

Acute Pulmonary Hemorrhage in Infants Associated With Exposure to *Stachybotrys atra* and Other Fungi

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Background: A geographic cluster of 10 cases of pulmonary hemorrhage and hemosiderosis in infants occurred in Cleveland, Ohio, between January 1993 and December 1994.

Study Design: This community-based case-control study tested the hypothesis that the 10 infants with pulmonary hemorrhage and hemosiderosis were more likely to live in homes where *Stachybotrys atra* was present than were 30 age- and ZIP code-matched control infants. We investigated the infants' home environments using bio-aerosol sampling methods, with specific attention to *S atra*. Air and surface samples were collected from the room where the infant was reported to have spent the most time.

Results: Mean colony counts for all fungi averaged 29 227 colony-forming units (CFU)/m³ in homes of patients and

707 CFU/m³ in homes of controls. The mean concentration of *S atra* in the air was 43 CFU/m³ in homes of patients and 4 CFU/m³ in homes of controls. Viable *S atra* was detected in filter cassette samples of the air in the homes of 5 of 9 patients and 4 of 27 controls. The matched odds ratio for a change of 10 units in the mean concentration of *S atra* in the air was 9.83 (95% confidence interval, 1.08-3 × 10⁶). The mean concentration of *S atra* on surfaces was 20 × 10⁶ CFU/g and 0.007 × 10⁶ CFU/g in homes of patients and controls, respectively.

Conclusion: Infants with pulmonary hemorrhage and hemosiderosis were more likely than controls to live in homes with toxigenic *S atra* and other fungi in the indoor air.

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Editor's Note: The epidemiology story is great reading. Rather than "Eleven Blue Men," we have 10 blue infants.

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PULMONARY hemosiderosis is a rare condition characterized by spontaneous pulmonary hemorrhage, often associated with iron-deficiency anemia.¹ The cause is most often unknown, although previous reports have linked a portion of childhood cases with cardiac or vascular malformations, infectious processes, immune vasculitis, trauma, or known milk protein allergies.²

During January 1993 to December 1994, 10 infants with acute pulmonary hemorrhage and hemosiderosis were seen at Rainbow Babies and Childrens Hospital, Cleveland, Ohio.³ The diagnosis was made by demonstrating alveolar hemosiderin-laden macrophages in biopsy specimens or in bronchoalveolar lavage 3 to 6 weeks after the initial hemorrhage.

All of the infants were black, and all but 1 of the infants were male. During the previous 10 years, 3 cases of idiopathic pulmonary hemosiderosis had been diagnosed among infants and children at this hospital.

The purpose of this investigation was to determine the cause of the high incidence of acute pulmonary hemorrhage and hemosiderosis among infants in Cleveland during these 2 years. The affected infants all lived in an area of eastern metropolitan Cleveland within 6 miles of the hospital.

Among the most striking features of this illness were its severity and its tendency to recur after hospital discharge. In 5 infants, acute hemoptysis necessitating readmission to the intensive care unit recurred within 1 day to 6 months of discharge. All infants' hemorrhages were so severe that they required admission to the pediatric intensive care unit. All but one underwent intubation. One infant died. In a previous report from this investigation, infants with pulmonary hemorrhage and hemosiderosis were found to be 16 times more likely than control in-

MATERIALS AND METHODS

All infants younger than 1 year who had been admitted to the hospital with idiopathic pulmonary hemorrhage and hemosiderosis between January 1993 and December 1994 were included. For each patient, we generated a list of potential controls from all infants born in Cleveland within 2 weeks of the patient and presently living in an area bearing 1 of the 6 ZIP codes in which all of the patients lived. The list of potential controls was generated from Cleveland birth certificates and records of the hospital continuity clinic. Infants' parents were telephoned to invite their participation in the study. For each patient, the first 3 potential controls whose parents agreed to participate were enrolled. Informed consent was obtained from the parents or guardians of all infants.

