period) given a live birth, and the conditional probability of dying in the remainder of the first year (the postneonatal period) given survival to 28 days of age. As such, our measure takes into account a portion of the distinctly nonlinear mortality function (with respect to age) which is so characteristic of infancy. Of course, if more detailed information is available, it becomes possible to specify the nonlinearity more closely. However, when the rates in infancy are very low, the increased precision is not worth the additional effort (as we pointed out in the previous communication). For example, I have recomputed the infant mortality rate for the United States in 1973 (a), using the neonatal and the postneonatal periods (b), and using 22 subdivisions of infancy (c), as given in our vital statistics.[1]

Infant mortality rate:		
(a) *per 100,000 births*	*(b)* *per 100,000 at risk*	*(c)* *per 100,000 at risk*
1,771.8	1,778.1	1,786.5

Another point worth mentioning is that in our previous communication we did not advocate the use of our measure. In fact, there was no discussion of it at all. The procedure was carried out to illustrate the difference in the overall risk of death in infancy when more precise information is used in the denominators. Clearly, it is useful and desirable to present the infant mortality rate per 1,000 births for comparative purposes. However, this does not preclude the use of other measures of risk which incorporate more "precise" information. The main point of our previous communication remains unchanged: it is useful to use the maximum information available to specify, more closely, the population at risk.

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REFERENCE

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Removal of ovaries with vaginal hysterectomy

To the Editors:

I would like to comment on a paper by Smale and co-workers.[1] The adnexa are removed by a technique similar to the one used at abdominal hysterectomy, but without peritonealizing the pedicle of the infundibulopelvic ligament. I wish to commend the authors on an excellent report which again documents the feasibility and safety of expanding the vaginal approach in the treatment of gynecologic conditions.

This paper is, however, presented as the first reported series in which the adnexa are removed at the time of vaginal hysterectomy. I would like to call attention to a paper[2] published 4 years ago which was apparently overlooked in their review of the literature. An alternative technique is described in this paper which would be of interest to the gynecologist. This technique is useful in vaginal hysterectomies associated with decensus, but is particularly applicable in patients where the ovaries are high and exposure difficult.

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Reply to Dr. Wright

To the Editors:

We are truly sorry our "off-line" search missed vaginal oophorectomy. We respectfully give credit where it is due!

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First description of vaginal agenesis

To the Editors:

The first description of vaginal agenesis is attributed to Realdus Columbus,[1, 3–7] although this anomaly was already known,[5, 8] but authors do not agree upon the date. Whereas Gray and Skandalakis[4] affirm that the first description was in 1559, other authors mention 1572[1, 3, 5, 6] and 1593.[7]

Columbus described a case of a woman with normal vulva but without a vagina and with a rudimentary uterus who suffered terrible pain upon coitus. The description can be found in the last book (Rare findings in anatomy) of Columbus' treatise[2] first published posthumously in 1559, a few months after author's death, by the Bevilacqua in Venice. There were further editions: in Paris by Wechelum and by Gillium and in Frankfurt by Lechlenum (1593).

We must therefore consider 1559 as the year of the first description of vaginal agenesis in medical literature.

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The syndromes of hydatidiform mole

To the Editors:

I must congratulate Drs. Szulman and Surti (AM. J. OBSTET. GYNECOL. 131:665, 1978) for their timely effort to dispel some of the confusion that surrounds this "awkward" entity of hydatidiform mole. The confusion has resulted largely from a lack of universally recognized diagnostic criteria and from an incomplete grasp of its nosology. The advent of cytogenetics and the recent recognition of partial moles have expanded our horizons but so far have failed to clear the air.

I agree with the authors that trophoblastic hyperplasia typifies the neoplastic mole and that early abortions, showing villous hydrops without trophoblastic overgrowth, should not be classified as moles. I am not clear about their views regarding "conceptuses with demonstrable embryo/fetus, variable pronounced hydatidiform changes, and focal mild to moderate trophoblastic hyperactivity." If depression of growth activity is fundamental in nonmolar heteroploid gestation, as shown biologically and pathologically,[1] the presence of "trophoblastic hyperactivity" is incompatible with such a diagnosis. I would like to suggest that all cystic placentas with trophoblastic hyperplasia, regardless of karyotype, should be classified as hydatidiform moles, i.e., neoplastic, especially as heteroploid moles are on record, as acknowledged by the authors. If trophoblastic hyperplasia, as stressed previously by Park,[2] is accepted as the crucial morphologic discriminant, pending validation or rejection in the future, it will allow consistency in diagnosis and comparability of reported series.

I was particularly interested in their unusual case of mole associated with 46,XX karyotype. They mention the karyotyped tissues as fetal but they make no mention of the sex of the fetus. I wonder if the possibility of maternal contamination was ruled out in this case.

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