دراسة تجريبية على الأجهاد الصيفى فى الأرانب

٣- التأثير الكمى والكيفى (للهرمون المحس لنمو الحويصلة اليضية) بمفرده وبمصاحبة هرمون الغده الدرقية على الدورة الخلوية المنوية فى الأرانب المجهدة تجريبيا

م. الشرى ، م. النجار ، سناء نصار

تم دراسة التأثير الكمى والكيفى لهرمون المحس لنمو الحويصلة البيضية بمفرده . وبمصاحبة هرمون الغدة الدرقية لازالة تأثير عوامل الأجهاد الصيفى الثلاثة ،ارتفاع درجة الحرارة ، وطول مدة الأضاءة ونسبة الرطوبة .

وقد سبب الهرمون المحس لنمو الحويصلة البيضية زيادة عدد ونسبة خلايا الاسبرماتوجونيا (نوع ب) . واعاد نقص خلايا الاسبرماتوسيتس فى الأرانب تحت الأجهاد الى المعدل الطبيعى . وقد زادت نسبتها الى خلايا سرتولى عن المعدل الطبيعى . وقد زود هذا الهرمون عدد خلايا والنسبة السرتولية للاسبرماتيد عن المعدل الطبيعى .

وكان لتأثير هذا الهرمون بمصاحبة هرمون الغدة الدرقية تأثير سىء على الدورة الخلوية المنوية . بحيث كانت الأنابيب المنوية مبطنة بخلايا الاسبرماتوجونيا فقط . وكان هناك زيادة عددية كبيرة لخلايا ليدج .

Depts. of Pathology, Gynaecology and Physiology,
Faculty of Vet. Med. Assiut University,
Heads of Depts. Prof.Dr.A.R.Khater, Prof.Dr.M.Osman & Prof.Dr.Y.Hamed.

EXPERIMENTAL STUDY OF SUMMER STRESS IN RABBIT

III- THE QUANTITATIVE AND QUALTATIVE EFFECT OF F.S.H. AND F.S.H. IN COMPBINATION WITH THYROXINE ON THE SPERMATOGENIC CELL CYCLE IN STRESSED RABBIT

(With 4 Tables and 5 Figures)

By

M.I. EL-SHERRY, M.A. EL-NAGAR and SANAA M. NASSAR*

(Received at 3/3/1980)

SUMMARY

The quantitative and qualitative effect of F.S.H. and F.S.H. in combination with thyroxine stress factors, elevation of temperature, length of photoperiod and relative humidy was studied. F.S.H. had increased the number and ratio of type B spermatogonia and had normalized the decrease of the total number of spermatocytes in stress. The Sertoli ratio was higher than normal. F.S.H. had increased the total number and ratio of the spermatids of the treated group even higher than normal.

Combination of thyroxine with F.S.H. had arrested the spermatogenic cell cycle. The tubules were lined only by spermatogonia. There was very prominant interstitial cell hyperplasia.

INTRODUCTION

The effect of the three summer stress factors temperature elevation, length of the photoperiod and relative humidity was demonestrated quantitatively and qualitatively to cause deliterious effect on the spermatogonial production, spermatocytogenesis and spermiogenesis (EL-SHERRY et al., 1980).

* Faculty of Medicine, Assiut University.

F.S.H. hormone alone and in combination with thyroxine hormone had been used to correct the spermatogenesis in experimental and farm animals. F.S.H. treatment caused extensive testicular epithelial repair and active meiosis in rats after chronic hypophysectomy (LOSTROH, 1963 and LOSTROH et al. 1963). F.S.H. increased the number of spermatogonial mitosis in immature rat and prevented physiological spermatogonial degeneration (HUCKINS et al., 1973). F.S.H. promoted spermatid development (REICHERT and NHALLA, 1974). The quantity and quality of semen was improved by F.S.H. treatment in ram (IZBASAROV and SIMANOV, 1969), frezian bulls (ROY CHAUDHERY et al. 1970) and buffalo bull (SAXENA and SINGH, 1973, 1974).

Thyroxine potentiates the action of gonadotrophins on the testis. MEITES and CHONDRASHAKER (1940) reported that semaltaneous treatment with thyroxine modified the action of gonadotrophins on the testis. treated with gonadotrophic hormone and triiodothyronine exhibited cell division beyound the spermatogenic stage and spermiogenesis established.

