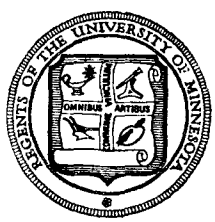
Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation



Intoxications in Neurology

Volume XXVI | Friday, February 11, 1955 | Number 16

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

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Published weekly during the school year, October to June, inclusive

The Bulletin is sent to members of the Minnesota Medical Foundation.
Annual membership fee - $10.00.

Address communications to: Staff Bulletin, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14, Minn.

I. INTOXICATIONS IN NEUROLOGY

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The problem of human exposure to the hazards of toxic materials is of ever increasing importance in all fields of medicine. Continually expanding industrialization, the introduction of chemicals into agricultural technique, and the growing utilization of therapeutic drugs are among the prime factors contributing to the high incidence of clinical intoxication. Accidental ingestion, as well as attempts at suicide or homicide are also prominent in the production of toxic symptomatology.

Among the most severe consequences of intoxication is nervous system damage of either temporary or permanent nature. In the classic neurologic involvement produced by toxic substances, a syndrome is produced which is characteristic of the activity of the offending material on nervous structures. However, in the majority of cases the reaction is complex and produces syndromes varying from the classical. As a consequence, it is often necessary to utilize great resourcefulness in the recognition and therapy of nervous system intoxications.

One factor contributing greatly to the complexity of nervous system intoxication is the accompanying involvement of other body organs. The failure of hepatic detoxifying mechanisms, the inability of renal excretion to proceed at a normal pace, the anoxemia resulting from depressed hematopoetic function, or other of the intricate interrelationships of these organs with nervous structures may result in additional nervous system findings.

In certain circumstances the intoxication may deviate from its expected clinical course and mimic such clinical entities as multiple sclerosis, anyotrophic lateral sclerosis, combined system disease, Guillain-Barré syndrome, cerebellar degeneration, intracranial neoplasms or nervous system infections. In other circumstances, some pre-existing neurologic disease may complicate the clinical response to nerve toxins. Exposure to some offending material may then result in exacerbation or accentuation of earlier symptoms.

Classification

Although large numbers of nerve toxins are as yet unidentified, the principal exogenous materials known to produce nervous system symptoms constitute an imposing list and may be classified in the following manner:[1]

TABLE I

Exogenous Toxins
Affecting the Nervous System

1. Metals and nonmetallic elements - arsenic, lead, mercury, manganese, thallium, antimony, zinc, barium, lithium, copper, silver, gold, and phosphorus

2. Organic solvents - Benzene, gasoline, toluene, alcohols, acetone, ethylene glycol, carbon disulfide, carbon tetrachloride, dioxine, trichlorethylene, tri-orthocresol and tetrachlorethane

3. Narcotics - opiates, synthetic drugs, cocaine, cannabis, mescaline and kava kava

4. Hypnotics and anticonvulsives - barbiturates, bromides, chloralhydrate, paraldehyde, hydantoins, diones and sulfones

5. Analgesics and antipyretics - salicylates, coal tar derivatives

6. Convulsants and anapleptics - strychnine, amphetamine, apomorphine, metrazol, picrotoxin, camphor, caffeine, bulbocapnine and absinthe

7. Autonomic nervous system drugs - belladona alkaloids, pilocarpine, physostigmine, prostigmine, mestinon

organic phosphorus insecticides and synthetic drugs

8. Antihelminthics - Oil of chemopodium, thymol, aspidium, santonin

9. Antimalarials - atabrine, quinine and quinidine

10. Chemotherapeutic and antibiotic drugs - sulfonamides, penicillin and streptomycin

11. Cardiac drugs - digitalis and quinidine

12. Cyanides and thiocynates

13. Food poisons - ergot, mushroom and lathyrysm

14. Miscellaneous - DDT and BAL

The incrimination of an agent responsible for nervous symptoms is often complicated by the fact that the patient has been exposed to a number of possible intoxicants. In some circumstances only one of the multiple agents is at fault, while in others the toxic symptoms may result from the action of several materials. This latter situation is frequently encountered in the organic solvent intoxications. These solvents are commonly employed in many industrial processes, in the home and in medical therapy. As a consequence this group of toxins represents an ever increasing source of nervous system intoxication in modern life.

