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أثر نقص فيتامين (أ) في العليقة على الحالة الاكلينيكية  
ومكونات الدم والأعضاء الداخلية في الجاموس البالغ

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تمت دراسة أثر نقص فيتامين (أ) في العليقة على عدد ٣١ من الجاموس البالغ في مزرعة القوات المسلحة بمدينة منقباد بمحافظة أسبوط . وقد اتضح من فحص الحيوانات المصابة اكلينيكيًا وجود اضطرابات في الحالة الصحية والنمو مع وجود قشور على جلد الحيوانات المصابة علاوة على اضطرابات في الجهاز التنفسي والبولي والهضمي ، كما كانت تعاني من عشى ليلي . كما تم دراسة مكونات الدم ولوحظ وجود تغيرات ملحوظة في نسب الجلوكوز وانزيمات الترانس امينيز وكذا الصوديوم والبوتاسيوم والكلور . كما لوحظ وجود نقص شديد في كمية فيتامين (أ) والكاروتين في الكبد .

كما أجريت دراسة هستوباثولوجية على الأعضاء الداخلية ثبت منها وجود تغيرات بثنولوجية في الكبد والكلى والرتتين .

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THE EFFECT OF DIETARY VITAMIN A DEFICIENCY ON THE CLINICAL  
CONDITION, BLOOD CONSTITUENTS AND INTERNAL ORGANS OF ADULT BUFFALOES  
(With 2 Tables & 4 Figures)

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SUMMARY

The effect of dietary vitamin A deficiency on 21 adult buffaloes was studied at Mankabad military breeding station. Clinical examination of the animals revealed disturbances in the animal health which appeared in the form of disturbances in the growth, pityriasis, night blindness with the appearance of respiratory disturbances, kidney troubles, and some animals were suffering from enteritis. Biochemical examination of the blood revealed significant changes mainly in blood glucose, serum transaminases and blood electrolytes. Marked decrease was noticed in vitamin A value in the liver.

Histopathological examination revealed a picture of lung affection with liver and kidney troubles.

INTRODUCTION

Vitamin A deficiency is of major economic importance in group of animals especially those fed on ration deficient in vitamin A or its precursors. This problem is of great significance in animal confined indoors fed on prepared ration (HOAR *et al.* 1968). The deficiency syndrome of vitamin A in ruminants includes a variety of symptoms depending on the degree of deficiency and the animal condition. The produced syndrome includes night blindness, corneal keratinization, pityriasis, defects in the hooves, loss of body weight and infertility (EATON, 1970). Primary vitamin A deficiency occurs most commonly because of lack of green feeds or failure to add vitamin A supplement to deficient diets (DAVIS *et al.*, 1970).

Vitamin A is essential for the maintenance of normal epithelial tissues, hence deprivation of the vitamin produces effects largely attributable to the disturbance of function of normal growth and maintenance of natural barriers (BLOOD and HENDERSON, 1974).

Both vitamin A and carotenes are important in insuring adequate supply of vitamin A to the tissues necessary for the occurring metabolic processes especially those concerned with carbohydrates, proteins, fats and minerals (MOORE, 1959). The pathological effects of vitamin A deficiency are so numerous and diverse that it is difficult to group them in a clear and unified picture, however, the work of (NELLSSEN *et al.*, 1966) showed that vitamin A deficiency in cattle may be accompanied with focal necrotic hepatitis.

Generally the literature on the necessity of vitamin A for cattle is too extensive to be reviewed in details, but as far as dealing with buffaloes the task of consideration will be lightened by data obtained in cattle to give as far as possible an explanation of the obtained results.

The aim of the present investigation is to study the effect of dietary vitamin A deficiency on adult buffaloes mainly the clinical condition, constituents of the blood and the occurring pathological changes.

MATERIAL AND METHODS

This work was conducted at Mankabad military breeding station on 21 adult buffalo (3 years old) suffering from vitamin A deficiency. This deficiency may be resulted from destruction of vitamin A value due to prolonged exposure (up to 6 months) of the ration to sunlight. This ration was in the form of concentrate mixture (30% cotton seed meal, 24% wheat bran, 38% crushed white maize, 2% lime stone, and 1% sodium chloride) and hay. Each animal was fed 16 Lbs of the concentrate mixture and 6 Lbs hay. The investigated animals were clinically examined and

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blood samples were taken to estimate serum blood constituents as follows:

Total serum proteins using Abe refractometer (MACFATE, 1972); Non protein nitrogen (OSER, 1965); Blood urea by nesslerization method (RAITSKA, 1970); Sodium and potassium using flame photometer (OSER, 1965); Chloride contents (SCHALES and SCHALES, 1941); glucose (COOPER and McDANIEL, 1970) and transaminases by test kits from C.F. Boehringer and Soenne Co. Mannheim, West Germany and after the method described by (REITMAN and FRANKEL 1957).

