still uniformly present in all testes 30 days after torsion repair.

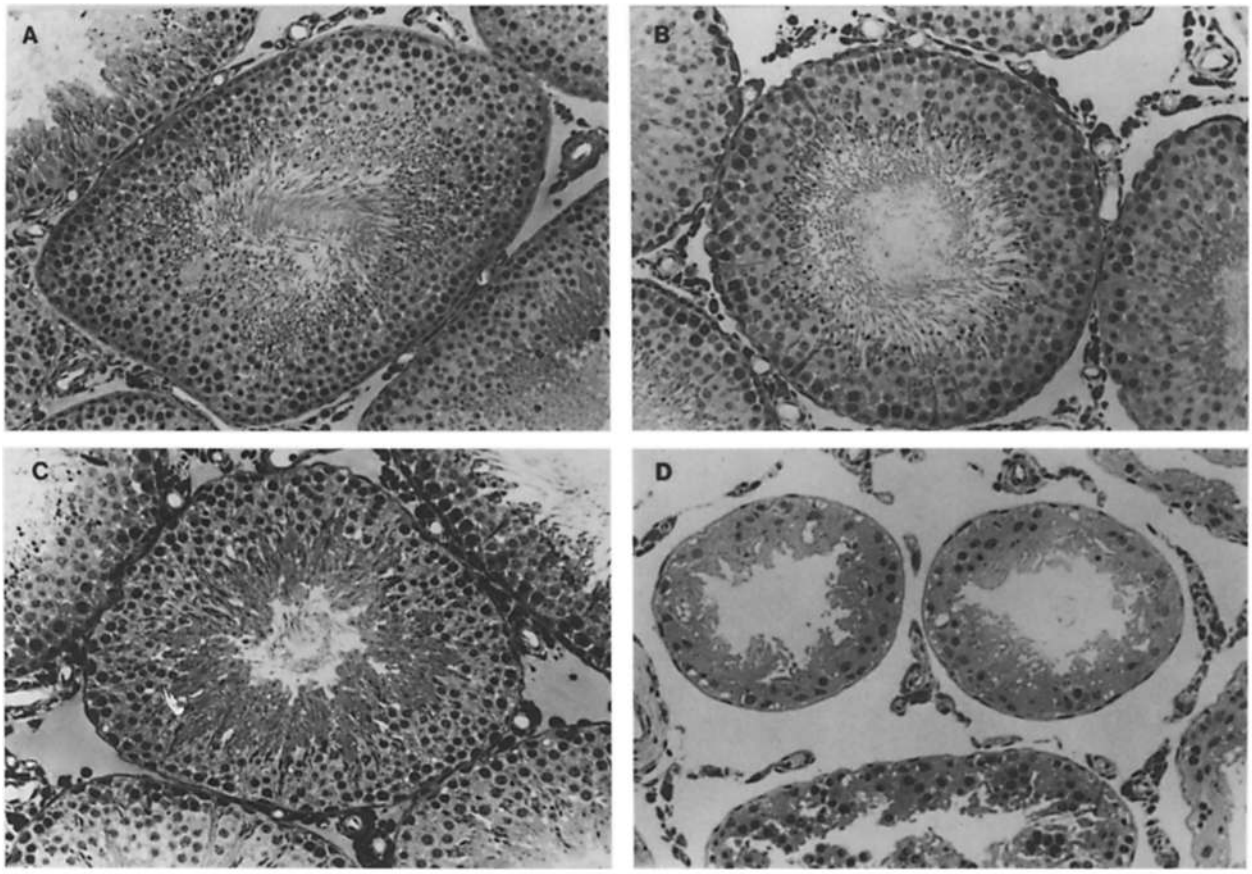

FIG. 1. Histology of rat testes 30 days after either 0.5-h or 1.0-h, 720° torsion. A and B) 0.5-h torsion, contralateral and ipsilateral, respectively. C and D) 1.0-h torsion, contralateral and ipsilateral, respectively.

When viewed under the operating microscope, the apparent hyperemia of the tissue at 2 and 30 days after torsion seemed due to focal enlargement of testicular microvessels immediately adjacent to focal areas of constricted microvasculature. This effect gave the testicular microvasculture a speckled or "patchy" appearance. Thirty days after 1-h torsion, the ipsilateral testes were reduced in size and contained some sclerotic-appearing seminiferous tubules. Contralateral testes in animals receiving 0.5-h or 1.0-h torsion were always normal in appearance.

