KEARNEY v PAUL 1983/ 1059 P. 219

THE HIGH COURT

JOHN KEARNEY

.v.

PAUL AND VINCENT LIMITED

Judgment of Mr. Justice Barron delivered the 30th day of July 1985

The Plaintiff is a married man with three children who farms approximately 100 acres in County Limerick. The Defendant is a manufacturer of animal foodstuffs. The Plaintiff claims damages for loss sustained by him as the result of feeding calves an acidified milk replacer marketed by the Defendants under the name Prolong Acidified Cream Food.

In the year 1977 the Plaintiff commenced to rear calves on a commercial basis. In that year the calves until weaned were fed by their mothers and the Plaintiff met with no unusual problems. In the following year 1978 the Plaintiff bought in 81 calves at between 3 to 5 weeks old to be reared over that season and the next and to be sold at approximately 1½ years of age in the autumn of 1979. The method of rearing employed by the Plaintiff was to keep the calves in pens for approximately 6 weeks during which period they were fed on milk replacer and gradually weaned until they were between 9 and 11 weeks old when they were let out to the fields. In the winter months they would be brought in and fed on silage and let out again to the fields in the spring. He met with no unusual problems but lost 6 of these calves through natural causes which was within an acceptable limit of loss.

In the year 1979 the Plaintiff bought in 85 calves for rearing with the intention of keeping them for a similar length of time as with his 1978 calves. As his wife was pregnant he did not have the same help with the calves as in the previous year and instead of using a milk replacer served warm in buckets to the calves he

changed over to acidifed milk replacer because this was served cold and involved considerably less work. The Defendant in line with its competitors had brought to the market that year a milk replacer which was fed to the calves on an on demand basis. The replacer was mildly acidified which meant that when mixed it would stay fresh for up to three days. This meant that it did not have to be fed in buckets but could be stored in containers and taken by the animals as they wanted it. They got it from two teats inside their pens which were attached to tubes leading to the containers in which it was stored.

The calves were kept in pens of 12 and were fed from two teats in each pen. In addition, in the course of weaning they received calf pencils, nuts and hay. The calves did not thrive as in the previous years. While some did alright, the majority did not. When they had been in the pens about three weeks, the Plaintiff drew the attention of one of the Defendant's representatives to the condition of the calves. At this stage as with conventional milk replacer, the Plaintiff was not using the product at full strength but at three quarter strength. The Defendant's representative pointed out that he should have been providing the product at full strength but did not otherwise make any other suggestions. The Plaintiff from then on used the Defendant's product at its recommended strength.

After the calves were put out to pasture, those which had not been thriving continued to do badly. In all the Plaintiff lost 16 of that season's calves, 7 in the first two months and the balance over the following nine months. He sought the advice of his veterinary surgeon who advised immunisation against septic pneumonia which was carried out. During that winter the calves were fed on silage and nuts, but stayed generally in poor

condition, some of them exhibiting signs of lameness, a complaint from which the previous year's calves had not suffered. Accordingly, he decided to sell the entire herd which he did.

In spring of 1980 the Plaintiff started again, this time he bought in 135 calves which he housed in pens of 19. He found the same problems as in the previous year. Essentially the majority of the animals did not thrive properly. A couple who would not feed from the teats were fed with conventional milk replacer from buckets and did thrive.

The results in 1980 were worse than in 1979. The numbers which were doing badly were substantially up on 1979. It is not necessary to go into any great detail in relation to the history of this herd. Sufficient that the Plaintiff became worried and sought to find a solution for the problems which he was meeting. Two animals which died in May 1980 were sent to the Regional Veterinary Laboratory for a report. One was found to have died from virus pneumonia, the other from salmonella. The Plaintiff while he did not contest these findings nevertheless doubted that these two diseases were prevalent in his herd. He said that coughing and scour which would have been the outward signs of such complaints were at a low level. He instanced the fact that there was a prevalence of black droppings which he took to be a sign of internal bleeding. The Plaintiff became convinced that there was something in the Defendant's product which was damaging his animals. In July or August of that year he made this allegation to the Defendant's representative and indicated that he would not use its product again.

