## STUDIES ON DIFFERENT CLINICAL DIAGNOSTIC TOOLS USED IN KHARTOUM STATE

BY

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بسم الله الرحمن الزحيم

## Dedication

To my parents To the soul of my grand parents to my brothers and sisters and my beloved children and husband G dedicate this work

Qarashat Alssarraj

#### **CONTENTS** Title Page Acknowledgement..... 1 iii Abstract Arabic abstract. vi INTRODUCTION AND OBJECTIVES..... 1 CHAPTER ONE: LITERATURE REVIEW..... 3 3 1.1 Goats..... 1.1.1 Geographical distribution of goats and sheep..... 3 1.1.2 Preferred Environment for Goats..... 3 1.1.3 Feeds and Feeding..... 4 1.1.4 Nutrient Requirements of Goats..... 4 4 1.1.4.1 Water Requirements..... 1.1.4.2 Dry Matter Intake..... 4 5 1.1.4.3 Energy..... 5 1.1.4.4 Protein 5 1.1.4.5 Minerals..... 1.1.4.6 Vitamins..... 6 1.1.5 Goats in The Sudan..... 6 7 1.1.5.1 Nubian Goats.... 7 1.1.5.2 Sudanese Desert Goats..... 1-1-5-3 Southern Sudan Goat..... 7 1.1.5.4 Mountain Goat. 7 1.1.6 Diseases and Parasites..... 8 9 1.2 Diagnosis.....

1.2.1 Allergic reactions.....

1.2.2 Clinical chemistry.....1.2.3 Bacteriological examination.....

1.2.4 Parasitological

1.2.5 Serological examinations.1.2.6 Hematological examination.

1.2.7 Analysis for poisons.....

1.2.9 Mechanical aids to diagnosis.....

1.2.8 Soil and Herbage.....

1.2.10 "Test therapy".....

1.3 Mastitis1.3.1 Definition and History

1.3.2 Clinical Signs.....

1.3.3 Diagnosis

1.3.4 Treatment.....

11

11

11

11 12

12

12

12

13

13 14

14

14

15

18

1.4 Pneumonia.			
1.4.1 Definition			
1.4.1.1 Contagious caprine pleuropneumonia (ccpp)			
1.4.1.2 Definition	22		
1.4.2 History	22		
1.4.3 Clinical signs	22		
1.4.4 Gross Lesions			
1.4.5 Diagnosis	24		
1.4.5.1 In the field	24		
1.4.5.2 Definite diagnosis	25		
1.4.5.3 Biochemical Tests	25		
1.4.5.4 Growth Inhibition Test	26		
1 4 5 5 Fluorescent Antibody Test	27		
1 4 5 5 Serological Tests	27		
1 4 5 7 Molecular Diagnosis	28		
1.4.6 Treatment for CCPP	20		
	2)		
CHAPTER 2: MATERIAL AND METHODS			
2 1 Animals	30		
2.1.1 Clinical cases (Group $\Delta$	30		
2.1.1 Chinear cases (Group R.	30		
2.1.2 Survey Group (Group D)	35		
2.1.3 Experimental Animals for Pneumonia group $(C = I)$	35		
2.1.3.1 Experimental Animals for Magtitis Group $(C = I)$			
2.1.5.2 Experimental Annuals for Masture. Group (C –II)			
2.2 Clinical examination.			
2.2.1 Detailed Clinical Examination for the Chest Region			
2.2.1.1 Inspection			
2.2.1.1.2 Nasal discharge and cougn	40		
2.2.1.1.3 Lymphatic glands			
2.2.1.2 Physical examination of the chest			
	4/		
2.2.1.2.2 Percussion	4/		
2.2.1.2.3 Auscultation	48		
2.3 Collection of Samples	54		
2.3.1 Blood Sampling			
2.3.3Clinical hematology			
2.3.3 Red blood cell picture			
2.3.3.1 Hemoglobin Estimation			
2.3.3.2 Red cell count.			
2.3.3.3 Micro-pipette Method of Dilution	55		
2.3.3.4 Preparation and Filling of the Counting Chamber			
2.3.3.5 Counting Procedure	57		

2.3.3.6 Calculations	57	
2.3.4 White Blood Cell Picture	58	
2.3.4.1 Total White Cell Count	58	
2.3.4.2 Micro-pipette Method.	59	
2.3.4.3 Counting Procedure	59	
2.3.4.4 Calculations	60	
2.3.4.5 Differential White Cell Count	60	
2.3.4.6 Staining Of Blood Film.		
2.3.4.7 Counting Technique.		
2.4 Experiments	64	
2.4.1 Animals Group C	64	
2.4.2 Sub group C-I	64	
2.4.2.1 Experimental infection of goats with pneumonic agents	64	
2.4.2.2 Source of bacteria.	65	
2.4.2.3 Way of administration	65	
2.4.3 Sub group (C – II)	65	
2 4 3 1 Collection of samples	65	
2 4 3 2 Clinical examination	70	
2 4 3 3 Bacteriology	70	
2 4 4 Treatment	74	
2.4.4.1 Drugs selected and used for both sub-groups C-I and C-II	74	
2.4.4.2 Drugs selected for treatment of mastitis.	74	
2 4 4 2 1 Local treatment	74	
2.4.4.2.7 Documentation		
2.4.4.3 Observation		
	, 0	
CHAPTER THREE: RESULTS		
3 1 Results of the first stage of the study	77	
3.1.1 Information Collected from Khartoum Veterinary Hospital	77	
3.1.1.1 Diagnostic methods other diagnostic procedures laboratory		
aidsto diagnosis	77	
3 1 1 2 Animals	77	
3 1 1 3 Diseases	77	
3.1.2 Information collected from Shambat Veterinary clinic	80	
3.1.2.1 Diagnostic methods other diagnostic procedures and	00	
laboratory aids to diagnosis methods	80	
3.1.2.2 Animals	83	
3.1.2.3 Disease	83	
3.2 Results from the second stage of the study (Survey)	85	
3.3 Result from the third stage of the study.	96	
3.3.1 Pneumonia experiment.	96	
3.3.1.1 Result from attempt 1	96	
3.3.1.2 Results from attempt 2	96	

3.3.2 Results from mastitis experiment		
3.3.2.1 Infected cases.	96	
3.3.2.1.1 case 1 (Animal A)	103	
3.2.1.1.1 History	103	
3.2.1.1.2 Clinical findings	103	
3.2.1.1.3 Bacteriology	103	
3.2.1.1.4 Treatment	103	
3.2.1.1.5 Response to treatment	103	
3.2.1.2 Case 2. (Animal B)	104	
3.2.1.2.1 History	104	
3.2.1.2.2 Clinical finding	104	
3.2.1.2.3 Bacterilogy	104	
3.2.1.2.4 Treatment	104	
3.2.1.2.5 Response to treatment	104	
3.2.1.3 Case 3 (Animal C)		
3.2.1.3.1 History		
3.2.1.3.2Clinical finding		
3.2.1.3.3 Bacterology		
3.2.1.3.4 Treatment	106	
3.2.1.3.5 Response to treatment	106	
3.2.2 Observation	106	
CHPTER FOUR: DISCUSSION	115	
CHAPTER FIVE: CONCLUSIONS AND RECOMMENDATIONS		
Conclusions	121	
Recommendations	122	
REFERENCES	123	

### LIST OF TABLES

Table	Title	Page
1	Daily clinical check for animals' sub group C-I (pneumonia)	
2	Groups for blood samples collection	
3	Blood check table for animals of sub group C-I (pneumonia)	
4	Data for animals 428, 437, 439 at the day of taking swaps for	
	creation of highly concentrated solutions of bacteria used for	
	experimental infection of pneumonia	
5	Blood data for animals 428, 437, 439 at the day of taking	
	swaps for bacterial gross and creation latr on highly	
	concentrated solutions used for experimental infection of	
	pneumonia.	
6	Analysis of the symptoms and clinical signs of the chief	
	respiratory diseases	
7	Compiled Heamatological values for adult sheep and goat -	
	means and distribution (standard for experiments in the	
	present study)	
8	Parameters took from animals' sub group C-II (mastitis) after	
	the beginning of treatment	
9	Daily check for the udder size, the milk quantity and the	
10	temperature	
10	Blood picture for the 3 animals sub group C-II (mastitis)	
11	before and after treatment	
11	Survey cases, species, gender, temperature, Hb gm, Hb%,	
10	RBCs, WBCs, complain, and treatment.	
12	2 Results from microbiological check for swabs collected	
12 1	randomly from sub group C-1 (trial 1)   1 Dlood chock result for onimals' sub-group C-1 (consumption)	
<b>13-1</b> Blood check result for animals sub group C-1 (pneur Experimental infection of pneumonic (attempt 1)		
	treatment	
13-2	Blood check result for animals' sub group C-I (nneumonia)	
15 2	Experimental infection (trial 1) – after treatment	
14	Parameters for animal C (sub group C-II mastitis) after one	
	month observation and develop of mastitis from chronic to	
	per acute	

### **LIST OF FIGURES**

Figure	Title	Page		
1	Survey exhausted ram suffering from mineral deficiency			
1	Survey, exhausted ram, surrering from inneral deficiency.			
2	Survey goat suffering from internal parasite			
2	Aborof area – Omdurman (caprine)			
3	Survey goat suffering from internal parasite			
5	Addibagha area – Omdurman (caprine)			
4	Survey, goat suffering from jaw abscess.			
	Shambat area (caprine)			
5-I	Survey, suspected heart water goat.			
	Shambat area (caprine)			
5-II	Survey.oedemated heart – suspected heart water. (caprine)			
5-III	Survey, enlarged gall bladder – suspected heart water goat.			
	Shambat area (caprine)			
6-I	Survey, allergic dog (itching in ears)			
6-II	Survey, allergic dog (skin allergy in all the body)			
6-III	Survey, allergic dog (general vision) before treatment.			
6-IV	Survey, allergic dog after treatment.			
7	Clinical, goat suffering from pneumonia			
	Shambat Vet Clinic, U of K			
8-I	Clinical, skin abscess (ovine) – veterinary clinic Shambat – U of			
	K			
8-II	Clinical, skin abscess (ovine) – closer vision			
9-I	Clinical, wounded udder			
9-II	Clinical, wounded udder (caprine) – Shambat Vet Clinic – U of K			
10	Clinical, (Abu regeba) symptoms of minerals deficiency (ovine)			
	Shambat Vet Clinic – U of K			
11	Experimental, sub group C-I (pneumonia), control group			
12	Experimental, sub group C-II (mastitis)			
13	Experimental, stable (housing) – sub group C-I (pneumonia)			
	control group			
14	Experimental, stable (housing), sub group C-I (pneumonia)			
15	Experimental, sub group C- II (mastitis), normal milk from			
	animals A and B			
16	Experimental, sub group C-II (mastitis), apparently healthy udder			
17	Experimental, sub group C-II (mastitis), apparently healthy			
	animal			
18	Experimental, sub group C-II (mastitis), animal C ( before			

	attacked by acute mastitis)			
19	Experimental group, sub group C-II (mastitis), animal C, chest			
	oedema			
20	Experimental animal, sub group C-II (mastitis), animal C,			
	asymmetric in udder			
21	Experimental group, sub group C-II (mastitis), animal C, normal milk			
22 Experimental, sub group C-II (mastitis), animal C				
	Left: normal milk from not affected right half udder			
	Right: blood tinged milk from affected left half udder			
23	Experimental, sub group C-II (mastitis), animal C (apparently healthy udder)			
24	Experimental, sub group C-II (mastitis), animal C (mastitic udder)			
25	Clinical, Khartoum veterinary hospital, distribution (by percentage) of sick animals that brought to the hospital in the period from 1 April to 2 June 2003			
26	Clinical, distribution of diseases affecting goats brought to			
	Khartoum Vet Hospital (by percentage) within the period from 1			
	April to 2 June 2003			
27	Clinical, distribution of diseases affecting dogs brought to			
	Khartoum Vet Hospital (by percentage), within the period from 1			
20	April to 2 June 2003			
28	Khartoum Vet Hospital within the period from 1 April to 2 June 2003			
29	Clinical, distribution of diseases (by percentage) affecting goats			
	brought to Shambat Vet Clinic within the period from 12 August			
	2003 to 10 April 2004			
30	Temperature for experimental animals sub group C-I			
	(pneumonia) in the period from 17 October to 10 November			
	2004, 17 – 26 October 2004			
31	Temperature for experimental animals sub group C-I			
	(pneumonia) in the period from 1/ October to 10 November			
22	2004, 27 October – 5 November 2004			
32	(nnoumonia) in the period from 17 October to 10 Nevember			
	(pneumonia) in the period from 17 October to 10 November 2004 6 11 November 2004			
33	Heart rate for experimental animals sub group $C_{-1}$ (nneumonia)			
55	in the period from 17 October to 12 November $17 - 26$ October			
	2004			
34	Heart rate for experimental animals sub group C-I (pneumonia)			
	in the period from 17 October to 12 November, 27 October – 5			

	November 2004	
35	Heart rate for experimental animals sub group C-I (pneumonia), in the period from 17 October to 12 November, 6 – 12 November 2004	
36	Pulse rate per minute for experimental animals sub group C-I in the period from 17 October to 12 November, $17 - 26$ October 2004	
37	Pulse rate per minute for experimental animals sub group C-I in the period from 17 October to 12 November, 27 October – 5 November 2004	
38	Pulse rate per minute for experimental animals sub group C-I in the period from 17 October to 12 November, 6 – 12 November	
39	Respiratory rate per minute for experimental animals sub group C-I in the period from 17 October to 12 November 2004, $17 - 26$ October 2004	
40	Respiratory rate per minute for experimental animals sub group C-I in the period from 17 October to 12 November 2004, 27 October – 5 November 2004	
41	Respiratory rate per minute for experimental animals sub group C-I in the period from 17 October to 12 November 2004, $6 - 12$ November 2004	
42	Day 6 was the last day before treatment, day 8 was a day after beginning treatment	
43	Experimental, variations of the milk quantity from the 3 animals' sub group C-II (mastitis)	
44	Case record	

### LIST OF ABBREVIATIONS

СА	Calceium
СВРР	Contagious Bovine Pleuropneumonia
ССРР	Contagius Caprine Pleuropneumonia
CFT	Compliment Fixation Test
СМТ	California Mastitis Test
ELISA	Indirect Immunosorbant Assay
FAO	Food and Agriculture Organization
FAT	Fluorescent Antibody Test
GIT	Growth Inhibitin Test
НАТ	Haemagglutination Test
IDF	International Dairy Federation
IFAT	Indirect Fluorescent Antibody Test
IHAT	Indirect Haemagglutination Test
LAT	Latex Agglutination test
MIC	Minimum Inhibitory Concentration
MWT	Modified White Side Test
NRC	National Research Council
Р	Phosphorus
PCR	Polymerase Chain Reaction
RNA	Ribosomal Nucleic Acid
SCC	Somatic Cell Count
U OF K	University of Khartoum
Vet	Veterinary

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#### ABSTRACT

This work was directed to investigate veterinary clinical work in Khartoum State and to evaluate the diagnostic methods and laboratory aids to diagnosis methods and tools in Khartoum Veterinary hospitals and Clinics with emphasis on caprine.

He study is based on three elements; the animal, diagnosis and laboratory aids to diagnosis methods and tools, and the disease. It consisted of three parts.

The first partwas atraining period in which informations and observations were collected, it was done in Khartoum Veterinary Hospital (2 months)and Shambat Veterinary Clinic (8 months). Findings were: Diagnostic methods used by Practitioner (Veterinarian) for clinical examination were auscultation and some special diagnostic procedures needed as the use of vaginoscope.

Laboratory aids to diagnosis were:

Parasitological examination Haematological examination Bacteriological examination

Animals cheched in Khartoum hospital:

The tptal number was 136, 71 animals (52% of these cases) were caprine, 37 were dogs (27.21 %), 20 cases were ovine (14.71 %), and 6 cats (4.41).

Pneumonia was the most common disease for the 4 species of animals examined.

Animals clinically checked, laboratory examined, diagnosed and treated, were 157 cases of caprine and ovine at Shambat clinic (College of Veterinary Medicine U of K); 81.01 % of them were caprine and 18.4 % were ovine.

The most common disease among all these cases was pneumonia 37.03 of the cases were suffering from it, and 25 % were suffering digestive system problems.

In the second part 35 goats and sheep were clinically checked, haematological and parasitological examinations were made for blood and faecal samples collected from them. These animals were from different areas in Omdurman and one farm in Shambat.

Most of these cases suffered from anaemia, internal parasites and pneumonia; one goat died before a diagnosis was made, one sheep died after the beginning of treatment and the rest of the cases showed good response to treatment.

He last part consisted of 2 experiments; the time for each was 2 months. The first one was designed to test the available diagnostic methods and laboratory aids to diagnosis of pneumonia; 12 goats aged 3-6 months were used. Experimental infection of pneumonia and observation of symptoms by routine clinical examination was done. Later test therapy was adopted as an aid to diagnosis.