The aim of this work is to evaluate the effect of F.S.H. and F.S.H. in combination with thyroxine on the spermatogenic cell cycle of experimentally stressed rabbits quantitatively and qualitatively.

MATERIALS AND METHODS

Two groups of Baladi male rabbits. Each were of four adult male of about 1½ : 2 Kg. Each group was put in a large thermostate; divided into four chambers. The ventilation was specially adjusted and dishes of water were included to produce relative high humidity. The ilumination were directed from surgical lamb of 400 wat to the glass window doors of the thermostate. The duration of the photoperiod were adjusted to start from 6 A.m. to 7 P.m. with longevity of 13 hours. Then the temperature were adjusted to be 39°C. One group was injected by F.S.H. hormone (prolane A, Bayer LEVERKUSEN, Germany) 200 I.U. subcutaneous every three days i.e. two doses per week. The other group was injected

by F.S.H. in the same manner and semultaneously treated by thyroid hormone (The Nile Co. For Pharmaceuticals and chemical Industries Cairo A. R.E.) 30 melligram day after day for the period of one week. At the end of the week, the animals were slaughtered. Testicular samples from both testicles were fixed in Suza and embeded in paraffin,Serial sections were stained by H and E. For estimating the Sertoli cell ratio,the different types of cells occuping the whole cross section of rounded ten seminiferous tubules were calculated. The ten tubules were representing the eight stages of the cycle and stage one and eight as a repetetion. A stage was typed and calculated when all the cell population of the cross section of seminiferous tubules was in the same stage (i.e.) passage between stages was not selected.

The diameter of 30 cross-section was measured for each case. The qualitative aspects of the cycle was observed. For comparison quantitative data of normal control rabbits and stressed rabbits without treatment were taken from previous work (EL-SHERRY et al., 1980). The difference between the groups were analysed statistically using the T test according to SEPTLIEV, 1968.

RESULT

1- THE EFFECT OF F.S.H.

The result of quantification of the cells number, Sertoli ratio and diameter of seminiferous tubules under the effect of F.S.H. on the stressed group is presented in (Table 3). The normal and stress group are presented in (Table 1,2).

F.S.H. injection nearly normalized the decrease in the diameter of seminiferous tubules caused by stress. ($P \not< 0.90$) F.S.H. injection had no effect on the Sertoli cells of the stressed group. It did not correct it to normal.

The total number of spermatogenia of stress treated group was insignificatly lowered than stress without treatment.(P/_ 0.90). The Sertoli ratio for the total number of spermatogonia of the normal, stress and stress treated group was more or less the same.

The number and ratio of type A spermatogonia of the F.S.H. treated group was lower than stress (P /_ 0.999) and normal (P /_ 0.999).The nubmer and ratio of type B spermatogenia in F.S.H. treated stress group was higher than stress (P/_ 0.999) and nearly as normal (P/_ 0.90) . This increase of type B beside the lower number of Sertoli are responsible for the normalization of the Sertoli of the total spermatogonia in F.S.H. stress treated group.

F.S.H. treatment of the stressed group had normalized the decrease of the total nubmer of spermatocytes of stress to normal (P/_ 0.999).The Sertoli ratio was higher than normal. This correction was observed for all types (i.e.) leptotene, zygotene, diplotene and secondary spermatocytes even above the normal level (P/_ 0.999) for all the mentioned types). Except the pachytene which was more or less not affected as the stress group without treatment (P/_ .90). The Sertoli ratio reflect the same character.

F.S.H. had increased the total number and Sertoli ratio of the spermatids of the stressed treated group even beyound the normal while stress sharply decreased them (P/_ 0.999). This correction was true for the number and ratio of all types (P/_ 0.999).

The result of qualification showed that the three animals were normally producing but with the following focal signs of stress degeneration. In one case the wall although normal but relatively of short hight (few layers of cells). In the other cases some tubules of stage seven and eight showed necrosis of partial numbers of elongated spermatids (Fig. 1). The cytoplasm of the cells of some tubules or part of the wall of some tubules was swollen and granulated. There was sporadic coaggulative necrosis of secondary spermatocytes and lysis of focal number of

spermatocytes (Fig. 2,3).

