PATHOLOGY OF THE LIVER IN QUAILS

(Coturnix coturnix japonica)

By

S. R. KRUPESHASHARMA

THESIS

Submitted in partial fulfilment of
the requirement for the degree

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University

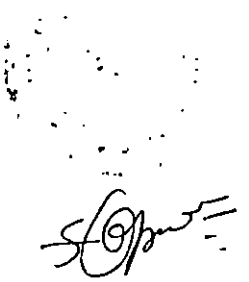
Centre of Excellence in Pathology
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
Mannuthy - Thrissur

1992

Dedicated to my Mother
Father and Sister

DECLARATION

I hereby declare that the thesis entitled **PATHOLOGY OF THE LIVER IN QUAILS (*Coturnix coturnix japonica*)** is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship fellowship or other similar title, of any other University or Society.



Mannuthy,
30 July 1992.

S.R. KRUPESHASHARMA

CERTIFICATE

Certified that this thesis, entitled **PATHOLOGY OF THE LIVER IN QUAILS (Coturnix coturnix japonica)** is a record of research work done independently by Sri. S.R. Krupeshasharma under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.

Dr. A. RAJAN
(Chairman, Advisory Committee),
Director,
Centre of Excellence in Pathology,
College of Veterinary & Animal Sciences,
Mannuthy.

Mannuthy,
30 July 1992.

CERTIFICATE

We, the undersigned members of the Advisory Committee of Sri. S.R. Krupeshasharma, a candidate for the degree of Master of Veterinary Science in Pathology, agree that the thesis entitled "**PATHOLOGY OF THE LIVER IN QUAILS (Coturnix coturnix japonica)**" may be submitted by Sri. S.R. Krupeshasharma, in partial fulfilment of the requirement for the degree.

Dr. A. RAJAN,
Chairman, Advisory Committee,
Director,
Centre of Excellence in Pathology,
College of Veterinary &
Animal Sciences, Mannuthy

Dr. K.M. Ramachandran,
Professor,
Centre of Excellence in
Pathology.

Dr.G.Krishnan Nair,
Associate Professor,
Department of Microbiology.

Dr. T. Sreekumaran,
Associate Professor,
Centre of Excellence in
Pathology.

External Examiner

ACKNOWLEDGEMENTS

I wish to express my deep sense of gratitude and indebtedness to **Dr.A. Rajan**, Director, Centre of Excellence in Pathology and Chairman of the Advisory Committee for his constant encouragement, valuable suggestions, proper guidance and critical advice throughout this work.

I am indebted to **Dr. K.M. Ramachandran**, Professor and **Dr. T. Sreekumaran**, Associate Professor, Centre of Excellence in Pathology for their help and encouragement as members of the Advisory Committee, throughout the course of this work.

I express my sincere gratitude to **Dr. G. Krishnan Nair**, Associate Professor, Department of Microbiology, for his valuable guidance and constant help, as a member of the Advisory Committee.

Grateful acknowledgements are made to the staff of the Centre of Excellence in Pathology for the help rendered during the period of my study.

I am thankful to **Dr. S. Sulochana**, Professor and Head, Department of Microbiology, for providing facilities for carrying out bacteriological work.

I am also thankful to the Director, Central Research Institute, Kasuali, for the serotyping of the isolate.

My sincere thanks are due to **Dr. T. Sooryanarayan**, Deejay Enterprises, Bangalore, for his help and encouragement during my study.

My sincere thanks are due to **Dr. K.C. George**, Head of the Department of Statistics, for the help rendered in statistical analysis.

I am grateful to **Dr. M. Krishnan Nair**, Director, Research Co-ordination, Kerala Agricultural University, for his help and encouragement throughout the study.

I am grateful to **Dr. S.K. Chaudhary, Dr. Sheela Yohannan, Dr. Fabiana Lobo** and **Dr. Mini Jose** for the help rendered during the period of this work.

I am also thankful to **Drs. Amir, Deepak, Shaji Antony, Manumohan, S., Sagar, Sunil, B.** and all my friends for their encouragement and gift of rejoicing togetherness, all the way.

I am indebted to the Indian Council of Agriculture Research for providing me the Junior Fellowship for the M.V.Sc. degree programme.

My sincere thanks are due to **M/s Blaise Computer Consultancy, Mannuthy** for the neat and flawless execution of the work.

S.R. KRUPESHASHARMA

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Introduction

1. INTRODUCTION

Quails are popularly known as Bater in India. The wild quails were hunted and their meat enjoyed as a delicacy for long, till they were declared as a Protected Species under the Wild Life Preservation Act, 1972. Rearing Japanese quail (Coturnix coturnix japonica) has now been taken up by Indian farmers on a large scale and it is getting more and more popular as years pass by. The introduction of Japanese quails in the country during the seventies has opened a new line of poultry production and created an alternative for diversification. In Kerala, of late, quail farming has been taken up on a large scale.

These birds are very useful for research in the laboratory because of their short generation interval, low feed and space requirement, and high rate of egg production. Quails can be managed successfully by methods which are used for rearing chicken. Japanese quail has been recommended as research model for chicks due to similar physiological and morphological characteristics.

Although, quails in their natural habitat are relatively resistant to many diseases, their domestication and artificial feeding have made them vulnerable to many pathogens. However, the incidence and nature of different

disease conditionswhich affect quails are not clearly known, particularly in Kerala.

Liver is an important organ which controls the health and productivity of birds. Several specific and non-specific disease conditions which affect quails involve the liver. No other organ is involved in as many complex interrelationships with other organs as the liver in maintaining the internal homeostasis and it holds a centralist position in the maintenance of internal milieu. These complex interrelationships and functions of the liver can be classified under five headings; secretory, haemopoietic, metabolic, defensive and excretory. Liver is concerned with the secretion of bile and is the site of erythropoiesis in the foetal life and extramedullary erythropoiesis in times of need during adult life. It is the primary organ concerned with protein, carbohydrate and fat metabolism. Many of the vitamins are synthesized and many are stored in the liver. Kupffer cells in the liver form a strategic defensive line against harmful microbial invaders. Another vital function of the liver is detoxification of toxins and excess hormones either by conjugation or inactivation.

Liver falls victim to malfunctions because of the complexity of structural organization and functional activities. Hepatocytes are highly specialized in their functions and this high degree of specialization makes them vulnerable even to apparently mild irritants. However, the effects of minor lesions of the liver are not manifested clinically. This is largely attributable to three important hepatic characteristics; a high degree of reserve functional capacity, a complex and multiple activity and its unusual ability for regenerations.

This organ which has direct control on the health and productivity of birds is of vital importance. The productive performance of birds is directly associated with the functional capacity of the liver. Hence, any malfunction of the liver will have a direct impact on the health and productivity of quails and will be responsible for causing serious set back to quail production programmes.

There has not been any systematic investigation on the hepatic disorders in quails and data are not available on the nature and prevalence of different types of hepatic disorders encountered in this species. In the light of the available information based on the autopsy records

maintained at the Centre of Excellence in Pathology, relating largely to the quails of the farm attached to the College and some of the neighbouring commercial quail farms, it was considered worth while to make more detailed studies on the pathology of the liver in order to gather more complete information of the disorders of the liver.

The pathological features encountered in the liver of quails were studied by systematic post-mortem examination of the quails brought for autopsy at the Centre of Excellence in Pathology. Besides this, experimental investigations were undertaken to elucidate the pathogenesis and pathology of some of the conditions encountered during the survey studies. The results obtained have been documented and discussed in the subsequent chapters.

Review of Literature

2. REVIEW OF LITERATURE

Although, no published reports specifically on the pathology of the liver in Japanese quails are available, considerable observations relating to many specific and non specific disease conditions affecting the liver primarily or secondarily have been reported from India and other countries. For the sake of reviewing the published literature, the pathologic conditions affecting the liver in quails were divided as follows:

1. Bacterial diseases.
2. Mycoplasmosis
3. Viral diseases
4. Fungal diseases
5. Protozoan diseases
6. Toxic conditions.

With an objective of providing a comprehensive information on quail diseases, based on the available literature, a general review of the diseases encountered in quails has been presented.

2.1. Diseases of quails

2.1.1. General

Edgar et al. (1964) examined Coturnix quails for susceptibility to several organisms that caused diseases in poultry. Quails were found to be susceptible to Fowl pox, Newcastle disease and Infectious bronchitis viruses, Salmonella pullorum, Salmonella gallinarum, Salmonella typhimurium, Pasteurella multocida, Escherichia coli and Aspergillus fumingatus. Domestically raised Japanese quails were found capable of carrying or were susceptible to visceral leucosis, Salmonella spp., Candida albicans, Mycoplasma, Borrelia anserina, E. coli and Raillietina echinobothrida (Bigland et al., 1965). Lohliger and Schubert (1967) described diseases of Japanese quails and attributed poor management as the cause of mortality in the first five days of life and diseases of reproductive organs, diseases of digestive system, and lymphoid leucosis in adults. Corrado (1970) could not infect quails with the viruses of Newcastle disease, Infectious bronchitis and Infectious laryngo tracheitis, or with Mycoplasma gallisepticum or Pasteurella multocida. Srinivasan et al. (1980) studied the mortality pattern in Japanese quails and observed that pneumonia was the main cause of death in the

younger age group while in adult females it was egg bound condition or peritonitis. In males the mortality was caused by different conditions such as hepatitis, pneumonia, enteritis, Colisepticaemia and Aspergillosis. Pradhan et al. (1983) conducted serological studies on some chicken viral infections in quails and observed that quails got infected with Infectious bronchitis, CELO, Reo and Infectious bursal disease viruses and Mycoplasma. They also found quails were refractory to Newcastle disease virus infection. Mortality pattern in quails as influenced by sex, age and month of the year was studied by Suneja et al. (1985). They observed highest mortality during the first week of life, followed by those aged one to six weeks. They did not find any significant difference in mortality between sexes. Pneumonia was the most prevalent cause of mortality. Sharma and Kaushik (1986) studied the incidence of diseases in Japanese quails. They encountered different disorders like pneumonia, Ulcerative enteritis, yolk retention, Colibacillosis, Egg peritonitis, hepatitis, Avian leucosis complex and Aspergillosis. Gangadharan et al. (1989) analysed the mortality pattern in quails and reported that omphalitis and hepatosis were the common disorders encountered in the younger age group, whereas in adults, hepatitis and enteritis were the common disorders.

2.1.2. Bacterial diseases

2.1.2.1. Ulcerative enteritis.

Durant and Doll (1941) reported Ulcerative enteritis(UE) in quails and attributed the etiology to an anaerobic gram positve bacillary organism. An outbreak of UE in wild California quails was reported by Buss et al. (1958). Peckham (1959) was able to isolate gram positive, spore forming rods from quails affected with UE. Twenty strains of gram positive, spore forming rods were isolated from chicken embryos from outbreaks of UE in chickens, poults and quails (Peckham, 1960). The disease was reproduced in quails by feeding the liver or intestinal suspensions of diseased quails. In Britain, UE was reported for the first time by Harris (1961). Davis et al. (1971) reported that quail could be the biological indicator in the differentiation of Ulcerative enteritis and necrotic enteritis. Davis (1973) showed Coccidiosis and stress as predisposing factors for UE. Berkhoff and Campbell (1974 a) studied the etiology and pathogenesis of UE in experimentally induced disease in quails. Berkhoff et al. (1974 b) isolated the causative organism of UE and observed that it differed from Clostridium perfringens. Berkhoff et al. (1974 c) described the characterization of the causative

anaerobe which caused UE, and tentatively named it as Cl. colinum. An outbreak of UE among quail chicks was reported by Srinivasan et al. (1981). Sharma et al. (1986) observed UE in 19.7 per cent cases during surveillance of diseases in quails between 1979 and 1983.

Other Clostridial infections

Cygan and Nowak (1974) were able to infect quails with Cl. perfringens type C isolated from chicks with acute necrotic enteritis. They observed that lesions in Japanese quails were similar to those in naturally infected chicks.

Mehler (1981) reported that quails were fifteen times more sensitive than mice to Cl. botulinum type C toxin.

2.1.2.2. Diseases caused by Pasteurella Spp.

Vallee et al. (1972) isolated Pasteurella anatipestifer from a flock affected by an outbreak of a disease with high mortality.

Panigrahy and Glan (1982) isolated Pasteurella multocida serotype 3 from one flock of Coturnix quails and

two flocks of bobwhite quails affected by acute fowl cholera which caused high mortality. Derieux (1983) studied the reaction of bobwhite and Coturnix quails to avirulent and virulent P. multocida and observed that the CV strain of P. multocida was moderately virulent for Coturnix quail by the oral route and highly virulent for bobwhites by stick wing. Myint and Carter (1988) reported an outbreak of fowl cholera for the first time in Burma, which caused heavy mortality. Polero et al. (1988) reported an outbreak of cholera in a breeding and egg producing flock of Japanese quails after the introduction of new birds from another flock. Glisson et al. (1989) isolated P. multocida from three flocks of Japanese quail which showed high mortality and described the lesions.

2.1.2.3. E. coli infections

The occurrence of E. coli infections in quails was reported by Bigland et al. (1965) and Sharma et al. (1986). Silva et al. (1989) reported the occurrence of Coligranulomatosis in a Brazilian flock of Coturnix quail which caused mortality and severe drop in egg production.

Sharma (1986) observed Colibacillosis in 10.6 per cent cases during the surveillance of diseases in Japanese quails. Ito et al. (1990) reported Colibacillosis in Japanese quails and described the gross and microscopic lesions.

2.1.2.4. Tuberculosis

Karlson et al. (1970) described the usefulness of Japanese quails for studies on experimental avian tuberculosis. They also described extensive tuberculous lesions, particularly in the liver and spleen in experimentally infected quails. Jsovsky and Svoboda (1973) were able to produce tuberculosis in Japanese quails and reported that Japanese quails were more resistant than the fowl to this infection. Histological examination showed that specific granulomas did not form in the organs.

2.1.2.5. Campylobacter infections

Minakshi et al. (1988) identified C. jejuni biotype 2 in caecal and cloacal swabs from four of 23 domestic quails. Maruyana and Katsube (1990) isolated C. jejuni from

the eggs and organs in experimentally infected laying Japanese quails.

2.1.2.6. Proteus infections

Sah et al. (1983) incriminated proteus infection as the cause of high mortality in a flock of quail chicks. The organism was identfied as Proteus mirabilis. Myint (1987) reported Proteus mirabilis infection in quail chicks in Burma which caused 65 per cent mortality.

2.1.2.7. Coxiella burneti infections

Schmatz and Busche (1977) produced Coxiella burneti infection in quails by nasal inoculation of a suspension of the organism. The agent was re-isolated from the infected birds.

2.1.2.8. Coryza

Reece et al. (1981) isolated Haemophilus paragallinarum from the infraorbital sinuses of the quail chicks which had mucoid to mucopurulent sinusitis, ulceration of the cornea, and conjunctivitis.

2.1.2.9. Listeriosis

Nikuradza (1970) was able to infect quails with a virulent culture of Listeria monocytogenes. None of the birds showed monocytosis in the circulating blood. It was reported that quail may play an important role in the spread or perpetuation of Listeriosis.

2.1.2.10. Infections caused by Salmonella Spp.

Salmonella gallinarum infection:

Awaad et al. (1981) infected Japanese quails with a streptomycin-resistant Salmonella gallinarum and observed lesions comprising of congestion and haemorrhages in parenchymatous organs and misshapen ovaries. It was reported that S. gallinarum infection was transmitted vertically in Japanese quails in a way similar to that in chicken. An outbreak of S. gallinarum infection in Japanese quail which caused high mortality in one to three day old quail chicks was reported by Sarma et al. (1988). Mathew and Sulochana (1990) were able to isolate S. gallinarum which caused heavy mortality among quails of all ages in a farm.

Salmonella pullorum infection:

In an experimental study, Edgar (1964) et al. reported that quails were susceptible to S. pullorum.

Paratyphoid infections:

Graham (1936) studied an outbreak of paratyphoid among quails in which Salmonella aranienburg was found to be the causative organism. In an experimental study, Edgar et al. (1964) showed that quails can be infected with Salmonella typhimurium. Bigland et al. (1965) reported that domestically raised Japanese quails were capable of carrying infections by Salmonella Spp. The species they isolated were S. anatum, S. give, S. infantis, S. london and S. kentucky. Poorciau and Pringer (1978) reported the frequency and duration of paratyphoid organism shedding by bobwhite quail chicks experimentally infected with S. urbana, S. infantis, S. newport, S. gaminura, S. braenderup and S. litchfield. A heavy mortality in quail chicks caused by S. bareilly was reported by Kapoor et al. (1980).

2.1.2.11. Erysipelas:

A severe outbreak of erysipelas causing high mortality in Coturnix breeder quails was reported by Panigrahy and Hall (1977).

2.1.3. Mycoplasmosis:

Bigland et al. (1965) reported that quails can be infected with Mycoplasma of the N-strain. Tiong (1978) was able to isolate Mycoplasma gallisepticum from the sinuses of three 45-day-old quails suffering from contagious purulent sinusitis. Nascimento and Nascimento (1986) isolated M. gallisepticum from the sinuses of Japanese quails with sinusitis. They observed that one of the isolates was pathogenic to chicken.

2.1.4. Viral diseases

2.1.4.1. Marek's disease

The natural occurrence in quail of Marek's disease (MD), earlier known as fowl paralysis, was first reported by Wight in 1963. Experimentally, Japanese quails were found susceptible to Marek's disease virus (MDV) (Dutton et al.,

1973; Figimoto et al., 1975 and Khare et al., 1975). Wight (1963) suggested the possibility of using Japanese quail and dometic fowl crosses to study the genetic susceptibility of quails to this disease. Narita et al. (1975) were able to find the MD-Specific agar gel precipitating antigen in the feather tips or feather follicular epithelium of some of the quails infected with JM strain of MD herpes virus. Natural cases of Marek's disease in Japanese quails were reported by Pradhan et al. (1985) and Nair et al. (1986). In both the cases, gross lesions were confined mostly to the liver and spleen. The epizootiological and pathological aspects of MD in Japanese quails were studied by Kobayashi et al. (1987). Imai et al. (1990) isolated MD virus from Japanese quails with problems of recurring outbreaks of lympho-proliferative disorders resembling MD. Kaul and Pradhan (1991) in a study of immunopathology of MD in quails, detected antinuclear antibodies in the sera of quails with MD. In addition to the presence of antinuclear antibody, immune complexes were also detected in the glomeruli of the kidney of quails infected with Marek's disease virus.

2.1.4.2. Leucosis complex

Bigland et al. (1963) described various manifestations of Leucosis complex in the original strain of quails in USA. Bigland et al. (1965), in a flock survey of diseases of Japanese quails, observed leucosis in 21.6 per cent cases. Wight (1963) gave an account of the macroscopical and histological lesions of naturally occurring fowl paralysis and lympoid leucosis in Japanese quails. Lohliger and Schubert (1967) (1967), Suneja et al. (1985), and Sharma et al. (1986) observed lymphoid leucosis during the analysis of mortality pattern in Japanese quails. The effect of Avian myeloblastosis virus in Japanese quails was studied by Moscovici and Macintyre (1966). They reported that the virus induced a range of neoplasms in Japanese quails similar to that in fowl with one exception of the total absence of acute myeloblastic leukemia in quail. They ascribed the cause of the difference to the heterogenicity of the virus and to the genetic composition of the quail cell. Lohliger and Schubert (1967) studied the etiology and pathology of lymphoid-cell leucosis in Japanese quails. Voronin et al. (1970) could not find viral infections in quails kept separate from fowl but could find avian leucosis agents in quails kept on chicken farms. Nishimura et al.

(1970) reported an Epizootic reticulam cell sarcoma which appeared suddenly in a colony of Japanese quails. Carlson et al. (1974) reported an outbreak of Reticuloendotheliosis which occurred in a flock of Japanese quails. The most striking and consistent lesion observed by them were thickening and nodular formations along the digestive tract. Nedyalkov et al. (1975) studied the experimental Erythroblastosis in Japanese quails with the Bulgarian E-26 strain (Sub group A). Yoshikawa et al. (1975) examined histologically, tumors induced in quails, by the Schmidt - Ruppin strain of Rous Sarcoma virus. They suggested an immunological reaction against tumour cells by lymphoid cells. A naturally occurring Lymphoproliferative disease in Japanese quails was reported by Schat and Gonzalez (1976). Ratnamohan et al. (1979) studied the transmission of an Australian strain of reticuloendotheliosis virus to adult Japanese quails. Bozhkov and Toshkov (1984) observed anisocytosis, poikilocytosis and erythroblastosis in quails produced by erythroblastosis virus strain E-26 from fowl.

2.1.4.3. Newcastle disease

Experimental infection of Japanese quail with B_1 and Grumble bony strains of Newcastle disease virus was reported

by Edgar et al. (1964). The natural outbreaks of Newcastle disease (ND) among Japanese quails were reported by Higgins and Wong (1968) and Hashimoto et al. (1969). Both the outbreaks were characterized clinically by high mortality, lowered egg production, and neurological signs. Zarzueto et al. (1969) suggested the importance of quails in the dissemination of ND. Corrado (1970) and Pradhan et al. (1983) could not induce infection in Japanese quails in experimental inoculation of ND virus. Kumaran and Venkatesan (1991) stated that Japanese quails got the infection of ND under stress conditions.

2.1.4.4. Quail bronchitis

Dubose et al. (1958) isolated a virus from bobwhite quails in a flock with respiratory symptoms and high mortality. Dubose and Grumbles (1959) observed that quail bronchitis virus and CELO virus produced a similar disease in bobwhite quails. These viruses were considered to be the same agent as they could not be differentiated. Outbreaks of quail bronchitis were reported from Singapore

(Chew-Lim, 1980) and in India (Pradhan et al., 1980). A fall in egg production by 10 to 15 per cent and respiratory distress were observed in these two outbreaks. Jack and Reed (1988) reproduced quail bronchitis experimentally in captive reared, one-week-old bobwhite quails inoculated with quail bronchitis virus. They also demonstrated large intranuclear inclusions, typical of adenovirus infection, in the trachea, lungs, liver, and bursa. The mortality pattern and clinical signs in experimentally induced quail bronchitis in bobwhite quails were studied by Jack and Reed (1989). The mortality rates were high in quails inoculated at one or three weeks of age than in quails inoculated at six or nine weeks of age. The histopathology of experimentally induced quail bronchitis was first reported by Jack and Reed (1990).

