colorectal cancer may join the group of diseases associated with HLA-B27. The increase in the frequency of this histocompatibility antigen was not higher in familial forms of colorectal cancer; the frequency of radiologic signs of ankylosing spondylitis among B27-positive patients with colonic cancer (three of 16) was comparable to that found by some of us among B27-positive healthy blood donors (16 of 40),[4] suggesting that the existence of colorectal cancer in B27-positive people has no influence on the incidence of radiologic signs of sacroiliitis.

These results raise the question of a possible causal role of B27 antigen in colonic carcinoma and ankylosing spondylitis. Since some microbial species (e.g., klebsiella) are thought to have a responsibility in the genesis of ankylosing spondylitis,[5] and since some others (*Streptococcus faecalis* and clostridia) are thought to be colonic carcinogens,[6,7] it is conceivable that B27 antigen favors some intestinal dysmicrobisms that would lead to different specific diseases. Finally, it is noteworthy that another potential colonic carcinogen, asbestos,[8] can give rise to another B27-associated disease, asbestosis.[9]

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PYRIDOXINE (B6) SUPPRESSES THE RISE IN PROLACTIN AND INCREASES THE RISE IN GROWTH HORMONE INDUCED BY EXERCISE

To the Editor: The role of monoamines in control of the secretion of anterior pituitary hormones has been widely studied in the past few years.[1] The hypothalamic dopaminergic pathways have been shown to be involved in the regulation of prolactin, growth hormone, ACTH, and gonadotropins.[2]

Pyridoxine (vitamin B_6) is one of the natural precursors of pyridoxal-5′-phosphate coenzyme in the decarboxylation and transamination of amino acids. As a coenzyme of dopa decarboxylase, this vitamin can induce the transformation of dopa to dopamine, thus sharing the endocrine effects of this amine. In fact, this drug has been reported to reduce prolactin levels and raise growth hormone levels,[3] although this action has not been confirmed.[4]

We conducted a study to evaluate the effect of pyridoxine on prolactin and growth hormone levels during exercise, since exercise provides a good model for the physiologic release of the adenohypophyseal hormones.[5,6] Six healthy subjects 25±3 years old volunteered to enter the study. Informed consent was obtained from all subjects. Each volunteer was tested at two different times separated by a seven-day interval (once with drug and once with placebo).

Physical exercise, performed on a bicycle ergometer, was monitored by an open-circuit gas analyzer. A spirometer included in the system was used to evaluate the static and dynamic lung volumes.[5]

Table 1. Plasma Levels of Growth Hormone and Prolactin before and after Exercise, Performed in Subjects Given a Saline or Pyridoxine Infusion.

Time	Growth Hormone *	
	with saline	with pyridoxine
	ng/ml	
Before exercise		
20 min before	2.2±0.9	4.8±0.8
At start (basal)	1.9±0.8	4.5±1.4
After exercise		
5 min	1.8±4.6	2.2±5.1
15 min	25.5±7.4	81.6±20.4 †
30 min	20.1±6.8	41.9±6.2 †
45 min	12.4±4.2	27.4±5.3 †
60 min	6.11±1.8	17.0±4.2 †
	Prolactin *	
	with saline	with pyridoxine
	ng/ml	
Before exercise		
20 min before	7.2±1.6	3.8±0.9
At start (basal)	5.4±1.4	4.5±0.8
After exercise		
5 min	16.9±2.1	4.8±0.9 †
15 min	23.0±4.1	3.2±0.6 †
30 min	12.1±1.6	3.8±0.6 †
45 min	6.4±0.4	4.8±0.9
60 min	6.1±1.4	4.0±1.2

*Mean ±S.D. in six subjects.
†Significantly different from value with saline infusion ($P<0.001$, Student's t-test).

The bicycle workload was adjusted so that the heart rate, monitored from a V_5 lead, was constant at a value corresponding to 80 per cent of the maximum heart rate of each subject (about 170 beats per minute), during the eight minutes of maximum workload. The exercise phase was preceded by a 20-minute rest period and followed by a 45-minute recovery phase. Saline or 600 mg of pyridoxine in 250 ml of saline was given by infusion for the duration of the test (rest, exercise, and recovery phases).

The subjects were not told which infusion was given. A catheter was introduced into a forearm vein to collect blood at the beginning of the 20-minute rest period, immediately before the start of exercise, and then five, 15, 30, 45, and 60 minutes after the end of the exercise period. After blood collection the test tubes were placed on ice; the plasma was immediately separated and frozen at −70°C. Levels of growth hormone and prolactin were measured by means of sensitive and specific radioimmunoassays.[7]

The results, analyzed with Student's t-test, are shown in Table 1. A significant increase in growth hormone and prolactin occurred in response to maximal effort during the test performed after the saline infusion. The hormonal response to physical exercise performed after the pyridoxine infusion showed that prolactin response was suppressed ($P<0.001$) whereas the growth hormone response was enhanced ($P<0.001$).

