patients with ovarian cancer seems to be a problem that needs further investigation.

Ole Mogensen
Bent Mogensen, D.V.M.
Institute of Cancer Research
Danish Cancer Society
Radiumstationen
DK-8000 Aarhus C, Denmark
Anders Jakobsen, M.D.
Department of Oncology
Vejle Hospital
DK-7100 Vejle, Denmark
Arne Sell
Department of Oncology
Aarhus Kommunehospital
DK-8000 Aarhus C, Denmark

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Measurements of delta optical density at 450 nm in SS disease

To the Editors:

I read with interest the recent article by Lindsay and Lupo concerning false elevations of amniotic fluid delta optical density at 450 nm in patients with SS disease and hyperbilirubinemia (AM J OBSTET GYNECOL 1985; 153:75). I first reported this observation in a sickle cell patient in 1978 (AM J OBSTET GYNECOL 1978;130:103). In addition, two additional cases have recently been reported from our laboratory.[1]

I think these collective cases should serve as adequate warning to obstetricians who are managing patients with potential maternal-fetal blood group incompatibilities and who coincidentally have maternal hyperbilirubinemia. The need for intrauterine transfusion and/or early delivery should be very cautiously deliberated.

I would like to make one last comment about the paper of Lindsay and Lupo. In their last paragraph they state that this false elevation of bilirubin level in the amniotic fluid documents indirectly the passage of bilirubin from the maternal to the fetal compartment. I do not think this is necessarily the case. The normal transport of bilirubin is from the fetal to the maternal compartment. An elevation in the fetal compartment as a result of a maternal elevation probably reflects a buildup in the fetal compartment because of inhibition of flow from the fetal to the maternal compartment rather than a flow from mother to fetus. Except in extreme cases of acute maternal hyperbilirubinemia, I doubt that the net flow is ever from the maternal to the fetal compartment.

George H. Nelson, Ph.D., M.D.
Department of Obstetrics and Gynecology
Medical College of Georgia
Augusta, Georgia 30912

REFERENCE

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Reply

To the Editors:

I would like to thank Dr. Nelson for his interest in our report. He presents a plausible mechanism for the elevation of bilirubin in the amniotic fluid, which unfortunately cannot be substantiated by experimental data. Lipsitz et al.[1] presents data which may indirectly support the passage of bilirubin from the maternal to fetal compartment.

In their report they noted that the level of cord blood and maternal serum unconjugated bilirubin was similar. If amniotic fluid elevation was from inhibition of transport, one would expect the level of unconjugated bilirubin in the cord blood to be higher than that in the maternal serum. In the Lipsitz report this was not the case, as the cord blood bilirubin was 9.8 mg/ml and that in maternal serum at the time of delivery was 11 mg/ml. Unfortunately in our report we did not have access to cord blood bilirubin levels. Certainly, further study is warranted.

Michael K. Lindsay, M.D.
Department of Gynecology and Obstetrics
Emory University School of Medicine
Atlanta, Georgia 30322
Virginia R. Lupo, M.D.
Department of Obstetrics and Gynecology
Hennepin County Medical Center
Minneapolis, Minnesota 55415

REFERENCE

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Breast-feeding in thrombocytopenic neonates secondary to maternal autoimmune thrombocytopenic purpura

To the Editors:

We would like to comment on the statement of Martin et al. (Martin JN, Morrison JC, Files YC. Autoimmune thrombocytopenic purpura: Current concepts and recommended practices. AM J OSTET GYNECOL

1984;150:86.) discouraging breast-feeding in thrombocytopenic infants born to women with autoimmune thrombocytopenic purpura.

