SEMEN ANALYSIS AS A PARAMETER OF MALE FERTILITY IN 266 INFERTILE COUPLES

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One of the major advances in the field of infertility in recent years has been the inclusion of the male partner in the routine investigation of a barren couple. Failure to conceive is due to deficiency in the husband in about 50% of infertile couples (Murphy and Torrano, 1965). Consequently, with the advent of new diagnostic means and methods, the male partners can now be adequately studied, and their condition accurately diagnosed.

The work-up of the male partner in the Gynaecologist's office, usually begins with a semen analysis with all its parameters. It is well known that speculations on fertility potential of the male based on semen analysis are dangerous. However, seminal study as a parameter of male fertility is the decisive factor in determining the necessity for further detailed study of the male partner. It accurately establishes the function of various glands that contribute to the formation of semen, and to some extent the excretory function of the male reproductive system.

During the period from August, 1975 to October, 1976, 266 couples who complained of childlessness were investigated in this centre. The duration of infertility ranged from 1 year and 6 months to 20 years. All the male partners submitted their semen for analysis. In all cases, the semen samples were studied and evaluated for all the parameters by the authors themselves. The observations of the authors, correlated with the physical findings, are presented in this report.

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Method of Investigation

The initial work-up of the infertile couple in our institution included a detailed history, a complete physical examination and semen analysis. If the semen sample proved to be defective, the analysis was repeated. Based on the seminal cytology and physical findings, the male was further evaluated with testicular biopsy and endocrine studies.

Collection of Semen: Semen was best obtained by masturbation. The collection was done after a period of sexual abstinence for 3 to 5 days. The semen samples were delivered for evaluation within 1-2 hours of ejacuation. The patient was instructed to collect the entire quantity of semen in a bottle.

Semen Analysis: The colour, odour and liquifaction were noted first and then the quantity of semen was measured. A fresh smear was made on a clean glass slide to study the sperm density, motility, morphology and the presence of immature cells. Sperm count was done by the haemocytometer method with reasonable precision. The following terminologies were employed in classification of the seminal samples:

Azoospermia: No sperms in semen.

Severe Oligospermia: Less than 20 million spermatozoa/ml.

Mild Oligospermia: 20 to 60 million spermatozoa/ml.

Stress pattern (of sperm morphology): presence of tapering and amorphous cells and plenty of exfoliated immature cells of the germinal line in the ejaculate.

Necrospermia: all spermatozoa found dead.

Observations

Age: The male partners were between the age group of 22 to 48 years (Table I); 50% of them were in the range of 30 to 39 years of age.

TABLE I

Age Group of the 266 Male Partners

No.	Age group	No. of cases
1.	Below 20 years	Nil
2.	20 to 29 years	83
3.	30 to 39 years	133
4.	40 to 49 years	50
	Total:	266

Sperm count: (Table II). Of the total number of 266 subjects, 62.3% (162 cases) revealed poor semen quality which could be accountable for the infertility. Forty-eight men had mild oligospermia with a mean count of 37.5 million per ml., in whom conception could be expected if their wives were found to be normal. Fifty-six men had good semen quality with a sperm count ranging from 61.4 million to 180 million per ml.

Severe oligospermia: Of the 162 men with poor semen quality, approximately 50% (79 men) had severe oligospermia with a sperm count below 20 million. Percentage of motility decreased markedly in severe oligospermia, with 75% showing poor motility. There was obvious increase in the abnormal forms and immature cells. 83% had normal sized testicles, majority of which were soft in consistency, whereas only 14% had small atrophic testicles (Table III). More than 75%

TABLE II

Semen Analysis of the 266 Subjects

No.	Seminal Cytology	No. of cases	Sperm count in million/ml	
			Mean	Range
1.	Azoospermia	78	—	—
2.	Severe oligospermia	79	6.24	2.00—19.20
3.	Mild oligospermia	48	37.50	21.0—58.00
4.	Normal sperm count	56	106.00	61.40—180.00
5.	Necrospermia	5	34.90	3.00—58.00

TABLE III

Testicular Morphology and Seminal Cytology

Testicular Morphology	No. of cases	Azoos-permia	Severe oligos-permia	Mild oligos-permia	Normal Count	Necro-Spermia
Normal size	208	41	66	43	54	4
Small size	49	35	11	2	–	1
Large size	3	–	1	2	–	–
Cryptorchidism	6	2 (bilateral)	1 (unila-teral)	1 (unila-teral)	2 (unila-teral)	–

(62 patients) had associated varicocele. In the presence of varicocele many of the patients with poor count demonstrated the typical stress pattern of sperm morphology with tapering and amorphous forms and plenty of immature cells, in the seminal pool.

Mild Oligospermia: Forty-eight patients had sperm count ranging from 20 to 60 million per ml., with an average count of 37.5 million per ml. About 90% of them (43 patients) had normal sized testicles and only 2 patients had small testicles. 65% of these men with mild oligospermia had varicocele, and at least one half of them revealed the stress pattern of sperm morphology and poor motility. When these patients were excluded, all the others with mild oligospermia had fairly good sperm motility and morphology.

Normal Sperm Count: There were 56 men with good semen quality, with a count ranging from 61 million to 180 million per ml,, good morphology and motility. All of them had normal sized firm testicles, even though 2 had unilateral cryptorchidism. Varicocele was an incidental finding in 22 subjects (about 39%) with normal seminal cytology.