The second experiment was designed for the same purpose to diagnose mastitis. Three lactating nannies aged between  $1\frac{1}{2} - 3\frac{1}{2}$  years were observed through daily routine clinical examination. 2 of these nannies were infected with sub clinical mastitis and showed good response to treatment, the third one showed clinical symptoms of mild mastitis and a little response to treatment.

In conclusion, it was found that the present used diagnostic methods and tools in Khartoum Hospitals and Clinics is not coping with the recent scientific advances and developments in techniques.

The study recommend more depth and more attention for rehabilitation of the present veterinary hospital care, introduction of more recent equipments and tools and up-dating the present diagnostic procedures and techniques.

#### ملخص الأطروحة

كان الغرض الأساسي من هذه الدراسة هو تقييم الآليات والطرق المستخدمة في التشخيص العيادي البيطري (الأكلينيكي) للأمراض في مدينة الخرطوم وعليه فقد ارتكزت الدراسة على البحث في ثلاثة عناصر وهي الحيوان – الآليات والمعدات الطبية المستخدمة ثم أخيراً المرض. وقد كانت من ثلاثة أجزاء.

الجزء الأول كان بمثابة فترة تدريبية تجمع فيها المعلومات والملاحظات المتعلقة بالدراسة قد تمت في مستشفي الخرطوم البيطري التعليمي (شهرين) وعيادة شمبات – كلية البيطرة – جامعة الخرطوم (8 شهور) ووجدت الدراسة الآتي:

الأدوات التى يستعملها الطبيب البيطرى في الفحص الإكلينيكي على الحيوان المريض هي السماعة الطبية Vaginoscope والثير موميتر وقفازات بلاستيكية وما تتطلبه الحالة مثلاً منظار مهبلي Stethoscope وكذلك بعض آليات التحكم في الحيوان مثل اللواشة وفاتح الفم

أما بالنسبة للكشف المعملي المساعد فكان:

Parasitological Examination فحص طفيلي

Bacteriological Examination فحص جر ثومي

Hematological Examination فحص الدم

بالنسبة لحالات الحيوان التى تم فحصها وتشخيص المرض فيها في الفترة التدريبية في المستشفي البيطري التعليمي الخرطوم كان عدد الحالات (136) منها 52ز 21% (71 حالة) من الماعز، 27ز 21% (37 حالة) من الكلاب، 14ز 71% (20 حالة) من الضأن، 4.41% (6 حالات) من القطط، وكان (37 حالة) من الكربية هو التهابات الجهاز التنفسي.

في عيادة شمبات البيطرية جامعة الخرطوم قمنا بالفحص العادي والمعملي ومتابعة العلاج في (157 حالة) من الماعز والضأن و (128 حالة) من الماعز (18ز 00% من عدد الحالات الكلي ومنها 84.4% من الإناث و 6.51% من الذكور) و (29 حالة) من الضأن (18ز 4% من الحالات). من حالات الماعز وهي نسبة الحالات هما الذكور) و (20 حالة) من الضأن (18ز 4% من الحالات). من حالات الماعز وهي نسبة الحالات من الذكور) من الحالات من الإناث من الخالات الماعز من الخالي ومنها 4.8% من الإناث من الخالات الكلي ومنها 4.8% من الإناث من الإناث من الذكور) و (29 حالة) من الضأن (18ز 4% من الحالات). من حالات الماعز وهي نسبة الحالات من الذكور) من الحالات من الضأن (18ز 4% من الحالات). من حالات الماعز وهي نسبة الحالات من الذكور) من الحالات تعاني إصابات في الجهاز التنفسي من الأعلي من بقية الحالات حيث كانت 25% من الحالات تعاني إصابات في الجهاز الهضمي و 17.9% من الحالات تعاني من الحالات تعاني من الحالات كانت تعاني من التهاب من التهاب من التهاب

الضرع و 7.03% من الحالات تعاني من التهاب المفاصل و 3.125% من الحالات تعاني من التهاب وإصابات في المسالك البولية.

في الجزء الثاني قمنا بالفحص العيادي والمعملي وعلاج 35 حيواناً من الضأن والماعز في مناطق متفرقة من ام درمان وكذلك مزرعة في شمبات وكانت معظم الحالات تعاني من فقد الدم، الطفيليات الداخلية وكذلك أمراض الجهاز التنفسي، ونفقت حالة من الماعز قبل مباشرة العلاج ونفقت حالة من الضأن بعد أعطاء الجرعة الأولى من العلاج، أما بقية الحالات فقد شفيت (إكلينيكيا).

الجزء الثالث: أحتوى على تجربتين كل تجربة استغرقت شهرين. التجربة الأولي أحدثت فيها الإصابة بالالتهاب الرئوي مرتين، عولجت في المرة الأولى مجموعة ولم تعالج الأخرى، كانت هناك أعراض كالإفراز ألأنفي وكحه وادماع وارتفاع في درجات الحرارة وقد استجابت الحيوانات المعالجة للعلاج والتى لم تعالج تماثلت للشفاء تلقائياً.

في المرة الثانية أحدث المرض بجراثيم معزولة من نفس الحيوانات قبل العلاج وقد استجابت للإصابة بحيث أظهرت أعراض إكلينيكية بسيطة وقد اختفت تلقائياً بدون علاج.

اما التجربة الثانية فقد اقتصرت على مراقبة عدد ثلاث معزات حلوب تتراوح أعمارها ما بين 11⁄2 سنة إلى 21⁄2 سنة أثبت الفحص العيادي لإحداهن إصابتها بالتهاب الضرع والتي اثبت التزريع البكتيري والحاليين 21⁄2 سنة أثبت الفحص العيادي لإحداهن إصابتها بالتهاب الضرع والتي اثبت التزريع البكتيري والحالتين Staph oreous و كما اثبت إصابة الأخريات بـ Staph oreous إلا خرين استجابتا للعلاج (اثبت ذلك الفحص البكتيريولوجي).

الحالة الأولى لم تستجب للعلاج فقد أثبت ذلك الفحص البكتيري وتحولت الحالة إلى التهاب ضرع مزمن ونقصت كثير اص كمية اللبن بعد شهر من تاريخ العلاج تحول الالتهاب المزمن إلى التهاب فوق الحاد وعولت كثير اص كمية اللبن بعد شفى الحيوان إكلينيكياً لكن تحجر معظم الضرع (تحجر في النصفين).

وأخيراً وضح أن المعدات المستخدمة في التشخيص الإكلينيكي والطرق لا تتواكب مع التغيرات التى تحدث يومياً في العام، حيث يومياً تظهر أمراض جديدة، تكتشف عثرات جديدة للفيروسات وتكتشف جراثيم ممرضة جديدة وكذلك تكتشف آليات تشخيصية للأمراض جديدة وعليه نوصى بالمزيد من الاهتمام في هذا الحقل.

#### **INTRODUCTION AND OBJECTIVES**

The term clinical diagnosis refers to the combination between the:

- Clinical examination (which is performed by means of senses of sight, touch, hearing and smell, and comprises two major parts: (a) the initial inspection, and the general and systemic examination of the animal. (b) The physical examination of the animal.) And
- 2. Suitable aid to diagnosis method, which is performed through laboratory examination by specialized technicians of a mechanical nature.

If these two parts used intelligently then a confirmative, precise and correct diagnosis can be obtained.

Although mastitis occurs sporadically in all species, it assumes major economic importance mainly in milk goats, late or wrong diagnosis considered to be one of the main reasons leading to chronic mastitis in them.

Deficiency of modern diagnostic and laboratory aids to diagnosis tools and methods is the most important problem affecting the field of veterinary owing to its significant effect on clinical diagnosis. In the Sudan the problem has become one of the major problems in recent years, given the fact that no confidence between animals' owners and veterinary governmental foundations. Although there are a considerable number of studies on many topics in many veterinary fields, very little attention has been paid to the role of clinical diagnosis in the reality of the veterinary field.

In the Sudan there are no any governmental or non governmental reports about the situation of the diagnostic and the laboratory aids to diagnosis methods and tools which were used, are used, and supposed or planned to be used in the future.

The main objective from this study was to find out what clinic work in the Sudan is, and what diagnostic tools are used and to try to evaluate it. For this purpose survey in veterinary hospitals and clinics, animals' markets, and visits to animals owners' houses were made. That was half the other half was evaluation for diagnostic tools through experiments.

# CHAPTER ONE LITERATURE REVIEW

#### 1.1 Goats

#### 1.1.1 Geographical distribution of goats and sheep

Both goats and sheep are widely distributed, from arid semi-desert to humid rainforest region, and represent 20.2 and 28 percent, respectively, of the total population of ruminant livestock in the tropics and sub-tropics. Although the total number of sheep is greater than that of goats, sheep in the tropics represent a lower proportion of the total world population. The Largest concentrations of goats are in Africa and in the Indian sub-continent. Devendra and Mcleroy. (1982)

#### **1.1.2 Preferred Environment for Goats**

The wide distribution of goats in the tropics and sub-tropics reflects their ability to adapt to a variety of environment. However, the preferred environment is on the lighter sandy soils in the drier tropics, rather than in the wet humid tropics. In the dry tropics they perform best and thrive in large numbers. The inherent characteristics of goats such as resistance to dehydration, preference for browse and wide ranging feeding habits, enable them to thrive in region that receive less than 750mm of rainfall.

In tropical Africa goats are found in the largest numbers in the drier steppe and savanna. Devendra and Mcleroy. (1982)

The dwarf goats, unlike other species, thrive throughout the humid tropics and must be specially adapted to humid environment. Devendra and Mcleroy. (1982)

#### **1.1.3 Feeds and Feeding**

Goats are very inquisitive animals, much more so than other ruminants and they can walk long distances in search of food. The wide distribution of goats is possibly due to their ability to feed on a wide variety of food stuffsmainly tree and shrub leaves and grasses. Goats can distinguish between bitter, sweet, salty and sour taste, and show a higher tolerance for bitter taste than cattle. They relish variety in their feed and do not thrive well when kept on a single type of feed for any length of time. The most important factor affecting choice of feed is the availability of a variety of feeds. Devendra and Mcleroy. (1982)

#### **1.1.4 Nutrient Requirements of Goats**

#### **1.1.4.1 Water Requirements**

Goats are efficient animals in the use of water. They have a low rate of water turn–over per unit of body weight. Ample quantities of clean water are essential for high milk production by lactating goats and for maximum growth and mohair production. The water requirements of meat animals are relatively less. Devendra and Mcleroy. (1982)

#### 1.1.4.2 Dry Matter Intake

The dry matter intake of goats indicates their capacity to utilize feed voluntarily. Intake depends on the breed of goat (meat or milk type) and on the environment. Dairy goats in the tropics usually consume dry matter at the rate of around 4 to 5 percent of live weight. Meat goats in the tropics seldom exceed an intake of 3 percent of live weight. Devendra and Mcleroy. (1982)

#### 1.1.4.3 Energy

The energy requirements for maintenance of goats is the same as that for sheep which for a 30 Kg live weight animal is 5.44 Mj/Kg daily. In addition to

this, goats require energy for production as well as for level of activity. Devendra and Mcleroy. (1982)

#### 1.1.4.4 Protein

In many parts of the tropics, protein rather than energy is the main limiting nutritional factor and so will need special attention. Although energy may be adequate, a deficiency of protein can interfere with voluntary feed intake and delay growth, estrus, puberty and reproduction. Devendra and Mcleroy. (1982)

#### 1.1.4.5 Minerals

Deficiencies or imbalance of minerals (macro or micro) exert a significant effect on the health and productivity of goats throughout the tropics. The mineral nutrition of goats, however, one of paucity Heinlein (1980). Adequate P is critical for normal reproductive function and deficiencies are likely to occur under range condition. Calcium is essential for normal body function. A Ca: P ratio of 1.2: 1.0 is recommended NRC (1981). Manganese deficiency may affect Ca utilization. Deficiencies of sodium chloride and potassium reduce feed intake, cause heat stress and a poor hair coat and therefore the performance of the animals. Concentrate supplements should contain 1 percent salt which is also a carrier of trace minerals such as zinc, manganese, iodine, cobalt, iron, molybdenum, copper and sulpher. Lactating goats require additional minerals Devendra and Mcleroy. (1982)

#### 1.1.4.6 Vitamins

Vitamin A and its precursor, carotene, are of particular importance for normal spermatogenesis in goats. Green forages and dried green forages are good sources of vitamin A. Vitamin D can affect Ca and p utilization leading to unthrifty kids. Vitamin E has beneficial role in reproduction and in preventing off-flavors in milk. The B vitamins are synthesized by rumen microbes Devendra and Mcleroy. (1982)

#### 1.1.5 Goats in The Sudan

In 1994-95, there were 33 million goats in the Sudan (on the net). Nomads in rural areas keep quite large flocks, but most low income groups both in rural and urban areas including the national capital (Research Area) keep a few goats in their house-holds for local milk consumption Food Agriculture Organization, FAO (1985).

Goats are considered to be the ruminant with the widest distribution in the Sudan near from the area of the river Nile, Desert areas, Rich savanna and Poor Savanna zones and the humid tropics in south Sudan. Producing life time of goats is about 8 years; it gives birth for 5 times within this period. Abas Belal

There are 4 types of goats in the Sudan according to Mason, Maul (1960) and Mufarah (1980), Fawzi (1970), Hassan and others (1970). These types are: The Nubian, The Desert, The Southern Sudan goat and Mountain goats. Madany. (Arabic rev. 1969)

#### 1.1.5.1 Nubian Goats

The Nubian is a distinctive breed, and although it originates in the Sudan it is widely distributed and found in many parts of Africa. It is the only African breed that has been selected for milk production.

It represents more than 50% of the total number of goats that are found in the Sudan Madany (Arabic rev. 1969).

#### 1.1.5.2 Sudanese Desert Goats

This type of goats is the Northern Sudan goats except that areas of river Nile, the cities and villages. It is found in the Desert area some times mixed with the Nubian goats. Mainly found with the Nomads tribes in the savanna zones especially in Darfur and Kurdufan. It can resist harsh conditions such as thirst that it can live on the tree leaves which grow in hills and sandy areas. It is said that it is originally from the goats of west Africa Madany (Arabic rev. 1969).

#### 1.1.5.3 Southern Sudan Goat

It is also called the Nilotic goats. It is originally from Dwarf goats of Africa. They are indigenous to the forest and drive savanna zones of west Africa and are well adapted to the humid zone. The Southern goats is small in size with a strong body (compact) Madany (Arabic rev. 1969).

#### 1.1.5.4 Mountain Goat

It is found in Jibal Annuba south Kurdufan – Al Angasana Mountains south of Al Jazzier, Jabal Marra west Darfur. It is also African Dwarf goat exactly like the Nilotic goat. It has short legs, short hair and short ears and is characterized with the quick movements and jumps Madany (Arabic rev. 1969).

#### **1.1.6 Diseases and parasites**

Wastage due to diseases and parasites is a source of serious economic loss and one of the main constraints to the development of goats. The disease incidence also becomes greater where a low level of nutrition causes reduced resistance. Poor sanitation and hygiene also affect the health and performance of goats.

Three categories of diseases affect goats, infections, non-infections and parasites. The infections disease includes peste due petits ruminants (PPR), contagious caprine pleuro-pneumonia and hemorrhagic septicemia. Non – infection diseases refer to various metabolic disorders, lameness and joint

infection. Parasites, especially internal parasites such as helminthes, seriously affect goats. Regular drenching is essential, using broad-spectrum drenche Payne (1990).

#### **1-2 Diagnosis**

Diagnosis is the foundation of both treatments of sick animals and of methods designed to control, eradicate and prevent animal disease. The importance of the veterinary profession is very largely dependent on the ability of its members to formulate an accurate diagnosis in respect of each and every disease problem. It can therefore be said that diagnosis is the principal thing Boddie (1969).

Diagnosis of disease comes from observation of symptoms and changes of the tissue of the animal. Some symptoms are characteristic of specific disease such as muscle spasms in tetanus and paralysis in milk fever, but more after a disease is identified only after a careful clinical examination of the animal or animals and after post-mortern and laboratory examination of smears and specimens have been made FAO (1985).

The basis of diagnosis is accurate in systemic case taking, without accuracy then method important points will be missed Kelly (1984).

The case record is a written statement of the information obtained by case taking. This record should be brief and concise, so that the salient points are immediately obvious. There are various methods of case taking and recording, all probably have merit. Perhaps the most important point to emphasis is that a method should be adopted and consistently followed in order that the habit of methodical examination can be developed. Any method of case recording should have a reasonable degree of flexibility so that the user may adapt it to the needs of the individual case Boddie (1969).

The basis of the study of clinical methods must be a sound knowledge of structure and function of the normal animal combined with a similar knowledge of the causation of the disease. Structure and function include anatomy, physiology and animal behavior. Causation of disease includes pathology, microbiology, parasitalogy and knowledge of environment and feeding. The veterinary clinician must possess an intimate appreciation of the appearance and behavior of healthy normal animals of the various species and breeds Boddie (1969).

