UNILATERAL CRYPTOCHIDISM; SUBSEQUENT EFFECTS ON FERTILITY

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Summary. Seminal studies carried out in thirty-four cases of untreated, post-pubertal, unilateral cryptorchidism have revealed a high incidence of sterility and severely impaired fertility, while the seminal findings of six cases of retractile testis showed no diminution of fertility.

Testicular biopsies, carried out in two retractile testes, confirmed their normality, while similar studies of seven pubertal retained testes and nine pubertal ectopic testes showed that spermatogenesis is more severely damaged in the former than it is in the latter. Further biopsy studies of eight contralateral gonads in unilateral cryptorchids have disclosed an unexpectedly high incidence of spermatogenic arrest in the so-called normally descended opposite testis.

INTRODUCTION

Although the imperfection of an undescended testis has been recognized since the time of John Hunter (1837), controversy still exists as to whether maldescent is the cause of this imperfection or the result of it. As long ago as 1891, Piana found that experimental cryptorchidism in rats resulted in the testes coming to resemble those of congenital cryptorchidism in horses. Moore & Oslund (1924) confirmed these histological changes in sheep, and Wangensteen (1927) showed that, in man, retention of the testes in the inguinal canal for a few years after puberty resulted in cessation of spermatogenesis. The bulk of the published work on unilateral maldescent has been concerned with the histological changes in the offending gonad; little or no attention has been paid to the histology of the so-called ‘normal’ opposite testis which has descended into the scrotum.

The association of sterility with post-pubertal bilateral cryptorchidism is widely accepted, but a review of the literature shows that few authors seem to appreciate the high incidence of sterility and severely impaired fertility that is associated with unilateral maldescent in the post-pubertal male. Recent studies of the histology of maldescended testes and their opposite descended partners (Scott, 1960a) suggest that this is due to a high incidence of tubular damage in the contralateral scrotal gonad. This paper sets out to confirm these findings, and to demonstrate that there appears to be a difference in the potential fertility of retained and ectopic testes at puberty.
Unilateral cryptorchidism

TYPES OF MALDESCENT

Retained testes are immature gonads that lie intra-abdominally or in the inguinal canal. They are frequently malformed when exposed at operation, and are commonly associated with abnormalities of the epididymis. They can seldom be palpated on clinical examination and cannot be manipulated into the scrotum.

Ectopic testes are gonads that have descended normally through the superficial inguinal ring but have been directed away from the scrotum by fusion of Scarpa’s fascia with the symphysis pubis. These testes are rarely abnormal when exposed at operation; they can generally be palpated in the line of the inguinal canal, and they cannot be manipulated into the scrotum.

Retractile testes are normal gonads that spend much of their life in the upper scrotum or in the superficial inguinal pouch (Browne, 1933), but they can be manually replaced at the lower pole of the scrotum, if gentle traction is used to overcome the contraction of the cremasteric and dartos muscles.

INCIDENCE OF MALDESCENT

There is wide divergence of opinion as to the incidence of maldescent at birth. Deming (1952) puts the figure at 1%, Scorer (1956) at 3.4%, and Counsellor (1933) at 10%. The incidence is considerably higher in premature infants than in those born at full-term (Hofstatter, 1912). Fifty per cent of cryptorchid testes descend within the first month of life, but Scorer (1955) considers that complete descent will not occur after the third month. Aird (1949) considers that spontaneous descent is unlikely after 1 year, and Grey Turner (1937) puts the ‘deadline’ at 3 years. On the other hand, Williams (1936) found that between the ages of 11 and 17 years, spontaneous descent occurred in 87% of maldescended testes. The incidence of cryptorchidism in adult life, as judged from examination of army recruits (McNab, 1955), appears to be in the region of two per thousand.

CLINICAL MATERIAL

This study was undertaken on thirty-four cases of post-pubertal, unilateral cryptorchidism and six cases of retractile testis attending the Male Subfertility Clinic of the Western Infirmary, Glasgow, together with sixteen cryptorchids attending the Urology Department of the same hospital for orchidopexy.

METHOD OF STUDY

Each of the forty cases attending the Subfertility Clinic had the type of maldescent personally appraised by the author who also performed seminal analyses on two or more samples of each patient’s semen within 5 hr of ejaculation. All specimens were produced by coitus interruptus after 3 days abstinence from intercourse, and the specimens were collected in clean glass receptacles, free from rubber attachments. The figures referred to in the text represent an average of two or more readings.

Twenty-four testes were biopsied at the time of orchidopexy, and biopsies
were also performed on two retractile testes. All the biopsy material was immediately immersed in Davidson's solution, in preference to Bouin's solution, and they were studied by one of two histologists who have, for several years, taken a special interest in testicular pathology.