A pediatrician (R.A.E., E.M., or D.G.D.) visited the homes of all patients and controls to administer a questionnaire comprising more than 200 items that included questions about the infant's health, infant care practices, and home environment characteristics. The questionnaire included specific questions about the infant's exposure to toxic agents (pesticides, paints, solvents, and gasoline) and structural characteristics such as water damage. A registered sanitarian performed an environmental survey of each home, with special attention to the infants' sleeping and living areas. The pediatrician and the sanitarian were not blinded to the case or control status of the infant.

From December 11 to December 19, 1994, at a separate visit, industrial hygienists unaware of case or control status of the homes performed environmental sampling to look specifically for the presence of *S atra* in the air and on surfaces. Residents were not at home during the sampling.

ENVIRONMENTAL SAMPLING METHODS

Bioaerosol sampling was performed at the homes of patients and controls to determine the presence of *S atra* and other fungi. All air and surface samples collected from each site for viable microorganisms were refrigerated at approximately 4°C before analysis.

The air samples were collected during 1 to 2 hours from the room where the infant was reported to have spent the most time. Various activities, including vacuuming carpets, pounding on furnace ducts several times, and walking on carpets, were performed at each residence in an effort to simulate household activities that could release dusts from ventilation systems and household surfaces. Air samples were collected to test for *S atra* spores and viable fungi.

AIRBORNE CONIDIA

Airborne conidia (spores) were collected using total dust sampling on cellulose ester membrane filters. Samples were collected using a Gilian pump (Gilian Instrument Corpo-

ration, Wayne, NJ) at a flow rate of 1.0 L/min for 6 to 8 hours. After sampling, each filter was positioned on a glass slide, and the entire area of each filter section was scanned using brightfield microscopy (approximate magnification, $\times 200$) to identify the presence of *S atra* spores. A standard reference slide of *S atra* spores was prepared in the same manner to aid in the identification of spores.

VIABLE AIRBORNE FUNGI

Continuous samples for viable fungi were collected using the CAMNEA filter method.⁷ Fungal propagules were collected on polycarbonate filters using a Gilian pump at a flow rate of 2.0 L/min for approximately 1 to 2 hours. By culturing serial dilutions of the filter washings, fungal spores from these samples were enumerated.⁸ Diluted filter fluids were plated on the following media: rose bengal streptomycin agar,⁹ cellulose agar (Czapek-Dox agar with sucrose and FeSO₄ omitted, containing 20 g/m powdered cellulose and 50 mg/L rose bengal and adjusted to pH of 8.0), 2% malt extract agar, and dichloran glycerol agar.¹⁰ The plates were then incubated at 24°C for 10 days. Colonies were classified into the following categories: *Aspergillus*, *Cladosporium*, *Penicillium*, *Stachybotrys*, and other. The other category included all other fungi observed. Concentrations are reported as colony-forming units per cubic meter of air sampled (CFU/m³).

SURFACE SAMPLES

Samples were collected from areas of suspected mold growth in homes of patients and controls by scraping surface materials into sterile centrifuge tubes or plastic bags. Serial 10-fold dilutions were prepared after adding 0.5-g portions of the sample to 49.5 mL of phosphate-buffered saline containing 0.1% polysorbate 80. Aliquots of these dilutions were plated as described, except that 2% malt agar was not used. The plates were incubated at 24°C for 10 days, the colonies were counted, and results were expressed as CFU per gram.

STATISTICAL ANALYSIS

Mean concentrations of fungi in the air and on surfaces were calculated for the homes of patients and controls by dividing the total number of CFU by the number of plates from each home. LogXact¹¹ was used to calculate the matched odds ratio (OR) for a change of 10 units in the mean concentration of *S atra* in the homes of the patients compared with the homes of the controls. Mean concentration of *S atra* is a continuous predictor in the logistic model. It follows that the slope coefficient for *S atra* gives the change in log OR for an increase of 1 unit in mean concentration. Since this was unlikely to be of interest biologically, we decided to consider a change of 10 units in mean concentration of *S atra*.¹² To test for interaction with environmental tobacco smoke, a multivariate logistic model was constructed that also controlled for the matching.

