significance. J. Med. Microbiol. *8*:149-166. 6. Fraser, D., J.S.D. Ritchie, A.F. Fraser. 1975. The term "stress" in the veterinary context. Br. Vet. J. *131*:653-662. 7. Gibson, E.A. 1961. 1. - Salmonellosis in calves. Vet. Rec. *73*:1284-1295. 8. Gibson, E.A. 1965. Disease of dairy cattle. Salmonella infection in cattle. J. Dairy Res. *32*:97-134. 9. Glickman, L.T., P.L. McDonough, S.J. Shin, J.M. Fairbrother, R.L. LaDue, S.E. King. 1981. Bovine salmonellosis attributed to *Salmonella anatum* - contaminated haylage and dietary stress. J. Am. Vet. Med. Assoc. *178*:1268-1272. 10. Hughes, L.E., E.A. Gibson, H.E. Roberts, E.T. Davies, G. Davies, W.J. Sojka. 1971. Bovine salmonellosis in England and Wales. Br. Vet. J. *127*:225-237. 11. Kahrs, R.F. 1978. Techniques for investigating outbreaks of livestock disease. J. Am. Vet. Med. Assoc. *173*:101-103. 12. Kahrs, R.F., J. Bentinck-Smith, G.R. Bjorck, D.W. Bruner, J.M. King, N.F. Lewis. 1972. Epidemiologic investigation of an outbreak of fatal enteritis and abortion associated with dietary change and *Salmonella typhimurium* infection in a dairy herd. A case report. Cornell Vet. *62*:175-191. 13. Khakhria, R., H. Lior. 1980. Distribution of phagovars of *Salmonella typhimurium* in Canada (1969-1976). Zbl. Bakt. Hyg., 1. Abt. Orig. A *248*:50-63. 14. Linton, A.H., K. Howe, S. Pethiyagoda, A.D. Osborne, 1974. Epidemiology of salmonella infection in calves (1): Its relation to their husbandry and management. Vet. Rec. *94*:581-585. 15. McDonough, P.L. 1982. Bovine salmonellosis in New York State. Veterinary Topics, April, 1982, pp. 5-11. 16. McDonough, P.L. 1985. Population diversity in strains of *Salmonella typhimurium* from animals in New York state. Ph. D. Thesis, Cornell University, Ithaca, New York. 17. McDonough, P.L., S.J. Shin, J.F. Timoney. 1986. Salmonella serotypes from animals in New York State, 1978-1983. Cornell Vet. *76*: in press. 18. Morse, E.V., M.A. Duncan, J.S. Baker, H.E. Amstutz, E.P. Myhrom, K.A. Gossett. 1975. Prevalence, clinical aspects, treatment and control of bovine salmonellosis, pp. 17-20. In: Proceed. Seventh Annu. Convention Am. Assoc. Bovine Practitioners, 1975. 19. O'Brien, T.F., J.D. Hopkins, E.S. Gilleece, A.A. Medeiros, R.L. Kent, B. O. Blackburn, M.B. Holmes, J.P. Reardon, J.M. Vergeront, W.L. Schell, E. Christenson, M.L. Bissett, E.V. Morse, 1982. Molecular epidemiology of antibiotic resistance in *Salmonella* from animals and human beings in the United States. N. Engl. J. Med. *307*:1-6. 20. Orskov, F., I. Orskov. 1983. Summary of a workshop on the clone concept in the epidemiology, taxonomy, and evolution of the *Enterobacteriaceae* and other bacteria. J. Infect. Dis. *148*:346-357. 21. Osborne, A.D., A.H. Linton, S. Pethiyagoda. *1974.* Epidemiology of salmonella infection of calves (2): Detailed study in a large beef rearing unit. Vet. Rec. *94*:604-610. 22. Richardson, A. 1973. The practical aspects of the epidemiology of salmonellosis in cattle, pp. 6-10. *In: The Veterinary Annual,* vol. 14. 23. Richardson, A. 1975. Salmonellosis in cattle. Vet. Rec. *96*:329-331. 24. Richardson, A. 1975. Outbreak of bovine salmonellosis caused by serotypes other than *S. dublin* and *S. typhimurium*. J. Hyg. (Camb.) *74*:195-203. 25. Richardson, A., W.A. Watson. 1971. A contribution to the epidemiology of *Salmonella dublin* infection in cattle. Br. Vet. J. *127*:173-182. 26. Robinson, R.A. 1965. Salmonellosis in young calves. New Zealand Vet. J. *14*:33-39. 27. Robinson, R.A., K.I. Loken. 1968. Age susceptibility and excretion of *Salmonella typhimurium* in calves. J. Hyg. (Camb.) *66*:207-216. 28. Threlfall, E.J., L.R. Ward, B. Rowe. 1978a. Spread of multiresistant strains of *Salmonella typhimurium* phage types 204 and 193 in Britain. Br. Med. J. *2*:997. 29. Threlfall, E.J., L.R. Ward, B. Rowe. 197b. Epidemic spread of a chloramphenicol-resistant strain of *S. typhimurium* phage type 204 in bovine animals in Britain. Vet. Rec. *103*:438-400. 30. Timoney, J.F. 1978. The epidemiology and genetics of antibiotic resistance of *Salmonella typhimurium* isolated from diseased animals in New York. J. Infect. Dis. *137*:67-73. 31. Tutt, J.B., D.I.B. Hoare. 1974. Disease associated with *S. typhimurium* in cattle. Vet. Rec. *95*:334-337. 32. Wray, C., W.J. Sojka. 1972. Bovine salmonellosis in England and Wales: Its control and prevention. State Vet. J. *27*:169-179.

