A PRACTISING VETS APPROACH TO THE HIGH CELL COUNT HERD

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SUMMARY

Dairy farmers currently use a variety of approaches to dealing with a high bulk tank cell count. Simply they may only consider each case of clinical mastitis individually, have some form of compliance with the five-point mastitis control plan or increasingly adopt a plan of strategic management. The cattle vet has a major role to play in the more sophisticated approaches need to plan mastitis control and to manage milk quality. The main component of the approach must remain the five-point mastitis control plan but with stricter attention to limiting the duration of infections and targeting of treatment for new infections. The vet now has many tools to deploy from obtaining good data to help understand the farm and its problems, easier identification of causative bacteria and introduction of a preventive medicine approach as part of a cattle health plan tailored to the individual farm.

INTRODUCTION

Udder health is central to profitable milk production. Not only are there significant losses from poor udder health but there are now significant penalties for failing to achieve particular levels of cell count. In recent years, the somatic cell count target levels have reduced and many producers are now penalised at levels as low as 150,000 cells/ml. Ten years ago many of many clients were comfortable with cell counts of 400-600,000 cells/ml. In 1990 33% of the national herd had average herd cell counts greater than 400,000 cells per ml. In October 1991 a penalty of 0.2 p/l was imposed on producers exceeding 700,000 cells/ml and a bonus was payable of 0.2p/l for milk quality better than 400,000 cells/ml (1). This improved milk quality and 2 years later only 26% of producers were producing milk with a bulk supply cell count above 400,000 cells/ml. (2). The penalties increased to 0.5 and 2.0 p/l for exceeding the thresholds of 400,000 and 1,000,000 cells/ml respectively. By 1996 the Milk Marque penalty for milk exceeding 400,000 cells/ml had increased to 2.0 p/l (3). Since introduction of the European Milk Directive, 92/46/EEC, milk with a cell count greater than 400,000 cells/ml is not acceptable for human consumption. The current penalties have now increased further with market place split between first purchasers pursuing a low somatic cell count milk with penalties ranging between 0.8 p/l to 1.8 p/l for slipping from 150,000 cells/ml to greater than 200,000 cells/ml. Other purchasers have a more lenient view on somatic cell count penalties and typically the penalty loss from below 250,000 to 350,000 cells/ml would be 0.2 p/l. Nearly all first purchasers levied a penalty of 1-10 p/l for somatic cell counts greater than 400,000 cells/ml. With this economic background the control of udder health has never been more important and the margin for error is minimal. The aim of this paper is to give a practising vet view of udder health control within the current standards and marketplace.

THE PROBLEM OF CONTROLLING UDDER HEALTH - A VETS VIEWPOINT

Not all clients will telephone the practice to seek advice when they develop a high cell count problem in the herd. Many farmers are not willing to accept defeat and often persevere with
managing a disease problem alone before eventually calling for help. All my clients have access to bulk cell counts and the majority will use individual cell counts on a monthly basis.

Mastitis has, typically, been poorly recorded in the past and more than half of the more keen dairy farmers have not had a system in place to record mastitis incidence and treatments. This is now changing and with the introduction of the BCVA Herd Health plan (3) and National Dairy Farm Assurance schemes this will be largely rectified.

The consequence of poor mastitis control and recording can be the development of a “shoot or treat” mentality to udder health control. The cowman, when presented with a problem cow, makes an “urgent” decision to treat the immediate problem on a case by case basis. Rarely is there a more measured approach to prevent future cases in the herd. This is not unique to farmers and the vet may also fall into the trap of treating the urgent clinical case and not initiating a proper preventive investigation due to pressures of clinical work.

The incidence of mastitis varies enormously within my client base with the annual incidence varying between herds from 5 to 100 cases/100 cows/year. In all these herds the bulk cell count will be less than 400,000 cells/ml. In some of the problem herds the overall bulk milk cell count is managed by increasing the cull rate by “brinkmanship culling” i.e. culling out high cell count cows with a high individual contribution to the bulk tank or not putting the cows’ milk into the bulk tank. In other herds the cell count may not be an issue as the problem is one of high incidence and low cell count. In my experience culling criteria change with the level of the bulk cell count and clients, when asked “what criteria you use to cull cows”, often answer that “it depends on the herd cell count”.

Good udder health depends on performing 20 or so small tasks correctly every day 365 days a year. Some farm systems achieve these standards easily. On others as litres sold per man and cow susceptibility to new infections increase maintaining udder health to current standards can be problematic.