fants to live in a house with a history of water damage from roof or plumbing leaks or flooding.⁴

The geographic clustering of the patients' homes, the history of recurrent bleeding, and the history of water damage in the homes led us to hypothesize that the infants had been exposed to a toxic substance in their homes. We found no differences between patients and controls

with respect to exposures to household pesticides, infant care products, or cocaine. After ruling out all other known causes of pulmonary hemorrhage (including coagulopathies and abuse), the finding of hemolysis on peripheral blood smears led us to consider the possibility that the infants had been exposed to toxins produced by indoor molds. Specifically, our a priori hypothesis was

Table 1. Most Common Presenting Symptoms Among Infants With Pulmonary Hemorrhage*

Prodrome	% of Patients	Onset	% of Patients
Abrupt cessation in crying	90	Acute hemoptysis	100
Limpness	86	Lethargy	100
Pallor	86	Grunting	90
Color change	90	Respiratory failure	100

*From Montaña et al.⁴

Table 2. Unmatched Analysis of Filter Samples*

Organism	Mean CFU/m ³	
	Patient Homes (n = 9)	Control Homes (n = 27)
<i>Aspergillus</i>	23 111	445
<i>Cladosporium</i>	1434	27
<i>Penicillium</i>	755	122
<i>Stachybotrys</i>	43	4
Other†	3880	109
Total viable fungi	29 227	707

*CFU/m³ indicates colony-forming units per cubic meter of air. Findings are averaged across all media; mean of means is not the total mean.

†Includes all other fungi observed.

that infants with pulmonary hemorrhage were more likely than controls to live in homes where *Stachybotrys atra* was present. This fungus is known to grow in water-damaged homes⁵ and to have toxins that produce hemorrhagic disease and hemolysis in animals.⁶

RESULTS

DESCRIPTION OF ILLNESS

Acute pulmonary hemorrhage occurred in infants who were previously in excellent health. Parents or caregivers noted that the infant abruptly stopped crying, became limp and pale, and then coughed up blood, started grunting, and stopped breathing (**Table 1**).⁴

COMPARABILITY OF GROUPS

Patients and controls appeared to be from relatively comparable socioeconomic settings. For example, mothers of 80% of patients and 83% of controls were receiving Medicaid assistance. Mean maternal ages (21.2 vs 24.3 years), mean maternal education (11.4 vs 11.5 years), and use of an air conditioner (25% vs 29%) were also comparable in both groups.⁴

DESCRIPTION OF HOMES

We were able to gain entry to homes of 9 of the 10 patients and 28 of 30 controls. Patients lived in homes that were an average age of 76 years (range, 59-89 years), whereas controls lived in homes that were an average age of 75 years (range, 35-95 years).

Table 3. Matched Odds Ratios (ORs) for Selected Variables Using Fungal Concentrations*

Variable	OR	Exact 95% Confidence Interval	P
Air Filter Samples†			
Mean fungal CFU/m ³	1.00	0.99-1.01	.07
Mean <i>Aspergillus</i>	1.00	0.99-1.01	.20
Mean <i>Cladosporium</i>	1.07	1.00-1.18	.02
Mean <i>Penicillium</i>	1.01	0.99-1.04	.07
Mean <i>Stachybotrys</i>	9.83	1.08-3 × 10 ⁶	.007
Mean other fungi‡	1.06	1.00-1.16	.009
In Surface Samples§			
Mean fungal CFU/g	1.20	1.03-1.74	.003
Mean <i>Aspergillus</i>	1.31	0.95-2.82	.10
Mean <i>Cladosporium</i>	1.16	0.86-1.92	.20
Mean <i>Penicillium</i>	1.38	0.87-3.14	.20
Mean <i>Stachybotrys</i>	1.35	0.99-1.5 × 10 ¹⁴	.20
Mean other fungi‡	1.16	0.99-1.45	.05

*CFU/m³ indicates colony-forming units per cubic meter of air sampled.

†The OR for a change of 10 units in the variable.

