دراسة تجريبية على الأجهاد الصيفى فى الأرانب

٥ - التقييم الكمى والكيفى لتأثير هرمون قشرة الغدة الكظرية على الدورة الخلوية المنوية للأرانب الطبيعية والأرانب تحت الأجهاد الصيفى التجريبى

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حقنت مجموعتان من ذكور الأرانب بهرمون قشرة الغدة الكظرية . المجموعة الأولى تمثل أرانب طبيعية ككنترول والمجموعة الثانية وضعت تحت الأجهاد الصيفى التجريبى برفع درجة حرارة واطالة فترة التعرض للضوء وزيادة نسبة الرطوبة . وحللت الدورة المنوية الخلوية نوعيا وكميا باستعمال نسبة خلايا سارتولى . أحدث الهرمون تحسن وزيادة فى عملية تخليق خلايا الاسبرماتوستيس . بينما سبب الهرمون تعطيل وتأخير لعملية التميز الشكلى والحركى لعملية تخليق الحيوانات المنوية لم ينجح الهرمون فى حماية الخصية من تأثير الأجهاد الصيفى . وبالرغم من أن عملية تخليق خلايا الاسبرماتوستيس قد تحسنت كميا الا أن هذه الخلايا كانت تقاسى من التخلل أو الموت التجلطى . ولوحظ موت معظم خلايا الاسبرماتيد الناضجة .

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EXPERIMENTAL STUDY OF SUMMER STRESS IN RABBIT V- THE QUANTITATIVE AND QUALITATIVE EFFECT OF GLUCOCORTICOIDS INJECTION ON SPERMATOGENIC CELL CYCLE OF NORMAL AND STRESSED RABBIT.

By

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SUMMARY

The qlucocorticoid hormone was injected in two groups of rabbits. A control group and a group suffered experimental stress by temprature elevation , long photoperiod and relative humidity. The spermatogenic cell cycle was analysed qualitatively and qualitatively using the Sertoli cell ratio. Glucocorticoid improved the process of the spermatocytogenesis and retarded the differentiation of the spermiogenesis in normal testicles. The hormone did not protect the testicles againest the experimental summer stress. Although the spermatocytogenesis quantitatively improved, all type of spermatocytes were suffering either lysis or coagulative necrosis. The mature spermatids were necrosed.

INTRODUCTION

Stress deleteriously affected the testis in rabbit (EWING et al. 1964). CHRISTIAN and DAVIS (1964) and CHRISTIAN et al. (1965), showed that the bad effect of stress was mediated through adrenal response. During enviromental stress; natural and experimental; the level of glucocorticoids was prooved to be deficiently lowered. (MARPLE, JUDGE, ABERLE, 1972; ALVAREZ, JOHSON, 1973 and THATCHER, 1973).

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The sperm cells are not the proper target tissue for glucorticoids action as KORNEL (1973) had enumerated liver tissue, lymphoid cells and fibroblast to be the proper target tissues for glucocorticoid action. The effect of exoganous adrenal steroids on the testis are highly contradictory in the literature. Glucocorticoids improved the testicular weight (LEROY, 1952; CONNER, 1959; LEROY, 1959; HANSON et al., 1957; D'ARCY and HOWARD,1961), increased sperm production (CUPPS et al. 1960), maintained spermiogenesis in hypophysectomised animals (LEROY and DOMM, 1952) and prevented toxins-induced degeneration of the testicles (CHATTERJEE, 1966).

No effect of glucocorticoids application on the testicles was reported by LI and EVANS (1947). LOFTS et al. (1968) and MADDACK et al. (1953).

Adverse effect of glucocorticoids were prooved by ANTOPOL, (1950), BOAS, (1958); RUPONEN and NAATANEN (1962), ZOR et al. (1966). Surveying the available to us literature, studies of the effect of glucocorticoids on indices other than testicular weight,sperm number were not available. The detailed changes on the spermatogenic cell cycle were laking.

The aim of this work is to study in details the quantitative and qualitative effect of glucocorticoids on the spermatogenic cell cycle in normal rabbits and in experimentally summer stressed pathologically altered cycle as a trial for correction.

