بسم الله الرحمن الرحيم

جامعة القاهرة

كلية الطب البيطرى

قسم الجراحة والتخدير والاشعة

قرار لجنة الحكم والمناقشة

قررت لجنة الحكم والمناقشة ترشيح السيد ط.ب / محمد عطية أحمد عزالـــــدين للحصول على درجة الماجستير فى العلوم الطبية البيطرية (الجراحة والتخدير والأشـــعة) من كلية الطب البيطرى – جامعة القاهرة.

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والمشرف على الرسالة

تحريرا فى : ١٩٩٦/١١/٧

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COMPARATIVE STUDIES ON THE USE OF SOME ANAESTHETIC DRUGS IN EQUINE

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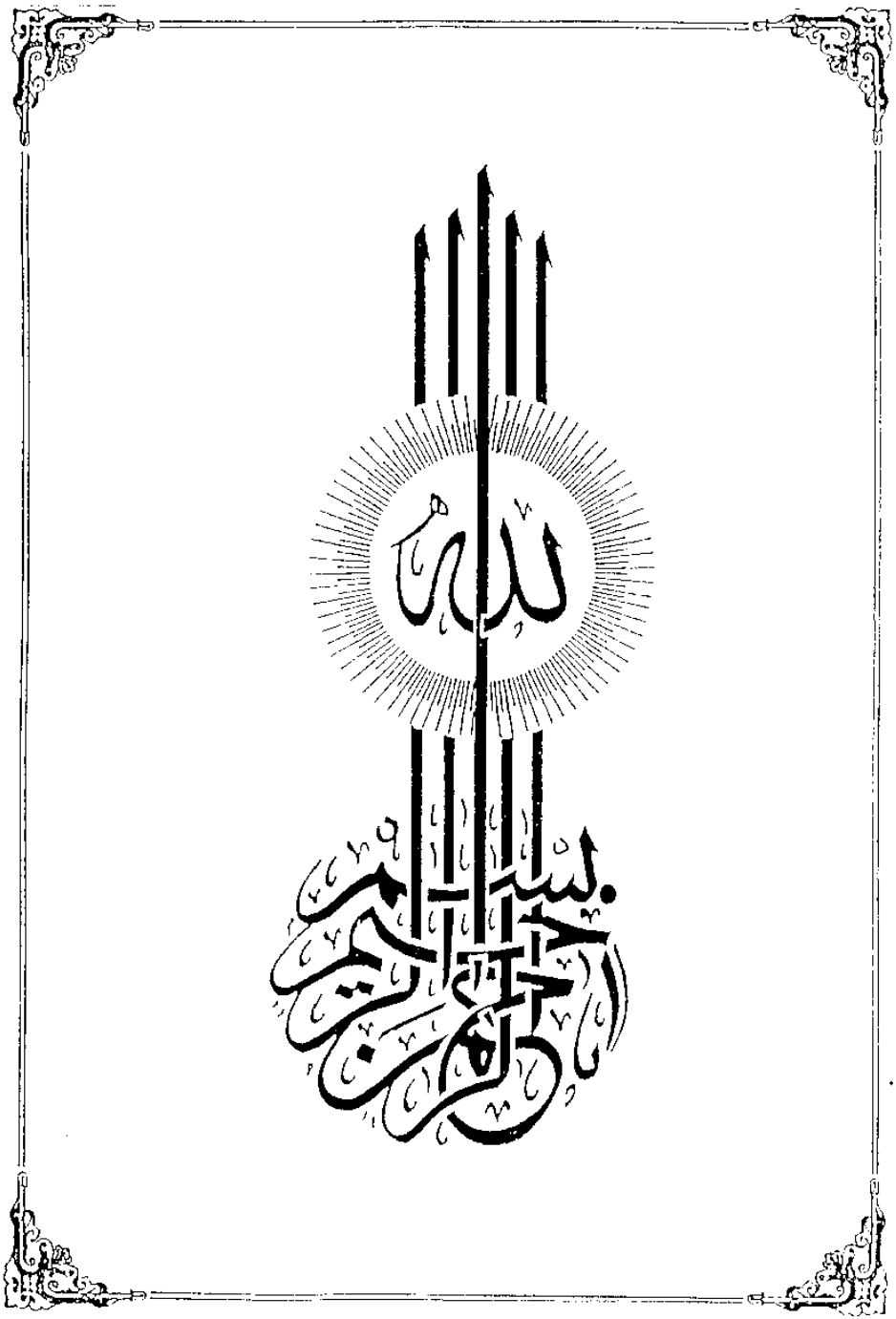
1996