‡Includes all other fungi observed.

§The OR for a change of 1 million units in the variable.

AIRBORNE CONIDIA

Microscopic analyses of dust for airborne *S atra* spores detected *S atra* spores in homes of 7 of 9 patients vs 9 of 28 controls.

VIABLE AIRBORNE FUNGI

The relative concentrations of the various categories of fungi in filter cassette samples from homes of patients and controls are shown in **Table 2**. An unmatched analysis shows that mean CFU counts for all fungi averaged 29 227 CFU/m³ in homes of patients vs 707 CFU/m³ in those of controls. The mean concentration of *S atra* was 43 CFU/m³ in homes of patients and 4 CFU/m³ in homes of controls when averaged across all media. *Stachybotrys atra* was detected in filter cassette samples from homes of 5 of 9 patients vs 4 of 27 controls. The matched OR for a change of 10 units in the mean concentration of *S atra* on the filters was 9.83 (exact 95% confidence interval [CI], 1.08-3 × 10⁶). In other words, if there was a 10-CFU/m³ increase in the concentration of *S atra* in the air the infant breathed, then the infant was 9.83 times more likely to be a patient (**Table 3**).

To test for interaction with environmental tobacco smoke, a multivariate matched analysis assessed the impact of *S atra* concentration and exposure to environmental tobacco smoke and showed an OR of 21 (95% CI, 1.07-7.5 × 10⁶) for an increase of 10 units in the mean concentration of *S atra* in the presence of environmental tobacco smoke.

SURFACE SAMPLES

The relative concentrations of the various categories of fungi in surface samples from homes of patients and controls are shown in **Table 4**. An unmatched analysis shows that the mean concentration of *S atra* was 20 × 10⁶ CFU/g

Table 4. Unmatched Analysis of Surface Samples*

Organism	Mean CFU/g	
	Patient Homes (n = 7)	Control Homes (n = 19)
<i>Aspergillus</i>	5.6×10^6	2.8×10^6
<i>Cladosporium</i>	1.9×10^6	3.9×10^6
<i>Penicillium</i>	1.9×10^6	0.84×10^6
<i>Stachybotrys</i>	20×10^6	0.007×10^6
Other†	11×10^6	7.9×10^6
Total viable fungi	40×10^6	15×10^6

*Data are averaged across all media. Mean of means is not the total mean.
CFU indicates colony-forming units.

†Indicates all other fungi observed.

Table 5. Detection of *Stachybotrys atra* in Patients' Homes Using Several Methods*

Set	Patient No.	<i>S atra</i> on Filters, Mean CFU/m ³	<i>S atra</i> on Surfaces, Mean CFU/g	<i>S atra</i> Spores Present
1	1	153	2.4×10^7	Yes
2	2	0	0	Yes
3	3	32	...	Yes
4	4	87	1.1×10^8	Yes
5	5	0	...	No
6	6	0	0	Yes
7	7	...	...	...
8	8	4†	92†	Yes
9	9	144	0	Yes
10	10	0	2.2×10^3	No

*Set designates the matched set to which each patient belongs.

CFU/m³ indicates colony-forming units per cubic meter of air;
ellipses, no sample obtained for analysis.

†Half the limit of detection was used when detected but too few to count
(divided by the number of plates).

and 0.007×10^6 CFU/g in homes of patients and controls, respectively. *Stachybotrys atra* was found in excess of 10^8 CFU/g in surface samples in homes of some patients. *Stachybotrys atra* was detected in surface samples from homes of 4 of 7 patients vs 10 of 19 controls (**Table 5** and **Table 6**). The matched OR for a change of 1 million units in the mean surface concentration of *S atra* was 1.35 (exact 95% CI, 0.99 - 1.5×10^{14}).

Homes of only 1 of the 9 patients failed to reveal the presence of *S atra* using at least 1 of these detection methods, in contrast to those of 12 of 28 controls (OR, 4.9 [95% CI, 0.5-255.6]) ($P = .26$).

