Effects of antimicrobial treatment at the end of lactation on milk yield, somatic cell count, and incidence of clinical mastitis during the subsequent lactation in a dairy herd with a low prevalence of contagious mastitis

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Objective—To determine whether treating cows with antimicrobials at the end of lactation would lower the incidence of clinical mastitis, improve milk production, and decrease somatic cell count (SCC) in the subsequent lactation.

Design—Randomized blind field trial.

Animals—233 Holstein cows from a single herd. All cows were in lactation 2 or greater.

Procedure—Cows were randomly assigned to treatment groups. Treated cows were given procaine penicillin G and novobiocin by intramammary infusion. Control cows were not treated. Farm personnel recorded cases of clinical mastitis. Milk yield and SCC were recorded during the subsequent lactation.

Results—Treatment did not significantly reduce the incidence of clinical mastitis when data for all cows were grouped or when data were stratified by lactation groups (lactation 2 vs lactation ≥ 3) or by last SCC (≤ 500,000 cells/ml vs > 500,000 cells/ml). Somatic cell counts (first, mean of first 5, maximum of first 5) for treated and control cows were similar, and proportions of treated and control cows with SCC > 500,000 cells/ml at least once were not significantly different. Treated cows produced 179 kg (394 lb) more milk during the first 17 weeks of lactation than did control cows.

Clinical Implications—Treating cows with antimicrobials at the end of lactation increased 17-week milk production during the subsequent lactation and, at current milk prices, was financially preferable to not treating them. (*J Am Vet Med Assoc* 1997;211:207–211)

During the last 30 years in the United Kingdom and United States, an increasing number of dairy producers have adopted various procedures, including teat dipping immediately after milking, antimicrobial treatment at the end of lactation, milking clean, dry udders, and regular milking machine maintenance, to control contagious mastitis. In some herds, milk samples collected from cows at the end of lactation, after calving, and when clinical signs of mastitis are apparent are submitted for bacteriologic culture to monitor the success of these procedures. These management techniques have greatly reduced the prevalence of contagious mastitis pathogens (*Staphylococcus aureus*, *Streptococcus agalactiae*, and *Mycoplasma* spp). Antimicrobial treatment at the end of lactation was intended to reduce the prevalence of infections at the cessation of lactation and to prevent new infections in the early nonlactating period. In herds with a high prevalence of contagious mastitis pathogens, this procedure has been an efficacious and economically effective method of reducing the frequency of intramammary infections.[1] With consistent application of these mastitis control programs, the prevalence of contagious mastitis pathogens has declined to a low level in many herds,[2] as is evidenced by bulk-tank somatic cell counts (SCC) of ≤ 300,000 cells/ml. Some owners of herds with a low prevalence of contagious mastitis pathogens have stopped treating cows at the end of lactation, and others are questioning the practice because of concerns about antimicrobial residues.[2] A recent bulletin[3] by the National Mastitis Council mentions treatment of only selected cows during this period, but recommends treatment of all quarters on all cows at the end of lactation. One study[4] of survey data indicated that antimicrobial treatment of cows at the end of lactation was not an economically justified management strategy. Oliver[5] and Smith et al,[6] however, found that such treatment was effective in preventing infections in the early nonlactating period and that the prevalence of environmental streptococcal and coagulase-negative staphylococcal infections was lower among treated than untreated cows. They also reported that antimicrobial treatment at the end of lactation was not effective in preventing coliform infections at any time or infections by other bacteria during the late part of the nonlactating period or during early lactation. Harmon et al[7] not only found that certain antimicrobials decreased the prevalence of coagulase-negative staphylococci, but also found that a high percentage of cows eliminated these organisms without treatment. These researchers could not measure any difference in 305-day mature equivalent (305ME) milk production between treated and untreated cows at the end of lactation.[5] Other researchers[8] reported a decrease of > 30% in quarter milk yield during the 4 milkings immediately after calving for cows with persistent infections during the nonlactating period and cows that acquired infections during the nonlactating period.

The purpose of the study reported here was to determine whether antimicrobial treatment of cows at

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the end of lactation would have an effect on the SCC, incidence of clinical mastitis, or milk yield during the first 120 days of the subsequent lactation in a herd that had a low SCC and low prevalence of mastitis attributable to contagious pathogens.