A straight forward clinical examination of the patient may in a proportion of cases reveal the nature of the malady from which the animal is suffering. These are, however, cases in which it is necessary to use one or more of the aids to diagnosis that are available. It cannot be too strongly emphasized that the various laboratory and specialized techniques of a mechanical nature that are available are aids to diagnosis and are not short cuts. If used intelligently these aids can be of great assistance and differential diagnosis, used indiscriminately they merely serve to increase confusion Boddie (1969).

The decision to use an aid to diagnosis must be based on the results obtained by clinical examination, the analysis of which indicates that a particular aid or aids may assist in diagnosis. This obviously implies knowledge of what information can be derived from an aid and how the results should be interpreted in the light of knowledge of the clinical findings. Some of the aids to diagnosis can be applied by the practitioner himself; others require special apparatus of the facilities of a fully equipped laboratory Boddie (1969).

There are now available a vast variety of aids to diagnosis. It is sufficient to the present purpose only to indicate the wide range of these with the object of emphasizing that this discrimination in their use is essential. It is convenient to group the test according to the type of information they furnish Boddie (1969).

#### **1.2.1 Allergic reactions**

These tests are applied to the individual animal. Today they are almost invariably used in the form of a skin test, that is to say, an interdermal test. Their chief use is in detection of sub clinical cases of a specific infection and are essential feature of disease eradication program (as a general rule, allergic tests are of little value in the differential diagnosis of specific disease in the individual clinically affected animal) Boddie (1969).

#### **1.2.2** Clinical chemistry

Urine analysis is of a considerable value in the diagnosis of disease of the urinary system, estimation of the blood content of certain constituents such as urea, glucose, calcium and magnesium may be of value in relation to the sick animal, but also are of importance in assessing the position in a herd or flock Boddie (1969).

#### **1.2.3 Bacteriological examination**

The identification of causal organisms can in a number of instances be achieved by microscopic examination of stained smears. In order to differentiate between the various forms of mastitis a bacteriological examination of the secretion from the affected udder is required. This is usually combined with tests for antibiotic sensitivity of the organisms concerned Boddie (1969).

#### **1.2.4 Parasitological**

Microscopic examination of skin scrapings permit the identification of ectoparasites such as manages mites, but cultural methods are often needed to identify the causal fungi of ringworm. Eggs of parasite helminthes can, within limits, be identified and the number of eggs in a given weight of faces can be estimated Boddie (1969).

#### **1.2.5 Serological examinations**

A serological examination requires the facilities and equipment to be found in a bacteriological laboratory Boddie (1969).

#### **1.2.6 Hematological examination**

A simple estimation of the hemoglobin level may be sufficient to confirm a tentative diagnosis of anemia. A more extensive hematological examination will be required to indicate the type of anemia and its possible cause. Investigation of white blood cell picture may indicate the type of response associated with the illness and may indicate a defensive reaction to infection. Information of this type is neither specific in relation to the type of infection nor is it indicative of the site of infection in the body Boddie (1969).

#### **1.2.7** Analysis for poisons

Confirmation of a tentative diagnosis of poisoning can only be based on the chemical identification of the suspected poison in material obtained from the animal and it is described to establish that the particular poison is present in the material thought to be the source Boddie (1969).

#### **1.2.8 Soil and Herbage**

Confirmation of trace element deficiency, e.g., cobalt deficiency, is based on a spectrographic analysis of the cobalt content of representative samples of herbage, soil analysis is desirable to ascertain whether, if cobalt is present in the material thought to be the source Boddie (1969).

#### **1.2.9** Mechanical aids to diagnosis

Radiological examination. A radiological examination of animals for purely medical reasons is largely confined to smaller animals such as dogs and cats, but with modern X-ray plants and the scope is being extended to larger animals Boddie (1969).

### 1.2.10 "Test therapy"

Success with group therapy could only be accepted as conclusive if untreated controls of the group failed to show any spontaneous recovery Boddie (1969).

#### 1.3 Mastitis:

#### **1.3.1 Definition and History**

Mastitis was defined by Cole (1962) as an inflammation of the udder caused by infection or by undue stress factor in the mammary tissue or by both. Newbould and Neave (1965) stressed that the mere presence of pathogens in milk without an elevated cell count doesn't indicate mastitis. According to the IDF (1971) the cell count of milk in the most important criterion. Mastitis affects particularly cattle, sheep, goats and occasionally other species of animals. Buxton and Frazer (1977). Other authors have reported that the isolation of a pathogenic organism from a respected milk sample in the presence of an elevated cell count is an indication of mastitis Anderson (1983). The term mastitis also refers to inflammation of the mammary gland regardless of the cause. It is characterized by physical, chemical and usually bacteriological changes in the milk and by pathological changes in the glandular tissue Blood (1983).

#### **1.3.2 Clinical Signs**

The clinical signs of mastitis include abnormalities of milk and udder with systemic reactions sometimes. Milk abnormalities are discoloration of milk, as a blood staining or/and wateriness. Milk abnormalities can be seen also in the presence of clots or flakes. The udder tissue may show various degrees of fibrosis, swelling and atrophy, hotness and asymmetric in udder shape.

The systemic reactions are fever, general depression, sometimes anorexia. Toxemia may occur also.

The disease in sheep and goats is similar to that in cattle. Particular care is needed in the clinical examination of goats' milk because of its apparent normality when there are severe inflammatory changes in the udder Blood (1987).

#### **1.3.3 Diagnosis of caprine mastitis**

The common screening tests used in the diagnosis of bovine mastitis include clinical examination, white side tests, rapid mastitis test, somatic cell count and bacteriological examination.

These tests have been studied by various workers to see if they were applicable to caprine mastitis.

Nesbakken (1976) reported his findings on the study of cell count in goat milk. Small variations in average cell count were observed in milk samples from the same herd but there were big variations in different herds of goats had an average cell count of 720.000 cells/ml while in hand – milk herds, the average cell count was 540.000.

Kapur and Singh (1977) compared results of California mastitis test (CMT). Modified white side test (MWT) and somatic cell count (SCC) in cows, buffaloes and goats. Counts over 500.000 cells/ml (considered positive) were found in 32%, 28% and 35% of cows, buffaloes and goats respectively. CMT gave 90.98%, 97.2% and 77.5% accuracy in the same order and suggested that this test was not reliable in goats.

The 500.000 cells/ml which is regarded as the threshold for diagnosis of bovine mastitis is not an arbitrary figure, but is a mean cell counts where rising cell count co inside with deviations from normal values of sodium, potassium, chloride, lactose and whey nitrogen in milk and of milk yield and it indicates disturbance in the secretion of mammary parenchyma Reichmuth (1975). With respect to mastitis in goats, threshold of 1.000.000 or 1.500.000 cells/ml in suggested as diagnostic.

According to Smith and Roguinsky (1977), diagnosis of bovine mastitis is based on the total number of somatic cells in milk, the counts indicates the level of leukocytes. Others found that the total count in goats did not correlate with the leukocyte count, as large numbers of epithelial cells may be present in milk at the various steps of lactation. Though epithelial cells are present at times in a severely mastitic cow, in such cases the leukocyte count is proportionally increased. Hinckley and Williams (1981). In a study of the cells count in goat's milk by Petersen (1981), the geometric mean cell count of healthy goat's milk in mid lactation was  $880 \times 10^3$  cell /ml by the direct microscopic count and  $690 \times 10^3$  cell/ml by the electronic cell count. A significant correlation was found between the cell counts and SMT. In contract, the bovine milk cell count by the direct microscopic count was low than that by the electronic cell count. In a similar study by Poutrol and Lerondelle (1983) the mean cell count of healthy goat milk in early and mid lactation was found to be  $404 \times 10^3$  cell/ ml by the coulter counter and  $614 \times 10^3$  cells/ ml by the Fossomatic method.

According to Thornton (1983), cell counts in goats by coulter counter are higher than those of cows' milk because goat's milk contains small crystals deposits known as corpora Amylacae. In a comparison of the average cell count in machine-milked goat and cows, he found that in 42.4% of the goat milk samples the count was less than 500.000 cells/ ml, 22.7% was  $\frac{1}{2}$  -1 million cell/ml and 35.9% was above 1 million cell/ml, the figures for cows was 71.9%, 12.4% and 15.8% respectively. Others regards counts over 1 million in goats to be significant.

According to Lerondelle and Poutrel (1984), the threshold of  $1 \times 10^6$  cell/ml was reliable for diagnosis of mastitis at mid lactation and they were able to diagnose 72% of major pathogens by using it. Manser (1986) reported that cell counts in goats within herds showed statistically significant difference in the geometric mean cell counts of halves with different infection status. According to Siddique et al (1988) there is a correlation of CMT with the number of neutrophils/ml and that CMT can be applied to goat milk. They also reported that there were no significant difference between the mean total microscopic counts for quarters free of infection and the ones that were infected.

#### 1.3.4 Treatment

In the early times, treatment consisted of bathing the udder, repeated stripping and injection of irritant acridine dyes. In the 1930's the therapy improved by use of parental treatment with sulphonamides. Mastitis treatment entered new era with use of penicillin for veterinary use in 1945 Edwand and Brownlee (1946). It was regarded then, that the mastitis problem was solved, as most of the infections in bovine were caused by *streptococcus agalactic* and it was possible to eradicate the infection from herds. Stableforth et al., (1935), Stableforth et al., (1949). This illusion did not continue for long as co-agulase positive *staphylococci* replaced *streptococcus agalactie* as main causative agent of mastitis where its treatment remains low. The search for better antibiotic formulation continued up to now.

According to Ziv (1980) the use of antimicrobial drugs for treatment of mastitis are for:

- a. Treatment of per acute and acute clinical mastitis in the individual cow.
- b. For treatment of recurrent, non responsive sub acute clinical mastitis.
- c. For herds producing milk of low quality.
- d. As a past of mastitis control program.

According to Craven (1987) the strategy in mastitis therapy is to achieve at the site of infection antibiotic levels in excess of the known in-vitro minimum inhibitory concentration (MIC) for the causative organism and keep this for a sufficient period of time, though in addition to the pharmacokinetics properties of the drug, the pathological changes such as blockage of milk ducts and swelling can impair the distribution of the drug. The methods of therapy devised by Wilson (1969) include:

- a. Intramammary treatment of clinical cases.
- b. Parental treatment of clinical cases.
- c. Intramemmary treatment of rub clinical cases.
- d. Dry cow treatment.

e. Prophylactic treatment at calving.

The use of parental or intramammary route for treatment remains controversial. Ziv (1980) regards parental treatment better, while Wilson (1981) considers the intra mammary route more efficient but generally parental treatment is advocated when systemic signs are present. Though antibiotic treatment may be enough but supportive treatment that include isotonic fluids, anti-inflammatory agents, stimulants and vitamins are advocated in acute and per acute mastitis Robinson (1981).

The colloquium on bovine mastitis (1977) categorized therapy into 3 headings.

- a. Therapy for acute and per acute cases- where parental medication is advised, to be supplemented by the use of oxytocin and frequent milking at 1-2 hours intervals to remove toxic material and maintain duct potency.
- b. Therapy of chronic or sub-acute case usually done by dairyman using intra mammary preparations.
- c. Therapy for dry cow to eliminate chromic infection and prevent new infection.

There are currently over 50 intra mammary products Wilson (1981) but the cure rates due to coincide with the commercial reports of manufacturers. Their cure rates depend on in-vitro sensitivity tests where the bacteria get abundant nutrients and are in an active growth phase being highly susceptible to antimicrobial used, but in-vivo the conditions are different Pyörälä (1988).

The treatment of lactating quarters still posses a problem. Though cure rates for streptococci are good, the best cure rate that can be achieved for staphylococci is about 65% Blood et al., (1985).In contrast, treatment of drying off is advocated.

According to Bywater (1977) treatment at drying off is more efficient due to slow release of antibiotic in the absence of dilution by continuous milk formation and due to treatment without loss of milk.
According to Wilson (1969), in *staphylococcus* mastitis less than 30% can be treated during clinical attacks, 50% if treated during lactation when subclinical and 70% can be cured by the same antibiotic at drying off.

There are many multiple antibiotic preparation and in some of these corticosteroids and enzymes are included but these have not proved more effective than preparations containing 2 antibiotics, one of which penicillin Robinson (1981)

When dealing with treatment of mastitis, spontaneous recoveries have to be taken into account. Griffin et al., (1982) reported that in investigation on commercial farms, spontaneous recoveries often equal the number of infections eliminated by antibiotic therapy, though there recoveries follow long periods of infections.

The literature on the treatment of caprine mastitis is scanty. According to Thornton (1983), the line of treatment in cows and goats is similar. Half an intramammary tube designed for cows is recommended for goats. As the teat canal of goat is narrow, a tube with narrow nozzle should be chosen or the contents have to be transferred to a sterile syringe and be administered using a cat catheter. With holding time for milk in goats 12 hours also as recommended for cows Long et al., (1984).

### 1.4 Pneumonia

## 1.4.1 Definition

The condition is defined as an inflammation of the lungs accompanied by bronchiolitis and pleurisy and affects all animal species Blood (1979).

# 1.4.1.1 Contagious Caprine pleuropneumonia (ccpp)

# 1.4.1.2 Definition

It is an acute highly contagious disease of goats caused by a mycoplasma, and characterized by fever, coughing, severe respiratory distress, and high mortality. The principal lesion at necropsy is fibrinous pleuropneumonia

# 1.4.2 History

The fist report on CCPP in goats was made in 1902 in Kassale Anon (1902). It was stated that the disease caused heavy losses in goats and was prevalent in many parts of the Sudan. The lesions were mainly in the lungs and strongly resemble those of CBPP. The first attempt to study the disease was made by Knowles (1923). He was able to isolate a pasteurella from 4 cases. The inoculation of this organism subcutaneously into goats produce a slight local reaction and he considered it a secondary factor in the causation of the disease and postulated that the cause of the disease was an ultra visible virus.

# 1.4.3 Clinical signs

The clinical signs described for CCPP from different parts of the world have varied enormously because at least 2 different mycoplasmas have been regarded as causative agents of the disease. In many fields the clinical picture has probable been farther complicated by the presence of viruses and other bacteria (e.g., pasteurella) as part of the etiological picture. The classical disease as caused by mycoplasma F-38 is a purely respiratory illness. Fever–of 106° F (41° c), coughing, and distinct loss of vigor, affected goats have labored breathing, later they may grunt or fleet in obvious pain. Frothy nasal discharge and stringy salivation are often seen shortly before death. In the acute disease, which occurs in fully susceptible population of goats, death occurs within 7 to 10 days of the onset of clinical signs. A more chronic form of the disease is often seen in endemic areas and lead to recovery of a higher percentage of infected animals, many of them carrier of the mycoplasmas. *M. mycoides. capri* tends to cause a more generalized infection in which septicemia is frequently seen. An acute or per acute septicemia form of the disease involving the reproductive, respiratory and alimentary tracts has been described.

#### **1.4.4 Gross Lesions**

The gross lesions in classical CCPP are confined to the thoracic cavity. Pea-sized yellowish nodules are seen in the lungs in early cases, whereas in more established cases there is marked congestion around the nodules. The lesion may be confined to one lung or involve both, and an entire lobe may become solidified. The pulmonary pleura become thickened, and there may be adhesions to the chest wall. Hutcheon (1889) emphasized that the lesions of CCPP do not resemble those of contagious bovine pleuropneumonia (CBPP), in that "no thickening of the interlobular tissue occurs" a classical lesion of CBPP, he described a CCPP diseased lung as resembling a "somewhat granular looking liver " which is his description of the massive hepatization seen in CCPP lungs.

In sharp contort. *M. mycoides. Capri* has been reported to cause lesions in a wide variety of organ systems and to produce lung lesion closely resembling those seen in CBPP.

The generalized lesions described include encephalitis, meningitis, lymphadenitis, splenitis, genitourinary tract inflammations, and intestinal lesions, none of which are a feature of clinical CCPP.

The lung lesions, which resemble those seen in CBPP, are usually confined to one lung and reflect various stages of fibrinous pneumonia.

Extensive pleuritis is usually present, and various stages of hepatization and marked dilation of interlobular septa is commonly seen. The cardiac and diaphragmatic lobes are the ones most commonly involved. Some describe this as a mild from of CCPP, others argue that it is not CCPP

### **1.4.5 Diagnosis**

#### 1.4.5.1 In the field

In field, diagnosis of mycoplasme pneumonia can not be established on clinical signs or on postmortem examinations alone. In out break of clinical acute CCPP, the high mortality and typical early thoracic lesion in goats are highly indicative of *M.capricolum subsp. Capripneumoniae* infection, but all cases of caprine mycoplasmosis need additional laboratory tests to establish a presumptive diagnosis. It may be difficult to distinguish CCPP from an infection by *M.mycoides subsp mycoides* LC on *M. mycoides subsp mycoides* SC, which have a pulmonary location. In the case of *M. mycoides subsp, mycoides* LC infection, thickening of the interlobular septa may be evident. These lesions are similar to those observed in the case of CBPP. Sometimes the thickening is absent or inconspicuous and laboratory confirmation is needed.

Recently, sequestrates in the lungs of goats infected with *M. mycoides subsp. mycoides* SC have been described Kusiluka et al., (2000).

