دراسة تجريبية على الأجهاد الصيفى للأرانب
٦- التأثير الكمى والكيفى لحقن الهرمونات المدمحة على الدورة الخلوية المنوية فى الأرانب المجهدة

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حقنت مجموعتان من الأرانب المجهدة برفع درجة الحرارة واطالة فترة التعرض الضوئى وزيادة نسبة الرطوبة بهرمونى :- الهرمون المحث للبويضة واللوتيئيزنج هرمون فى مجموعة . وحقنت المجموعة الأخرى بنفس هادين الهرمونين مع هرمون قشرة الغدة الكزرية وتم أصلاح قطر الأنابيب المنوية فى المجموعتين . ولم يصلح التكسير والنقص فى عدد خلايا السرتولى . لم يصلح العدد والنسبة السرتولية لخلايا الاسبرماتوسيت بالهرمون المحث للبويضة واللوتينيزنج هرمون ولكن هذه المؤشران أصلحوا فى المجموعة التى حقنت بالهرمونات الثلاثة . وقد تم أصلاح نسبة وعدد خلايا الاسبرماتيد فى المجموعتين .

وكانت نوعية التكوين المرضى لتأثير هرمونى . الهرمون المحث للميض واللوتينيزنج هرمون هى اعتراض هجرة خلايا الاسبرماتيد . واستطالة ونمو الاسبرماتيد فى مكانهم . واختلال الانفصال السيتوبلازمى وكان خاصا بمجموعة الهرمونات الثلاثة وجود جيل واحد من خلايا الاسبرماتوسيت . أو جيل واحد من خلايا الاسبرماتيد خلال مراحل الدورة كلها .

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EXPERIMENTAL STUDY OF SUMMER STRESS IN RABBIT

VI- THE QUANTITATIVE AND QUALITATIVE EFFECT OF HORMONAL ASSOCIATION INJECTION ON SPERMATOGENIC CELL CYCLE OF STRESSED RABBIT.

(With 4 Tables and 7 Figures)

By

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SUMMARY

Two groups of rabbits stressed by elevated temperature, length of photoperoid and relative humidity were injected by F.S.H. and L.H. in one group and these two hormones in combination with glucocorticoid in the other group. In both groups the diameter was corrected to normal. The damage and decrease in the Sertoli cell number was not corrected, also the spermatogonial damage. The number and Sertoli ratio of spermatocytes were not corrected by F.S.H. and L.H. But it was corrected by the group of the three hormonal association. In both groups the Sertoli cell ratio and total number of spermatids were corrected.

The qualitative pathogenesis of hormonal action for the F.S.H., L.H. group was blokage of migration of spematids, elongation and maturation of spermatids in situe and disturbance of cytokinesis. Peculiar to the seconed group of the three hormonal association was, one generation of spermatocyte or one generation of spermatid through out the whole cyclic stages.

INTRODUCTION

Spermiogenesis is F.S.H. depenent (REICHERT and BHALLA, 1974). F.S.H. acts on the Sertoli cells and initiates androgen binding proteins which shares in the transfere of androgen to the spermatocytes, the target tissue for androgens (HANSSON, et al. 1974 and FRENCH et al., 1974).

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Androgens combined with the nucleus of the sperm cells will direct and control the further process of spermiogenesis (DORRINGTON and FRITZ, 1973).

L.H. acts on it's target cells, leydge cells and will stimulate and regulate the secretion of androgen (HALL, 1970). It was found that L.H. injection stimulates spermatogonial and spermatocyte meiosis (EL-SHERRY et al., 1980 d).

Naturally,the spermatogenic cell cycle is L.H.and F.S.H. dependent. The three hormones F.S.H., L.H. and androgens synergetically control the spermatogenic cell cycle. These hormones were proved to be deficient in environmental stress (KATANGOLE et al.1974). During enviromental stress, natural and experimental, the level of glucocorticoids was proved to be deficiently lower (MARRLE,JUDG & ABERLS 1972, ALVAREZ, 1973 and THATCHER 1973).

The aim of this work is to study the synergetic action of F.S.H. and L.H. on the spermat ogenic cell cycle of stressed rabbit and also the effect of the three hormonal associations F.S.H., L.H. and glucocorticoids on stressed rabbit trying to correct this pathological condition.

MATERIALS AND METHODS

Two groups of adult male Baladi rabbits (1½:2 Years old) weighing (1½:2 Kg) were used. Each group was composed of four animals. One group was injected by F.S.H.and L.H. simulatanously.The dose of F.S.H. Hormone (Prolane A, Bayer Leverkusen, Germany) was 200 I.U. subcutanously every three days (i.e.) two doses per week. The dose of L.H. (chorion gonadotrophin - Mucos, Emulsions gesell shaft m 6 H. 8022 Grunwold her Munich) was three melliliters 600 I.U. were injected subcutanously every three days. The animal received two doses during the week.

The other group was injected semultanously with glucocorticoids beside the above two gonadotrophic hormones. The glucocorticoid used was sodium prednisolone 21- hemisuccinate (Solu - Decortin Merck). The dose

was 100 mg subcutanously. The animal received 2 doses per week.