2.1.4.5. Inclusion body hepatitis

Jack et al. (1987) were able to isolate group I adenovirus from the liver of dead farm-reared bobwhite quails. The liver of these quails had multiple pale white foci in the liver. Jack and Reed (1990) studied the avian adenovirus associated with Inclusion body hepatitis (IBH) in bobwhite quails and stated that this virus was

indistinguishable from quail bronchitis virus. it was indicated that IBH of bobwhite quails was a manifestation of quail bronchitis.

2.1.4.6. Avian pox

Rinaldi et al. (1969); EI Dahaby et al. (1971); and Rinaldi et al. (1972) reported that Japanese quails were susceptible to infections with pox viruses. Cross immunity tests showed that pox viruses naturally infecting quails were more closely related to pigeon pox virus than to fowl pox virus. (EI Dahaby et al., 1971). One strain of pox virus (241-63-PV) isolated by Rinaldi et al. (1972) was found to be different from the other pox viruses in its biological properties and experimental inoculation by the intracerebral route caused encephalitis and high mortality in quails. Avian pox in California quail in Oregon was first reported by Crawford (1979). The prevalence of avian pox in adult and immature California quail was studied by Crawford (1986).

2.1.4.7. Infectious Bronchitis

Edgar et al. (1964) reported briefly on experimental Infectious bronchitis infection in quail. Quails were found

to be susceptible but the details of the experiment were not available. Corrado (1970) could not produce Infectious bronchitis (IB) in quails. Pradhan et al. (1983) demonstrated that quails were susceptible to IB virus.

2.1.4.8. Avian influenza

Many strains of type A influenza virus were isolated from outbreaks of respiratory disease in quail flocks in Italy (Mandelli et al., 1968 and Nardelli et al., 1970). Rossi et al. (1972) described another outbreak in Italy where the whole colony was affected after the introduction of a group of breeding birds. By the haemagglutination inhibition and complement fixation tests, the virus was found to be antigenically related to type C influenza virus. This was of interest as type C influenza virus was not otherwise isolated from a non-human host.

2.1.4.9. Rabies

There was one report from France concerning rabies virus surviving and retaining its virulence for 112 days in one of the six intracerebrally inoculated quails (Villemin, 1972). Histological examination of this bird revealed the presence of Negri bodies.

2.1.4.10. Eastern equine encephalomyelitis (EEE)

In one single report of EEE virus infection where more than 90,000 birds died (Eleazer et al., 1978), the prominent clinical signs were paralysis and torticollis, and the birds died within few hours after the manifestation of symptoms. This case report was of interest because quails could be used as an experimental animal model for the study of atleast some arbovirus infections in birds (Ratnamohan, 1985).

2.1.4.11. Avian encephalomyelitis

Apparent natural infection and also experimental infection with avian encephalomyelitis virus (AEV) were reported by Hill and Raymond (1962).

2.1.5. Fungal diseases

2.1.5.1. Candidiasis

Bigland et al. (1965), in a flock survey of diseases of Japanese quails, observed candidiasis in 5.7 per cent cases.

2.1.5.2. Aspergillosis

Natural occurrence of aspergillosis in Japanese quails was reported by Olson in 1969. The susceptibility of Japanese quails to Aspergillosis was reported by Edgar et al. (1964), Srinivasan et al. (1980) and Sharma & Kaushik (1986). Ghori and Edgar (1973) observed that Coturnix quails exposed to Aspergillus fumigatus spores at hatching suffered greater mortality from the ensuing disease than did chicken and turkeys exposed at the same time. Reece et al. (1986) in a study on mycosis of commercial quail, ducks and turkeys, reported brain lesions in some quails showing nervous signs. Chaudhary et al. (1988 a) were able to observe characteristic gross and microscopic lesions by intratracheal inoculation of young quail chicks with Aspergillus fumigatus spores. Re-isolation of the fungus was consistently achieved from the lungs, airsacs, and trachea upto 14 days. The clinical signs and haematological changes in experimental Aspergillosis in Japanese quails were studied by Chaudhary and Sadana (1988 b).

2.1.6. Protozoan diseases

2.1.6.1. Coccidiosis

Mazurkiewiez et al. (1967) described anorexia, death and decreased reproduction in quails infected with Eimeria tsunodai. The life cycle of E. bateri, a parasite of the Indian quail, was described by Norteon and Peirce (1969). Shah and Johnson (1971) could not transmit E. bateri from quail to chicken. They reported that this coccidium is host specific. Acedo and Reguera (1972) reported an outbreak of coccidiosis among two-month-old Japanese quails caused by E. bateri. Pathogenicity of E. tsunodai for Japanese quails and the susceptibility of the coccidium to certain drugs was studied by Tsutsumi and Tsunoda (1972). The oocyst size and description of the coccidia identified as E. tsunodai and E. taldykargamica in quails were reported by Svanbaev and Utebaeva (1973). Zapryanov (1979) reported that E. tenella was able to develop in two-week-old quails, but the quail coccidium, E. kufoidi, was unable to develop in chicks. Ruff and Fegan (1984) identified the coccidia recovered from field outbreaks in the commercially raised Japanese quails as E. uzura, E. tsunodai and E. taldykargamica and studied their pathogenicity. Zuo and Chen (1986) were the first to

report Coccidiosis in quails in China, caused by E. bateri. They described and illustrated the oocysts. The interaction between E. uzura infection and aflatoxicosis in Japanese quails was studied by Rao and Sharma (1990). According to them, the combination of E. uzura infection and aflatoxicosis in Japanese quails caused significant weight loss and increased oocyst production and reproductive potential. The prevalence of coccidial species affecting the Japanese quails in India based on single oocyst isolation studies was reported by Rao and Sharma (1992).

2.1.6.2. Histomoniasis:

Bigland et al. (1965) found that quails were refractory to experimental infection with Histomonas meleagridis. Dhillon et al. (1970) reported an outbreak of histomonad infection in a flock of bobwhite quails which caused 95 per cent mortality. They reproduced the disease in young bobwhite quails. Ellis (1967) reported that quails may not be significant in the spread of Heterakis and Histomonas, whereas Kellong (1970) reported that quails were vectors of H. meleagridis. Zeakes et al. (1981) reported that the susceptibility of bobwhite quails to Histomonas meleagridis increased after exposure to sevin insecticide.

2.1.6.3. Cryptosporidiosis

Cryptosporidial infection in a flock of four-week-old quails was diagnosed by Tham et al. (1982). Protozoan parasites attached to the affected epithelium were observed by electron microscopy as Cryptosporidium Spp. A fatal cryptosporidiosis was reported by Hoerr et al. (1986). An acute enteric disease of young bobwhite quails was studied by Ritter et al. (1986) in which Cryptosporidium Spp. and reovirus were identified in affected quails.

2.1.7. Parasitic diseases

Enigk and Dey H.A (1970) reported that quails were susceptible to mature eggs of the nematode, Syngamus trachea.

Reed et al. (1981) reported a severe non suppurative meningo-encephalitis with multifocal areas of malacia in a flock of bobwhite quails with neurologic disorders. The histologic changes, which were confined to the brain, were associated with the presence of nematode larvae of the genus Bayliscascaris.

Githkopoulos et al. (1983) were able to find Capillaria contorta and large numbers of eggs in the thickened and inflammed crop and oesophageal wall of quails.

2.1.8. Toxic conditions

2.1.8.1. Aflatoxicosis

Sawhney et al. (1973) observed lesions in the liver by feeding aflatoxin to 72-day-old laying quails for six weeks. The interaction of graded levels of B_1 aflatoxin and Floxaid was studied by Damron et al. (1977). They reported improvement in body weight in quails dosed with aflatoxin. Chang and Eisen (1979) studied the nature of resistance to aflatoxin in Japanese quails and suggested that there was greater potential for selecting birds resistant to aflatoxin. However, resistance to aflatoxin did not confer resistance to ochratoxin. They stated that the genetic approach to studying the nature of resistance to aflatoxin was feasible. Marks and Wyatt (1979) studied the genetic resistance to aflatoxin in Japanese quail. The influence of aflatoxin on growth and mortality of diverse lines of quails was studied by Marks and Wyatt (1980). It was found that feeding 5 or 10 ug AF/g diet from four to seven weeks of age

resulted in significant decrease in body weight. Blankford et al. (1982) studied aflatoxicosis in Juvenile Japanese quails. They reported that the increase in testicular cholesterol was due to interference with androgen synthesis. Chang C-F and Hamilton (1979) observed 50 per cent mortality in quails fed 20 ug/g aflatoxin. According to them, the acute oral LD_{50} was 19.5±4.8 mg AF/kg. Tissue distribution and hepatic ultrastructural effects of AF B_1 in Japanese quail were studied by Shanks (1986). The in vitro metabolism of AF B_1 catalyzed by heptatic microsome isolated from control or 3-methyl cholanthrene-stimulated quails was studied by Neal et al. (1986).

2.1.8.2. Ochratoxicosis

Burns and Dwivedi (1984) were able to induce teratogenic effects in Japanese quails by feeding ochratoxin A (OA). Maxwell and Burns (1987) examined ultrastructural changes in the liver and kidney of 11 week old quails, fed from the day of hatch with a diet containing OA. They stated that OA was more hepatotoxic and nephrotoxic in quail than in broilers. Fuchs et al. (1988) studied Carbon-14-OA distribution in Japanese quail using whole body autoradiography.

2.1.8.3. Insecticide toxicity

McFarland (1969) studied the physiological and endocrinological effect of insecticide kepone in Japanese quails. The most characteristic abnormality observed was enlarged liver. An EM study of kepone toxicity with regard to fatty change and hepatic cell excretion in quail liver was reported by Atwal (1973).

Smith et al. (1969) reported 50 per cent mortality in adult quails on consumption of 400 ppm DDT for 30 days. Mcblain et al. (1974) observed limited accumulation of DDT in eggs, fat and liver of Japanese quails.

Mumtaz et al. (1986) studied the comparative metabolism and fate of fenvalerate in Japanese quails.

2.1.8.4. Cadmium toxicity

Richardson and Spivey Fox (1974 a) were able to produce intestinal lesions in quails, which were limited to the proximal small intestine, by feeding cadmium. According to them, these lesions were comparable to similar lesions occurring in human jejunum in various malabsorption

syndromes. Richardson et al. (1974 b) observed testicular hypoplasia, growth retardation, anaemia, hyperplasia of the bone marrow, and enteropathy of the small intestine in quails fed cadmium.

2.1.8.5. Monensin toxicity

Sawant et al. (1990) were able to determine liver damage as measured by changes in the activities of serum enzymes and liver microsomal enzymes in quails fed dietary monensin.

2.1.8.6. Furazolidone toxicity

Arbid et al. (1990) studied the toxicological and biological aspects of furazolidone toxicity in Japanese quails and found that Japanese quails were more sensitive to toxicity of furazolidone than chicken.

2.1.9. Nutritional deficiencies

Chang and McGinnis(1967) in an experimental study of Vitamin D deficiency in quails observed that the requirement of Vitamin D for adult male quails is low or non-existent.

Howell and Thompson (1970) observed lesions in the nervous tissue and bone in vitamin A deficient quails.

Kling and Suares (1980) observed decrease in hatchability and fertility in vitamin E deficient quails.

Morphological effects of magnesium deficiency on liver cells and general aspects of its influence on metabolism in young quails were investigated by Dider et al. (1984).

2.1.10. Miscellaneous disease conditions

2.1.10.1. Glycogen storage disease

Matsui et al. (1983) reported a generalized glycogen storage disease in two Japanese quails.

2.1.10.2. Sertoli cell tumors

Sertoli cell tumours of the testes in Japanese quails were reported by Gorham and Ottinger (1986).

2.2. Diseases affecting the Liver Primarily or Secondarily

2.1.1. Bacterial diseases

2.2.1.1. Ulcerative enteritis

Peckham (1959) was able to isolate gram positive rods from the affected quail liver which had either small, sharply demarcated, necrotic, yellow foci, or large, poorly delineated, light yellow areas on the borders. During further studies on the causative organism of UE in quail, Peckham (1960) observed mottled liver which often contained one to two mm size grey, necrotic areas, or larger, irregular yellow, necrotic areas. A smear of the necrotic liver lesions revealed gram positive rods with free spores and rods with subterminal spores. In an outbreak of Ulcerative enteritis among quails reported by Harris (1961), six of the twelve specimens had hepatic involvement which consisted of innumerable pin point foci of necrosis scattered widely throughout the parenchyma. Compressed clearly demarcated foci of necrosis scattered widely throughout the parenchyma were observed histologically. Berkhoff et al. (1974 b) observed more or less extensive liver necrosis in experimentally induced UE in quails, whereas Srinivasan et al. (1981) noticed only congestion of the livers in a natural case of UE.

2.2.1.2. Pasteurellosis

Myint and Carter (1988) observed swollen liver which contained multiple small focal areas of heterophilic infiltrations in quails affected with fowl cholera. Glisson et al. (1989), in a study of natural case of fowl cholera in quails, did not find gross lesions in the majority of the birds that had died. An occassional bird had small, faint, multifocal pale areas in the liver. Histologically, there were multifocal areas of necrosis of coagulative to fibrinoid type and multifocal areas of reticuloendothelial hyperplasia. P. multocida was isolated from the affected liver. The lesions in the experimentally infected birds were generally more pronounced than in the field cases.

2.2.1.3. E. coli infections

Silva et al. (1989) described multiple nodules of three to five mm diameter, which were whitish-grey with smooth surface and firm consistency, in liver of quails affected by Coligranulomatosis. Histologically, the chief cells involved in the process were macrophages and epithelioid cells, mononuclear lymphocytes, plasma cells, and scarce heterophils. There were small areas of caseous necrosis,

haemorrhage, and proliferation of fibrous connective-tissue characteristic of a granuloma. Ito (1990) described hepatomegaly, thickening of the liver capsule, and necrosis of the hepatocytes. E. coli was isolated from the liver.

2.2.1.4. Tuberculosis

In an experimental study of susceptibility of Japanese quails to avian tuberculosis, Karlson et al. (1970) observed enlarged liver which almost filled the abdomen. There were innumerable miliary and nodular foci in the liver; these foci were yellowish or greenish, depending on the stasis of bile. Few birds had rupture of the liver. Multinucleated cells at the periphery of the developing tubercles were seen histologically. Langhan's giant cells, as seen in the mammalian tubercles, were not evident.

2.2.1.5. Proteus infections

Sah et al. (1983) observed congestion of the liver in a septicaemic Proteus mirabilis infection in Japanese quails. Sections stained with MacCallum-Goodpasture's stain revealed slender gram negative rods. Myint (1987) also reported congested liver in Proteus mirabilis infection in quails.

Pure cultures of gram negative rods were isolated from the liver.

2.2.1.6. Listeriosis

Nikuradze (1970) reported focal necrosis in the liver of quails infected with Listeria monocytogenes. The organism was isolated from the liver.

2.2.1.7. Infections caused by Salmonella Spp.

Kapoor et al. (1980) observed necrotic foci on the surface of the liver of quails infected with Salmonella bareilly. Awaad et al. (1981) reported congestion and haemorrhage in the liver of quails infected with Salmonella gallinarum. Histologically, focal necrosis, microgranuloma in the liver parenchyma and perivascular lymphocytic infiltrations in the portal areas were observed. Subcapsular infarcts associated with thrombophlebitis were also evident. In a study of outbreak of S. gallinarum infection in Japanese quail, Sarma et al. (1988) reported liver lesions which consisted of congestion, perihepatitis, and necrotic foci. The organisms were successfully isolated from the liver. Mathew and Sulochana (1990) could not find any specific post-mortem lesions in the liver in an outbreak

of fowl typhoid among quails; but they were able to isolate S. gallinarum from the liver.

2.2.1.8. Erysipelas

Hepatic congestion and haemorrhages were reported by Panigrahy and Hall (1977), in a study of outbreak of Erysipelas in Coturnix quails.

2.2.2. Mycoplasmosis

Nascimento and Nascimento (1986) observed mild haemorrhagic lesions in the liver of quails affected by M. gallisepticum.

2.2.3. Viral diseases

2.2.3.1. Marek's disease

Dutton et al. (1973) observed enlarged livers which protruded beyond the caudal and lateral limits of the thorax of quails affected by Marek's disease. The liver exhibited white nodules or diffuse enlargement with a yellowish 'nut meg' pattern. They also reported that the liver appeared grossly normal in the very mild cases. Histologically,

lymphoid cells with occassional plasma cells were predominant in neoplastic accumulations. The lymphoid cells were characterized by a round nucleus which was surrounded by a moderate rim of basophilic cytoplasm. Mitotic figures were frequent. Perivascular infiltrations were more commonly observed in the grossly normal or slightly enlarged liver. Nodular infiltrations were not perivascular in distribution.

Fugimoto et al. (1975) on the other hand did not find grossly visible lesions in any of the visceral organs or tissues of many quails affected with MD virus. In some quails, however, the livers were slightly enlarged and sometimes had scattered or diffuse foci on their surface and in their parenchyma, but no solid, large tumours. Histologically, the livers had small, diffuse foci or extensive invading masses of lymphoreticular cells. The neoplastic masses consisted of a mixture of small and medium lymphoid cells, lymphoblastic cells, and reticulum cells.

Khare et al. (1975) observed enlarged 'nut meg' liver in quails surviving of MD. They observed a definite relation between sex and liver lesions. Almost all female survivors had liver lesions, yet these liver lesions were infrequently observed in males.

Pradhan et al. (1985) in a study of natural case of MD in Japanese quails, observed involvement of the liver in 93 per cent cases. The liver had a granular appearance. Microscopically, pleomorphic lymphoid cells in the liver and other organs were found. According to them, spleen, proventriculus, liver and duodenum were the target organs for MDV in quails.

Nair et al. (1986) observed moderately and diffusely enlarged liver which almost filled the abdominal cavity. Livers were soft and contained many circumscribed greyish-white nodules varying in size from 1-2 cm both on the surface as well as in the parenchyma. Histologically, in the liver, in many areas, large sheets of hepatic cells were seen replaced by lymphocytes. The impression smear from liver tissue revealed pleomorphic lymphoid cells by Methyl green-Pyronin stain.

2.2.3.2. Leucosis

Wight (1963) found enlarged liver with numerous small yellowish white foci visible on the surfaces and in the substances. These foci resembled small areas of bacteraemic necrosis. Histologically, the liver had small diffuse foci

or extensive invading masses of a uniform type of cells resembling the lymphoblasts of the domestic fowl. These cells had a large nucleus, rather prominent nucleolus, and relatively large amount of basophilic cytoplasm; mitotic figures were numerous. Those hepatic cells which had survived destruction by the proliferating neoplastic masses, generally appeared to be morphologically normal. However, in one case, the liver had several small abscesses consisting of a necrotic core surrounded by giant cells, external to these were a thick zone of mature lymphocytes and small numbers of heterophils. These abscesses on the liver were considered to be probably due to a coincidental bacteraemia.

Moscovici and Macintyre (1966), in a study of the effect of avian myeloblastosis virus in Japanese quails, observed extramedullary haematopoiesis in the liver. This consisted of diffusely infiltrated lymphoma cells which permeated the vessel lymphatics and protruded into the lumen of the blood vessels as a microtumour.

Nishimura et al. (1970) observed small, focal, greyish-tan infiltrations in the liver of quails affected by Epizootic reticulum cell sarcoma. They also observed

virion-like, double walled, spherical bodies in the nuceoplasm of hepatic cells under electron microscope.

Naturally occurring lymphoproliferative disease in Japanese quails was reported by Schat and Gonzalez (1976). They observed enlarged livers which had white to yellowish-white foci varying from few to confluent. Microscopically, the affected organ had lymphoproliferative infiltrations some times completely replacing the normal liver structure. In some quails, large dark-staining cells were present, whereas in other birds medium-sized cells with open vesicular nuclei were prevalent. In few cases, a fine connective tissue stroma was present in the foci. Mitotic cells were frequent.

Ratnamohan et al. (1979) observed enlarged liver which contained multiple red and white spots in quails inoculated with an Australian strain of reticuloendotheliosis virus.

2.2.3.3. Quail bronchitis

Olson (1950) observed small necrotic foci in the liver of chick embryos inoculated with a virus isolated from bobwhite quails with respiratory symptoms and high mortality.

Chew-Lim (1980) noticed evidence of bile stasis or necrotic foci in the liver of quails with bronchitis.

Jack and Reed (1988) observed large intranuclear inclusions, typical of adeno virus infection in the liver which also had focal necrotizing hepatitis.

Jack and Reed (1990) in a study of experimentally induced quail bronchitis noticed multiple disseminated pale foci in the liver with necrotizing hepatitis. In some birds foci were most prominent, on the margins of the affected liver lobes, though most foci were randomly distributed. The most severely affected liver was diffusely pale, enlarged and friable. Necrotizing hepatitis was present in 60-70 per cent of the birds. Histologically the affected liver showed multiple necrotic foci infiltrated by lymphocytes and heterophils. Some of the livers contained discrete foci of coagulative necrosis with minimal leucocytic infiltration whereas, many other affected livers had extensive coalescing zones of necrotizing hepatitis. Hepatocytes adjacent to the necrotic zones frequently contained large basophilic intranuclear inclusions.

2.2.3.4. Inclusion body hepatitis

Jack et al. (1987) noticed multiple disseminated one to two mm size pale foci in the liver of farm-reared bobwhite quails affected with inclusion body hepatitis. Histologically, these areas represented foci of hepatocellular necrosis.

2.2.4 Fungal diseases:

2.2.4.1. Aspergillosis

Chaudhary et al. (1988,a) studied experimental aspergillosis in Japanese quails. They noticed congestion of the liver in affected quails but did not find any characteristic histopathological alterations at any stage of the experiment. In the initial stages, they noticed congestion, focal haemorrhage and fatty change in the hepatic parenchyma but thereafter the sections of the liver were comparable to those of the controls. At the 35th day of infection, multiple foci of necrosis with mononuclear cell infiltration were seen in the liver of one bird, but they could not demonstrate any fungus in these necrotic areas.

2.2.5. Protozoan diseases

2.2.5.1. Histomoniasis

Dhillon et al. (1980) in a study of an outbreak of histomonad infection in a flock of bobwhite quails observed pathologic alterations which were most prominent in the liver. The lesion consisted of disseminated white foci of necrosis. There were circular to irregular raised subcapsular areas and roughly spherical foci within the liver parenchyma. Microscopically, liver revealed multifocal necrosis and associated infiltration with the heterophils, lymphocytes, and monocytes. Variable numbers of protozoan organisms were present in areas of hepatic necrosis. These were per-iodic acid Schiff-positive and present singly or in clusters.

Zeakes et al. (1981) reported that the susceptibility of bobwhite quails to Histomonas meleagridis increased after exposure to sevin insecticide. They observed discoloured liver when sevin was given @ 10 ug/day for 5 days. This indicated only slight damage.