MATERIALS AND METHODS

Two groups of adult male Baladi rabbits (1½ : 2 years old weighing 1½ : 2 Kg. Each group was composed of four animals. One group was injected by glucocorticoids. Each rabbit was injected subcutaneously by 100 mg sodium prednisolone 21- hemisuccinate. Solu-Dacortin Merck.The animal received two doses per week.

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The second group was put in thermostate with a 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 am. to 7 Oclock pm. to represent the medium duration of summer day light. The temperature adjusted to 39°C for day and night. The animals were injected by gluco-corticoids as the first group.

At the end of the week the animals were slaughtered. Testicular specimens were fixed in Suza. From each block serial sections 5 micron thickness were stained by Harris Haematoxylene and eosin.

The spermatogeneic cell cycle was qualitatively evaluated.For their quantitative evaluation 10 rounded cross sections of seminiferous tubles 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 control group were taken from previous work (Table 1,2, EL-SHERRY et al. 1980). For evaluation of the diameter 30 rounded C.S. were selected and measured. The results were statistically analysed and compared to the result of control group by T test according to (SEPETLIEV, 1968).

RESULTS

1- The effect of glucocorticoids on normal testicles:-

The results of quantification of the seminiferous epithelial cycle and diameter of seminiferous tubules of both the glucocrticoids normal treated group and stressed group were presented in (Table 3&4).

Glucocorticoids increased the diameter of seminiferous tubules ($P < 0.999$). Glucocorticoids had no effect on the number of Sertoli cells. The hormone decreased the Sertoli ratio and number of total spermatogonia. This was true for both type A and B with different significance ($P < 0.999$) and ($P < 0.98$) resectively.

Glucocorticoids increased the Sertoli cell ratio and number of total spermatocytes. This was true for all types except the pachytene where the number and ratio decreased; P / 0.999 for all types.

Glucocorticoids increased the number and ratio of type A spermatid. The other types B, C and D were decreased (P / were respectively / 0.999, / 0.90 and / 0.90).

Pathological examination of the quality of the semiferous epithelial cycle showed that glucocorticoids affected the process of spermiogenesis in three cases. Normal spermiogenesis in the foruth case reflected individual variation. Retarded differentiation of spermatids was reflected variably. In one cases all the seminfierous tubles were deviod from elongatting or elongated spermatids, only rounded spermatids were present.

In the other two cases the presence of elongated spermatids is very few. Different stages of spermatid differentiation are present in the same cross section. Many cross sections demonestrated the rounded, elongating and elongated spermatids in the same time (Fig. 1). The frequency of the advanced stages are very low. If present, mature spermatids were very few or absent. The interstitial cells were more or less normal. Mild degree of hyperaemia was present in the four cases.

2- The effect of glucocorticoids on stressed testicles:-

The result of quantification of the seminiferous epithelial cycle and diameter of seminiferous tubules of the glucocorticoid treated stressed group and stress without treatment are presented in (Table 3 & 4).

The hormone normalised the diameter of the stressed treated group (P/ 0.999). There was no difference between the normal and stressed glucocorticoids treated group (P/ 0.90). The treatment normalized

the decreasing effect of stress (P/_ 0.999) on the Sertoli cells probably by preventing their damage.

The hormonal treatment lead to further depresion in the number and ratio of spermatogonia than the stress without treatment. The effect was the same for the two types of spermatogonia. Type A was decreased (P/_ P/95). In spite of the fact that stress caused increase of type A number (P/_ 0.90) and the ratio doubled. The Sertoli ratio for type B was lower than stress. Although the number non significantly (P/_ 0.90) increased.

The Sertoli cell ratio and total number of spermatocytes were increased (P/_ 0.999) and was corrected by the hormonal treatment nearly to normal. The leptotene type of the prophase was decreased in number and ratio (P/_ 0.999). In contrast to the effect of glucocorticoids on the normal where the leptotene increased. The hormone treatment increased the zygotene number (P/_ 0.999) and ratio in the stressed treated group to above the normal level. This is because the number of zygotene were constant in stress and their Sertoli ratio was higher. The pachytene number did not change in treated group. Their Sertoli ratio decreased. The number and ratio of diplotene and diakinesis were increased in stress treated group (P/_ 0.999). The secondary spermatocytes were corrected to the normal level (P/_ 0.999).

