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OB/GYN CLINICAL ALERT®

A monthly update of developments in female reproductive medicine

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Risk of Leukemia After Platinum-Based Chemotherapy for Ovarian Cancer

ABSTRACT & COMMENTARY

Synopsis: *Platinum-based chemotherapy increases the risk of secondary leukemia. Nevertheless, the substantial benefit that platinum-based treatment offers patients with advanced disease outweighs the relatively small excess risk of leukemia.*

Source: Travis LB, et al. *N Engl J Med* 1999;340:351-357.

Platinum-based chemotherapy is the standard postoperative treatment for most patients with epithelial ovarian cancer. Although there is extensive information regarding the risk of leukemia after chemotherapy for ovarian cancer, it principally relates to alkylating-agent chemotherapy. Except for case reports, there has been no large-scale study of the risk of leukemia in association with platinum-based chemotherapy. Travis and colleagues have conducted a case-control study of secondary leukemia in a population-based cohort of 28,971 women in North America and Europe who had received a diagnosis of invasive ovarian cancer between 1980 and 1993. Leukemia developed after the administration of platinum-based therapy in 96 women. These women were matched to 272 control patients. The type, cumulative dose, and duration of chemotherapy and the dose of radiation delivered to active bone marrow were compared in the two groups. Among the women who received platinum-based combination chemotherapy for ovarian cancer, the relative risk of leukemia was 4.0. The relative risks for treatment with carboplatin and for treatment with cisplatin were 6.5 and 3.3, respectively.

Travis et al found evidence of a dose-response relation, with relative risks reaching 7.6 at doses of 1000 mg or more of platinum. Radiotherapy without chemotherapy did not increase the risk of leukemia. Travis et al conclude that platinum-based treatment of ovarian cancer increases the risk of secondary leukemia. However, they found that the substantial benefit that platinum-based treatment offers patients with advanced disease outweighs the relatively small excess risk of leukemia.

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INSIDE

COMMENT BY DAVID M. GERSHENSON, MD

Platinum-based chemotherapy is the mainstay of postoperative treatment for the majority of patients with epithelial ovarian cancer. In the United States, the standard practice is to advise this therapy for all such patients, except those with low-risk, early-stage disease. For several years, we have known that alkylating agent chemotherapy-standard treatment for ovarian cancer is particularly associated with an increased risk of secondary leukemia. Certain drugs are more likely to lead to secondary acute nonlymphocytic leukemia than are others. For example, prior studies have indicated, for instance, that melphalan is more leukemogenic than cyclophosphamide. We know that, by its mechanism action of producing intrastrand and interstrand cross-links, platinum drugs function much like alkylating agents. Both carboplatin and cisplatin were found to be associated with an increased risk of leukemia. Intuitively, it makes sense that carboplatin was more leukemogenic than cisplatin since its effects are much more pronounced on the bone marrow. And the same trend has held, namely, that cumulative dose and duration appear to influence the degree of risk. It is important to note that Travis et al have placed their results in the proper context—the substantial benefit of platinum-based chemotherapy far outweighs the small excess risk of leukemia. Nevertheless, this report will evoke anxiety among ovarian cancer patients and their families. ❖

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Luteal Phase Deficiency and Anovulation in Recreational Women

ABSTRACT & COMMENTARY

Synopsis: *In sedentary women, 90% of all menstrual cycles were ovulatory, but in exercising women, only 45% were ovulatory. Of the remainder, 43% of cycles demonstrated luteal phase deficiency and 12% were anovulatory.*

Source: De Souza MJ, et al. *J Clin Endocrinol Metab* 1998;83:4220-4232.

The purpose of this study was to determine menstrual cycle characteristics in women participating in moderate, recreational exercise who had regular menstrual cycle intervals. For three consecutive menstrual cycles, subjects collected daily urine samples for analysis of follicle stimulating hormone (FSH), estrone conjugates, pregnanediol-3-glucuronide, and creatinine. Urinary estrone is a metabolite of and marker for estradiol. Pregnanediol is a metabolite of and marker for progesterone. The 11 sedentary women and 24 recreational exercisers were similar in age, weight, gynecologic age, and menstrual cycle length. Despite regular menstrual cycle intervals of 28.8 days, 10% of sedentary women consistently had luteal phases that were less than 10 days and associated with decreased peak pregnanediol (< 3 mcg/mg creatinine). None of the sedentary women had anovulatory cycles. In comparison, recreational exercisers were more likely to display ovarian compromise (55%) than to meet criteria for ovulation (45%). In exercising women, energy availability was most decreased in those with anovulatory cycles. Menstrual interval was preserved in women with luteal phase deficiency because follicular phase lengths were longer when luteal phase durations were shorter. As a group, exercisers ran 17 miles a week and participated in five hours of other exercise weekly.