Spermatogenesis appeared histologically normal in ipsilateral and contralateral testes 30 days after 0.5-h torsion (Fig. 1, A and B). Thirty days after 1-h torsion, contralateral testes were qualitatively normal (Fig. 1C), but seminiferous tubule profiles in ipsilateral testes were depleted of germ cells or contained only degenerate germ cell-Sertoli cell associations (Fig. 1D).

Quantitative Results: Testis Weights, Daily Sperm Production, and Testicular Blood Flow

Control rat testis weights were approximately 2 g, and DSP was approximately 20×10^6 sperm/g testis/day (Table 1). Thirty-minute torsion had no significant effect on either testis weight or DSP at either time point studied, but both testis weight and DSP were significantly reduced by 1-h torsion (Table 1). Thirty days after torsion, ipsilateral testis weights were approximately 62% of contralateral testis values or 67% of control values. DSP values were 42% of contralateral testis values or 36% of control values (Table 1).

Testicular blood flow values in ipsilateral and contralateral control testes were 17.1 ± 1.5 ml/min/100 g tissue and 18.9 ml/min/100 g tissue, respectively. During the period of torsion, ipsilateral testicular blood flow was severely restricted to less than 1 ml/min/100 g tissue (Fig. 2). This value was significantly lower than other ipsilateral values and the corresponding contralateral value ($p < 0.05$). Contralateral testis blood flow values during the torsion period tended to be lower but were not significantly different from control blood flow values (Fig. 2).

Four hours after the repair of 1-h 720° torsion, ipsilateral testicular blood flow values were not significantly different from contralateral values and were significantly higher than the blood flow values obtained during torsion; nevertheless, ipsilateral blood flow values were still significantly lower than those obtained from control testes or from those ipsilateral testes examined over the next several time periods

TABLE 1. Effect of 0.5-h and 1.0-h testicular torsion on ipsilateral and contralateral testes wts. and daily sperm production (DSP) 2 and 30 days after repair of torsion.

Group	n	Testis wt. (g)		DSP (Sp × 10 g/day)	
		Ipsilateral	Contralateral	Ipsilateral	Contralateral
Control*	7	1.89 ± 0.03	1.91 ± 0.05	21.2 ± 0.6	19.7 ± 0.9
0.5/2†	6	1.98 ± 0.07	1.89 ± 0.03	18.9 ± 0.07	20.4 ± 0.8
0.5/30	6	1.96 ± 0.12	1.96 ± 0.04	20.3 ± 3.1	21.6 ± 1.7
1.0/2	6	1.89 ± 0.16	2.10 ± 0.07	20.4 ± 0.5	19.8 ± 1.6
1.0/30	6	1.27 ± 0.08[a]	2.03 ± 0.06	7.6 ± 1.8[a]	18.9 ± 0.2

*For control animals, data from right and left testes are placed in this table under Ipsilateral and Contralateral, respectively.

†For each group code, the number before the diagonal represents the hours of 720° torsion and the number after the diagonal represents the number of days after torsion repair the data were collected.

[a]Significantly lower than other ipsilateral values and significantly lower than contralateral value ($p < 0.05$).

(Fig. 2). At 2, 7, and 15 days after repair of 1-h 720° torsion, testicular blood flow values in ipsilateral and contralateral testes were not different from each other or from control testis values (Fig. 2). Thirty days after torsion repair, ipsilateral testicular blood flow values were not significantly different from contralateral values, but were significantly lower than control testicular blood flow values (Fig. 2; $p < 0.05$).

While testicular blood flow, expressed as ml/min/100 g tissue, remained relatively stable after torsion, total blood flow to the testis did not. Ipsilateral testis weights decreased significantly between 7 and 15 days after repair of 1-h 720° torsion (Fig. 3); thus, total-organ blood flow declined over the same period (Fig. 4). Total testicular blood flow was approximately 0.30 ml/min/testis in controls (Fig. 4). After torsion repair, these blood flow values returned to control levels for a time, but a trend toward lower total blood flow was noticeable by 7 days after torsion repair and became statistically significant ($p < 0.05$) 15 and 30 days after torsion repair. Contralateral testis weights and total-organ blood flow values from animals after torsion repair were never significantly different from control values (Figs. 3 and 4).

