THE AETIOLOGY AND IMPORTANCE OF SALPINGITIS IN LAYING HENS

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Summary

Acute and chronic bacterial infections of the oviduct (salpingitis) are recorded in most commercial laying flocks and may account for losses of from 1% to 8% over a laying year. Evidence collected over 23 years in laying experiments at the University of New England showed that, out of a total of 12500 hens housed alone in single bird laying cages, none died from salpingitis. Typical commercial losses from vent-peck and salpingitis were recorded in laying hens housed on deep litter (11000 birds) or in three-bird laying cages (48000 birds). The fitting of polypeepers markedly reduced the incidence of vent-peck and salpingitis. From these results it is postulated that salpingitis is an ascending infection of the oviduct following pecking damage to the oviduct by pen or cage mates.

I. INTRODUCTION

Both acute and chronic infections of the oviduct of hens have been recorded from laying flocks ever since mortality records have been kept. The disease appears in nearly all flocks of layers causing losses of from between 2 and 8% over a laying year with a mean incidence of about 4%. The condition has been variously called salpingitis, peritonitis and reproductive breakdown and varies from acute to chronic with characteristic lesions.

In acute cases, post mortem inspection generally confirms that the bird is in good condition, the ovary and oviduct active, and that the bird has obviously very recently been in production. The carcass appears fevered and the breast muscles are darker than normal. The most prominent lesion is marked venous congestion of the oviduct, which may contain small (1 to 4 mm) floccules of yellowish pus. There may be similar floccules of pus in the peritoneal cavity where the blood vessels are also often congested (acute peritonitis).

In the chronic form the bird is usually emaciated with an inactive ovary and the oviduct distended with pus. This may be in fluid form but often occurs as concentric layers of inspissated material. Evidence of a chronic form of peritonitis is often present. The incidence of the different types and degrees of condition varies from flock to flock.

II. METHODS

The laying hens from which the data presented in this paper were derived, were kept at the poultry farm of the University of New England. The objective was to give the birds good practical management as they were part of field demonstrations. These demonstrations, which commenced in 1961, included the production and maintenance of Mycoplasma gallisepticum (MG) free chickens under field conditions, as well as comparing various methods and degrees of feed restriction of the growing pullets between 6 and 18 weeks of age. Some trials included comparisons of breeds and some also compared feeds, feeding regimens and other management practices.

The commercial layer pullets were mostly obtained as day old chicks from various commercial hatcheries in Australia. The layer stock used initially from 1961 to 1970, were principally White Leghorn X Black Australorp crossbreds. Thereafter a greater variety of

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194
breed and strain crosses were available, including White Leghorn X New Hampshire, New Hampshire X Black Australorp and strain cross White Leghorn. The pullets were reared on the floor on litter in a brooder shed to 6-8 weeks of age. They were then transferred to deep litter rearing sheds until 18 weeks of age, when they were transferred to their laying quarters. These consisted of single bird laying cage, (544 birds) or 20 deep litter sheds each housing 50 birds (total 1000 birds). The pullets were reared under natural day length but received 16 hours of light per day when in the laying quarters. From 1972, a new saw-tooth layer cage shed was available, with commercial three birds caged measuring 300 mm wide by 530 mm deep and 425 mm high. This shed housed 4000 birds and the deep litter pens were no longer used after a couple of years.

The chickens received commercial chicken starter diets to 8 weeks of age and then commercial pullet developer diets until transferred to their laying quarters. Commercial or experimental laying diets were fed from about 18 weeks of age. The pullets in the single bird cages received the same diets and management as the birds on deep litter or in the three bird cages.

All dead birds were autopsied to determine the cause of death where possible. The flocks were serologically free from Salmonella pullorum and Mycoplasma gallisepticum. When Marek's Disease vaccines became available in the mid seventies, the pullets were vaccinated at the hatcheries before despatch. Infectious bronchitis vaccines were used at three and fourteen weeks of age when they became available. The pullets were beak trimmed at 10 days of age and again at 14 to 16 weeks of age.