2.2.6. Toxic conditions

2.2.6.1. Aflatoxicosis

Sawhney et al. (1973) in a study of experimental aflatoxicosis in laying Japanese quails observed changes only in the liver. The liver varied from normal size to greatly enlarged, two to three times the normal size. The colour varied from brown to tan. The only significant pathological change besides fatty change was the hyperplasia of the bile duct.

Blankford et al. (1981) reported that the relative weights of the liver of aflatoxin fed quails were not significantly different from that of the controls.

Chang and Hamilton (1982) observed enlargement of the liver of quails fed aflatoxin. The enlargement was not associated with a significant increase in the liver lipid content. According to them, aflatoxicosis appeared almost like a different disease in quail and chicken.

2.2.6.2. Ochratoxicosis

Maxwell and Burns (1987) while describing the ultrastructural changes in ochratoxicosis in quails,

observed that the changes in the mitochondria and glycogen deposition in the liver were the main pathological changes. They noticed enlarged mitochondria which contained 'crystals'. These were regarded as products of degenerative changes.

2.2.6.3. Monensin toxicity

Sawant et al. (1990) in an experiment to study the liver damage in quails caused by dietary monensin toxicity, observed changes in the activities of serum enzymes and liver microsomal enzymes. It was suggested that monensin at lower level induced hepatic microsomal drug-metabolizing enzymes, whereas at higher levels (330 ppm) it inhibited the microsomal enzymes and produced liver damage. The authors attributed the hepatotoxicity produced by monensin fed at levels higher than 330 ppm to monensin-induced disruption of cell membrane and consequent cell death.

2.2.6.4. DDMU toxicity

Tarrant et al. (1989) observed changes in hepatic morphology in quails fed 1, 1-Di(p. chlorophenyl)-2-chloroethylene (DDMU), a metabolite of DDT, which included cytoplasmic and nuclear degeneration in the hepatocytes,

followed by severe lipid accumulation and hepatocellular hypertrophy.

2.2.6.5. Furazolidone toxicity

Arbid et al. (1990) observed hepatotoxicity in Japanese quails fed toxic doses of furazolidone, which was evidenced by an increase in serum aspartate aminotransferase, alanine aminotransferase and alkaline phosphatase and a decrease in serum total protein, in addition to the degenerative changes in the hepatocytes.

2.2.7. Deficiency disease conditions

2.2.7.1. Magnesium deficiency

Dider et al. (1984) studied the morphological effects of magnesium deficiency on liver cells. Magnesium deficiency was characterized by depressed growth, high mortality rate, decrease in the number of mitochondria per cell section of the liver and an increase in the average area of a mitochondrion.

Materials and Methods

3. MATERIALS AND METHODS

3.1. Survey studies

The Japanese quails brought for autopsy to the Centre of Excellence in Pathology from the quail farm attached to the college and also from the suburban farms during the period 1990 to 1991 were examined in detail for the presence of gross lesions in the liver. To study the hepatic disorders a total of 575 livers with gross lesions were examined.

The post-mortem reports maintained at the Centre of Excellence in Pathology during the period 1986 to 1991 were also made use of for the study. In a retrospective study, from the autopsy records, different disorders affecting the liver of quails were identified. The hepatic disorders encountered were classified under different heads according to the age of the quails.

3.2. Collection of materials

Bacteriological studies

From the fresh carcasses, materials were collected from the liver for bacteriological examination taking care to

avoid extraneous contamination. Representative samples of liver tissue were collected in sterile Petri-dishes for bacteriological examination from the livers with well defined lesions, before removing the other organs. Bacteriological cultures were made in Tryptose soya agar, MacConkey agar and blood agar and incubated at 37°C for 24 hours. The genera of the isolates were identified as described (Cowan, 1974). For systematic typing and identification, the cultures were sent to the National Salmonella and Escherichia Centre, CRI, Kasuali.

Histopathological studies

From the fresh carcasses, after studying the gross lesions, representative samples of liver were collected in 10 per cent neutral buffered formalin for histopathological examination. The tissues were processed by routine methods, embedded in paraffin and sections cut at 3-5 u thickness were stained with hematoxylin and eosin (Luna, 1968). Wherever necessary, tissues were also stained with special stains which included staining for fat (Disbrey and Rack, 1970), reticulum staining (Luna, 1968), staining for fungus (Luna, 1968) and Ziehl-Neelsen method of staining for acid-fast bacteria (Luna, 1968).

3.3. Experimental studies

Based on the results of the autopsy findings the following experimental studies were also undertaken.

A) Hepatitis caused by experimental infection with Salmonella typhimurium

S. typhimurium which was isolated from three quails died of hepatitis was used for evaluating the pathogenicity of the strain and to study the pathological features of hepatic lesions caused by the organism.

Experimental design

Thirty, three-month-old healthy male quails were randomly divided into five groups of six quails each and maintained in cages during the experimental investigation. Before the experimental infection, all the birds were tested and found negative for Salmonella pullorum and Salmonella gallinarum infections by rapid plate test using Pullorum colored antigen obtained from the Institute of Animal Health and Veterinary Biologicals, Bangalore.

The inoculam used was a saline suspension of 18 h old Tryptose Soya Agar culture containing 10^8 organisms per ml as determined by McFarland Nephelometer standards (Sonnenwirth, 1990). The bacteria were injected into the crop of the quails using a sterile tuberculin syringe. Group A was administered 0.3 ml of the suspension per bird; Group B was administered 0.6 ml of the suspension per bird; Group C was administered 0.9 ml of the suspension per bird; Group D was given cortisone acetate @ 6 mg/bird, intramuscularly for three days, to induce immunosuppression and on the fourth day each quail was given 0.3 ml of the saline suspension containing the organisms and Group E was kept as control. The control group was housed separately from the treatment groups.

The quails were observed for fifteen days. The birds which died during the course of the experiment and the remaining birds which were sacrificed on the 15th day were subjected to detailed post-mortem examination and the hepatic lesions were recorded. Representative samples of liver were collected for histopathological and bacteriological examination. The results were recorded. The weight of the quails of the groups A, B, C and control was recorded on alternate days. The data were analysed using CRD (Snedecor and Cochran, 1967).

B. Hepatic toxicity of dietary monensin in quails

Twenty four, four-month-old Japanese quails obtained from the University quail farm were randomly divided into two groups. Group I consisted of eighteen birds and Group II six birds. The birds were maintained in cages. Coban-100 (Monensin Sodium) obtained from Ventri Chemicals, Pune was used for this study. This was given in the feed to one group consisting of eighteen birds (Group I) at the rate of 250 ppm daily for three months. The other group (Group II) served as control. Monensin was well mixed with the feed and then given to the quails. Feed free from monensin was given to the control birds. Six birds and two birds from the treatment and control group respectively were sacrificed at monthly intervals and the liver was examined for the presence of gross and microscopic lesions. The birds which died naturally during the course of the experiment were autopsied and the liver lesions were recorded. Representative samples of liver tissue were fixed in 10 per cent neutral buffered formalin for histopathological studies. Paraffin sections were prepared as described earlier and stained with hematoxylin and eosin. Weight of the control and experimental birds was recorded at fortnightly intervals and data were analysed using Student's 't' test (Snedecor and Cochran, 1967).

Results

4. RESULTS

During the course of this investigation, 575 livers from the quails of different age groups brought for autopsy were examined at autopsy for various gross and histologic lesions. The following investigations were undertaken.

1. Liver from 575 quails out of 10,628 quails autopsied during the period 1990-91 at the Centre of Excellence in Pathology.

2. Data pertaining to the incidence of hepatic disorders in Japanese quails were collected from the post-mortem records maintained at the Centre of Excellence in Pathology and analysed.

3. Experimental studies which included the pathogenesis and pathology of hepatitis caused by experimental S. typhimurium infection and hepatic toxicity induced by dietary monensin.

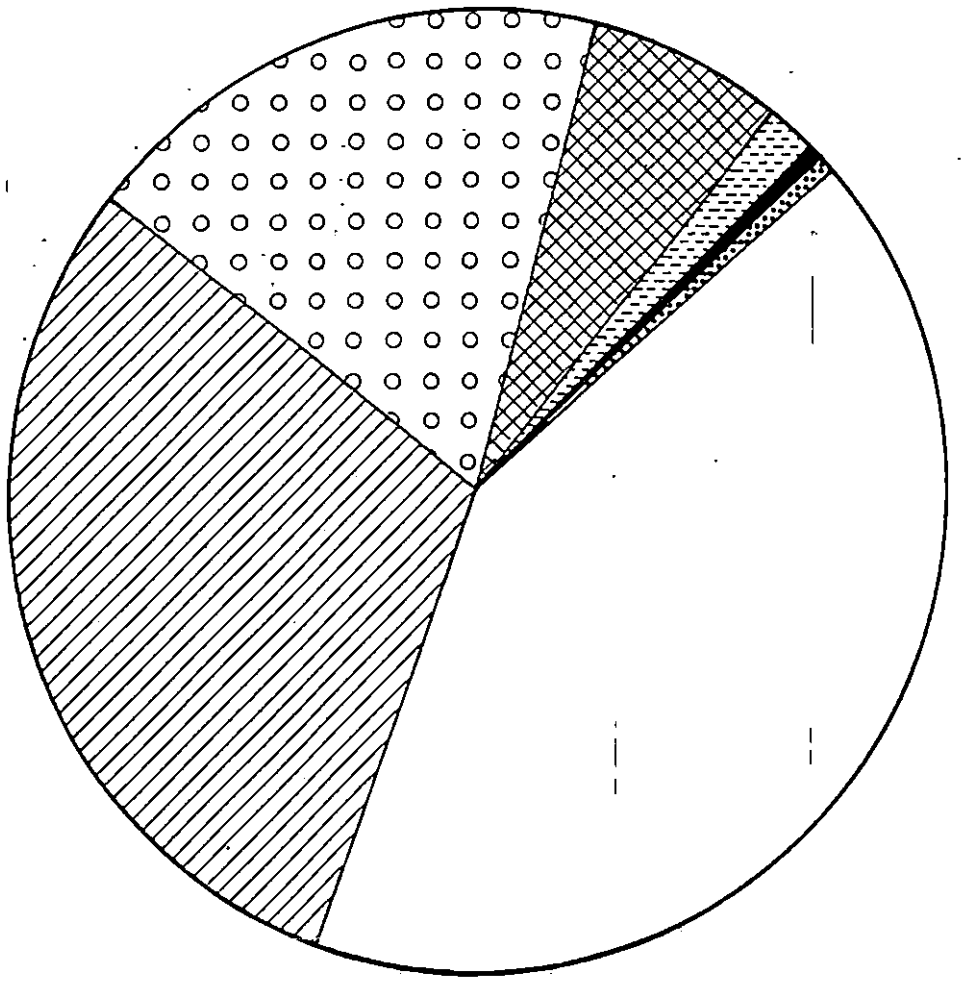
The criteria for collecting tissues for histopathological examination were the presence of well defined lesions such as discolouration of the liver, diffuse or focal enlargement, presence of greyish-white raised or depressed foci or patches, presence of brownish-black spots or mottling of the liver.

Analysis of the data collected from the autopsy records during the period 1986 to 1991 revealed hepatic involvement in 34.09 per cent cases (Table I). Age-wise incidence of hepatic disorders collected from the post-mortem records maintained at the Centre of Excellence in Pathology is shown in table II, Fig. 2. Year-wise incidence of hepatic disorders collected from the autopsy records is presented in table III.

The percentage incidence of various hepatic lesions encountered during the post-mortem examination of quails brought to the Centre of Excellence in Pathology is shown in table IV. The various hepatic disorders encountered were congestion (42.08 per cent), hepatic steatosis (30.26 per cent), necrosis (6.78 per cent), Marek's disease (18.43 per cent), hepatitis (1.73 per cent), abscesses (0.34 per cent) and subcapsular haemorrhage (0.34 per cent). The overall incidence of histopathological lesions is presented in table V. The incidence of various disorders has been diagrammatically represented in Fig. 1.

The severity of the histopathological lesions observed in the different age groups in randomly selected histologic sections is shown in table VI.

Fig.1 Percentage of hepatic lesions



Congestion (42.08%)

Fatty change (30.08%)

MD (18.43%)

Necrosis (6.7%)

Hepatitis (1.73%)

Subcapsular haemorrhage (0.34%)

Abscess (0.34%)

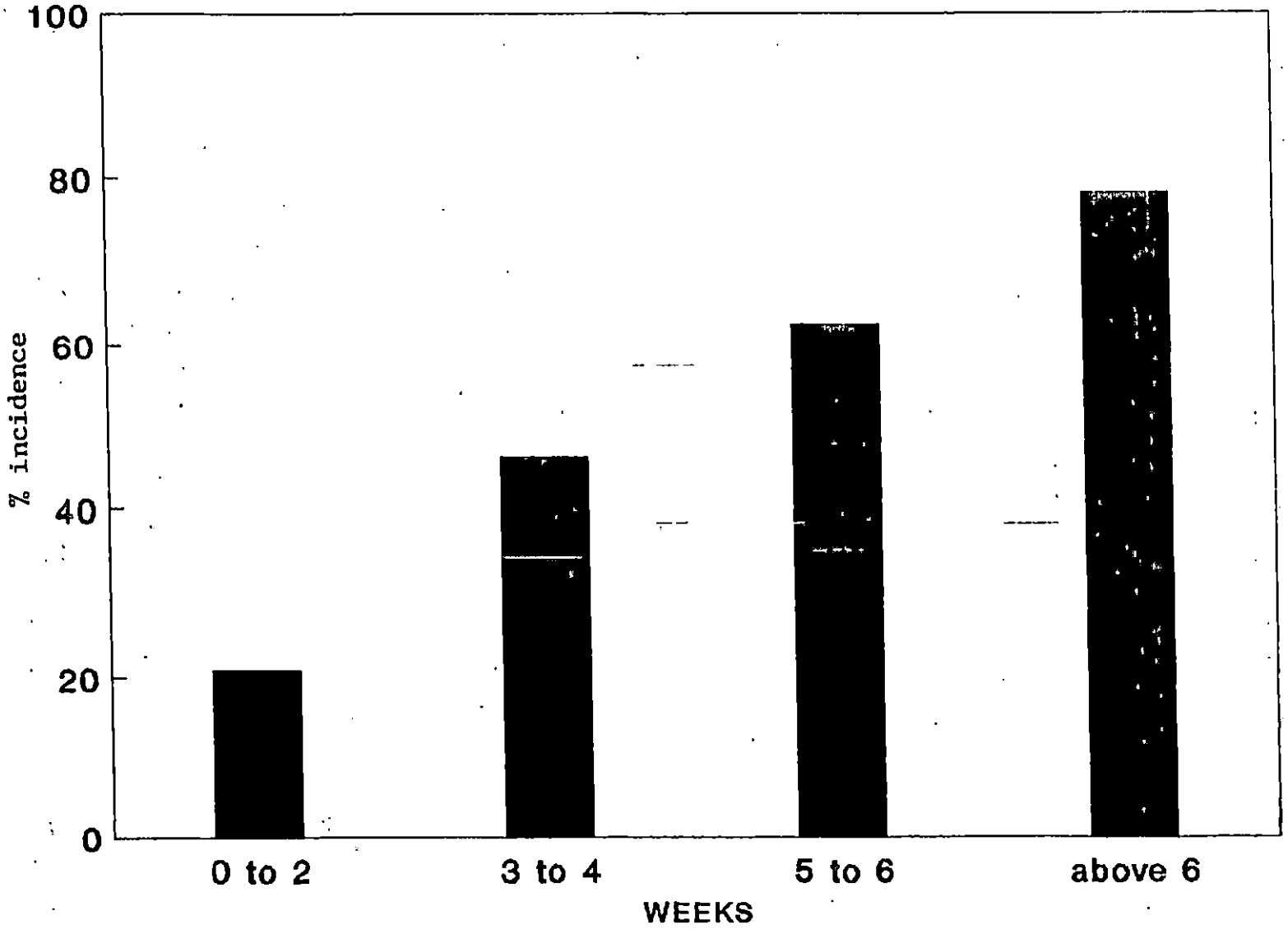


Fig.2. Age-wise incidence of hepatic lesions

4.1. Autopsy studies

4.1.1. Hepatic abscess

4.1.1.1. Incidence

Hepatic abscesses were encountered in two adult quails out of 575 livers examined. The incidence was 0.34 per cent.

4.1.1.2. Gross Pathology

Gross lesions were seen in only one case out of the two cases of hepatic abscesses observed. Multiple abscesses of varying size were seen on both the lobes (Fig. 3). Most of the abscesses were superficially located and few were embedded deep in the parenchyma of the liver. The abscesses had well defined encapsulated border. They were greyish-white in colour. On incising the abscesses, yellowish inspissated mass was visible. The larger abscesses measured two cm in diameter.

The other quail which had hepatic abscess histologically showed moderate congestion grossly.

4.1.1.3. Histopathology

There was extensive central area of caseation necrosis in the liver surrounded by a thin band of fibro-vascular connective tissue. The central necrotic area consisted of necrotic tissue, degenerated and necrotic cells and infiltration of mononuclear cells. Besides this, in some abscesses there were also clumps of bacteria in the central zone. The dense bands of fibro collagenous tissue was seen infiltrated with heterophils, macrophages, lymphocytes and few giant cells. The surrounding hepatic tissue was compressed (Fig. 4). In focal areas, bile duct hyperplasia was evident. This was characterized by proliferation of biliary epithelium along with the formation of many small bile ducts.

The microscopic changes in the liver of the other quail which did not have gross hepatic abscess consisted of islands of necrotic areas containing clumps of bacteria. Surrounding the focal necrotic zone, there was dense infiltration of heterophils. There were many microabscesses embedded within the parenchyma.

4.1.2.Hepatic congestion

4.1.2.1. Incidence

Congestion as the predominant lesion was observed in 42.08 per cent of cases. The age-wise incidence of congestion is shown in table IV. It was higher in quails aged 0-2 weeks (65.58 per cent) and lowest in adults (19.40 per cent). Marek's disease was associated with congestion in 89 per cent cases. Subcapsular haemorrhages were also associated with congestion. Chronic venous congestion was observed in two cases and these were associated with Marek's disease. Fatty change was also associated with mild or moderate congestion in most instances. In quail chicks, congestion was associated with fatty change in 25.58 per cent cases and with haemorrhage in 6.9 per cent cases. In quails aged 3-4 weeks, congestion was associated with fatty change in 33.75 per cent cases whereas in quails aged 5-6 weeks, it was 39.5 per cent and in quails aged above 6 weeks, it was 32.4 per cent.

4.1.2.2. Gross pathology

In 95 per cent of cases congestion was associated with other lesions and in 68 per cent cases congestion was the

only gross change observed. When congestion was the only gross lesion and when it was mild or moderate, there was no enlargement of the liver, but when the liver was severely congested, it had slight to moderate enlargement. When there was hepatic enlargement the borders were rounded. In quail chicks, in most of the cases, congestion was confined either to the right or the left lobe whereas in adults, both the lobes were equally involved. On incision large quantity of blood tinged fluid oozed out.

4.1.2.3.Histopathology

In mild and moderate cases there was slight engorgement of few sinusoids with blood. The dilated sinusoids contained erythrocytes which in some cases packed the sinusoids. The blood filled spaces were of different sizes and were lined by endothelial cells and surrounded by a thin compressed band of hepatic cells. In some of the specimens the erythrocytes had undergone lysis to form a homogenous eosinophilic mass with deposition of hemosiderin pigments. The blood filled spaces which were many were seen separated by cords of compressed hepatic cells. In severe cases the areas of congestion were found to be large spaces filled with erythrocytes (Fig. 5). Most of the vessels were

severely dilated and contained erythrocytes some of which had undergone lysis. Sinusoids especially around central veins were distended with blood. Chronic venous congestion was observed in two cases. This was associated with Marek's disease (Fig. 6). In these cases the wall of the vessel was very much thickened due to the fibrous tissue formation in the perivascular tissue. The engorged vessels were surrounded by compressed hepatocytes and neoplastic lymphocytes.

4.1.3. Subcapsular haemorrhage

4.1.3.1. Incidence

Haemorrhage under the capsule was seen in two quails out of 575 livers examined. The incidence of subcapsular haemorrhage was 0.34 per cent. These were seen in adult quails.

4.1.3.2. Gross pathology

This lesion was encountered in one quail aged 5-6 weeks and the other quail aged above six weeks. In one case the liver showed dark-brown raised areas of 0.5 to 1 cm diameter on one lobe. Besides this, many small dark-brown raised

foci were seen on both the lobes (Fig. 7). In the other case, five to six dark-brown raised foci were seen on both the lobes. On incision a clot of blood was seen under the capsule.

4.1.3.3. Histopathology

Areas of haemorrhage were noticed just below the capsule. The capsule was elevated. In certain areas the haemorrhages were seen to extend deep into the parenchyma separating the hepatic cells. Islands of hepatic cells were seen encircled by erythrocytes. The hepatocytes around the heamorrhagic zone showed fatty change and focal necrosis. There was moderate to severe congestion. In one quail the subcapsular haemorrhage was associated with Marek's disease. Here there was diffuse proliferation of pleomorphic lymphocytes. Moderate to severe congestion was noticed in both the cases.

4.1.4. Hepatic necrosis

4.1.4.1. Incidence

The incidence of hepatic necrosis is shown in tables IV and V. The incidence of necrosis as the predominant hepatic lesion was higher in adults than in chicks. Hepatic

necrosis as the only lesion was not observed in quails aged 3-4 weeks and 5-6 weeks. In all age groups, in most cases, necrosis was associated with congestion. In adult birds diffuse infiltration of neoplastic cells in Marek's disease was associated with focal necrosis. Focal necrosis was also associated with diffuse or focal fatty change in many cases.

4.1.4.2. Gross pathology

Affected liver revealed the presence of pale or yellowsih areas of varying sizes. But in most of the cases, when the necrosis was associated with severe acute congestion and petechial haemorrhages, gross lesions consisted of moderate enlargement of the liver with reddish spots all over and few greyish-white depressed foci. Moderate congestion and five to six greyish-white foci of necrosis were seen in three cases from which Salmonella typhimurium was isolated. In one case there was diffuse greyish white patches of varying size on the surface of the liver which did not show any change other than necrosis. The livers did not show any considerable enlargement when the necrosis was not associated with any other lesions other than Marek's disease or severe acute congestion.

In quail chicks, the necrotic areas were confined either to the right or the left lobe, where as in the case of adults, it was confined to both the lobes. On dissection, necrotic changes were found to be extending deep into the parenchyma in 60 per cent of the cases whereas in others it was confined to a small area under the capsule. Massive type of necrosis was not observed.