COMMENT BY SARAH L. BERGA, MD

Is energy availability the prime regulator of reproductive function? At least two types of reproductive compromise are due to altered metabolism. Polycystic ovary syndrome is associated with excessive energy efficiency and a proclivity toward weight gain. In the absence of insulin resistance, which is primarily a function of excess weight, women with polycystic ovary morphology remain ovulatory.[1] Thus, too much energy availability can lead to reproductive compromise and increase the risk of diabetes in women with "thrifty metabolism."

Most women who experience restricted energy availability develop reproductive compromise. The only exception might be women with polycystic ovary syndrome. This study demonstrated that even when the menstrual cycle interval is preserved, restricted energy availability due to moderate running and recreational exercise can compromise ovulatory function. This result is not entirely unanticipated, but it is alarming. What should we tell young women? Certainly, those attempting conception might want to reduce or even cease recreational running because the occurrence of regular cycles does not mean that ovulatory function is sufficient to support implantation. What about those not immediately interested in pregnancy? It is even more difficult to know what to say. Does luteal phase deficiency due to recreational exercise have long-term deleterious consequences such as reduced bone accretion? In a separate study, De Souza and associates found no decrement in bone density as long as estrogen levels were not diminished.[2] Perhaps restricted energy availability confers protection against cardiovascular disease and cancer by reducing the "stress" of oxidative metabolism. If an exercising woman with regular menstrual cycles is not seeking pregnancy, it seems doubtful that her reproductive status would be an issue for the patient or physician. Thus, most reproductive compromise in exercising women is likely to escape clinical detection. Until deleterious consequences are revealed, and in the absence of oligomenorrhea or amenorrhea, it is prudent not to scare women by advising against exercise. ❖

References

1. Robinson S, et al. *Clin Endocrinol* (Oxf) 1993; 39:351-355.
2. De Souza MJ, et al. *J Clin Endocrinol Metab* 1997;82:2867-2876.

VBAC Success After Failure to Progress in the Second Stage

ABSTRACT & COMMENTARY

Synopsis: *Women have an excellent chance for a successful vaginal birth after a prior cesarean delivery.*

Source: Jongen VHWM, et al. *Br J Obstet Gynaecol* 1998;105:1079-1081.

To determine the likelihood of a vaginal birth after a prior cesarean delivery (VBAC) performed for delay in descent in the second stage of labor, Jongen and colleagues performed a retrospective follow-up study of primiparous women who delivered at a medical center in the Netherlands between 1986 and 1998. Women were not included in the study if they had a cesarean delivery early in the second stage or because of a non-reassuring fetal heart rate tracing. Of 132 women eligble for the study, 29 had a repeat cesarean delivery while 103 elected to have a trial of labor. Eighty-two (80%) were successful, while 21 (20%) required a second cesarean delivery. Forty-one of 55 women (75%) who had experienced a failed instrumental delivery during their prior labor had a successful VBAC. One uterine dehiscence occurred. No difference was noted in mean birth weight between those patients who had an elective cesarean delivery and those who underwent a trial of labor.

Jongen et al conclude that women who had an arrest of descent in the second stage of labor requiring a cesarean section for their first delivery, including women who had a failed instrumental vaginal delivery birth, have an excellent chance for a successful vaginal birth after a prior cesarean delivery.

COMMENT BY STEVEN G. GABBE, MD

This study from the Netherlands confirms several earlier reports, namely, that women who have required cesarean delivery for failure to progress after reaching full dilatation have a higher probability of a successful VBAC. Even women whose history included a prior failed instrumental birth delivery had a success rate of 75%. These findings are in conflict with those of Hoskins and colleagues who reported a 13% success rate in women who required a cesarean delivery at full dilatation.[1] However, that group included only 32 women. Hoskins et al did note that women whose first cesarean delivery was performed at 5 cm or less had a successful VBAC rate of 67%, and those who were delivered by cesarean section at a dilatation of 6-9 cm had a 69% success rate. Jongen et al do not provide data on the difference in birth weight between babies delivered by cesarean section in the index pregnancy and those delivered after a successful VBAC. Women who have required a cesarean delivery after reaching full dilatation are often reluctant to undergo a trial of labor. Despite this fact, the study is very encouraging. ❖

Reference

1. Hoskins IA, et al. *Obstet Gynecol* 1997;89:591-593.

Estrogen Supplementation Attenuates Glucocorticide and Catecholamine Responses to Mental Stress in Perimenopausal Women

ABSTRACT & COMMENTARY

Synopsis: *Estrogen supplementation of hypoestrogenic perimenopausal women attenuated blood pressure, cortisol, and catecholamine responses to acute psychological challenge.*

Source: Komesaroff PA, et al. *J Clin Endocrinol Metab* 1999;84:606-610.