Each group was put in thermostate with glass doors partitionally divided into four chambers, one for each rabbit. Ventilation was specially adjusted and dishes of water were included to produce relative high humidity. Artificial illumination started from 6 Oclock a.m. to 7 Oclock p.m. to represent the medium duration of summer day light. The temperature adjusted to 39°C for day and night.

At the end of the week, the animals were slaughtered. Testicular specimens were fixed in suza. From each block serial section 5 micron thickness were stained by haematoxylene and eosin. The spermatogenic cell cycle was quantitatively evaluated. For their quantitative evaluation 10 rounded cross sections of seminiferous tubules representing the eight stages of the cycle and a repetition of stage one and eight was selected. The number and Sertoli cell ratio for each type of cells were calculated. The Sertoli cell ratio of stressed rabbits groups without treatment and normal control group were taken from previous work (EL-SHERRY et al.,1980 a&b). For evalution of the diameter 30 rounded cross section were selected and measured. The results were statistically analysed and compared to the result of the control group by T test according to (SEPETLIEV, 1968).

RESULTS

I- Effect Of Combination Of F.S.H. and L.H. On Stressed Testicles .

Combination of F.S.H.and L.H. had corrected the diameter of seminiferous tubules nearly to the normal (P/_ 0.90). It did not correct the decrease of the Sertoli cell njmber of stress.

The Sertoli cell ratio and numer of total spermatogonia were lower than the number in case of stress without treatment (P/_ 0.999). Type A

spermatogonia decreased to half the normal number (P/ 0.999). The Sertoli ratio was decreased to half the value in case of stress. Type B number had been corrected to a slight degree below normal and the Sertoli ratio was typically as normal. (P/ 0.999).

The combination of F.S.H. and L.H. did not effect the number and Sertoli ratio of the total spermatocytes. They were the same like in stress without treatment. The number and Sertoli ratio of leptotene were normalized (P/ 0.999). The number and ratio for zygotene had been increased above the stress without treatment and normal.The pachytene were deleteriously affected and sharply lowered (P/ 0.999) than in case of stress group without treatment. The number and ratio of diplotene diakinesis were doubled (P/ 0.999). The number and ratio of secnodery spermatocytes were normalized (P/ 0.999).

F.S.H. and L.H. had increased the total number (P/ 0.999) and Sertoli ratio of spermatids but did not reached the normal level. It had doubled the number of type A (P/ 0.999) and its ratio above the stress without treatment and the normal. The number and ratio of the type B slightly increased above the stress but it did not reach the normal.The number and ratio of type C and D were significantly higher (P/ 0.90) than stress.

The testis demonstrated producing tubules in two cases. Most of the seminiferous tubules were in the normal first three stage of the cycle. But the Sertoli cytoplasm with the inserted cells were highly swollen and granulated. Coagulative necrosis or lysis of primary and secondary spermatocytes were observed with the start of stage four and five. In these stages the elongated spermatids were more or less healthy.Most of the tubules of higher stages. 6,7,8 showed necrosis of the spermatid Contents. Normal tubules with these stages were present.

In the other two cases, all the seminiferous tubules were producing but the cycle did not proceed stage three because the cytokinesis failure. This is evidenced by the following facts:

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1- Early formation of spermatid gaint cells in stage one. 2- Nuclei of the rounded spermatids start to aquire acrosomes and the underlying chromatin was without elongation. Their cytoplasm start to become acidophilic (Fig. 1). 3- In some cross section the elongation of spermatid nuclei occured but without migration to form bundles in the Sertoli cytoptasm (Fig. 2). The associated spermatocytes were not corresponding to stage two but of higher stages. In other cross sections, the rounded spermatid nuclei with acrosomes start to aquire the dusty chromatin and maturation. (i.e) failure of cytokinesis block the seminiferous epithelial cycle, althouth the differentiation of spermatid nluclei were proceeding. Few number of tubules were lined by Sertoli and spermatogonia only.

Interstitial hyperemia was predominant in four cases. The interstitial cells in the four cases were swollen with foamy cytoplasm. Some cells showed necrobiotic changes (Fig. 3).

II- The Effect Of The Three Hormonal Association F.S.H., L.H. And CLUCO-CORTICOIDS On Stessed Testicles.

The association treatment of stressed group had Corrected the decreased diameter caused by stress to normal ($P/\_$ 0.999). The Sertoli cells were not effected by treatment and they were the same as in the stress group without treatment.

The Total number of spermatogonia and their Sertoli ratio were lower than in stress ($P/\_$ 0.95). Type A spermatogonia number and ratio were decreased to the half quantity of the stress and normal goups. Type B number was significantly ($P/\_$ 90) lower than normal while, the Sertoli ratio was normalized.

The number of total spermatocytes was normalized ($P/\_$ 90). The Sertoli ratio was higher than normal. The number and Sertoli ratio of leptotene was the same as that of stress without treatment. The zygotene ratio and number were increased nearly double the amount of stress and normal groups ($P/\_$ 0.999). The pachytene were sharply decreased in number ($P/\_$ 0.999) and ratio than normal and stress group. The number of the

diplotene and diakinesis were normalized (P/ 0.90). The ratio was double that in case of stress and higher than in case of normal. The number and ratio of secondary spermatocytes were double the normal.