The effect of the hormonal treatment on the total number and ratio of spermatids was no effective as on the spermatocytes. The Sertoli cell ratio of the spermatids was lower in the stress treated group than the stress without treatment. The number non significantly increased (P/_ 0.90).Differential for the type of the spermatids, type A increased above the normal level. The other three types were badly effected (P/_ 0.999). Although the number and ratio of type D increased than stress (P/_ 0.999) but it is still lower than normal.

The pathological examination demonestrated sever testicular degeneration in all the four cases. It is meant here by severe degeneration the frequency of tubules affected and not the extent of damage of individual tubule (LAGERLOF, 1934). In one case the cellular wall of the seminiferous tubules was totally necrosed to the basement membrane. Only spermatid giant cells, swollen Sertoli nuclei and or pycnosed zygotene cells could be observed (Fig. 2). There was severe interstitial hyperaemia and the interstitial cells were suffering from necrobiotic changes. In the seconed case, there was rich infilteration of the interstitium by eosinophiles and neutrophils (necrotic orchitis). In the third and fourth case the seminiferous tubules showed various cyclic stages but the following pathollgical features were observed. There was coagulative necrosis of spermatids types D or during the last three staqes of the cycle in nearly all the seminiferous tubules. Aspermiogenesis is evident in some tubules (Fig. 3). The Sertoli cytoplasm with the cytoplasm of the inserted cells was swollen and granulated. Necrosis of the primary and secondary spermatocytes was observed in nearly all stages (Fig. 4). During the first three stages some zygotene and pachytene spermatocytes were lysed which gave the wall of the seminiferous tubules vacoulated appearance (Fig. 5). Some secondary spermatocytes of stage four were usually coagulatively necrosed. Dead cells with mitotic figures and acidophilic coagulated chromosomes were observed. The sporadic spermatocyte coagulative necrosis were observed in higher stages.

The interstitial hyperaemia was a feature. The interstitial cells although possesed normal nuclei, the cytoplasm was totally vacuolated.

DISCUSSION

Glucocorticoids application to normal testicle had bad effect on both types of spermatogenia. The hormone improved the process of spermatocytogenesis. The hormone retarded the process of differentiation in spermiogenesis. The increase in type A spermatid is explained by the

increase of spermatocytes pool as type A is the result of the meiotic division of secondary spermatocytes and also morphologically the majority of cross section of the seminiferous tubules were lined by the rounded type A spermatid. The other types: B (stage of accumulation of dusty chromatin), type C (stage of elongation of spermatids)and type D (stage of maturation) were decreased. It is obvious that differentiation and maturation in the process of spermiogenesis is deleteriously retarded but division are not affected as the total number of spermatid were more or less constant.

Retarded differentiation and maturation of spermatid can be interpreted in the light of the mode of action of glucocorticoids on the target tissues and cells. Glucocorticoids inhibites the synthesis of nucleic acid and protein in lymphocytes and fibroblasts (SEIFER and HILZ, 166). But contradictory to this fact will be the explanation of improved spermatocytogenesis because increased spermatocytes necessiates increase synthesis of D.N.A. and R.N.A. althouth the action of glucocrticoids on target tissue and cells is also contradictory as there were lymphocyte sensetive and lymphocyte resistant and fibrocyte sensetive fibrocytes resistant (HARRIS, 1970). Vice versa to the action of glucocorticoid on lymphocyte and fibroblast. It increases the R.N.A. and protein synthesis in liver tissue (MAKMAN et al. 1968).

However our interpritation of increased spermatocyte number is that the majority of the tubules are lined by spermatocytes in association with lower stages of the cycles. Confirmatory was the decrease in the pachytene spermatocytes (Tetraploid cells with the maximum quantity of D.N.A.), and are associated mainly with higher stages of the cycle.