The present study tested the hypothesis that estradiol reduces cortisol and catecholamine responses to stress. Twelve women within two years of their last menses with hot flashes were designated as being perimenopausal. Women with known cardiovascular disease, including hypertension, were excluded. These women were then randomized to 12 weeks of estradiol valerate, 2 mg daily by mouth, or placebo. Outcome variables that were determined before and after estradiol use included cortisol, adrenocorticotropic hormone (ACTH), epinephrine, norepinephrine, blood pressure, and heart rate. Estradiol levels rose from about 35 pg/mL to 250 pg/mL, which is well above physiological levels. After estradiol supplementation, the increases in both systolic and diastolic blood pressure in response to mental stress were reduced, and cortisol, ACTH, epinephrine, and norepinephrine responses were attenuated.

COMMENT BY SARAH L. BERGA, MD

There are several mechanisms by which estrogen protects against cardiovascular disease. One of these is thought to be reduced endocrine and vascular reactivity to psychological challenge. Indeed, estrogen exerts direct effects upon the vessel wall and vasomotor tone. Previous studies by Komesaroff and colleagues found that estrogen supplementation of perimenopausal women enhanced basal nitric oxide release from the vessel wall and reduced norepinephrine-induced vasoconstriction. In the present model, Komesaroff et al extend their previous work by asking whether estrogen supplementation alters cardiovascular and endocrine responses to psychological challenge.

The use of a challenge paradigm has advantages over studies in which a given vasoconstrictor agent is directly infused. Multiple factors regulate vasomotor tone and cardiovascular reactivity, and psychological challenge is thought to activate all or many of these mechanisms, including cortisol and catecholamine release. Further, basal endocrine and cardiovascular parameters do not adequately reflect what happens when an individual is confronted by the mundane trials of daily living. Therefore, to better approximate what happens in response to minor stress, Komesaroff et al used a psychological challenge, performing difficult arithmetic tasks in a distracting milieu.

Komesaroff et al found that estrogen administration reduced cardiovascular and endocrine reactivity. They suggested a direct link between reduced adrenal secretion and reduced blood pressure during challenge. While their discussion focused on the implications of reduced endocrine reactivity for cardiovascular risk, reduced cortisol and catecholamine secretion in response to mundane challenges could have other benefits as well. Another likely benefit of reduced endocrine reactivity is a lower risk of depression and dementia. Bone health is reduced by chronic glucocorticoid elevations, even when the glucocorticoid exposure is from an endogenous source. Chronic adrenal activation leads to reproductive compromise and hypothalamic hypothyroidism. Animal tests suggest that sustained increases in endocrine reactivity accelerate the aging process in general, possibly by increasing programmed cell death (apoptosis). Obviously, it is impossible to rid ones life of stress, so the prudent course is to take measures to reduce ones endocrine and cardiovascular reactivity to such pressures. While many of the ways to reduce mental stress involve psychological mechanisms such as "attitude readjustment" (in hypogonadal women at least), one should ensure that estrogen levels are adequate.

This study nicely demonstrates the profound effect of estrogen administration upon cardiovascular and endocrine reactivity, but, like all good studies, it raises certain questions. As Komesaroff et al point out, the effect of progestins in this model have not been determined. Perhaps more important, the effect of phytoestrogens, tamoxifen, or raloxifene upon these parameters should be studied. Many women have chosen these and other estrogen alternatives in the belief that their use would confer the benefits of estrogen while reducing the risk of breast cancer. Given that we are still engaged in specifying the multiple mechanisms underlying the benefits (and risks) of postmenopausal estrogen use, it is impossible to know what to expect from the long-term use of estrogen alternatives. However, the development of investigative paradigms, such as the one in this study, may well allow for informative comparisons that are less

labor- and time-intensive than long-term, large-scale epidemiological trials. ❖

Latest Bone Data from the Annual Meeting of the American Society for Bone and Mineral Research

CONFERENCE COVERAGE

By Leon Speroff, MD

The reports at the annual meeting of the American Society for Bone and Mineral Research in December 1998 provide us with the latest data from the on going studies of alendronate and raloxifene.

