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# Torsion of only spermatic cord in the absence of testis and/or epididymis results in contralateral testicular hypoxia

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**Abstract** Unilateral spermatic cord torsion in the presence or absence of ipsilateral testis causes hypoxia in the contralateral testis. An experimental study was conducted to find the most important structure that causes contralateral testicular hypoxia following ipsilateral twisting. In five groups each consisting of 10 rats sham operations, epididymoorchiectomy, spermatic cord torsion, spermatic cord torsion following subepididymal orchiectomy or spermatic cord torsion following epididymoorchiectomy were performed. Lactic acid, hypoxanthine and thiobarbituric acid reactive products of lipid peroxidation (TBAR) were determined in the contralateral testis. While lactic acid, hypoxanthine and TBAR values did not differ significantly following sham and epididymoorchiectomy procedures, evaluation of other groups revealed significantly increased values compared with sham and epididymoorchiectomy groups. Since torsion of only spermatic cord and testicular vasculature causes contralateral testicular hypoxia, testis and epididymis do not seem to be mandatory for occurrence of contralateral testicular hypoxia. Testicular artery under distress seems to be the most important structure that results in contralateral testicular hypoxia following torsion.

**Key words** Spermatic cord torsion · Testis · Ischemia

## Introduction

Although some authors disagree, it is generally accepted that unilateral spermatic cord torsion may cause contralateral testicular damage and result in diminished fertility [5, 26]. However, the mechanism of contralateral testicular injury is still controversial [5].

Recent studies have revealed a decrease in contralateral testicular blood flow [15, 16, 20, 22] and an increase in biochemical indicators of tissue hypoxia during ipsilateral spermatic cord torsion [3]. Those changes have been reversed, and fecundity has been improved by chemical sympathectomy prior to torsion [10, 11]. Torsion of only epididymis and spermatic cord following subepididymal orchiectomy has shown that the elevation of indicators of tissue hypoxia in the contralateral testis and histological deterioration have not mandated the presence of ipsilateral testis [4, 9].

Those findings have suggested that contralateral testicular injury following ipsilateral spermatic cord torsion may be a hypoxic damage through a reflexive decrease in blood flow. However, the most important structure that causes a decrease in contralateral testicular blood flow following twisting remains to be determined.

Therefore an experimental study was planned to find, through the biochemical indicators of tissue hypoxia, the most important structure that causes contralateral testicular hypoxia following twisting.

## Materials and methods

Fifty male adult albino rats were used in this experiment. Rats were randomly divided into five groups each containing ten rats. The experiment followed the "Principles of Laboratory animal care" of the NIH. The surgical procedures were performed under ether anesthesia employing a sterile technique. Left-sided inguinoscrotal incisions were used, and clockwise torsion of 720° was applied in each group.

Group 1: Sham operation was performed. The testis was exposed and a 4/0 atraumatic silk suture was placed through the tunica albuginea.

Group 2: Epididymoorchiectomy was performed.

Group 3: Testicular torsion was performed, and a 4/0 atraumatic silk suture was placed to maintain torsion.

Group 4: Subepididymal orchiectomy was performed. Remaining epididymis and spermatic cord were twisted and a 4/0 atraumatic silk suture was placed to maintain twisting.

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Group 5: Epididymoorchiectomy was performed. Remaining spermatic cord and vascular structures were twisted, and a 4/0 atraumatic silk suture was placed to maintain twisting.

After harvesting 24 h later, all right testes were placed in glass bottles with rubber caps, labeled and stored in deep freeze. Lactic acid, hypoxanthine and thiobarbituric acid reactive products of lipid peroxidation (TBAR), which are indicators of tissue hypoxia, were determined in testicular tissue according to biochemical methods published elsewhere [4]. One-way analysis of variance was used for statistical analysis of the data among groups. Values of $P < 0.05$ were accepted as statistically significant.

## Results

Intratesticular lactic acid, hypoxanthine and TBAR values following sham and epididymoorchiectomy operations did not differ significantly ($P > 0.05$) (Table 1). Lactic acid, hypoxanthine and TBAR values were significantly elevated following spermatic cord torsion in the presence of testis and epididymis compared with sham and epididymoorchiectomy operations ($P < 0.01$) (Table 1). Torsion of epididymis and spermatic cord in the absence of testis also revealed increased levels of lactic acid, hypoxanthine and TBAR values ($P < 0.01$) (Table 1).

Torsion of spermatic cord only in the absence of testis and epididymis, similarly resulted in elevated levels of lactic acid ($P < 0.05$), hypoxanthine ($P < 0.01$), and TBAR ($P < 0.01$) values in the contralateral testes compared with sham and epididymoorchiectomy operations (Table 1). While torsion in the presence of testis and epididymis caused significantly elevated TBAR values compared with torsion in the absence of testis and epididymis ($P < 0.01$), lactic acid and hypoxanthine levels of these groups were not significant ($P > 0.05$) (Table 1).