The Plaintiff lost 29 calves in all during the first months of that season but many of the calves which survived had not thrived properly and were clearly underweight for their age.

Another carcass was sent to the laboratory in November and the animal was found to have died from enteritis. The animals when put on silage that winter were also fed nuts, given supplementary proteins and treated for fluke and worms. It was found that after some weeks many of the animals became lame and their hoofs and joints became infected. The condition of such animals was considerably worse than the condition of the animals similarly affected the previous year. He sought advice from his veterinary surgeon and as a result blood samples were sent to the laboratory for analysis. These showed low levels of calcium though the cause was not ascertained. When the animals were put out to pasture in the spring the calcium level rose again and tests in April showed them to be normal. The calves that did well, however, continued to do well and calves which he bought in to replace those that had died also did well. As a result of the drop in calcium levels and the fact that the animals did worse when fed on silage the Plaintiff believed that the Defendant's product was the cause of his troubles. He associated the trouble with increased acidity in the stomach brought about in the first instance by the acidity in the milk replacer and secondly by the acidity in the silage. He took the view that in some way the animals were unable to cope with increased acidity and were unable to neutralise it.

In April 1981, he sought to impose liability on the Defendant.

On the 6th April, 1981 he wrote to the Defendant as follows:

"Dear Sirs,

For the past two years, I have been using your acidified milk replacer - Prolong - to rear calves, and in those two years I have incurred substantial losses together

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"with many stunted survivors.

After exhaustive veterinary investigations over 1979/80 it was only discovered recently by the veterinary laboratory in Knockalisheen that a substantial number of the surviving calves were calcium deficient.

According to the agricultural experts I have been in contact with and they are in agreement that this deficiency was caused by the milk replacer or the calf nuts - both of which were supplied by you.

I wish to point out that my losses are in the region of £16,000 and I will be pressing for compensation.

I contacted your Kilmallock branch on the 16th March and on the 20th a representative called and I showed the problem to him. He was to arrange for an expert to call to see the calves but I have heard nothing since.

Yours faithfully".

A reply was received from Mr. Tim O'Riordan the Defendant's Technical Services Manager dated the 21st April, 1981 which was as follows:-

'Dear John,

Thank you for your letter which I received recently.

I note the contents of your letter of April 6th and I am very much aware from our discussions on your farm that you suffered severe losses in your calf enterprise.

I want to thank you for your courtesy in showing me around your farm and giving me the opportunity to see your cattle.

The Company is giving your problem careful consideration and we will be in contact with you within the next few weeks.

Wishing you every success in your farming in the future.

Yours sincerely".

This letter was followed up by a further letter dated the 10th July, 1981 from Mr. O'Riordan which was as follows:-

"Dear John,

Sorry for the delay in answering you as promised in my last letter.

We have looked into your calf problem with great consideration.

We regret that you have suffered losses of such a magnitude.

However, we feel we cannot accept any liability for the deaths of your calves.

Wishing you every good luck in your future farming.

Yours sincerely".

This prompted a reply from the Plaintiff dated the 13th of July 1981 as follows:-

"Dear Sir,

Thank you for your letter of the 10th inst.

I cannot understand that you will not accept any liability for our losses as we can establish that your product was the sole cause of our problems.

As I have already pointed out to your Accounts Department if our cheque comes through our Bank I will be publishing the facts and I am now in contact with the I.F.A. on this matter.

I have also told your Accounts Department that I will be taking action against you in the High Court.

Yours faithfully".

A reply was received on the 17th of July intimating that Mr. O'Riordan was on holiday and would receive the Plaintiff's letter on his return. There was no further correspondence

that year.