The glucocorticoids application on stress did not protect the testicles. Although the quantification demonestrated increased spermatocytogenesis but the spermatocytes when produced were suffering in their majority eigher lyses or coagulative necrosis. The mature spermatids

were necrosed. The variation between individuals was of the same magnetitude as the stress without treatment. The mature spermatids formed were necrosed.

In conclusion glucocorticoids inhanced the process of spermatocytogenesis and retarded the differentiation of spermatids in the normal testicles. The hormone did not protect the testicles from the stressing influence of the three factors of summer stress.

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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 normal glucocorticoid 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 semineferous tubules in/U
1	6.0	7.1	7.0	14.1	16.8	14.0	10.9	6.2	4.9	52.8	48.5	4.8	13.3	7.2	73.8	200
2	7.0	9.9	1.9	11.8	9.4	17.6	15.3	5.8	9.7	57.8	44.1	10.7	8.1	37.7	100.6	229
3	9.3	7.9	4.0	11.9	10.9	18.8	29.0	6.2	21.6	86.5	33.1	35.4	11.6	33.9	114.0	233
4	6.6	10.8	4.4	15.2	6.0	10.3	20.6	0	1.8	38.7	45.6	3.6	0	6.8	53.0	215
Mean	7.2	8.9	4.3	13.3	10.8	15.0	18.9	4.6	9.5	58.95	42.1	13.6	8.3	21.4	85.4	219.3
S.D.	1.2	1.5	1.8	1.5	3.9	3.2	6.7	2.6	7.5	17.4	5.6	12.8	5.1	14.5	23.6	13.0
S.E.	±0.2	±0.2	±0.3	±0.2	±0.6	±0.5	±1.1	±0.4	±1.2	±2.8	±0.9	±2.0	±0.8	±2.3	±3.7	±1.2
Sertoli ratio	-	1.2	0.6	1.8	1.5	2.1	2.6	0.6	1.3	8.2	5.8	1.9	1.2	3	11.9	-

S.D.: Standerd Deviation

S.E.: Standerd Error.

Table 4: Average number of cells, their Sertoli ratio and diameter of seminiferous tubules in stress glucocorticoid 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	6.0	11.1	0.7	11.8	0	19.0	11.2	0	0	30.2	4.8	4.9	6.6	9.4	25.7	152
2	8.5	6.8	2.8	9.6	0	17.9	19.6	6.5	4.6	48.6	31.8	0	6.3	25.2	63.3	173
3	6.9	7.6	1.2	9.0	0	19.4	9.3	7.9	3.5	40.1	24.1	4.3	0	22.0	50.4	182
4	10.1	10.0	3.8	13.8	0	32.3	14.4	12.5	0	59.2	14.0	0	0	13.5	29.2	215
Mean	7.9	8.9	2.1	11.1	0	22.2	13.6	6.7	2.0	44.5	18.7	2.3	3.2	17.5	42.2	180.5
S.D.	1.6	1.7	1.2	1.9	0	5.9	3.9	4.5	2.1	10.7	10.2	2.3	3.2	6.3	15.5	22.6
S.E.	±0.3	±0.3	±0.2	±0.3	0	±0.9	±0.6	±0.7	±0.3	±1.7	±1.6	±0.4	±0.5	±1.0	±2.5	± 2.07
Sertoli ratio	-	1.9	0.3	1.4	0	2.8	1.7	0.9	0.3	5.6	2.4	0.3	0.4	2.2	5.4	-

