Without broadening the issues, how many of the current modality measurements used in ORs and ICUs have been subjected to vigorous clinical trials which establish efficacy and safety? Scientific validation is not synonymous with widespread noncritical acceptance of various forms of management.

My "bias" is not directed at home apneic monitoring. It is directed at the mass and noncritical use of a host of management modalities. Home apneic monitoring is by no means the only offender.

The data base that is quoted was the one that is available. If you have a better data base, it should be published by all means. I offer my column as a vehicle.

The summary of Harsell's studies is in the public domain. It can be obtained from the NICHD. What you may mean is that the study has not been peer-reviewed and published in a journal. This raises an interesting, if tangential, issue. Several years ago, the University of Utah completed a small but important cost/benefit analysis of home apneic monitoring. The study concluded that the cost/benefit and risk/benefit ratios were unfavorable. It was universally rejected for publication. My impression is that the major reason for rejection was that the experts in the field could not accept the idea that their endeavors were not patient-effective.

In addition to Harsell's study there are two well-documented cases of infanticide or attempted infanticide as a result of home apneic monitoring. In addition, I have been contacted by a number of mothers, each with her story of severe psychosocial harm from monitoring, anecdotal to be sure, but nevertheless persuasive (to me.)

The only other report I could find in the literature which looks at psychosocial risk was from a group who found only "appropriate" emotional responses by mothers whose infants were being monitored. The study was conducted using a ghetto population in a large Eastern city. I contacted one of the authors, who informed me that ghetto mothers did not seem to be as concerned with the welfare of their children as more affluent mothers. I found this conclusion to be unacceptable as a scientific evaluation and to display a degree of racism which raised important issues about the objectivity of the study, and I have consequently decided the data are not acceptable.

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A Case History

To the Editor:

It is with great interest that I read the article, "Pulmonary Eosinophilia and Coccidioidal Infections" (Chest 1987; 91:734). Patient 2 was originally admitted to our hospital and we transferred her to Stanford. The course of her hospitalization was as follows.

The patient was in excellent health until the onset of pleuritic chest pain on September 16, 1985, while on vacation. The next day, pulmonary rumbles were noted, but not fever or pain. On September 20, she presented with fever and dry cough. Four days later, she was admitted to the hospital with a white blood cell count of 19,000, including 73 segs, 10 percent eosinophils and diffuse bilateral interstitial pneumonia on chest x-ray film. Coccidiomycosis topped the differential diagnosis, and skin tests and serologies were performed. The cold agglutinins were positive at 1:32. Two days later, skin test results came back negative, and an open lung biopsy frozen section showed no inflammation. The Health Department reports were negative for fungus on direct stains of the lung biopsy specimen. The next day, permanent sections revealed eosinophilic pneumonitis, with coccidiomycosis of primary concern. IgE level was drawn. Special stains of lung biopsy specimens were negative for organisms. Therapy with SoluMedrol was started, and lung slides were sent to Dr. Luise Katzenstein for pulmonary pathology consultation. On September 30, the IgE level was 12 X normal. Reexamination of all sputum samples for coccidiomycosis spurels was negative. Therapy was begun for oral Candidiasis. On October 4, the patient was still intermittently febrile, but the lungs and sputum had cleared. Herpes sore appeared on the lip. Another hypotensive flushing reaction with IV nafillin, 19 percent eosinophils and PCN allergy. Dr. Pappagianis reported that the coccidiomycosis serologies were negative by the immunodiffusion and comp fix methods. Dr. Katzenstein's consultation was received on October 7, advising that it was not typical of eosinophilic pneumonia, and she suspected a hypersensitivity reaction to some inhaled substance, with a diagnosis of eosinophilic infiltrate with necrosis. The next day, serologies were sent to Dr. Fink, Milwaukee, for inhaled allergic precipitants. The eosinophilia had cleared, but respiratory symptoms still required therapy with prednisone, 60 mg daily, to control. The patient was discharged on October 11 on therapy with ketoconazole and prednisone. A week later, fungal culture of the open lung biopsy specimen reported Aspergillus fumigatus. The patient became symptomatic again by November 5, with severe reticulonodular infiltrates bilaterally, anemia with 6.8 WBC count, toxic granulations, a left shift and no eosinophils in peripheral blood. Repeat fungal cultures were obtained, and serologies sent to Dr. Pappagianis. Therapy with prednisone, 3 mg tid, and Imuran was instituted. Two days later, sputum was negative for eosinophils and the patient's condition had subjectively improved. On November 11, we consulted with Dr. Raffin of Stanford and Dr. Fauci of the NIH. They suggested an immunologic phenomenon, similar to Wegener's, and recommended switching from Imuran to Cytoxan. The patient deteriorated and was transferred to Stanford on November 14. Three days later, the Public Health Service reported Candida albicans from the November specimen. Dr. Pappagianis reported, on November 22, that serologies from samples we submitted as well as those submitted from Stanford were positive for Coccidioides. On December 16, the Public Health Service further reported that the final fungal culture we had submitted was Candida albicans, and mold was also present although they were unable to isolate it from the yeast.

In the article, the authors state that "serologic studies and skin tests play an important role in diagnosis of Coccidiomycosis as well as other infections. In the second case appropriate serologic studies and skin testing could have led to the diagnosis of a coccidioidal infection." I submit that appropriate skin and serologic tests were performed expeditiously. Although there is a three to four week delay in receiving final fungal culture results, our initial open lung biopsy and sputum cultures were negative, so hastening the results would not have helped.

Since IgE is not the first antibody to be formed in response to an antigen challenge (IgM is first), why was the IgE level elevated but IgM and IgG Coccidiomycosis serologies negative at initial admission? Perhaps this patient was first exposed to Coccidiomycosis in the four weeks between discharge and readmission, while on prednisone therapy. It is equally likely that Aspergillus precipitated the initial eosinophilic microabscessing condition, as is much more commonly reported. Faced with all the negative study results and worsening status of the patient, plus her response to steroid therapy, that treatment was logical and, in fact, necessary in this patient.

Is it acceptable editorial practice to allow publication of an article in which the authors did not consult the primary care physicians before publishing to obtain all facts?

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Communications to the Editor