2- EFFECT OF F.S.H. AND THYROXINE

In this group two animals only survived to the end of the week and will constitute the material of our observation. The result of quantification of the number and Sertoli ratio of the epithelial cell cycle and diameter of seminiferous tubules is presented in (Table 4).

This type of treatment had a drastic effect on the testicles. The diameter of the seminiferous tubules was lower than stress without treatment ($P/\_ 0.999$).

The Sertoli cells were slightly higher than in stress ($P/\_ 0.999$) and lower than normal. The seminiferous tubules were lined only by type A spermatogomia whose number and ratio was higher than the normal. No other cells were present.

The qualification showed that the testicle of the two cases were severely degenerated. All the seminiferous tubules were totally denuded and lined by Sertoli and spermatogonia. In the two cases there was very prominant interstitial cell hyperplasia (Fig. 4,5) with hyperaemia.

DISCUSSION

F.S.H. did not correct the decrease of the number of the Sertoli cells of stressed group. Although the Sertoli are the target cells for the action of F.S.H. F.S.H. stimulates adenylate cyclase on the membrane receptors of the Sertoli which in turn increases the cyclic adinosine monophosphate. This evidences stimulate R.N.A. and protein synthesis (MEANS, A.R., HUCKINS, C. 1974). F.S.H. stimulates the production of androgen binding proteins (HANSSON, et al.,1974), and thus increase the binding and accumulation of androgen within the seminiferous tubules (FRENCH et al., 1974).

F.S.H. increased the number of type B spermatogonia. This, beside the lower number of Sertoli are responsible for the normalization of the

Sertoli ratio of total spermatogonia. Some cytochemical studies(MANCINI, et al., 1967) and by determination of F.S.H. sensetive adenyclase activity (BRAUN, 1974) had prooved localisation of F.S.H. in the spermatogonial cells beside the Sertoli cells. The mode of action of F.S.H. on spermatogonial cell is still speculative. HUCKINS, et al., (1973): Found that F.S.H. prevent the degeneration of A_0, A_1, A_2 spermatogonia. Our results did not coincide with this finding as type A was lower than stress without treatment. F.S.H. increases the number of spermatogonial mitosis (MEANS, 1974). Probably, the increase of the number of type B in this data was through the activation of mitosis at the level of intermediate and/or type B spermatogonia. But not at the level of type A as (BRAUN, 1974) had suggested. This fact is inaggrement with the interpretation of MEANS (1974) that F.S.H. control the size of the differentiating spermatogonial pool and subsequently the number of cells which enter the spermatocyte pool (BRAUN, 1974).

The total number of spermatocytes was corrected and this is true for all types except the pachytene cells.The increasing effect of F.S.H. on the number of spermatocytes per tubule was reported by GREEP et al. (1936), LOSTROH, (1963) and LOSTROH et al.,(1963) who observed active meiosis after F.S.H. injections and suggested that F.S.H. stimulates primary spermatocytes. It is of interest to note that STEINBERGER et al. (1974) proved that spermatocytes had no F.S.H. receptors. The result of our quantification showed that the total number of spermatocytes increase can be explained by two facts: a). Increase of type B spermatogonia entering the spermatocyte pool b). The increase in the number of secondary spermatocyes indicating active meiotic division. . The pachytene cells were not increased probably because it is the most sensetive cell to stress (EL-SHERRY, et al. 1980).

F.S.H. increased the total number and Sertoli ratio of the spermatids of the stress treated group even higher than normal. Many data supported the necesssity of F.S.H. to the development of the spermatids (LOSTROH et al. 1963; MEANS, 1974, REICHERT and BHALLA, 1974). There are

no receptors for F.S.H. in the spermatids but the action is mediated through the Sertoli cells (DORRINGTON et al. 1974). The action probably is through two ways: 1) Activation of meiosis and production of young spermatids 2) Exerting a permesive effect by enhansing the binding and transfere of androgen to the spermatid (DESJARDINS et al.,1974). The process of spermoiogenesis is androgen dependent.

Although quantification demonestrated the enhancing effect of F.S.H. on the spermatogonial production, spermatocytogenesis and spermiodenesis. The effect of stress was not completely eleminated by F.S.H. treatment. Partial necrosis of maturing spermatids at stage seven and eight and individual necrosis of secondary spermatocytes were observed. In normal bulls F.S.H. had increased the total number of spermatozoa, improved the motility and percentage of living spermatozoa SAXENA and SIGN,1974).

