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Talc and Asbestos Contaminant of Rice

To the Editor.—Sacks of rice intended for consumption by Japanese in the markets of the Los Angeles area bear a warning that talc is added, and that the rice should be rinsed carefully before cooking. This means that asbestos is a likely additive, since asbestos is a frequent contaminant of talc. The two minerals are produced by the same geologic processes and both occur in the same deposits. A recent publication by the World Health Organization warns of the possible hazards of this contamination when talc is ingested.

Selikoff and his group have shown that asbestos exposure results in increased incidence of gastrointestinal tract cancer.[1] All this suggests that asbestos-contaminated talc may play a causative role in cancer of the stomach in Japanese. The incidence of this cancer in Japan is much higher than in other countries. Rice itself has been suspected for some time, since cancer of the stomach appears to follow roughly the rice-eating habits of the areas within Japan. Japanese who abandon their national eating habits on migrating to this nation lose their increased rate of cancer.

This relationship has long been suspected since there is an abundant although somewhat contradictory experimental literature demonstrating the production of lesions in the stomach of rats when fed polished rice.

In view of the established carcinogenicity of asbestos, the role of asbestos-contaminated talc in Japanese rice in the causation of the high rate of stomach cancer in that nation seems an attractive hypothesis.

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1. Selikoff IJ, Bader R, Bader M, et al: Asbestosis and neoplasia. *Amer J Med* 42:487-496, 1967.

Illegal Use of Psychoactive Drugs

To the Editor.—Illegal use of psychoactive drugs by young people has received great publicity in both lay and medical publications. Much interest has been expressed about what physicians can and cannot do about this problem. Paralleling the use of these illegal drugs is the massive consumption of prescription psychoactive drugs. American pharmacists filled 225 million prescriptions for stimulants, sedatives, and tranquilizers in 1970 (*New York Times*, March 14, 1971, p 36). Until recently, this phenomenon received very little attention.

Several issues require discussion. Does such widespread use reflect the common incidence of emotional problems seen by physicians? Are prescription drugs always good treatment for these problems? Are there more appropriate substitutes, such as counseling by the physician or referral to another professional for psychological evaluation and treatment? Is there a relationship between the massive use of legal prescriptions and nonprescription psychoactive drugs by adults and illegal drug use by young people? What responsibilities do physicians, drug companies, medical journals, and government agencies have in dealing with this problem?

Mind-affecting drugs are now an integral part of American life. Prescriptions are a major distribution route. Physicians must decide what role these drugs will play in their practices.

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Intracranial Hemorrhage In Hemophilia

To the Editor.—In a recent LETTER TO THE EDITOR (216:518, 1971), Quick made a statement to which I must take strong exception. He said: ". . . intracranial hemorrhage in hemophilia is rare."

I am of course aware of Dr. Quick's many contributions in the field of the coagulopathies. And, of course he is literally right in his statement. Intracranial bleeding in the hemophiliac *is* rare, compared with hemarthrosis, intramuscular hematoma, or renal bleeding. On the other hand, when it occurs, it is not infrequently the cause of death.

I feel that a known hemophiliac who has received trauma to the head should be observed closely for evidences of bleeding, and should be treated vigorously on the first appearance of such signs. I have on several occasions treated such patients preventively with infusions of factor VIII.

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Mercury Poisoning From Application to Omphalocele

To the Editor.—In 1957, Grob of Zurich recommended painting the exposed sac of an omphalocele with merbromin (Mercurochrome).[1] We employed this treatment for a giant omphalocele in a newborn, only to have the patient die of mercury poisoning. While this appears to be an infrequent occurrence, this potentially fatal means of treatment should be reported.

Report of a Case.—The patient was a 5 lb 8 oz black infant born to a gravida 1, para 0, O-positive, 28-year-old mother with normal results of serologic tests, at 36 weeks gestation. A giant omphalocele was present with an intact sac, and the base measured 5 × 6 cm. The distance from the xiphoid process to the pubic symphysis was 9 × 6 cm. The liver was within the omphalocele as well as were most of the intestines. The abdominal cavity appeared extremely small. Aside from bilateral undescended testicles all was normal. Initial treatment included intravenous fluid, penicillin, kanamycin sulfate, and, through misunderstanding, the exposed sac was painted with 1% merbromin every three hours for a total of three treatments. This was changed to 2% merbromin every 12 hours on the following day, when oral feedings were started and intravenous fluid administration stopped. Oliguria on the second day of life led to anuria on the third day and there was an episode of pink urine. Diffuse sclerema began on the third day. Because of a diagnosis of sclerema, pink urine, and anuria, mercury poisoning was considered, and an exchange transfusion was carried out. Because of the possibility of dehydration and anuria, the patient was challenged with a water load which resulted in 5 cc of urine. The omphalocele sac was painted with tincture of benzalkonium chloride (Zephiran); hydrocortisone and dimercaprol were administered. On the fourth day of life the sclerema seemed diminished, the infant was more active, and urinary output increased slightly. Respiratory arrest occurred on the fifth day of life and the infant died.

The pink urine, progressive anuria, and appearance of sclerema on the third day of life with oliguria and death on the fifth day exactly parallels the course of the patients who died under similar circumstances reported by Schippan and Wehram.[2] The level of mercury in the urine was 16 mg/liter in Schippan and Wehram's patients and 30 μg/deciliter in the blood of our patient. Lundgren et al[3] have found that blood levels of mercury in nor-

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