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Letters to the Editor

Can Crib Death Be Prevented?

Sir:

There is a growing and substantial body of investigative evidence that most infants who die suddenly, unexpectedly, and inexplicably are not, as previously thought, entirely normal. When examined at autopsy meticulously and methodically and in large numbers, together with appropriate controls, they reveal subtle morphologic abnormalities, including increased muscle mass in the walls of small arteries in the lungs [*1,2*], undue retention of periadrenal brown fat [*3,4*], inappropriate hepatic extramedullary hematopoiesis [*3,5*], and gliosis involving the brain stem, pons, and medulla [*6,7*]. All of the above references have suggested to some extent that affected babies die after protracted or oft-repeated episodes of hypoxia and hypoxemia. Clinical studies of infants who have eventually died of the syndrome or who are considered to be at risk for crib death, such as subsequent siblings of victims or the so-called near-miss infants, have revealed that they are functionally different from normal, particularly with regard to vital respiratory and cardiac function [*8-11*]. Less extensively documented but also recorded are evidences of their inability to cope with external threats to their airway [*12*], difficulty with swallowing [*8*], abnormal cries, and failure to grow as well as matched controls [*9,13*].

Nowadays most interested scientists believe that the sudden infant death syndrome is not one entity but instead represents a group of obscure deaths that probably result from a number of causes or mechanisms. For example, Dr. Arnon [*14*] has recently demonstrated that a certain small number, perhaps as many as 5%, may be due to infant botulism, heretofore unrecognized. Yet there is a sizable group, perhaps a majority, who at necropsy show morphologic evidences of their having been physiologically compromised, possibly for all of their lives. It is this large group that is most interesting to clinicians and the public at large since it makes very real the possibility that such an infant might be identifiable in the first days or weeks of life and his sudden death as an infant prevented. Toward that end the National Institute of Child Health and Human Development has recently funded one large and admirably organized prospective project, now underway, in which a thousand newborns a year will be examined extensively from the first day of life onward; the intent is to identify and refine procedures to recognize vulnerable babies. In other centers, infants already diagnosed as being at special risk because of repetitive attacks of protracted apnea are being maintained, even at home, with monitors; there is controversy as to whether or not these programs are actually saving lives that would otherwise have been lost, but it seems that they may be.

Dr. Raven's recent article [*15*], like those she has written in the past [*16,17*], demonstrates once more her long-standing conviction that most crib deaths are the result of interstitial pneumonia of viral etiology in infants presumed to have been well prior to their precipitous terminal illness. Most pediatric pathologists have, through the years, been reluctant to accept her interpretation of the morphologic features of these infants' lungs, illustrated in the photomicrographs (Figs. 4*d* and 4*f*) in her most recent article [*15*]. It is true that the alveolar walls seen in these instances may seem to be thicker and more cellular than normal—as judged by adult standards—but that appearance is commonplace in the lungs of infants in the early months of life. In addition, several excellent investigations, directed at the recovery of viruses from the lungs of such infants, have resulted in the isolation of relatively few agents [*18-20*] and those are a mixed lot, with no one viral agent predominating. Hence there is neither strong morphologic nor microbiologic support for Dr. Raven's contention.

The presence of antibody-producing cells in the lungs of these infants is not surprising.

Antibody-producing cells are normally scattered throughout the body and are not simply called into being to participate in specific local inflammatory reactions. They have been demonstrated in these infants' lungs before [*21*], and even in the lungs of normal infants [*21*]. In fact, it seems unusual that they were not observed in the sections of lung from Dr. Raven's two control infants. It is surprising furthermore that no cells were encountered producing IgA or IgM; one wonders about the efficacy of the anti-human IgA and IgM immunoglobulin antiserum employed.

In summary, I find myself agreeing with Dr. Mueller [*22*], who, in his recent letter to the editors of this journal, took issue with Dr. Raven. In addition, I should like to thank him for his gift of what is, to me, a new word—procrustean. It is one I had never encountered before but which I shall cherish, not that one would need to use it very often but there seems to be no substitute for it in this situation.