Combination of thyroxine with F.S.H. gave a dreastic effect.The spermatogeonic cell cycle did not proceed after type A spermatogonia. Contrasting was the prominant interstitial cell hyperplasia. GOSWAMI (1962) had similar report that no benificial effect of adminsestration of P.M.S. and thytoxin on lipido and spermiogenesis of buffalo bulls. There was prominant interstitial cell hyperlasia. The interstitial cells are the target cells for L.H. action. Hyperplasia can probably be atributed to increased L.H. stimulation. Relation between L.H. and thyroxine was established. CHU and YOU (1945) reported increased pitutary L.H. level by thyroxine treatment. Vice versa; there was decreased level of pitiutary L.H. in the thytoectomised rabbit (CHU,1944 & THORSOE, (1962).

Now why the spermatogenic cell cycle is arrested under the condition of F.S.H. and thyroxine treatment? Are the hyperplastic cells not androgen secreting? What is here harmfull? is still an enigma.

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Table 1: Average number of cells, their Sertoli ratio and diameter of seminiferous tubules in normal

Case Number	Sertoli	Spermatogonia Type A	Spermatogonia Type B	Total Spermatogonia	Spermatocytes Leptotene	Spermatocytes Zygotene	Pachytene	diplotene diakinesis	Secondary Spermatocytes	Total Spermatocytes	Spermatids A	Spermatids B	Spermatids C	Spermatids D	Total Spermatids	Diameter of seminiferous tubules in μ
1	8.5	10.8	4.3	15.1	2.3	10.8	16.3	3.5	3.2	36.1	6.4	29.6	12.9	25.5	74.4	221
2	5.1	8.0	8.1	16.1	3.1	13.7	22.5	1.8	2.0	37.8	4.0	38.9	9.7	1.5	73.1	167
3	6.3	10.1	7.2	17.3	4.0	10.8	25.4	2.0	1.1	39.3	9.2	52.9	7.4	29.2	98.7	178
4	9.3	12.6	3.7	16.3	3.7	9.1	27.2	4.2	2.6	46.8	2.3	46.9	8.3	28.4	89.5	179
Mean	7.3	10.3	5.8	16.2	3.3	11.1	22.9	2.9	2.2	40.0	5.5	42.0	9.6	25.2	83.9	186.3
S.D.	1.6	1.6	1.8	0.8	0.6	1.7	4.1	1.0	0.8	4.1	2.6	8.7	2.1	4.6	10.7	23.8
S.E.	±0.3	±0.3	±0.3	± 0.1	±0.1	±0.3	± 0.7	±0.2	±0.1	± 0.7	±0.4	± 1.4	±0.3	±0.7	± 1.7	± 2.2
Sertoli ratio	-	1.4	0.8	2.2	0.5	1.5	3.1	0.4	0.3	5.5	0.8	5.8	1.3	3.5	11.5	-

S.D. Standerd Deviation.

S.E. Standerd Error.

Table 2: Average number of cells, their Sertoli ratio and diameter of seminiferous tubules in stress.

Case Number	Sertoli	Spermatogonia Type A	Spermatogonia Type B	Total Spermatogonia	Spermatocytes Leptotene	Spermatocytes Zygotene	Spermatocytes Pachytene	Spermatocytes diplotene diakinesis	Secondary Spermatocytes	Total Spermatocytes	Spermatids A	Spermatids B	Spermatids C	Spermatids D	Total Spermatids	Diameter of seminiferous tubules in U
1	6.6	9.4	1.8	11.2	1.9	4.5	2.2	0	0	8.6	5.8	0	0	0	5.8	161
2	1.5	18.8	0	18.8	0	7.1	4.9	0	0	12.0	0	0	0	0	0	99
3	5.8	9.3	1.4	10.7	0	19.4	15.6	0.9	2.9	38.8	10.9	4.8	6.2	14.5	36.4	149
4	6.0	7.2	4.1	11.3	5.4	13.2	32.3	5	0.5	56.4	5.3	42.1	10.8	16.8	75	189
Mean	5	11.2	1.8	13.0	1.8	11.1	13.8	1.5	0.9	29.0	5.5	11.7	4.3	7.8	29.3	149.5
S.D.	2.0	4.5	1.5	3.4	2.2	5.8	11.8	2.1	1.2	19.7	3.9	17.7	4.6	7.9	29.8	32.6
S.E.	±0.3	±0.7	±0.2	±0.5	±0.3	±0.9	±1.9	± 0.3	± 0.2	±3.1	±0.6	± 2.8	±0.7	±1.3	± 4.7	± 2.9
Sertoli ratio	-	2.2	0.4	2.6	0.4	2.2	2.8	0.3	0.2	5.8	1.1	2.3	0.9	1.6	5.9	-