Torsion following orchiectomy caused significantly elevated levels of hypoxanthine compared with torsion following epididymoorchiectomy ($P < 0.05$). However, levels of lactic acid and TBAR did not differ between those two groups ($P > 0.05$) (Table 1).

## Discussion

It is known that levels of lactic acid, hypoxanthine and lipid peroxides increase in hypoxic tissues [1, 2, 8, 21]. The present study has also revealed increases in the levels of lactic acid, hypoxanthine and TBAR values of contralateral testes following unilateral spermatic cord torsion compared with control operations. While epididymoorchiectomy did not cause a difference in the levels of lactic acid, hypoxanthine and TBAR values of contralateral testes compared with sham operations, torsion following subepididymal orchiectomy or epididymoorchiectomy resulted in the elevation of biochemical indicators of tissue hypoxia in the contralateral testis.

The tissue hypoxia in the contralateral testis following torsion in the presence or absence of testis and/or epididymis has been significant. Therefore the presence of only spermatic cord and adherent testicular vasculature undergoing torsion in the ipsilateral side seems to be sufficient for the occurrence of hypoxia in the contralateral testis.

Since hypoxia reflects a depressed tissue perfusion, the present study also suggested a decrease in contralateral testicular blood flow. Among the mechanisms that decrease blood flow, vasospasm preceded by sympathetic activation is most likely. However, the exact role of the sympathetic system remains to be determined, and catecholamine levels in contralateral testis are currently under evaluation in our labaratory. If their role is exactly defined, further studies including sympathectomy, alpha receptor blockage or peripheral vasodilation are required to minimize the hypoxia. On the other hand the present study, did not address the biological effects of torsion of only spermatic cord and adherent vasculature upon the contralateral testis, and this subject is also under evaluation through DNA flow cytometry in our laboratory.

It is known that not only ipsilateral spermatic cord torsion, but also undescended testes [19], incarcerated inguinal hernia [23–25], varicocele [14] and obstruction of the vas deferens [17] may cause contralateral testicular deterioration. Therefore a common pathway of contralateral testicular deterioration instead of different pathways for various conditions seems to be more feasible. Contralateral testicular injury following experimental spermatic cord torsion makes less likely the proposals of preexisting congenital abnormality and incidental occurrence of other conditions such as varicocele and subclinical attacks of contralateral torsion [5]. The occurrence of contralateral testicular hypoxia and histological injury in the absence of testicular and spermatogenic material makes the place of autoimmunity questionable [9]. The present study suggests that contralateral testicular hypoxia may develop following spermatic cord torsion even in the absence of testis and

**Table 1** Lactic acid, hypoxanthine and thiobarbituric acid (TBAR) values in the contralateral testis (mean ± 1SD). *T*, testis, *E*, epididymis, *S*, spermatic cord

| Groups ($n = 10$) | Lactic acid (μmol/g wet tissue) | Hypoxanthine (μmol/g wet tissue) | TBAR (nmol/g wet tissue) |
|---|---|---|---|
| Control | 2.6840 ± 0.4989 | 24.4700 ± 5.3655 | 68.2800 ± 7.4474 |
| Epididymoorchiectomy | 2.8260 ± 0.6234 | 26.8300 ± 6.4276 | 67.4960 ± 7.8276 |
| Torsion (T+E+S) | 3.9720 ± 1.0629 | 47.6300 ± 7.0350 | 98.7800 ± 7.9127 |
| Torsion (E+S) | 3.9973 ± 0.6430 | 37.8273 ± 5.6161 | 80.4273 ± 5.5939 |
| Torsion (S) | 3.6350 ± 1.1080 | 44.5500 ± 7.6531 | 77.7700 ± 20.6083 |

epididymis. Tissue hypoxia in the contralateral testis has also been suggested in other pathologies affecting ipsilateral testis such as failure to descend [12, 13], varicocele [7] and vas deferens obstruction [6]. Therefore hypoxia seems to be the common pathway of contralateral testicular deterioration following pathologies damaging the ipsilateral testis. Attempts at restoring the contralateral testicular blood flow would have beneficial effects upon contralateral testis.

Ligation and transection of the ipsilateral testicular artery does not cause contralateral testicular deterioration [18]. However, in our experimental design twisting spermatic cord and testicular vasculature following epididymoorchiectomy resulted in contralateral testicular hypoxia. This suggests that an additional distressing factor on the testicular artery is essential to initiate afferent impulses to cause contralateral testicular hypoxia. Therefore, a testicular artery under distress seems to be mandatory for the occurrence of contralateral testicular hypoxia.

Contralateral testis undergoes hypoxia following torsion of only spermatic cord and testicular vasculature in the absence of testis and epididymis. A testicular artery under additional distress seems to play the most important role in the occurrence of contralateral testicular hypoxia encountered during unilateral testicular torsion.

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