The effect of alendronate on osteoporosis-related fractures was reported from the follow-up of 4432 women for an average of 4.2 years.[1] A statistically significant reduced risk of fracture was demonstrated only in women with initial T-scores of -2.5 or less, with 36% reduction in all fractures, and a 50% reduction in vertebral fractures. Calcium and vitamin D supplementation with alendronate treatment had no added effect as long as women have a minimal intake of 800 mg daily. (*See Table 1.*)[2]

Table 1

Effect of Alendronate on Osteoporosis-Related Fractures

T-Score	Vertebral Fractures	All Fracture Fractures
Less than -2.5	0.50 (0.31-0.82)	0.64 (0.50-0.82)
-2.5 to-2.0	0.54 (0.28-1.04)	1.03 (0.77-1.38)
-2.0 to -1.6	0.82 (0.33-2.07)	1.14 (0.82-1.60)

Adapted from: Bonnick S, et al. 1998;http://www.asbmr.org.

There are two important interpretations of these data:

- Treatment obviously benefits women who already have an osteoporotic low bone density or previous vertebral fractures, meaning, women who already have osteoporosis; and
- If alendronate benefits women who do not already have osteoporosis, it will take more than four years of treatment to observe the effect. This is a reasonable prediction, based on the recognized positive impact of alendronate on bone density.

The Early Postmenopausal Interventional Cohort (EPIC) study indicated that over a four-year period, alendronate and hormone therapy in the United States produced similar bone density results.[3] The greater increase noted in Europe with hormone therapy probably reflects the use of 19-nortestosterone progestins, which are known to have an additive effect on bone density when combined with estrogen. (*See Table 2.*)

Table 2

Results of the EPIC Study

	Spine	Hip
Placebo	-2.65%	-1.69%
Alendronate, 5 mg	+3.78%	+2.89%
E/P, USA	5.15	2.78
E/P, Europe	7.55	4.20

Adapted from: Ravn P, et al. 1998;http://www.asbmr.org.

Combining alendronate and hormone therapy produces an added gain in bone density. When women who were already taking hormone therapy also received alendronate (10 mg) for one year, the gain in bone density ranged from 0.9% in the femoral neck to 2.6% in the spine.[4] In women with osteopenia, combined therapy with alendronate 10 mg and 0.625 mg conjugated estrogens produced a 1-2% greater gain in bone density over a two-year period of treatment.[5] However, it is by no means certain that these findings will translate into a difference in the incidence of fractures later in life. Indeed, it is unlikely. There is a further theoretical concern that oversuppression of resorption can ultimately yield more brittle bones. This emphasizes the importance of long-term follow-up studies with fracture outcomes. Unfortunately, most bone studies end after about four years when bone density differences are demonstrated.

Compliance with alendronate has been overestimated by the clinical trials. It is well-recognized that participants in clinical trials are better motivated, better supported, and perform better. In the Kaiser Permanente Medical Care Program in California, about one-third of patients had acid-related complaints and one in eight required treatment.[6] About 50% of patients do not comply with instructions and about 50% discontinue therapy by one year.[6,7] These compliance data are a strong argument for the use of bone marker or bone density measurements to assess the effectiveness of treatment.

The Multiple Outcomes of Raloxifene Evaluation (MORE) study of raloxifene administration to osteoporotic women has now accumulated results from two and three years of follow-up. (*See Table 3.*)[8]

Table 3

Results of the MORE Study

	Vertebral Fractures	Nonspinal Fractures
Low T-score	0.53 (0.36-0.80)	0.97 (0.72-1.15)
Previous vertebral fracture	0.52 (0.41-0.65)	0.97 (0.73-1.28)

Adapted from: Ensrud KE, et al. Bone 1998;23(Suppl 5):S174.

Women with low T-scores or previous vertebral fractures have approximately a 50% reduction in vertebral fractures with raloxifene treatment. Thus far, there has been no evidence of a reduction in hip fractures. The reduction in vertebral fractures is similar to that seen with alendronate. Why is there no decrease in hip fractures, despite a bone density response that is only slightly less than that associated with alendronate? Is it because the slight difference in bone density comparing raloxifene with alendronate is clinically significant?[9,10] Like alendronate, we have no fracture data in treated women who originally had normal bone densities. Will the effect of alendronate and raloxifene in osteoporotic women be similar to that achieved with long-term treatment of normal women?