During the winter of 1981/82 the Plaintiff found a similar pattern to that of the previous year when the animals were fed on silage. In February 1982 he asked for examination of the pancreas of a deceased animal. However, death was diagnosed as caused by salmonella and worms and no test was made on the pancreas because unfortunately the laboratory had neither the equipment, nor the technicians the expertise, to do so. About this time also tests showed a calcium phosphorus imbalance. In December, 1982 when one of the animals died it was found that the stomach was black and the pancreas in pieces while the liver was normal. However, no laboratory examination was made because by the time it could be arranged the organs had become decomposed. One of the animals from 1980 which had not thrived was still on the Plaintiff's farm in 1984. It died and when opened showed similar signs as those shown by the animal which had died in December 1982 but again no laboratory examination was made because the organs were already too decomposed. In both cases the examination of the animal took place three to four days after death and I am satisfied from the evidence given on behalf of the Defendants that the organs would have been too decomposed at that stage for any reliable diagnosis to have been obtained.

From 1981 on the Plaintiff sought the opinions of many experts, several of whom came to his farm, but all to no avail. No one was apparently able to provide him with a satisfactory explanation for his problems. Nevertheless, he remained convinced that the Defendant's product was responsible. The following year he determined to find out the cause for himself. He read textbooks on the subject and from these satisfied himself that he had discovered the scientific reason for the damages

sustained by his animals. In October 1982 he again sought to impose liability for his losses upon the Defendant. Mr. O'Riordan and another representative of the Defendant met the Plaintiff and discussed his problems. Regrettably, the Defendant who from the evidence adduced on his behalf at the hearing before me must have been reasonably well aware of the factors contributing to the Plaintiff's problem chose once again to be unhelpful. Following the meeting Mr. O'Riordan wrote to the Plaintiff on the 12th October 1982 as follows:-

"Dear Mr. Kearney,

I wish to thank you for meeting Tom Bell and myself last Wednesday, 6th October, 1982. We followed with interest what you said to us and took action by speaking to the contacts that you gave us. We have now completed a detailed analysis of all aspects of your case and on the representation of our Company Nutritionist we are satisfied that our milk replacer "Prolong" did not in any way damage your cattle as calves. We therefore wish to emphasise to you once again that Paul and Vincent Limited disclaim any liability for the problems you have had with your cattle.

Yours sincerely."

Not being able to obtain any satisfaction from the Defendant, the Plaintiff issued proceedings on the 15th February, 1983, seeking damages for breach of contract and for negligence. The Statement of Claim delivered in those proceedings set out in essence the scientific theory which the Plaintiff believed from his researches was the cause of his problems. Other than claims for damages, the Statement of Claim was in the following terms:

"The Plaintiff claims that he purchased in 1979 and 1980

"from the Defendant a product called Prolong Acidified Cream Food designed for ad-lib cold feeding systems for calves. The Plaintiff further claims that above said product had acid applied to it at time of purchase and that there was no specified low temperature below which it should not be fed either with instructions with product or by advertisement. The Plaintiff claims that he fed above said product as designed and that it caused an abnormally low temperature in the calves and this was indicated by calves shivering shortly after starting to drink the product and for a considerable time after consuming the product. The Plaintiff claims that because of the low temperature the result of which would be poor enzyme activity causing a failure of the hydroxide mucus to neutralise the increased acidity close to the lining of the stomach resulting in damage to said organ.

The Plaintiff claims that the same occurrence would take place in the duodemun resulting in damage to said organ. This would also cause a failure of pancreas juices and bile salts to neutralise the increased acidity of stomach resulting in overexertion of pancreas thereby damaging said organ.

The Plaintiff claims the result of this damage is high mortality, poor performance of the animals throughout their life depending on the amount of said damage done. This would also result in the inability of the animal to handle the normal acidity of the silage and a calcium phosphorus imbalance when the level of feed was increased on silage.

The Plaintiff claims that he found said damage to the

"stomach and pancreas of an animal and that he requested the defendant to send a representative to inspect the said damage.