S.D.: Standerd Deviation

S.E.: Standerd Error.

Table 3: Average number of cells, their Sertoli ratio and diameter of seminiferous in Stressed F.S.H. treated rabbits.

Case Number	Sertoli	Spermatogonia Type A	Spermatogonia Type B	Total Spermatogonia	Spermatocytes: Leptotene	Spermatocytes: Zygotene	Spermatocytes: Pachytene	Spermatocytes: diplotene diakinesis	Secondary Spermatocytes	Total Spermatocytes	Spermatids A	Spermatids B	Spermatids C	Spermatids D	Total Spermatids	Diameter of seminiferous tubules in/U
1	4.5	5.5	6.0	11.5	2.1	14.0	9.7	4.2	4.1	34.1	11.1	40.8	6.6	37.1	95.6	209
2	6.1	7.6	6.9	14.5	9.0	17.3	12.3	4.1	2.2	49.0	16.7	65.9	17.6	37.1	137.3	177
3	5.1	5.6	5.3	10.9	6.3	16.3	8.8	4.0	4.3	39.7	2.3	20.4	5.7	29.6	88.0	156
Mean	5.2	6.2	6.1	12.3	5.8	15.9	4.1	3.5	3.5	40.9	10.0	52.4	10.0	34.6	107.0	180.7
S.D.	0.7	1.0	0.7	1.6	2.8	1.4	0.1	0.1	1.0	6.1	5.9	10.3	5.4	3.5	21.7	21.8
S.E.	±0.2	±0.2	±0.2	± 0.3	±0.5	±0.3	±0.2	±0.2	±0.2	±1.1	±1.1	±1.9	±1.2	±0.6	± 3.9	± 2.3
Sertoli	-	1.2	1.2	2.4	1.1	3.1	0.8	0.8	0.7	7.9	1.9	10.1	1.9	6.7	20.6	-

S.D.: Standerd Deviation S.E.: Standerd Error.

Table 4: Average number of the cells, their Sertoli cell ratio and diameter of seminiferous tubules in F.S.H. and thyroxin stress.

Case Number	Sertoli	Spermatogonia Type A	Spermatogonia Type B	Total Spermatogonia	Spermatocytes Leptotene	Spermatocytes Zygotene	Spermatocytes Pachytene	Spermatocytes diplotene diakinase	Secondary Spermatocytes	Total Spermatocytes	Spermatids A	Spermatids B	Spermatids C	Spermatids D	Total Spermatids	Diameter of seminiferous tubules in/U.
1	5.1	11.6	-	-	-	-	-	-	-	-	-	-	-	-	-	274
2	7.8	14.3	-	-	-	-	-	-	-	-	-	-	-	-	-	329
Mean	6.5	13	-	-	-	-	-	-	-	-	-	-	-	-	-	301
S.D.	1.4	1.4	-	-	-	-	-	-	-	-	-	-	-	-	-	27.5
S.E.	±0.3	±0.3	-	-	-	-	-	-	-	-	-	-	-	-	-	±3.5
Sertoli ratio	-	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-

S.D.: Standerd Deviation.

S.E.: Standerd Error.