#### **1.4.5.2 Definite diagnosis**

It is made by the isolation of *M. capricolum subsp. Capripneumoniae* from clinical samples, usually lung tissue and may be a long and difficult process. The success of isolation depends primarily on the attention that is given to sample collection. The main difficulties in isolating *M. capricolum subsp, capripneumoniae* is that it grows very poorly in vitro and samples are often contaminated by other mycoplasmas. Freundt (1983), Thiaucourt et al., (1996) which are generally faster growing and overgrow *M. capricolum subsp* 

*capripmeumoniae*. In addition to this the frequent use of antibiotic therapy has impaired the cultivation of these mycoplasmas from clinical material. An immunobinding assay that detects *M. capricolum subsp capripneumoniae* in pleural fluids that over comes some of these problems has been described. Guerin et al., (1993). Liquid medium and a solid agar medium which allows the presumptive identification of *M. capricolum sub.sp capripneumoniae* by the production of colored colonies are available commercially Bashiruddin and Windsor (1998). An antigen detection system using latex coated antibiotics has also been described March et al., (2000).

#### **1.4.5.3 Biochemical Tests**

Only a limited number of biochemical tests perform a useful function as a preliminary screening system and are based on specific enzyme activities on nutritional capabilities, for instance, digitonin sensitivity distinguishes mycoplasmas from acholeplasmas, and serum digestion differentiates members of the *M. mycoides* cluster from all other small ruminants mycoplasmas Freundt (1983 b). Also, phosphate production separates *M. capricolum subsp capricolum* from other members of the cluster Bradbury (1983). Substantial metabolic differences between *M. capicolum subsp capricolum* and *M. capricolum subsp capripneumoniae* exist, but differences in glucose metabolism were described between strains of *M. capricolum rubsp capripneumoniae* Abu- Groun et al., (1994) These tests can not differentiate *M. capricolum subsp capripneumoniae* from all members of *M. mycoides* clustes Bjlske (1995) the interspecies variation in some biochemical reaction is often considerable, rendering their application values Jones (1989), Rurangirwa (1996)

#### **1.4.5.4 Growth Inhibition Test**

The growth inhibition (GI) test is the simplest and most specific, but the least sensitive of the tests available. It depends on the direct inhibition of

mycoplasma growth on solid media by specific hyperimmune serum, and detects primary surface antigens Dighero et al., (1970). The GI test is particularly useful in identifying *M. capricolum subsp capripneumoniae* because they appear to be serologically homogeneous, and antiserum to the type strain produces wide inhibition zones free of break through 0% calonis against field isolation from divers' sources. *M.capricolum subsp capripneumoniae* cross-reacts with leach 5% Bg 7, *M. equigenitalium* and *M. primatum* in the GI test, but since these cross-reactive species do not occur in goats, they present no difficulties when identifying field isolates,

However, a small proportion of *M. capricolum subsp capripneumonae* isolates also cross-react in the GI test with antiserum to *M. capricolum subsp capricolum* which may confuse the identification of field isolates. A monoclonal antibody has been produced which specifically inhibits the growth of *M. capricolum subsp capripneumoniae* but not of other members of the *M. mycoides* cluster Rurangirwa et al., (1987C). It was later demonstrated that this monoclonal antibody was not specific. Cross reactions with some strains of Bg 7 were observed with the GI test and with a strain of *M. capricolum subsp capricolum subsp capricolum* in the immunofluorescence test Beltion et al., (1994).

## 1.4.5.5 Fluorescent Antibody Test

The direct and indirect florescent antibody tests over are of the most effective, simple and rapid serological methods of identification for most mycoplasma Rosendal and Black (1972). Several forms have been described, the most commonly used one is the indirect fluorescent antibody (IFA) test which is applied to unfixed colonies on agar.

## **1.4.5.6 Serological Tests**

The complement fixation test (CFT) was used for the detection of CCPP infection Macowan (1976), MacOwan and Minette (1977), and it was to found

more specific, though less sensitive, than the indirect heamagglutination test Muthomi and Rurangirwa (1983). The latex agglutination test uses latex beads sensitized with the polysaccharide produced by M. capricolum subsp capripneumoniae in culture supernatant in a slide agglutination test Rurangirwa et al., (1987a). The use of the more defined antigen such as the polysaccharide provides greater sensitivity without cross-reactivity with sera against the other three principal caprine mycoplasmas. The indirect haemagglutination test (IHA) Cho et al., (1976) has been used for the diagnosis of CCPP Muthomi and Rurangirwa (1983). The specificity of IHA test for the *M. mycoides* cluster has been evaluated and results were found to show cross-reactivity between these organisms Jones and Wood (1988). An indirect imminosorbent assay (ELISA) was developed to screen goat sera at a single dilution of antibody to M. capricolum subsp capripreumonoae Wamwayi et al., (1989). Some problems due to cross reactions from other members of the *M. mycoides* cluster were encountered, but in spite of these, ELISA was more sensitive than CFT in detecting antibodies in serum. More recently, a competition ELISA (e- ELISA) was developed which permitted the specific detection of antibodies in sera from animal, affected y ccpp Thiaucourt et al., (1999). Analysis of field sera showed that sero-conversion did not occur in all animal, what ever test was used. The percentage positive animals in affected herds varied between 30% and 60% with this test. The test was therefore unsuitable as an individual screening test Thiaucourt et al., (1996).

### **1.4.5.7 Molecular Diagnosis**

Until recently, isolation was the only way to confirm the presence of CCPP. A DNA probe which differentiate *M. capricolum subsp capripneumoniae* from other members of the *M. mycoides* cluster was developed Taylor et al., (1992). Diagnostic system based on PCR have been developed for the rapid detection, identification and differentiation of members of the *M. mycoides* 

cluster and specific identification of *M. capricolum subsp capripreumonoae* Bashiruddin et al., (1994), Hotzd et al., (1996). The sequence of the gene for 16S ribosomal RNA has also been used to develop a PCR-based test where the final identification of *M. capricolum subsp capripneumoniae* is made dependant on the pattern of the products after digestion of the PCR product with the restriction enzyme Pst1 Bascu -ana et al., (1994), Bjlske et al., (1996).

### **1.4.6 Treatment for CCPP**

The mycoplasmas are sensitive to several broad spectrum antibiotics (notably the Tetracyclins, Tylosin, and Tiamulin)

Treatment also described in Blood (1987) as Tylosin tartrate 10 mg/kg BW or Oxytetracyclin 15 mg/kg BW daily.

The macrolids (Erythromycin, Spiramysin, and Tylosin)

Tetracycline, Quinolons and Chloramphinicol are active against *M.capricolum subspp.caprioneumoniae*. Tylosin, Tetracycline, Tiamulin or Streptomycin are recommended Hassan et al., (1984); Onovarian (1974) but their success depend on early intervention and treatment.

# **CHAPTER 2**

# **MATERIAL AND METHODS**

#### 2.1 Animals

### 2.1.1 Clinical cases (Group A)

These were all ill animals that their owners brought them to the Veterinary Hospital of Khartoum and later on whom brought to Shambat Veterinary Clinic Fig (7), (8-I, II), (9-I, II), and (10). The owners were complaining about abnormality in the behavior of their animals, such as recumbence, anorexia, coughing, hair loss, emaciation, etc...

The period in Khartoum veterinary hospital was from 1 April 2003 until 2 June 2003. A clinical check and diagnosis were made for 136 cases (most were caprine and ovine).

The period in Shambat Veterinary clinic was from 12 August 2003 until 10 April 2004. This time a clinical check, confirmative diagnosis and treatment were done for 157 cases (they were sheep and goats and most of them suffering respiratory disturbance)

## 2.1.2 Survey Group (Group B)

This group consisted of animals for households in Omdurman and sheepmen in Azzariba market (Omdurman west), also some farms in Shambat area. The size of household and ownership ranged from 4-10 goats and 4-20 sheep. Samples of blood and faeces were collected and checked for each case and then treatment was given to each. Total number of this group was 35 sheep and goat and most of them were suffering disturbances in respiratory and digestive systems.



Fig 7: Clinical, goat suffering from Pneumonia. Shambat Vet Clinic – U of K (Caprine) Fig (8-I): Clinical, Skin abscesses (Ovine) – Veterinary Clinic (Shambat)- University of Khartoum



Fig (8-II): Clinical group, Skin abscesses (Ovine), closer vision



Fig (9-I)



Fig (9-I), (9-II): Clinical group – Wounded udder (Caprine) – Shambat clinic University of Khartoum



Fig (10): Clinical group – (Abu regeba) symptoms of minerals deficiency. (Ovine)- Shambat Vet Clinic – University of Khartoum

### **2.1.3 Experimental animals (Group C)**

These were the animals of group (C-I), experimental animals for pneumonia, and the animals of group (C -II), experimental animals for mastitis.

### 2.1.3.1 Experimental Animals for Pneumonia, group (C – I)

A total of 12 goats' males and females (About 3-6 months in age) were utilized as experimental and control animals Fig (11). They were divided into 3 groups each group was housed in a separate stable from the other groups Fig (13), (14). 2 groups were composed of 5 animals (Groups 1, 2) and the control group composed of 2 animals as the following:

Group 1	2 females; 440 & 429. 3 males 428, 156 & 158
Group 2	3 females; 437, 157 & 129. 2 males 439 & 160
Group 3	1 female; 438. 1 male; 430

They all were housed in stables at the department of veterinary medicine, pharmacology and toxicology, faculty of veterinary science, University of Khartoum. They were fed on barseem, Abu eshreen and seeds.

### 2.1.3.2 Experimental Animals for Mastitis. Group (C –II):

A total of 3 unsuspected of mastitis and healthy looking milking nannies were utilized as experimental animals Fig (12). They were housed as one group in one big stable at the department of veterinary internal medicine, pharmacology and toxicology. College of veterinary, U of K), they were fed on barseem, abueshreen and seeds.



Fig (11): Experimental group, Sub-group C-I (Pneumonia), control group



Fig (12): Experimental group – Sub-group C-II (Mastitis)



Fig 13: Experimental group – Stable (housing) – Sub group C-I (Pneumonia) (Control group)



Fig (14): Experimental group – Stable (housing), Sub group C-I (Pneumonia)

# 2.2 Clinical examination

A general clinical examination was carried for each case in both group A and group B, a detailed examination for different systems was carried when needed. The general clinical examination and inspection involves detailed consideration of the following:

Distinguishing marks Physical condition General appearance and demeanor Posture Gait Behavior Body temperature Pulse (per minute) Heart rate (per minute)

Respiration

If needed a regional or systemic clinical examination involves the application of the various clinical methodologies (sensory and physical) must be done to the various regions or system of the body. These will be dolt with in this order:

Coat and skin Head and neck Thorax Abdomen Urinary system Reproductive system Blood and blood forming organs

For these cases a case record was used. Fig (46)

For group C (Experimental animals) for both sub groups C-I and C-II a daily clinical check and general observation were done for each animal (Temperature, Pulse, Respiratory, and heart rate were taken every day). For sub group C-I (Pneumonia experiment) it was organized in a table. Table (1)

Blood sample was collected from each animal every 4 days; animals in sub group C-I were divided into 4 groups Table (2) & (3). for this purpose ; so the blood samples for each animal were collected every 4 days. That was a random division, just to organize the animals into a system for blood samples taking.

### 2.2.1 Detailed Clinical Examination for the Chest Region

#### 2.2.1.1 Inspection

#### 2.2.1.1.1 Respiration

For that animals suspected of pneumonia, the inspection carried out in the preliminary general check required to be supplemented by detailed clinical check for the chest region. So, the rate the rhythm and the character of the respiratory movement were carefully observed.

The rate was be observed by noting the excursion of the ribs and abdominal wall or by listening to the passage of air in and out of the nostrils.

Because pronounced variations in the rhythm appear in animals that are excited or subjected to any unusual or frightening influence. It was therefore important to study the respiratory rhythm before disturbing the animal or handling it in any way when it was of an excitable nature.

A careful observation for the combined movement of the thoracic and and abdominal wall was done as to decide the type of respiration

Attention was paid to all that abnormal sounds arising in the respiratory system and discernible by simply listening to the animal breathing these sounds were **Snuffling noises** which is an indicator to the presence of a big mass of nasal discharge in the nasal passages, or edema of the nasal mucous membrane or deformity of the nasal passages caused by chronic inflammatory changes or tumor growth, accompanied by exudation, snuffling is usually present during both inspiration and expiration.

**Snoring or stertorous** breathing which indicates an alteration in the contours of the respiratory passages so that obstruction is presented to the free passage of air ; then may be present in the nasal cavities, pharynx, larynx, or trachea. If only one side of nasal passage is involved the sound will be stopped by occluding the nostrils on that side. Ulceration of the respiratory mucosa, esp. if in the larynx causes a whistling or roaring sound that may be heard either on inspiration or expiration.

**Spasm of the diaphragm** (hiccup) causes an abrupt sound, varying in volume according to the size of the animal, and also a peculiar jerky movement of the animal body.

#### 2.2.1.1.2 Nasal discharge and cough

Attention should be paid to the presence of nasal discharge and cough, it was noticed whether the nasal discharge was watery, mucoid, or purulent, and if it had an offensive smell. For cough it was obviously necessary to ascertain the source of the cough. And whether it consists of a simple act or if paroxysms of coughing occur that also should be known whether the cough was chronic or not, also if the cough was dry, harsh and painful or it was moist and soft and whether it was deep and powerful in character.

Sometimes coughing is just a result of chocking, and sometimes cardiac diseases over- driving of fat sheep, may also result in acute congestion of the lungs, which in addition to marked dyspnoea causes much coughing.

### 2.2.1.1.3 Lymphatic glands:

Examination of the respiratory system was always including inspection and palpation of the superficial lymphatic glands of the head and neck. The glands most commonly involved were those of the pharyngeal region. The sub maxillary glands were frequently involved.

## 2.2.1.2 Physical examination of the chest

It consists of palpation, percussion and auscultation.

#### 2.2.1.2.1 Palpation

Provides relatively little direct information regarding the state of the thoracic organs, e.g. it reveals abnormal sensitivity of the thoracic wall and the intercostals spaces is most pronounced in inflammatory conditions affecting the pleura. Oedema of the chest wall is found by palpation

#### 2.2.1.2.2 Percussion

It is done by using the plexor and the plexometer, but can be done also using the finger in one hand as a plexor and the other one as a plexometer. it is done in the triangular area of percussion ; the superior limit is marked by the anterior portion of a line joining the posterior angle of the scapula to the coxal tuber of the ilium. The anterior limit is formed by a line joining the olecranon process of the ulna to the posterior angle of the scapula. The anterior limit of the area can be moved forward to a useful extent in small animals if the fore-leg is drawn foreword. The third side of the triangle is formed by a line connecting the olecranon to the superior limit at the second last intercostals space.

The texture of the chest wall has therefore a considerable influence on the success attending percussion and the information that can be obtained by it, so the force used in percussion must be varied to suit the thickness of the chest wall and the depth of the underlying tissues in the different animal species. It is necessary to compare the corresponding area on either side of the same animal.

Using the same degree of force in each side. It was found most satisfactory if in percussion the most resonant part is percussed first and percussion is continued from the resonant to the less resonant area.

In all animals the state of the coat has an effect on percussion.

# 2.2.1.2.3 Auscultation:

It was done by using the stethoscope, almost the same area of percussion, the chest piece of the binaural stethoscope was sufficiently firmly applied to the chest, all consideration for the coat or hair was done. The chest was methodically explored, moving the stethoscope from place to place, and retained at least until one complete respiratory movement was performed by the patient at any particular point. Different parts of the same lung may require being compared one with another; also corresponding portions of the lung on both sides was compared one with the other. Attention was paid to the respiratory sounds, the vesicular and the bronchial sounds, the normal and the alteration from the normal; abnormal vesicular sounds as exaggeration and harsh in character (in fever, pain), (cog weel) Jerky interrupted as a result of fear. Interrupted vesicular sounds localized to particular areas which indicate fibrosis of a part of the lung.

Barely audible or inaudible vesicular sound, in an animal showing symptoms of respiratory disease involving the lung, are indicative of defective expansion of the lung. Extension of the bronchial sound into those parts of the lung where normally only the vesicular sound should be heard indicates that the lung contains less air than normal.

Attention was paid also to Adventitious sounds (abnormal sounds) arising from disease of the bronchi, lungs or pleura. These adventitious sounds fall into four main categories – Dry Sounds, Moist Sounds, Crepitations and Friction Sounds.

Dry Sounds are indicative for a partial obstruction of the bronchial tubes.

**Moist sounds** may be produced in the bronchi, bronchioles or alveoli. Indicate the presence of secretion or exudates, and of a bubbling character. The sound was heard prior the coughing.

**Crepitations** are very fine sharp sounds only during inspiration and usually towards the end of it. Crepitations is done by the sudden separation of the walls of alveoli that have become adherent on account of the presence of exudates.

**Friction sounds** are of a creaking or rubbing character and indicative for the presence of inflamed roughened visceral and parietal layers of the pleura rubbing together when there is no sufficient exudates to separate the two layers.