The Defendant further claims that at no time did the Defendant make any effort to advise or help the Plaintiff as to the nature of the problem with his animals so as reduce the Plaintiff's losses to a minimum and prevent any reoccurrence of the problem in the future."

The Defendant denies that its product was in any way dangerous or was not properly tested. It maintained that the causes of the Plaintiff's losses were to be found in the Plaintiff's own poor husbandry and his failure to follow the instructions contained in each sack of the Defendant's product. Under the first heading the Defendant contended that the housing of the calves was poor, that inadequate bedding was provided, and that the quality of silage and of the spring pasture was poor. Under the second heading the Defendant contended that insufficient calf pencils were fed to the calves and that the maximum recommended number for group feeding was exceeded.

The evidence given by the Plaintiff himself dealt with the history of his herds and the reasons why he believed that the defendant's product was the source of his ills. No evidence was adduced from any professional witness or any witness with any experience of investigating similar problems, to support his theory. The Plaintiff based his theory upon the calcium deficiency which was discovered in some animals during the winter of 1981. From this he deduced from his textbook readings that there was acidity in the duodenum. Had there not been, and, had this track been alkaline, the calcium solids entering it would have dissolved and thereby prevented calcium deficiency

in the bloodstream. This acidity also meant that pancreatic juices flowed continuously to neutralise the acidity in the duodenum thereby damaging the pancreas. The cause of acidity in the duodenum he believed arose because the acidity of the stomach was not being neutralised. He stated that the acid contents of the stomach were normally neutralised by the alkaline mucus contained in the stomach which reaction was catalysed by enzymes produced by the stomach. These enzymes were not produced in as large a quantity when the temperature of the stomach dropped, and that such temperature drop was taking place when the animals ingested the acidified milk replacer.

The Plaintiff's main complaints were lack of body thrive, excessive shivering when ingesting the acidifed milk replacer and black droppings. His belief was that the temperature drop in the stomach as evidenced by the excessive shivering led on the basis indicated to acidity in the duodenum. This in turn brought about the calcium deficiency and also organic damage evidenced by body fading and black droppings.

In support of the Plaintiff's case evidence was given by two neighbouring farmers who had similar experiences to that of the Plaintiff when using the Defendant's product. The animals shivered excessively after consumption of the product and became unthrifty. Even when taken off the product they remained unthrifty. One of these witnesses also had the same poor results as the Plaintiff when the unthrifty animals were fed on silage the following winter. This evidence established no more than that other animals suffered similarly to those of the Plaintiff, but it was of no assistance in seeking to determine the cause.

Two Veterinary Surgeons were called on behalf of the Plaintiff. They supported the Plaintiff's evidence as to the

poor quality of many of his animals but were unable to diagnose the cause though they did give advice which was followed as to how to alleviate the symptons. Grace Lane a bachelor of Dairy Science gave evidence that tests were made in September 1981 showing that the grass fed to the animals was within normal limits and that silage was good but less acid than normal. Laurence Murnane an ACOT adviser also gave evidence that he was unable to account for the condition of the Plaintiff's animals. He said that the silage which he tested had a low digestibility and would have been required to be supplemented by other feeding. The condition of some of the animals which he saw could have been caused by their being fed on such silage without supplementary feeding. He also gave evidence to the effect that the drainage in the Plaintiff's calf houses was poor and that he would have needed to use more straw to keep the floor dry than would otherwise have been necessary. A further factor involving the need for further straw was the fact that animals take more of the acidified milk replacer than of conventional milk replacers and accordingly produce more urine. Dealing with the question of the deformity of the hooves of the Plaintiff's animals he was of the opinion that this was caused by some deficiency but was not in a position to indicate what. William Ryan an Agricultural Officer with the Department of Agriculture also gave evidence to the effect that though the calf houses of the Plaintiff looked suitable they would have needed plenty of straw.