Materials and Methods

Study population—The study was conducted on a 1,200-cow, commercial, Holstein dairy in the central valley of California. The dairy was an open-shed, freestall operation with sand bedding. Cows were milked 2 times/d in a double-18, rapid-exit parlor. The dairy had not treated cows with antimicrobials at the end of lactation for the 3 years prior to the start of the trial. Cows were always vaccinated with a commercial bacterin for the J-5 mutant of *Escherichia coli*[a] at the end of lactation, during the middle of the nonlactating period, and at calving. Mean (± SD) bulk tank SCC during the 13 months of the study was 238,330 ± 38,480 cells/ml. Milk samples were collected from cows and submitted for bacteriologic culture at calving and when clinical signs of mastitis were evident as part of the routine herd health program recommended by the herd veterinarian (JPR). During the study, none of these cultures were positive for *Streptococcus agalactiae* or *Mycoplasma* spp and < 0.2% were positive for *Staphylococcus aureus*.

Enrollment of cows and treatment—Each month for 13 months, 18 cows at the end of their lactation periods were brought into the parlor for the last milking. We calculated that we would need at least 108 cows/treatment group to detect significant ($\alpha = 0.05$; power = 0.90) differences in mastitis incidence rates (15 vs 25% in treated and untreated cows, respectively) and to detect a difference of at least 125,000 cells/ml in SCC between the 2 groups. Cows were eligible for inclusion in the study if they were free from clinical signs of mastitis at the end of lactation and were expected to calve and milk for at least 150 days in the subsequent lactation. Cows with clinically apparent mastitis that were not likely to calve and milk for at least 150 days or that had obvious health problems, such as lameness or other physical conditions that might lead to early culling or affect trial outcome, were excluded. Cows were washed in the sprinkler pen and allowed to drip-dry. Teats were dried with single-service paper towels, and teat ends were cleaned with 70% alcohol before samples were collected. Cows were randomly assigned to treatment or control groups by drawing stall location numbers. Quarter milk samples were collected aseptically prior to the last milking and submitted for bacteriologic culture. Personnel collecting milk samples wore latex gloves during sample collection and dipped their gloved hands in iodine solution before collecting samples from each cow. Samples were submitted to the University of California-Davis Food Safety Laboratory, and bacteriologic culture was performed according to National Mastitis Council guidelines.[9]

After all cows were milked, teat ends were again cleaned with 70% alcohol. An iodine teat dip was applied after the last milking, before cows were moved to the nonlactating pen. Control cows were not treated with antimicrobials. In treated cows, 200,000 U of procaine penicillin G and 400 mg of novobiocin were infused into each quarter.[b] Procaine penicillin G and novobiocin were chosen because the withdrawal time (30 days after infusion, 72 hours after calving) was shorter than that for other antimicrobials, and the herd owner was concerned about the possibility of residues if cows calved early. Also, research by Matthews et al[10] indicated that 65 and 74% of *Staphylococcus* spp isolated from bovine mammary glands were susceptible, in vitro, to penicillin and novobiocin, respectively, and that 100% of *Streptococcus* spp were susceptible to penicillin and novobiocin.

Follow-up and data collection—Ear tag numbers of cows that developed mastitis and date of onset of mastitis were recorded by the herdsman and entered into the farm computer. Milk samples were aseptically collected by the herdsman from cows with clinical signs of mastitis, and samples were submitted to the laboratory for bacteriologic culture. Cows with mastitis were treated by the herdsman with hetacillin potassium or cephapirin. Data on milk yield, SCC, and protein and fat contents were collected monthly by Dairy Herd Improvement Association technicians and were downloaded from the Data Records Processing Center[c] to the farm computer by use of a commercial record-keeping program.[d] Cows were followed up for at least 5 months after calving or until they died or were culled. Cows that died or were culled for reasons unrelated to mastitis were censored for survival analysis. The owner and herdsman knew which cows were in the trial, because red leg bands were placed on study cows to alert them if a cow calved early. They did not, however, know to which treatment group cows belonged.