RESULTS

SEMINAL STUDIES

No significant changes were noted in either sperm morphology or ejaculate volume and, unlike varicocele (Scott, 1958, 1960b), there was no apparent effect on sperm motility. The sperm counts of the thirty-four cases of post-pubertal unilateral maldescent are shown in Table 1. The seminal findings of six cases with retractile testes are shown in Table 2.

| Table 1 | SPERM COUNTS OF THIRTY-FOUR UNILATERAL CRYPTORCHIDS |
|---------------------------------------------|
| **Sperm count in millions/ml** | **No. cases** |
| Over 20 | 10 |
| 11 to 20 | 4 |
| 6 to 10 | 2 |
| 1 to 5 | 3 |
| Under 1 | 8 |
| Azoospermia | 7 |

| Table 2 | SEMINAL FINDINGS IN SIX MEN WITH RETRACTILE TESTES |
|---------------------------------------------|
| **Case No.** | **Sperm count in millions/ml** | **Sperm motility** |
| 1 | 145 | Poor |
| 2 | 61 | Good |
| 3 | 49 | Good |
| 4 | 48 | Good |
| 5 | 22 | Good |
| 6 | 21 | Poor |

HISTOLOGICAL STUDIES

For convenience of analysis, the biopsied testes were divided into four study groups as follows:

**Group 1: Retractile testes.** This group was represented by two testes; normal spermatogenesis was present in both of them.

**Group 2: Ectopic testes.** This group consisted of nine cases, representing the ectopic testis in the pubertal phase (Table 3). Leydig cells were either absent or rare in four of the testes, although, in a previous study (Scott, 1960a), I found that Leydig cells were diffusely increased in both ectopic and retained testes after puberty. Spermatogenesis was completely absent in three cases and was
arrested at spermatogonia level in two, but four cases went as far as the spermatocyte level.

**Table 3**

**BIOPSY FINDINGS IN ECTOPIC TESTES AT PUBERTY**

<table>
<thead>
<tr>
<th>Age in years</th>
<th>Level of spermatogenesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Arrested at spermatogonia level</td>
</tr>
<tr>
<td>11</td>
<td>Sertoli cells only</td>
</tr>
<tr>
<td>12</td>
<td>Sertoli cells only</td>
</tr>
<tr>
<td>13</td>
<td>Sertoli cells only</td>
</tr>
<tr>
<td>13</td>
<td>Arrested at spermatogonia level</td>
</tr>
<tr>
<td>13</td>
<td>Arrested at primary spermatocyte level</td>
</tr>
<tr>
<td>14</td>
<td>Arrested at primary spermatocyte level</td>
</tr>
<tr>
<td>16</td>
<td>Arrested at secondary spermatocyte level</td>
</tr>
<tr>
<td>17</td>
<td>Arrested at secondary spermatocyte level</td>
</tr>
</tbody>
</table>

**Group 3: Retained testes.** This group consisted of seven cases, representing the retained testis in the pubertal phase (Table 4). Leydig cells were again either absent or rare in most cases. Spermatogenic arrest at spermatogonia level was present in three testes; in the remaining four, spermatogenesis was completely absent.

**Table 4**

**BIOPSY FINDINGS IN RETAINED TESTES AT PUBERTY**

<table>
<thead>
<tr>
<th>Age in years</th>
<th>Level of spermatogenesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Sertoli cells only</td>
</tr>
<tr>
<td>13</td>
<td>Arrested at spermatogonia level</td>
</tr>
<tr>
<td>13</td>
<td>Arrested at spermatogonia level</td>
</tr>
<tr>
<td>14</td>
<td>Sertoli cells only</td>
</tr>
<tr>
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<td>Sertoli cells only</td>
</tr>
<tr>
<td>14</td>
<td>Sertoli cells only</td>
</tr>
<tr>
<td>16</td>
<td>Arrested at spermatogonia level</td>
</tr>
</tbody>
</table>

**Group 4: Contralateral testes.** This group consisted of eight cases, representing the histological picture of the normally descended contralateral scrotal testis

**Table 5**

**BIOPSY FINDINGS IN THE CONTRALATERAL SCROTAL TESTIS IN CASES OF UNILATERAL CRYPTORCHIDISM**

<table>
<thead>
<tr>
<th>Age in years</th>
<th>Level of spermatogenesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>Arrested at spermatogonia level</td>
</tr>
<tr>
<td>16</td>
<td>Arrested at spermatogonia level</td>
</tr>
<tr>
<td>26</td>
<td>Mature spermatozoa</td>
</tr>
<tr>
<td>27</td>
<td>Mature spermatozoa</td>
</tr>
<tr>
<td>30</td>
<td>Mature spermatozoa</td>
</tr>
<tr>
<td>31</td>
<td>Sertoli cells only</td>
</tr>
<tr>
<td>32</td>
<td>Arrested at spermatid level</td>
</tr>
<tr>
<td>32</td>
<td>Arrested at spermatid level</td>
</tr>
</tbody>
</table>