Analysis of the Symptoms and Clinical signs of the chief Respiratory Diseases was used as assistant to clinical check of the respiratory system. Table (6).

## **2.3 Collection of Samples**

## **2.3.1 Blood Sampling**

Small quantities of blood were required for blood smears, cell counts and hemoglobin determination. This was obtained by shaving an area on the outer surface of the pinna of the ear near the margin and wiping the exposed skin with ether or alcohol and allowing it to dry, and then puncture the marginal ear vein with a sharp- pointed surgical blade or needle.

But a little larger quantities were collected for the same purpose, so venepuncture was performed in the jugular vein with a hypodermic needle 1.5 inches long and diameter size 20 in standard wire gauge, also after removing the hair over it. 1 ml blood was collected from each animal and transferred to small bottle glasses containing Edeta.

### 2.3.2 Clinical hematology

A routine blood examination usually consists of two inter-related parts; the red cell picture which comprises hemoglobin estimation, packed cell volume and total red cell count, and the white cell picture which comprises total and differential white cell counts. Later on a need for further examinations will be determined either by clinical features of the case or by the findings of the routine blood examination.

In this study only hemoglobin estimation, total red cell count and total and differential white cell count were of interest.

#### 2.3.3 Red blood cell picture

## 2.3.3.1 Hemoglobin Estimation

**Sahli method**: This method depends on the formation of acid haematin. For that 20 c.mm of blood using a special hemoglobin pipette to was added to N/10 hydrochloric acid filled to the 20 mark on the graduated Sahli tube, a standard time of five to ten minutes followed that. and the amount of acid haematin formed was estimated by comparing with known standards, using some form of colorimeter or comparator. This method was simple but not possessed of a high degree of accuracy. It was adequate for clinical purposes.

#### 2.3.3.2 Red cell count

The red cell count was carried out on blood diluted to such an extent that the number of cells was conveniently counted under the microscope. The blood was diluted by means of micro pipette dilution method. As a general rule a dilution of 1 in 200 was used.

**Dacies'fluid** had been found satisfactory for diluting blood for erythrocyte counts as it keeps well and preserves the shape of the red cells. The fluid was prepared by adding 1ml. of formalin (40%. Formaldehyde) to 99ml. of a 3%. Aqueous solution of sodium citrate.

**Strong's fluid** contains 1g. Sodium citrate, 0.6g. Sodium chloride, 1.0ml. Formalin and 98ml. of distilled water.

### 2.3.3.3 Micro-pipette Method of Dilution:

Using the thoma red count pipette, well mixed samples of blood containing anti-coagulant were ready to be checked. The pipette was filled by means of a rubber suction tube. Blood was drawn up until it reached the 05 (0.5) on the stem of the pipette (if the patient was obviously very anemic blood would be drawn up to the 1 mark). The blood was not allowed to pass the mark and no air bubbles were allowed to enter the pipette. Only the tip of the pipette was held in the blood sample; any blood on the outside of the pipette was carefully removed by wiping with a clean piece of absorbent cloth. The pipette was then filled with the diluting fluid till it reached the 101 mark above the bulb of pipette. The contents of the pipette were thoroughly mixed with a twisting or rotating motion for at least three minutes. Mixing was assisted by the bead in the bulb of the pipette which was colored red. After that diluting fluid was seen in the stem of the pipette which was discarded before starting to fill the counting chamber. that was accomplished because the first four or five drops of the diluted blood were blown out of the pipette.

#### **2.3.3.4** Preparation and Filling of the Counting Chamber

The counting chamber and the cover-slip were thoroughly cleaned and freed from grease. The raised bars on either side of the ruled counting areas were smeared with a trace of saliva and the cover-slip quickly placed on top so that it covered the counting areas. Light pressure with the thumbs was sufficient to fix the cover-slip in position. It was properly secured so it remained in position when the counting chamber was inverted. The red cell pipette with freshly mixed blood was placed with the free end at an angle of approximately 45° to the chamber so that the fluid flowed under the cover-slip by capillary

attraction. The ruled area of the chamber was completely filled; care was taken that excess does not run into the throughs and that no air bubbles appeared under the cover-slip. If any of these faults occured the counting chamber was emptied, and the cover-glass cleaned and dried and the procedure of filling was repeated. The counting chamber when satisfactory filled was allowed to stand for three minutes to allow the erythrocyte to settle. The chamber was placed under the microscope and examined with a 2/3- in (10). Objective to ensure that there was an even distribution of the cells.

#### **2.3.3.5** Counting Procedure

The cells were counted with the 1/6-in. objective. The central area of the counting chamber marked R at its four corners and in the centre was 1 sq.mm.and was divided into 25 squares which in turn were divided into 16 smaller squares. The area of each of the small squares was 1/400 of a sq.mm. The depth of the counting chamber was 1/10 mm. The squares in the central area of the counting chamber were used for the erythrocyte count and the red cells were counted in 5 sets of small squares each counting 16 smaller squares making a total of 80 smaller squares in which the red cells were counted. If a start was made with the top left hand square, those cells which lie wizen the left hand and upper lines were counted . Cells touching the right hand or bottom lines were excluded from the count in that square. This process was repeated until the required number of squares have been counted. In order to attain a reasonable degree of accuracy at least 500 cells were counted. In cases of anemia to attain this minimum it was necessary to count more squares, when it is probably best to count double the number of squares.

Alternatively the blood dilution may be limited to 1 in 100.

#### 2.3.3.6 Calculations

In the improved Neubauer counting chamber each small square =

1/400 sq. mm. The depth of the counting chamber = 1/10 mm. The volume represented by one small square = 1/4000 c.mm. The dilution of the blood is 1 in 200 (or 1 in 100).

Let *N* represent the number of erythrocytes counted in *R* small squares:

Then R/4000 c.mm of diluted blood (1 in 200) contains N erythrocytes.

1 c.mm of undiluted blood contains N  $\times$  4000/R  $\times$  200 erythrocytes.

If the number of small squares counted is 80 as suggested and total of at least 500 cells were counted in them the formula can be simplified:

 $N \times 4000/80 \times 200 = N \times 10,000$  per c.mm

The human error inherent in counting blood cells can only be reduced to a minimum by meticulous attention to technique. It must be realized that the error increases with fatigue if many counts have to be made in one working period. Electronic blood cell counters replaces the laboratories task of counting cells. Though costly the apparatus has the merit of being time saving.

### 2.3.4 White Blood Cell Picture

#### 2.3.4.1 Total White Cell Count

The principles involved in producing a total white cell count were the same as those used in making a red cell count. An appropriate dilution of the blood was made and in a special counting chamber the number of white blood cells were counted in a pre-determined volume of diluted blood. The diluting fluid recommended for this purpose is either made by adding 1 ml. of a 1% of gentian violet to 100 ml. of 2% acetic acid, or 1% hydrochloric acid can be used instead of acetic acid.

### 2.3.4.2 Micro-pipette Method

Using the white pipette, blood from a well mixed sample containing anticoagulant was drawn up to the 05 (0.5) mark. (If leucopenia was suspected the blood would be drawn up to the mark 1). Precautions in regard to air bubbles and subsequent cleaning of the stem of the pipette were the same as when making a red cell count. The pipette was then filled with the leucocyte diluting fluid to the 11 mark. The dilution attained was 1 in 20 when 05 mark was used and 1 in 10 when 1 mark is used to measure the amount of blood. The contents of the pipette were thoroughly mixed by a twisting or rotating motion and in the process of mixing the erythrocytes would become haemolysed. The contents of the stem of the pipette were discarded and the counting chamber was filled in the same way as described in doing a red cell count.

### **2.3.4.3** Counting Procedure

When the chamber was filled, it was examined under the 2/3-in. objective of the microscope to make sure that the cells were evenly distributed. The chamber was then allowed to stand on the microscope for at least three minutes to allow the cells to settle. The squares in the four areas marked W on the diagram of the ruled area, are each 1 sq. mm. in area. All the cells in the 1 sq. mm. areas were counted and at least four squares were counted, but if less than 120 cells had been recorded more areas should be counted.

#### **2.3.4.4 Calculations**

Each square has an area of 1 sq.mm. and a depth of 0.1mm. giving a volume of 0.1 c.mm.

Let W= the total number of cells counted in 4 squares 4 squares= 0.4 c.mm. o.4 c.mm. contained W cells 1 c.mm. of diluted blood would contain W/0.4But blood was diluted in 20 **So 1** c.mm. of blood contained ( $W \times 20$ ) / 0.4 =  $W \times 50$ 

### 2.3.4.5 Differential White Cell Count

In order to obtain a differential white cell count a blood smear was required. For this purpose a thoroughly clean dry slide was essential. A drop of recently collected blood containing anti-coagulant was placed towards the end of the slide. A "spreader slide" was made by cutting off the corners of one end. The narrower or tapered end of the spreader slide was placed against the surface of the first slide at an angle of 40 and in front of the drop of blood. The spreader slide was then drawn towards the top of blood; when it touched it, the blood flowed along the tapered edge. The spreader was pushed gently and steadily forwards drawing the blood along and spreading it out into a thin film. The smear thus formed was at once waved in the air to dry rapidly and prevented distortion of the cells. Smears made by this method were thin and at least 2mm. it was left between the edges of the film and the slide. The blood film was never allowed to spill over the edge of the slide.

#### **2.3.4.6 Staining Of Blood Film**

Leishman's was probably the most useful and convenient for routine staining of blood film. The stain was obtained ready for use. With a teated pipette the prepared slide was covered with undiluted stain. The preparation was left for ½ to 1 minute so that the alcohol in the stain fixed the blood film. Buffered distilled water (pH 6.8) was then added to the stain on the slide; ideally the volume of distilled water was twice the volume of stain used. The diluted stain was left on the slide for 5, 10 or 15 minutes; the time was largely governed by the known activity of the stain. The stain was then washed off with distilled water and the surface flooded with distilled water for a few seconds until the smear appeared to have a rose- pink color. The water was decanted, excess moisture removed with blotting paper and the smear dried in air.

### 2.3.4.7 Counting Technique:

The slide was scanned with the 2/3-in. objective and an area selected where the smear was of uniform thickness and evenly stained. As a general rule

identification and counting was carried out with 1/12-in. oil immersion objective.

There were two main methods of examining the slide; the parallel strip method and the battlement or four field meander method. The first was the one used in this study. The slide was moved in one direction with the mechanical stage until the strip of the selected area was traversed. The slide was then moved to the side until the next contiguous field was in view. The slide was then moved in the opposite direction to that for examining the first strip. In this way a series of parallel and contiguous strips were examined, the cells identified and counted. At least 200 cells in all were counted. The need for counting of a larger number may be indicated by the clinical findings of the case or from evidence acquired while counting 200 cells, such as noticeably uneven distribution, an unusual preponderance of one type of cell or the presence of unusual types of cell.

The cells was classified in simple classifications. The following classification has been found convenient and effective.

Neutrophils Non-lobulated

Lobulated

Eosinophils Basophils Lymphocytes Monocytes

If a large number of blood samples were being examined a digital laboratory counter could be used to record the various types of cells. A counter was not available; the cells were recorded on a sheet of paper divided into six columns, one for each type of cell.

The classification of neutrophils into non-lobulated and lobulated was necessarily somewhat arbitary but was based on the extent to which the nucleus had undergone change and was separating into lobules. All neutrophils in which the nucleus was divided into segments although the segments are joined by threads or bands were classified as lobulated. The purpose of the classification was that the non-lobulated neutrophils were young or immature cells, while the lobulated neutrophils were older or mature cells.

Table (7) was used as standard in this stud

## **2.4 Experiments**

## 2.4.1 Animals Group C

This group was composed of 2 subgruops; the first was subgroup C-I, animals used for pneumonia experiment. The second was sub group C-II, animals used to study mastitis in goats.

## 2.4.2 Sub group C-I

A clinical check and general observation were done as daily routine for each animal. Blood samples were collected from each group every 4 days.Tables (2), (3)

# 2.4.2.1 Expermental infection of goats with pneumonic agents

## Attempt 1

A sick animal with unknown pneumonic agent was housed with the animals in the stable. Observation for the groups was done for one week after, and treatment was given later on to the animals with the ear tags 440, 429, 428, 156, 158, and 437 only.

### Attempt 2

The animals with the ear tags: 439 & 429 were infected with Staph . aurious

The animals with the ear tag 428, 440, and 160 were infected with Streptococcus spp.

The animals with the ear tags 158, 156, 159, 437 and 157 were infected with Pseudomonas spp.

#### 2.4.2.2 Source of bacteria:

The experimental animals it selves; after being experimentally infected using a kid suffering from pneumonia, swabs of nasal discharge and lacrimation were taken randomly from 3 of them. These three animals were with the numbers 428, 437, and 349. The daily clinical check showed their parameters at that day in Table No (4), result for the blood check in Table No (5). High concentrated solutions were created in sterile media by adding bacteria from its gross to a sterile normal saline (each bacterium was added to a separate sterile normal saline bottle).

#### 2.4.2.3 Way of administration:

**Nose drops**: From the gross of Staph. aurieus, Streptococci spp, and Pseudomonas spp. A sterile syringe was used as a dropper for each bacteria. 2 mm were given to each animal in its nostrils.

#### 2.4.3 Sub group (C – II):

Milking goats  $1 \frac{1}{2} - 3 \frac{1}{2}$  years old were used Fig (19). They were brought from the local market (suq Shaabi, Omdurman) They were observed for a period of 2 months, a bacteriological check was done to its milk, treatment was given to it, and a daily check for them all was done temperature, udder size and milk quantity.

## 2.4.3.1 Collection of samples:

The milk samples examined in this experiment were collected aseptically. The udder was washed with soap and water and dried with clean cloth. The teats and teat orifices were swabbed with 70% alcohol and dried. Milkers hands were swabbed with 70% alcohol and dried between milkings .

Clinically affected halves were milked last. The first few streams of milk were discarded and 5 - 10 ml was collected in sterile McCartney bottles. From this experimental animals milk was collected from both halves together in one bottle. Each sample was labeled appropriately for identification. Clinical examination was done after collection of milk samples.



Fig (15): Experimental group – Sub group -II (Mastitis), normal milk from a Animals A & B



Fig (16): Experimental group – Sub group-II (Mastitis), apparently healthy udder.


Fig (17): Experimental group – sub group-II (Mastitis), apparently Healthy animal

#### 2.4.3.2 Clinical examination

As a general clinical examination was carried out for each animal, a detailed udder examination was taken for these animals. The udder was first inspected and observations made on the size; size was checked before and after milking Fig (18), the quantity of milk yeald was checked daily Fig (17), presence of scars, abscesses, lacerations and any other abnormalities in addition to temperature and blood haemogram as shown in Tables (9) & (10).

The udder tissue was then palpated with fingers to note indurations, fibrosis, heat, pain and other abnormal changes, both supramammary lymph nodes were palpated.

According to Pyöräla and Syvajarvi (1987), systemic signs, local signs on the udder and milk appearance were divided into 3 classes that ranged from 1(normal) to 3(severe) clinical signs were recorded.

#### 2.4.3.3 Bacteriology

Milk samples were collected from all 3 nannies. From each, a milk sample was taken from both halves, using sterile screw cap bottles. The examination of milk samples for isolation and identification of bacterial causative agents and for testing the sensitivity of the isolated bacteria to specific antibiotics, were done by laboratory of bacteriology at the department of microbiology, faculty of veterinary medicine, U of K. (It was done on commercial basis).

#### 2.4.4 Treatment

#### 2.4.4.1 Drugs selected and used for both sub-groups C-I and C-II

#### **Parental treatment :**

Ox tetracycline 5% (oxtra Italy) each 100 ml contains :

Oxytetracycline Hydrochloride

Lindocaine Hydrochloride	2 gm
Propylene Glycol q.s	100 m

Doses: 5 cc for 5 days, the second dose was given after 12 hours from the first one administration. The rest of doses was given once daily.

Adminstration route: Through intramuscular route. (It is a product of)

#### 2.4.4.2 Drugs selected for treatment of mastitis

#### 2.4.4.2.1 Local treatment

Neomastipra – J5, manufactured by laboratories Hipra, S. A, Spain. Composition per syringe of this drug:

Benzyl Penicillin procaine	100.000 I.U
Dihydro streptomycin (Sulphate)	62.4 mg
Neomycin (Sulphate)	36 mg
Polymyxin B ( sulphate )	50.000 I.U
Sulpha dimidine (Sodium)	250 mg
Sulphathiazole	250 mg
Hydrocortison	20 mg

**Dosages**: (Note: Although in – vitro sensitivity test results were taken into account in treatment trials, the latter were initiated before results were available to save the life of the animal particularly in acute cases and to reduce tissue damage.) so it was used: Half syringe for each half every 12 hours ; one syringe per each mastitis suspected goat ; the doses were twice daily for 5 days .

Withdrawal period:

Milk: 3 days (6 milking)

#### **Special precautions:**

The affected and suspected halves were treated after a careful milking, cleaning and disinfection of the teat, the equipments and milk man's hands.

Emptying the mammary gland was avoided after infusion, in order not to loose the infused drug, so the goat under treatment was not milked within 6 hours after infusion.