The buffaloes were slaughtered and samples were taken from the liver to determine vitamin A values according to (DANN and EVELYN 1938) described by (OSER 1965). Histopathological studies were conducted on samples taken from the lungs, kidneys and liver using H&E stain (CARLETON and SHORT, 1954).

## RESULTS

### A- Clinical picture:

The affected animals exhibited general disturbances in their health which appeared in the form of rough dry coat with heavy deposits of bran-like scales on the skin. Hooves were dry and scaly and there were also night blindness. Some of the buffaloes were suffering from enteritis and others showed respiratory disturbances and urinary tract troubles. Respiratory troubles were rhinitis, bronchitis ending with pneumonia in some cases. Urinary tract troubles appeared as attacks of acute abdominal pain with downward arching of the back and in some cases there were dysuria.

### B- Biochemical examination:

Chemical constituents of blood serum of adult buffaloes suffering from vitamin A deficiency are demonstrated in table I; while vitamin A and carotene level in the liver is demonstrated in table II.

### C- Histopathological examination:

In the kidney Bowman's capsules were dilated and glomerular tufts were atrophied (Fig. 1). There was accumulation of homogenous highly acidophilic albuminous materials in many tubular lumina. In addition necrotic changes were seen in tubular epithelium of some tubules. There was proliferation of histocytes and lymphocytes causing thickening of the interstitial tissue. Examination of the liver showed dilatation and congestion of the portal and central veins. The bile ducts walls showed signs of metaplasia. Necrobiotic changes were clearly seen in the liver cells especially those located at the peripheral zone of liver lobules. Proliferation was seen in the cells of the portal triad (Fig. 2).

The lung showed squamous metaplasia in the epithelial cells of some bronchi and bronchioles. Other bronchi showed evidence of destruction in the epithelial lining. External to the bronchi there was a zone of relatively dense fibrous tissue which was less vascular with rare cellular infiltration. The alveolar spaces were widened and some of them were ruptured. (Fig. 3 and 4).

## DISCUSSION

The evidence presented here would seem to demonstrate the effect of dietary vitamin A deficiency in adult buffalo whether in the clinical manifestation or other biochemical and histopathological findings. The appeared clinical syndrome in general agrees with that reported by (BLOOD and HENDERSON 1974) with exception of absence of xerophthalmia. This may be attributed to the fact that xerophthalmia is seen at latter stages of vitamin A deficiency.

The determined vitamin A value in the liver (  $3.09 \pm 0.17$  mg% vitamin A and  $0.33 \pm 0.04$  mg% carotene ) of the affected buffaloes pointed out to the occurring deficiency as far as 7.25 mg%, vitamin A is considered to be the limit below which the animal will suffer from vitamin A deficiency (AWAD *et al.*, 1979 and McDONALD *et al.*, 1979).

The changes in total proteins, nonprotein nitrogen, glucose and electrolytes in the blood serum come in agree with (MOORE 1959) in that vitamin A and carotenes are important for the occurring metabolic processes especially those concerned with proteins, carbohydrates and minerals, and hence deficiency in vitamin A value will eventually leads to changes in the concentrations of such blood constituents. The appeared changes in the total blood serum proteins (slight decrease) and glucose (marked increase) if compared with the normal values obtained by (HASSAN 1980) (  $7.26 \pm 1.25$  gr.% and  $64.0 \pm 6.80$  respectively ) can be explained by the fact that glucose level is abnormally increased in hepatic and renal insufficiency (MILLER, 1966). The histopathological picture in the liver may give further explanation to such behaviour of glucose, proteins and minerals where the liver is mainly responsible for

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the metabolic processes of these constituents. This occurring hepatic and renal insufficiency resulted from lowering of natural barriers by disturbances in epithelial lining of the gastrointestinal mucosa. Vitamin A is necessary for healthy epithelial tissue and consequently its deficiency will affect the efficiency of the epithelial lining and hence pathogenic and toxic agents passed to the liver and kidney and other vital organs setting up such troubles leading to their insufficiency.

The percentages of sodium, potassium and chlorides are relatively decreased if compared with the normal values obtained by (HASSAN 1980) which are  $157.96 \pm 5.62$  and  $108.9 \pm 10.48$  mEq./litre respectively. This can be explained on the bases that in pulmonary affections and kidney troubles the percentages of these electrolytes decrease (MILLER, 1966). But our results however contradict with the findings of (LANTER 1975) in that kidney affection may interfere with the process of excretion of sodium and potassium and these electrolytes may be retained in the blood. The results of morphologic alterations in the kidney parenchyma detected in the examined animal may support the disturbance in the estimated electrolytes levels.