Marie Valdes-Dapena, M.D.
Department of Pathology D-33
University of Miami School of Medicine
P.O. Box 016960
Miami, Fla. 33101

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Discussion of "Spermatozoa in the Anal Canal and Rectum and in the Oral Cavity of Female Rape Victims"

Sir:

I read with interest the technical note by Enos and Beyer in a recent issue (Vol. 23, No. 1, Jan. 1978, pp. 231-233). Their conclusions about the significance of semen on anal/rectal smears from rape victims are very similar to those reached in this laboratory from analysis of some of the information available from past case work.

Seventeen cases that fulfilled the following conditions were found during a survey [1]:

(1) the victim was raped,
(2) there was no allegation of buggery,
(3) the doctor took an anal or rectal swab, and
(4) there was semen on the vaginal swab.

In nine of these cases semen was found on either the anal or rectal swab. The amount found was small in comparison to that on the vaginal swab. However, unlike the observations of Enos and Beyer, our records indicate that a small quantity of semen is the normal finding when anal or rectal swabs from victims of buggery are examined. At present we do not usually know if the victim has defecated before examination although this is part of the information we try to elicit from police surgeons.

In the same issue of your journal there was a letter from White et al [2] on the subject of vaginal phosphoglucomutase (PGM). Some of our results are also pertinent to this letter in that they too demonstrate both the existence of vaginal PGM and the disproportionate weighting towards the female PGM type of grouping results from mixtures of semen and vaginal debris in vaginal samples.

While studying grouping results from past cases, I have found 26 cases where successful PGM typing has been obtained from mixtures of semen and vaginal debris on a vaginal swab and at least one other item in each case (usually clothing or bedding, but on one occasion a floor swab). In only 2 of the 26 cases did the vaginal swab give reactions for a PGM group different from that of the female concerned, whereas 13 of the cases contained other items where the grouping result was partially or completely due to seminal PGM activity (Table 1).

Frequencies of the more common PGM_1 phenotypes in the British population [3] indicate

TABLE 1—*Results from 26 cases in which PGM typing of a vaginal swab and one other item was successful.*

Victim's PGM Phenotype	PGM Result from Vaginal Swab	PGM Result from Other Item	Suspect's PGM Phenotype (If Known)
1	1	1 (pants)	1
1	1	1 (pants)	1
1	1	1 (pants)	1
1	1	1 (pants)	1 and 2-1
1	1	1 (pants)	unknown
1	1	1 (pants)	unknown
1	1	1 (pants)	unknown
1	1	1 (coat)	1
1	1	1 (tights)	unknown
1	1	2-1 (pants)	1 (excluded)
1	1	2-1 (pants)	2-1
1	1	2-1 (pants)	2-1
1	1	2-1 (pants)	unknown
1	1	2-1 (pants)	unknown
1	1	2-1 (sheet)	2-1
1	2-1	2-1 (sheet)	2
2	2	2-1 (sheet)	1
2	2	2-1 (towel)	1
2	2	2-1 (pants)	unknown
2	2	2-1 (tights)	unknown
2-1	2-1	2-1 (pants)	1
2-1	2-1	2-1 (pants)	1
2-1	2-1	2-1 (pants)	1
2-1	2-1	2-1 (pants)	unknown
2-1	2-1	1 (pants)	1
2-1	1	1 (floor swab)	1

that in 47% of PGM typings of semen/vaginal debris mixtures it will not be possible to determine the origin of the PGM activity because both parties will be of the same phenotype. In 29% of cases at least an indication of the seminal PGM type should be obtained. In the remainder of cases (24%) there will be the possibility of the female heterozygous type (PGM 2-1) masking the seminal homozygous types. However, such masking does not always occur (see the last example in Table 1). Either constituent of a semen/vaginal debris mixture may not contribute to the final result. When vaginal swabs are typed the seminal PGM is least likely to react, possibly because of its destruction by some factor in the vaginal environment as well as its dilution by vaginal secretions. Mixtures on other items are much more likely to give reactions enabling partial or complete typing of the seminal PGM.

Mrs. Anne Davies
The Metropolitan Police Forensic Science Laboratory
109 Lambeth Rd.
London SE1 7LP, England

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