COMMENT

The results of our study suggest that infants with acute pulmonary hemorrhage were more likely than controls to live in homes that had molds, including *S atra*, in the air. The spores of *S atra* contain potent mycotoxins, and we hypothesize that pulmonary hemorrhage occurred after the infants inhaled these spores.

A unique set of circumstances in Cleveland may explain, in part, why this cluster of cases of pulmonary hemorrhage occurred there. The neighborhood in which most

Table 6. Detection of *Stachybotrys atra* in Control Infants' Homes Using Several Methods*

Set	Control	<i>S atra</i> on Filters, Mean CFU/m ³	<i>S atra</i> on Surfaces, Mean CFU/g	<i>S atra</i> Spores Present
1	11	3	8.3×10^4	Yes
1	12	0	...	No
1	13	0	0	No
2	14	35	3.2×10^4	Yes
2	15	...	4.4×10^5	No
2	30	0	0	No
3	16	38	4.9×10^5	No
3	17	0	...	No
3	33	0	0	Yes
4	34	0	0	No
4	18	0	...	Yes
4	19	0	...	No
5	20	0	...	Yes
5	21	0	2.8×10^3	No
5	22	0	1.9×10^3	Yes
6	23	0	...	No
6	24	0	0	No
6	32	0	1.3×10^4	No
7	25	0	1.2×10^5	No
7	26	...	...	...
7	31	0	0	No
8	27	...	...	...
8	28	0	...	Yes
8	29	0	0	No
9	35	0	0	No
9	39	23	...	Yes
9	40	0	2.7×10^3	No
10	36	0	7.7×10^4	No
10	37	0	0	Yes
10	38	0	...	No

*Set designates the matched set to which each patient belongs.

CFU/m³ indicates colony-forming units per cubic meter of air;
ellipses, no sample obtained for analysis.

of the patients resided consisted of older homes, some in poor repair. Roof and plumbing leaks and flooding with standing water in many of basements were commonly reported, resulting in conditions suitable for the growth of a variety of fungi, including toxigenic *S atra*. Many of the forced-air heating systems of these homes were designed so that return air for the furnaces was pulled from the basements. Because of limited resources, the patients' caregivers reported that water-damaged items were not removed from the homes.

The numbers of CFU per cubic meter of air sampled for all categories of fungi studied were consistently higher in homes of patients than those of controls when the air samples were collected using filter cassettes (Table 2), suggesting that conditions in these homes favored exceptional levels of fungal contamination.

In some homes, we were able to culture *S atra* from the air but did not find it on surfaces. This may be because the fungus was growing in areas not visible to the investigators, such as behind wallpaper or inside walls. In other homes, we cultured *S atra* from surfaces but did not find it in the air. This may be because the fungus, which is slimy and not easily aerosolized, was not in the air at the time of our sampling. Aerosolization may be an intermittent phenomenon.

Although *S atra* was found on surfaces in homes of 10 controls, we think that it may be necessary to aerosolize spores to put an infant at risk for pulmonary hemorrhage. The presence of this fungus on surfaces therefore may not be clinically relevant unless it is disturbed or becomes aerosolized.

Stachybotrys atra requires water-saturated, cellulose-based materials for growth in buildings.⁵ Its spores contain a variety of toxins,¹³ including the most potent members of a large family of mycotoxins called trichothecenes.¹⁴ Two specific trichothecenes produced by *Stachybotrys*, satratoxins G and H, are among the most potent protein synthesis inhibitors known.¹⁵

A study of the toxigenic potential of strains of *S atra* from the homes of Cleveland patients grown in the laboratory in pure culture on rice demonstrated that these isolates of *S atra* produced satratoxins G and H and a variety of other trichothecene mycotoxins.¹⁶

Species of the genera *Aspergillus* and *Penicillium* were abundant in the homes studied, which suggests the possibility that metabolites of *S atra* and of other fungi may be present together. Some of these species are also known to produce mycotoxins, eg, *Aspergillus ochraceus*, *Aspergillus versicolor*, *Penicillium aurantiogriseum*, and *Penicillium chrysogenum*.¹⁷ However, the matched ORs in Table 3 demonstrate that, in our study, there were no differences in concentrations of *Aspergillus* or *Penicillium* between patient and control homes.