A recent patient of ours with autoimmune thrombocytopenic purpura and previous splenectomy was referred to our center for management of the disease during her third pregnancy. She had a first child by vaginal delivery with neonatal thrombocytopenia and cerebral hemorrhage with persistent neurologic deficit, and a second child died after vaginal delivery with intracranial hemorrhage. She received steroids in the last 3 months of her pregnancy because of severe thrombocytopenia (<10,000/mm^3) and started labor at 34 weeks. The premature baby was delivered by cesarean section and showed disseminated bruises. The baby's platelet count was <10,000/mm.^{3} Platelet transfusion was given and exsanguination transfusion was performed, corticosteroid therapy was started, 1 mg/kg/day, and platelet transfusions were continued for 2 weeks. The clinical status deteriorated because of sepsis and respiratory distress. Antibiotics and oxygen were administered, and the clinical situation improved. The baby was first fed by nasogastric tube with the mother's milk when he was 5 days old; when he was able to suck, breast-feeding was started without deterioration of his platelet count. Corticosteroids were stopped after 2 weeks and breast-feeding was continued; the platelet count remained at 145,000/mm^3 and no more platelet transfusions were required.

Although it is only one case, we have evidence that in this severely thrombocytopenic baby, breast-feeding did not accentuate the thrombocytopenia. In a letter of Kelemen et al.[1] there is only a suggestion that platelet counts in the babies reported by Jones et al.[2] were lowest between the second day and fourth day as a result of breast-feeding, but there is no proof that this was the cause. In fact, no absorption of breast milk antibodies could be demonstrated in humans,[3] so we think that more cases are needed to establish whether breast-feeding is safe or not in these cases.

Susana Meschengieser, M.D.
María Angela Lazzari, M.D.
Department of Hemostasis and Thrombosis
Instituto de Investigaciones Hematológicas
"Mariano R. Castex"
Academia Nacional de Medicina
Pacheco de Melo 3081
(1425) Buenos Aires, Argentina

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3. Hanson LA. The mammary glands as an immunological organ. Immunol Today 1982;3:168.

Reply

To the Editors

We would like to thank Drs. Meschengieser and Lazzari for communicating their recent experience with a parturient who had autoimmune thrombocytopenic purpura after splenectomy and who, after cesarean birth of her thrombocytopenic preterm infant, contributed breast milk and later breast-fed without apparent adverse effect on the neonatal platelet count.

Their case report serves to reinforce the likelihood of severe fetal thrombocytopenia in the splenectomized parturient with autoimmune thrombocytopenic purpura. Their finding that breast-feeding beginning on the fifth day did not worsen the neonatal thrombocytopenia is valuable information. We agree that further study and discussion is needed to clarify this issue and suspect that the benefits of breast-feeding in the preterm infant especially may outweigh theoretical or actual risk.

James N. Martin, Jr., M.D.
Division of Maternal-Fetal Medicine
Department of Obstetrics and Gynecology
The University of Mississippi Medical Center
2500 North State Street
Jackson, Mississippi 39216

Mobiluncus-specific antibodies in a postoperative infection

To the Editors:

Mobiluncus, a bacterium closely related to bacterial vaginosis, has during the last year been reported by two different authors to give deep infections.[1, 2] Serologic studies have, however, not yet been able to demonstrate antibodies against *Mobiluncus* in patients with bacterial vaginosis.[3] In this letter we present the result of the case of an abdominally hysterectomized woman with postoperative infection of *Mobiluncus,* in which we have used an indirect immunofluorescence assay to measure *Mobiluncus*-specific antibodies in serum.

Whole untreated bacterial cells were used as antigen, and screening for the isolate with highest antigenic activity was performed by enzyme-linked immunosorbent assay. The chosen isolate, *KB,* gave a reaction two twofold dilutions higher than the type strain of *Mobiluncus curtisii* and five twofold dilutions higher than the isolate with the poorest reaction. In indirect immunofluorescence assay approximately 10^5 bacteria per milliliter from 3-day-old cultures suspended in 0.01mol/L of phosphate-buffered saline solution were placed on microscopic slides, dried in air, and used without fixation. To each well on the microscopic slide 20 μl of serum diluted 10-fold and further diluted twofold (10, 20, 40, etc.) was applied. After incubation in a moist chamber for 20 minutes at 37° C, the slides were washed in phosphate-buffered saline solution and the slides were stained with fluorescein isothiocyanate–labeled sheep antihuman immunoglobulin (National Bacteriological