On behalf of the Defendants, Tim O'Riordan, their Sales Manager, gave evidence that when he saw the herd on the 8th April 1981 it was in poor condition, that the animals were very dirty and had very little hair. He said that he indicated that conditions

on the farm were poor but that the Plaintiff insisted that they were normal. He said that the loss of hair was caused by constant lying in dung and that 70% of the animals were in that condition. He said that animals which should have weighed from five hundredweight upwards ranged from between three and six hundredweight. His view was that there was overstocking in 1980 and that this predisposed the herd to both pneumonia and salmonella. He also gave evidence as to the purchase of calf pencils to show that the calves did not receive these in recommended quantities.

Doctor Norman Kenny, the Chief Nutritional Adviser to the Defendant, gave evidence that acidified milk replacer was developed and launched in Holland in 1976 and that trials were carried out by the Defendants at a Research centre in England in 1978. He indicated that the product had a number of advantages over conventional milk replacer and, in particular, that it saved the farmer time. He said that the animals drank twice as much as of conventional milk replacer but that it did not suit all calves and that the farmer setting up an ad-lib system required a lot of advice. Dealing with the scientific theory of the Plaintiff in relation to the product he said there was an acid sensor mechanism in the duodenum which would prevent excess acidity passing from the stomach. In relation to the question of the acidity from silage he said that this would have been neutralised in the rumen and would not have reached the stomach as increased acidity. He said that he was unaware of calcium problems with the silage. Dealing with the question of ad-lib feeding he said that if the product ran out he would expect the animals to take larger quantities than was suitable for them and that this would cause problems. He accepted that shivering

would follow ingestion of the product as this was a normal reaction to any cool or cold liquid, but this would not have harmed the animals in any way.

Doctor Joe Harte, a Senior Research Officer with the Agricultural Institute, also gave evidence to this effect. He said that a true ad-lib feeding was very important as otherwise the animals would go at the feed too quickly. He said that having too many calves meant that the bins containing the acidified milk replacer would have to be filled more often and that also it would be difficult to pick out those which were not feeding properly. He said that he had seen the Plaintiff's housing and he was not satisfied as to its quality.

Professor Barry Leek gave evidence to the effect that there were no enzymes in the stomach which would catalyse the neutralisation of acidity. He said that the pancreatic secretions entered the duodenum at a point below that at which the sensor mechanism in the duodenum indicated an increase in acidity. He also said that calcium was more likely to be absorbed in acidity than in alkalinity. His view of the causes of the Plaintiff's problems were that there was a deliberate attempt to restrict the amount of milk replacer and that the animals were fed nuts instead of calf pencils. He said that pus in the joints was evidence of the presence of salmonella and that the pancreas could not be turned on permanently as suggested by the Plaintiff. His view was that the animals were under nutritional stress. Generally speaking he said that the infection must have been brought to the herd in 1979, that the Plaintiff's problems were compounded that year by serving the product at three quarter strength, that there was a housing problem, lack of straw and, finally, nutritional stress.

I am satisfied from the evidence that the scientific theory propounded by the Plaintiff as to the cause of the damage to his animals is untenable. I am satisfied that there is no enzyme produced in the stomach to assist in neutralising the acidity of its contents. I accept the evidence given on behalf of the Defendant that the animals would have shivered after ingestion of the product and that this was natures way to ensure that stomach temperature would rise following any drop. I accept that any drop in stomach temperature would have been short-lived and would not have affected the neutralisation of the acidity of its contents. If the contents of the stomach passing into the duodenum showed excess acidity then there was a sensor mechanism in the duodenum itself which would have slowed down the flow of such acid juices. This sensor mechanism is situate in the duodenum before the point at which the pancreatic juices reach the duodenum and accordingly the duodenum would not in any circumstances have imposed through acidity an excess demand on the pancreas for production of the pancreatic juices. So far as the acidity in the silage is concerned I accept the evidence that this would have been neutralised in the rumen and that no excess acidity would have reached the stomach as a result of the ingestion of silage.

