The YEAR BOOK of

Obstetrics and Gynecology

1982

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Introduction

Special articles in the 1982 YEAR BOOK OF OBSTETRICS AND GYNECOLOGY address two of the most common clinical problems in obstetrics and gynecology.

Postdate pregnancy often presents a dilemma for the obstetrician. Though Clifford is usually credited with the first recognition of the "postmaturity syndrome" in the early 1950s, the Scottish obstetrician Ballantyne, writing in the *Journal of Obstetrics and Gynaecology of the British Empire* a half-century earlier, gave as succinct and accurate description of the condition as has ever been written. When pregnancy extends beyond 42 weeks, the risk of antepartum and intrapartum death rises to a slight, albeit significant, degree. While aware of this risk, the obstetrician is also cognizant of the dangers of ill-timed intervention, inadvertent prematurity and prolonged induction of labor. Given the uncertainties and imponderables of dating gestation in the usual clinical situation, the major problem with postdate pregnancy involves establishing the diagnosis. Dr. Allan B. Weingold, of George Washington University, has prepared an excellent review of the topic in which he outlines his approach to this common and troublesome condition.

Among current controversies in gynecology, none is as prominent as that of menopausal estrogen replacement therapy. On the one hand, there are certain clear benefits—alleviation of vasomotor symptoms, correction of vaginal atrophy, and maintenance of normal bone metabolism. On the other hand, certain risks, chiefly an increased propensity to endometrial cancer, are established quite clearly. Dr. Stanley J. Birnbaum, of Cornell University, has prepared a thorough review, written from the point of view of an experienced and thoughtful clinician, of the risks and benefits of exogenous estrogen treatment. It should provide the basis for an informed decision, based on risk-benefit analysis, in the individual case.

ROY M. PITKIN, M.D.
FRANK J. ZLATNIK, M.D.

Current Literature Quiz

The questions below are an informal test of your knowledge before and/or after reading the YEAR BOOK. The questions are answered by locating the appropriate article in the text by its reference number, which appears in parentheses after each question. The reference numbers indicate the chapter in which the article appears and its numerical order within the chapter.

1. What hormones may be responsible for increased insulin resistance during pregnancy? (1–1)
2. What is the relationship between the maternal concentration of human chorionic gonadotropin and fetal sex? (1–5)
3. What is the biochemical basis for the observed sex difference in risk of respiratory distress syndrome? (1–10)
4. What happens to blood levels of anticonvulsant drugs during pregnancy? (1–19)
5. How might high maternal hemoglobin levels adversely affect the fetus? (1–21)
6. What is the treatment of choice of proliferative retinopathy accompanying diabetic pregnancy? (2–3)
7. Significant deterioration in renal function with pregnancy occurs in what proportion of gravidas with kidney transplants? (2–11)
8. What is the effect of pregnancy on viral hepatitis? (2–15)
9. What is the treatment of herpes gestationis? (2–21)
10. What blood magnesium levels may be anticipated with intravenous administration of 1 and 2 gm/hour? (3–3)
11. What happens to maternal blood levels of complement and C-reactive protein after premature rupture of the membranes? (3–8)
12. What are risk factors for group B streptococcal infections in infants? (3–10)
13. What proportion of infants with nonimmunologic hydrops have congenital malformations? (3–14)
14. Which technique of external cardiotocography gives the best results in terms of readable tracings? (4–1)
15. What treatment is indicated for fetal supraventricular tachycardia? (4–10)
16. What ultrasound measurement is most accurate in determining gestational age in the first trimester? (4–17)
17. What is the effect of blood, meconium, and vaginal secretion on phosphatidylglycerol levels? (4–24 and 4–25)
18. How soon after fetal death is amniotic fluid creatine kinase elevated? (4–27)
19. Do oral β-adrenergic drugs prevent or postpone the recurrence of threatened premature labor? (5–11)
20. What is the most significant maternal complication of β-adrenergic drug treatment of threatened premature labor? (5–13)
21. What is the effect of a distended bladder on uterine activity? (5–23)
22. How often is the volume of the intrapartum fetomaternal bleed larger

than that which one vial of Rh-immune globulin is designed to counteract? (5–26)