A Scandinavian assessment of cost effectiveness concluded that it is not cost effective to treat a 55-year-old woman of average risk for fractures if the only benefit is skeletal.[11] If this woman already has osteopenia, then it is cost effective, and a treatment that has multiple benefits. (*See Table 4.*)

Table 4

Cost of Treating a 55-year-old Woman:

Effect	Cost Per Year of Life Gained
Hip fracture only	$250,000
Hip fx + CVD	$87,000
Hip fx + CVD + breast	$35,000
Hip fx only, osteopenia	$23,500

Adapted from: Jönsson B, et al. Bone 1998;23(Suppl 5):S203.

These data reinforce my belief that the treatment of choice for the early postmenopausal years (age 50-65) is hormone therapy because of its broad spectrum of benefits—most notably, symptomatic relief and protection against cardiovascular disease and also because we have no current data that confirm alendronate and raloxifene given to women with normal bone densities will prevent fractures in old age and, if they do, how they compare to hormone therapy. Around age 65, I recommend measurement of bone density. Low bone density should be treated with a drug chosen during a clinician-patient dialogue reviewing the advantages and disadvantages of each drug (estrogen, alendronate, raloxifene). ❖

References

1. Cummings SR, et al. *JAMA* 1998;280:2077-2082.
2. Bonnick S, et al. 1998;http://www.asbmr.org.
3. Ravn P, et al. 1998;http://www.asbmr.org.
4. Lindsay R, et al. *Osteoporos Int* 1998;8(Suppl 3):12.
5. Greenspan S, et al. *Bone* 1998;23(Suppl 5):S174.
6. Ettinger B, et al. 1998;http://www.asbmr.org.
7. Faulkner DL, et al. *Osteoporos Int* 1998;8(Suppl 3):21.
8. Ensrud KE, et al. *Bone* 1998;23(Suppl 5):S174.
9. Meunier PJ, et al. *Bone* 1998;23(Supp 5):S295.
10. Bjarnason NH, et al. *Osteoporos Int* 1998; 8(Suppl3):11.
11. Jönsson B, et al. *Bone* 1998;23(Suppl 5):S203.

Special Feature

Primary Care in Obstetrics and Gynecology

By Kenneth Noller, MD

There are few topics that engender as much debate among obstetrician-gynecologists as the decision to include training in primary care in ob/gyn residency programs. Even though such training has been in all programs for several years, and despite the fact that there is absolutely no serious discussion about removing the requirement from residency training, many practicing ob/gyns do not understand why surgical specialists are required to learn about such things as immunizations, diabetes, hypertension, and other responsibilities. Perhaps the reasons behind this decision can be understood better by reviewing the concept of primary care in more detail.

A dramatic change in healthcare in the United States occurred shortly after the end of World War II. The advances in the biological sciences along with advances in surgical techniques (which had their roots in World War II) caused more and more physicians to seek specialty training rather than pursuing the time-honored "general practitioner" career. Specialty training soon spawned subspecialty training, and physicians eventually became so subspecialized that, often, they failed to view the patient as a whole. Patients became more and more dissatisfied with the fragmentation of their medical care; they might be forced to see five or six different physicians to accomplish little more than an annual health assessment.

Of course, there were still primary care physicians, and some prestigious institutions such as the Mayo Clinic recognized that each patient must have one physician who remained in overall control of the individual patients care. Approximately 30 years ago, the field of family practice was recognized as a separate medical specialty, primarily because of the large void that had been created by subspecialization. This group of physicians came forward and said "we can manage over 80% of a patients medical problems." This specialty became successful and was quickly joined by primary care internists. These physicians became the main providers of first contact healthcare for men and women in the United States.

During this same time period, ob/gyns became more scientific, established three subspecialties, and focused on operative deliveries and pelvic surgery. However, advances in medical care have dramatically reduced the need for hysterectomy, operative deliveries other than cesarean sections have almost vanished in many parts of the United States, Pap smear screening has markedly reduced the incidence of cervical cancer, and advances in in vitro fertilization have decreased (nearly eliminated) the need for tubal surgery. One needs only to review the case lists of young ob/gyns taking their oral boards to recognize how little gynecologic surgery is now being done by the non-subspecialist.