#### 2.4.4.2.2 Parental treatment:

Penivet forte Injection. (Note: Was not used for all animals.) Composition: Each 50 ml vial contains:

Penicillin G Procaine	3,000,000 I.U
Penicillin G Sodium	1,000,000 I.U
Dihydro Streptomycin Sulphate	5gm
Sterile diluent ( in separate 50 ml vial )	30 ml

**Dosages and Administrations:** This drug was given through intramuscular route; the dose rate was 7.5 ml per 100 kg body weight, daily for 5 days (4 ml for 5 successive days).

#### 2.4.4.3 Observation:

After treatment for both subgroups C-A and C-B an observation for a period of a month was done for them both and notes were recorded.

## **CHAPTER THREE**

#### RESULTS

#### 3.1 Results of the first stage of the study

3.1.1 Information Collected from Khartoum Veterinary Hospital

# **3.1.1.1** Diagnostic methods, other diagnostic procedures, laboratory aids to diagnosis:

Diagnostic methods were Inspection, palpation, percussion, Auscultation, and the use of sense of smell. Other diagnostic procedures were passage of sounds and catheters, exploratory puncture, and metal examination .Laboratory aids to diagnosis was Parasitological examination.

#### 3.1.1.2 Animals

All 136. Fig (25)		
Caprine	71 cases	53%
Dog	37	28%
Ovine	20	15%
Feline	6	4%

#### 3.1.1.3 Diseases

Goats. Fig (26)		
Gynecological problems	15 cases	20 %
Pneumonia	19	26%
Digestive system problem	21	28%
Mastitis	9	12%
others	10	14%

The rest were 2 cases diagnosed as weakness, 3 cases tumor, 3 eye infection, one case arthritis, abscess, lameness, horn fracture, titanus, and 2 cases suspected rabies.

#### **Dogs**. Fig (27)

Tic infestation	35.13%
Digestive system problem	21.62%
Pneumonia	18.9%

The rest of the cases were tumors, otitis, and paralysis in hind legs.

# Sheep. Fig (28)Pneumonia30%Digestive system problems30%External parasite15%Heart water10%Sheep pox10%

The rest were mastitis, lameness and congenital defects and a vaginal prolapse case.

#### Cats

3 cases were Pneumonia, one case dystocia, muscle weakness, Tumor in hind quarter.

#### **3.1.2 Information collected from Shambat Veterinary clinic:**

**3.1.2.1** Diagnostic methods, other diagnostic procedures, and laboratory aids to diagnosis methods

Diagnostic methods were the same of that used in Khartoum Veterinary hospital and the other diagnostic procedures also the same laboratory aids to diagnosis were:

Parasitological examination. Hematological examination. Bacteriological examination.

#### 3.1.2.2 Animals

All animals (Sheep and Goat) were 157, all goats were 128, 108 of it were females only 20 were males, the sheep were the rest.

#### 3.1.2.3 Disease:

#### Goats. Fig (29)

Pneumonia	41 cases	31 %
Digestive system problems	32	25%
Gynaecological problems	23	18 %
Mastitis	20	16 %
Artheritis	9	7%
Urinary tract problems	4	3%

#### 3.2 Results from the second stage of the study (Survey)

They were: 1 dog. Fig (6-I, II, III, IV) 16 0vine.Fig (1) 18 caprine. Fig (2), (3), (4), and (5-I, II, III) Hemoglobin estimate was done to 31 of them Red blood cell count was done to 21 of them White blood cell count was done to 23 of them More details will be found in Table (11)



Fig (1): Survey – Exhausted ram, suffering from mineral deficiency. (ovine) Sug Libia Fig (2): Survey – Goat suffering from internal parasite. (Caprine) Aborof area - Omdurman





Fig (3): Survey – Goat with internal parasite. (Caprine) Addibagha area- Omdurman



Fig( 4): Survey – Goat with jaw abscess. ( Caprine )- Shambat area



Fig (5-I): Survey – Suspected heart water goat. ( Caprine ) – Shambat area





Fig (5-III): Survey - Enlarged gall bladder- suspected heart water. (Caprine)

Fig (5-II): Survey – oedemated heart- suspected Heart water. (Caprine)

Fig (6-I): Survey, Allergic dog ( Itching in ears)





Fig (6-II): Survey, Allergic dog (Skin allergy in all the body)

Fig (6-III): Survey – Allergic dog (general vision) before treatment





Fig (6-IV): Survey – Allergic dog after treatment

# **3.3** Result from the third stage of the study

3.3.1 Pneumonia experiment

#### 3.3.1.1 Result from attempt 1

All experimental animals showed symptoms of respiratory system defect; coughing, nasal discharge, and rise in temperature, but no anorexia.

Results of Bacteriological examination for random samples took from the group (and were used for the second experimental infection) will be found in Table (12).

Analysis for routine daily clinical check (parameters) for animals sub- group C-I will be found in charts; Figures (32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, and 43).

Treatment was given to the animals with the ear tags numbers: 429, 156, 15, 158, and 159 only.

All animals witch were treated recovered completely within one week, animals witch were not treated also recovered semoltanasly after one week. Blood haemogram for all animals before and after treatment will be found in Table (13)

#### 3.3.1.2 Results from attempt 2

All animals that were experimentally infected with pneumonic agents showed mild symptoms of respiratory problem such as coughing, nasal discharge, mild rise in temperature and then disappeared semoltanasly and no treatment was given to it.

#### 3.3.2 Results from mastitis experiment

**3.3.2.1 Infected cases** 

3.3.2.1.1 Case 1 (Animal A) 3.2.1.1.1 History: Local breed goat (Nubian goat), about 3 years in age, more than 4 calving, fair milk production, according to the owners (sellers), no previous disease and no previous treatment .

#### **3.2.1.1.2** Clinical findings:

No any Clinical sing for disease. Normal parameters, normal milk, good appetite.

#### 3.2.1.1.3 Bacteriology:

culturing of milk sample took from both halves revealed *Staphylococcus aurious*.

#### **3.2.1.1.4** Treatment

Treatment was done by Neomastipra which applied locally as mentioned before and the response was as the following:

#### **3.2.1.1.5** Response to treatment

#### 48 hours after treatment:

temperature 39.0 C° heart rate 92/min, pulse rate 84/min, respiratory rate 44/min

#### 72 hours after treatment:

Temperature 39.2 C°, heart rate 72/min, pulse rate 64/min, respiratory rate 30/min.

#### **10 day post treatmeant:**

Temperature 38.6C° heart rate 48/min, pulse rate 44/min, respiratory rate 32/min milk quantity was normal again 136 cc.Complete recovery both clinically and bacteriologically was achieved.

#### **3.2.1.2 Case 2. (Animal B)**

#### 3.2.1.2.1 History:

local breed goat (Seams like a mixture between Nubian and the mountain tagaz goat)

1<sup>1</sup>/<sub>2</sub> years in age, only one calving, bought from Suq Shaabi Omdurman North. According to the owner (sellers) no previous disease so no previous treatment.

#### **3.2.1.2.2** Clinical finding:

looks healthy, no sign for any suspected disease, normal parameters, normal milking and good appetite.

#### 3.2.1.2.3 Bacterilogy

Bacteriological culturing of milk samples from both halves revealed B. aurious.

#### 3.2.1.2.4 Treatment

Neomastipra was used for treating this case, it was applied locally as mentioned before and the response was as follows:

#### **3.2.1.2.5 Response to treatment**

#### 48 hours after treatment:

Temperature 39.6 C° heart rate 104/min, pulse rate 100/min, respiratory rate 80/min, appetite was normal.

#### 72 hours after treatment:

Temperature 39.4 C°, heart rate 92/min, pulse rate 96/min, respiratory rate 76/min.

#### **10 days post treatment:**

Temperature 38.9 C° heart rate 60/min, pulse rate 64/min, respiratory rate 36/min and the milk quantity was 65 cc, so complete recovery both clinically and bacteriologically was achieved.

#### **3.2.1.3 Case 3 (Animal C)**

#### 3.2.1.3.1 History

Local breed goat (it seams like a mixture between Nubian and desert goat),  $2\frac{1}{2}$  years in age, more than 3 calvings, bought from Suq Shaabi Omdurman North. According to the owner (seller), no previous disease so no previous treatment. The udder teats were tied firmly both separately to prevent its kid from milking it.

#### **3.2.1.3.2**Clinical finding:

when it was bought: looked healthy, good milk production, good appetite.

After 3 days :Pneumonia signs ; coughing, nasal discharge, temperature was 40.7 C°. And then appeared continouns drop in milk production, growing of hard small masses in the udder and enlargement of udder lymph nodes.

#### 3.2.1.3.3 Bacterology:

Bacteriological culturing of milk samples from both halves revealed *Staphylococcus spp* 

#### **3.2.1.3.4 Treatment:**

Treatment was done by Neomastipra which applied locally as mentioned before and the response was as fallows:

#### **3.2.1.3.5** Response to treatment

#### 48 hours after treatment:

Temperature 39.3 C° heart rate 120/min, pulse rate 68/min, respiratory rate 112/min, appetite was normal.

#### 72 hours after treatment :

Temperature 39.0 C°, heart rate 120/min, pulse rate 84/min, respiratory rate 24/min.

#### **10 days post treatment:**

Temperature 39.6 C° heart rate 64/min, pulse rate 64/min, respiratory rate 24/min, produced milk quantity was 76 cc. The animal generally clinically recovered but the udder did not recover not clinically still there were hard masses in it) bacteriologically that bacteriological culturing revealed growth of *Staphlylococcus spp*.

#### **3.2.2 Observation:**

After one month Observation animal C was severely attacked with mastitis again; the attack was a develop from mild to per acute mastitis in a very severe way that treatment could help the animal to survive the systematic signs only Fig (21), (22), and (24) but the udder was completely destroyed Fig (22) and (26), parameters after second time treatment for animal C will be found in Table (14).

The animal A and B were completely recovered clinically and bacteriologically Table (8) and Fig (44) & (45).



Fig (20): Experimental group – Sub group 2 – Mastitis Animal C ( Before attacked by acute mastitis)

# Fig (21): Experimental group - Sub group II (Mastitis) Animal C, chest Oedema





Fig (22): Experimental animal – Subgroup II (Mastitis) Animal C, asymmetric in udder



Fig (23): Experimental group, Subgroup-II (Mastitis) Animal C – Normal milk



Fig (24): Experimental group – Sub group-II (Mastitis), animal C

Left : Normal milk from not affected right half udder Right: Blood tinged milk from affected left half udder

Fig (25): Experimental group – Sub group-II (Mastitis), animal C (Apparently healthy udder)





Fig (26): Experimental group, Sub group-II (Mastitis), animal C (Mastitic udder)

# CHAPTER FOUR DISCUSSION

The present study was performed to study the clinical diagnosis in Khartoum veterinary hospitals and clinics, emphasis diagnostic tools and laboratory aids to diagnosis methods and tools with special consideration for goats. Diagnosis

continues to be the main problem for every practitioner and animal owner in these contries which are not well developed in its diagnostic mechanical techniques. This study performed in 3

stages, The first stage was in Khartoum Veterinary Hospital and Shambat Vet Clinic, Collage of Veterinary medicine U of K. The second stage was survey in some areas around Omdurman, and the 1st stage was 2 experiments; one for pneumonia and the other for mastitis . The first stage was for training and collecting information about diagnostic methods and laboratory aids to diagnosis methods, animals, and the most common diseases. For this, it is found that in Khartoum vet hospital beside the stethoscope and thermometer, the laboratory aids to diagnosis was parasitological diagnosis and a bacteriological diagnosis which later on was established. It was the same in Shambat Vet Clinic but the aids to diagnosis were parasitological, bacteriological and laboratory hematological diagnosis. And for disease information which were collected; 25.24% of the whole sick animals received in Khartoum Vet Hospital had digestive system problem, 25% had respiratory system problems (diagnosed as pneumonia). It seems that digestive system problems and pneumonia were the most common diseases through two months in Khartoum Vet Hospital because the rest diseases like mastitis, arthritis, external parasites etc, were of small

percentages . Through the same period in Khartoum Vet Hospital it was found that 52.21% of the cases were caprine, 27.21 % were dogs, 14.71% were ovine, and 4.14% were cats. A period of 8 months in Shambat Vet Clinic , the concentration was poured on caprine and ovine , the whole number of both species was 157 , 81.01% were caprine and 18.4% were ovine . The most common disease among goats was pneumonia 32.03%, 25% was digestive system problems , gynecological and congenital problems 17.96% , mastitis 15.62% , arthritis 7.03% and urinary system problems 3.125 .

Information was continued to be collected through the second stage of this study ; 40 cases from random areas in Omdurman were checked clinically , blood and faecal samples were collected from them , and parasitological and haematological examinations were done to it all , also bacteriological examination were done to some of it . Most of these cases suffered from anaemia, internal parasites, and pneumonia, treatment was done to most of it, one goat was died before a clear diagnosis was fixed for it, one sheep was died after treatment.

In this study a routine clinical examination ,haematological and parasitological examination were choose as laboratory aids to diagnosis methods , the treatment used was broad spectrum drugs ( umbrella treatment ).

Before we go further to the problems and difficulties in the first and second stages in this study a question must be answered or must be kept in the mind; were these results true, or it came out just according to our capacity in diagnosis?

Problems and difficulties faced the first and second stages from the study:

The case history told by animal owner can not be reliable, no disease records, no birth records, and no any reliable information about the animal.

Most cases were emergent cases, because they were brought to veterinary help whether after long neglicanse until many complications took share in the sick animal problem , or when the sickness was so severe , or the animal about to die and the owner looses hope of the recovery of the animal .``

The last stage, results gained from pneumonia experiment:

Experimental infection by using pneumonic animal: 10 animals out of 12 animals were infected, showed symptoms of pneumonia like high temperature, nasal discharge, coughing, but no anorexia. Bacteriological examination for random samples collected from them showed Streptococcus spp, corynbacteriuem spp, pseudomonas spp , and gram –ve rods .

All animals in treated group showed response to treatment in different degrees, the animals in untreated group recovered spontaneously.

Infection repeated again for all animals, this time using pathogenic agent as nose drops. Infected animals showed mild clinical findings, as a little rise in temperature and nasal discharge but it disappeared spontaneously within 2 days, treatment was not needed.

It is easy to diagnose pneumonia as respiratory system infection, but some diseases as (ccpp) Contagious Caprine Pleuropneumonia as respiratory system infection can not be diagnosed this way. From observation for the three animals in mastitis experiment, it was found that sub clinical mastitis can not be discovered by routine clinical examination and hematological examination only , many tests should be used for detection of mastitis , bacteriological culturing is very important as strong evidence of mastitis and also because it is important for mastitis treatment to know the causative agent . In goats, when fibrosis appears in udder, the recovery seems to be impossible.

Evaluation for diagnostic methods and laboratory aids to diagnosis methods:

Clinical examination methods used are:

Inspection

Palpation

Percussion

Auscultation

Use of sense of smell

Other diagnostic procedures: special diagnostic procedures include the passage of sounds and catheters, exploratory puncture, and rectal examination are used, the rest as the use of oesophagoscope, ophthalmoscope, and mechanical aids to diagnosis such as X-rays and the electrocardiography are not well known and are not used.

Laboratory aids to diagnosis employed are:

Haematological examination

Parasitological examination

Bacteriological examination

"Test therapy "can hardly be called an accurate aid to diagnosis.

The rest which are not commonly play a clear role in diagnosis in vet hospitals and clinics in Khartoum:

Allergic reactions Clinical chemistry Serological examination Analysis for poisons Soil and herbage Mechanical aids to diagnosis (Radiological examination)

In this study haematological examination "Test therapy "were used as laboratory aids to diagnosis, it is found that it can slightly help in accurate diagnosis because :

Hemoglobin examination, a simple estimation of the haemoglobin level may be sufficient to confirm a tentative diagnosis of anaemia . A more extensive haematological examination will be required to indicate the type of anaemia and its possible cause. This more extended investigation should only be sought when the clinical examination has eliminated blood loss as a cause of anaemia and the study of environment has eliminated the possibility of trace element deficiency in ruminants. Investigation of the white blood cell picture may indicate the type of response associated with the illness and may indicate a defensive reaction to infection. Information of this types neither specific in relation to the type of infection nor is it indicative of the site of infection in the body.

For haemoglobin estimation Sahli method is used, no colorimeter is used but a comparator. For red and white cell count no electronic blood cell counters are used , also for differential white cell count no digital laboratory counter is used , so it is time wasting , not accurate , as done manually which means lot's of human errors can take place also . It is very difficult to make complete haematological examination for more than two blood samples in one working period. The "Test therapy " was used in this study for that ( pneumonia ) group of 12 animals , but it could not be accepted as conclusive because untreated controls in the group showed spontaneous recovery .

Bacteriological aid to diagnosis : milk samples culturing was effective in mastitis diagnosis and treatment , but the result coming from laboratories considered to be not specific , it gives the big lines only for the causative agent for example Staph spp , Gram –ve rods . Diagnosis for some diseases depend on causative agent isolation for example CCPP, its diagnosis will be confirmed only if M.C.capripneumoniae is isolated.