In animals, exposure to trichothecenes has been associated with hemorrhaging and anemia, but this has not been reported previously in human infants

Young or immature animals are more susceptible to the toxic effects of trichothecenes than adults, and hemorrhage and karyorrhexis are conspicuous in rapidly dividing cells.¹⁸ It is possible that very young infants may be unusually susceptible because their lungs are growing rapidly. Conceptually, local inhibition of protein synthesis during the formation of the endothelial basement membrane is likely to lead to capillary fragility and subsequently to stress hemorrhage. Male animals may be more susceptible to these mycotoxins than female animals.¹⁹

In an earlier report from this investigation, exposure to environmental tobacco smoke appeared to increase the risk for acute pulmonary hemorrhage. Nine (90%) of the 10 patients were exposed to tobacco smoke in the home, whereas 16 (53%) of 30 controls were so exposed. In a matched analysis, exposure to tobacco smoke in the home showed an OR of 7.9 (95% CI, 0.9-70.6).⁴ Although no association between pulmonary hemosiderosis and environmental tobacco smoke exposure has been reported previously, idiopathic hemosiderosis has been linked to active smoking²⁰ in a 15-year-old boy. Secondary stressors such as tobacco smoke or other illnesses may play an important role in triggering overt pulmonary hemorrhage.

A variety of investigators have described the effects of exposure to *S atra* among adults.^{21,22} Bloody nasal discharge has been documented among adults with occupational exposure to *S atra*. Forgacs and Caril⁶ described "se-

vere pharyngitis, or burning sensation in the nose accompanied by bloody nasal discharge and a moderate to severe cough" in workers in whom illness developed after inhaling dusts from *Stachybotrys*-contaminated straw.

In animals, exposure to trichothecenes has been associated with hemorrhaging and anemia,²³ but this has not been reported previously in human infants. In mature mice, studies of intranasal administration of *S atra* spores demonstrated severe alveolar and interstitial inflammation with hemorrhagic exudate in the alveoli.^{24,25} Studies of the effects of inhalation exposure of another trichothecene, a biological warfare agent called T-2 toxin, have been made in several animal models. The effects of inhalation were noted to be much greater (>10 times) than those of intravenous exposure.²⁶

Stachybotrys atra is thought to be uncommon in North American homes. A study in California found about 3% of 70 homes to have this fungus.²⁷ A study of 52 homes in eastern Canada found *S atra* in 1 home.²⁸ A recent Canadian study surveyed 401 single-family homes in Wallaceburg, a largely rural community of 12 000 in southern Ontario, during the winter of 1994. Approximately 280 species of molds were recovered from dust samples collected in the living areas of the homes. *Stachybotrys* was found in 3 homes.²⁹ Thus, in large surveys of residential environments, *Stachybotrys* has not been listed among the most common fungi found indoors.

There are several limitations of our study. Home sampling for fungi occurred after the infants' hemorrhages, and the conditions at the time of sampling may not have reflected conditions during development of the hemorrhage. However, the fact that none of the patients' parents reported clean-up of water damage suggests the presence of long-standing mold problems. In previous studies, the concentrations of fungi in the air of residences was shown to differ considerably from week to week.³⁰

Since each home was sampled only once, it is possible that we may have misclassified some homes as negative for *S atra* when in fact they were positive. Air spore counts are known to increase with construction work and vacuuming of carpets.³⁰ It is well known that spores released in 1 part of a home can rapidly spread throughout the home on air currents.³¹ Since the sampling was performed with the environmental hygienists unaware of case status, however, any misclassification would have made an association between the presence of *S atra* and infant pulmonary hemorrhage less likely. Another limitation is that we cannot rule out association of pulmonary hemorrhage with exposure to other toxigenic fungi that we did not uniformly speciate and quantify.