The obtained results concerning GOT and GPT enzymes pointed to an increase activity of both enzymes and the first recorded relatively higher levels. These additional facts may give further explanation of the involvement of the liver under the stress of vitamin A deficiency for the pathological changes in these parameters.

Finally it can be concluded that deficiency of vitamin A can lead to disturbances in the natural barriers as far as vitamin A is necessary for healthy epithelial tissue. Furthermore such deficiency can lead to disturbances in the metabolic processes especially those concerned with proteins, carbohydrates and minerals.

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Table (I)

Chemical constituents of blood serum of adult buffalo suffering from vitamin A deficiency

Items	No. of examined animals	Mean values
Total proteins (gr.%)	21	6.16 $\pm$ 0.31
Urea (mg%)	21	38.9 $\pm$ 0.67
Nonprotein nitrogen (mg%)	21	29.4 $\pm$ 0.61
Glucose (mg%)	20	105.4 $\pm$ 2.16
Chloride (m.Eq./litre)	21	85.3 $\pm$ 0.72
Sodium (m.Eq./Litre)	21	162.4 $\pm$ 2.19
Potassium (m.Eq./Litre)	21	4.18 $\pm$ 0.14
Glutamic oxalic transaminase	20	38.70 $\pm$ 1.01
Glutamic pyruvic transaminase	20	9.55 $\pm$ 0.13

Table (II)

Vitamin A and carotene level in the liver of adult buffalo suffering from vitamin A deficiency

Items	No. of examined animals	Mean values
Vitamin A (mg%)	20	3.09 $\pm$ 0.17
Carotene (mg%)	20	0.33 $\pm$ 0.04

 $\pm$  = Standard error.



Fig.(1):Kidney: X400. shows atrophied glomerular tuft, necrobiotic and metaplasia of renal epithelium.

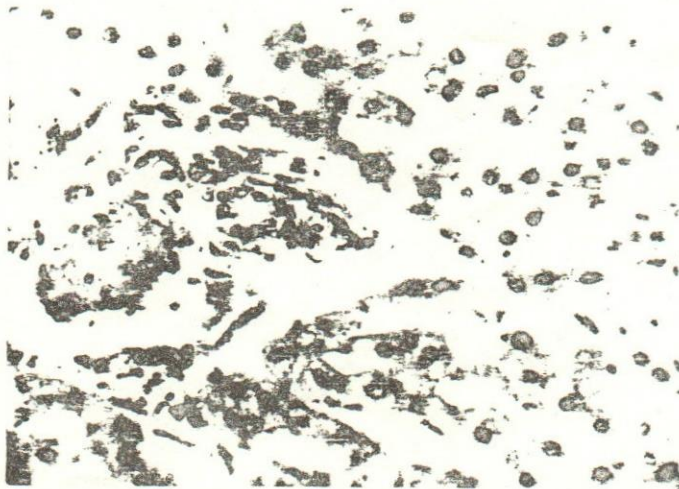
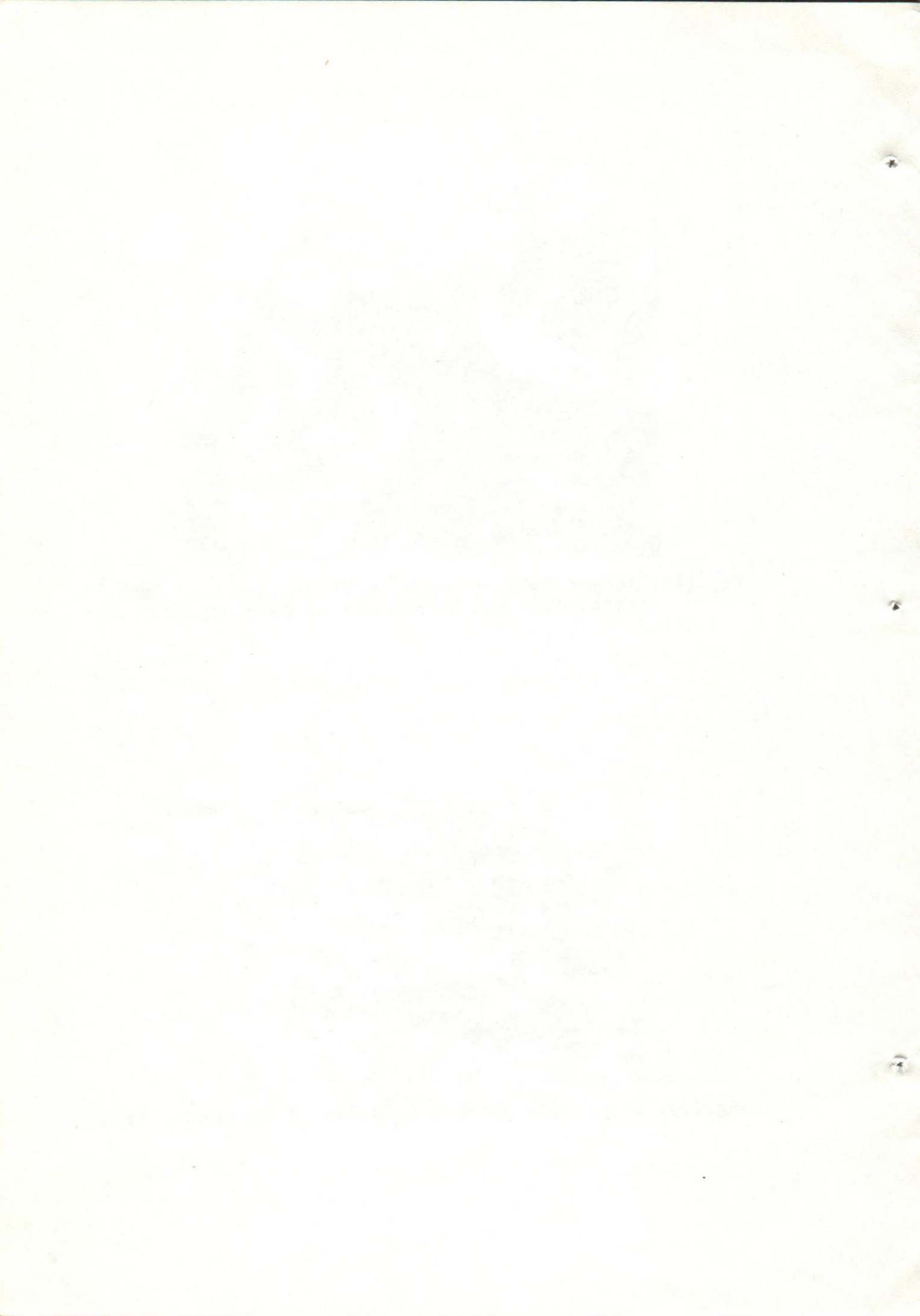