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

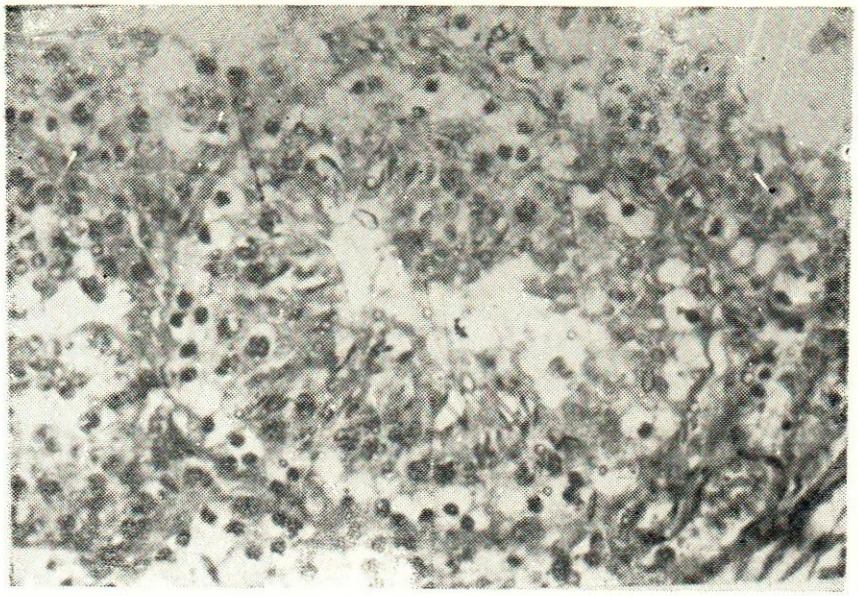

Fig. 1 : Retarde diffsrentiation of spermatids : rounded, elongated and elongating spermatid. (H & E. 20 x 12.5).

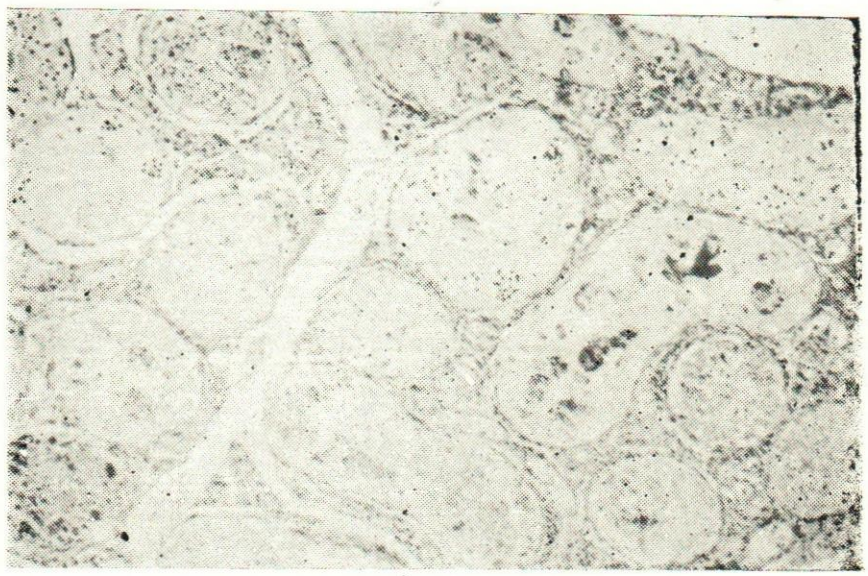

Fig. 2 Total necrosis of the cellulur wa'l of the seminiferous tubules Spermatid giant ce'l and pycnosed spermatocytes were present. (H & E 10 x 12 5).

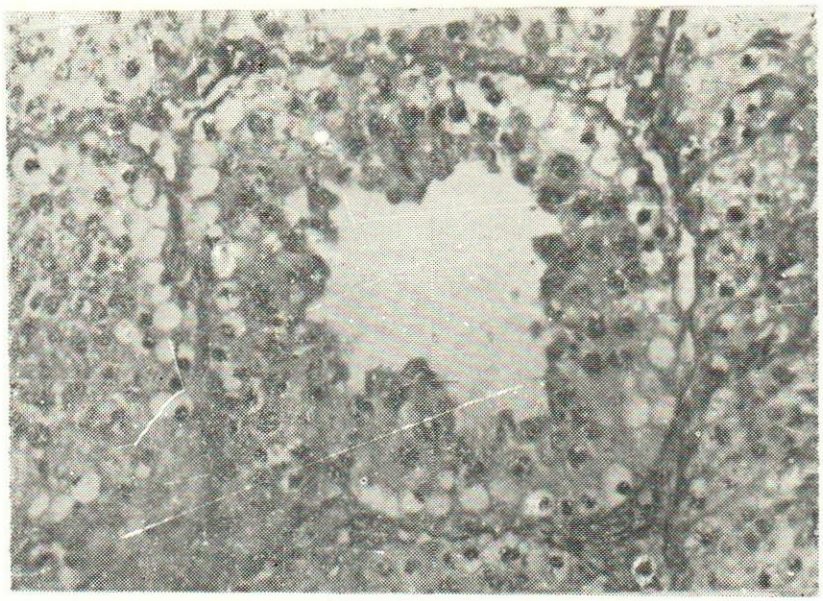

Fig. : 3 Aspermiogenesis. (H & E. 20 x 12,5)