**A TYPICAL AD HOC APPROACH.**

In the majority of herds the main method of mastitis control remains the 5-point plan. This is applied with varying degrees of efficiency and success. Even 30 years after the plan was devised it is not uniformly understood. Multiple use udder cloths are still used in some herds and these clients can be difficult to convince of the benefits of the 5-point plan. In herds with diligent stockmen and well-maintained housing and plant udder health is not a major problem. However, achieving a consistently low bulk milk cell count, below 150,000 cells/ml, is a challenge as a single missed case of clinical mastitis may put the herd bulk milk SCC above the bonus band. High levels of culling and treatment can become the norm rather than be seen as broadly unacceptable and requiring of investigation. This is largely due to a conspicuous failure of the dairy farmer to understand the true costs of mastitis and a false perception of high costs of investigation (typically around 0.02 p/l compared to the financial losses of more than 0.5 p/l).
Treatment of clinical and sub clinical cases is usually based on previous responses to a particular antibiotic preparation or historic bacteriological investigations and seldom based on individual culture on a planned basis.

Currently most intramammary tubes are licensed to be used as a short duration treatment with the aim to reduce milk losses rather than produce a high cure rate during lactation, especially to pathogens such as *Streptococcus uberis* and *Staphylococcus aureus*.

Increasingly sub-clinical cases may be treated based on individual quarter or cow cell counts and/or California Milk Tests without reference to bacteriology on grounds of expense. Again the focus can be over reliance on therapy rather than prevention.

**THE STRATEGIC APPROACH**

In order to tackle a problem cell count herd the 5-point plan is still the main basis of the investigation. The broad aims are:

1. Prevent new infections. Rigorous implementation of the 5-point plan and attention to environment.

2. Remove existing infections from the herd/bulk tank. This is achieved by culling, early drying off, or removing the cow from the milking herd, for treatment or to suckle calves.

3. Treat existing infections more effectively. Improved lactation and dry cow therapy. Both 2 and 3 will reduce the exposure to infection of susceptible cows and in turn reduce the new infection rate.

**THE TACTICS**

The method of investigation has been covered in more detail in previous papers (5,6,7,8,9) The approach I use is via a questionnaire and the current one in use was developed by five of us in conjunction with Pfizer Animal Health (10).

1. Establish the extent of the problem. This is based on a study of mastitis records, intramammary tube usage and cell count data. The records, combined with knowledge of the farm, will often give strong indications of the areas of weakness.

2. Identify the pathogens. This is central to success. Unless the main pathogens are identified, the strategy cannot be implemented.

3. Perform an in-depth investigation on farm, based on discussions with the farmer, milker and observations at milking.

4. Discuss the results with the farmer and herdsman and agree a list of recommendations that will be applied.
5. Review and monitor progress. Encourage compliance with recommendations on an ongoing basis.

DIAGNOSING THE BACTERIA INVOLVED IN ELEVATING CELL COUNTS: PRACTICAL CONSIDERATIONS

- Bacteriology of individual quarter samples of all cows having a persistent monthly cell count more than 200,000 cells/ml is the best way to determine the causative bacteria. In many instances this is prohibitively expensive in large herds or herds with high numbers of cows with a high cell count. Complications come if many infections more than one type of bacteria. Intermittent excretion of bacteria can complicate accurate diagnosis when using single samples.

- The numbers can be reduced by selecting the highest 10 cows, or the 10 cows with the most recent new sub clinical infection (i.e. an increased in cell count less than 200,000 to more than 200,000 cells/ml) or by using the CMT test to identify individual quarters.

- Bacteriology of bulk milk samples may give an indication of the organisms present in the herd. Although this may seem to be a “broad brush” approach it has the advantage of screening the whole herd. The results will not allow an action plan for each cow more an overview of the key organisms involved. One advantage of the bulk milk examination is that it provides useful information plant cleaning and general levels of organisms in the milk (11).

- Bacteriology of samples from the ten most recent clinical cases, frozen if necessary and submitted to the lab in bulk, is often most useful. The clinical cases may not always be representative of the bacteria causing the elevated cell counts as low grade infections by some bacteria may not feature as causes of clinical mastitis. Bacteriology on 3 samples or any problem clinical case may not be representative. Determination of the bacteria involved will help to identify appropriate treatment protocols (12,13).

COMMON PITFALLS OF AN INVESTIGATION

These are some of the key difficulties that I have experienced when investigating problem herds. This is not comprehensive list.

- **Do not jump to conclusions.** The clinical presentation of mastitis may be different to the cause of high cell counts in the herd. The severe *Escherichia coli* mastitis cases are obvious and the assumption may be made that the key organisms involved in the sub-clinical mastitis are environmental. This may not be the case. A fuller investigation is necessary.