4.1.4.3. Histopathology

Hepatic necrosis observed was of coagulative type. Both focal, multifocal and diffuse necrosis were noticed. The affected areas were homogenous and eosinophilic with hyperchromatic pyknotic nuclei. Four of the specimens showed diffuse type of necrosis. In many of the hepatic cells the nucleus had completely disintegrated and disappeared retaining only the outline of the cell. Diffuse necrosis was always associated with severe acute congestion. When necrosis was the only lesion, it was of focal type. The areas of necrosis were found to be extending to the periphery in certain specimens. In few cases focal areas of coagulative necrosis were associated with diffuse fatty change.

4.1.5. Hepatic steatosis

4.1.5.1. Incidence

Fatty liver as the only predominant hepatic lesion was observed in 30.26 per cent cases. Hepatic steatosis was the common lesion observed other than congestion. It was highest in quails aged five to six weeks (39.53 per cent) and lowest in quail chicks (33.40 per cent).

4.1.5.2. Gross pathology

Most of the affected livers were moderately and diffusely enlarged and had rounded borders. The affected livers were pale or yellow when fatty change was not associated with congestion, otherwise it was dark-red in colour. When fatty change was severe and diffuse, the liver was diffusely pale in colour (Fig. 8). Mottling of the liver was always associated with fatty change and congestion.

In general, the liver was friable and easy to cut. Cut surface bulged out and was oily in appearance.

4.1.5.3. Histopathology

The hepatocytes contained small to medium sized globules of fat in the cytoplasm. This displaced the nucleus towards the periphery giving a signet ring appearance to the hepatocyte. Diffuse fatty change was observed in 60 cases (Fig. 9) and focal fatty change was observed in 114 cases In 12 cases it was associated with hepatitis. Congestion was the common lesion associated with fatty change (Fig. 5). Infiltration with pleomorphic cells in MD was also associated with fatty change in many cases (Fig. 10). In few instances fatty change was associated with coagulative necrosis (Fig. 11). In one case few small globules fused together to form a fatty cyst (Fig.12).

The hepatic steatosis was confirmed by staining the cryostat sections with Sudan III and IV. The fat globules were stained bright pink (Fig. 13).

Reticulum staining revealed collapsed and fragmented reticulum fibres (Fig. 14).

4.1.6. Hepatitis

4.1.6.1. Incidence

The incidence of hepatitis was 1.73 per cent. It was highest in the quails aged above 6 weeks (3.37 per cent). Hepatitis was not observed in quails aged 3-4 weeks. In quails aged 0-2 weeks, the incidence was 0.46 per cent. The incidence of hepatitis is shown in table IV and V. The overall incidence of hepatitis was low.

4.1.6.2. Gross pathology

Grossly the liver was severely congested. On dissection, blood oozed out. In some cases there was mottling. In two cases petichiae were seen.

4.1.6.3. Histopathology

In two cases hepatitis observed was fibrinous peri hepatitis. Extensive fibrin deposition in the subcapsular region, extending into the parenchyma was evident (Fig. 15). There was moderate congestion. Focal areas of necrosis were evident. In other cases there was focal infiltration of heterophils. In 12 cases this was associated with moderate fatty change. In one case there was multifocal haemorrhage.

4.1.7. Marek's disease

4.1.7.1. Incidence

The incidence of hepatic lesions in MD in quails is shown in table IV and V. The incidence was highest in quails aged above six weeks (41.35 per cent). The incidence of Marek's disease in the age group of five to six weeks was 18.60 per cent.

4.1.7.2. Gross pathology

The hepatic lesions were characterized by diffuse enlargement which was moderate to severe. In three cases, the liver was three times the normal size (Fig. 16). The hepatic enlargement was associated with or without splenomegaly. There was moderate to severe congestion of the liver. In few cases there was mottling of the liver which was diffusely enlarged with or without the presence of greyish-white foci (Fig. 17). Nodular lesions were not noticed. But in many cases the diffuse enlargement was associated with the presence of small greyish-white foci which appeared like necrotic foci. These were distributed evenly throughout the surface of the parenchyma and within the substance of the liver tissue (Fig. 18). In one case the liver was severely enlarged, pale and friable and it had

ruptured. In the other case, the liver was moderately enlarged and contained numerous small greyish-white foci and five to six foci of subcapsular haemorrhages. The haemorrhagic foci were uneven in size.

4.1.7.3.Histopathology

In most of the cases the capillaries were dilated (Fig.19). The central veins were prominent. Histologic lesions consisted of both diffuse and focal proliferation of pelomorphic neoplastic lymphocytes. Focal proliferations were mostly associated with the presence of greyish-white foci, which were evident grossly. In the case of diffusely proliferating type, most of the hepatocytes were replaced by pleomorphic neoplastic lymphocytes. Few lymphocytes in mitosis were also seen. Regenerating hepatocytes were evident in many cases (Fig. 21). The neoplastic cells were uniformly proliferative in nature. The cellular composition consisted of diffusely or focally proliferating small to medium sized lymphocytes and lymphoblasts. The cell types of the tumors were same in all cases, eventhough, the gross pattern of involvement varied.

In three cases, the hepatic lesions were associated with chronic venous congestion. Here the proliferating

cells were found within the congested vessel and also outside of it. The vessels were severely dilated. Proliferating cells outside the vessel were mostly perivascular in distribution (Fig. 6).

In few cases the proliferating lesions were associated with focal areas of coagulative necrosis. The necrotic areas were surrounded by proliferating neoplastic cells. In some cases, necrotic areas contained erythrocytes.

In many cases the neoplastic lesions were associated with fatty change (Fig. 12). This was mostly seen in focally proliferating type of lesions. The fatty change was moderate to severe and multifocal or diffuse. The hepatocytes were containing medium to large sized vacuoles which stained bright pink with Sudan III and IV.

Out of 106 MD cases diagnosed 16 livers did not show either gross enlargement or presence of greyish-white foci. But histologically infiltration of pleomorphic lymphocytes was evident.

4.2. Experimental studies

4.2.1. Hepatitis caused by Salmonella typhimurium

4.2.1.1. Clinical signs

For the experimental studies S. typhimurium isolated from spontaneous cases was used. Within 24 h of the administration of Salmonella organisms, all the quails which received oral infection became slightly dull. The droppings from these quails were more fluid in consistency. Diarrhoea and reduced feed consumption from the 2nd to 8th day were the features of infection in all the infected quails. All of them became dull and progressively lost weight as against the steady weight gain noticed in the control birds. The mean weights of the infected and the control quails are shown in table VII. Loss in weight was more severe in the quails which received the higher dose, whereas clinical symptoms were less pronounced in the group which received the lower dose. Compared to the quails whch did not receive corticosteroid but did receive bacterial suspension, the corticosteroid treated birds became severely depressed within 24 h of commencement of the experiment (Fig. 22). Out of the six corticosteroid treated quails, two birds died within 24 h and remaining four quails died on the 3rd day of

experimental infection with Salmonella. Two quails from the group C died on the second day. One bird from the group B developed torticollis and it died on the 5th day of the experiment. Remaining quails from all the groups were sacrificed on the 15th day of the experiment.

4.2.1.2. Gross pathology

The quails which died within 24 hours of treatment had moderate to severe hepatic congestion and two to three greyish-white depressed necrotic foci. The quails which were sacrificed on the 14-15th day had moderate hepatic congestion and few to many greyish-white necrotic foci scattered throughout the liver. Both the lobes were equally involved. The necrotic spots were numerous in the group which received the higher dose. An exception to this was the corticosteroid treated group. All the birds in this group died within 72 h of infection with Salmonella. The liver from these quails was severely congested and had many discrete pin head sized greyish-white foci of necrosis. Liver from one bird had numerous small pin head sized greyish-white foci scattered evenly throughout the parenchyma in addition to severe congestion (Fig. 23). The spleen and the heart also had moderate to severe congestion. There was catarrhal enteritis.

4.2.1.3. Histopathology

The histologic changes were typical of bacterial hepatitis. There were focal to multifocal areas of coagulative necrosis. In focal areas infiltration by heterophils was evident without necrosis. The liver tissue from group C showed extensive necrosis of the hepatocytes (Fig. 24). Compressed hepatocytes surrounding this was evident. In severely congested cases sinusoids were stuffed with erythrocytes, otherwise there was localized dilation of capillaries. The quails which died within 24 h of commencement of the experiment did not show infiltration by inflammatory cells but did show severely congested capillaries. In eighteen of the twenty-four cases there was moderate to severe fatty change in the liver. Most of the hepatocytes were containing small to medium sized fatty vacuoles. All the corticosteroid treated quails had severe congestion, multifocal areas of necrosis and moderate fatty change. Two of the six birds had infiltration by heterophils in focal areas in addition to the above changes. Here the infiltration was mostly perivascular (Fig. 25).

In the cases which had long survival period, in addition to infiltration of heterophils there was also infiltration with lymphocytes and macrophages. There was

also a linear zone of fibrous tissue proliferation towards the outer zone.

In the spleen there was reticular hyperplasia particularly around the periarteriolar sheath. There were small foci of collections of lymphocytes and macrophages in focal areas. There was moderate to severe congestion.

In the heart there was mild myocardial degeneration. Capillaries were engorged.

Liver from the control birds did not reveal any gross or histologic changes.

4.2.2. Hepatic toxicity of dietary monensin

From the 15th day of commencement of the experiment all the quails which received monensin gained less weight than the birds which were kept as control. The growth curve for the next 45 days of the experiment showed the depressing effect of monensin on the rate of growth of experimental group of quails. The average body weight of the quails which received dietary monensin and control quails is shown in table VIII. No clinical signs other than growth

depression were observed. On the 45th day one bird died and on the 50th day two birds died. Six quails and two quails were sacrificed from the treatment and control group respectively at monthly intervals and subjected to detailed post-mortem examination.

4.2.2.1. Gross pathology

The quails which died on the 45th day of the experiment had hydropericardium and mottling of the liver. The quail which died on the 50th day of the experiment had discoloured liver which was diffusely greenish in colour. All the other quails from the treatment group which were sacrificed had mild congestion and mottling of the liver. Other organs did not reveal any gross changes.

4.2.2.2. Histopathology

In all the quails which received monensin, the liver was affected. There was moderate to severe fatty change. Most of the hepatocytes were containing small to medium sized vacuoles. The hepatocytes contained either many small vacuoles or large sized vacuole. The vacuoles were confirmed as fat on staining the sections with Sudan III & IV. All the vacuoles stained pink with Sudan III & IV. There was no infiltration with inflammatory cells.

Table I. Year-wise and age-wise incidence of hepatic lesions (Autopsy records: 1986 to 1991).

Year	Age group (weeks)			
	0 to 2	3 to 4	5 to 6	Above 6
1986	94/279 (33.6)	24/70 (34.2)	30/52 (57.6)	94/279 (33.6)
1987	94/249 (37.7)	27/51 (52.9)	4/6 (66.6)	108/152 (71)
1988	268/1211 (22.1)	75/215 (34.8)	13/22 (59.0)	276/397 (69.5)
1989	390/2673 (14.5)	179/412 (43.4)	185/316 (58.5)	602/809 (74.4)
1990	449/3099 (14.4)	89/138 (64.4)	30/81 (37)	414/505 (81.9)
1991	1597/6264 (25.4)	592/1253 (47.2)	41/100 (41)	1059/1116 (94.8)

(Figures in the bracket indicate percentage)

Table II. Age-wise incidence of hepatic lesions in Japanese quails (Autopsy records: 1986 to 1991).

	Age group (weeks)			
	0 to 2	3 to 4	5 to 6	Above 6
No. of quails examined	13775	2139	577	3258
Number of quails which had hepatic lesions	2892	986	303	2553
Percentage incidence of hepatic lesions	20.9	46.0	62.5	78.3

Table III. Year-wise incidence of hepatic lesions (Autopsy records : 1986 - 1991)

Age group	1986	1987	1988	1989	1990	1991
No. of quails examined	680	458	1845	4210	3823	8733
Number of quails which had hepatic lesions	242	233	632	1356	982	3289
Percentage incidence of hepatic lesions	35.58	50.87	34.25	32.20	25.68	37.66

Table IV. Age-wise incidence of hepatic lesions (Histopathological lesions: 1990 to 1991)

Type of lesion	Age group (weeks)			
	0 to 2	3 to 4	5 to 6	Above 6
Congestion	141/215 (65.58)	43/80 (53.75)	12/43 (27.9)	46/237 (19.40)
Fatty change	72/215 (33.40)	24/80 (30)	17/43 (39.53)	61/237 (25.73)
Marek's disease	--	--	8/43 (18.60)	98/237 (41.35)
Necrosis	2/215 (0.93)	11/80 (5.23)	4/43 (9.3)	22/237 (9.28)
Subcapsular haemorrhage	--	2/80 (2.5)	--	--
Abscess	--	--	--	2/237 (0.84)
Hepatitis	1/215 (0.46)	--	1/43 (2.32)	8/237 (3.37)

(Figures in the bracket indicate percentage)

Table V. Incidence of hepatic lesions in quails (Histopathological lesions: 1990 to 1991)

Sl. No.	Type of lesion	Total number of quails examined-575	
		Number of livers which had lesions	Percentage
1.	Congestion	242	42.08
2.	Fatty change	174	30.26
3.	Marek's disease	106	18.43
4.	Necrosis	39	6.78
5	Hepatitis	10	1.73
6.	Subcapsular haemorrhage	2	0.34
7.	Abscess	2	0.34

Table VI. Severity of hepatic lesions (Histopathological lesions: 1990 to 1991)

Type of lesion	Age group (weeks)			
	0 to 2	3 to 4	5 to 6	Above 6
Congestion	+++	++	++	++
Fatty change	++	+	++	+++
Marek's disease	-	-	+	+++
Necrosis	+	+	+	++
Subcapsular haemorrhage	-	-	-	-
Abscess	-	-	-	-
Hepatitis	+	-	-	+

+++ Severe

++ Moderate

+ Mild

- Not observed

Table VII. Average body weight (Mean±SE) of quails in experimental Salmonellosis (g)

Group	Days							
	0	2	4	6	8	10	12	14
A	91±0.73^{a}	90±0.58^{a}	88±0.82^{a}	89±1.10^{a}	90±1.50^{ab}	91±1.15^{a}	92±1.15^{b}	95±0.86^{a}
B	89±0.86^{a}	88±0.58^{a}	88±0.93^{a}	85±0.82^{b}	87±1.71^{ac}	90±0.52^{ab}	90±1.26^{a}	91±0.71^{ab}
C	89±0.86^{a}	84±1.15^{b}	83±1.78^{b}	84±0.86^{c}	87±1.21^{bc}	88±0.63^{b}	90±0.71^{a}	91±1.10^{b}
Control	90±0.97^{a}	98±0.93^{c}	104±1.71^{c}	109±2.10^{d}	112±1.39^{d}	115±1.03^{d}	118±0.73^{d}	121±1.53^{d}

Means with common letters do not differ significantly.

Table VIII. Average body weight (Mean±SE) of quails which received dietary monensin (g)

Group	Days						
	0	15	30	45	60	75	90
Monensin treated quails	130±0.60	140±0.85	155±0.78	160±1.05	164±1.15	168±0.77	171±0.59
Control	135±0.89	148±2.62	164±0.73	175±1.37	189±1.41	205±1.15	218±2.31
't' value	2.065	3.8476**	6.3321**	11.6828**	15.0**	23.2805**	19.45**

** Significant at 1% level

Discussion

5. DISCUSSION

There has not been any systematic study undertaken to elucidate the incidence and nature of hepatic disorders in quails in the country. This investigation was undertaken to study the prevalence and nature of hepatic disorders in quails. Analysis of the data on diseases of 19749 quails collected from the post-mortem records maintained at the Centre of Excellence in Pathology for the last six years revealed hepatic involvement in 34.09 per cent cases. This indicated that the hepatic disorders were relatively common in quails. Another significant finding was an increase in hepatic disorders in quails of the advanced age group. The incidence of hepatic disorders in quail chicks was 20.9 per cent while it was 78.3 per cent in adult quails. This is an observation which clarified that hepatic disorders were the common lesions encountered in adult quails.

The disorders encountered in the liver are in fact a reflection of adverse health of the quails. The liver is an organ closely associated with health and productivity of quails and when the liver is damaged it is bound to cause lowered production and growth rate. The high incidence of hepatic disorders encountered in adults in this study would suggest that there is general lowered productivity in quails

in the farm. This points out the need for taking urgent, effective control and prophylactic measures to check the hepatic disorders. The incidence of hepatic disorders was high in adult quails. This may be explained by the fact that the adult quails would have been exposed to the noxious agents like the plant poisons, chemicals and bacteria for a longer period compared to the young quails and the liver injury was a manifestation of cumulative toxicity of these agents. And also in case of Marek's disease, which will not affect young quails, and which affects adult quails of all ages, there is every possibility of adult quails being exposed to the MD virus and suffering liver damage.

The incidence of hepatic lesions was lowest in quails aged 0-2 weeks. The cause of death in this age group was mainly pulmonary congestion and oedema which reflect the improper brooder management and in such situations the liver is less likely to be involved. Therefore, these observations points to the fact that hepatic disorders are generally age dependent. The more mature the quail, there is more likelihood of the liver getting damaged.

In the present study, data of 10628 quails which were subjected to post-mortem examination during the period

1990-91 were analysed. The overall incidence of hepatic disorders was 35.52 per cent. Out of 10628 quails autopsied, a total of 575 livers with well defined gross lesions were examined for histologic lesions. Among the hepatic lesions encountered the percentage incidence of various types of hepatic lesions in the decreasing order of frequency was congestion (42.08 per cent), fatty change (30.08 per cent), Marek's disease (18.43 per cent), hepatic necrosis (6.7 per cent), hepatitis (1.73 per cent), subcapsular haemorrhage (0.34 per cent) and hepatic abscess (0.34 per cent).

Congestion was the most common lesion encountered and this would points out the possibility of a bacterial infection and septicaemia. No systematic bacteriological examination was made to isolate bacterial agents possibly associated with this condition. However, the observation of high incidence of hepatic congestion points out the need for undertaking bacteriological studies to identify the various organisms associated with this condition so that prophylactic and therapeutic measures can be chalked out.

The relatively high incidence of hepatic steatosis associated with congestion also suggests the involvement of

toxic agents. Therefore, this aspect also needs further investigation.

Hepatic abscesses were seen in 0.34 per cent cases. These were seen in adult quails aged more than six weeks. The incidence of hepatic abscesses in this age group was 0.84 per cent.

The scattered nature of distribution of microabscesses and the presence of bacterial colonies in histologic sections suggest that the organisms were distributed through the blood stream. Such small microabscesses were also observed by Wight (1963). They suggested the cause of these abscesses as a coincidental bacteraemia. In the other bird which had abscesses grossly, hyperplasia of the bile duct was also evident which suggests the severity of the hepatic response to the agent that was responsible for the abscess formation and the chronic nature of the abscess. Karlson et al. (1970) observed innumerable miliary hepatic nodules in experimentally induced tuberculosis in quails. However, although the nodules observed in the present study resembled tuberculous nodules, the histologic picture was not suggestive of tuberculosis. Specific staining for acid-fast organisms and fungal organisms did not reveal the

involvement of these agents. Silva et al. (1989) observed multiple nodules in the liver of quail affected by Coligranulomatosis. The histologic lesions were similar to the observations made in the present study. Although bacterial isolation was not attempted from this liver, the possibility of E. coli infection being responsible for the abscess formation cannot be overlooked.

Congestion was the most common lesion encountered in quail chicks. In the case of quail chicks the incidence of hepatic congestion as the predominant finding was higher when compared to the adult quails. In adult quails, in most of the cases, hepatic congestion was associated with other lesions like fatty change and Marek's disease. But in these cases, congestion was the only change observed grossly, and in few cases gross congestion was associated with diffuse enlargement or mottling except in the case of Marek's disease, where greyish-white foci were associated with above gross changes. Awaad et al. (1981) observed only congestion of the liver from which they could isolate Salmonella gallinarum.

The reason for the high incidence of congestion as the only lesion in quail chicks can be two-fold: quail chicks

would have died before the congestion progressed to fatty change or necrosis, or, as discussed previously, the usual cause of death in quail chicks in this farm is pulmonary congestion and oedema and the hepatic congestion may be a part of generalized congestion. In adult quails, the congestion was, in most of the cases, associated with other lesions like fatty change, toxic factors from the feeed and or bacterial infections can be attributed as responsible factors. The earliest response of any organ to injury is congestion. Therefore, it is likely that hepatic congestion may be a part of generalized congestion associated with an acute systemic toxaemia.

Srinivasan et al. (1981) observed only hepatic congestion in a natural case of Ulcerative enteritis. Sah et al. (1983) and Myint (1987) observed congested liver of quails in Proteus mirabilis infection. But in the present investigation efforts were not made to isolate bacterial organisms from the liver which had only congestion.

Subcapsular haemorrhage was observed in two cases out of 575 livers examined. There are no reports regarding subcapsular haemorrhage in the liver of quails. Haemorrhage under the capsule of the liver might be due to mechanical

factors inflicted during transportation of quails from one pen to another, fight between the birds or during flying. Toxic principles could also cause such haemorrhages. In this study the subcapsular haemorrhage was associated with severe congestion; during flight due to mechanical factors there is every possibility of rupture of the capillaries in the subcapsular region.

Hepatic necrosis was observed in 39 cases out of 575 livers examined. The incidence of hepatic necrosis was 6.7 per cent. The incidence was highest in adult quails (9.28 per cent) and lowest in quail chicks (0.93 per cent). Hepatic necrosis, in most of the instances, was associated with acute congestion or fatty change or both. This explains the possibility of toxic liver damage. The earliest manifestation of liver to noxious agents being fatty change, this might have progressed to the stage of necrosis. Focal necrosis was also associated with bacterial hepatitis in three cases, from which S. typhimurium was isolated. Necrotic changes were associated with congestion and infiltration of heterophils.

Necrosis of hepatic cells was not always associated with the presence of greyish-white foci, macroscopically.

Histologically, hepatic necrosis was also seen in livers which had severe congestion, grossly. But in these cases the necrotic changes were confined to a place deep in the liver parenchyma. The livers from which S. typhimurium was isolated had pin head sized greyish-white foci on the surface. Kapoor et al. (1980) observed greyish-white foci on the surface of the liver of quails infected with S. bareilly. Hepatic necrosis was observed in Ulcerative enteritis (Peckham, 1960), Listeriosis (Nikuradze 1970), Pasteurellosis (Myint and Carter, 1988) and E. coli infections (Ito, 1990). Berkhoff et al. (1974 b) observed extensive hepatic necrosis in experimentally induced UE in quails. Extensive liver necrosis was not observed in this study. Packham (1959) was able to isolate gram positive rods from the quail liver which had small greyish-white foci. However, in the present study, gram positive rods could not be isolated from the livers which had small greyish-white foci when subjected to bacteriological examination.