The present data indicate the efficacy of vitamin B_6 in stimulating growth hormone and suppressing the rise in prolactin induced by exercise. This phenomenon is probably due to the increase of dopaminergic activity by pyridoxine.[3]

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STORAGE OF VANCOMYCIN ORAL SOLUTION

To the Editor: According to the manufacturer's recommendation, vancomycin oral solution is stable for seven days under refrigeration.[1] Since vancomycin is a very expensive drug, a study was initiated to determine the stability of reconstituted oral vancomycin hydrochloride at 0°C, 4°C, and 25°C for periods of up to 90 days after repackaging of the solution in amber-glass unit-dose vials.

A high-pressure liquid chromatographic procedure has been described for assaying vancomycin[2,3]; a modified assay was used to analyze the vancomycin samples. A bottle of vancomycin was repackaged in 50 unit-dose vials, which were stored at 0°C, 4°C, and 25°C. Two of the unit-dose vials stored at 4°C and 25°C were analyzed on each of the following: Days 0, 3, 6, 7, 18, 20, 35, 58, 85, and 90. Two unit-dose vials kept at 0°C were analyzed on Days 7, 18, 35, 58, and 90.

The results of this study (Table 1) indicated that reconstituted oral vancomycin solution was stable for 58 days at room temperature (25°C) and for at least 90 days under refrigeration (4°C) and freezing (0°C). However, at room temperature, a precipitate was observed on Day 6 in the vials. After filtration of the precipitate, no decrease in the potency of the vancomycin was found until Day 58 (room temperature), when degradation was detected by chromatographic determination. An increase in the pH of the vancomycin solution was observed over the study period; the greatest increase occurred in the solution maintained at room temperature. The microbiologic activity of the vancomycin samples maintained at room temperature was determined on Days 0, 3, 21, and 35. The samples were tested against *Staphylococcus aureus* penicillinase-producing organisms. The results from the assay indicated a retention of 98 per cent activity by the four samples tested. No decrease in activity was observed.

The data presented in this study indicate that reconstituted oral vancomycin solution can be stored for a period of at least 90 days at 0°C and 4°C without an appreciable loss in potency. (The *United States Pharmacopoeia* [20th ed.] indicates that oral vancomycin solution should contain the equivalent of not less than 90.0 per cent and not more than 115.0 per cent of the labeled amount of vancomycin.) It is not recommended that the solution be stored at room temperature, since we observed a precipitate on Day 6 of the study. Savings can be made if the unused portion is stored at 0°C and 4°C.

Table 1. Stability of Reconstituted Oral Vancomycin Stored at 0°C, 4°C, and 25°C.

No. of Days Stored	(25°C) Room Temperature		(4°C) Refrigeration		(0°C) Freezing	
	pH	*% retained*	*pH*	*% retained*	*pH*	*% retained*
0	3.65	100.0	3.65	100.0	3.65	100.0
3	3.67	100.2	3.72	102.8		
6	3.76	98.5	3.79	101.3		
7	3.75	100.9	3.70	100.1	3.74	99.7
18	3.98	98.3	3.95	102.1	3.96	101.7
20	3.96	102.3	3.86	107.4		
35	4.03	97.1	3.82	102.4	3.83	103.2
58	4.30	93.2	3.92	100.0	3.96	97.6
85	4.28	96.7	3.88	102.7		
90	4.32	83.8	3.92	96.6	3.90	98.8

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A MISINTERPRETATION OF THE EFFECTS OF NALOXONE ON SENSITIVITY TO CO_2

To the Editor: Chernick erred in his interpretation (October 15, 1981, issue[1]) of our results in determining the effect of naloxone on carbon dioxide responsivity in fetal lambs,[2] and he has since acknowledged this fact to us. The correct citation and interpretation of these results are that although the carbon dioxide sensitivity of previously apneic fetuses was 57.8 ± 13.7 Σtorr/min · PCO_2 torr^{-1}, (Table 1 of our paper), naloxone increased this sensitivity by 56.7 ± 12.5 Σtorr/min · PCO_2 torr^{-1}. To quote from our paper (Table 2) the latter figure is "based on paired comparison analysis between fetal response to CO_2 in the presence of . . . naloxone administration on the one hand, and fetal response to CO_2 alone, on the other." Thus, naloxone produces a significantly greater response ($P<0.025$ [Table 2]) than that obtained with carbon dioxide alone.

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WHAT'S WRONG WITH RADIOLOGY

To the Editor: In the February 25 issue Dr. Richard Heilman correctly evaluated the dangers of "bigness" in the hospital radiology department.*

However, what Heilman is obviously unaware of is that most radiologists in moderate-sized hospitals have plenty of opportunity and take the time to establish rapport with referring physicians. In doing so, we radiologists practicing in 300-bed to 400-bed community hospitals spend a great deal of effort advising our clinicians on just which examinations will be most suitable in the clinical setting. These recommendations are made in formal lectures, on an individual consulting basis, and within the body of the radiology report.

Furthermore, in the groups with which I am familiar, most radiologists are trained to interpret nuclear scans, ultrasound scans, and CT scans in addition to other routine x-ray procedures. Thus, we are able not only to correlate for ourselves the radiologic findings but also to present them to the clinician in a comprehensive manner. There is therefore no need for the clinician to wander from one "imaging" department to the other, and there is very little reduplication in the diagnostic workup.

*Heilman RS. What's wrong with radiology. N Engl J Med. 1982; 306:477-9.