- **What is mastitis?** Carefully discuss with the milker what are his criteria for diagnosing mastitis and what treatment protocol he applies. This may be different to what you and or the farmer think it is. In some instances the change of incidence of mastitis in the herd is more a reflection of a change in criteria of case selection rather than a change in incidence. A milker can become sensitised to the occurrence of clinical mastitis and embark on a campaign of
treatment which will effect results. There may be no system in place to diagnose mastitis save for visual examination of the udder, which has its limitations.

- **Check the tube usage against the clinical records.** This will give an indication of tubes used per case and is relatively easy to do.

- **Clarify the culling policy.** This policy can be an absolute muddle on some farms, ranging from culling on the basis of one or two elevated cell counts to an absence of any culling policy. The policy is often fluid and varies with the number of replacement heifers available rather than rigid scientific criteria. More use of bacteriological cultures prior to deciding to cull may aid the decision process.

- **Liner changes.** Commonly on problem herds the milking machine is taken for granted and there is often not a system in place to ensure that liners are changed at the appropriate interval. Even if the liner life is calculated and agreed putting a system in place to carry out the changes at the required frequency is often difficult. Milking the liner “till it splits” is not the ideal option!

- **There is no substitute for observing milking.** This gives a general overview of what the milking routine and approach is like. Partial teat dipping and spraying is the norm rather than the exception and in many herds the criteria appears to be if there is a drip of teat dip on the end of the teat this is enough. Covering the whole teat per se may not be seen as part of the procedure. Checking the volume of teat dip or spray used is useful.

- **Dry cow therapy.** Establishing how this is done is essential. This can be very rough and ready and the concept of bacteria being introduced at drying off is difficult to explain. Just checking that DCT is in use is not enough.

- **Producing a report.** Written reports and then a verbal follow up is essential. If you are asking someone to change the habits of a lifetime, then you must carefully explain the benefits of the change and why they are necessary. Writing down the recommendations and methods will emphasise the points but is no substitute to discussion.

- **Defining expectations.** Do not promise success quickly. The speed of response will depend on the cause of the problem, the solutions available and the diligence with which they are applied. The aim is not to go for a quick fix but a lasting solution.

- **Encouragement and follow up.** Giving ongoing guidance is part of the solution.

**BACTERIOLOGY OF SUB CLINICAL AND CLINICAL CASES - AN OBSERVATION IN A HERD**

The herd had 380 cows calving all year round, housed on straw yards, an averaging 7500 litres. The straw yards were introduced 2 years earlier to a building designed for cubicles. The straw yards were overcrowded. The rolling bulk tank cell count was 315,000 cells/ml. There was a rapid increase in the incidence of clinical mastitis, to more than 100 cases in 100 cows.
Table 1. Results of clinical cases sampled for culture March – June 2000

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>% Quarters</th>
<th>No. Quarters</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus uberis</em></td>
<td>67</td>
<td>21</td>
</tr>
<tr>
<td>CNS</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>Bacillus sp.</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td><em>Corynebacterium bovis</em></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><em>Escherichia coli</em></td>
<td>16</td>
<td>5</td>
</tr>
<tr>
<td><em>Streptococcus dysgalactiae</em></td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Totals</td>
<td></td>
<td>31</td>
</tr>
</tbody>
</table>

Initial investigation of 12 clinical cases of mastitis revealed *Str. uberis* in 10 cows, CNS in 4 cases and *Staphylococcus aureus* in one case. The herd was chosen to be involved in a trial run by Mo Milne and Andy Biggs to evaluate the use of pulse therapy on failed initial treatment of clinical mastitis caused by *Str. uberis*. Sampling of clinical cases continued and the results suggested a problem caused by *Str. uberis* (Table 1). A further screen of 113 cows with individual cell counts more than 200,000 cells/ml was performed to establish the pathogens isolated from 452 individual quarters (Table 2).

Table 2. Results of a screen of 113 cows, quarter samples taken from cows with cell counts above 200,000 cells/ml.