But what about the patient? For a young woman in good health, it is not unreasonable to assume that the majority of her healthcare during the next few decades will involve her reproductive system. She will likely become pregnant, may develop a disorder of menstruation, and will seek screening for breast and cervical diseases. Thus, many young women see their ob/gyn as the primary, if not sole, provider of healthcare. Yet, those of us who were trained in the '50s, '60s, and '70s were ill-prepared to function as primary care physicians. While a woman may primarily seek our services on an annual basis for breast and pelvic screening, it may be far more important, for example, for her to be certain that her immunizations are up-to-date. Few ob/gyns in the past have performed cardiac auscultation at the time of an "annual pelvic examination," and even fewer have screened for hyperlipidemia. While we might inquire about lifetime sexual partners and family history of breast cancer (both important issues), rarely have we inquired about use of seat belts, presence of hand guns in the home, and other issues of personal security. While we have provided excellent, absolutely outstanding reproductive healthcare, in the past we have done a rather poor job of providing care for the whole patient.

Given all of this background, I personally believe that the American College of Obstetricians and Gynecologists and the Residency Review Committee for Obstetrics and Gynecology showed great foresight in introducing the requirement for training in primary care into OB/GYN residency programs. There is no doubt that, in the future, there will be less pelvic surgery as there have become fewer and fewer indications for hysterectomy. Of course, there will always be genital neoplasia (primarily handled by gynecologic oncologists) and pelvic relaxation (now often handled by urogynecologists), but fewer and fewer general ob/gyns will spend much time in the operating room. More and more of our colleagues have decided to cease any operative activities and devote themselves completely to primary care medicine. Indeed, I do not think that it is at all unreasonable to expect that, at some time in the future, there may be another dramatic change in ob/gyn training with separate tracks for those who wish to pursue solely an ambulatory career, and for those who wish to pursue a surgically oriented career. However, there is virtually no likelihood that the requirement for primary care training in womens health will be dropped from training in the specialty of obstetrics and gynecology. ❖

Suggested Reading

1. American College of Obstetricians and Gynecologists, Washington, DC, 1993.
2. Horton J, et al. *Primary Care Update for OB/GYNs* 1, 1994;5:212-215.
3. Department of Health and Human Services. Centers for Disease Control and Prevention. Advance Data: Office Visits to Obstetricians and Gynecologists: United States, 1989-1990, No 223. Washington, DC, 1993.
4. Benson Gold R, et al. Improving the Fit: Reproductive Health Services in Managed Care Settings. http://www.agi-usa.org.
5. McGee G, et al. *Am J Man Care* 1996;2:71-74.

CME Questions

19. Long-term effects of platinum-based chemotherapy include an increased risk of:

a. sexual dysfunction.
b. arthritis.
c. dental caries.
d. leukemia.
e. alopecia.

20. Which of the following statements is true?

a. Sedentary women with normal menstrual cycle intervals do not ever have luteal phase deficiencies.
b. A woman who runs 10 miles weekly and has regular menstrual cycle intervals does not have luteal phase deficiencies.
c. Luteal phase deficiencies are associated with reduced bone accretion.
d. Anovulation due to excessive exercise always presents as amenorrhea.
e. Energy availability is a likely regulator of reproductive competence.

21. In a recent study from the Netherlands, the VBAC success rate after a prior cesarean delivery for delay of descent in the second stage of labor was:

a. 40%.
b. 50%.
c. 60%.
d. 70%.
e. 80%.

22. Potential benefits of estrogen-induced reductions in cortisol and catecholamine reactivity in response to psychological challenge include all of the following *except*:

a. reduced cardiovascular risk.
b. increased risk of breast cancer.
c. reduced risk of dementia.
d. reduced risk of depression.
e. increased risk of osteoporosis.

23. The main reason for the inclusion of primary care training in residencies in obstetrics and gynecology was to:

a. improve patient care.
b. preserve income.
c. prolong residency training.
d. prevent loss of patients.

24. The following statements are true about treatments to prevent osteoporosis and fractures *except*:

a. Alendronate is increasingly effective with decreasing pre-treatment bone density levels.
b. Estrogen therapy increases bone density to higher levels compared with alendronate.
c. The combination of alendronate and estrogen produces an even greater increase in bone density, but it is not known if this changes the fracture rate.
d. Raloxifene differs from alendronate in not having an effect on the hip fracture rate after 2-3 years of treatment in women with osteoporosis.

In Future Issues:

Risk Factors for Postpartum Incontinence