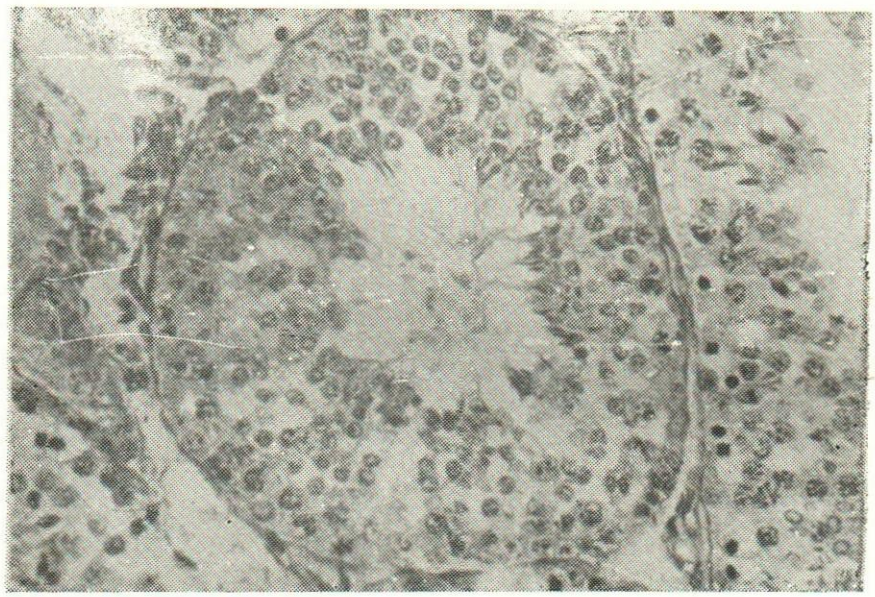


Fig. 1 : Stage VIII partial necrosis of mature sparmatids. (H & E. 20 x 12.5).

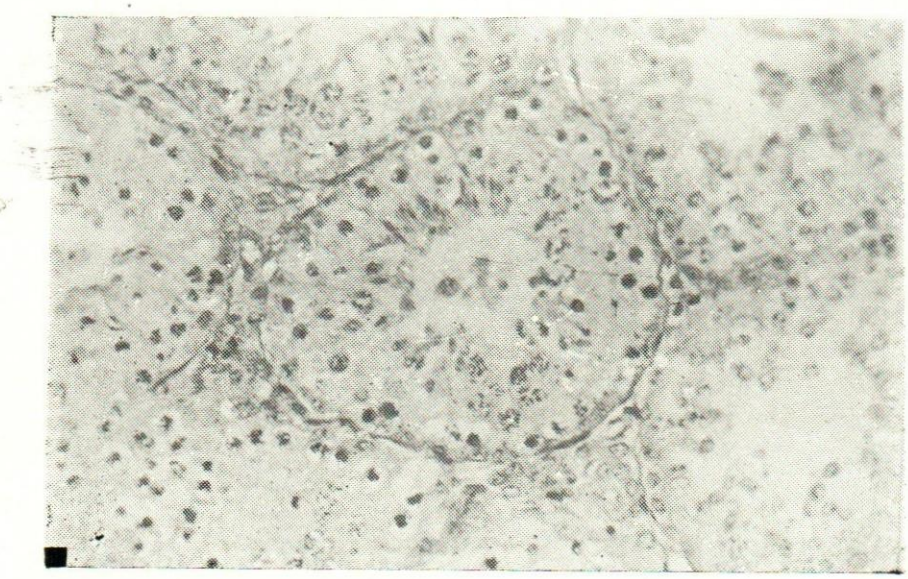


Fig. 2 : Cytoplasmic swelling and granulation. Focal necrosis of secondary spermatocytes. (H & E. 20 x 12.50)