Association treatment had normalized the total number of spermatids of stress group (P/ 0.999). It is insignificantly higher than normal (P/ 0.90). Although the Sertoli ratio is significantly higher than normal. The number and ratio of type A spermatid were highly increased (P/ 0.999) four times the normal level. Type B number highly increased than stress (P/ 0.999), and approached the normal level (P/ 0.99). The Sertoli ratio was higher than normal.The nubmer of type C was insignificantly higher (P/ 0.90) than normal. The Sertoli ratio was higher than normal. Type D number and ratio were nearly normalized (P/ 0.90).

Pathological examination revealed that the interstitial cells were normal. There was mild degree of hyperaemia. The seminiferous tubules were producing but with the following cyclic disturbances: Some Sertoli cells were swollen with lysed nuclei. Cytoplasmic swelling and granulation was a feature.

Stage one: In two cases, there was abscence of one generation of spermatocytes. This was contant in all the seminiferous tubules of stage on (Fig. 4).

Stage two: In two cases, the elongation of the spermatids and their migration to from bundles in the Sertolian cytoplasm were stopped or retarded. In some seminiferous tubules, the spermatid is started to be mature in their places and aquired the dusty chromatin and acrosomes preserving the rounded appearance and their position of stage one(Fig. 5). In some seminiferous tubules elongation occured without migration (Fig. 6). In very few numbers of seminiferous tubules elongation occured but associated with advanced stages of spermatocytes and only one generation of them i.e. cycle retardation.

Stage 3: The migration and elongation of spermatids take place in situe. Some seminiferous tubules showed the zygotene, diplotene and the

rounded spermatids of stage one.

Stage 4: In the three rabbits, the cross section of this stage had only secondary spermatocytes. The zygotene spermatocytes of normal stage four were abscent. The elongated spermatids still not migrated to bundles.

Stage 5: The seminiferous tubules of this stage were lined by young spermatids type A or B with few elongated spermatids type D (Fig. 7). No generation of primary spermatoctes were observed. Stage 6. Rarely present with few mature spermatids. Stage seven and eight were absent.

DISCUSSION

In both groups F.S.H. and L.H. treated and the other combined three hormones, the diameter was corrected to normal. But it is clear from the above qualification and quantification that spermatogenic cell cycle is not perfectely normalized. The diameter index for evaluation of testicular function is valuable only when it decreased. It do mean pathological condition. Normal diameter bears, the two probability either perfect normal cycle or disturbed cycle. A clear example was the action of glucorticoids alone on the stressed testis where glucocorticoids enhanced quantitatively the process of spermatocytogenesis and hence the height of the wall of the seminiferous tubules and the diameter was normal. Qualitatively the spermatocytes were suffering either degnerative changes or necrosis (EL-SHERRY et al., 1980 e).

Treatment by hormonal association in both group did not correct the damage and decrease of the Sertoli cell number in stress. Damage of Sertoli is central in the pathogenesis of any disturbance of the spermatogenic cell cycle, as it is the cell which Co-ordinates, regulates and associates the various types of cells in the cycle. The Sertoli number do not respond to the action of F.S.H. alone (EL-SHERRY et al. 1980 a). Although the Sertoli was prooved to be the target cell for the metabolism of F.S.H. (MEANS and HUGKINS 1974). The effect of L.H. alone on the

Sertoli was conflecting. L.H. injected to normal animal decreased the Sertoli cell number. L.H. normalised the Sertoli number of stressed treated testicle (EL-SHERRY et al., 1980 d). Damage of the Sertoli was prevented by glucocorticoids alone (EL-SHERRY et al., 1980 c).

The hormonal association treatment in both group did not correct the spermatogonial damage of stress. Glucocorticoid alone was harmfull to the spermatogonia (EL-SHERRY et al., 1980 e). On the contrary F.S.H. highly enhanced the spermatogonial production through activation of mitosis as both types of spermatogonia were increased (EL-SHERRY et al. 1980 c). L.H. also normalize the spermatogonial damage but here beside activation of division, influence on differentiation was clear as type B was increased in stress more than normal.

The combination of F.S.H. and L.H. did not affect the number and Sertoli ratio of total spermatocytes in stress. These two hormones with combination of glucocorticoids normalized the total number of spermatocytes and the ratio was higher than in normal. For interpretation of this correction of the later group, must be taken in consideration, the action of each hormone quantitatively and qualitalively.

It was clear form the prevous work (EL-SHERRY et al. 1980 d) that L.H. increases the spermatocytogenesis through the activation of meiosis. F.S.H. increase the spermatogenesis through activation of spermatogonial mitosis and thus increase the spermatogonia which enter the spermatocyte pool. The increase of the spermatocytes under glucocorticoid injection (EL-SHERRY et al.,1980 e) was only numerical but qualitatively not true. This is also the same in the condition of the three hormonal association as the frequency of the lower stages of the cycle prevailed than in the higher stages. The lower stages of the cycle posses two generation of spermatocytes. The higher stages of the cycle posses one generation of spermatocytes and that is why number of pachytene cell sharply decreased and the number of zygotene diplotene & diakinesis increased and hence the numerical increase of the spermatocytogenesis do not reflect increase

efficiency of spermatogenic cell cycle. These results dectates another index, the frequencies of each cycle stage for full evaluation of the productivity of the cycle.