Fatty change was the most common finding next to congestion. The incidence of fatty change as the predominant histologic finding was 30.26 per cent. In most of the cases fatty change was associated with congestion.

Congestion being the first response of any organ to the noxious agents, liver would respond by the accumulation of lipids in the hepatocytes as the next stage. Toxic factors from the feed may be attributed as the causative factors, while at the same time bacterial infections leading to fatty change cannot be overlooked. Fatty change was a consistent lesion in experimental salmonellosis, in this investigation. Also fatty change was the consistent finding observed in hepatic toxicity of dietary monensin in quail. Extensive fatty change was observed by Tarrant et al. (1983) in the liver of quails fed DDMU, a metabolite of DDT. It can be reasonably suggested that in the case of quails fatty change could be the significant hepatic response to any noxious agents. In few instances diffuse fatty degeneration was associated with focal necrosis. This may be because of the continued action of the toxic agent which was responsible for inducing congestion and fatty change.

Hepatitis observed in few cases was also associated with fatty change. This could suggest that the quails either would not survive till the fatty change is progressed to the stage of inflammation or those with liver having highest regenerating capacity would withstand the noxious agents, if these were not serious.

In many cases the fatty change was so diffuse and extensive that all most all the hepatic cells were containing fatty vacuoles. This extensive infiltration caused disintegration of the reticular fibres which was evident in sections stained with Gomori's method of staining for reticulum.

Liver which had only congestion as the gross change also had mild to moderate fatty change, histologically. Therefore, possibility of fatty change cannot be ruled out when congestion of the liver is the only gross change.

Except in few cases, Marek's disease was always associated with fatty change. This may be explained by the fact that Marek's disease begins as an inflammatory reaction and as in other cases, this inflammatory reaction may also induce lipid accumulation in the hepatocytes, or fatty change observed in Marek's disease affected liver may be an incidental finding caused by other factors and not associated with MD.

The incidence of hepatitis was 1.73 per cent. The overall incidence of hepatitis was less. This indicated that hepatitis was less commonly encountered in quails. It was highest in quails aged above six weeks. Hepatitis was not

observed in quails aged 3-4 weeks. In quail chicks the incidence was 0.46 per cent. In adults the incidence of hepatitis was more when compared to quail chicks. S. typhimurium was isolated from the liver which had hepatitis. Most of the adult chicken affected with Salmonellosis are carriers which had survived the infection when they were chicks or acquired a chronic localized infection during growth. When the birds are immunosuppressed because of some other disease conditions or any other stress factors, the organisms may flare up and produce hepatitis (Gordon, 1977).

S. typhimurium was isolated from three cases of hepatitis. This has significance because S. typhimurium also affects human beings and it has zoonotic importance (Clarenburg, 1964). This investigation has therefore, brought to light the prevalence of this important zoonotic disease in the quail farm of the University and there is urgent need to take measures to control the disease by serological screening and culling of the affected quails.

In two cases of hepatitis, there was fibrinous perihepatitis. E. coli or Salmonella can be attributed as causative agents for this condition even though no efforts

were made to isolate the bacterial organisms from these livers as only congestion could be appreciated grossly. Sarma et al. (1988) reported liver lesions which included perihepatitis, in an outbreak of S. gallinarum infection in Japanese quails.

In many cases hepatitis was associated with fatty change. As discussed earlier, the fatty change observed here may be a stage in the development of hepatitis.

Marek's disease was diagnosed in 106 cases out of 575 livers examined. The percentage incidence was 18.43. The incidence was high. This is because the quails received for autopsy are from the farm where chicken and quails are reared in an intensive system. MD is prevalent in chicken in the farm. Therefore, there is every possibility of quails getting exposed to MD virus. The quails are not vaccinated against MD. The prevalence of MD in this farm was reported earlier by Nair et al. (1986).

In this investigation, MD was diagnosed in quails aged more than five weeks and in most of the instances it was diagnosed in quails aged more than one year. This is a feature not seen in chicken. In chicken MD is seen in birds of the younger age group. Therefore, the age criterion for

differentiating Marek's disease and lymphoid leukosis cannot be considered valid in the case of quails.

Gross lesions of MD in quails were also different from those seen in chicken. Unlike in chicken nodular lesions were not seen. But diffuse enlargement was the characteristic lesion seen in many cases. In many instances greyish-white foci, which appeared like necrotic foci, were evident. Fugimoto et al. (1975) also observed slightly enlarged livers with scattered or diffuse foci on their surfaces. Pradhan et al. (1985) observed liver which was granular in appearance, in a natural case of MD in Japanese quails. The observations made in the present study were similar to those observed by the above authors. Nair et al. (1986) noticed enlarged liver of quails, which had many circumscribed greyish-white nodules. This is in contrast to the observations made in this study. In three cases, the liver was enlarged three times the normal size. Dutton et al. (1973) also observed severely enlarged liver in quails due to MD. In one case there was also rupture of the enlarged liver.

Histologic lesions were always associated with severe congestion. The pleomorphic lymphoid cell infiltration was similar to those found in chicken. Both focal and diffuse

infiltration were found. Focal infiltration was associated with the presence of greyish-white foci, grossly. In the case of diffusely proliferating type, most of the hepatocytes were replaced by pleomorphic neoplastic lymphocytes. Nair et al. (1986) also observed large sheets of hepatic cells replaced by lymphocytes.

Out of 106 MD livers examined histologically, 16 livers did not show either gross enlargement or the presence of greyish-white foci. But on histologic examination they were confirmed as MD. Fugimoto et al. (1975) also could not find grossly visible lesions in any of the visceral organs or tissues in many quails affected with MD. This observation clarified that gross involvement need not be a criterion for diagnosing MD.

In the case of chicken, in MD besides the liver, other organs like the kidney, ovary, spleen, heart etc. are involved. In the case of quails, in this investigation, besides the liver, the only other organ found to be involved was the spleen. It would appear that in the case of quails, liver is an important target organ for the MD virus. This observation has significance and needs further investigation to clarify the organ specificity.

Experimental salmonellosis

This investigation has established the prevalence of Salmonellosis in quails and it was also clarified that the organism involved was S. typhimurium. There has not been any reported cases of S. typhimurium infection in quails from India. Edgar et al. (1964) were also able to infect quails experimentally with S. typhimurium. Experimental studies in the present investigation indicated that stress plays a significant role in enhancing the pathogenicity of the organism. In the experimentally induced stress with cortisone acetate, the quails were found to be very susceptible, the distribution of lesions were extensive and mortality was high and earlier. This implies that in natural infections when quails are under stress they will pick up the infection easily. Also the experimental model using cortisone acetate has brought to light the importance of stress in precipitating the disease. It was reported by Siegel et al. (1983) that critical antigen concentration may be capable of inducing maximal humoral antibody responses in moderate environments but which allow these responses to be suppressed by stress factors that stimulate increased pituitary-adrenal activity. Therefore there is need to prevent stress during rearing of quails. This would help to prevent disease outbreaks. Farmers have to be cautioned about the importance of stress in precipitating disease outbreaks.

There was progressive loss of body weight from the second day onwards. Quails failed to gain optimum body weight till the 15th day, eventhough feed consumption was normal after eighth day. Early infection was followed by transient bacteraemia. These observations are similar to the findings of Brown et al. (1975) in cockerels experimentally infected with S. typhimurium.

After the 8th day, the feed consumption was normal and the quails which were sacrificed on the 15th day were healthy but had localized necrotic foci in the liver indicating a chronic phase. This indicates quails are relatively less susceptible to S. typhimurium infection.

The multiple gross distribution of lesions and the perivascular location of the lesions indicated a bacteraemic phase and localization of the organisms in the liver. The necrotic foci were similar to the classical lesions described in chicken in Salmonellosis (Gorden, 1977). The immunosuppression however, naturally caused more extensive and severe lesions in the liver.

S. typhimurium was isolated from the liver upto the 15th day. This was in contrast to the observations made by Brown et al. (1975) in cockerels. They were able to isolate

the organisms from the cockerels infected with S. typhimurium upto 5 days, and after that a fall in the recovery rate occurred. This would mean that the infected quails continue to excrete the organisms for a long period of time and this would serve as a source of infection in the flock. Therefore, there is need to screen the flock for salmonella infection and take urgent measures to control the infection in the flock.

Hepatic toxicity of dietary monensin

Monensin is employed as a feed supplement to control Coccidiosis in chicken. This is also used in some of the quail farms for this purpose. Therefore, it was decided to study the toxicity of this chemical in quails.

Depression in the growth occurred after 15 days of commencement of the experiment. It became more pronounced after 60 days. The depression in the growth rate could be attributed to the direct adverse effect of monensin on the quail.

Fatty change in the liver was the consistent finding. As discussed elsewhere, fatty change in the liver is the significant response of the liver to noxious agent in the

case of quails. Sawant et al. (1990) reported hepatotoxicity in quails based on the alterations in the enzyme profile when monensin was administered for a period of 15 days at the dose rate of 330 ppm. They did not undertake any histopathological studies. In this investigation, when 250 ppm of monensin was given for a period of three months, there was definite indication of hepatotoxicity characterized by fatty change. The hydropericardium observed in one bird may be because of toxic liver damage thereby producing hypoprotenemia. No other lesions were observed in the heart. This is in contrast to the lesions observed by Wagner et al. (1983) in the heart of the chicks fed monensin. They observed focal heterophil infiltration, focal epicardial thickening and subepicardial haemorrhage. But in the present study focal heterophil infiltration was observed in the liver of two quails. The diffuse fatty change observed demonstrated that liver is the target organ of toxicity of monensin in quails. Hepatotoxicity in quails fed monensin as reflected by changes in the activities of serum enzymes and liver microsomal enzymes was reported by Sawant et al. (1990). It could, therefore, be summarised that monensin has hepatotoxic effect at the dose level employed and it should be used as a feed additive for the quails only with caution.

Summary

6. SUMMARY

An investigation was undertaken to study the incidence and nature of hepatic disorders in Japanese quails. Experimental studies were also undertaken to elucidate the pathogenesis and pathology of Salmonella typhimurium infection and the hepatic toxicity of dietary monensin in Japanese quails.

The data on the incidence of hepatic lesions recorded in 19,749 quails during the period 1986-1991 at the Centre of Excellence in Pathology were analysed.

The overall incidence of hepatic lesions for the above period was 34.09 per cent. The incidence was high in adults (78.3 per cent). In quails aged 5-6 weeks, the incidence of hepatic lesions was 62.50 per cent, in quails aged 3-4 weeks it was 46 per cent and in quail chicks the incidence of hepatic lesion was 20.90 per cent.

Besides this, 575 livers with well defined gross lesions were systematically examined for histologic changes during the period 1990-91. Among the hepatic lesions encountered, the percentage of incidence of different types of liver lesions in the decreasing order of frequency were congestion (42.08 per cent), fatty change (30.26 per cent), Marek's

disease (18.43 per cent), necrosis (6.78 per cent), hepatitis (1.73 per cent), subcapsular haemorrhage (0.34 per cent) and abscesses (0.34 per cent).

During the course of investigation hepatic abscesses were seen in two quails. In one case gross lesions were evident and in the other case microabscesses were seen, histologically.

Hepatic congestion was the most common histologic lesion encountered (42.08 per cent). It was more common in quail chicks (65.58 per cent) and less in quails aged above 6 weeks (19.40 per cent). The histopathological features of congestion were described, the pathogenesis was elucidated and the aetiological factors were indicated.

Subcapsular haemorrhage was observed in two instances. The gross and histologic features and probable causative factors were discussed in detail.

Hepatic necrosis was seen in 39 cases out of 575 livers examined histologically. The incidence was more in adults. The necrosis observed was of coagulative type. The gross and histologic features were discussed in detail.

Fatty change was the most common finding encountered in adults. The overall incidence of fatty change was 30.26 per cent. In quails aged above 6 weeks the incidence was 25.73 per cent. It was pointed out that in quails fatty change was the most consistent hepatic response to toxic agents. The histologic features of fatty change were described in detail and the possible causative factors were discussed. Disintegration of reticular fibres in cases of extensive hepatic steatosis was demonstrated by Gomori's method of staining for reticulum.

Marek's disease lesions were noticed in 106 livers out of 575 specimens examined. The incidence was 18.43 per cent. Marek's disease was mostly seen in quails aged more than one year. The distinguishing features of liver lesions of MD in the quail and chicken were brought to light and its importance was discussed.

Ten cases of hepatitis were seen out of 575 livers examined histologically. The incidence of hepatitis was found to be less in quails. Histological changes were described in detail.

Salmonella typhimurium was isolated from three adult quails which died spontaneously. This is the first record

of S. typhimurium infection in quails from India. Experimentally Salmonellosis was induced in quails using this isolate with and without immunosuppression with cortisone acetate. The lesions were more severe and extensive in immunosuppressed quails. The lesions were predominant in the liver. Induced stress was found to enhance the pathogenecity of the organism. The role of stress in precipitating the infection in the field situation was clarified. Zoonotic importance of S. typhimurium infection was discussed. Hepatic necrosis was a consistent lesion. Pathogenesis of the condition was clarified and the pathological features were described in detail.

By an elegantly designed experimental study the hepatic toxicity of dietary monensin was demonstrated. Reduction in the body weight was noticed from the 15th day of the commencement of the experiment. Hepatic steatosis was the consistent feature. The importance of the toxicity was highlighted and the need to caution the farmers in using monensin as a feed additive was pointed out.

References

REFERENCES

*Acedo, C.S. and Reguera, V.G. (1972). Identification of E. bateri in quail (Coturnix coturnix japonica). Revista Iberica de Parasitologica 32 (34): 271-276.

Arbid, M.S., El-Habbak, M.M.E. and Hanafy, M.S.M. (1990). Toxicological and biological studies on Japanese quails fed graded levels of furazolidone. Acta. Veterinaria Hangurica. 38(4): 271-279.

Atwal, O.S. (1973). Fatty changes and hepatic excreation in avian liver. An EM study of kepone toxicity. J. Comp. Path. 83(1): 115-129.

*Awaad, M.H.H., Hafez,H.M., El-Dimerdash, M.Z. and Krauss H. (1981). Zentralblatt fur Veterinarmedizin. 28(9/10): 704-712.

Berkhoff, G.A. and Campbell, S.G. (1974 a). Etiology and pathogenesis of Ulcerative enteritis (quail disease): The experimental disease. Avian Dis. 18(2): 205-212.

Berkhoff, G.A., Campbell, S.G. and Naylor, H.B. (1974 b). Etiology and Pathogenesis of Ulcerative enteritis (quail disease): Isolation of the causative anaerobe. Avian. Dis. 18(2): 186-194.

Berkhoff, G.A., Campbell, S.G., Naylor, H.B. and Smith, L.D. (1964 c). Etiology and pathogenesis of Ulcerative enteritis (quail disease): Characterization of the causative anaerobe. Avian Dis. 18(2): 195-204.

Bigland, C.H. (1963). Leucosis complex in Japanese quails. Personal communications. In Vet. Rec. 75(27): 685-687. Lymphoid Leucosis and fowl paralysis in the quail.

Bigland, C.H., Damassa, A.J. and Woodard, A.E. (1965). Diseases of Japanese quail (Coturnix coturnix japonica) - A flock survey and experimental transmission of selected avian pathogens. Avian Dis. 9: 212-219.

Blankford, M.B., Ottinger, M.A. and Doerr, J.A. (1981). Aflatoxicosis in juvenile Japanese quails. Poult. Sci. 60(7): 1626.

Bozhkov, S. and Toshkov, I. (1984). Dynamics of morphological changes in Japanese quails infected with avian leucosis virus strain. Obshecha isravniteina Pathologita 15(0-3): 16-25.

Brown, D.D., Ross, J.G. and Smith, A.F.G. (1975). Experimental infection of cockerels with Salmonella typhimurium. Res. vet. Sci. 18: 165-170.

*Burns, R.B. and Dwivedi, P. (1984). Proceedings of the V meeting on mycotoxins in human and animal health, Edinburgh, pp. 11-12.

*Buss, I.O., Conrad, R.D. and Reilly, J.R. (1958). Ulcerative enteritis in wild California quail. J. Wildl Mgt. 22: 446-447.

*Carlson, H.L. Seawright, G.L. and Pettit, J.R. (1974). Reticuloendotheliosis in Japanese quail. Avian Pathol. 3(3): 169-175.

*Chang, S.I. and McGinnis, J. (1967). Vitamin D deficiency in adult quail and chicken and effects of estrogen and testosterone treatments. Proc. Soc. exp. Biol. Med. 124: 1131-1135.

Chang, C-F. and Eisen, E.J. (1979). Nature of resistance to aflatoxin in Japanese quail (Abstr.). Poult. Sci. 58: 1042.

Chang, C-F. and Hamilton, P.B. (1982). Experimental aflatoxicosis in young Japanese quails. Poult. Sci. 61(5): 869-874.

Chaudhary, S.K., Sadana, J.R. and Pruthi, A.K. (1988 a). Sequential pathological studies in Japanese quails infected experimentally with Aspergillus fumigatus. Mycopath. 103: 157-166.

Chaudhary, S.K. and Sadana, J.R. (1988 b). Experimental aspergillosis in Japanese quails (Coturnix coturnix japonica) clinical signs and haematological changes. Mycopath. 102: 179-184.

Chew-Lim, M. (1980). Adult Coturnix quail bronchitis. Avian Dis. 24(2): 520-526.

Clarenburg, A. (1964). Zoonoses. Elsevier Publishing Company, London, P.145.

*Corrado, A. (1970). Susceptibility of Japanese quail (Coturnix coturnix japonica) to fowl pathogens. Nuova. Vet. 46: 49-52.

Cowan, S.T. (1974). Manual for the identification of medical bacteria. Cambridge University Press, London. 2nd Ed. pp. 103-106.

Crawford, J.A. (1979). Avian pox in California quail J. Wildl. Dis. 15: 447-449.

Crawford, J.A. (1986). Differential prevalence of Avian Pox in adult and immature California quail. J. Wildl. Dis. 22(4): 564-566.

*Cygan, Z. and Nowak, J. (1974). Necrotic enteritis in chickens. II. Toxigenic properties of Cl. perfringens type C strain and experimental infection of Japanese quail. Weterynaryjna 30(5): 262-265.

Damron, B.L., Manley, J.G., Wilson, H.R. and Harms, R.H. (1977). Floxaid and Aflatoxicosis of bobwhite quail chicks (Abstr.). Poult. Sci. 56: 1708.

Davis, B.R., Brown, J. and Dawe, L.D. (1971). Quail-Biological indicators in the differentiation of Ulcerative enteritis and Necrotic enteritis in chickens. Poult. Sci. 50(3): 737-740.

Davis, B.R. (1973). Ulcerative enteritis in chickens: Coccidiosis and stress as predisposing factors. Poult. Sci. 52: 1283-1287.

Derieux, W.T. (1983). Reaction of bobwhites, Coturnix quail, guinea fowl and mallards to avirulent and virulent strain of P.multocida. Avian Dis. 27(2): 539-541.

Dhillon, A.S., Winterfield, R.W., Tacker, H., Kazacos, K.R. and Alby, C.J. (1980). A typical Histomoniasis in bobwhite quails. Avian Dis. 24(2): 510-516.

Dider, R., Gueux, E. and Rayssiguier, Y. (1984). Magnesium deficiency in the Japanese quail. Comp. Biochem. Physiol. 79A(2): 223-227.

Disbrey, B.D. and Rack, J.H. (1970). Histological laboratory methods. E. & S. Livingstone, Teviot Place, Edinburgh. pp. 160-161.

Dubose, R.T., Grumbles, L.C. and Flowers, A.I. (1958). The isolation of a non bacterial agent from quail with a respiratory disease. Poult. Sci. 37: 654-658.

Dubose, R.T. and Grumbles, L.C. (1959). The relationship between quail bronchitis virus and CELO virus. Avian Dis. 3(3):321-344.

*Durant, A.J. and Doll, E.R. (1941). Ulcerative enteritis in quail. Missouri Agr. Exper. Sta. Res. Bul. p. 325.

Dutton, R.L., Kenzy, S.G. and Becker, W.A. (1973). Marek's disease in Japanese quails. Poult. Sci. 52(1): 139-143.

Edgar, S.A., Waggoner, R. and Flanagan, C. (1964). Susceptibility of Coturnix quail to certain disease producing agents common to poultry. Poult. Sci. 43: 1315.

*El Dahaby, H., El Sahbagh, A.H. and Nassar, M.I. (1971). Some studies on the role of wild birds in the transmission of avian pox. J. Egypt. vet. med. Ass. 31: 97-104.

Eleazer, H.T., Blalock, H.G. and Warner, H.J. (1978). Eastern Equine Encephalomyelitis outbreak in Coturnix quail. Avian Dis. 22: 522-525.

Ellis, F.E. (1967). Histomoniasis in Japanese quails. Arch. Exp. Vet. Med. 1: 231-245.

*Enigk, K. and Dey-Hazra, A. (1970). The host specificity of Syngamus trachea. Zentbl. Vet. Med. 17B: 840-847.

Fuchs, R., Appelgren, L.E., Hagelberg, S. and Hult, K. (1988). Carbon-14-Ochrotoxin A distribution in Japanese quail monitored by whole body autoradiography. Poult. Sci. 67(5): 707-714.

Fugimoto, Y., Mikami, J., Narita, M. and Okada, K. (1975). Pathological studies of Marek's disease in Japanese quail. Jap. J. vet. Res. 23(4): 119-124.

Gangadharan, B., Valsala, K.V., Maryamma, K.I., Ramachandran, K.M., Nair, M.G. and Rajan, A. (1989). An analysis of mortality in quails (Coturnix coturnix japonica). Kerala J. vet Sci. 20(1): 44-49.

Ghori, H.M. and Edgar, S.A. (1973). Comparative susceptibility of chicken, turkeys and quails to Aspergillosis. Poult. Sci. 52(6): 2311-2315.

*Githkopoulos, P.R., Liakos, V.D., Punagiotidou-Mamalouka, V. and Lekkas, S. (1983). Capillariasis of the crop and oesophagus in quails (Coturnix coturnix japonica) Partridge and pheasant. Hellenike kteniatrike, 26: 287-299.

Glisson, J.R., Cheng, I.H.N., Rowland, G.N. and Stewart, G.R. (1989). Pasteurella multocida infection in Japanese quails. Avian Dis. 33(4): 820-828.