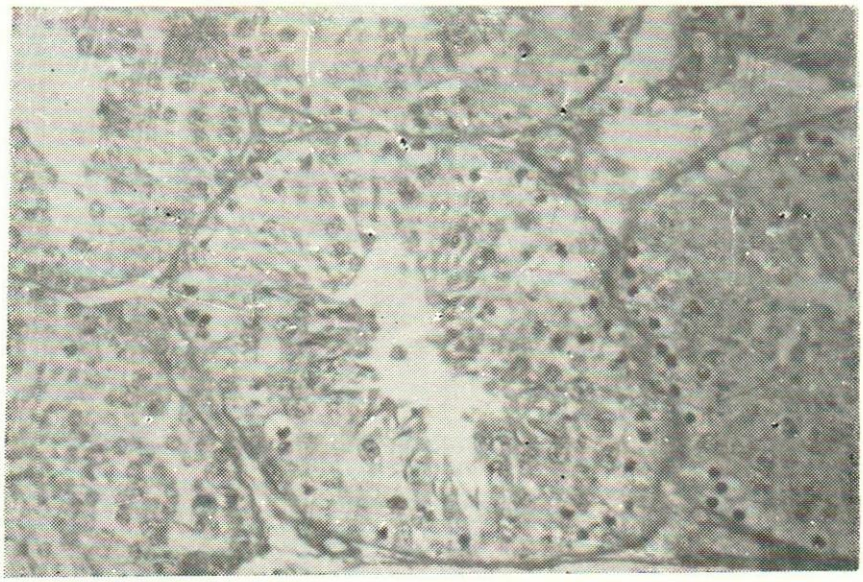


Fig. 3 : Focal Karyloysis of spermatocytes.
(H & E. 20 x 12.5).

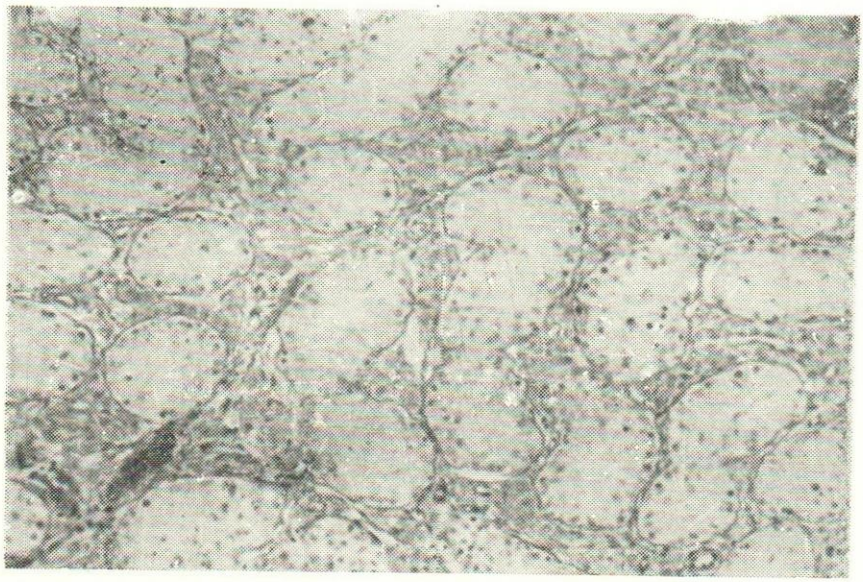


Fig. 4 : The seminiferous tubules were lined by S ertoli and spermatogonia. Diffuese intestitial cell hyperplasia.
(H & E. 10 x 12.5).