It is worthy to note here that in the group F.S.H. and glucocorticoid, the cycle in two cases showed peculiar character (i.e.) through out the whole stages of the cycle. The majority of the tubules showed either one generation of spermatocyte in the lower stages or one generation of spermatid in the higher stages i.e the Capacity of the cycle had been normalized but to the half of it's full capcity.

Qualitatively F.S.H. is still the hormone of choice which normally corrected the spermatocyte production. In L.H. treatment, although the correction of meiosis were morphologicaly prooved by increase number of nuclei and aggregation of nuclei, but failure of cytokinesis was the defect. Glucocorticoids caused lysis or coagulative necrosis on the sporadic spermatocytes.

L.H. and F.S.H. although corrected the number and ratio of the total number of spermatids, but this correction is on the expense of type A (rounded spermatids) and the other types.; type B (stage of acculation of D.N.A.) and type C (elongating spermatid), type D (maturing spermatid) were not corrected. In the group of the three associated hormones quantitatively, the four types of spermatids were corrected but qualitatively pathological disturbance in the process of spermiogonesis was evident by 1- Retarded differentiation of spermatids; as elongating spermatids of stage two observed with higher stages of the cycle. 2- The migration of the spermatids to form bundle was highly disturbed. Failure of cytokinesis blocked this phenomen in many places. The rounded spermatids mature and differentiate in situe without migration.

In conclusion, the pathogenesis of the spermatogenic cell cycle can be divided into the following successive stages: 1- Spermatogonial mitosis and differentiation where F.S.H. stimulates the mitosis. F.S.H. and thyroxin (EL-SHERRY et al.1980 c) block the process of the spermatogonia

production to type A only. 2- Spermatocytogenesis including the two meiotic division, the first resulting the prolonged stages of meiotic prophase; leptotene, zygotne, pachytene, diplotene, diakinesis and secondary spermatocytes; the result of the seconed meiotic divison. In these two process accumulation of D.N.A. is central. Glucocorticoids are harmful to spermatocytogenesis by blockage probably D.N.A. synthesis and cause necrosis of spermatocytes. L.H. is the main factor controling the meiotic divison and their differentiation. F.S.H. is also necessary for meiotic divison and differentiation. The two hormones sporadically corrected the damaging effect of stress with it's sensetive stages; the pachytene diplotene transformation,the zygotene pachytene transformation and the secondary spermatocytes.

The pathogenesis of spermiogenesis inculed three main categories. The second meiotic division with production of spermatid type A, augmented by F.S.H. hormone. In the other hormonal treatment, they were numerically increased through the predominant frequency of the lower stages of the cycle. Type B and type C prooved to be heat sensetive. They were deliteriously affected by glucocorticoids. L.H. promotes these two processes of differentiation. F.S.H. normalized these two process of differentiation. Type D (the maturation of spermatid) was totally blocked by glucocorticoid alone. F.S.H. normalize the maturation. L.H. enhanced the maturation and differentiation but disturbed the phenomena through failure of cytokinesis.

The three indices entroduced, diameter, quantification and qualification was helpful in explaining the pathogenesis of heat stress and hormonal action on the spermatogenic cell cycle. For future work, we suggest the frequency of the cyclic changes to be combined to the three indices. These four endices will be helpful in the evaluation of the pathogenesis of the spermatogenic cell cycle due to other causes and will give high posibilities than the simple classification slight, mild and severe testicular degeneration of LAGERLOF, 1934; or commonly used spermiogenesis or aspermatocytogenesis (focal and diffuse)...

The work has revealed an important aspect in the pathogenesis of the hormonal action. Probably lower doses of L.H. and the accurate timing of injection simulating nature as L.H. episodicaly secreted will give us activated meiosis without cytokinesis. Also the dose of F.S.H. and injection before or after L.H. injection will still a question? Association with androgen is also required to correct the process of spermiogenesis.

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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	Secondary 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 tubules in stress F.S.H. and L.H. treated rabbits.

Case Number	Sertoli	Spermatogonia Type A	Spermatogonia Type B	Total spermatogonia	Spermatocytes Leptotene	Spermatocytes Zygotene	Spermatocytes Pachytene	Spermatocytes diplotene diakinesis	Secondary Spermatocyte	Spermatocytes	Spermatids A	Spermatids B	Spermatids C	Spermatids D	Total Spermatids	Diameter of seminiferous tubules in/U
1	5.8	8.1	6.8	14.9	13.4	9.8	13.5	5.6	9.0	51.3	17.1	39.0	9.1	29.7	94.9	187
2	4.6	4.9	4.0	8.9	1.0	13.5	8.8	2.8	0	26.1	9.0	11.9	7.5	17.6	46.0	175
3	5.0	4.3	3.1	7.4	0	14.9	0	8.7	0	23.6	0	0	7.0	0	48.9	165
4	4.1	2.9	2.2	5.1	0	15.0	0	0	0	15.0	15.0	17.7	0	0	21.2	195
Mean	4.9	5.1	4.0	9.1	3.6	13.3	5.6	4.3	2.3	29.0	10.3	17.2	5.9	11.8	52.8	180.5
S.D.	0.6	1.9	1.7	3.6	5.7	2.1	5.8	3.2	3.9	13.6	6.6	14.1	3.5	12.6	26.6	11.4
S.E.	±0.1	±0.3	±0.3	±0.6	±0.9	±0.3	±0.9	±0.5	±0.6	±2.2	±1.0	±2.2	±0.6	±2.0	±4.2	± 1.04
Sertoli ratio	–	1.1	0.8	1.9	0.7	2.7	1.1	0.9	0.5	5.9	2.1	3.5	1.2	2.4	10.8	–