Gordon, R.F. (1977). Poultry Diseases. The English Language Book Society, London, Ist Ed. pp. 1-337.

Gorham, S.L. and Ottinger, M.A. (1986). Sertoli cell tumors in Japanese quails. Avian Dis. 30(2): 337-339.

Graham, R. (1936). Salmonella isolated from baby quail. J. Am. vet. med. Assoc. 88: 763-764.

Harris, A.H. (1961). An outbreak of Ulcerative enteritis amongst bobwhite quail. Vet. Rec. 73(1): 11-12.

*Hashimoto, K., Kaneko, S., Onoshi, H., Kobyashi, K., Maeda, M. and Kita, E. (1969). An outbreak of Newcastle disease in Japanese quails (Coturnix coturnix japonica). Bull. natn. Inst. Anim. Hlth. 58: 7-14.

Higgins, D.A. and Wong, F.S.F. (1968). Newcastle disease in a flock of Japanese quails. Vet. Rec. 83(17): 437-440.

Hill, R.W. and Raymond, R.G. (1962). Apparent natural infection of Coturnix quail hens with the virus of Avian encephalomyelitis - case report. Avian Dis. 6: 226-227.

Hoerr, F.J., Current, W.L. and Haynes, T.B. (1986). Fatal Cryptosporidiosis in quail. Avian Dis. 30(2): 421-425.

Howell, J. and Thompson, J.N. (1970). Lesions in nervous tissue and bone in a toxic vitamin A deficient quail. Acta neuropath 16: 285-292.

*Imai, K., Yuasa, N., Kobayashi, S., Nakamara, K. and Tsukamoto, K. (1990). Isolation of MD virus from Japanese quail with Lymphoproliferative disease. Avian Pathol. 19(1): 119-129.

Ito, H., Okada, S., Kobayashi, S., Sugiyama, H. and Natsume, M. (1990). Colibacillosis in Japanese quails in the Higashimikawa district. J. Jap. vet. med. Assn. 43(9): 661-665.

Jack, S.W., Reed, W.M. and Bryan, T.A. (1987). Inclusion body hepatitis in bobwhite quails (Colinus virginianus). Avian Dis. 31(3): 662-665.

*Jack, S.W. and Reed, W.M. (1988). Pathology of quail bronchitis. Proc. Westn. Poult. Dis. conf. 37: 102-104.

Jack, S.W. and Reed, W.M. (1989). Observations of experimentally induced Quail bronchitis. Avian Dis. 33(3): 446-450.

Jack, S.W. and Reed, W.M. (1990). Pathology of experimentally induced Quail bronchitis. Avian Dis. 34(1): 44-51.

Jack, S.W. and Reed, W.M. (1990). Further characterization of an avian adenovirus associated with IBH in bobwhite quails. Avian Dis. 34(3): 526-530.

*Jsovsky, H.J. and Svoboda, S. (1973). Experimental infection of quail with M. avian. Archiv Fur Experimentelle Veterinarmedizin. 27: 926 -937.

Kapoor, K.N., Chauhan, H.V.S. and Gupta, B.R. (1980). Epidemiological and pathological studies in outbreaks of S. bareilly infection in chicken and quails. Indian vet. J. 57(7): 536-538.

Karlson, A.G., Thoen, C.O. and Harrington, R. (1970). Japanese quail: susceptibility to avian Tuberculosis. Avian Dis. 14: 39-44.

Kaul, L. and Pradhan, H.K. (1991). Immunopathology of Marek's disease in quails: presence of antinuclear antibody and immune complex. Vet. Immun. Immunopath. 28: 89-96.

Kellong, F.E. and Reed, W.M. (1970). Bobwhite quails as possible reservoir hosts for Black head in turkeys. J. Wildl. Mgt. 34:155-159.

Khare, M.L., Grun, J. and Adams, E.V. (1975). MD in Japanese quail - a pathological virological and serological study. Poult. Sci. 54(6): 2066-2081.

Kobayashi, S., Kobayashi, K., Itoh, H. and Mikami, T. (1987). Some epizootiological and pathological observations on MD in Japanese quail. Prev. vet. Med. 5(2): 141-150.

Kling, L.J. and Suares, J.H. (1980). Vitamin E deficiency in Japanese quail. Poult. Sci., 59(10): 2352-2354.

Kumaran, K. and Venkatesan, R.A. (1991). Characterization of a strain of RD virus isolated from Japanese quails. Indian J. Anim. Sci. 61(5): 499-500.

*Lohliger, H.C. and Schubert, H.J. (1967). Spontaneous diseases in Japanese quail. Z. Versuchstierk. 9: 87-94.

*Lohliger, H.C. and Schubert, H.J. (1967). Aetiology and pathology of Lymphoid cell leucosis in Japanese quail. Dt. tierarztl. Wschr. 74: 154-158.

Luna, L.G. (1968). Manual of Histologic staining methods of the Armed Forces Institute of Pathology. McGraw-Hill Book Company, New York, 3rd Ed. pp. 1-258.

Mandelli, G., Rinaldi, A., Nardelli, L., Cervio, G., Cessi, D. and Valeri, A. (1968). Observazioni epidemiologi chesu diun focolaio di influenza ganglia domestica. Atti della Societa Italiana delle Scienze Veterinarie. 22: 787-792.

Marks, H.L. and Wyatt, R.D. (1979). Genetic resistance to aflatoxin in Japanese quail. Science 206(4424): 1329-1330.

Marks, H.L. and Wyatt, R.D.(1980). Aflatoxin susceptibility of selected and control Japanese quail lines. Br. Poult. Sci. 21: 29-36.

Maruyana, S. and Katsube, Y. (1990). Isolation of C. jejuni from the eggs and organs in experimentally infected laying Japanese quails. Jap. J. vet. Sci. 52(3): 671-674.

Mathew, E.S. and Sulochana, S. (1990). Isolation of Salmonella gallinarum from Japanese quails (Coturnix coturnix japonica). Indian J. Comp. Microbiol. Immunol. Infct. Dis. 11(1): 53-54.

Matsui, T., Kuroda, S., Mizutani, M., Kiuchi, Y., Suzuki, K. and Ono, T. (1983). Generalized glycogen storage disease in Japanese quail (Coturnix coturnix japonica). Vet. Pathol. 20(3): 312-321.

Maxwell, M.H. and Burns, R.B. (1987). Ultrastructural study of ochrotoxicosis in quail. Res. Vet. Sci. 42(2): 228-232.

*Mazurkiewicz, M.D., Podlewska and Wachnik, Z. (1967). Coccidiosis in Japanese quails. Med. Vet. 23: 536-537.

McBlain, W.A., Lewin, V. and Wolfe, F.H. (1974). Limited accumulation of DDT in eggs, fat and livers of Japanese quail. Poult. Sci. 53(1): 84-88.

*McFarland, L.Z. (1969). Physiological and endocrinological effects of the insecticide kepone in Japanese quail. Sch. vet. Med. 15: 441-443.

*Mehler, M. (1981). Susceptibility of Japanese quail to Cl. botulinum toxins. Inaguaral Dissertation, Justus Liebig Universitat, Giessen. 97 pp.

Minakshi, Dogra, S.C. and Ayyabari, A. (1988). Isolation of C. jejuni from quail: an initial report. Br. vet. J. 144(4): 411-412.

Moscovici, C. and Macintyre, E.H. (1966). Effect of avian myeloblastosis virus in Japanese quail. J. Bact. 92: 1141-1149.

Mumtaz, M.M., Menzer, R.E. and Tiong, S.K. (1986). Comparitive metabolism and fate of fenvalerate in Japanese quails and rats. Comp. Biochem. Physiol. 34(6) 929-936.

Myint, A. (1987). Proteus mirabilis infection in quail in Burma. Trop. Anim. Hlth. Prod. 19(2): 75-76.

Myint, A. and Carter, G.R. (1988). Fowl cholera in quail in Burma. Trop. Anim. Hlth. Prod. 20(1): 35-36.

Nair, G.M., Jayakumar, P.M. and Valsala, K.V. (1986). Marek's disease in Japanese quails. Kerala J. vet. Sci. 17(1): 146-148.

*Nardelli, L., Rinaldi, A., Pereira, H.G. and Mandelli, G. (1990). Influenza virus infections in Japanese quail. Arch. exp. Vet. Med. 24: 231-249.

Narita, M., Okada, K. and Fugimoto, Y. (1975). Pathogenic and serologic studies of Japanese quails infected with JM strain of MD herpes virus. J. natn. Cancer Inst. 54(3): 607-614.

Nascimento, G.F. and Nascimento, E.R. (1986). Infectious sinusitis in Coturnix quail in Brazil. Avian Dis. 30(1): 228-230.

Neal, G.E., Judah, D.J. and Green, J.A. (1986). The in vitro metabolism of aflatoxin B1 catalyzed by hepatic microsomes isolated from control or 3-methyl cholanthrene-stimulated rats and quail. Toxic. appl. Pharmac. 82(3): 454-460.

*Nedyalkov, S., Bozhkov, S. and Todorov, G. (1975). Experimental erythroblastosis in Japanese quail induced by E-26 leukosis strain. Acta Veterinaria Brno 44(1/2): 75-78.

*Nikuradza, T.I. (1970). Susceptibility of quail to listerosis. Veterinariya. 4: 70-72.

Nishimura, E.T., Ross, E. Leslie, G., Hong-Yiyang and Hokama (1970). Epizootic reticulum cell sarcoma in a sequestered colony of Japanese quail. Cancer Res. 30: 2119-2126.

Norten, C.C. and Peirce, M.A. (1969). The life cycle of E. bateri in the Japanese quail (Coturnix coturnix japonica). J. Protozool 18: 57-62.

*Olson, N.O. (1950). A respiratory disease (bronchitis) of quails caused by a virus. Proc. U.S. Livestock. San. Assn. 54: 171-174.

Olson, L.D. (1969). Aspergillosis in Japanese quail: case report. Avian Dis 13:225-227

Panigrahy, B. and Hall, C.F. (1977). An outbreak of erysipelas in coturnix quail. Avian Dis. 21: 708-710.

Panigrahy, B. and Glan, S.E. (1982). Outbreaks of Fowl cholera in quail. Avian Dis. 26(1): 200-203.

*Peckham, M.C. (1959). An anaerobe, the cause of Ulcerative enteritis (quail disease). Avian Dis. 3: 471-477.

*Peckham, M.C. (1960). Further studies on the causative organism of Ulcerative enteritis. Avian Dis. 4: 449-456.

*Polero, B.J., Smitsaart, T.E., Vena, M.M. and Rivero, U. (1988). Outbreaks of fowl cholera in Japanese quail. Revista de Medicina Veterinaria 69(4): 219-223.

*Poorciau, S.S. and Springer, W.T. (1978). Frequency and duration of paratyphoid organism shedding by experimentally infected bobwhite quail. J. Wildl Dis. 14(2): 203-207.

Pradhan, H.K., Mohanty, G.C., Verma, K.C. and Panda, B.K. (1983). Evidence of some chicken viral infections in Japanese quails based on serological studies. Indian J. Poult. Sci. 18: 81-84.

Pradhan, H.K., Mohanthy, G.C. and Mukit, A. (1985). Marek's disease in Japanese quails - A study of natural cases. Avian Dis. 29(3): 575-582.

Pradhan, H.K., Panda, B.K. and Sah, R.L. (1980). Preliminary studies on pathology of quail bronchitis in Japanese quail. Indian vet. J. 57: 707-708.

*Rao, J.R. and Sharma, N.N. (1990). Interaction between Eimeria uzura infection and Aflatoxicosis in Japanese quail (Coturnix coturnix japonica) Vet. Para. 35(3): 259-267.

Rao, J.R. and Sharma, N.N. (1992). Coccidiosis in Japanese quails in India. Indian J. Anim. Sci. 62(1): 51-52.

Ratnamohan, N., Bagust, T.J. and Spradbrow, P.B. (1979). Transmission of an Australian strain of reticuloendotheliosis virus to adult Japanese quail. Aust. vet.J. 55(10): 5060.

Ratnamohan, N. (1985). The management of Japanese quail and their use in virological research: A review. Vet. Res. Communs. 9(1): 1-14.

Reece, R.L., Barr, D.A. and Owen, A.C. (1981) The isolation of H. paragallinarum from Japanese quail (correspondence). Aust. vet. J. 57(7): 350-351.

Reece, R.L., Taylor, T.K., Dickson, D.B. and Kerr, P.J. (1986). Mycosis in commercial Japanese quail, ducks and turkey. Aust. vet. J. 63(6): 196-197.

Reed, W.M., Kazacos, K.R, Dhillon, A.S, and Winterfield, R.W. (1981). Cerebrospinal nemetodiasis in bobwhite quail. Avian Dis. 24(4): 1039-1046.

*Richardson, M.E. and Spivey Fox, M.R. (1974 a). Dietary cadmium poisoning and enteropathy in Japanese quails. Histochemical and ultrastructural studies. Lab. Invest. 31(6): 722-731.

*Richardson, M.E., Spivey Fox, M.R. and Fry, B.E. (1974 b). Pathological changes produced in Japanese quails by ingestion of cadmium. Lab. Invest. 104(3): 323-328.

*Rinaldi, A., Mahnel, H., Nardelli L., Mandelli, G.C., Cervio, G. and Valeri, A. (1969). Richerche sai virus vaiolosi della guaglia (Coturnix coturnix japonica). Atti della societa Italiana delle Scienze Veterinarie 23: 1063-1066, 1066-1070.

*Rinaldi, A., Mahnel, H., Nardelli, L., Mandelli, G.C., Cervio, G. and Valeri, A. (1972). Pox virus of quail. I. Strains with characteristics resembling those of fowl pox virus. II. A strain with properties differing from those of known avian viruses. Atti Soc. ital. Sci. Vet. 23: 1063-1066.

Ritter, G.D., Ley, D.H., Levy, M., Guy, J. and Barnes, H.J. (1986). Intestinal cryptosporidiosis and reo virus isolation from bobwhite quails with enteritis. Avian Dis. 30(3): 603-608.

*Rossi, G.A., Simonella, P., Acciarri, C. and Casalema, B. (1972). Preliminary studies on an influenza outbreak in quail. Atti della Societa Italiana delle Scienze Veterinarie 26: 561-565.

Ruff, M.D. and Fegan, J.M. (1984). Pathogenicity of coccidia in Japanese quail. Poult. Sci. 63(1): 55-60.

Sah, R.L., Mall, M.P. and Mohanthy, G.C. (1983). Septaecemic proteus infection in Japanese quail chicks. Avian Dis. 27(1): 296-300.

Sarma, B.J., Dhanalakshmi, K., Reddy, D.B. and Chetty, S.M. (1988). An outbreak due to Salmonella gallinarum in Japanese quails. Indian vet J. 65(12): 1139-1140.

Sawant, S.G., Terse, P.S. and Dalvi, R.B. (1990). Toxicity of dietary monensin in quail. Avian Dis. 34(3): 571-575.

Sawhney, D.S., Vadehra, D.V. and Baker, R.C. (1973). Aflatoxicosis in the laying Japanese quail (Coturnix coturnix japonica). Poult. Sci. 52(2): 465-473.

Schat, K.A. and Gonzalez, J. (1976). A Lymphoproliferative disease in Japanese quails. Avian Dis. 20(1): 153-161.

*Schmatz, H.D. and Busche, R. (1977) Coxiella burneti infections in birds - Experimental infection of quail. Deutsche Tienarzthche Wochense Hrift 84(10): 375-376.

Shah, H.L. and Johnson, C.A. (1971). Eimeria bateri Bhatia, Pandey, 1965 from the Hungarian quail in the United States and its attempted transmission to the chicken. J. Protozool. 18: 219-220.

*Shanks, E.T., Statkiewicz, W.R., Lewelleyn, G.C. and Dashek, W.V. (1986). Tissue distribution and hepatic ultrastructural effects of aflatoxin B1 in Japanese quail. Polskie Archiwum Weterynaryjne 26(1/2): 117-131.

*Sharma, N.K. and Kaushik, R.K. (1986). Surveillance of diseases in Japanese quails. Indian J. vet. Med. 6(1): 48-50.

Siegel, H.S., Latimer, J.W. and Gould, N.R. (1983). Concentration of S. pullorum antigen and the immunosuppressive effect of Adrenocorticotrophin in growing chicken. Poult. Sci. 62(5): 897-903.

Silva, P.L. DA., Coelho, H.E., Ribeiro, S.C.A. and Oliveira, P.R. DE. (1989). Occurrence of Coligranulomatosis in coturnix quail in Uberlandia, Minas Gerais, Brazil. Avian Dis. 33(3): 590-593.

Smith, S.I., Weber, C.W. and Reid, B.L. (1969). The effect of high levels of dietary DDT on egg production, mortality, fertility, hatchability and pesticide content of yolk. Poult. Sci. 48: 1000-1004.

Snedecor, G.W. and Cochran, W.G.(1967). Statistical Methods. The Iowa State University Press, U.S.A. 6th Ed. pp. 91-296

Sonnenwirth, C.A. (1990). Gradwohl's Clinical laboratory methods and diagnosis. B.I. Publications Ltd., New Delhi, 8th Ed. p. 1363.

Srinivasan, V.A., Katra, D.S. Kharole, M.V. and Dwivedi, P. (1980). Mortality pattern in Japanese quails (Coturnix coturnix japonica). Indian vet. J. 37: 778-779.

Srinivasan, R., Shanmugam, A.M. and Nedunchelliyan, S. (1981). A note on the control of Ulcerative enteritis in quails. Cheiron. 10(1): 50-52.

*Suneja, S., Sadana, J.R. and Aggrawal, C.K. (1985). Studies on mortality patterns in quails as affected by sex, age and month of the year. Haryana vet. 22(2): 82-86.

*Svanbaev, S.K. and Utebaeva, M.K. (1973). Infectivity of pheasant and quail with coccidia. J. Acad. Sci. Kazak Soc. Stud. Reprod. Biol. Ser. 1: 62-68.

Tarrant, K.A., Bunyan, P.J., Stanley, P.I. and Westlake,G.E. (1983). The effects of 1, 1-Di(P-Chlorophenyl)-2-chloroethylene (DDMU) on hepatic morphology of Japanese quail. Toxic. appl. Pharmac. 67: 184-192.

*Tham, V.L., Kniesberg, S. and Dixon, B.R. (1982). Cryptosporidiosis in quails. Avian Pathol. 11(4): 619-626.

Tiong, S.K. (1978). Isolation of Mycoplasma gallisepticum from sinuses of three quails (Coturnix coturnix japonica). Vet. Rec. 103(24): 539.

Tsutsumi, Y. and Tsunoda, K. (1972). Pathogenicity of E. tsunodai for Japanese quail (Coturnix coturnix japonica) and susceptibility of the coccidium to some drugs. Jap. J. vet. Sci. 34(3): 115-121.

*Vallee, A., Le Turdu, Y., Gullion, J.C. and Le Menec (1972). Natural Pasteurella anatipestifer infection in aflock of quail. Bulletin de l'Academie Veterinaire de France, 45(7): 343-347.

*Villemin, M. (1972). Conservation du virus rabique chez la caille domestique (Coturnix coturnix japonica) apres inoculation intracerebral. Bulletin Academy de vet. 45: 237-238.

*Voronin, E.S., Dzagurov, S.G., Smirnova, N.E., Morozov, K.V. and Elekow, K.A. (1970). Seralogical investigations on some wild and domestic birds for avian infectious and oncogenic viruses. Voprosy Virusologii. 15: 213-217.

Wagner, D.D., Furrow, R.D. and Bradley, B.D. (1983). Subchronic toxicity of Monensin in broiler chickens. Vet. Pathol. 20: 353-359.

Wight, P.A.L. (1963). Lymphoid Leucosis and fowl paralysis in the quails.Vet. Rec. 75(27): 685-687.

*Yoshikawa, V., Yamanou, Takahashi, R. and Fugiwara, K. (1975). Histopathological observations of spontaneous regression of Rous sarcoma virus in Japanese quails. Jap. J. med. Sci. Bio. 28(3): 189-200.

*Zapryano M. (1979). E. tenella infection in young quails and E. kofoidi infection in chicks. Veterinarnomeditsinski Navki 16(4): 36-40.

*Zarzueto, E. and Gutierrez and Galiano, F. (1969). Wild birds as carriers and disseminators of Newcastle disease. Revta Patron Biol. Anim. 13: 49-66.

Zeakes, S.J., Hansen, M.F. and Robel, R.J. (1981). Increased susceptibility of bobwhite quail to Histomonas meleagridis after exposure to sevin insecticide. Avian Dis. 25(4): 981-987.

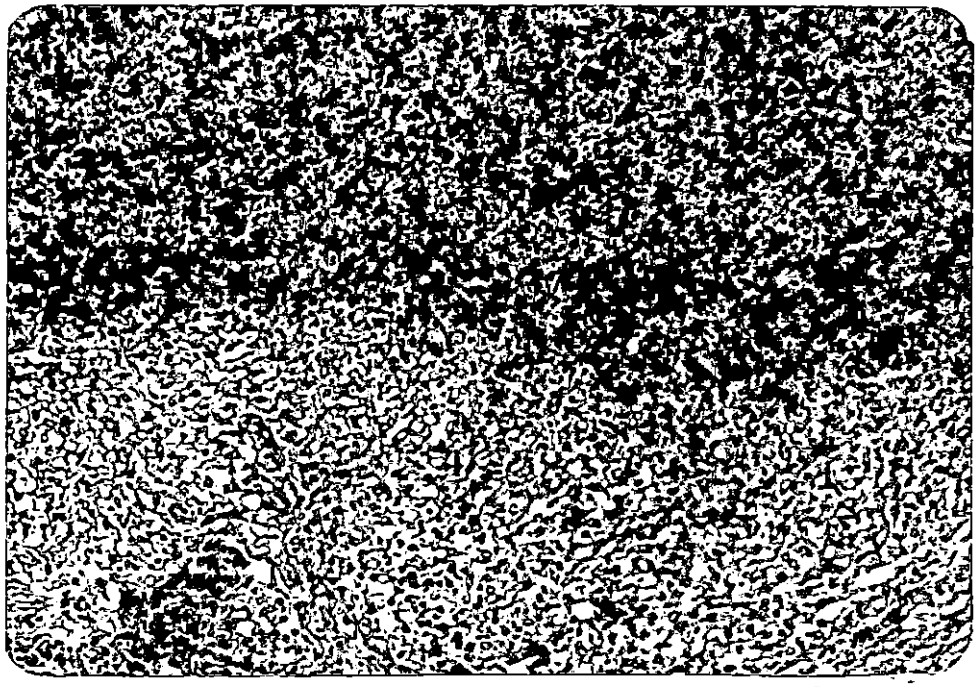
Zuo, Y.X. and Chen, F.Q. (1986). Coccidia of quail Eimeria bateri. Chinese J. vet. med. 12(2): 15-17.