It is quite clear from the evidence that there was nothing in the chemical composition or the chemical reaction caused by the Defendant's product which in any way damaged the Plaintiff's cattle. I accept the evidence of Doctor Norman Kenny as to the development and testing of the Defendant's product. I accept the evidence that the advantages of the acidified milk replacer are that considerable time is saved both in the mixing of the product and in the daily or twice daily feeding to the

animals. The product when mixed would keep for three days and ensure that the milk was preserved and did not separate. It was an ad-lib system which meant that the animals took smaller meals and this in turn conferred health advantages. It was designed to reduce scour and to prevent the proliferation of bacteria. I accept that consideration had been given to the possibility of problems arising from the product being served cold but that no known problems had been ascertained. Since the animals drank about twice as much liquid as with conventional milk replacers they passed twice as much urine. This led to a greater potential for management problems and also a need for greater observation of the animals. I also accept that any farmer setting up an ad-lib system would need a lot of advice and that twelve animals for every two teats was the optimum. I also accept that the product did not necessarily suit all calves. I accept that the product itself was properly tested before it was put on the market and that it is a suitable product to feed to calves. Nevertheless, it is also clear that farmers going in for calf rearing need special advice if they intend to use an acidified milk replacer to ensure that the product will meet their demands and that they will not have any unnecessary problems.

I am satisfied from the evidence that the 1979 herd suffered somewhat more than the normal quota of disease which might have been anticipated. I am also satisfied that the 1980 herd suffered in a similar fashion to that of the 1979 herd but in a much aggravated form. Evidence has been given by Professor Barry Leek that such a build-up of disease was to have been expected. However, it was essential to his evidence that the disease would have remained in the Plaintiff's herd through

animals which were carriers and not just independently in the breeding sheds. The Plaintiff disposed of his 1979 herd before he purchased his 1980 herd. In addition, some of the affected animals from his 1980 herd remained on his farm during 1983 when he again commenced a calf rearing programme. This herd was apparently unaffected. It seems to me that on the probabilities the Plaintiff's problems with the 1980 herd must spring from the same basic factors as caused his troubles in 1979. Since the problems were aggravated in 1980, it seems reasonable to attribute this to a predisposing factor which itself was more prominant in 1980 than in 1979.

In each of these years the housing and general level of husbandry would have been about the same. There was one major difference between the 1979 herd and the 1980 herd and that was their relative numbers. It seems to me that the aggravation of the Plaintiff's problems must have been rooted in this circumstance. It would have meant that the housing conditions which on the evidence even of the Plaintiff's witnesses was border-line would have been a predisposing factor towards lack of thrive and disease. Poor bedding - and I accept the evidence of the condition of the animals in April 1981 as being attributable in part to this cause - would have been a further predisposing cause. Both factors, however, would have become relatively more likely causes the greater the number of animals being reared. A further factor not involving the feeding would have been that, with greater numbers, the Plaintiff - who in the main had lost the help of his wife through pregnancy and the need to look after her child - would have been less able to supervise the animals and to ensure that problems would be dealt with promptly. In these factors alone, there is sufficient

evidence to indicate that results in 1980 would have been worse than in 1979.

The final possible factor is the manner in which the acidified milk replacer was fed to the calves. On the evidence, I am satisfied that it should be fed on a truly ad-lib basis and that this enables the calves to take the replacer in relatively small quantities. If they are deprived of product then they are inclined to take larger amounts when it does become available which is detrimental to them.

On the Plaintiff's own evidence I am satisfied that he did deprive the calves of product deliberately because he was of the view that they were taking too much. The obvious result of such a policy was that the feeding ceased to be ad-lib and that the calves had in effect to compete for their food.