Increasing the time of 720° torsion from 1 to 2 to 4 h was not associated with a reduction of return testicular blood flow 48 h after repair of torsion, whether expressed as ml/min/g of tissue or ml/min/testis (Fig. 5). In fact, both contralateral and ipsilateral testicular blood flow tended to be higher 48 h after 2- or 4-h torsion than after 1-h torsion (Fig. 5). This was especially true for the 4-h torsion group examined 48 h later (Fig. 5).

Average cardiac output, typically between 15 and 25 ml/min/100 g BW in these groups of rats, was unaccountably elevated in the 4/48 (hours of torsion/hours after torsion repair) group to 35 ml/100 g/BW; average ipsilateral kidney flow, typically between 250 and 350 ml/min/100 g tissue, was elevated to 408 ml/min. Thus, when expressed relative to kidney blood flow or to cardiac output, the 4/48 testicular blood flow values were not different from those in the other groups (data not shown). Other cardiac output values and reference organ blood flow values among the

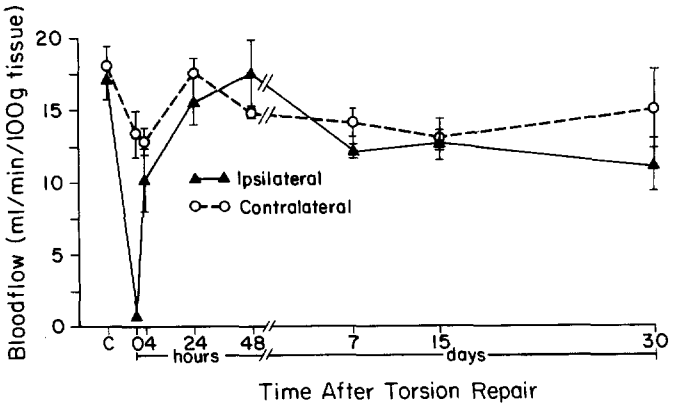

FIG. 2. Testicular blood flow (ml/min/100 g tissue) in control animals; in those experiencing 1-h, 720° testicular torsion (0 h after torsion repair); and in those examined 4 h, 24 h, or 2, 7, 15, or 30 days after repair of 1-h, 720° torsion. Blood flow is severely reduced during torsion but returns toward or to normal after torsion repair.

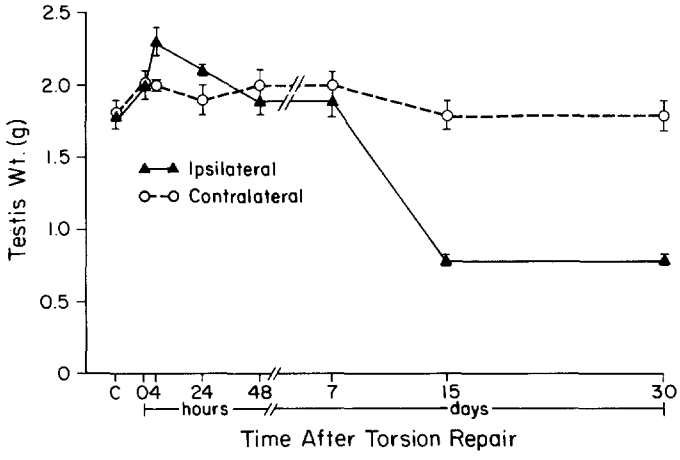

FIG. 3. Testis weight in control animals; in those experiencing 1-h, 720° testicular torsion; and in those examined 4 h, 24 h, or 2, 7, 15, or 30 days after repair of 1-h, 720° torsion. Ipsilateral testis weights declined significantly by 15 days after torsion repair. Contralateral testis weights are not affected.