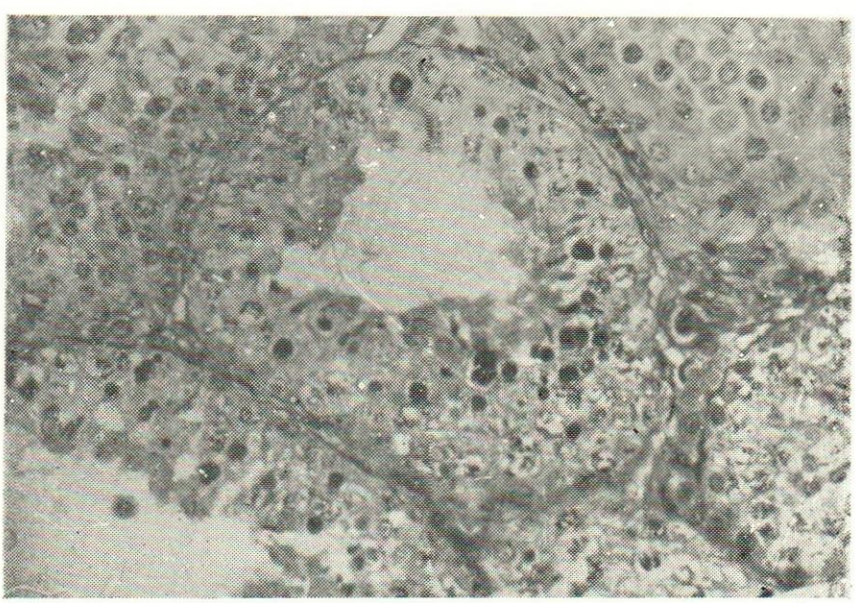

Fig. : 4 Primary spermatocytes & coagulative necrosis.
(H & E. 20 x 12.5),

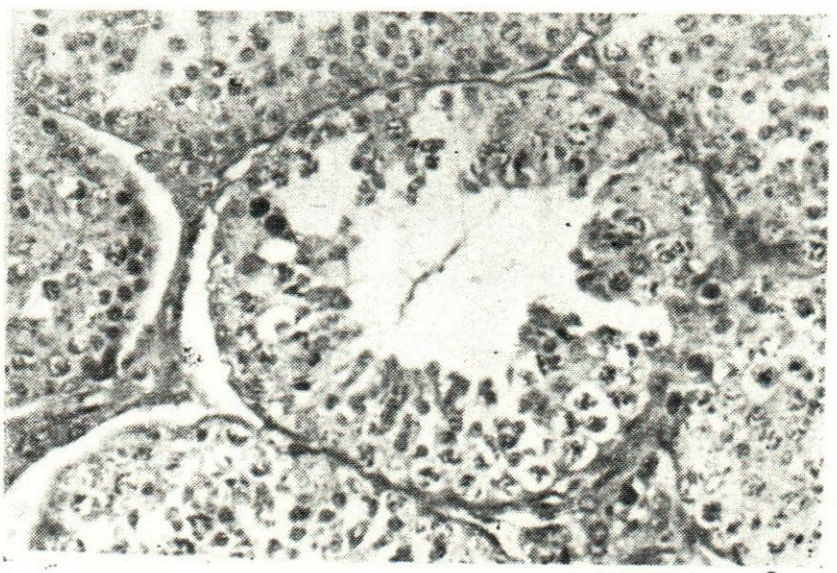

Fig. 5 : Lysis of spermatocytes. Reticulated appearance of the wall. Necrosis of spermatids. (H & E. 20 x 12,5).