23. Is there a difference in prognosis with "thin" versus "thick" meconium staining? (5–28 and 5–29)
24. At what interval prior to delivery is meperidine administration to the laboring gravida most apt to be associated with neonatal depression? (6–1)
25. Is cimetidine effective prophylaxis against Mendelson's syndrome? (6–5 and 6–6)
26. Is the risk of genetic disease increased with increasing paternal age? (7–2 and 7–3)
27. What is the recurrence risk for neural tube defect when a couple has had a previous child who was affected? (7–6)
28. What amniotic fluid test may be helpful in intrauterine diagnosis of cystic fibrosis? (7–11 and 7–12)
29. Is Bendectin teratogenic? (7–17 and 7–18)
30. What are the effects of intrauterine diethylstilbestrol exposure on subsequent pregnancy outcome? (7–19)
31. What is the effect of nursing on maternal levels of oxytocin, prolactin, vasopressin, and prostanoids? (8–2 and 8–4)
32. What is the relationship between maternal diet and milk content of fat and protein? (8–6 and 8–7)
33. What constituent(s) of milk is associated with neonatal jaundice? (8–11)
34. How frequently do *Mycoplasma* organisms cause puerperal fever? (8–15)
35. What are the relative frequencies of *Chlamydia* organisms and gonococci as causes of neonatal ophthalmia? (8–17)
36. What is the relationship between time of onset of fetal growth retardation and later intellectual impairment? (9–3)
37. What are the effects of cesarean delivery on lung volumes and mechanics? (9–6)
38. What proportion of infants with brachial plexus injury at birth ultimately recover completely? (9–11)
39. Should exclusively breast-fed infants receive supplemental iron? (9–17)
40. Is the incidence of ectopic pregnancy increasing, decreasing, or staying about the same? (10–1)
41. What is the failure rate for sterilization by fibriectomy? (10–10)
42. What is the pathogenesis of tuboperitoneal fistulas after tubal sterilization procedures? (10–11)
43. What is the best means of closure of the abdomen to prevent wound disruption? (10–15)
44. Is obtaining a urine culture after the treatment of uncomplicated cystitis in women worthwhile? (11–1)
45. How can detrusor instability be distinguished from true stress incontinence? (11–10 and 11–11)
46. Describe the effects of estrogen therapy on the urethral pressure profile in postmenopausal women. (11–13)
47. What histopathologic finding in vulvar cancer best correlates with prognosis? (12–5)
48. What is the failure rate of cryotherapy of CIN III? (12–7, 12–8)
49. How does cell type (adenomatous, squamous, and adenosquamous) relate to prognosis in carcinoma of the cervix? (12–12)
50. In patients with endometrial cancer, what is the prognostic significance of prior estrogen usage? (12–18 and 12–19)
51. Does estrogen-progesterone receptor status correlate with the response of endometrial cancer to gestagens? (12–23)

52. What is the risk of associated malignancy in benign cystic teratomas after the menopause? (12–28)
53. Is in vitro chemotherapy testing more accurate in ovarian cancer when a favorable or an unfavorable response is predicted? (12–32)
54. What levels of pregnancy-specific placental proteins are found in gestational trophoblastic disease, and what do they mean? (12–39 and 12–40)
55. What causes thyrotoxicosis in choriocarcinoma? (12–42)
56. How often is the vagina involved in recurrent genital herpes infection? (13–1)
57. Why does metronidazole work in "nonspecific" vaginitis? (13–2)
58. What is the single-dose metronidazole regimen for treating vaginal trichomoniasis? (13–4)
59. Prepuberal gonorrhea infrequently is related to sexual assault. True or false? (13–5)
60. Are specimens for culture obtained at culdocentesis useful in managing acute salpingitis? (13–12)
61. How may endorphins be involved in the regulation of the menstrual cycle? (14–1 and 14–2)
62. Is 2-hydroxyestrone administration associated with an increase in serum prolactin levels? (14–4)
63. What is the effect of hyperprolactinemia on bone density? (14–5)
64. What is the relationship between obesity and serum sex steroid-binding globulin levels? (14–20)
65. What factors would suggest that acute adolescent menorrhagia might be a manifestation of an underlying coagulation disorder? (14–22)
66. Does gastrointestinal transit vary with the phase of the menstrual cycle? (14–24)
67. What is the correct dosage of danazol for the treatment of endometriosis? (14–29)
68. If estrogen is contraindicated, what can be used to treat menopausal symptoms? (14–44 and 14–45)
69. How well does laparoscopic recovery of sperm from the pouch of Douglas correlate with the postcoital test? (15–4)
70. How does the basal body temperature vary with the time of awakening? (15–6)
71. What role might the hamster egg serve in the evaluation of human infertility? (15–11)
72. Are there functional sequelae in the male to in utero diethylstilbestrol exposure? (15–12)
73. Why does "escape" ovulation not necessarily mean pregnancy in users of oral contraceptives? (16–3)
74. Why does rifampicin therapy decrease the efficacy of oral contraceptives? (16–7)
75. What is the rate of spontaneous abortion? (17–1)
76. Which pregnancy complications occur with increased frequency after induced abortion? (17–7)