Statistical analysis—A *t*-test was used to test for differences between groups in regard to days between treatment and calving and to compare baseline values for last SCC and previous 305ME milk production. An ANOVA was used to test for differences in SCC during the lactation after treatment. For this analysis, we collected data for at least 5 months after calving and analyzed for differences in first SCC, mean of the first 5 SCC, and maximum of the first 5 SCC. Somatic cell counts of 0 were treated as missing data, and cows with < 4 SCC were excluded from the latter 2 analyses. A χ^2 test was used to compare proportions of cows with 0, 1, or > 1 SCC > 500,000 cells/ml during the first 5 months of lactation. For this analysis, SCC of 0 were treated as missing data, and cows with < 3 SCC were excluded. A χ^2 test was used to evaluate differences in percentages of treated versus control cows that had ≥ 1 episode of mastitis during the first 120 days of lactation. The Kaplan-Meier product-limit method of estimating a survival curve[11] was used to compare the cumulative proportions of cows that did not develop mastitis during the first 120 days of lactation. This method accounted for censoring of cows that were known to be free of clinical mastitis when they were removed from the herd. Two-way ANOVA, with previous 305ME milk production as a covariate, was used to test for differences between groups in total milk produced during the first 17 weeks (119 days) of the subsequent lactation. The 305ME milk production in the previous lactation was used as a covariate, because it was a predictor for 305ME milk production in the subsequent lactation. This decreased the likelihood of overestimating the increase in milk production attributable to treatment. The previous 305ME milk production was higher for treated cows than for control cows, although the difference was not significant. Interaction of treatment and lactation group was assessed. All analyses were performed by use of statistical software.[12]

Results

Total number of cows treated was 233, instead of 234, because only 17 cows were available for allocation during 1 visit. Five cows (4 treated, 1 control) were culled prior to calving (3 were not pregnant, 2 had an illness other than mastitis). Two cows were eliminated because ear tag numbers were misrecorded, and 3 were eliminated because they were starting their second lactation during the trial. Therefore, 223 cows (111 treated, 112 control) began a lactation after treatment. Of these 223 cows, 49 were culled prior to the 120th day of lactation (25 treated, 24 control). Thus,

Table 1—Mean somatic cell count (SCC) for cows treated with procaine penicillin G and novobiocin at the end of the previous lactation and for untreated control cows

SCC	Treated cows		Control cows	
	Mean ± SEM	No.	Mean ± SEM	No.
First				
All cows	164.2 ± 40.2	92	260.1 ± 60.6	88
Cows in lactation 2	133.5 ± 49.6	42	160.6 ± 33.4	48
Cows in lactation ≥ 3	189.9 ± 61.3	50	379.5 ± 125.5	40
Mean of first 5				
All cows	336.0 ± 67.1	86	250.1 ± 52.6	86
Cows in lactation 2	277.1 ± 98.4	41	222.7 ± 68.5	49
Cows in lactation ≥ 3	389.7 ± 91.3	45	286.2 ± 82.7	37
Maximum of first 5				
All cows	948.2 ± 201.0	86	668.8 ± 139.0	86
Cows in lactation 2	733.2 ± 277.9	41	609.0 ± 199.7	49
Cows in lactation ≥ 3	1,144.1 ± 288.7	45	748.0 ± 189.8	37

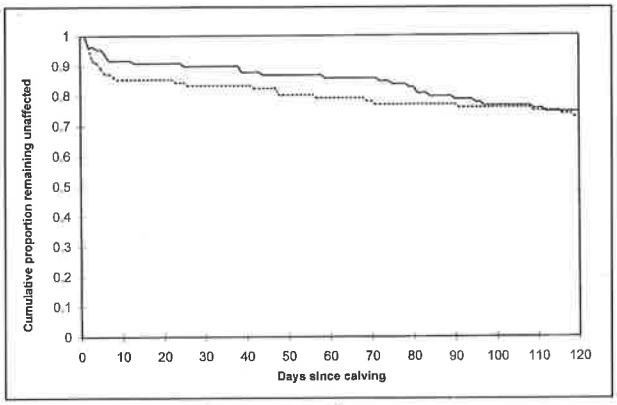

Figure 1—Cumulative portion of cows that received (solid line; n = 112) and did not receive (dotted line; n = 111) procaine penicillin G and novobiocin at the end of lactation and did not develop clinical signs of mastitis.

174 cows completed the study. Of the 49 cows that were culled, 7 of 25 treated cows and 8 of 24 control cows were culled because of mastitis.

Analysis of baseline data confirmed that groups were comparable prior to treatment. Last SCC (mean ± SEM) for treated cows was not significantly (P = 0.329) different from last SCC for control cows (248,900 ± 29,000 vs 212,600 ± 23,000 cells/ml, respectively). Previous 305ME milk production was 10,134 ± 136 kg (22,295 ± 299 lb) for treated and 9,830 ± 150 kg (21,626 ± 330 lb) for control cows (P = 0.135). Mean duration of the nonlactating period for treated cows was similar (P = 0.62) to that for control cows (56.9 ± 1.8 vs 58.6 ± 2.2 days, respectively).