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Cows Number</th>
<th>Cows %</th>
<th>Quarters Number</th>
<th>Quarters %</th>
<th>Quarters per cow</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>2</td>
<td>1.8</td>
<td>2</td>
<td>0.4</td>
<td>1.0</td>
</tr>
<tr>
<td>CNS</td>
<td>69</td>
<td>61.1</td>
<td>197</td>
<td>43.6</td>
<td>2.9</td>
</tr>
<tr>
<td><em>Streptococcus uberis</em></td>
<td>60</td>
<td>53.1</td>
<td>94</td>
<td>20.8</td>
<td>1.6</td>
</tr>
<tr>
<td><em>Streptococcus dysgalactiae</em></td>
<td>29</td>
<td>25.7</td>
<td>40</td>
<td>8.8</td>
<td>1.4</td>
</tr>
<tr>
<td><em>Corynebacterium bovis</em></td>
<td>41</td>
<td>36.3</td>
<td>70</td>
<td>15.5</td>
<td>1.7</td>
</tr>
<tr>
<td>Coliforms</td>
<td>6</td>
<td>5.3</td>
<td>6</td>
<td>1.3</td>
<td>1.0</td>
</tr>
<tr>
<td><em>Streptococcus faecalis</em></td>
<td>40</td>
<td>35.4</td>
<td>67</td>
<td>14.8</td>
<td>1.7</td>
</tr>
<tr>
<td>Bacillus spp.</td>
<td>59</td>
<td>52.2</td>
<td>114</td>
<td>25.2</td>
<td>1.9</td>
</tr>
<tr>
<td>No growth</td>
<td>15</td>
<td>13.3</td>
<td>20</td>
<td>4.4</td>
<td>1.3</td>
</tr>
<tr>
<td><em>Streptococcus agalactiae</em></td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

It is interesting to note the relatively low prevalence of *Str. uberis* from the quarter samples compared to the clinical incidence (20% cases versus 67% quarter isolation rate) and the absence from the 31 cows sampled for clinical mastitis of *Corynebacterium bovis*. *Streptococcus dysgalactiae* occurred in 26% of the quarter samples from sub clinical cases and only 6% of the clinical cases. Coagulase negative staphylococci were mainly associated with sub clinical cases.

Care must be taken in assuming that the pathogen isolated from the clinical sample is representative of all the bacteria present in the udders. Many types of bacteria appear to be
present at a sub clinical level and depending on the bacteria may contribute to the overall bulk tank SCC. This neatly illustrates the difficulty of identifying the causative organism and there is a potential in some herds for the bacteria such as *C. bovis* and CNS to be cause of cell count problems at the bulk tank level of 150,000 cells/ml.

**CONCLUSIONS**

The control of udder infections using the 5-point mastitis control plan is as relevant today as it ever has been. The targets have changed and lower cell counts are now expected. The increasingly high standards of milk quality and medicine usage that farmers have to achieve require complete adherence to the mastitis control strategies.

The key issues vets and farmers need to address, are:

- Increasingly sub clinical udder infections will cause significant financial problems for the farmer as even low-grade may jeopardise the milk price bonus payable. Treating clinical mastitis alone may not always be sufficient and treatment of sub clinical infections may be required in some circumstances. There are new questions being raised on how low cell counts should be, the possibility of an increase in susceptibility to more severe mastitis in low cell count cows, and if there is an optimal balance between any target SCC and the likelihood of increased antibiotic usage in the milking cow.

- To achieve the higher standards being demanded, a higher level of management and attention to detail is required and given these difficult financial times this can be difficult to achieve as more output per man is expected, for no increase in income to deliver any capital investment necessary. Although the 5-point plan has been in existence for 30 years it can not be assumed that all farmers know the importance of the 5-point plan in detail.

- As sub clinical infections become more relevant financially to the farmer the use of the California Milk Test, individual cow cell counts and quarter cell counts become more relevant in the approach to udder health. Early detection of udder infection may be more important than visual changes in the milk. This is currently difficult to achieve. The farmer needs a better guide as to the SCC of the milk before it enters the tank. This is not easily achieved either.

- More effort should be spent in the investigation of problem herds aiming to prevent new infections rather than simply coping with the consequences.

- If treatment and culling are required they should be based on scientific approaches and involve bacteriology and detailed veterinary advice rather than as responses to an empirical cell count.

- Treatment and culling should be seen as failure to prevent rather than accepted as the main part of mastitis control strategy in some herds. These control measures are only part of the 5-point plan.
More sampling of cows to identify pathogens will aid not only in diagnosis but also in decision making for the individual cow. For instance improved cure rates with augmented dry cow therapy with Tylosin in cows infected with Str. uberis and S. aureus (14) have been described. A proportion of cows identified for culling on the basis of SCC alone might be saved if more effort on correct diagnosis was applied. It is a maxim that 3 cases in one quarter is a criterion for culling. A more sophisticated approach may reduce wasted culls.

The whole area of disease control should move from treating the urgent case to developing a more lasting preventive approach, which is much more profitable and essential in the increasingly competitive market place. This will only occur with vets and farmers working closer together to achieve these aims.

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REFERENCES