The evidence as a whole satisfies me that calves when fed in the pens did suffer nutritional stress and I fully accept the evidence of Professor Leek to this effect. This nutritional stress resulted in lack of thrive and greater susceptibility to disease. Nutritional stress also manifested itself when the animals were brought in from pasture and fed on silage. The Plaintiff attributes this latter condition to damage sustained by the calves by reason of their consumption of the Defendant's product. I am unable to accept this contention. In the first case it is based upon the existence of organic damage which he has failed to establish. Secondly, there is no evidence that feeding on silage rather than on other foodstuffs would aggravate any pre-existing lack of thrive. I accept the evidence that nutritionally the silage fed to the calves was deficient, since once the animals were again put out to pasture their calcium deficiency ceased. If they did

get supplementary feed they did not get sufficient of it. Undoubtedly the animals which had already suffered were again those that suffered from the deficiency in the silage. But I find no connecting link in the evidence that any further damage sustained by the animals is attributable to their earlier history.

I am satisfied that the problems sustained by both the 1979 herd and the 1980 herd had their origins in nutritional stress, aggravated by border-line housing, less than satisfactory bedding and poor husbandry. The nutritional stress was brought about by failure to provide the acidified milk replacer in sufficient quantity and a similar failure with calf pencils. In 1979, there was reduced product supply because it was given at three quarter strength for the first three weeks. In 1980, the Plaintiff admits that he deliberately cut down the amount of product because it seemed to him the animals were taking more than if they had been on a conventional milk replacer. This in part explains why the Plaintiff put 19 calves in a pen in which in the previous year he had placed only 12. It seems on the probabilities that he also cut down on supply the previous year. Once there was a curtailment of supply, then it followed that the animals could not have been fed on an ad-lib basis. In both years there was an insufficiency of calf pencils.

The Defendant cannot be responsible for loss sustained by the Plaintiff because of either border-line housing, less than satisfacoty bedding or poor husbandry. Nevertheless, there is evidence to suggest that it may have some responsibility for the nutritional stress because of the failure of the Plaintiff to appreciate differences between the Defendant's

product and the type of milk replacer previously used by the Plaintiff, and in particular that the calves consumed twice the quantity of acidified milk replacer than a conventional milk replacer and the need for feeding to be truly ad-lib.

A manufacturer of a product which is not dangerous in itself is not absolved from all duty of care to the users of his product. This duty arises upon the basis of the neighbour principle established in Donoghue .v. Stevenson 1932 A.C. 562 and approved by the Supreme Court in McNamara .v. E.S.B. 1975 IR 1. Henchy J. at page 24 quotes the following passage from the judgment of Lord Atkin in Donoghue .v. Stevenson:

> "You must take reasonable care to avoid acts or omissions which you can reasonably foresee would be likely to injure your neighbour. Who, then, in law is my neighbour? The answer seems to be persons who are so closely and directly affected by my act that I ought reasonably to have them in contemplation as being so affected when I am directing my mind to the acts or omissions which are called in question."

Later on the same page he says:

> "It needs to be stressed that the existence of, or falure to observe, a duty of care should not be determined with the hindsight derived from the accident but in the light of the circumstances, actual and potential, that ought to have been present to the mind of a reasonably conscientious occupier of property before the trespass took place."

The extent of the duty of a manufacturer whose product is alleged to have caused damage is not to be determined therefore with regard to the damage alleged to have been sustained but with regard to what he knew or ought to have known when he released the product on to the market. In McNamara .v. E.S.B. the Court

was dealing with the duty of care towards a trespasser. In the present case, it seems to me that the duty of care should be analogous to that of the duty owed by an invitor to an invitee as in both cases the relationship between the parties arises in the interests of the party owing the duty.

There must be many matters which come or should come to the notice of a manufacturer placing a new product on the market which, if he gave or had given proper consideration to such matters he should have realised were such that the user of the product required to be told about them because otherwise in ignorance of them he might use the product in such a way as to sustain damage as a result. It is his duty to ensure that such matters are brought to the attention of the users of the product.

In the present case the bag in which the product was supplied indicated that the product was "Prolong Acidified Cream Food". Each bag also contained the following warning "This acidified product is designed for ad-lib cold feeding systems. Instructions inside the bag".