PART ONE

OBSTETRICS

1. Maternal and Fetal Physiology

1-1 **Correlation of Hyperprolactinemia With Altered Plasma Insulin and Glucagon: Similarity to Effects of Late Human Pregnancy.** Pathologic increases in serum prolactin (PRL) levels have been associated with elevated plasma insulin levels. Anthony B. Gustafson, Michael F. Banasiak, Ronald K. Kalkhoff, Thad C. Hagen, and Hak-Joong Kim (Milwaukee) assessed plasma glucose and insulin responses to oral glucose and intravenous tolbutamide tolerance tests in 9 women with hyperprolactinemia and amenorrhea-galactorrhea syndrome (AGS) and in 9 controls matched for age and body weight. Nine women in the third trimester of pregnancy also were studied for their response to oral glucose tolerance tests.

Glucose tolerance curves, baseline insulin levels, and postchallenge plasma insulin responses to oral glucose tolerance tests were markedly higher in AGS subjects than in controls. Plasma glucagon levels were similarly suppressed in both AGS subjects and controls, though the suppression was more sustained in the AGS group. The response to oral glucose tolerance tests in the 9 pregnant women was similar to that for the AGS women. Intravenous tolbutamide produced a similar degree of hypoglycemia in controls and AGS women, though plasma insulin responses in the AGS group were significantly higher than in controls at 5 and 30 minutes.

The results suggest that hyperprolactinemia may contribute to the development of hyperinsulinemia and marked glucagon suppression in response to glucose that is characteristically found in the third trimester of pregnancy.

▶ [Resistance to the actions of insulin characterizes late pregnancy and, in order to maintain glucose tolerance, the gravida increases her insulin levels, both in the basal state and in response to a glycemic stimulus. Substances thought to be responsible for this physiologic hyperinsulinism include human placental lactogen estrogen, progesterone, and cortisol. To that list, prolactin apparently can now be added for, as indicated by this study, there are quite marked similarities in insulin release in response to an oral glucose challenge between women in late pregnancy and those with hyperprolactinemic amenorrhea. Because prolactin levels increase with gestation, perhaps prolactin may be responsible for insulin augmentation, but it is probably, at most, a contributory cause. Moreover, if prolactin calls forth an insulin response, perhaps a "borderline" diabetic might decompensate with development of hyperprolactinemia.] ◀

1-2 **Bromocriptine Treatment During Early Human Pregnancy: Effect on Levels of Prolactin, Sex Steroids, and Placental Lactogen.** During human pregnancy, the level of prolactin exceeds the nonpregnant level after 32–36 days from conception. It is not known whether this prolactin rise is crucial for the normal progress of preg-

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(1–1) J. Clin. Endocrinol. Metab. 51:242–246, August 1980.
(1–2) Acta Endocrinol. (Copenh.) 95:412–415, November 1980.

nancy and the normal production of sex steroids and peptide hormones. O. Ylikorkala, S. Kivinen, and L. Rönnberg (Univ. of Oulu) studied the role of prolactin in the endocrinology of early pregnancy.

Of 50 healthy women admitted for abortion at 6 to 9 weeks' gestation, 28 were given 5.0 to 7.5 mg of bromocriptine daily for 2 weeks between weeks 6 and 9 of gestation; 22 served as controls. Blood samples were collected before and 1 and 2 weeks after the start of the trial for analysis of plasma prolactin, estradiol-17β, progesterone, testosterone, and human placental lactogen levels. All women then underwent uncomplicated abortions.

Bromocriptine treatment induced a prolactin depression at 1 week (7.3 vs. 23.7 ng/ml) and at 2 weeks (5.3 vs. 31.9 ng/ml). Estradiol-17β, progesterone, testosterone, and human placental lactogen levels were not significantly different between the two groups. Two women receiving bromocriptine (7.1%) and 1 control woman (4.5%) experienced spontaneous incomplete abortion during the study period, but these 3 already had a low estradiol-17β level and a low or undetectable placental lactogen level at the beginning of the study.

Neither maternal hypoprolactinemia nor bromocriptine treatment during early human pregnancy interferes with the normal progress of pregnancy or with the normal synthesis of sex steroids and human placental lactogen at this time.