(Table 5). In each case, the other testis was maldescended. Mature spermatozoa were only detected in three testes. Spermatogenic arrest, at various levels, was
present in four testes and, in one case, the scrotal testis showed complete absence of spermatogenesis.

DISCUSSION

Of the thirty-four untreated unilateral cryptorchids who underwent seminal studies, 21% were undoubtedly sterile due to complete azoospermia, while a further 23% were almost certainly sterile as they had only occasional non-motile malformed sperms. Nine per cent had sperm counts under 5 million/ml, and only 29% could be considered fertile. This high incidence of impaired fertility associated with post-pubertal unilateral cryptorchidism is not generally appreciated, but was more than substantiated by Mack (1953) who found that, of thirty-seven cases, 59.4% were either sterile or very severely impaired. In a collected series of seventy-two published cases of untreated unilateral cryptorchidism (Hansen, 1949; Mack, 1953), 41% were considered to be either sterile or very severely impaired.

Of the six cases that were referred to my Clinic as ‘undescended’ or ‘ectopic’ testes but which, on closer inspection, proved to be high retractile testes, five were fertile and only one was considered slightly subfertile due to a count of 21 million/ml being associated with poor motility. It is obvious, therefore, that great care must be taken to distinguish these cases from true maldescent.

Charny, Conston & Meranze (1952) presented an excellent paper on a histological study of the testes from birth to maturity and showed that, in the maturation phase — starting at the age of 10 years and going on to the end of puberty — active spermatogenesis was usually present, and mature spermatozoa were frequently detectable. All my histological studies were from this age group and it is obvious that both retained and ectopic testes show histological evidence of retarded spermatogenesis in the pubertal phase.

I have always been impressed by the fact that, when exposed at orchidopexy, the majority of ectopic testes are well developed anatomically, in striking contrast to the frequency with which abnormalities of the testis and its associated epididymis are encountered in exploration of retained testes. The difference between these two types of maldescent is further borne out by my biopsy findings during the pubertal phase. Six of the nine ectopic testes showed evidence of spermatogenesis in a high percentage of the tubules examined, whereas it was only detected in very occasional tubules in two of the seven retained gonads. Furthermore, spermatogenesis frequently went as far as the spermatoocyte level in the ‘ectopic’ group, but never went further than the spermatogonia level in the ‘retained’ group.

The biopsy findings in the contralateral scrotal testes may shed some light on the aetiology of the high incidence of impaired fertility in unilateral cryptorchidism because although, clinically, most of these testes appeared to be normal in size, consistency and position, only three out of eight had full spermatogenesis. Spermatogenic arrest at various levels was present in four testes, one showed complete absence of spermatogenesis, and in a further four unilaterally cryptorchid men — whose fertility depended solely on the opposite testis as the maldescended gonad had been surgically removed — two were...
sterile, one had a sperm count of 2 million/ml, and one had a count of 11 million/ml.

It would therefore seem that the normally descended opposite testis is more frequently abnormal, in this condition, than has hitherto been supposed.

CONCLUSIONS

Although little interest has previously been shown in the fertility of unilateral cryptorchids, the present studies show that this condition is associated with a high incidence of sterility and severely impaired fertility. A possible explanation for this finding has been brought to light by the testicular biopsy studies of the contralateral gonads which showed that there is a hitherto unexpectedly high incidence of spermatogenic arrest in the so-called, normally descended, opposite testes. While supporting a common belief that maldescent is the result of testicular imperfection rather than the cause of it, these studies also suggest that there may be an inherent bilateral defect which clinical examination fails to detect.

The importance of distinguishing retractile testes from examples of true maldescent is once again stressed, as the present seminal and histological studies of retractile testes have confirmed their normality in terms of fertility.

The biopsy studies of ectopic and retained testes in their pubertal phases have shown that spermatogenesis is more advanced, and much closer to normal, in the former than it is in the latter. In my opinion, this finding, together with the relatively normal appearance of most ectopic testes at surgical exploration, may explain the better post-operative fertility results which, in my experience, follow pre-pubertal orchidopexy of ectopic testis than those that follow scrotal placement of retained gonads.

ACKNOWLEDGMENTS

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