S.D.: Standerd Deviation.

S.E.: Standerd Error.

Table 4: Average number of cells, their Sertoli ratio and diameter of seminiferous tubules in stress F.S.H., L.H. and glucocorticoids treated rabbits.

Case Number	Sertoli	Spermatogonia Type A	Spermatogonia Type B	Total Spermatogonia	Leptotene	Zygotene	Pachytene	diplotene diakinesis	Secondary Spermatocytes	Total Spermatocytes	Spermatids A	Spermatids B	Spermatids C	Spermatids D	Total Spermatids	Diameter of seminiferous tubules in/U
1	6.2	5.7	9.6	15.3	4	28.4	25.8	6.5	5.9	70.6	27.2	44.1	4.3	48.7	124.3	210.8
2	5.0	6.9	2.6	9.5	0.8	18.2	0.3	2.3	6.9	28.5	15.8	24.0	4.4	14.0	58.2	166.3
3	4.3	4.7	0.4	5.1	0.9	14.1	0.7	2.5	1.3	19.5	23.4	29.0	24.9	10.5	87.8	173.8
Mean	5.2	5.8	4.2	10	1.9	20.2	8.9	3.8	4.7	39.5	22.1	32.4	11.2	24.2	90.1	183.6
S.D.	0.8	0.9	3.9	4.2	1.5	6.0	11.9	1.9	2.4	22.3	4.7	8.5	9.7	17.2	27	19.5
S.E.	±0.2	±0.2	±0.7	±0.8	±0.3	±1.1	±2.2	±0.4	±0.4	±4.1	±0.8	±1.5	±1.8	±3.1	± 4.9	± 2.1
Sertoli ratio	-	1.1	0.8	1.9	0.4	3.9	1.7	0.7	0.9	7.6	4.3	6.2	2.2	4.7	17.3	-

S.D.: Standerd Deviation

S.E.: Standerd Error.

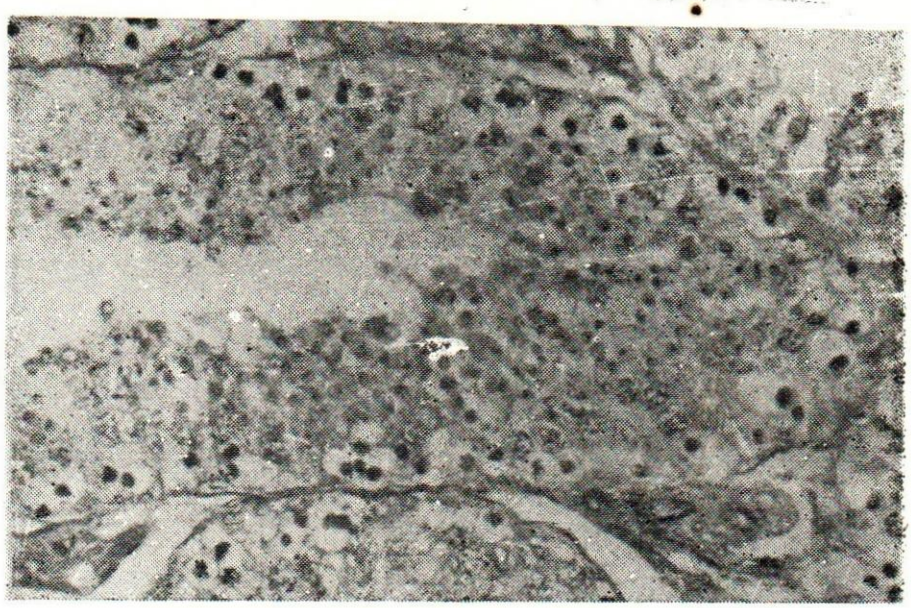


Fig. 1 : The rounded spermatids aquired acrosomes and dusty chr matin without elongation or migration. (H.&E. 20 x 12.5).