In 1969, in an attempt to reduce the losses from vent-peek and salpingitis, polypeepers were fitted to the pullets in half the pens in the deep litter shed. Polypeepers are plastic devices which prevent forward vision of the birds and are attached by a C clip, that locks into the nostrils of the beak. They were fitted to the pullets 10 to 14 days after the birds had been introduced to their laying quarters and had become familiar with their new environment. In 1973 polypeepers were fitted to half the pullets in the three bird laying cages, care being taken to randomise the treatment groups across the shed.

III. RESULTS

(a) 1961 to 1971

This period involved 5440 hens in single bird laying cages and 11000 hens on deep litter. Over this period total annual losses of about 30% were recorded which were essentially similar to those observed in commercial layer flocks. Losses were consistently lower in the birds housed in the single-bird cages than in those on deep litter. This difference was due to the complete absence of any cases of vent-peek or salpingitis in the birds housed singly. Total mortality on deep litter was 27.3%, with vent-peek contributing 6.5% and salpingitis 3.6%. In the single-bird cages total losses were 19%, with no cases of vent-peek or salpingitis.

(b) 1972 – 1984

This period involved 6500 hens in single bird laying cages and 48000 hens in three bird cages. Overall mortality was dramatically reduced when Marek's Disease vaccines were introduced. Again, no cases of vent-peek or salpingitis were recorded in the birds housed singly. Overall mortality was considerably lower in the single-bird cages (11%) than in the three-bird cages (17%) and was essentially due to the difference in combined mortality from vent-peek and salpingitis.
The fitting of polypeepers to the birds on deep litter and in the three bird cages markedly reduced the incidence of vent-peck and salpingitis mortalities. Total losses in birds on deep litter not fitted with polypeepers, were 21%, with vent-peck contributing 11% and salpingitis 1.0%. Fitting polypeepers reduced these losses to 9.5%, 1.1% and 0.2%. Thus on deep litter, polypeepers significantly reduced but did not entirely eliminate mortality due to vent-peck or salpingitis. In the three bird cages, in birds without polypeepers, total losses were 17%, with losses from vent-peck, 8% and salpingitis 1.1%. Fitting polypeepers reduced these losses to 10%, 1.0% and 0.1% respectively.

IV. DISCUSSION

Over the entire twenty three years not a single case of vent-peck or salpingitis was recorded in the 12512 birds housed in the single bird cages. The 59000 pullets housed on deep litter or in the three bird cages, suffered constant and significant losses from vent-peck and salpingitis. The incidence of vent-peck varied from 2 to 8% over the years on deep litter and in the three bird cages. This variation was possibly in part due to the varying skills of the persons carrying out the beak trimming. However some strains of birds had consistently high mortalities and others consistently low mortalities from vent-peck. The incidence of salpingitis mortality was typically between 10 and 50% of that due to vent-peck. On two occasions, however, when vent-peck losses were low (3.2 and 3.8%) the losses from salpingitis were comparatively high, 2.6 to 2.4% respectively. The overall incidence of salpingitis and vent peck mortality was markedly reduced when polypeepers were fitted.

Taking all this evidence into consideration, i.e. the complete absence of salpingitis if vent-peck is eliminated and the reduction in salpingitis if vent-pecking is reduced, it is postulated that salpingitis is an ascending infection of the oviduct following pecking damage to the oviduct by pen mates. The course of the infection that follows varies and is probably due to the degree of damage and contamination of the wounded area. This idea was first proposed by Cumming (1974) and more recently a possible correlation between vent-peck and salpingitis was suggested by Jordan and Pattison (1996).

Losses from vent-peck are one of the major problems in modern laying hens, particularly in the single layer Californian style sheds widely used in Australia which have such a high light intensity. To this loss we can now add the deaths from salpingitis, at roughly one third the number of birds dying from vent-peck. Birds dying from salpingitis may succumb in a few days but, in chronic cases, the birds may survive for up to 150 days (Gross and Siegel 1959). These chronic cases will produce no eggs but will eat food for several weeks and even months thus further increasing the loss to the egg producer.

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REFERENCES