The number of possible intoxications is too extensive to enter into a detailed description of the clinical aspects of each, and the manifestations and therapy of many of the more common intoxicants are familiar to the clinician. Therefore this presentation will be devoted to varieties of intoxication encountered in the Division of Neurology of the University Hospitals, together with a discussion of selected cases. Ethyl alcohol intoxication represents a separate and complex neurologic problem and will not be considered at this time. Carbon monoxide poisoning is similarly not considered since its adverse effects are considered to be of anoxic origin.

In the five and one half year period between January 1, 1949, and June 30, 1954, 43 cases were encountered on the neurologic service, warranting a diagnosis of toxic involvement of the nervous system. (Table 2) In 14 of these, the diagnosis was made by laboratory procedures added to the history of exposure. The laboratory procedures consisted principally of chemical identification of excessive amounts of the offending material in the blood or urine. In an additional 16 cases, the diagnosis was reached by a careful survey of the historical evidence of known exposure to excessive amounts of a nervous system toxin associated with nervous system symptoms. In the remaining 13 cases, the clinical findings led to a strong suspicion of some responsible toxic agent, but no definitive material could be incriminated.

The multiplicity of possible offending agents is well demonstrated by several cases in this study. Three of the six patients in whom excessive quantities of lead were demonstrated in the urine also exhibited elevation of the urinary arsenic to toxic levels. Intoxications consequent to exposure to organic solvents particularly well demonstrate the presence of multiple offending agents, since solvents employed in industry generally consist of a number of chemical compounds. Virtually all patients with organic solvent intoxications in this study demonstrated such a multiple exposure. One intoxication followed the use of a spray type permanent wave preparation in which a number of organic solvents were included. In another case the toxic symptoms followed exposure to tractor fuel employed as a degreasing agent. Episodes of intoxication also occurred during operation of the tractor in which the factors of breakdown products of the fuel and carbon monoxide were added. In one of the remaining cases in this group intoxication followed the ingestion of shoe polish containing a number of

TABLE 2

Nervous System Intoxication

Division of Neurology, University Hospitals
1/1/49 - 6/30/54

I. Diagnosis by history of exposure and chemical demonstration of the toxin - 14

Lead	6
(lead alone	3)
(lead plus arsenic	3)
Barbiturate	4
Mercury	2
Bromides	2

II. History of heavy exposure to toxins related to the appearance of neurologic symptoms - 16

Mercury	2
Barbiturates	2
Benzene	1
Toluene	1
Lead	1
Arsenic	1
DDT	1
Sulphur dioxide	1
Desoxyn	1
Artane	1
Spray Net (cold wave preparation)	1
Tractor Fuel	1
Heet, antifreeze, etc.	1
Shoe polish	1

III. Strong suspicion of nervous system intoxication without identification of the toxic agent - 13

organic solvents. Another instance of intoxication occurred in a chronic alcoholic so undiscriminating in dietary habits as to ingest Heet, containing methyl alcohol, ethylene glycol anti freeze, and any other liquid suspected of having inebriating qualities. Even the two cases listed as benzene and as toluene intoxication were complicated by exposure to other organic solvents.

Organic Solvents: Widespread utilization of various organic solvents results in the exposure of large segments of our population to possible intoxication with these materials. Six of the 30 cases in which the toxic agent was known resulted from excessive exposure to organic solvents. Solvents are widely employed in a large number of industrial processes, in dry cleaning procedures, as a degreasing agent in garages and other such mechanical enterprises, as an antifreeze agent, as motor fuels, and in fire extinguishers. Intoxication has resulted during industrial use as well as during medical usage as an antihelminthic, analgetic or anesthetic agent, as a therapeutic measure in leukemia or polycythemia, as a liniment or sponge bath, as a plast cast substitute, and as a solvent in removing adhesives from the skin. Intoxications have also been noted when

organic solvents have been employed in hair sprays, as a shampoo, as an insecticide, following aspiration during siphoning, sniffing or ingesting the material as an inebriant and on occasion as a suicidal agent.[1]