Fig.(2): Liver: 40x shows congestion of the portal veins





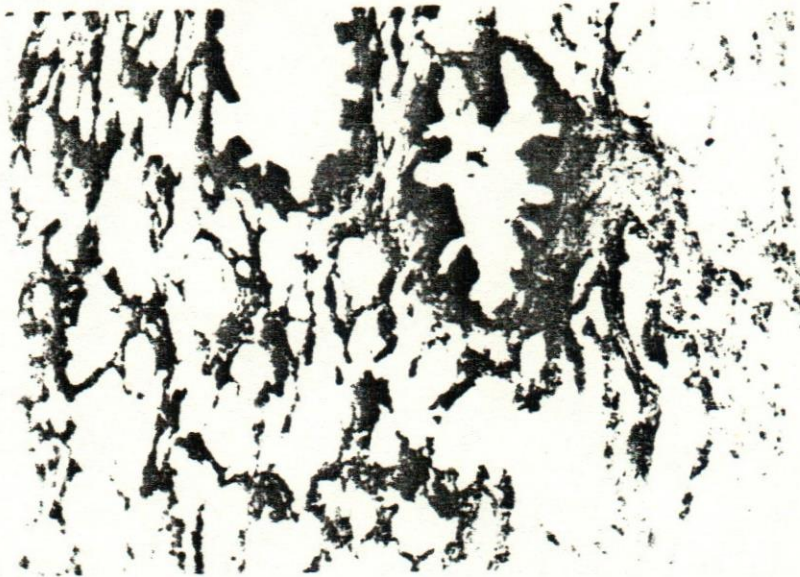


Fig.(3): Lung: 40x, shows squamous metaplasia in bronchiolar and alveolar walls with prebroncheal proliferation of histogenic cells.

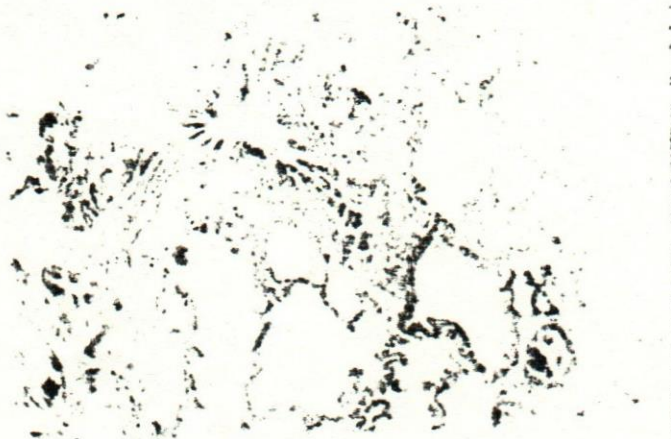


Fig.(4): Lung: 10x shows shredding of bronchial cells.