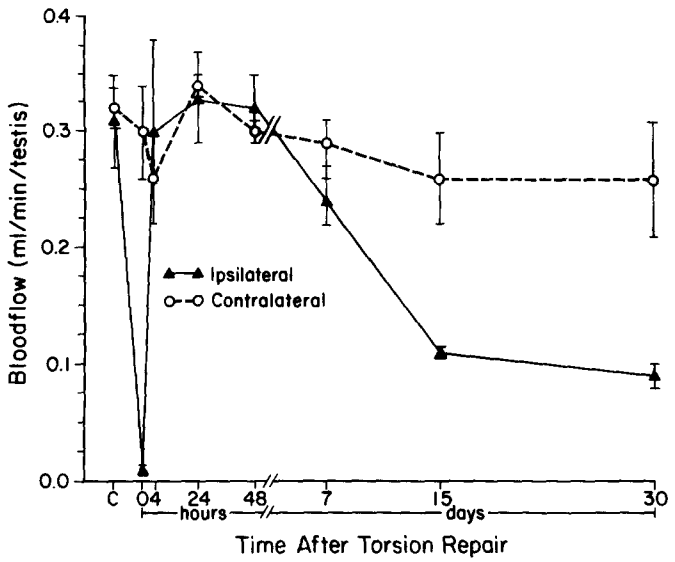

FIG. 4. Total-testis blood flow (ml/min/testis) in control animals; in those experiencing 1-h, 720° torsion; and in those examined 4 h, 24 h, or 2, 7, 15, or 30 days after repair of 1-h, 720° torsion. Ipsilateral, total testis blood flow declined significantly by 15 days after repair of torsion. Contralateral values were not significantly affected.

various groups were not significantly different except in two instances. Liver blood flow, typically 20–30 ml/min/100 g tissue in these rats, was elevated to 57 ml/min in the 1/24 group; and prostatic flow values, typically 15–20 ml/min/100 g tissue in these rats, was reduced to 8 ml/min in the 1/48 group. Since these changes stood alone and were not associated with any other trends or apparent pathologies, they were not used to eliminate groups from the study.

DISCUSSION

Testicular torsion results in testicular ischemia, in both the clinical [2] and the experimental [8] setting. Early investigations of testicular ischemia induced by testicular artery ligation in the rat demonstrated that increasing periods of ischemia from minutes to hours resulted in progressive damage to the testis, with significant, long-lasting effects on spermatogenesis occurring as a result of 1 h of ischemia [14, 15]. Minor, transient effects on the seminiferous epithelium were found after time periods as short as 10 min [14]. As one would anticipate, increasing the duration of testicular torsion is also clearly associated with increasing severity of testicular damage [2, 8, 16].

In previous investigations of experimental torsion, sham, or "0 time," 720° torsion (twist with immediate repair) of the rat testis had no effect on ipsilateral testis weight or cauda epididymal sperm concentrations at several time periods after repair of torsion [8, 17], but increasing the time of torsion to 1, 2, and 4 h caused a progressive loss in testis weight measured 30 days after torsion repair and caused an almost total loss of spermatogenesis at both 30 and 60 days after torsion repair. Similarly, in the present work it