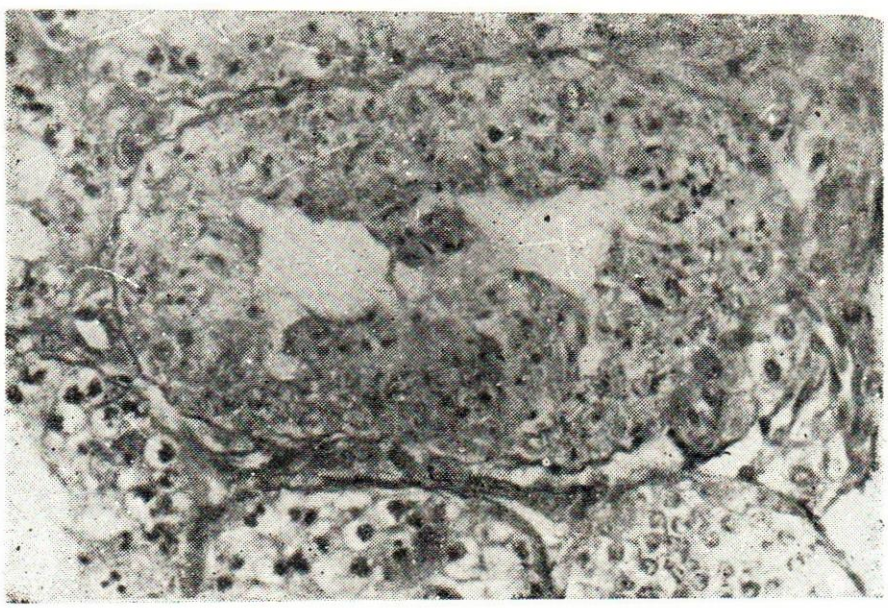
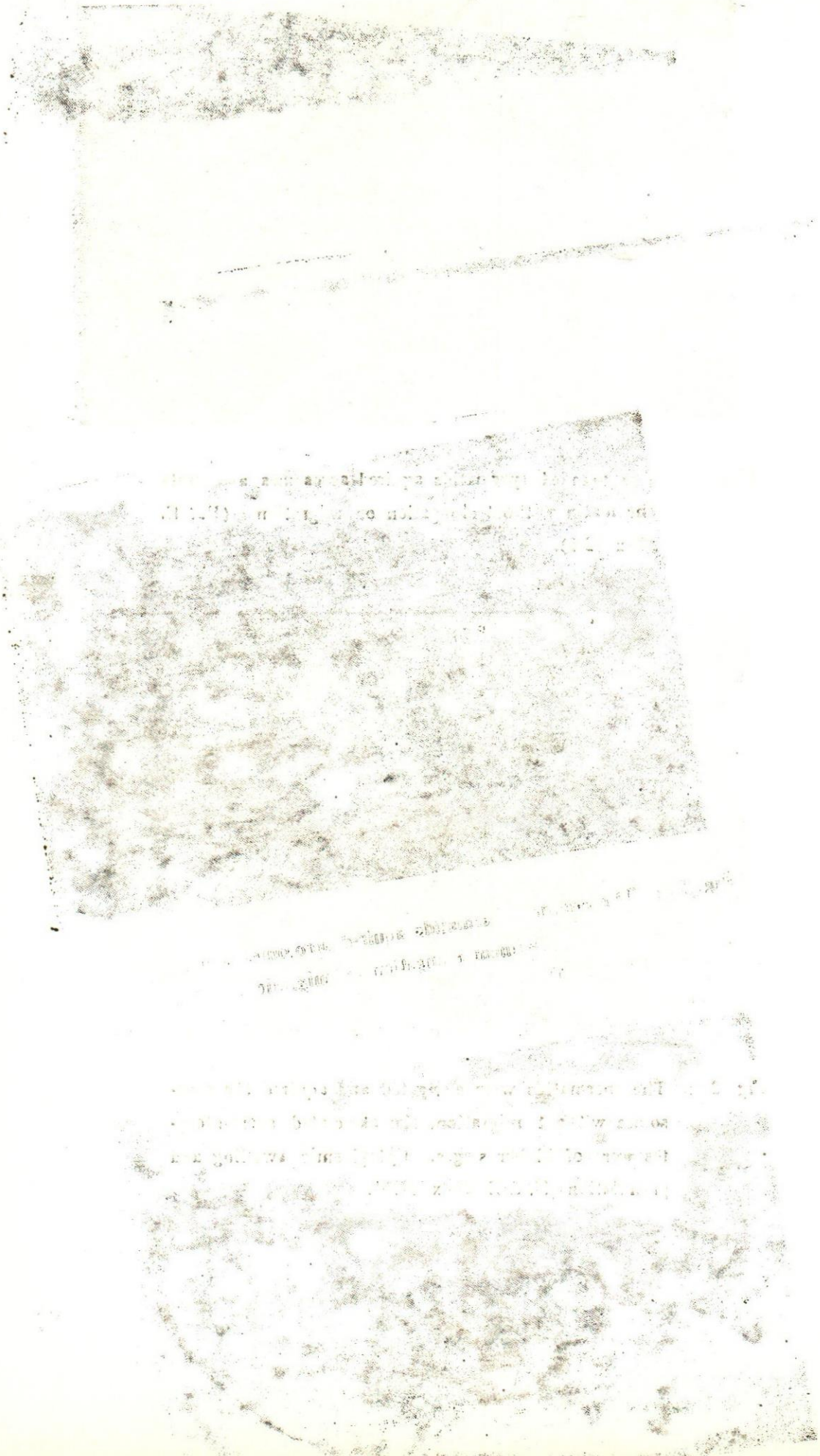
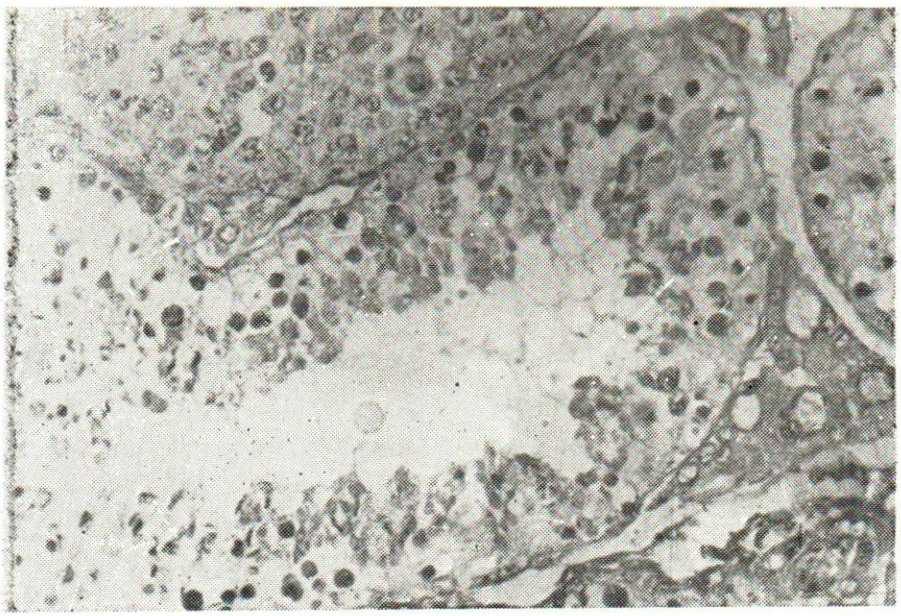