Last no official information is available to give any idea about this topic, and to show the present situation for what laboratory aids to diagnosis are used, what is needed, and what is planned to be used or imported for the coming years.

# CHAPTER FIVE CONCLUSION AND RECOMENDATIONS

## **Conclusions:**

- 1- The goat is a very important animal in the Sudan; it plays a major part in the supply of fresh milk in nearly all parts of the country. The Nubian goat which is 50% of the goats in the Sudan is the only milk goat breed in Africa.
- 2-Mastitis is a dangerous problem in goats, and only preventive detection for sub clinical mastitis can help in it .
  - 3- The present used diagnostic methods and tools are not coping with the continuous development in the world; every day new diseases appear, new pathogenic agents discovered and new methods are employed.
  - 4- There is some deficiency in Diagnostic methods and the laboratory aids to diagnosis needs to be compensated to enable the veterinarian practitioner to fulfill his duty

# **Recommendations:**

- 1- More and special attention must be paid to field work and clinical diagnostic methods and tools in veterinary hospitals and clinics.
- 2- Rehabilitation and modernization of the veterinary practice is demanding.
- 3- More funding and budgeting is a necessity for improved and developed veterinary practice.
- 4- Special attention is needed for small ruminants particularly goats being wide spread and mostly raised by families.



Fig (25): clinical, Khartoum vet Hospital, distribution (by percentage) of sick animals brought to within the period from 1 April to 2 June 2003



Fig (26): clinical, distribution of diseases affecting goats brought to Khartoum Hospital (by percentage within the period from 1 April to 2 June 2003)



Fig (27): clinical, distribution of diseases affecting dogs brought to Khartoum vet Hospital (by percentage) within the period from 1 April to 2 June 2003



Fig (28): clinical, distribution of diseases affecting sheep brought to Khartoum vet Hospital (by percentage) within the period from 1 April to 2 June 2003


Fig (29: clinical, distribution of diseases (by percentage) affecting goats brought to ShambatVet Clinic within the period from 12 August 2003 to 10 April 2004







Fig (31): 27 October- 5 November 2004



Fig (32): 6-10 November

Temperature for experimental animals sub group c-I (Pneumonia) in the period from 17 October to 10 November 2004



Fig (33): 17-26 October 2004



Fig (34): 27 October - 5 November 2004



Fig (35): 6-12 November 2004

Heart rate for experimental animals sub group c-I (Pneumonia) in the period from 17 October to 12 November 2004



Fig (36): 17-26 October 2004



Fig (37): 27 October- 5 November 2004



Fig (38): 6- 12 November

Pulse rate per minute for experimental animals sub group c-I (Pneumonia) in the period from 17 October to 12 November 2004



#### Fig (39): 17-26 October 2004



Fig (40): 27 October- 5 November 2004



Respiratory rate per minute for experimental animals sub group c-I (Pneumonia) in the period from 17 October to 12 November 2004

12

438



Fig (42): Day 6 was last day before treatment Day 8 was last day after begining treatment

Fig (43): Variations in milk quantity from the 3 animals sub group C-II



Fig (44)

# Case record

Case number:				
Date:				
Locality: Khartoum Area:				
Species:	Sex:	Ад	e:	
Physical condition:				

# History

1-	Chief	complain:

# 2- Previous state of health:

Previous	disease:					
When:		Durat	tion			
Nature of it :	Seasonal (	)	or	Continuous	(	)
Previous treat	ment:					
Results:			• • • • • • •			

# Present condition:

### **General condition** :

Temp: c	Pulse :	per minute
Respiratory rate :		per minute
Heart rate :		per minute

Appetite: Good ( )	Fair ( )	Bad (	)
Desire for water: Good (	) Fair (	) Bad (	)

Diet:		
<b>Environmental condition</b> : Housing: Indoors ( ) Use of the animal:	Outdoors ( )	
Tentative		diagnosis:
Laboratory examination results:		
Blood sample: Faecal sample: Other samples:		
Confirmative		diagnosis:

### **Table (1):**

Dung chineur chech tuble for sub Broup Off (Incumoniu)									
Case	Date	Time	Temp	Pulse/min	Heart/min	Resp/min	Notes		
No									

### Daily clinical check table for sub- group C-A (Pneumonia)

### **Table (2):**

# groups for blood sample collection

Group (1)	429, 428, and 440
Group (2)	437, 439, and 160
Group (3)	158, 159, and 156
Group (4)	430, 438, and 157

### Table (3):

### Blood check table for animals of sub-group C-I (Pneumonia)

Date	Group	Case	RBC	WBC	Hb/g	Hb%	Neuts.m	Neuts.im	Lymph	Mono	Eosin	Baso
		No										

Table (4)

Data for animals 428,437,439 at the day of taking swaps for creation of highly concentrated solutions of bacteria used for experimental infection of Pneumonia

Case	Temperature	Pulse/min	Heart/min	Resp/min	Notes
No					
428	38.9	64	60	24	Lacrimation – watery
					and mucoid
437	38.9	64	72	16	Diarrhoea with
					offensive smell
439	40	52	92	20	Nasal discharge and
					lacrimation

Table (5): Blood data for animals 428, 437, and 439 at the day of taking swaps for bacterial gross and creation later on highly concentrated solutions used for experimental infection of Pneumonia

Case no	WBCs	RBCs	Hb%	Hb/g
428	8,700	18,240,000	59%	8.4
437	3,500	19,040,000	60%	8.4
439	4,350	16,940,000	46%	6.4

### Table (6)

# Analysis of the Symptoms and Clinical signs of the chief Respiratory Diseases.

	Nasal catarrh	pharyngitis	bronchitis	Lobular pneumonia	Interstitial pneumonia	Chronic alveolar emphysema	Pleurisy 1 <sup>st</sup> stage	2 <sup>nd</sup> stage
1-nasal discharge	1-serous 2-mucoid 3-purulent	May be mucoid or purulent	May be mucoid or purulent	May be mucoid or purulent				_
swalling		May be difficult	—		—	—		—
cough	absent	1-dry and harsh 2-moist and soft	1-dry and harsh 2-moist and soft May be paroxysms	Copious expectorate. Cough may be very frequent		Deep hollow cough		
respirations	normal	normal	Dyspnoea may be severe	Dyspnoea may be severe	Rapid shallow	Double expiratory effort. Dyspnoea on exertion	Mainly abdominal. Pleuretic line present	Dyspnoea if exudates voluminous
fever	Usually only slight	May be marked	May be marked	May be marked	slight	absent	present	Present
Regional(throat)l.g	May be swollen	May be swollen	—		—	—		—
Chest. Vesicular sounds			may be exaggerated	May be exaggerated	exaggerated	Increased in volume. Crackling noises		
Bronchial sounds			Sonorous and sibilant ronchi	Sonorous and sibilant ronchi	loud		_	_
Moist sounds.	_	_	present	present	—	—		—
crepitation				present	May be crackling due to alveolar emphysema	No true crepitation		
Friction sounds		_				_	Present normal	absent
Percussion.			normal	General reduction in resonance. May be dull areas due to confluence	Loss of resonance develops in upper third	May be an increase in resonance and size of area	normal	Dull area in lower part of chest

	RBC	P.C.V	Hb.g.per	WBC.per	Neuts.mature	Neuts.immature	Lymph	Mono.	Eosin	Baso
			Cu.mm	Cu.mm.	Per.cu.mm	Per.cu.mm	Per.cu.mm	cu.mm	Per.cu.mm	per.cu.mm
Sheep	8-16	24-50	8-16	4,000-	700-6,000	rare	2,000-9,000	0-750	0-1,000	0-300
	12	38	12	12,000 8,000	2,400(30%)		4,950(62%)	200(2.5%)	400(5%)	50(0.5)
goat	12-20	24-48	8-14	4,000-	1,200-7,200	rare	2,000-9,000	0-550	50-650	0-120
8	15	35	11	13,000 9,000	3,250(36%)		5,000(56%)	250(2.5%)	450(5%)	50(0.5%

# Heamatological values shouts used as standard in this study

# Table (8)Parameters took after the beginning of treatment (Mastitis experiment)

Day	Animal	Pulse/min	Heart/min	Resp/min	TemperatureC	Milk/cc	Notes
9 <sup>th</sup>	А	84	92	44	39		48 hours
	В	100	104	80	39.6		after
	С	68	120	112	39.3		beginning
							treatment
10th	А	64	72	30	39.2		72 hours
	В	96	92	76	39.4		after
	С	84	120	24	39		beginning
							treatment
	А	44	48	32	38.6	136	10 days
	В	64	60	36	38.9	65	after
	С	64	64	24	39.6	76	beginning
							treatment

#### Animal L of udder L of udder W of W of Milk Notes Day Temperature udder B udder A quantity В A R R L R L L R L 19 19 19 7 210 39.3 19 12 12 6.5 1 А 4.5 5.5 6 В 39.3 15 15 16 16 6 157 19.5 С 180 39.5 18 18 19 8 7 7.5 8 2 19 20 19 20.5 6.5 7.5 6.5 194 39.0 Udder А 7 palpation reveals presence of small hard masses 192 39.2 14 16.5 15 5.5 В 16 6 5.5 Udder 6 С 19.5 8.5 18 20.5 20 7 6.5 181 38.8 palpation 8 reveals presence of hard masses and enlarged lymph nodes 22.5 154 39.0 3 21 19.5 20.5 6.5 7 6.5 6 А 39.0 В 14.5 16 15.5 17 6 161 6 6 6 С 18 20 15 21.5 6.5 7 6 7 119 39.1 7 172 20.5 21.5 7 39.1 4 А 20.5 21 6 5 В 16 17 15.5 6.5 5.5 6 158 39.2 16 6 С 20 21 19.5 21 115 39.3 8 8 8.5 6 7 39.2 5 А 19 21 17 19 6.5 6.5 6 200 В 15 17 11 13.5 6 5 5.5 182 39.1 6 С 16 17.5 15.5 7.5 7 160 39.0 17 6 6 5.5 Last day 17 19.5 6.5 201 39.0 6 А 16 18 6.5 6 B 12 13 before 13.5 12 5.5 4.5 161 39.1 5 7 treatment С 15 16 12.5 14 7.5 7.5 201 39.1 8 5 Hard masses became bigger and the L.N became more enlarged, last day before treatment 17.5 18.5 16 18 5.5 230 39.2 First day 7 7 7 6 А

# Table (9)Daily check for the udder, the milk, and the animal temperature

	В	12	14	11	12.5	5	5.5	4	4.5	160	39.1	after
	С	13.5	15.5	16	19	6	6.5	5.5	6.5	176	39	beginning
												treatment
14	А	15.5	17	16.2	17.5	6	6	5	5.5	96.0		The 8 <sup>th</sup>
	В	14.5	17.5	12.5	15	5.5	5.5	4	5	98.0		day after
	С	16.5	16.5	15	17	6	6.5	6.5	7	76.0		beginning
												treatment

Timing	Animal	Hb%	Hb/gm	RBCs	WBCs	L%	M%	N%	<b>B%</b>	E%
Before	А	35	5	6,930,000	4200	57.073	16.585	18.048	00	8.292
treatment	В	40	5.2	13,870,000	8400	53.554	7.109	34.597	00	4.739
	С	33	4.8	10,930,000	8650	43.069	9.405	43.564	0.495	3.465
After	А	53	5	11,770,000	5450	3.214	82.857	12.857	00	1.071
treatment	В	32	4.4	21,610,000	8200	0.89	88.02	5.98	2.09	2.99
	С	33	4.8	13,190,000	7650	1.694	83.05	13.559	00	00

Table (10): Blood picture for the 3 animals' sub group C-II (Mastitis experiment) before and after treatment (Mastitis experiment)

Ca	C	Com	<b>A</b> mag	Та	TTL/	TTL	Dha	W/L	Comulain/Di	4
Ca	spec	Gen	Area	Ie	ΠD/		RDC	VV D	Complain/Di	treatment
se	ies	der		mp	gm	%0		c	agnosis	
no				C						
1	Capr	Fem	Addib		10	65	5,910,0	545	Internal	Albendazo
	ine	ale	agha			%	00	0	parasite	le
2	Capr	Fem	Addib	38.	6.0	48	4,530,0	1,88	Hair	Albendazo
	ine	ale	agha	8			00	0	loss,anaemia,i	le
									nt parasite	
3	Capr	Fem	Addib	39.	9.29	62	7,100,0	2,23	Int parasite	Albendazo
	ine	ale	agha	6			00	0 Ó	1	le
4	Capr	Fem	Addib	39	9 89	68	6 790 0	3 68	Int parasite	Albendazo
-	ine	ale	agha	8		00	00	0	int parasite	le
5	Capr	uie	Aborof	39	10.1	75	00	Ŭ		10
5	ine		1100101	5	10.1	15				
6	Copr		Aborof	20	71	51				
0	Capi		AU0101	39. 1	/.1	51				
7	Com		A 1	4	7	40				
/	Capr		Aboroi	39.	/	49				
0	ine		A.1 C	1	7.4	(7	7 100 0	0.75		
8	Capr		Aborof	38.	7.4	67	7,100,0	8,75		
	ine			8			00	0		
9	Capr		Aborof	38.	5.2	39				
	ine			7						
10	Capr		Aborof		5.8	40				
	ine									
11	Capr		Aborof		7.0	50				
	ine									
12	Capr		Aborof		7.0	49				
	ine									
13	Capr		Aborof		7.2	50.				
	ine					3				
14	Capr		Aborof		7.4	49	5.940.0	18.4		
	ine						00	00		
15	Ovin	Male	Sug		92	65	3 600 0	21.3		
10	e	white	lihia		1.2	05	00	50		
16	Ovin	Male	Sug		5	34	6 2 2 0 0	7 20		
10	Ovini e	whate	libia		5	54	0,220,0	0,20		
17	Ovin	Fom	Sug		6	12	4 5 1 0 0	5 15		
1/	0vm		Jibio		0	42	4,510,0	5,15		
10	C Orvin	Lore	filla		0	50	4.070.0	7.00	Ita hida diad a	
18	Ovin	Fem	Sug		8	39	4,970,0	7,90	Its kids died a	
	e	ale	1101a				00	0	rew days after	
									been	
1.0					6.0	4-	4.0.10.0		delivered	
19	Ovin	Male	Sug		6.3	45	4,940,0	1,55	Caziated	
	e		libia				00	0	lymph node	
									upper foreleg	
20	Ovin	Male	Sug		4.3	33	2.200.0	16.7	Nasal	

Table (11): Survey cases, species ,gender, temperature, Hb gm, Hb%, Rbc, Wbc, Complain and treatment

	e		libia				00	50	discharge, dehydration	
21	Ovin	Male	Sug		2.9	20	4 060 0	5 20	Exhaustion	Ivomec
	e	mare	libia		>		00	0	ext parasite	hipravet
2.2	Ovin		Sug		32	25	2 690 0	3 60	•• p ••	Ivomec
	e		libia		5.2		00	0		hipravet
23	Ovin		Sug		39	27	3 580 0	615		Ivomec hi
	e		libia				00	0		pravet
24	Ovin		Suglibi		5.1	36	5,980.0	6.15		Ivomec.
	e		a				00	0		hipravet
25	Ovin		Sug		9.4	69	11,080,	12,7		Ivermectin
	e		libia				000	00		, hipravet
26	Ovin		Sug		4.1	30	4,080,0	8,85		Ivermectin
	e		libia				00	0		, hipravet
27	Ovin		Sug		4	28		300		Hipravet,
	e		libia					0		Ivermectin
28	Ovin		Sug		4.1	30	4,110,0	5,95		Ivermectin
	e		libia				00	0		, Hipravet
29	Ovin		Sug		3.2	24	4,730,0	5,90		Ivermectin
	e		libia				00	0		, Hipravet
30	Ovin		Sug		4.9	35	4,470,0	3,35		Hipravet,
	e		libia				00	0		Ivermectin
31	Capr	Male	Shamb	39.	4	30.				
	ine		at	2		1				
32	Capr	Fem	Shamb	39.					Arthritis,	
	ine	ale	at	4					corneal	
									obacity, and	
									mastitis	
33	Capr	Fem	Shamb	40.					Diarrhea	Antiflad,
	ine	ale	at	2						Antiacids
34	Dog	Male	Aborof						Allergy	Dexameth
			north							azone,
										Tetracycli
2.5	0	Г	C1 1	20						n ointment
35	Capr	Fem	Shamb	<i>3</i> 9.					Chest	
	ine	ale	at	0					oedema,	
1								1	recumbant	

# Table (12): Results from microbiological check for swabs collected randomly from sub group C–I (trial 1)

Animal	swap	Bacteria
428	Nasal discharge	Staph aureus & streptococcus spp
	Lacrimation	Corynbacterium spp
437	Nasal discharge	Pseudomonas spp
439	Nasal discharge	Staph aureus & gram negative rods

### Table (13-1)

Case no	RBCs	Hb gm	Hb%	WBCs	L%	N%	M%	Е%	В%
430	19,950,000	6	42	10,250	58.695	37.826	2.173	0.869	0.434
156	20,350,000	6.8	48	10,600	74.626	14.925	9.425	0.497	0.497
429	21,850,000	7.8	55	9,250	69.856	22.966	6.698	0.478	0
157	16,250,000	4.6	42	8,900	88.744	10.433	1.731	2.597	2.597
159	21,560,000	6.9	49	7,300	88.88	2.173	1.304	3.381	3.864
158	11,850,000	4.6	34	8,050	55.445	6.93	0.99	3.465	33.168
438	21,640,000	6.4	53	4,950	61,962	8.256	2.293	16.973	10.55

Blood check result for animals'sub group C-I (pneumonia)

Experimental infection of pneumonia (trial 1) before treatment

### Table (13- 2): After treatment

Case no	RBCs	Hb gm	Hb%	WBCs	L%	N%	M%	Е%	B%
430	11,750,000	6	44	17,100	68.619	13.807	12.33	3.765	1.673
156	22,590,000	8.2	57	9,580	82.042	9.507	5.281	1.405	1.760
429	17,930,000	6.4	46	7,450	70.722	10.266	17.11	1.52	0.380
157	14,160,000	7.2	52	6,900	72.684	12.037	5.095	4.166	6.018
159	16,270,000	7	50	13,400	72.243	18.250	4.185	3.422	1.901
158	13,730,000	601	42	18,400	67.566	28.828	2.252	1.351	0
438	20,780,000	8	56	6,150	52.438	44.390	9.268	2.439	1

#### Table (14)

Parameters for animal C (subgroup C-II mastitis), after observation for one month and develop of mastitis from chronic to per acute

Day	TempC°	Pulse/min	Heart/min	Resp/min	Notes
$1^{st}$	39.8	120	128	28	First day after beginning treatment
$2^{nd}$	39.8	116	124	32	Second day after beginning treatment
3ed	39.8	92	100	40	Third day after beginning treatment
$4^{\text{th}}$	39.2	88	100	52	Forth after beginning treatment
$5^{\text{th}}$	39.4	80	92	48	Firth after beginning treatment
6 <sup>th</sup>	39.4	64	64	32	Sixth after beginning treatment
7 <sup>th</sup>	39.3	88	92	36	Seventh after beginning treatment

### REFERENCE

Abu-Groun, EAM.; Taylor, RR.; Varsani, H.; Wadher, BJ.; Leach, RH.; Miles, RJ. (1994). Biochemical diversity. Within the Mycoplasma Mycoides cluster'. Microbiology (Reading). 140(8):2033 – 2042.