Treated cows had lower first SCC, higher mean SCC, and higher maximum SCC than did control cows, but none of the differences were significant (Table 1), even after stratification by lactation group (lactation 2 vs lactation ≥ 3). Proportions of treated and control cows with ≥ 1 SCC > 500,000 cells/ml during the first 5 months of lactation were not significantly different when data for all cows were pooled or when data were stratified by lactation groups.

Clinical signs of mastitis were not detected in cows in either group during the nonlactating period or at calving, but during the first 120 days of lactation, 27 of 112 (24.1%) treated cows and 28 of 111 (25.2%) control cows developed mastitis. Differences in incidence of mastitis were not significant when data for all cows were pooled (P = 0.85) or when data were stratified by lactation group ($P > 0.30$) or last SCC ($P > 0.25$). To detect a significant difference in incidence of clinical mastitis at the observed rates would have required about 24,300 cows/group ($P < 0.05$; power = 0.80), and such a difference would not have been clinically meaningful.

The cumulative proportion of treated cows that did not develop mastitis was not significantly (P = 0.703) different from the proportion of control cows (Fig 1). Cows in lactation 2 had a similar pattern to that for all cows, and treated cows in lactation ≥ 3 had a longer time to first development of mastitis, but the difference was not significant (P = 0.54). When survival curves were stratified by last SCC, treated and control cows were still not significantly (P = 0.55) different.

Bacteria were isolated from 183 of 915 (20%) quarter milk samples collected from cows at the end of lactation (Table 2). Any sample with > 2 bacterial isolates was considered contaminated. Bacteria were also isolated from 86 of 256 (33.6%) composite samples col-

Table 2-Results of bacteriologic culture of quarter milk samples collected at the end of lactation, of composite samples collected at the time of calving, and of quarter samples collected when clinical signs of mastitis were first evident from cows given procaine penicillin G and novobiocin at the end of lactation and from untreated control cows

Culture result	Treated cows		Control cows	
	No.	%	No.	%
End of lactation				
Negative/contaminated*	369	80	363	80
Staphylococcus spp	73	16	81	18
Streptococcus spp	5	1	9	2
Other	10	2	0	0
Coliform	0	0	0	0
Mixed†	5	1	0	0
At calving				
Negative/contaminated*	94	71	76	62
Staphylococcus spp	24	18	26	21
Streptococcus spp	5	4	5	4
Other	3	2	7	6
Coliform	5	4	3	2
Mixed†	2	1	6	5
Clinical mastitis				
Negative/contaminated*	9	35	10	43
Staphylococcus spp	4	15	0	0
Streptococcus spp	5	19	3	13
Other	2	8	4	17
Coliform	3	11	2	9
Mixed†	3	11	4	17

*Contaminated samples were those with ≥ 3 different bacterial species.
†Most were staphylococci or streptococci in combination with 1 other bacterial species.

Table 3—Milk production (kg) of cows treated with procaine penicillin G and novobiocin at the end of the previous lactation and of untreated control cows

Weeks of lactation	Treated cows		Control cows	
	Mean ± SEM	No.	Mean ± SEM	No.
4	922 ± 14	95	870 ± 14	89
8	2,051 ± 27	95	1,950 ± 28	87
12	3,138 ± 38	92	3,025 ± 39	87
16	4,207 ± 48	87	4,037 ± 48	86
17	4,458 ± 51	87	4,279 ± 51	86

To convert to milk production in pounds, multiply values by 2.2.

lected at calving and from 30 of 49 (61.2%) samples collected from cows with clinical signs of mastitis. The proportion of samples from which bacteria were isolated was similar (P = 0.13) for treated and control cows at each of the 3 sampling times.

Treated cows produced significantly (P = 0.014) more milk than did control cows (Table 3). During the first 17 weeks of lactation, treated cows produced 179 kg (394 lb) more milk than did control cows. Milk production up to the time of culling was used for cows culled prior to the 120th day of lactation. There was no interaction between treatment and lactation group (P = 0.79).

Discussion

The finding that there was no difference in SCC attributable to antimicrobial treatment at the end of lactation in the study reported here agrees with findings from a study[13] of Ohio dairy herds in which lower SCC was not associated with treatment of cows at the end of lactation or with teat dipping after milking, but was associated with use of separate cloth towels for wiping teats of each cow.