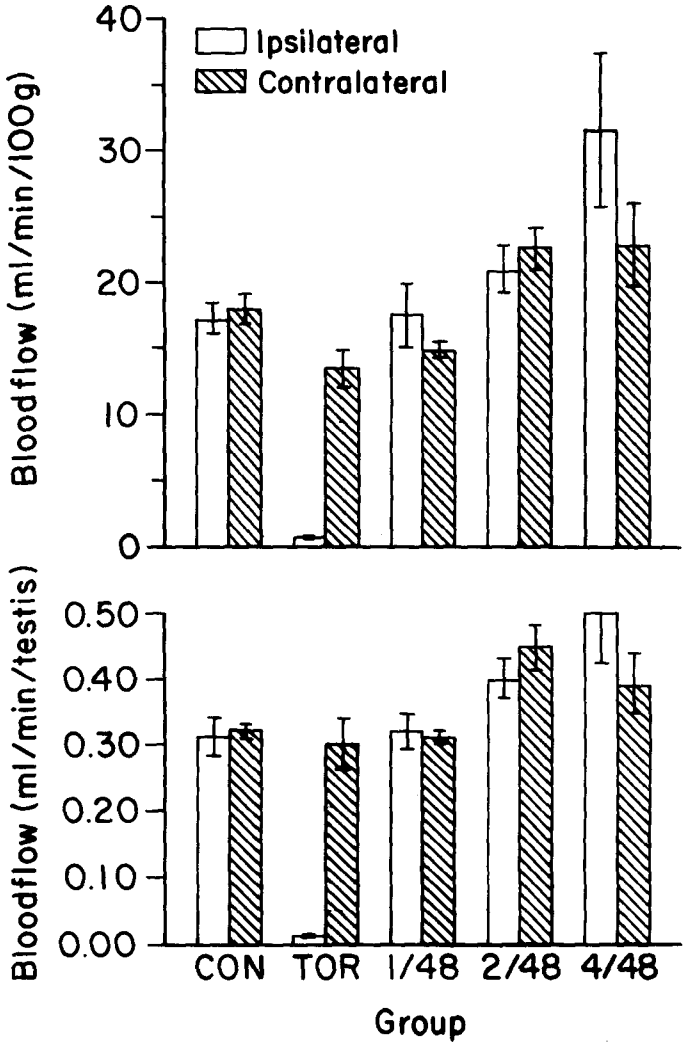

FIG. 5. Testicular blood flow in control animals; in those experiencing 1-h, 720° torsion; and in those examined 48 h after repair of 1-h, 2-h, or 4-h, 720° testicular torsion. Increasing the time of torsion did not impair the return of testicular blood flow as measured 48 h after torsion repair.

was demonstrated that 1-h 720° torsion significantly reduced ipsilateral testis weight and caused a significant decrease in ipsilateral spermatogenesis as demonstrated by histology (Fig. 1) and by DSP (Table 1). Additionally, the microvasculature of all testes subjected to 1-h torsion had a qualitatively abnormal appearance at both 2 and 30 days after torsion repair (see *Results*).

In contrast, 0.5-h 720° torsion caused no significant reduction in ipsilateral testis weights or ipsilateral DSP 30 days after torsion repair, even though all these testes appeared slightly hyperemic 2 days after torsion repair and half were still judged to be slightly hyperemic 30 days after torsion. It was concluded that the critical time of vascular-occluding torsion after which severe testicular damage occurs in the rat model is between 30 min and 1 h. For subsequent studies in the rat, 1 h was selected as the minimum effective time of induction for significant, persistent testicular damage.

Neither testis weight nor DSP was significantly affected by 1.0-h torsion two days after torsion repair, possibly because reactive edema compensated for any acute loss of tissue weight and not enough time had passed to allow testicular passage of sloughed condensed spermatids and spermatozoa.

In the blood flow experiments, testicular blood flow during the torsion period was reduced to 3–4% of control values. Unuler et al. [18], using rabbits and ^{133}Xe-clearance as a measure of testicular blood flow, found that 720° torsion reduced blood flow to 7–16% of control values; thus, while it is clear that experimental, 720° torsion severely restricts testicular blood flow, there is some variation, both within and between species, in the absolute amount of persistent blood flow to the testis. It is unclear whether there is a different biological consequence between there being no flow during torsion and there being essentially no flow during torsion.

Average ipsilateral testicular blood flow values had largely returned toward control values within 4 h of relief of torsion (Fig. 2), but individual testes varied in their blood flow rate at 4 h after torsion. Approximately half the testes demonstrated normal flow values and approximately half evidenced less flow. Tzika et al. [9] reported a similar variability in a study using different techniques to examine testicular blood flow 3 h after similar torsion. We speculate that the return to control blood flow values occurs gradually and that some testes are more retarded in return flow at 4 h after torsion repair than are others.

Ipsilateral testicular blood flow values had returned to control values by 24 h after torsion repair (Figs. 2 and 4). These data are consistent with previous data from this laboratory [8]. Other descriptions of return flow at or later than 24 h after relief of torsion have not been found. Generally, ipsilateral testicular blood flow (per gram of testicular parenchyma) was not significantly altered from control or contralateral values at 2, 7, or 15 days after torsion repair; but the effect of torsion on blood flow 30 days after repair is not clear. As in the 2-, 7-, and 15-day groups, ipsilateral testicular blood flow values 30 days after torsion repair were not significantly reduced from contralateral values; but in the 30-day group, ipsilateral blood flow values were significantly reduced relative to control values (Fig. 2). Considering all the data across the times studied, we suggest the interpretation that the effects of 1-h 720° torsion on blood flow per gram of testis is limited to a tendency toward reduced flow by 30 days after torsion repair. Since spermatogenesis is clearly disrupted before this time (Fig. 1, Table 1), resulting in significantly reduced testis weights by 15 days after torsion repair (Fig. 3), we conclude that the slight reduction of blood flow detected at 30 days after repair of torsion is a result, not a cause, of the destruction of the normal seminiferous epithelium. Of course, total flow to the ipsilateral testis is significantly reduced in parallel with the loss of testis weight (Figs. 3 and 4).

