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LEAD POISONING — THE SILENT EPIDEMIC

LEAD poisoning is a serious, sometimes fatal, illness of known cause, readily diagnosed and treated and completely preventable in most cases. The disease presents clinically with a continuum of rather nonspecific gastrointestinal symptoms (anorexia, constipation, nausea, vomiting or colic) or central-nervous-system symptoms (irritability, confusion, lethargy, coma or convulsions). It is often misdiagnosed initially unless the physician has a high index of suspicion. Routine laboratory studies, which may reveal anemia, basophilic stippling and "lead lines" in long bones, should serve as clues but are not invariably present, particularly in the more acute cases. Lead presumably exerts its deleterious effects by inhibiting essential enzyme systems. It has been shown to interfere with hemoglobin synthesis and specifically to block delta aminolevulinic acid (δ ALA) dehydrase.[1] However, tests based on the finding of elevated urinary coproporphyrin or δ ALA have not proved sufficiently reliable for diagnosis although they may be helpful in some cases. The diagnosis of lead poisoning is essentially based on the finding of increased whole-blood lead levels; values in excess of 40 μg per 100 g are suspicious and 60 μg per 100 g or greater are diagnostic. Treatment consists of the separation of the patient from the source of lead, supportive measures and in cases with marked elevations of blood lead or severe symptoms, treatment with chelating agents (BAL, calcium EDTA and penicillamine).[2] There is as yet no general agreement about a precise level of blood lead at which to initiate chelation therapy, and clearly this is an area demanding prompt definitive investigation. Similarly, a rapid, precise micromethod for the determination of blood lead is sorely needed, as well as investigation of alternative diagnostic approaches.

In their illuminating article in this issue of the *Journal*, Klein and his co-workers have called attention to the serious potential hazard of lead poisoning from ceramic glazes. As they point out, this problem has been recognized throughout recorded history, and its recurrence now may reflect more the alertness of the authors than a major increase in the prevalence of the disease. This comment in no way should minimize their important contribution but rather serves to reiterate the adage that those who do not learn from history are destined to repeat its mistakes. The question of who now has the responsibility to test and certify earthenware as safe for use with food remains unanswered. Furthermore, who will make available to those of us already employing such dishware a means of testing its safety? Apparently, the Lead Industry Association and the United States Pottery Association have initiated a program of testing and certifying commercially produced dinnerware, but this presumably applies only to domestic large-scale production.[3] These questions

deserve prompt answers by the Food and Drug Administration. Beyond our national interest there is the equally serious question of education and safety standards in other countries, particularly Mexico, where large-scale daily use of such ceramics must pose a considerable danger.

As the authors have noted, lead poisoning in children is well known, being associated in this country almost exclusively with deteriorated housing, the defective walls and woodwork of which provide a limitless source of ingestible old lead paint.* Children between the ages of one and six constitute the majority of cases. Of those who present with encephalopathic signs and symptoms approximately a third have well documented permanent mental retardation, and many will require institutional care for life.[4] Aside from the shocking human tragedy, institutional care for life may cost society in excess of $100,000 per child! In studies conducted in New York City, Chicago, Baltimore and Philadelphia over the past decade, 5 to 10 per cent of ghetto children in the age group from one to six years have been found to be poisoned, and in selected housing units frequencies as high as 20 per cent have been noted. In spite of these facts only a few cities have any rational approach to this silent epidemic. New York City, in response to relentless citizen and professional pressure, has developed the most serious effort to date to identify poisoned children. New York's blood lead screening is currently obtaining about 4000 samples a week. In an effort to prevent re-poisoning the City health code now requires repair of the child's apartment by the landlord (with tax-abatement incentive) and, failing his compliance, by the City's own housing department. The screening program has been most effective in areas where efforts have been made to work closely with ghetto residents, including paid teen-age workers. Skepticism remains over whether the repair program will prove adequate to the increasing demand, and already serious delays are being encountered. It should also be noted that this major effort is directed at finding already poisoned children and in no way effectively deals with the problem of prevention of poisoning in families not yet affected. Yet New York's efforts are probably the best in the country. An as yet unpublished study recently concluded by the New York Scientists Committee for Public Information reveals that in only seven of 50 states and six of 30 major cities is lead poisoning a reportable disease. This study found only two states and seven cities with screening programs, and three of these cities have only pilot programs. This observation may be considered against a background of crude estimates in excess of 200,000 currently poisoned children in this country!

Failure to attack this epidemic seriously is a national disgrace.† One cannot help noting the fact that the great bulk of affected children are poor and nonwhite. The charge of genocide by neglect has already been leveled against the health profession regarding this situation. This charge cannot be readily dismissed. The problem of lead poisoning in ghetto children will not be solved until we deal with our failure to provide truly comprehensive family care with an emphasis on preventive medicine for all our citizens. Furthermore, in this particular instance of hazardous urban pollution we must reconsider our current attitudes toward housing, which here is clearly the epidemic vector. The solutions will of necessity be radical, but we are dealing with a major epidemic. It would be a beginning for those reading this editorial to explore what their hospital and community are doing about this epidemic made possible by shameful neglect. In this particular situation, it should be clear to all who call themselves health professionals that "if you are not part of the solution, you are part of the problem!"

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*After World War II, titanium oxide paint became competitive and to a large extent replaced lead-containing inside house paint.

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†Currently several bills in congressional committee are directed toward providing funds for lead-poisoning programs. These include HR9191, 9192, 11699 (Ryan), HR17027 (Green), HR17234 and 17260 (Barrett), and S3216 (Kennedy).

THE CRYSTAL BALL

RESEMBLING certain members of the animal species, more than a few new drugs, while demonstrating admirable familial traits, also indulge in streaks of abnormal behavior. The volatile inhalation anesthetics provide no exception to this rule although early in the history of use, their deviations are apt to be concealed in the complications of surgery. Over the last 15 years chemists have concentrated on the synthesis of new halogen-substituted hydrocarbons in an effort to eliminate undesirable features of the primordial anesthetics. The result has been the introduction into clinical anesthesia of several compounds incorporating the most wanted characteristic — inertness, therefore nonflammability and freedom from toxicity.

The best known of the new breed is halothane (Fluothane). However, readers of this and other periodicals must have become convinced of the association between this volatile liquid and the development of post-anesthetic hepatic necrosis, presumed by many to be a sensitizing phenomenon.[1]