Intoxication by organic solvents most commonly results from inhalation although skin absorption and ingestion provide other avenues of entrance of the toxic material into the body.[1] One of the most hazardous present uses of solvents is in the form of degreasing agents. Most commonly the degreaser remains in an open vat; the greasy parts are immersed and scrubbed in the liquid by hand. Inhalation of the concentrated fumes while bending over the vat provides the greatest intake of the intoxicant; however, skin absorption also plays a role. It is frequently difficult to ascertain the exact toxin responsible for degreaser intoxications. The materials most commonly employed are commercial preparations usually prepared by the petroleum industry. The trade name for such substances may remain constant, but the exact contents are often varied periodically depending upon the solvents available at any given time as a by-product of refining processes. Solvents most commonly employed in degreasing preparations include trichlorethylene, carbon tetrachloride, alcohols, petroleum hydrocarbons, and on occasion benzene. Degreaser intoxications may be further complicated in garage mechanics and others in whom carbon monoxide exposure is added to that of the organic solvent.

Symptoms of intoxication by organic solvents may follow acute exposure to excessive amounts of the toxin or may result consequent to repeated contact with lesser concentrations. Great variations exist in the exact quantity of the agent required to produce intoxication. On a given exposure one individual may exhibit severe intoxication while others may be relatively unaffected. Experimental animal intoxications now in progress have emphasized this individual variation as well as demonstrating that any individual animal may exhibit varying degrees of toxic reaction to identical quantities of the solvent on different exposures. These variations occur in the absence of any detectable physical alteration. However, concomitant or preceding disease of almost any nature may lower tolerance to the toxin.

The symptoms of acute intoxication vary dependent upon the nature and amount of the solvent concerned. The neurologic symptoms most commonly observed include rapidly developing headache, euphoria, or depression, memory defects, confusion, delerium, ataxic gait, pupillary dilatation, cranial nerve pareses, convulsions, psychotic behavior, and ultimately unconsciousness, respiratory difficulties and death.

Chronic involvement by the organic solvents exhibits a more variable picture. The common findings include confusional states, memory defects, personality alterations, loss of libido, irritability, insomnia, retrobulbar neuritis which may lead to ultimate optic atrophy and blindness, cranial nerve pareses, convulsions, parasthesias and hypesthesias, and parkinsonian symptoms. As the intoxication progresses, secondary infections are common and may prove fatal.

In most organic solvent intoxications specific therapy is unavailable. The patient must be removed from all sources of the intoxicant. Symptomatic therapy and diligent nursing care are essential. In benzene intoxication, therapy must be directed against bone marrow depression as well as nervous symptoms, while in carbon tetrachloride and tetrachlorethane toxicity treatment must be instituted to combat hepatic involvement. In methyl alcohol intoxication early alkalinization with intravenous sodium bicarbonate or sodium lactate is essential to combat dangerous acidosis.

Case 1

An illustrative case of organic solvent intoxication occurred in a 37 year old white male arsenal worker

presenting a history of gradual onset of incoordination first noted in his right lower extremity about three years prior to hospitalization. This difficulty gradually progressed to involve the left lower extremity and the fingers of both hands. Approximately two years before admission, he first noted transient periods of loss of sight in the lower half of the left eye. His speech also became indistinct and became progressively worse until his hospitalization in June, 1952. Neurologic examination on admission revealed visual scotomata on the left. There was a right peripheral facial weakness and the speech was dysarthric. The right deep tendon reflexes were hyperactive and extensor Babinski responses were present bilaterally. Incoordination was elicited in the lower extremities as well as in the right upper extremity. Tremor was noted in both hands. The gait was ataxic and the Romberg test was positive. Further interrogation of the patient revealed a four year exposure to heavy concentrations of degreasing agents containing large amounts of benzene. Marked improvement followed withdrawal from contact with the toxin and the administration of BAL (2,3 dimercaptopropanol). Two years following hospital discharge the patient manifested no symptoms other than a slight tremor of the hands.