Thanks God

Great thanks
to the member of my
family
and the spirite of my
father

ACKNOWLEDGMENT

I would like to express my profound gratitude and high appreciation to ***Prof. Dr. Adel Hassanein****, Prof. of Surgery, Faculty of Vet. Med., Cairo University for his constructive* Critism*, faithful guidance, advice and unfailing patience through the progress of the work.*

I wish to forward my acknowledgment and grateful thanks to ***Prof. Dr. Ahmed S. Soliman,*** *Prof. of Surgery, Faculty of Vet. Med., Cairo University for his keen supervision, valuable help and encouragement.*

I sincerely offer my grateful thanks to ***Prof. Dr. Sabry A. Awadalla****, Prof. of Food Hygiene, for his helpful suggestions and cooperation.*

Many thanks are also due to the stuff of ``Brooke hospital for poor animals`` for their help.

The author is greatly indebted to all members of the Surgery Department, Faculty of Veterinary Medicine, Cairo University.

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INTRODUCTION

Introduction

Equine anaesthesia has lagged behind human and small animal anaesthesia because of traditional reliance upon various methods of physical restraint, an absence of appropriate and safe equipments and the conservative nature of equine surgeons **(Muir and Hubbell, 1991).**

It is well known that, there is no one drug can produce alone a Satisfactory anaesthesia which fulfill all the requirements of unconsciousness, analgesia and muscle relaxation in order to provide an immobile patient on which various medical or surgical procedures can be performed.

It is important to mention that, with any anaesthetic regimen, the practitioner looks for cardiopulmonary stability, analgesia and uneffected induction and recovery periods.

The present investigations aims at applying balanced general anaesthesia in equine under Egyptian relations in which most of equine populations are still used as draught animalsfor poor farmers and carters.

In this respect several drugs will be tested to select the best combinations that can produce satisfactory anaesthesia taking in consideration the economic point of view, their efficacy and easiness of application by the general veterinary practitioners.

REVIEW OF LITERATURE

Review Of Literature

According to the available literature; the drugs used, in the present study, for production of general anaesthesia in equine were classified into the followings:

I. Preanaesthetics

A. Tranquillizers:

1- Phenothiazine derivatives:

a- Chlorpromazine hydrochloride.

b- Propionyl promazine.

c- Promazine hydrochloride.

2- Benzodiazepines:

Diazepam.

B. Analgesics:

Nefopam hydrochloride.

II. Narcosis and Anaesthetics.

A- Narcosis:

Chloral hydrate

B- Anaesthetics:

1- Ether.

2- Ketamine hydrochloride.

3- Thiopental sodium .

I. Preanaesthetics

A- Tranquillizers:

1- Phenothiazine derivatives:

a- Chlorpromazine hydrochloride

(2 chloro-10- 93- dimethyl amino propyl phenothiazine hydrochloride)

The pharmacological action:

-Effect on the central nervous system:

Terzian (1952 / 1953); Aron, Chambon, and Voisim (1953); Courvoisier, Fournel, Ducrot, Kolsky and koetschet (1953); fritsch (1955); Bovet, Longo and Silvestrini (1957); Demaar, Martin and Unna (1958); Wright (1971); Nicholas and leslie (1982) and Goodman and Gilman (1991) reported that chlorpromazine hydrochloride has a powerful central and peripheral depressant action. The central effect is probably through its action on the basal ganglia, hypothalamus and substantia reticularius of the brain and the ascending reticular tissue formation of the mid brain which contains the centers of vomiting and temperature control. **Wright (1958) and Raker and Sayers (1959)** mentioned that the drug acts by depressing the brain stem or lower nervous system (thalamus, hypothalamus and reticular formation) rather than cerebral cortex. Concerning the effect of the drug on the reticular system **Bovet et al., (1957) and DeMaar et al., (1958)** believed that the drug has a direct depressive action on this system.