* Originals not consulted

Photographs

Fig. 3. Hepatic abscesses

Fig.4. Heptaic abscesses - Suppurative foci and degenerating heptaic tissue (H & E x 160)



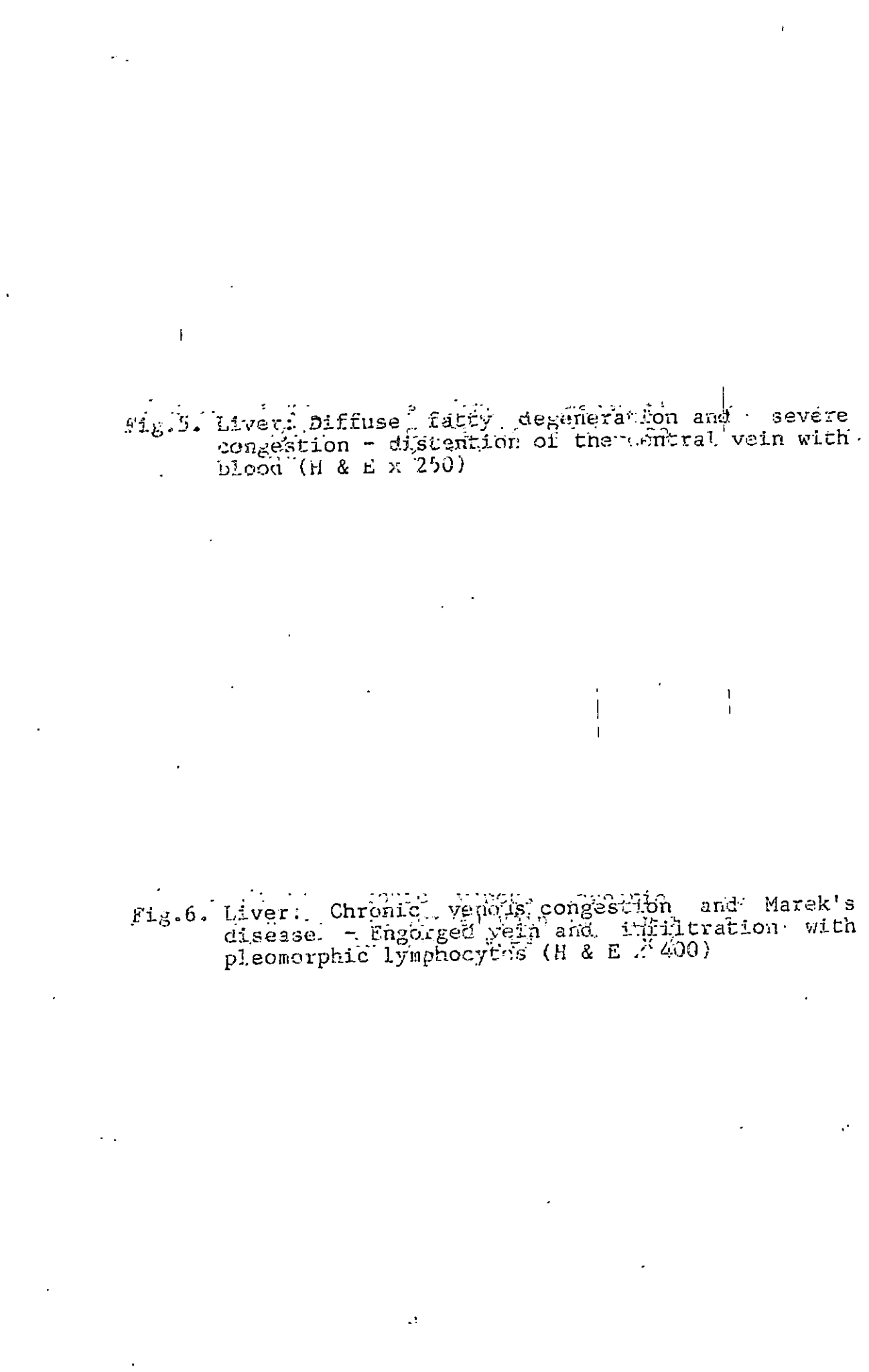


Fig.5. Liver: Diffuse fatty degeneration and severe congestion - distention of the central vein with blood (H & E x 250)

Fig.6. Liver: Chronic venous congestion and Marek's disease - Engorged vein and infiltration with pleomorphic lymphocytes (H & E x 400)

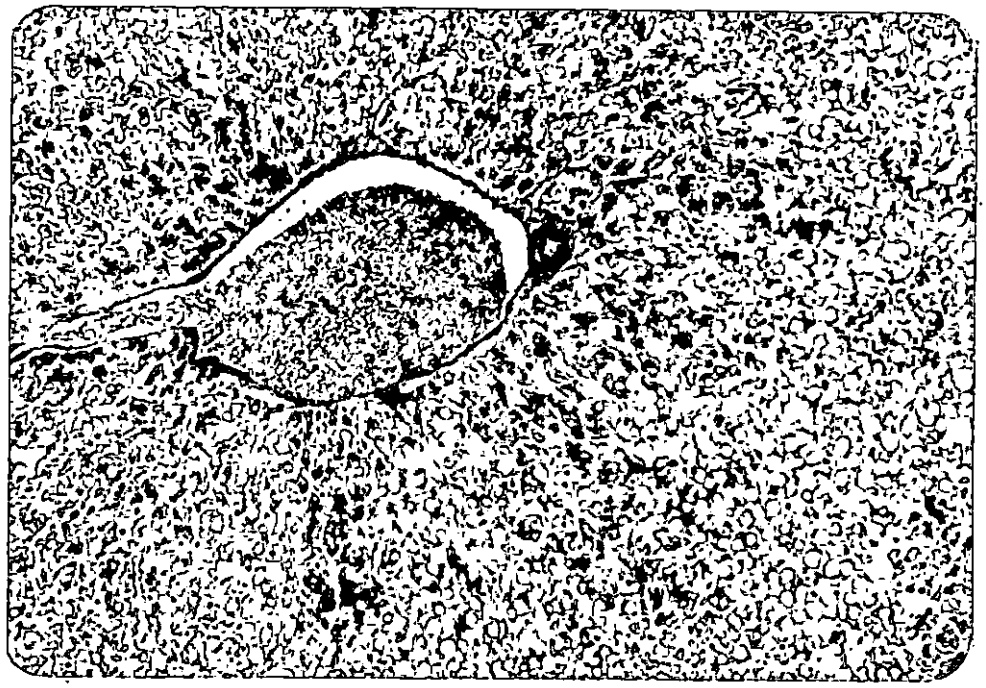
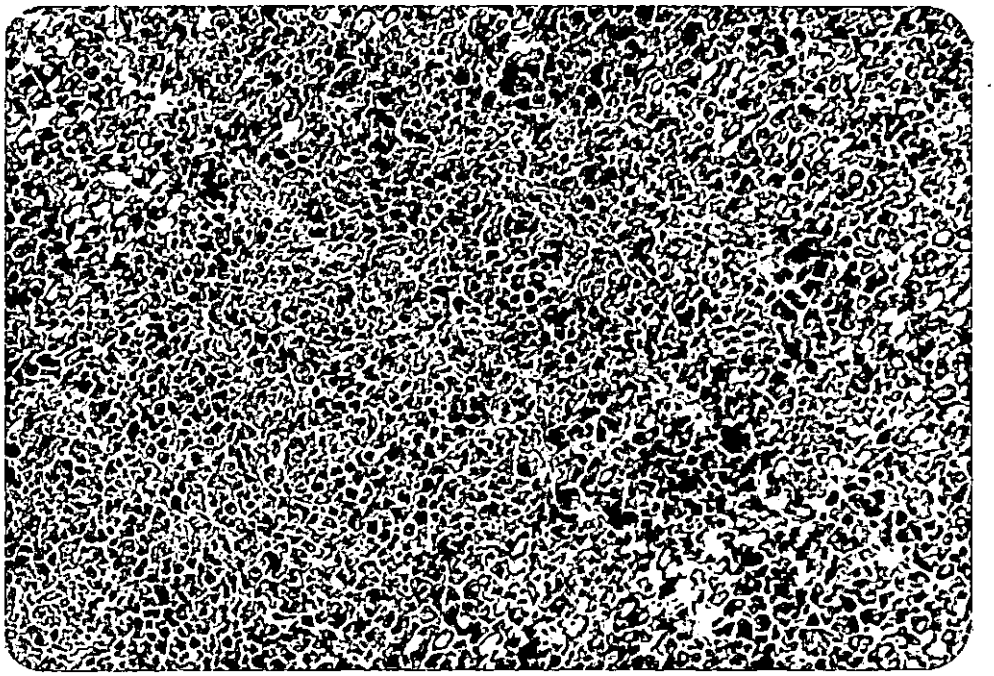


Fig.7. Liver: Subcapsular haemorrhage and Marek's disease.

Fig. 8. Liver: Hepatic steatosis - Diffusely enlarged pale liver.

Fig.9. Liver: Diffuse fatty change and necrosis (H & E x 160).

Fig.10. Liver: Marek's disease and focal fatty change (H & E x 250)



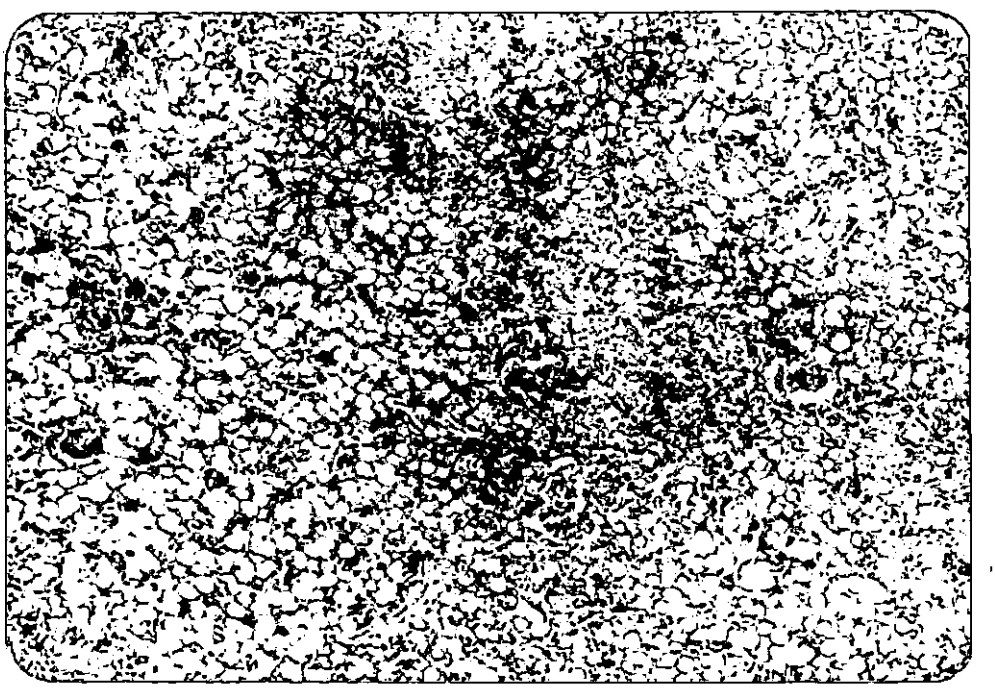
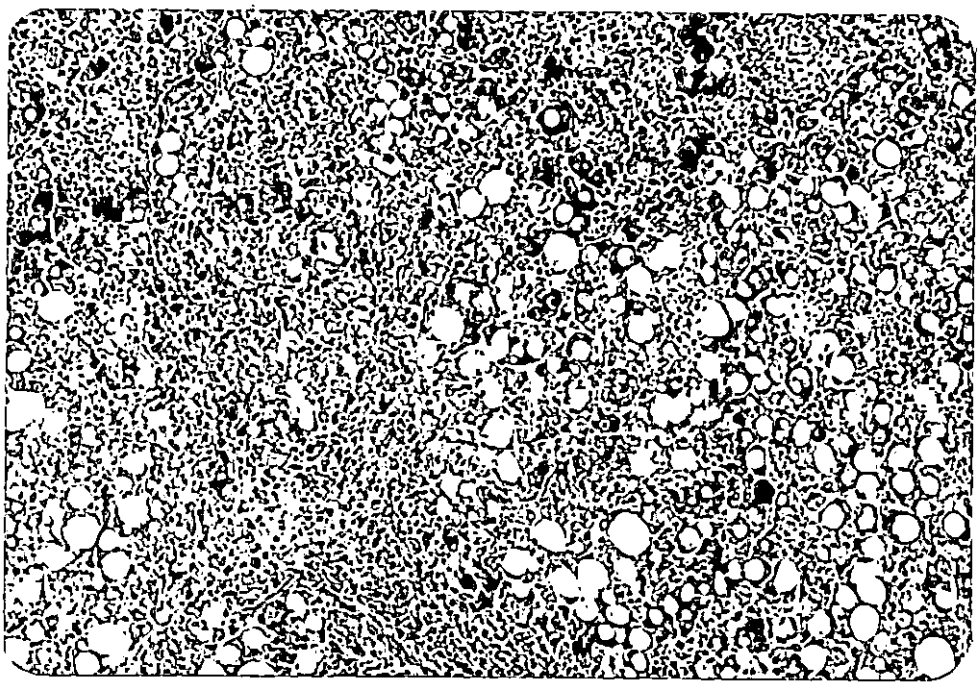


Fig.11. Liver: Focal areas of necrosis and diffuse fatty change (H & E x 250)

Fig.12. Liver: Fatty degeneration and Marek's disease - Fatty cysts are evident (H & E x 250)



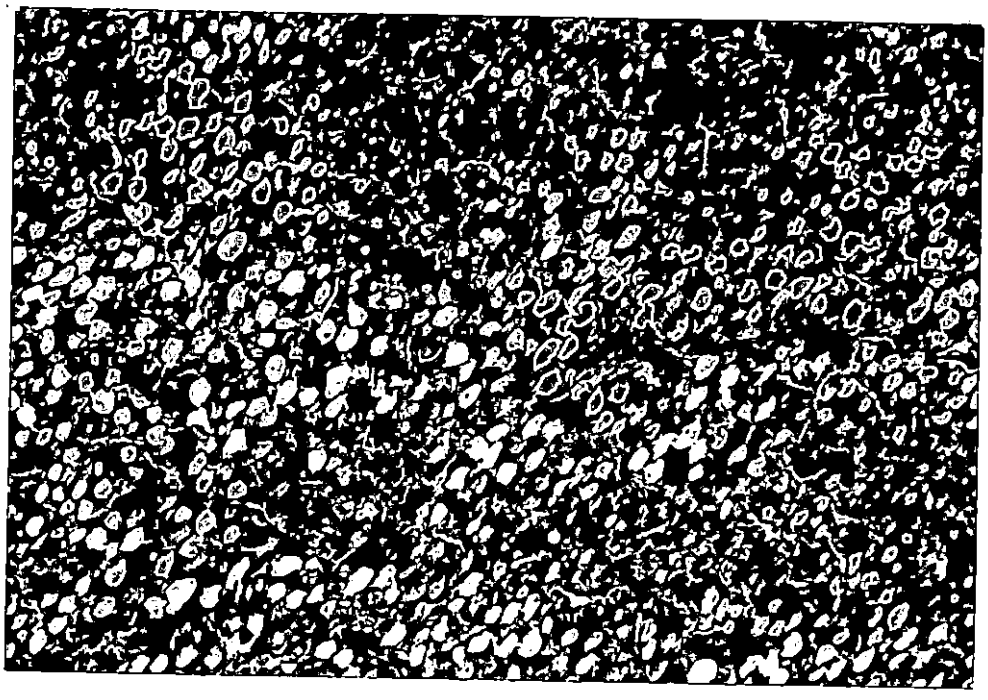
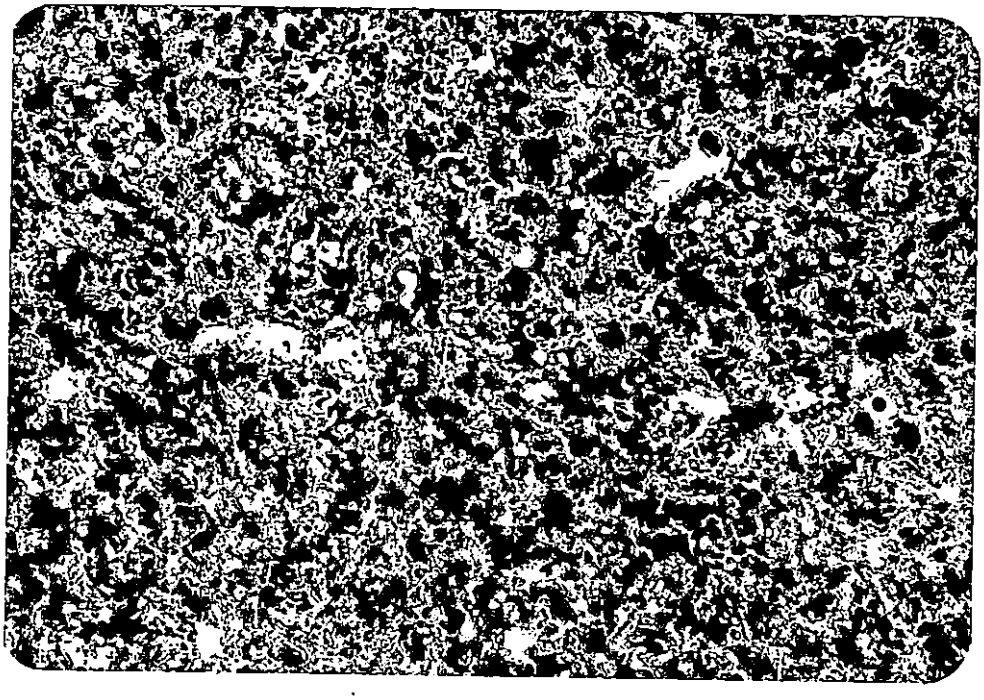


Fig.13. Liver: Hepatic steatosis - Hepatocytes containing fat globules in the cytoplasm(Sudan III&IV x 250)

Fig.14. Liver: Fatty change - Frag mentation of reticular fibers (Gomori's reticulum stain x 250)



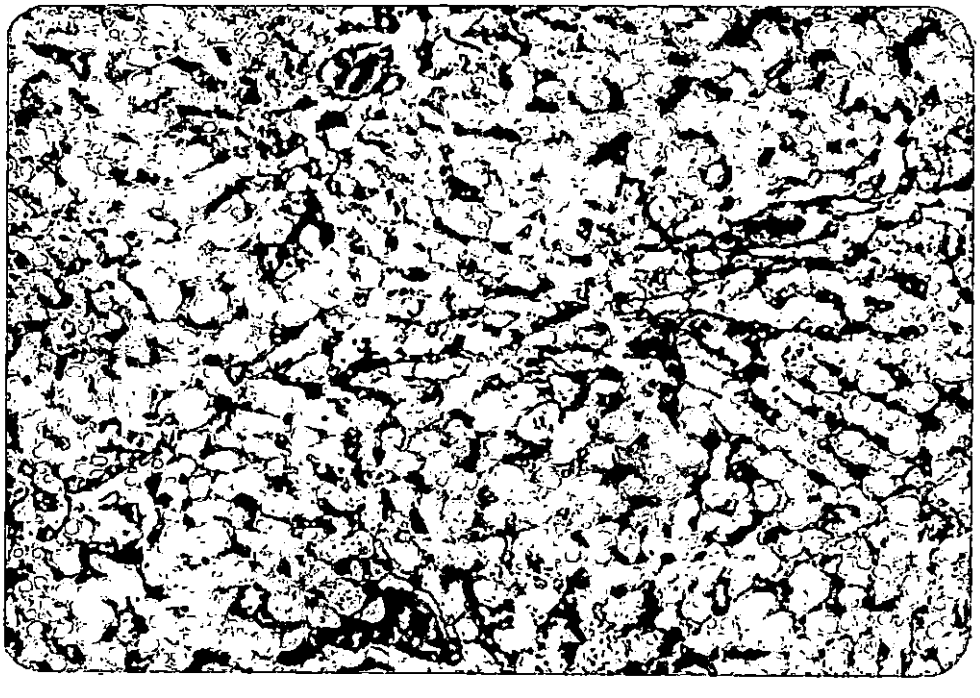


Fig.15. Liver: Fibrinous perihepatitis - A thick band of fibrinous exudate on the surface of the liver (H&Ex160)

