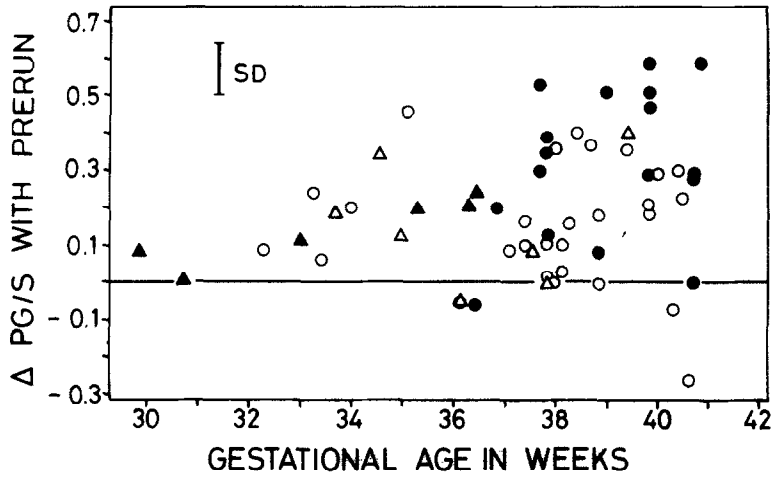

Fig. 1. Amount of pseudo-phosphatidylglycerol found in amniotic fluid, expressed as a ratio to sphingomyelin (*PG/S* ratio). The values are coded as to outcome as discussed in Reference 3, with *closed triangles* representing cases of respiratory distress syndrome; *open triangles*, mild transient difficulty; and *closed circles*, absence of any respiratory difficulty (delivery within 48 hours).

In addition, we have recognized and recently reported the presence of a substance found in most amniotic fluids which imitates phosphatidylglycerol with this system.[3] The substance ("pseudo phosphatidylglycerol"), of which Dr. Barnes and associates may have been unaware, is present in many cases in amounts greater than the genuine phosphatidylglycerol. In a sample of 75 fluids that had appeared phosphatidylglycerol-positive with the original procedure, we found 20 to be phosphatidylglycerol-negative after the removal of the contaminating substance.

The accompanying Fig. 1 shows the amount of pseudo phosphatidylglycerol we found, expressed as a ratio to sphingomyelin. Notably, the cases of respiratory distress syndrome in this population had pseudo phosphatidylglycerol as great as a ratio of 0.2, but of these cases, four were shown to actually be phosphatidylglycerol-negative after removal of the pseudo phosphatidylglycerol.

We believe that the confusion of pseudo-phosphatidylglycerol for phosphatidylglycerol with this system may be an important factor in the results that Barnes et al. report. The authors may wish to reanalyze the specimens, using the modification we have developed to eliminate the interference of "pseudo phosphatidylglycerol" in the Helena method.

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Reply to Cotton and Spillman

To the Editors:

The concept of "pseudo-phosphatidylglycerol" mentioned by Drs. Cotton and Spillman is intriguing (see Reference 3 in their letter). It remains an unidentified substance, a co-migrator with phosphatidylglycerol found resistant to digestion by *Bacillus cereus* phospholipase C. It is suggested that a prechromatography step of developing the sample on the plates for 70 minutes, equilibrating with the solvent for 10 minutes, and air-drying for 10 minutes removes pseudo-phosphatidylglycerol.

Cotton and Spillman describe a sample of 75 amniotic fluids reported initially as phosphatidylglycerol positive; however, 20 fluids (27%) were subsequently found to be, in fact, PG negative with use of the prechromatography step. We find this figure high and would expect a greater incidence of the respiratory distress syndrome with positive phosphatidylglycerol by use of our current one-dimensional thin-layer chromatography method with the Helena Tek 200 system with that percentage of error from contamination. Our experience has been that respiratory distress syndrome is rare in cases reported as phosphatidylglycerol positive. Whit-

tle et al.,[1] in a series of patients from whom amniotic fluid had been collected within 72 hours of delivery, reported only three of 532 (0.6%) babies developed respiratory distress syndrome when phosphatidylglycerol was present.

It is of interest that it is documented[2] that the diagnostic value of phosphatidylglycerol may be valid only when the phospholipids are measured by two-dimensional thin-layer chromatography. However, Semmer et al.[3] report an analysis with the use of one-dimensional thin-layer chromatography without the prechromatography step of Cotton and Spillman. In 159 transabdominal amniotic fluid samples the phosphatidylglycerol indicated a 98% prediction rate for absence of respiratory distress syndrome with a 1.8% false positive result corrected to 0%; they concluded that the presence of >3% phosphatidylglycerol by means of Painter's[4] method of readily available one-dimensional thin-layer chromatography is consistent with fetal lung maturity. The advantages of the unidirectional method are a shorter procedure time, the ability to analyze multiple samples on a single plate, and the simplicity of a single-solvent system. Whether greater accuracy for phosphatidylglycerol interpretation is dependent on standard quantitative evaluation of phosphatidylglycerol or the removal of pseudo phosphatidylglycerol from amniotic samples may still require investigation.

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Are CA125 and placental alkaline phosphatase the same antigen?

To the Editors:

We have read with interest the paper entitled "Elevation of serum CA125 in carcinomas of the fallopian tube, endometrium, and endocervix" (AM J OBSTET GYNECOL 1984;148:1057), and it would appear that the monoclonal antibody OC125 has many properties similar to monoclonal antibodies directed against placental alkaline phosphatase[1] (also unpublished observations). Both CA125 and placental alkaline phosphatase are membrane-bound glycoproteins and the 'M' form of placental alkaline phosphatase has a similar molecular weight as CA125.[2] Their distribution in various tumors is similar,[3-5] although as far as we are aware placental alkaline phosphatase has not been reported in association with fallopian tube carcinoma. CA125 was initially reported as being absent from all normal human tissues, including fallopian tube, cervix, and endometrium,[6] but a later study found it to be present in these tissues[7] with a distribution identical to placental alkaline phosphatase.[8] Placental alkaline phosphatase has also been demonstrated in normal lung, testis, and thymus.[9] However, no comment was made in the later study[7] about the expression of CA125 by these organs. Both placental alkaline phosphatase and CA125 are found in association with inflammation,[1,7] and a coelomic epithelial origin is suggested for both.

Using the monoclonal antibody $NDOG_2$, which recognizes the three common allelic forms of placental alkaline phosphatase, we have found placental alkaline phosphatase in association with 60% of endometrial carcinoma, 60% of ovarian carcinoma, and 30% of cervical carcinoma—a roughly similar distribution to CA125. Have the authors investigated the possibility that their monoclonal antibody binds a placenta-like alkaline phosphatase activity and may therefore be equated with one or many of the forms of placental alkaline phosphatase present in normal and tumor tissue?

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