Increasing the time of torsion did not impair the return of testicular blood flow as measured 48 h after repair of torsion (Fig. 5). This is in contrast to previous findings from this lab; in that study, testes observed 1 and 2 h after repair of 4-h 720° torsion were qualitatively judged to be clearly infarcted [8], and the histology of those testes was progressively worse than that of testes exposed to only 1-or 2-h torsion [8, 17].

In a further examination of this point in five testes, the qualitative appearance of ischemia induced by 4-h 720° torsion was observed, as was the degree of ischemic appearance 5 min, 30 min, 1 h, and 2 h after repair of torsion. Four of the five testes were clearly and markedly ischemic with all vessels being dark purple in color after the 4-h torsion, but one testis was less markedly ischemic with oxygenated blood evident in the main trunk of the testicular artery. One of the ischemic testes evidenced little return blood flow by 2 hr after torsion repair, but the other four testes did qualitatively exhibit return flow in large vessels after torsion repair. Nevertheless, examination under the operating scope revealed focal areas of no perfusion through the microvasculature and recalled the "patchy" appearance of the post-torsion testis vasculature mentioned earlier. It is likely, therefore, that return of blood flow to normal values after 4-h torsion, and perhaps after other long-term but non-infarcting torsion, is relatively protracted. This flow is still very sluggish within 2 h of torsion repair, is more vigorous 4 h after torsion repair (Fig. 2), and is not back to normal until some time between 4 h and 24 h. We conclude that in the rat model, testes that appear to be dark and infarcted at time of operation often have significant return blood flow several hours later and can have normal blood flow rates within 48 h of torsion repair (Fig. 5). This may be true in other species as well.

The finding that testicular blood flow in the 4/48 group was actually higher than in the other groups is perhaps artifactitious due to the high cardiac output in that group, but this does not affect the observation that at 48 h after the repair of 4-h torsion these testes were not infarcted.

Interestingly, neither contralateral testis gross appearance, histology, testis weight, DSP, nor testicular blood flow was ever significantly affected by ipsilateral torsion. This is consistent with previous results from this lab [8, 17] as well as many others (see [17] for review, [19, 20]), but is in contrast with the oft-repeated notion that contralateral effects result from ipsilateral torsion (see [21] for review, [22, 23]).

Cosentino and colleagues have presented both early [24, 25] and later reports [22, 23] of the contralateral effects of ipsilateral torsion, and, importantly, theirs is one of the few laboratories in which repeated experiments have been performed over many years. Most of their work has been on 35-day-old, prepubertal rats [22–25], but in a recent study [26] they expanded their studies to younger and older animals. The investigators also show no contralateral effect in adult animals and limit their claim of contralateral effects

to rats in a narrow age window (>35 days, <50 days). Importantly, these investigators see significant fertility effects only when the torted testis is left untreated for periods far longer (9–12 h [24, 26]) than that required to induce ipsilateral testicular necrosis [8, 14, 15]. This is an important consideration, since immunological phenomena have been called upon as the rationale for contralateral effects of ipsilateral torsion [21, 23]. Any ipsilateral, testis-related immune response should be activated by necrosis-inducing 1–4 h torsion, but this apparently does not occur to any significant degree. Cosentino et al. [25] did report some changes in qualitative histological scores of contralateral testis after a period of 3-h torsion, but these changes were apparently not sufficient to affect fertility and their meaning is unclear.

Contralateral effects of ipsilateral torsion, although claimed by various investigators to occur in both adult and prepubertal animals, are seen by others in neither adult nor perpubertal animals (see [21] for review). In the present study in adult animals and previous reports from this lab [8, 17], only normal physiology and function have been found in contralateral testes. One would appreciate seeing evidence that contralateral effects, when found, especially in young animals, are not due to untoward effects of experimental protocols inducing long periods (9–12 h) of torsion.

The important findings of the present study are that testicular blood flow (ml/g/min) generally returns to control values in the non-infarcted testis after torsion repair. This was certainly not necessarily to be expected, and the results lead to further questions regarding cellular responses to testicular ischemia followed by reperfusion. Since the seminiferous epithelium is severely disrupted in testes reperfusing after torsion, the primary injury must occur during the ischemia or upon reperfusion. Reperfusion injury (cell damage due to reactive oxygen species) has been intensely studied in many tissues [27], but has received little attention in the testis [7, 28]. Investigations in this area, as well as investigation of specific Leydig cell and Sertoli cell responses to graded periods of torsion, are currently underway in our laboratory.

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