Comparison of Oral and Intravenous Fluid Therapy in Neonatal Calves with Experimental Colibacillosis

C. L. Guard, *D.V.M., Ph.D.*
B. C. Tennant, *D.V.M.*
Department of Clinical Sciences
New York State College of Veterinary Medicine
Cornell University
Ithaca, NY 14853

Fluid therapy is widely recognized as the primary means of correcting the abnormalities of volume, electrolyte and acid-base status that occur in severe diarrhea. Commercially available oral electrolyte preparations are commonly used and likely prevent the loss of many dehydrated, scouring calves. Intravenous fluid therapy is effective in restoring body fluid and electrolytes but has the disadvantages of 1) greater expense, 2) less availability of supplies to farmers, 3) more skill needed to administer and 4) more time required for proper delivery. Most would agree that calves in coma or severe shock will not respond favorably to oral therapy. These clearly require immediate intravenous fluids. Those calves that are dehydrated and depressed but not yet shocky represent a grey area. Will oral fluids be utilized adequately or must therapy be more aggressive? These questions prompted our study comparing oral and intravenous therapy in experimentally produced diarrhea.

The Model

Male Holstein calves (n=25) were purchased from nearby farms and obtained within 3 hours of birth. Blood samples were collected and a fixed quantity of pooled colostrum given. The calves were transported to an isolation unit and prepared for quantitative collection of feces and urine. At 12 hours of age they were inoculated orally with a log-phase broth culture of *Escherichia coli* 0101:K(A), originally isolated from a calf with severe diarrhea. Calves were allotted in advance to 1 of 4 treatment groups. Treatments

were oral or intravenous (IV) fluid therapy beginning either at the onset of diarrhea or when 8% of initial body weight was passed as diarrheic stood. IV fluids were Ringer's bicarbonate and oral fluids were 20 mM KCl, 60 mM NaCl, 30 mM $NaHCO_3$ and 111 mM glucose. Rehydration was for 48 hr at a rate calculated to provide maintenance water requirements and correct water volume deficits. The calves were then switched to whole milk feeding for a 48 hr maintenance period. Samples of blood, all urine and all feces were collected at 6 hour intervals during therapy and at 12 hour intervals during maintenance.