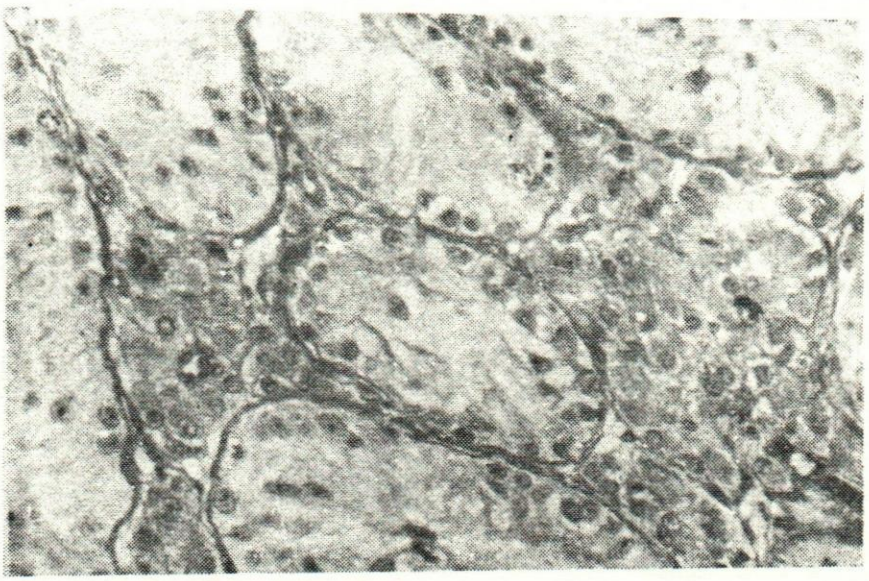
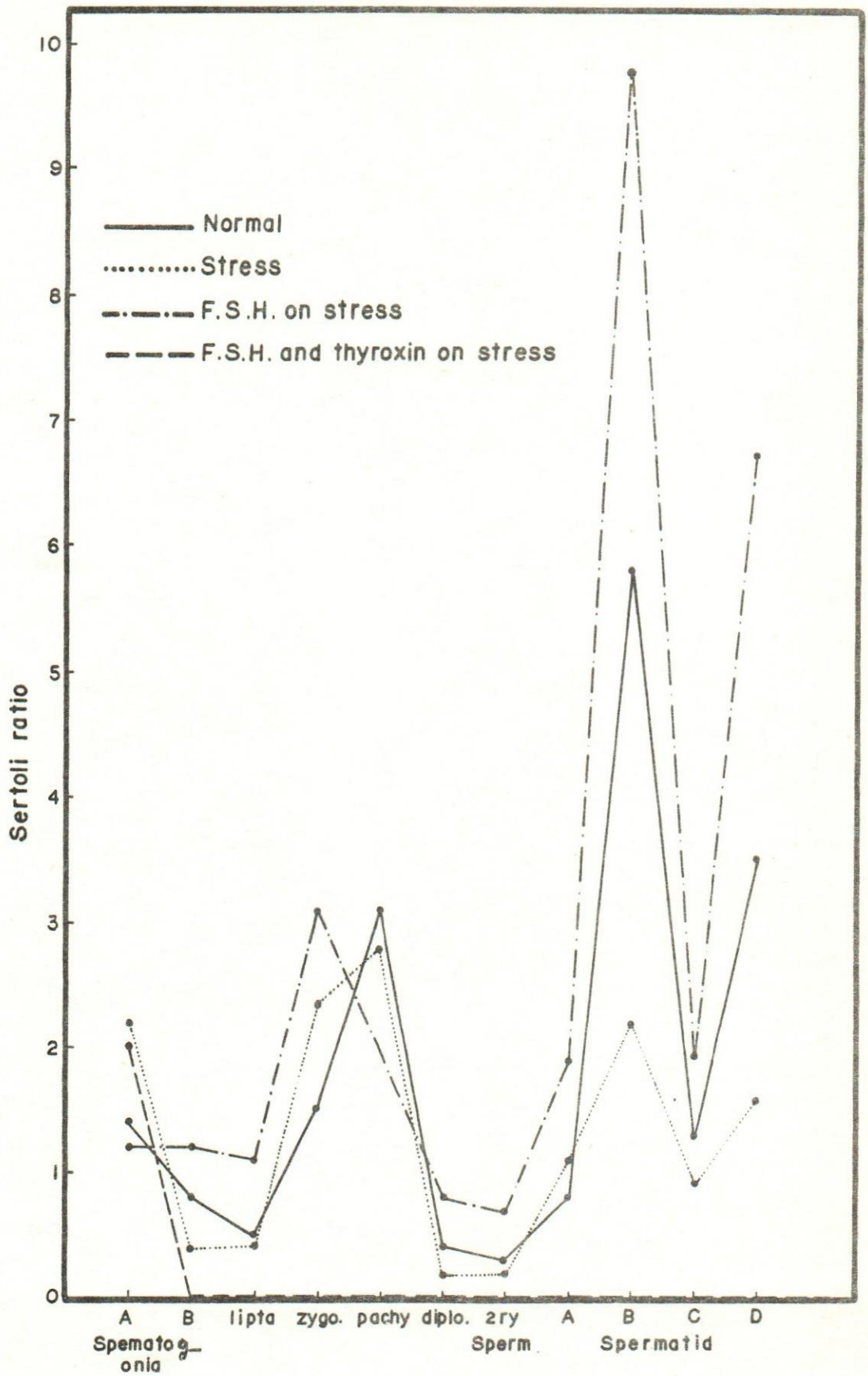


Fig. 5 : Interstitial cell hyperplasia and denueded seminiferous tubules. (H & E. 20 x 12.5).



Graph(2): Sertoli cell ratio F.S.H. and thyroxin on stress