Fig.16. Liver:Marek's disease -severe hepatomegaly and patchy pale areas

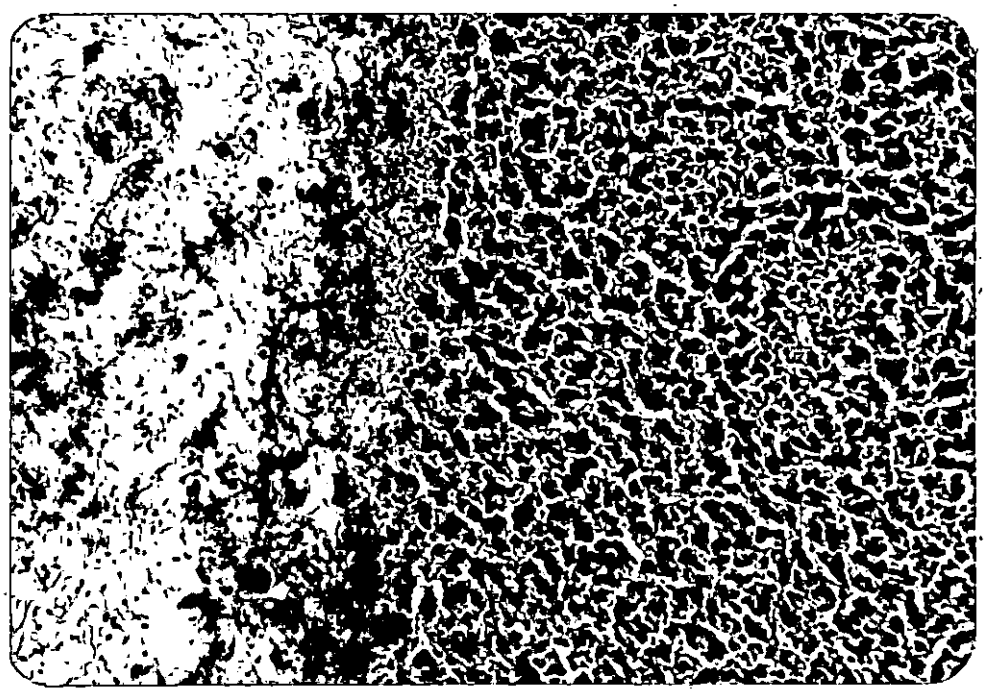
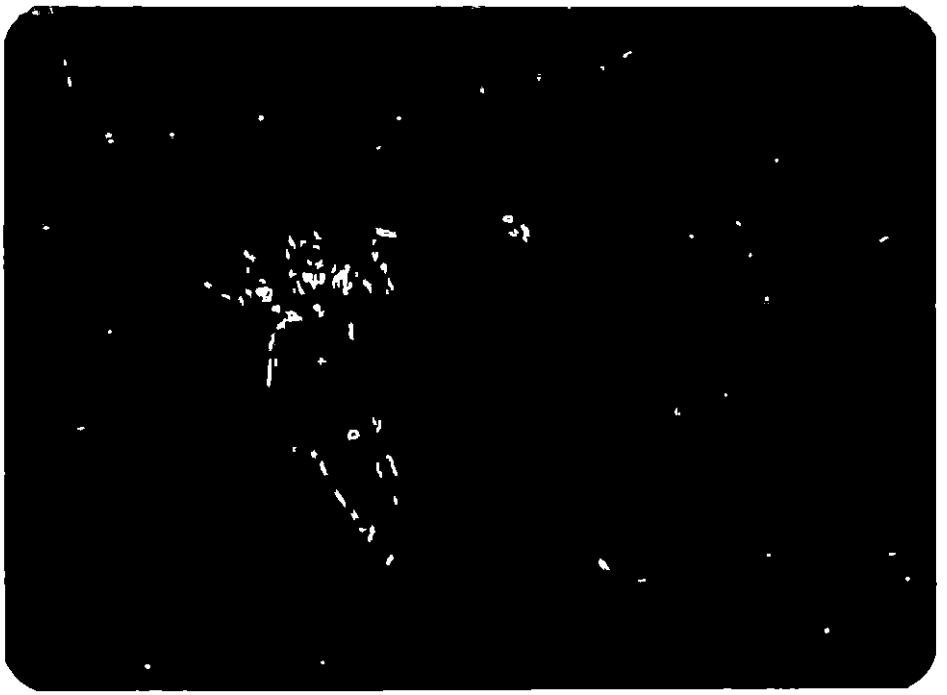
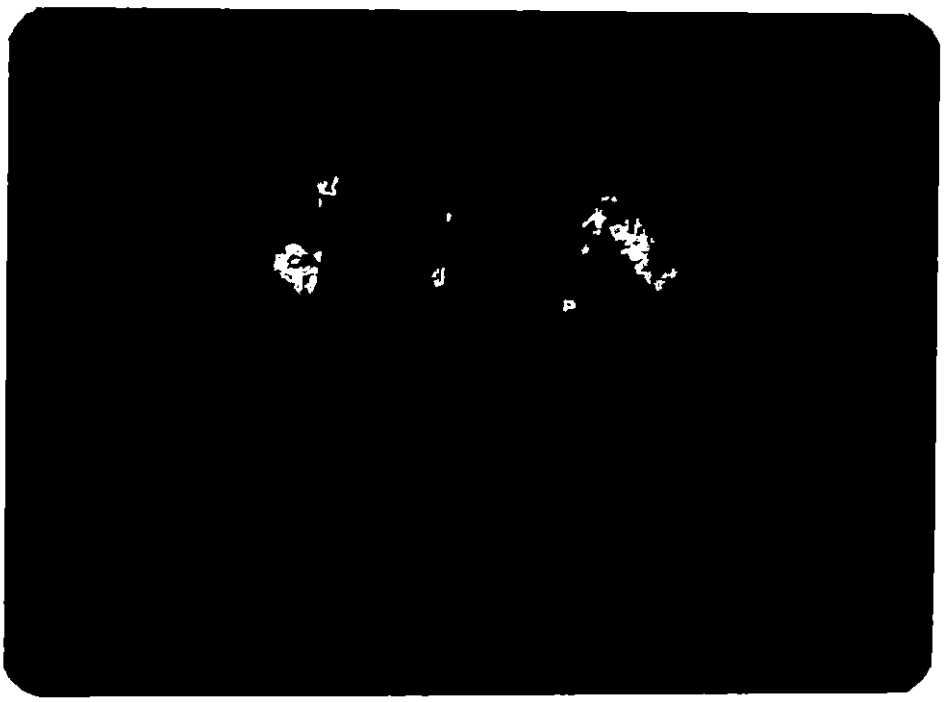


Fig.17. Liver: Marek's disease - diffusely enlarged liver with greyish-white foci and mottling

Fig.18. Liver: Marek's disease - diffusely enlarged liver with greyish-white foci and patches





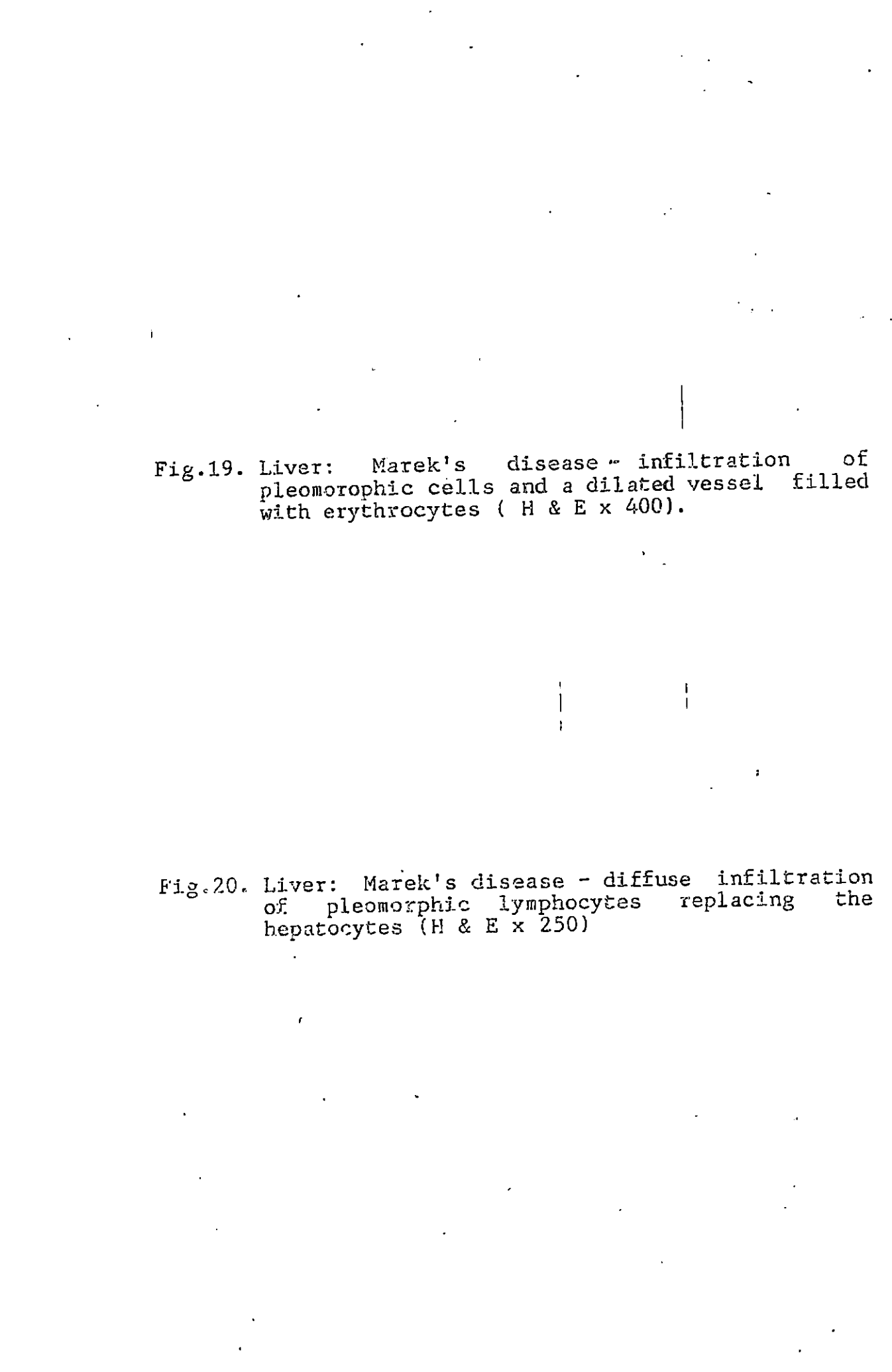
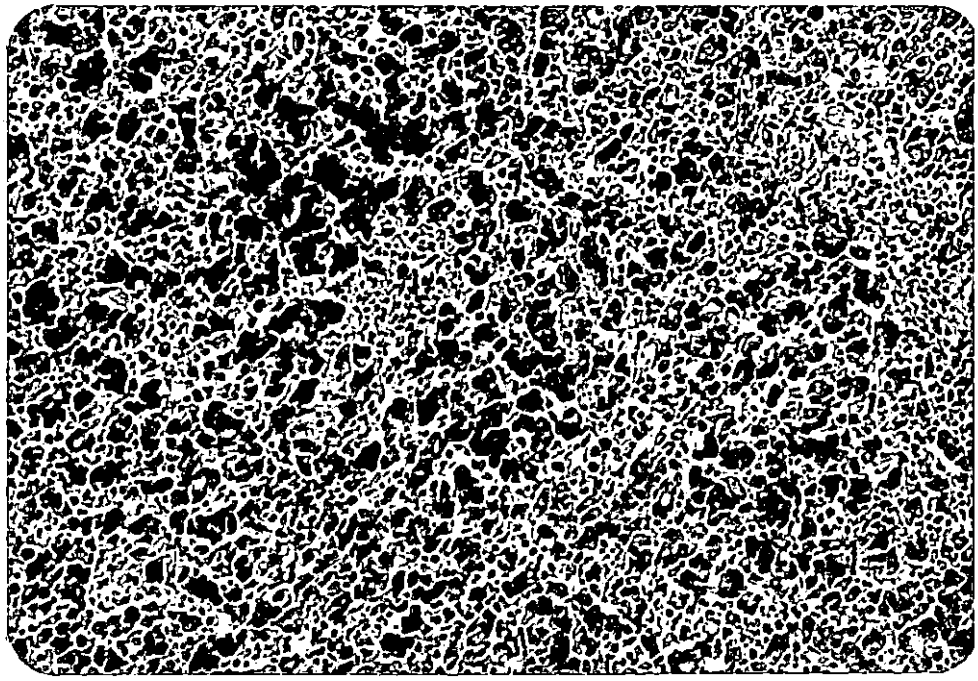


Fig.19. Liver: Marek's disease - infiltration of pleomorophic cells and a dilated vessel filled with erythrocytes (H & E x 400).

Fig.20. Liver: Marek's disease - diffuse infiltration of pleomorphic lymphocytes replacing the hepatocytes (H & E x 250)



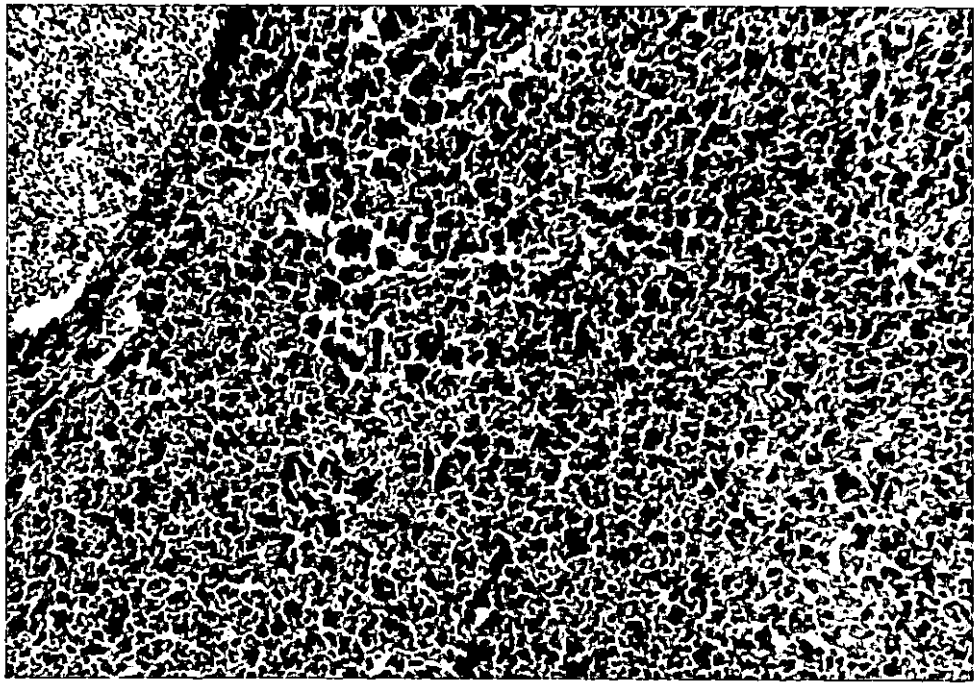


Fig. 21. Liver: Marek's disease - diffuse infiltration with pleomorphic lymphocytes and regenerating hepatocytes (H & E x 250)

Fig. 22. Experimental Salmonellosis : second day - Immunosuppressed quails which are dull and depressed. A quail treated with Salmonella but not immunosuppressed is also seen standing.

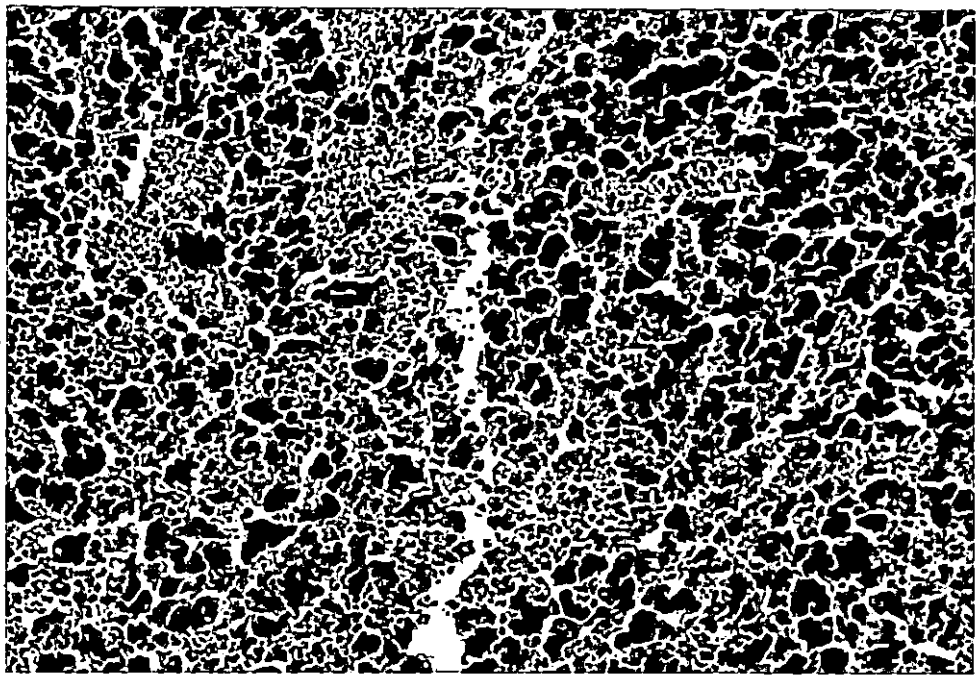


Fig.23. Experimental Salmonellosis: moderately congested liver with numerous pin-head sized necrotic foci.

Fig.24. Liver: Experimental Salmonellosis - circumscribed areas of necrosis and focal areas of infiltration with heterophils (H & E x 250).

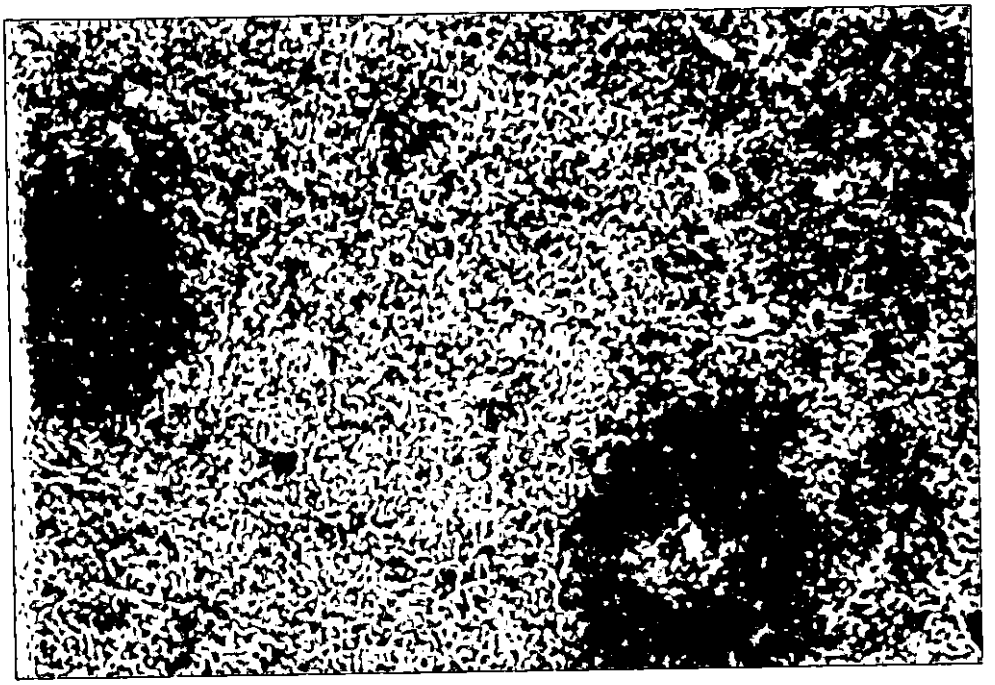
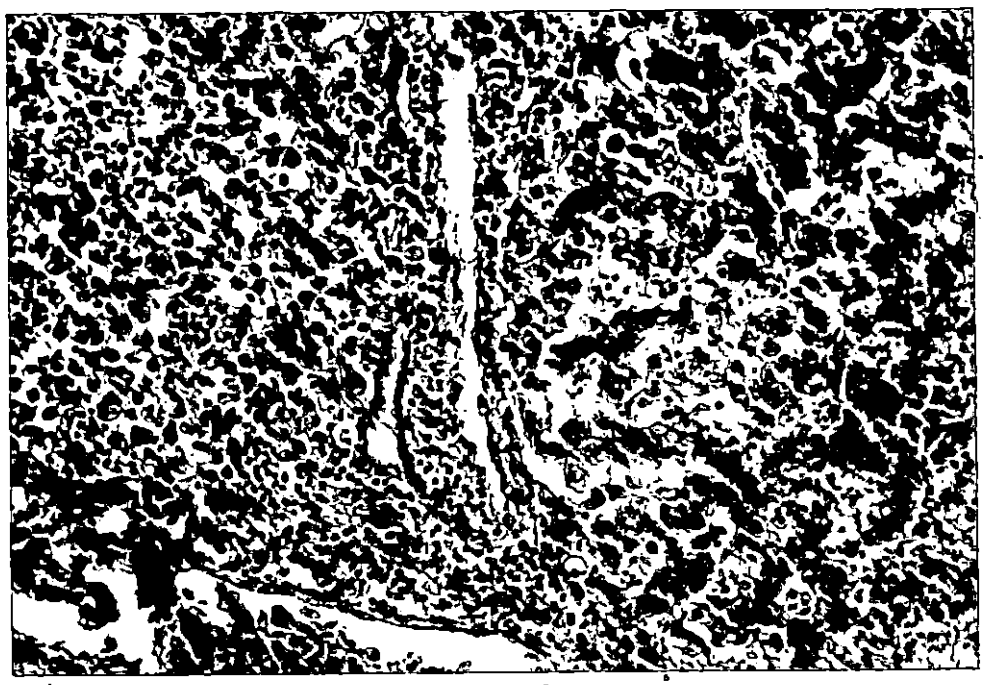


Fig.25. Liver: Experimental Salmonellosis - infiltration of heterophils in the perivascular area (H&E x 400).



PATHOLOGY OF THE LIVER IN QUAILS

(Coturnix coturnix japonica)

By

S. R. KRUPESHASHARMA

ABSTRACT OF A THESIS

Submitted in partial fulfilment of
the requirement for the degree

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University

Centre of Excellence in Pathology
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
Mannuthy - Thrissur

1992

ABSTRACT

A study was undertaken to assess the prevalence and nature of different types of lesions encountered in the liver of Japanese quails.

To study the incidence of hepatic disorders in Japanese quails data were collected from the autopsy records maintained at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy for a period of six years from 1986-1991. The overall incidence of liver lesions was 34.09 per cent. The incidence was high in quails aged above 6 weeks and low in quail chicks.

During the course of this investigation for the period 1990 to 1991, 575 livers with well defined gross lesions were examined for histopathological changes. The different type of lesions encountered were congestion (42.08 per cent), fatty change (30.26 per cent), subcapsular haemorrhage (0.34 per cent), abscesses (0.34 per cent), necrosis (6.78 per cent), Marek's disease (18.43 per cent) and hepatitis (1.73 per cent).

Congestion was the most common lesion observed. The incidence of congestion was 42.08 per cent. It was highest in quail chicks. In adults, in many cases, congestion was

associated with other hepatic lesions whereas in quail chicks, congestion was observed as the only change in most of the cases.

Out of 575 livers examined, hepatic abscesses were seen in two livers. In one liver gross abscesses were seen, where as in the other case abscesses were seen only in histologic sections.

The incidence of fatty change was highest in adults. It was found that fatty change was the significant hepatic response to various toxic conditions in quails. Disintegration of the reticular fibres in case of extensive fatty change was demonstrated.

Subcapsular haemorrhage was seen in two livers. Hepatitis was relatively uncommon in quails. It was recorded in 10 cases out of 575 livers examined.

Marek's disease lesions were noticed in 106 livers. Pathological features of Marek's disease lesions in the liver of quails, were discussed in detail.

S. typhimurium was isolated from three cases of hepatitis. Experimental infection was induced in quails with and without immunosuppression. It was observed that the hepatic lesions were more extensive and mortality was higher in immunosuppressed quails. The possible role of stress in enhancing the pathogenicity in field situations was clarified. The zoonotic importance of S. typhimurium infection was discussed.

Hepatic toxicity of dietary monensin was studied. Hepatic steatosis was the consistent finding. It was clarified that liver is the target organ in monensin toxicity in quails. The practical importance of this toxicity was highlighted.