Fig 2 : The spermatids were elongated and aquired the acrosomes without migration, the associated spermatocytes were of higher stages. Cytoplasmic swelling and granulation (H, & E. 20 x 12.5).





Fig, 3 : The interstitial cells showed swollen vaculated nuclei and swollen granulated cytoplasm, coagulative necrosis of spermatocytes and spermatids. (H. & F. 20 x 12.5).

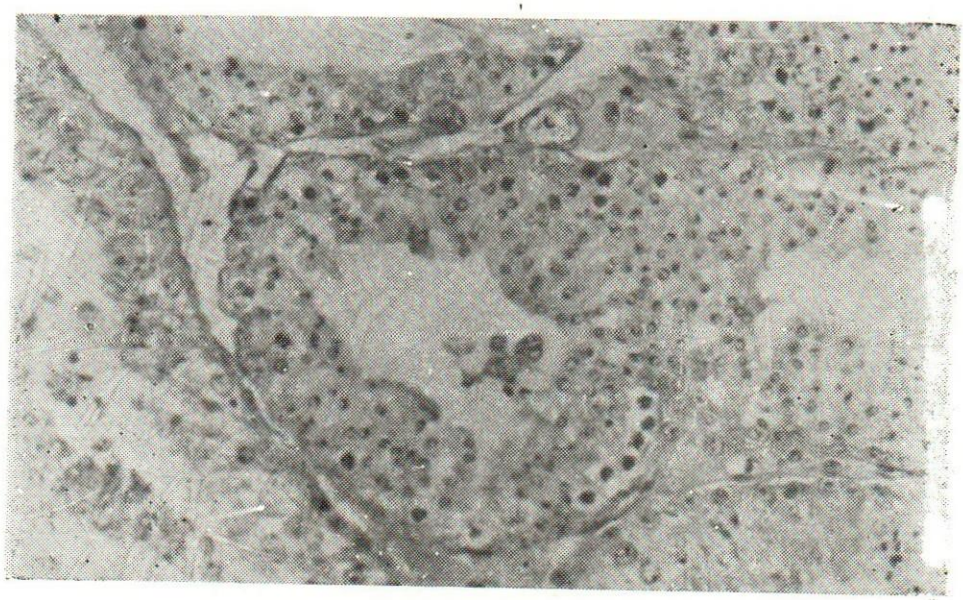


Fig. 4 : Association group. Stage I. Absence of pachytene generation of spermatocytes. H. & E. 20 x 12.5).