Killam (1962) believed that chlorpromazine hydrochloride stimulates the reticular system which increases the input and conduction of the reticular formation with a decrease in the response to stimuli.

On the autonomic nervous system, **Fritsch (1955)** mentioned that the drug has a strong sympatholytic and weak parasympatholytic effect sufficient to reduce the intestinal spasm and peristalsis, bronchial and salivary secretion. **Preston (1956) and Witzel (1956)** reported that the drug can block conditioned response to unconditioned stimuli such as electric shock.

Bradly (1963) claimed that the sensory collateral rather than the reticular system itself were depressed by the drug. This explains the indifference and unresponsiveness to sensory stimuli without sedative effects which is the basis for its tranquilizing action.

Chlorpromazine hydrochloride acts as a skeletal muscle relaxant as mentioned by **Henatsch and Ingvar, 1956; Neff and Rossi, 1962; Scheidy and Mcnally, 1958; Nicholas and Leslie, 1982 and Goodman and Gilman, 1991).**

On the skeletal muscle the drug posses a depressant effect on muscle tone which is probably related to the motor tracts in the brain stem **(wright, 1958)** and also cause relaxation in some type of spastic condition **(Goodman and Gilman, 1991).**

-Effect on the cardiovascular system:

The drug produces direct effect on the heart and blood vessels, and has an indirect one through its actions on central nervous system and autonomic reflexes. The drug has an antifibrillatory effect upon the heart,

this may be due to a quinidine-like action or to a local anaesthetic effect similar to that seen with procain. It may also be related to an adrenergic blocking action **(Goodman and Gilman, 1991).**

The drug can prevent epinephrine-induced ventricular fibrillation during the use of halogenated anaesthetics similar to α-adrenergic blocking agent, or due both to quinidine like action or to a local anaesthetic effect **(Nicholas and Leslie, 1982 and Goodman and Gilman, 1991).**

Reflex tachycardia was commonly seen following administration of chlorpromazine hydrochloride **(Wright, 1971 and Goodman and Gilman, 1991).**

Hypertension, most commonly seen, is primarily due to inhibition of centrally mediated pressor reflexes, but peripheral α-adrenergic blockade may also play a role. The drug has a vasodilating action due to its effects on the autonomic nervous system and a direct action on blood vessels. For this it antagonises and sometimes reverses the pressor and vasoconstrictor action of adrenaline **(Donnet and Garnier, 1955).**

Hershy et al (1956) and Zweifach and Antopal (1956) said that although chlorpromazine protects against haemorrhagic hypotension, the drug tends to abolish the carotid sinus reflex which regulate blood pressure and respiratory rate.

During chlorpromazine hydrochloride medication, a fall in blood pressure was beyond 10-20%. Furthermore **Lumb and Jones (1973)** said that the hypotensive effect of the drug may produce shock.

-Effect on the respiratory system:

It tends to abolish the carotid sinus reflex which regulates blood pressure and respiratory rate **(Hershy et al, 1956).**

The highest concentration of chlorpromazine hydrochloride was found in the lung followed by the liver, the adrenals and the spleen **(Walkanstein and Seifter, 1959).**

-Effect on the liver:

Salzmann and Brodie (1956) mentioned that chlorpromazine hydrochloride is partly detoxicated in the liver. **Wright (1958) and Muller and Lettow (1959)** found no hepatic damage in their experimental studies on horses. **Westhus and Fritsch (1964) and Huang, Yeh and Muni (1970)** said that the action of the drug is usually enhanced in animals suffering from liver damage.

Also, in this respect **Jarvik (1968)** noticed an increase in bile viscosity without clinical signs. **Walkenstein and Seifter (1959) and Soma (1971)** said that about 60-70% of an administrated dose of chlorpromazine hydrochloride was rapidly removed from the portal circulation by the liver and there was a very active hepatic circulation.

-Effect on the kidney:

Acute and cumulative toxicity tests showed that chlorpromazine hydrochloride is well tolerated in domestic animals **(S.K. F., 1953; Dubost and Pascal, 1953; Dobkin, Gilbert, and Lamoureux, 1954 and Scheidy and Mcnally, 1958).**