On the other hand, the finding that there was no difference in the incidence of clinical mastitis during the first 120 days of lactation in the study reported here is in contrast to results of an Australian study[14] in which cows that did not have mastitis at the end of lactation and that were treated with antimicrobials had a higher incidence of clinical mastitis during the first 3 to 5 months of the subsequent lactation than did cows that were not treated. The difference was not significant, but the authors speculated that this difference was related to problems with hygiene during infusion of antimicrobials. Those researchers also found that cows that had mastitis at the end of lactation had a lower incidence of clinical mastitis in the subsequent lactation if all quarters were treated rather than only infected quarters. A subsequent study[15] by the same researchers showed that selective treatment of infected cows (all quarters) resulted in use of fewer treatment tubes per infection cured than did blanket treatment of all cows (all quarters) or selective treatment of only infected quarters in infected cows. They also found, however, that the prevalence of infection during the first 3 months of lactation was almost the same for all treatment groups, and they concluded that a reduction in the prevalence of infection from 1 lactation to the next would require more than just antimicrobial treatment of nonlactating cows. In the present study, we did not have data on infection status at the time of assignment to treatment groups. In large dairy herds, it would be impractical to implement selective treatment of infected cows or selective treatment of infected quarters, because bacteriologic culture of samples collected prior to the end of lactation would be difficult and more expensive than blanket treatment of all cows. Researchers[16] in the Netherlands found a high incidence of clinical mastitis during the early nonlactating period in cows that were not treated at the end of lactation. They postulated that milk leakage might have been a risk factor for clinical mastitis during the nonlactating period, because they observed that cows that leaked milk had a high incidence of clinical mastitis. Management of large dairies does not lend itself to frequent observation of nonlactating cows or collection of samples for bacteriologic culture during the early part of the nonlactating period, because lactation is usually stopped abruptly and cows are moved to a pen where they are infrequently observed prior to calving.

The finding that there was no difference in time to first development of mastitis between treated and control cows in the study reported here agrees with that of others.[13,14] New Zealand researchers[17] found that cows that were not infected at the end of lactation had a lower incidence of clinical mastitis attributable to *Streptococcus uberis* when treated at the end of lactation than when not treated, but that treatment did not reduce the incidence of clinical mastitis in cows that were infected at the end of lactation. Incidence of clinical mastitis after treatment of cows at the end of lactation may vary with product used and specific bacteria causing mastitis.[7,18]

Harmon et al[7] found that treatment of nonlactating cows was effective in eliminating infections caused by minor pathogens during the nonlactating period, but they did not detect any difference in 305ME milk production. Smith et al,[8] however, reported that quarters that became infected during the nonlactating period produced less milk during the subsequent lactation. It would seem reasonable that antimicrobial treatment at the end of lactation cured some infections or prevented some of the early, transient infections that would be detrimental to udder involution and early lactation milk yield. In the present study, after adjustment for milk production in the previous lactation, treated cows produced 179 kg (394 lb) more milk than did control cows during the first 17 weeks of lactation. The value of the additional 179 kg of milk would be $43.40 (at a rate of $11.00/45.4 kg [$11.00/100 lb]), assuming that there were no additional feed or other costs. Estimated cost would be approximately $6.00/treatment ($4.50 for antimicrobial tubes, $1.50 for labor). In other words, it would take about 25 kg (55 lb) of extra milk to pay for the cost of nonlactating cow treatment. On the basis of results from this herd, we anticipate that herds with a low prevalence of contagious pathogens may benefit from treatment of all cows with antimicrobials at the end of lactation.

[a]*Escherichia coli* bacterin J-5 strain, The Upjohn Co, Kalamazoo, Mich.
[b]Albadry plus suspension, The Upjohn Co, Kalamazoo, Mich.
[c]AgriTech Analytics, Tulare, Calif.
[d]Dairy Comp 305, Valley Agricultural Software, Tulare, Calif.

References

1. Heald CW, Jones GM, Nickerson SC, et al. Mastitis control by penicillin and novobiocin at drying-off. *Can Vet J* 1977;18:171–180.

2. Kirk JH, Degraves F, Tyler J. Recent progress in treatment and control of mastitis in cattle. *J Am Vet Med Assoc* 1994;204:1152–1158.

3. National Mastitis Council. *Dry cow therapy*. Bulletin C-12254. Arlington, Va: National Mastitis Council Inc, 1995.

4. Miller GY, Bartlett PC. Economic effects of mastitis prevention strategies for dairy producers. *J Am Vet Med Assoc* 1991;198:227–231.

5. Oliver SP. Importance of the dry period in the control of intramammary infections by environmental mastitis pathogens, in *Proceedings*. Annu Meet Natl Mastitis Counc 1987;81–92.

