The above letter was referred to the authors of the Editorial Retrospective in question, who offer the following reply:

To the Editor: Dr. Hrachovy and his colleagues are correct in taking exception to our statement implying that sudden infant death is always quiet cessation of breathing during sleep. Clearly, we are not in a position to assume that this is *always* the case. On the other hand, we recognize that it often is, on the basis of reports that no sounds were heard while an infant was close to the parents, who were awake at the time, and the fact that in most cases, death occurred when the infant was assumed to be asleep. Certainly, there is no uniform circumstance of death and no way in which one can make the prospective observations that would be relevant. Evidence that the sudden-infant-death syndrome is usually associated with a normal sleeping period comes from the studies of Beckwith.*

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*The sudden infant death syndrome. Rockville, Md.: Department of Health, Education and Welfare, 1975. (DHEW publication no. (HSA)75-5137.)

AN ASSOCIATION BETWEEN CRYPTOSPORIDIUM AND GIARDIA IN STOOL

To the Editor: Cryptosporidium species have recently been recognized as a cause of gastrointestinal disease in both immunocompromised and immunocompetent persons.[1,2] Since February 1, 1983, all stool specimens submitted to our Clinical Parasitology Laboratory for ova and parasite examination have been screened for cryptosporidium by a modified acid-fast method.[3] We wish to report an association between the presence of cryptosporidium and *Giardia lamblia* in stool.

As of November 1, 1983, cryptosporidium had been identified in stool specimens from 33 of 1290 patients (2.6 per cent). For five patients, positive findings were verified by Sheather's sugar-flotation method.[4] Five of the 33 patients had the acquired immunodeficiency syndrome, and 28 had no known immunocompromising diseases or treatments. Of the 33 patients, 10 (all immunocompetent) also had giardia trophozoites or cysts or both identified in at least one stool specimen that also contained cryptosporidium. Four patients with cryptosporidium and giardia were members of one family. Five patients, four with cryptosporidium and one (nonindex) with cryptosporidium and giardia, were members of another family. Excluding the nonindex cases of each family, 6 of 26 patients (23 per cent) with cryptosporidium had giardia found concurrently in their stool. Over the same period, giardia without cryptosporidium was identified in stool specimens from 101 of 1283 patients (7.9 per cent). These data indicate a significant association between cryptosporidium and giardia by Fisher's exact test ($P<0.02$). If Southeast Asian refugees are excluded from the total, then 5 of 25 patients (20 per cent) with cryptosporidium had giardia, whereas 48 of 1040 patients (4.6 per cent) had giardia without cryptosporidium ($P<0.006$ by Fisher's exact test). Other pathogens isolated from the stools of patients with cryptosporidium were *Entamoeba histolytica*, *Isospora belli*, ascaris, shigella, and salmonella, but none appeared to be significantly associated with cryptosporidium.

Our observations with crytosporidium and giardia are supported by the data of Jokipii et al.,[5] who described 14 immunocompetent patients with cryptosporidium. Three of the 14 (21 per cent) had giardia, with one also having *Yersinia enterocolitica*. Only 45 of 1422 stool specimens (3.2 per cent) examined in their laboratory had giardia. The reason for an association between the presence of cryptosporidium and giardia is not known, but the connection might represent a simultaneous infection from a common source, or infection with one parasite might predispose to infection or colonization with the other.

Our data also indicate that cryptosporidium may be identified relatively frequently in stool from immunocompetent patients, many with symptoms of gastroenteritis (unpublished observations), from the northeastern United States as well as from regions of Australia[1] and Europe.[5] We urge the medical community to look for cryptosporidium with or without giardia (or other pathogens) in stool specimens from symptomatic patients.

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MAJOR SURGERY AND SCLEROMYXEDEMA

To the Editor: Scleromyxedema (lichen myxedematosus) is a rare progressive skin disease characterized by deposition of mucin and proliferation of fibroblasts in the upper dermis. The fibroblasts contain abnormal fibrils, raising questions concerning their function.[1] Since fibroblasts are important for wound healing, patients with scleromyxedema may not tolerate major surgery. A review of the literature, including the most recent comprehensive reports,[2,3] since this disease was first described[4] in 1906, reveals only one report of a patient subjected to a major surgical procedure. In 1946 a young woman had an abdominal hysterectomy, salpingo-oophorectomy, and appendectomy without apparent difficulty.[5] Our recent surgical experience with a patient with severe systemic scleromyxedema prompted this report.

A 46-year-old white man presented in 1983 with a poorly differentiated squamous-cell carcinoma of the bladder invading the rectosigmoid colon. In 1972 a diagnosis of papular mucinosis had been established by identification of mucin deposits within papules involving the forehead, hands, perianal region, and feet. A slow progression of disease occurred, characterized by generalized xerosis, thinning of the hair, atrophic glistening epidermis with diffuse, marked thickening of the skin and subcutaneous induration, scattered papular lesions, flexion contractures of both hands and angles of the mouth, and sclerodactyly of the fingers. The patient also had sensory peripheral neuropathy, a prior myocardial infarction, grand mal seizures, and repeated bouts of pneumonia. Treatment with melphalan was begun in 1974 for associated paraproteinemia but was stopped in 1978 because of lack of effect. Intermittent corticosteroids were continued.

On November 9, 1983, after 2000 rad had been delivered to the whole pelvis preoperatively, the patient underwent a pelvic lymphadenectomy, radical cystectomy, and resection of the adjacent involved large bowel, with primary colorectal anastomosis and construction of a ureteroileal urinary conduit. The wound and all anastomoses healed per primum, and the patient was discharged on the 10th postoperative day. Despite deformity of the hands, manual dexterity gained as a ticket vendor allowed him to master his stoma appliance.

This report and the earlier one suggest that selected patients with scleromyxedema can tolerate major surgery despite the severity of their skin manifestations. It is intriguing to speculate on the etiologic relations among the underlying disorder, prolonged exposure to an alkylating agent, and development of squamous carcinoma. One other patient who had received chemotherapy was reported to have squamous carcinoma of the lip contiguous to a distinct lesion of lichen myxedematosus.[6] Too few cases have been described for causal relations to be inferred, but the association is worthy of observation as more patients are followed.

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