Necrospermia: In 5 patients sperm motility was totally absent. They had normal sized testicles, and one of them had varicocele. The average sperm count in this group was 34.9 million per ml.

Azoospermia: Among the 266 men whose semen samples were studied, about 30% (78 subjects) were azoospermic; 44% of whom had small atrophic testicles, while 2 had bilateral cryptorchidism. Twenty-nine patients had associated varicocele.

Seminal Cytology in Varicocele: Varicocele was a common physical deformity to be noticed at the time of the clinical examination of the men who attended the infertility clinic. One hundred and forty-four subjects had varicocele (Table IV), the incidence being 77.5% in severe oligospermia patients and 63.3% in mild oligospermia group. 39% with normal semen pattern had varicocele. Ninety-two patients with varicocele had mild to severe degree of oligospermia and of them 77 had the typical stress pattern of sperm morphology and poor sperm motility.

TABLE IV
Seminal Cytology in Varicocele (144 Cases)

Seminal Cytology	No. of cases	Percentage
Azoospermia	29	20.10
Severe oligospermia	62	43.10
Mild oligospermia	30	20.80
Normal sperm count	22	15.30
Necrospermia	1	0.70
Total cases:	144	100.00

Size of the Testes: Semen quality was invariably poor in all the 49 men who had small sized atrophic testicles. Indeed, about 50% of the men with normal sized testicles also had poor semen quality.

Discussion

Farris (1950) defined male fertility in terms of the number of motile sperms in the total ejaculate. He concluded that men having a minimum of 80 million motile sperms in the total ejaculate should be classified as having normal fertility.

Murphy and Torrano (1965) selected "20 million motile sperms per ml." as the dividing line between normal and less than normal fertility. Since the average volume of semen per ejaculation is close to 4 ml., the figure is the same as that of Farris, but the calculation is more simplified. MacLeod (1962), who has perhaps studied critically more semen samples than any one else, demonstrated that sperm count per ml of ejaculate is a better gauge than the total ejaculate volume.

According to MacLeod (1955, 1952, 1962), 2 peaks of male fertility are present; the first level is at 20 million per ml; and the second level is at 60 million per ml. (Above this there is no significant rise in male fertility). He also demonstrated that above a level of 20 million cells per ml., the sperm count by itself was not the most important factor. On the contrary, the potential fertility of any individual above a certain sperm count could not be equated with the sperm count alone. Another conclusion was that there is a far closer relationship between the quality of motility (the rate of forward migration of the cells) and potential or actual fertility. The pregnancy rate rises rapidly to 40% active sperms and flattens off thereafter. Yet another observation was that, while extremes of abnormal sperm may play an important part in certain cases of human male infertility, sperm morphology is less significant than motility. Over 60% normal forms represents a good line of demarcation between a fertile and infertile count.

In our study, about 30% of the men investigated had a sperm count of less than 20 million per ml. (severe oligospermia). Since the count was very low, with a mean value of 6.24 million per ml., the fertility potential was directly related to the count. But other factors, such as reduced motility and abnormal sperm morphology, also contributed to the infertility. Percentage of motile forms decreased markedly when the count was below 20 million per ml. About 75% of the subjects with poor count had less than 40% active sperms in the ejaculate. In these cases there was significant increase in the abnormal forms. Hence, by all the 3 parameters, sperm motility, morphology and count, these men can be grouped as "poor" husbands. Fertility may be anticipated only by further evaluation and proper treatment.

Out of the 266 men, 18% had mild oligospermia, with a range of 21.40 million to 58 million sperms per ml. Fifteen patients in this group had stress pattern of sperm morphology. When these men were excluded, all the others had more than 40% motile sperms and reasonably good sperm morphology. The fertility status in them may be considered as directly proportional to the increased percentage of motile spermatozoa and decreased percentage of abnormal forms, and not much related to the count.

Varicocele, one of the commonest cause of male infertility was detected in 144 subjects. The incidence of this anomaly in infertility practice is variously reported as 26.67% (Rajan, 1976) and 39% (Dubin and Amelar, 1971). The seminal picture has been delineated by MacLeod (1965). Oligospermia of varying degree was noted but more important was the marked impairment of sperm motility as well as stress pattern of sperm morphology, with increase in immature and tapering sperms in the ejaculate. In our series, 62 men (43.10%) had severe oligospermia and 30 men (20.80%) had mild oligospermia. All the severely

oligospermic men and 15 men with mild oligospermia (total of 77 men with varicocele) demonstrated stress pattern in the cytology. One patient with varicocele had necrospermia. 15.30% of men with varicocele had good semen quality. Varicocele ligation was not recommended in those men with normal count because their over-all semen quality was such that conception could be expected if their wives were found to be normal. In those men who were azoospermic the testicular morphology should be studied, before attempting ligation, since varicocele may not be responsible for azoospermia in all cases.

Summary

A count of the number of sperms in the semen samples of 266 childless men showed about 30% to have less than 20 million sperms per ml. of semen. The sperm motility decreased and abnormal sperm cells increased with the severity of oligospermia. Varicocele was found to be the commonest cause of poor semen quality.

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