6. Smith KL, Hogan JS, Schoenberger PS, et al. A practical look at environmental mastitis, in *Proceedings*. Annu Meet Natl Mastitis Counc 1987;102–109.

7. Harmon RJ, Crist WL, Hemken RW, et al. Prevalence of minor udder pathogens after intramammary dry treatment. *J Dairy Sci* 1986;69:843–849.

8. Smith A, Dodd FH, Neave FK. The effect of intramammary infection during the dry period on the milk production of the affected quarter at the start of the succeeding lactation. *J Dairy Res* 1968;35:287–290.

9. Brown RW, Barnum DA, Jasper DE, et al. *Microbiological procedures for use in the diagnosis of bovine mastitis*. Washington, DC: National Mastitis Council, 1981;1–35.

10. Matthews KR, Oliver SP, Jayaroa BM. Susceptibility of staphylococci and streptococci isolated from bovine milk to antibiotics. *Agri-Practice* 1992;13:18–24.

11. Kaplan EL, Meier P. Non-parametric estimation from incomplete observations. *J Am Stat Assoc* 1958;53:457–481.

12. Dixon WJ. *BMDP statistical software manual*. Los Angeles: University of California Press, 1992.

13. Bartlett PC, Miller GY, Lance SE, et al. Environmental and managerial determinants of somatic cell counts and clinical mastitis incidence in Ohio dairy herds. *Prev Vet Med* 1992;14:195–207.

14. Browning JW, Mein GA, Barton M, et al. Effects of antibiotic therapy at drying off on mastitis in the dry period and early lactation. *Aust Vet J* 1990;67:440–442.

15. Browning JW, Mein GA, Brightling P, et al. Strategies for mastitis control—dry cow therapy and culling. *Aust Vet J* 1994;71:179–181.

16. Schukken YH, Vanvliet J, Vandegger D, et al. A randomized blind trial on dry cow antibiotic infusion in a low somatic cell count herd. *J Dairy Sci* 1993;76:2925–2930.

17. Williamson JH, Woolford MW, Day AM. The prophylactic effect of a dry-cow antibiotic against *Streptococcus uberis*. *N Z Vet J* 1995;43:228–234.

18. Hogan JS, Smith KL, Todhunter DA, et al. Efficacy of dry cow therapy and a *Propionibacterium acnes* product in herds with low somatic-cell count. *J Dairy Sci* 1994;77:3331–3337.

From My Armchair: W. W. Armistead

Myths of academia

Humorist Josh Billings said, "The trouble with most people is they know so many things that ain't so." We tend to see what we are looking for. We believe things because we want to believe them, often in the absence of proof—sometimes even in the face of evidence to the contrary.

Grades earned in college have little or no relationship to career success—This is one of academia's most durable myths. Of course, most of us know highly successful people who were poor students in college. And there may be a few individuals with PhD degrees on skid row or in prison. But these are exceptions. Still valid is the general rule that better students tend to be more successful in their later careers. This should not be so surprising; the success shown in coping with tasks in college tends to be characteristic of the success shown in dealing with life's other challenges. Both are to a large extent measures of motivation.

Small liberal arts colleges provide a better education than do large, state universities—Like most generalizations, this one breaks down under close examination. There are poor small colleges and excellent large universities. True, it probably could be demonstrated that the typical graduate of a Harvard or Brown is more successful than is the typical graduate of a Colossal State or Podunk Poly—if "typical" is understood to mean "average." But the Harvards and Browns are much more selective in student admissions. When only top graduates are compared, institutional differences tend to disappear.

Good researchers generally are poor teachers and good teachers generally are poor researchers—Put either way, this is nonsense. Enthusiasm for the subject, an inquiring mind, and a propensity for problem solving are desirable traits both of teachers and of researchers. What it all boils down to is that many faculty members are very good at both teaching and research, some are better at one or the other, and some, alas, are not very good at either. A remarkably human distribution of talents!

A pass is a pass is a pass—The belief is widespread that the time required to learn a subject is relatively unimportant—that, so far as acquiring knowledge is concerned, a pass after two or three tries is about as good as a pass on the first try. One might even argue that more than one exposure to the same course results in better education. This may be true for courses like history or English grammar, which do not involve problem solving. But in veterinary education, more is involved than just mastery of course material. Performance in veterinary courses also gives clues to the student's mental quickness, grasp of fundamentals, and ability to synthesize.

If ever I am taken suddenly, dangerously ill, I hope that the physician summoned for me will have graduated near the top of the class—and be in the habit of getting things right the first time.

W. W. Armistead