Further research is needed to determine whether this association is causal. Although the association meets several of the epidemiologic criteria for causation (ie, strength of the association, specificity, biologic plausibility, and coherence), other criteria (ie, temporality and consistency with other studies) have not yet been fulfilled.³² Additional research is needed to determine whether exposure to toxigenic fungi such as *S atra* is associated with acute pulmonary hemorrhage in infants in other areas. Such work would be aided by the development of methods to detect spores of *Stachybotrys* or trichothecene metabolites in human tissue.

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they have discovered, not what spills off our tongues. Creating a comfortable rapport, asking a thoughtful question, and counting silently to 6 to allow thought are much better educational techniques than an "I know it all" speech and a slide full of data. Need I say that you yourself are unlikely to discover, to create new knowledge, if you don't have quiet in which to think?

3. Quiet balances perspectives. You have spent much of your time during the last 7 or so years with medical professionals. When you have ventured out into the "real world," you may have found yourself answering medical questions. You may notice increasingly that when people discover you are a physician, they become quiet, they defer. Without even realizing it, you can be seduced into believing that your narrow perspective is the only way of looking at the world and that your expertise is paramount.

During the past year, I witnessed a painful meeting. A young physician came to the School of Public Health to discuss a particular topic. Unfortunately, he only talked; he did not ask or listen. In the room were a dozen of the world's experts on the subject about which he was expounding. He was naive. He was rude. By playing doctor, he gained not one whit from the encounter. It was a wasted and embarrassing hour. Unless physicians preserve silence during which to listen to other persons, they are deprived of course-correcting information. Quiet helps one maintain perspective.

4. Quiet allows others to draw close. Having just completed the rigors of residency you are acutely aware that this profession can easily consume, or actually demand, all of you. It is difficult to maintain high-quality relationships. Being a full partner or parent or family member, as well as a full physician, will require quiet—quiet during which the ones you love can count on your presence. I mean something far more than making certain you are there in time to sing "Happy Birthday"; to see at least one touchdown, basket, or goal during the season; or to walk her down the aisle. I mean periods of unscheduled, unoccupied, availability.

Last week my oldest child graduated from high school. I know there have been special occasions I shall always regret having missed, but I also know that it has been during the quiet times we have spent together that we have gained the most. I remember, for example, questions that have come from him during the deep silence of a nighttime car trip. Questions like, "What do you really believe about God?" Questions that came because we had been quietly side by side long enough to be at ease. Sometimes there must be no telephone, no beeper, no schedule, no plan. For without true quiet, there is no opportunity for closeness.

5. And finally, quiet restores the soul. The practice of pediatrics draws from emotional as well as intellectual self. It beckons you to a privileged sphere, privileged because it allows you to dwell not only in your own life, but in the lives of others. I hope it does not sound trite when I say it; you may share full measure in the triumphs and tragedies of your patients. It will be the quiet times you grant yourself that allow you to cope with your experiences. Quiet times for reflection will be your protection against becoming so overwhelmed that you must build walls against strong feelings.

I have listed for you 5 important reasons to keep, that is, to treasure, quiet. Quiet allows your patients to speak. Quiet promotes learning. Quiet balances perspectives. Quiet allows others to draw close. Quiet restores the soul. You may be adding to this list even now. And so, if I have one wish as you leave us, it is that you will somehow recognize the importance of keeping quiet. I think it an essential, life-preserving skill for a pediatrician.

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Correction

Error in Acknowledgments. In the article titled "Acute Pulmonary Hemorrhage in Infants Associated With Exposure to *Stachybotrys atra* and Other Fungi," published in the August issue of the ARCHIVES (1998;152:757-762), the names of 3 additional authors were omitted from the Acknowledgments on page 762. A paragraph should have been included that reads as follows: "Additional authors include David R. Olson, PhD, of the Centers for Disease Control and Prevention, Atlanta, Ga; Bruce B. Jarvis, PhD, of the Department of Chemistry and Biochemistry, University of Maryland, College Park; and J. David Miller, PhD, of Agri-Canada, Ottawa, Ontario." The journal regrets the error.