The instructions inside the bag were as follows:-

"Feeding instructions for Prolong Acidified Cream Food Code No. 177

Prolong Acidified Cream Food is designed for cold ad-lib self feeding systems and will keep fresh for up to three days when mixed.

The calf should receive biestings from its mother in the normal way and can then go on to cold ad-lib feeding from one week of age.

Add Prolong Cream Food to water at room temperature - not

higher than 40° C (104° F).

Recommended Mixing

The rate is: 1 pound in 1 gallon water., i.e. a 10% concentration. It is preferable, during the first three weeks, to have the feed available to appetite. A new mix can satisfactorily be emptied on top of a small residue in the container, but never mixed with a large residue. In this way, the container need not be emptied, if so wished; however, it is advisable to wash it out once a week.

On the fifth and sixth weeks restrictions should be placed on the feeding of Prolong Cream Food to encourage the rumen development by the calfs and therefore an increased intake of calf weaner pencils.

Offer calf weaner pencils from the first week, together with hay and water, and continue throughout the rearing period up to twelve weeks."

The instructions further indicated how to use the product with a single unit system or with a group feeding - "dustbin" system. In relation to a single unit system the instructions were as follows:-

"Most farmers are already geared for individual penning of baby calves in their housing systems. This enables better assessment of food intake, observation of calves and disease control than does a group system. Bins with a single teat may be fixed to the front of an individual calf pen."

In relation to group feeding the instructions were:-

"Fix teats to the wall of the calves pen. Connect tubes (narrow bore) to the teats and run them through holes in the lid of the dustbin, ensuring that they reach close to the bottom of the dustbin. The teats should be at least 24" above floor level. Up to twelve calves can be service

"by two teats. Before setting up a group feeding system, please consult our advisory service."

While these feeding instructions indicate that the product is to be fed on an ad-lib basis there is no warning as to the dangers inherent in setting up the system other than on an ad-lib basis. The use of the words "it is preferable" and their reference to "the first three weeks", if anything, indicates the contrary. Again, when considering the instructions in relation to single unit systems and group feeding systems there is no warning on the need to create a true ad-lib system. If anything, the implication from the instructions is that those matters such as assessment of food intake, observation of calves and disease control may be more difficult with a group feeding system and that advice should be obtained to ensure that these matters can be properly monitored.

The Plaintiff contends that these instructions should have warned against the dangers inherent in feeding product at low temperature. Since I do not accept that there was any such danger, the instructions arenot inadequate on that account. It may be that these instructions should have been more detailed. However, such an issue has not been raised by the Plaintiff and the Defendant has had no opportunity to consider nor to adduce evidence to meet any such case. It is not, therefore, open to me in the absence of an amendment to the pleadings to decide whether or not these instructions ought to have gone further, nor, if they should, that such failure caused or contributed to any damage sustained by the Plaintiff. I have given consideration to the question as to whether or not I should even at this late stage allow the pleadings to be amended and then hear further evidence.

I think I should not. Such a course would be clearly prejudicial to the Defendant and even though the Plaintiff presented his own case, the Defendant cannot be penalised on that account.

The Plaintiff claims that he should have been warned of the dangers inherent in feeding the Defendant's product at a low temperature to his calves and that when the Plaintiff's herds suffered damage the Defendant should have given him advice to overcome his problem and to minimise his loss. As I am satisfied that the losses sustained were not caused by feeding the Defendant product to the calves at low temperature, this aspect of the Plaintiff's claim fails. If the Plaintiff had established liability on the part of the Defendant, this would have entitled him to damages. The Defendant having been offered an opportunity to give advice and assistance would not, if such advice and assistance had been refused, have been in a position to complain if alternative efforts by the Plaintiff to minimise his loss had proved more expensive for them. As liability has not been established under the first head of claim, the Defendant was not under any legal liability to advise or assist the Plaintiff. This head of claim also fails.

Henry Barron
30/7/85