Ahmed, O.A. (2003). Bovine Mastitis Aetiology, Clinical Aspects and Treatment with Special Reference to Actinomyces Infection. Ph.D. Thesis U of K

Anderson, J.C. (1983). Mastitis in Goats. Goat Veterinary Society Journal. 4:17-20

Anon (1902). Manual Report of the Veterinary Department, Sudan Govarnment.

Bashiruddin, JB.; Taylor, TK.; Gould, AR. (1994). A PCR-based test for the specific identification of mycoplasma mycoides subspecies mycoides SC. Journal of veterinary Diagnostic Investigation, 6(4):428-434; 14 ref.

Bashirudin, JB.; Windsor, GD. (1998). Coloured colonies of mycoplasma mycoides subsp. mycoiodes Sc and Mycoplasma capricolum sub sp capripneumoniae on solid agar media for the presumptive diagnosis of CBPP and CCPP. In: Proceedings of the ARC-Onderstepoort OIE International congress with WHO -cosponsorship on Anthrax, Brucellosis, CBPP, Clostridial and Mycobacterial diseases. 9-15, August 1998, Kruger National Park, South Africa, 226-229.

Bascu-ana, CR.; Mattsson.; JG. Bjlske, G.; Johansson, K-E. (1994). Characterisation of the 16S rRNA genes from mycoplasma sp.strain F38 and development of an identification system based on PCR. Journal of Bacteriology, 176:2577-2586.

Belton, D.; Leach, RH.; Mitchelmore, DL.; Rurangirwa, FR.; (1994). Serological specificity of a monoclonal antibody to Mycoplasma capricolum strain F38, the agent of contagion caprine pleuropneumonia .Veterinany Record, 134(25): 643-646.

Bjlske, G. (1995). Respiratory mycoplasmosis in goats: especially with regard to contagious caprine pleuropneumonia .Ph D. thesis, Uppsala, Sweden.

Bjlske, G.; Mattsson, JG.; Bascun<sup>macron</sup>~ana, CR.; Bengstr<sup>5</sup>m, K.; Wesonga, H.; Johansson, KE. (1996). Diagnosis of contagious caprine pleuropneumonia by detection and identification of mycoplasma capricolum subsp. Capripneumoniae by PCR and restriction enzyme analysis. Journal of Clinical Microbiology, 34 (4):785-791.

Blood, C.D.; Radostitis, O.M. and Henderson, J.A. Veterinary Medicine .5<sup>th</sup> Edition. (1979). 6<sup>th</sup> Edition (1983, 1985). Bailliere Tindall, Landon .451- 493.

Boddie, GEO. (1969). Diagnostic methods in Veterinary Medicine. Six Edition. (1969)

Bradbury, JM. (1983). Phosphatase activity. In: Razin S, Tully JG, eds. Methods in Mycoplasmology Vol. 1. Mycoplasma characterization. New York, USA: Academic Press, 363-366. Buxton, A. and Frazer, G. (1977) .Animal Microbiology volume I. Blackwell scientific publication, Oxford.

Bywater, R.J. (1977). Antibiotics and Mastitis. Veterinary Annual. 17:55

Cho, HJ.; Ruhnke, HL. And Langford, EV. (1976). The indirect haemagglutination test for detection of antibodies in cattle naturally infected with mycoplsma. Canadian Journal of Comparative Medicine, 40: 20-29.

Cole, H.H. (1962) – Introduction to Livestock Production .San Francisco and London W.H Free man and Co., 1962.

Craven, N. (1987). Efficacy and financial value of antibiotic treatment of bovine clinical mastitis during lactation. A Review .British Veterinary Journal. 143: 410-422.

Cripps, P. (1986). Prevention and Control of Mastitis in Goats. Goat Veterinary society Journal .7 (2):48 – 51.

Devendra, C. and McLeroy, G.L. (1982). Goat and sheep production in the tropics. Longman: London.

Dighero, MW. Bradstreet, PCM. And Andrews, BE. (1970). Dried paper discs for serological identification of human mycoplasmas. Journal of Applied Bacteriology, 33:750-757.

Edwards, S.J. and Brownlee, A. (1946). Therapeutic treatment of Bovine Mastitis .Veterinary Record. 58: 335-343.

Epestein, H. (1971). The origin of domestic animals of Africa, 2. African Publishing Corporation: NewYork.

Food and Agriculture Organization FAO (1985), Manual for animal Health auxiliary Personnel

Freundt, EA. (1983). Culture media for classic mycoplasmas. In: Razin S, Tully JG, eds. Methods in Mycoplasmalogy Vol. 1. Mycoplarma characterization. New York, USA: Academic Press, 127-135.

Freundt, EA. (1983). Preteolytic activity. In: Razin S, Tully JG, eds, Methods in Mycoplasmology .Vol.1. Mycoplasma characterization. New York, USA: Academic Press, 367-371.

Guerin, C.; Thiaucort, F.; Mady, V.; Breard, A.; Lefevre, PC. (1993). Rapid diagnosis of contagious caprine pleuropneumonia in pleural fluids by immunobinding assay. Small Ruminant Research, 12 (2):193-200.

Griffin, T.K.; Dodd, F.H. and Bramley, A.J. (1982). Antibiotic therapy on the control of mastitis. British Cattle Veterinary Association 1981-1982. Proceedings, 137 – 152.

Haenlein, G.F.W. (1980) Mineral nutrition of goats. J. Dairy Sci., 63, 1729 – 48.

Hall, H.T.B. (1985). And Devendra and Mcleroy (1982). Diseases and Parasites of livestock in the tropics (2<sup>nd</sup> edn) Longman: London.

Harbi, MSMA. And El-Tahir, Ms. (1981) Mycoplasma strain F38 and contagious caprine pleuropneumonia in the Sudan .Veterinary Record 108: 261.

Hassan, SMel. ;Harbi, MSMA. And Bakr, MIA. (1984). Treatment of contagious caprine pleuropneumonia. Veterinary Research Communications, 8(1):65-67.

Hinckley, L. and Williams, L. F. (1981). Diagnosis of mastitis in goat. Veterinary Medicine/ small Animal Clinician .76: 711 – 712.

Hutcheon, D. (1889). Contagious pleuro-pneumonia in goats at cape colony, South Africa. Veterinary journal, 29: 399-404

Ibrahim, A.E.A E.T. (1962). A Preliminary Survey of the Aetiology of Mastitis among Goats and Sheep around Khartoum. M.V.Sc Thesis U of K (1962)

International Dairy Federation I.D.F (1971). A monograph on bovine mastitis. I. D. F. Document 60, 1-40

Jones, GE. Wood, AR. (1988). Microbiological and serological studies on caprine pneumonias in Oman .Research in Veterinary Science, 44 (1):125-131.

Jones, GE. (1989). Contagious caprine pleuropneumonia. Technical Series – Office International des Epizooties, No. 9: 63 pp.

Kapur, M.P. and Singh, R.P. (1977). Diagnosis of mastitis-a comparative study of four indirect tests. Haryana Veterinary. 16 (2): 69-73.

Kelly, W.R (1984). Veterinary Clinical Diagnosis. Third Edition. Bailliereand Tindal, London

Knowls, R.H. (1923). Annual Report of the Veterinary Department, Sudan Government.

Kusiluka, LJM.; Semuguruka, WD. and Kazwala, RR. et al., (2000). Demonstration of Mycoplasma capricolum subsp. capripneumoniae and Mycoplasma mycoides subsp. mycoides, small colony type in outbreaks of caprine pleuropreumorina in eastern Tanzania .Acta Veterinaria Scandinavica, 41: 331-319.

Lerondelle, C. and Poutrel, B. (1984). Characteristics of non – clinical mammary infection of goat's .Vet. Bull. (1984) 54 (10) : abstract 6535.

Long, P.E.; Heavner, J.E; Z.V, G. Gelata, I.N. and Nepote, K. (1984). Depletion of antibiotics from the mammary gland of goats. Journal of Diary sciences 67 (63):707 – 712.

MacOwan, KJ. (1976). A mycoplasmsa from chronic caprine pleuropneumonia in Kenya. Tropical Animal Health Production, 8:28 – 36.

MacOwan, KJ. And Minette, JE. (1977). The role of Mycoplasma strain F38 in contagious caprine pleuropneumonia (CCPP) in Kenya. Veterinary Record, 101:380-381.

Manser, P.A. (1986). Prevalence, cause and laboratory diagnosis of subclinical mastitis in goats. Veterinary record. 118 (20): 552-554

March, JB.; Gammack, C. and Nicholas, R. (2000). Rapid detection of contagious caprine pleuropneumonia using a Mycoplasma capricolum subsp. capripneumoniae capsular polysaccharide specific antigen detection latex agglutination test. Journal of Clinical Microbiology, 38:4152-4159.

Mason and Maul, J.P. (1960). The Indigenous Livestock of Eastern Africa and Southern Africa. I: L

Mba, A.U.; Akinsoyinu, A.O. and Olubajo, F.A. (1974). studies on comparative utilization of urea and groundnut cake rations by West African Dwarf goats. I.N-balance and growth. Nigerian J .Anim Prod., 1, 209-16.

Muthomi, EK.; Rurangirwa, FR. (1983). Passive haemagglutination and complement-fixation as diagnostic tests for contagious caprine pleuropnumonia caused by F-38 strain of mycoplasma. Research in Veterinary Science, 35: 1 - 4.

Nesbakken, T. (1976). The cell content in milk of goats. Nordisk Veternaer Medecin 28: 550 – 556.

Newbould, F.H.S and Neave, F.K. (1965). The response of bovine mammary gland to an infusion of staphylococci. Journal of Dairy Research 32: 163-170.

National Research Council NRC. (1981) Nutrient requirements of goats. National Academy of sciences Press: Washington, DC.

Omar, M.Kh. (1990) – Clinical Studies and Treatment Trails on Caprine Mastitis. M.V.Sc Thesis Uof K (1990)

Onovarian, O. The comparative efficacy of some antibiotics used to treat experimentally induced mycoplasma infection in goats. Vet Record, 1974; 94:418-420

Payne, W.J.R. An Introduction to Animal Husbandary in the Tropics. Forth edition (1990).

Pettersen, K.E. (1981). Cell content in goat's milk Acta Veterenaria Scandanaviea. 22: 226 – 237.

Poutrel, B. and Lerondelle, C. (1983). Cell content of goat milk. California Mastitis Test, Coulter Counter and Fossomatic for predicting help infection. Journal of Diary Science. 66:2575 -2579.

Pyörälä, S. (1988). Clinical aspects of bovine mastitis and treatment during lactation. Academic Desertation. College of Veterinary Medicine, Helsinki, Finland.

Robinson, T.C. (1981), Therapy for acute and per acute mastitis. In: Mastitis control and herd management. Technical Bulletin 4. National Institute for Dairying .Reading, England. 128-134.

Reichmuth, J. (1975). Proceeding of a seminar on bovine mastitis. I.D.F. document 85, 93.

Roganisky, M. and Grandmey, T. (1977). Characteristic of caprine mastitis staphylococci. Veterinary Bulletin 1980. 50 (1): Abstract 4707.

Rosendal, S. and Black, FT. (1972). Direct and indirect immunofluorescence of unfixed and fixed mycoplasma colonies. Acta Pathologica et Microbiologic Scandinavica, 80:615 – 622.

Rurangirwa, FR. (1996). Contagious caprine pleuropnuemonia. In: Manual of standards for Diagnostic tests and Vaccines. Office International Epizootic, 374 – 383.

Rurangirwa, FR.; McGuire, TC.; Kibor, A. and Chema, S. (1987). A latex agglutination test for field diagnosis of contagious caprine pleuropneumonia. Veterinary Record, 121(9): 191-193

Schalm, O.W. (1965). Veterinary haematology. Second edition. (1965)

Siddique, I.H.; Hafeez, M. and Gbadamosi, S.G (1988). Screening for subclinical mastitis in goats. Testing the tests. Veterinary Medicine. (1988) .83 (1): 87-88.

Smith, M.C. and Roguinsky, M. (1977). Mastitis and other diseases of the goat's udder. Journal of the American Veterinary Medical Association.

Stableforth, A.W.; Edwards, S.J. and Minett, E.C. (1935). Further observation on the control of chronic streptococcus mastitis. Journal of Comparative Pathology and Theraputic. 48: 300-315

Stableforth, A.W.; Husle, E.C.; Wilson, C.D.; Chodkowski, A. and Stuart, P. (1949). Herd Eradication of Streptococcus agalactiae by simultaneous treatment of all cows with 5 doses of 10,000 units of penicillin at daily intervals and disinfection. Veterinary record. 61:357-362

Taylor, TK.; Bashiruddir, JB. And Gould, AR. (1992). Relationships between members of the Mycoplasma mycoides cluster as shown by DNA probes and sequence analysis. International journal of Systematic Bacteriology, 42 (4): 593 -601

Thiaucourt, F. and Bjlske, G. (1996). Contagious caprine pleuropreumonia and other pulmonary mycoplasmoses of sheep and goats. Revue Scientifique et Technique – Office International des epizooties, 15 (4): 1397 – 1414.

Thiacourt, F.; Bjlske, G.; Libeau, G.; Goff, Cle. And Lefevre, PC. (1994). The use of monoclonal antibodies in the diagnosis of contagious caprine pleuropneumonia (CCPP). Veterinary Microbiology, 41 (3): 191 – 203; 36 refs.

Thornton, D.A.K. (1983). Diseases of the mammary gland in goats. Gout Veterinary Society Journal. 4 (1): 12-16.

Wamwayi. HM.; Wafula, JS.; Litamoi, JK. And Nandokha, EN. (1989). Detection of antibody to mycoplasma F38 in goat sera by an enzyme – linked immunosorbent assay. Tropical Animal Health and Production, 21 (1):43 – 49.

Wilson, C. D. (1969). Therapy of bovine mastitis. Veterinary Annual .10: 139 – 148.

Wilson, C.D. (1981). Antibiotic therapy in mastitis control. In: mastitis control and herd Management Technical Bulletin 4. 113 – 127.

Ziv, G. (1980). Drug selection and use in mastitis: Systemic VS local therapy. Journal of the American Veterinary Medical Association .176 (10): 1109 -1115.

### المراجع العربية

د. محمد أبو العزائم مدنى (1969). الثروة الحيوانية و الأنتاج الحيواني في السودان.

د. محمد بشير مفرح. الماعز في السودان. تقرير أعد للندوة المقامة بواسطة أكساد (المركز العربي) 12-17 ديسمبر 1981 (دمشق).

عباس بلال. الماعز و أثره على الغطاء الشجري.