Results and Discussion

Two calves did not develop diarrhea and 1 calf died before diarrhea developed. In 22 calves diarrhea developed in 6 to 18 hours after inoculation with *E. coli.* All calves receiving intravenous therapy survived the 96 hour experimental period. One calf receiving immediate oral therapy died at 18 hours after the onset of diarrhea, one at 48 hours, 1 at 54 hours and 1 at 60 hours for a total of 57% mortality. All calves receiving delayed oral therapy died within 18 hours of the onset of diarrhea. Serum potassium was maintained within normal range for the treatment and maintenance periods for both IV groups but rose above 6 mEq/l by 12 hours of treatment for the oral groups and remained elevated through maintenance. Serum sodium ion stayed in the normal range for the immediate IV group, fell to just below normal in the delayed IV group and fell to 128 mEq/l in the immediate oral group. Serum sodium was normal in the delayed oral group. Plasma glucose rose to 80 mg/dl or more in all groups during the development of diarrhea. Immediate oral rehydration maintained glucose above 75 mg/dl. Plasma glucose fell in both IV groups during rehydration and rose again when they were returned to the maintenance diet of milk. Serum urea nitrogen (BUN) rose from baseline values throughout the experimental period for all groups to about twice the high-normal value at the end of the maintenance period. Blood pH remained closest to 7.4 during rehydration in both IV groups. It varied between 7.29 and 7.35 during rehydration for the immediate oral group. Blood pH decreased in all groups during maintenance to between 7.25 and 7.32. The lowest values were observed in the non-surviving, delayed oral group with a mean of 7.16 at 12 hours of treatment.

During the 48 hr rehydration period the IV treatment groups were in substantial positive water balance (3-4 l/48 hr) whereas the immediate oral group was nearly in balance. The delayed oral group, although alive for only a portion of the treatment period, was in positive water balance. This positive balance probably was from fluid administered and retained in the gut at the time of death. Potassium balance was achieved for the oral treatment groups but a large deficit (180 to 210 mEq) occurred during rehydration for the IV treatment groups. Calves receiving IV treatment were in positive (ca. 450 mEq) sodium balance during rehydration while a net loss of almost equivalent magnitude occurred during maintenance. A 400 mEq sodium deficit developed in both delayed treatment groups between the onset of diarrhea and the beginning of rehydration. The immediate oral group had a 350 mEq sodium deficit during rehydration but recovered most of this during maintenance.

Summary

Severe, acute diarrhea was produced in 22 of 25 calves by oral inoculation of a broth culture of *E. coli.* Those calves receiving IV fluid therapy at the onset of diarrhea had the least deviations from normal blood chemistry values and all survived. Those receiving delayed IV therapy had minor abnormalities in blood chemistry values and also all survived. Those calves receiving oral therapy beginning at the onset of diarrhea developed moderate acidosis, hyperkalemia and hyponatremia; they had a cumulative 57% mortality by the end of the maintenance period. Calves orally rehydrated when 8% of body weight had been lost as diarrheic stool developed severe acidosis, hemoconcentration and hyperkalemia; all calves in this group died within 18 hr of the onset of diarrhea.

The challenge to neonatal calves posed by this experimental model of enteric colibacillosis represented an extreme of the naturally occurring disease. In the model only those groups of calves receiving IV rehydration had no deaths. The oral solution used for rehydration was better able to balance the potassium losses and maintain plasma glucose than the IV solution. The IV solution was better for maintaining water and sodium balance and correcting acid-base disturbances. When calves have severe diarrhea as typified by this model intravenous rehydration is required to restore homeostasis. If the challenge to the calf is less severe, oral therapy may be adequate. At this time no predictor for field use is available for deciding in advance which route of therapy will prove most efficient.

Questions & Answers:

Question: What did you feed them?

Answer: They were on milk until they developed diarrhea. Actually for the group that was maintained until they developed 8% diarrhea, they got milk up until the point at which they were 8% dehydrated. Sorry for leaving that out.

Question: How much?

Answer: The quantity was estimated. It wasn't the same. It varied depending on their fecal losses during the preceding period, plus an estimate of their maintenance requirements. So they could have received as much as maybe 15 liters over 48 hours time, based on how much fluid they lost earlier. In some earlier experiments we tried to replace mililiter per mililiter what calves lost, and essentially they drowned. So volume at each feeding was generally on the order of 2-3 liters every 6 hours of oral electrolytes.