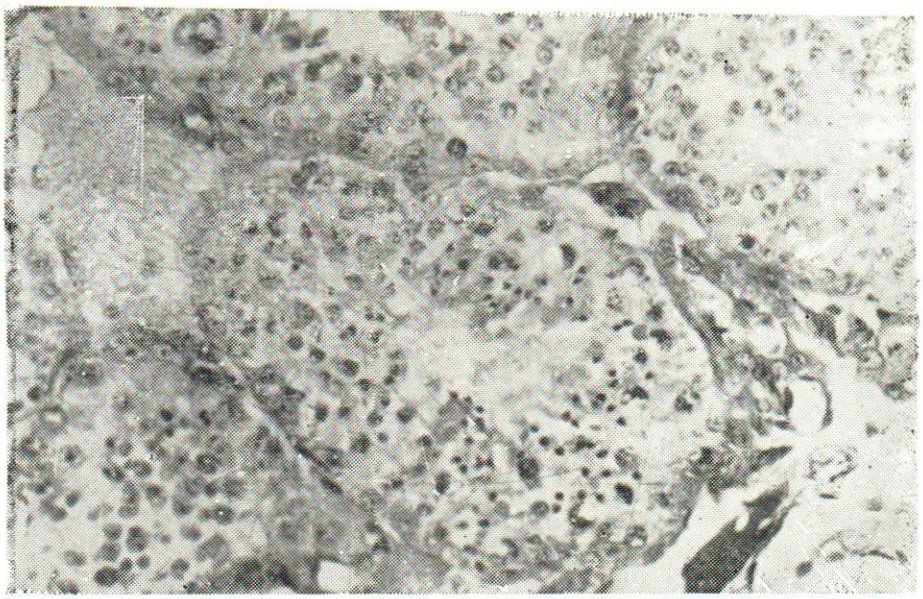


Fig. 5 : Maturation of spermatids in situ. (H & E. 20 x 12.5).

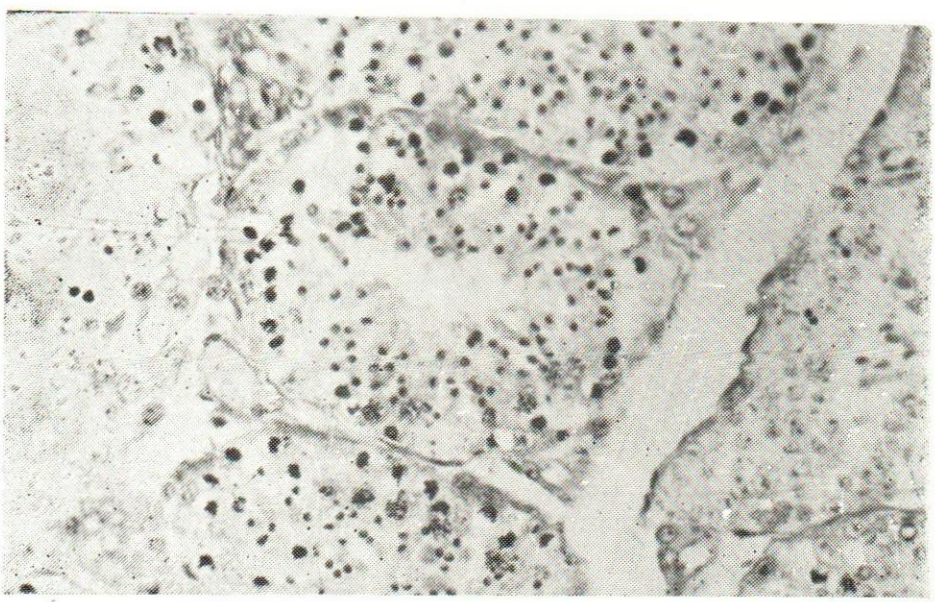


Fig. 6 : Presence of zygotene and diplotene spermatocytes of stage three and associated with rounded spermatid. Elongation of weakly expressed, no migration to (H. & E. 20 x 12.5).

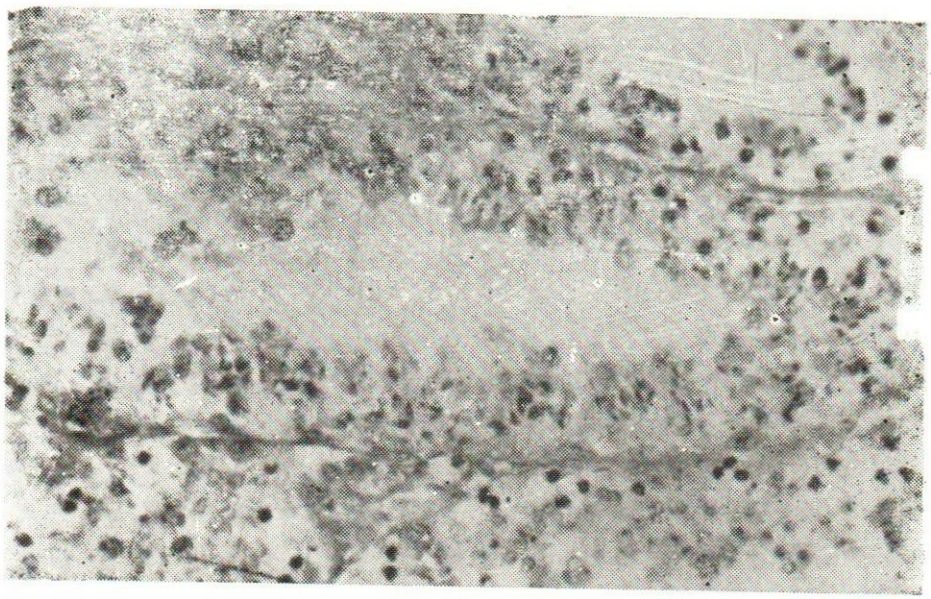


Fig. 7 : The seminiferous tubules were lined by elongated spermatid. The second generation of rounded spermatid were absent. One generation of pachytene spermatocytes. (H. & E. 20 x 12.5).