► [These results indicate that bromocryptine administered during normal early gestation lowers prolactin levels but does not affect estradiol, progesterone, or placental lactogen values. Current labeling regulations, at least in the United States, require the statement that bromocriptine is contraindicated in patients wishing to conceive, but these results should provide some reassurance for the patient who does become pregnant while under therapy.] ◄

1–3 **Plasma Prostaglandin F Metabolite Concentrations Following Cervical Encerclage.** Rapid increases in plasma 13,14-dihydro-15-keto-prostaglandin $F_2\alpha$ (PGFM) concentrations after vaginal examination and amniotomy after the 37th week of pregnancy have been documented. P. S. Cocks, I. S. Fraser, R. Markham, M. Robinson, and R. P. Shearman (Univ. of Sydney) measured peripheral plasma concentrations of PGFM in 7 women having elective cervical encerclage in an otherwise normal pregnancy (gestation 12–16 weeks) to determine whether an increase in plasma PGFM levels occurred during this operation performed in the second trimester. To provide comparison, 2 patients having amniotomy at term and 4 patients having suction termination of pregnancy (2 under local and 2 under general anesthesia) also were studied. Ten ml of venous blood was collected prior to anesthesia, after anesthesia but before surgery, and at intervals during the 60 minutes from commencement of surgery. In group I (7 patients), a further specimen was collected at 24 hours. After addition of tritiated PGFM to monitor recovery, plasma samples were extracted, applied to microcolumns to separate PGFM from other prostaglandins, and then assayed with tritiated PGFM and a specific PGFM antiserum.

Only 3 women in group I showed a substantial rise in PGFM within

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(1–3) Prostaglandins 20:493–502, September 1980.

60 minutes, and the others showed no change. All women showed a substantial fall by 24 hours, which was significantly less than both preoperative control value ($t = 2.57$, $P < .05$) and the 60-minute postoperative value ($t = 8.78$, $P < .001$). No other postoperative values were significantly different from either set of control values. Two patients at term had a substantial rise in plasma PGFM within 10 minutes of low amniotomy. There was a similar rise within 15 to 30 minutes of cervical dilatation and termination of first-trimester pregnancy.

Results suggest that most patients do not have a significant rise in PGFM levels following cervical encerclage. Since none of these subjects had any clinical evidence of uterine contractions, and since the safety of prostaglandin-inhibiting drugs in pregnancy has not been established, the authors conclude that routine administration of prostaglandin-inhibiting agents at the time of cervical encerclage is not justified. The extent of the rise in PGFM levels may be related to the degree of cervical manipulation, since the 4 first-trimester patients having elective termination of pregnancy with cervical dilatation to 9 mm showed significant rises in prostaglandin levels. It is not clear why there should be a statistically significant fall in PGFM from the preoperative to the 24-hour postoperative level.

▶ [Although elevated PGFM levels previously have been reported to result from cervical cerclage (1980 YEAR BOOK, pp. 163–164), the present study is not confirmatory. Most patients did not show an increase in metabolite concentration after the procedure.] ◀

1–4 **Decreased Prostacyclin Production: A Characteristic of Chronic Placental Insufficiency Syndromes.** Decreased production of vascular prostacyclin (prostaglandin I_2 or PGI_2) in maternal and placental circulation has been advanced as an etiologic factor underlying the pathophysiologic changes in preeclampsia. However, since fetal PGI_2 synthesis may be related to gestational age, it is questioned whether a deficiency in fetal prostaglandin metabolism is unique to preeclampsia or whether it occurs in other acute or chronic placental insufficiency syndromes.

Marie J. Stuart, Shirazali G. Sunderji, Thelma Yambo, David A. Clark, Judith B. Allen, Haim Elrad, and Jeffrey H. Slott (SUNY, Upstate Med. Center, Syracuse) measured production of 6-keto-$PGF_{1\alpha}$ (stable end product of PGI_2) in the umbilical arteries of neonates born at various gestational ages from 28 weeks to term and compared that with PGI_2 levels in neonates born of pregnancies complicated by abruptio placentae, preeclampsia, intrauterine growth retardation (IUGR), or essential hypertension. The PGI_2 levels were reflected by conversion of ^{14}C-arachidonic acid (AA) to 6-keto-$PGF_{1\alpha}$. Uptake of AA was also measured to determine if this could account for differences in PGI_2 production.

Uptake of ^{14}C-AA was similar in all groups. Thus, the differences in conversion rates to 6-keto-$PGF_{1\alpha}$ were not the result of differences in uptake, nor were they related to gestational age. The amounts of conversion of ^{14}C-AA to 6-keto-$PGF_{1\alpha}$ were similar in neonates born

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(1–4) Lancet 1:1126–1128, May 23, 1981.