Comment

This patient was classified in group II as exhibiting nervous symptoms compatible with intoxication, together with a history of heavy exposure to a known central nervous system toxin. The scattering of symptoms and signs including optic neuritis, cranial nerve, cerebellar, and long tract findings mimicked the syndrome of multiple sclerosis, lacking only the factor of recurrence. BAL was employed because of its known effect against enzyme poisons such as the heavy metals. Its efficacy in these cases is presumed to be a consequence of its ability to supply sulfhydryl groups with which the toxic metal preferentially unites, thus sparing similar groups in the enzyme molecule and permitting the continuation of enzyme activity. However, no evidence exists that organic solvents produce symptoms by such a mechanism.

Metals and Nonmetallic Elements: Metals and nonmetallic elements are among the more common toxins producing nervous system symptoms.[1] Lead, mercury, and arsenic alone accounted for 11 of the 30 cases in this series in which the toxic agent was identified. Under certain circumstances intoxication by some materials in this group results in a characteristic and readily identifiable clinical picture. Chronic arsenic poisoning is often characterized by a chronic dermatitis accompanied by headache, peripheral neuropathy, and a neurasthenia-like picture. Lead toxicity in children commonly produces an acute encephalopathy with evidence of increased intracranial pressure. In adults the clinical appearance is characteristically manifested by gastrointestinal complaints accompanied by mental impairment and peripheral neuropathy, particularly with bilateral wrist drop. A lead line at the gingival margin, anemia with basophillic stippling of erythrocytes, a dense band at the growing margin of the long bones roentgenographically, an elevated urinary excretion of coproporphyrin and demonstrably increased amounts of lead in the urine aid in the detection of intoxication from this metal. Thallium poisoning is characterized by alopecia accompanying neurologic symptoms, while chronic manganese intoxication manifests itself chiefly by a parkinsonian syndrome, often accompanied by pneumonitis.

Mercurial intoxication has been noted during industrial use, during medical therapy with mercurial diuretics or cathartics, following accidental ingestion and occasionally when the metal is employed as a suicidal or homicidal agent. Mercury intoxication is now appearing in agricultural workers who employ mercury-containing fungicides such as Ceresan in the preparation of certain grain seeds.[2] Acute mercury intoxication generally manifests itself by a severe renal

impairment with oliguria or anuria. However, on occasion the most prominent manifestation may be an acute psychosis.[1] More commonly the intoxication is chronic and is manifested by a gradually increasing weakness, tremor or personality change often accompanied by a neurasthenic picture with extreme lethargy and somnolence, dysarthria, and other neurologic findings. On occasion, as demonstrated by the following case report, the intoxication may be manifested by a syndrome characteristic of another neurologic entity such as amyotrophic lateral sclerosis. In addition, mercury poisoning has recently been implicated as the cause of acrodynia in infants.[3,4] The diagnosis of mercurial poisoning is aided by the demonstration of increased amounts of the metal in the urine and often by the presence of a brown or bluish line at the gingival margin.

As with other intoxicants, the therapy of involvement by metals and non-metallic elements is begun with removal of the patient from the source of intoxication. BAL (2,3 dimercaptopropanol) is effective in increasing the urinary excretion of the toxin in cases of arsenic, lead, mercury, thallium and gold intoxication and often results in alleviating the clinical symptoms.[1] Attention has recently been directed to the therapeutic use of "chelating" agents capable of binding lead in a nontoxic form in which it may be excreted. Enthusiastic reports concerning the use of disodium calcium versenate (ethylene diamine tetra-acetate) in lead intoxication have appeared in the recent literature.[5,6,7]

Four cases of chronic mercurialism have been encountered in the present series, all as a result of Ceresan exposure. The following case illustrates one of the more severe intoxications.

Case 2

A 39 year old white male farmer first noted weakness of both arms in June 1951. Over the next four months there was progressive wasting of the muscles of the hands, arms, chest, abdomen, and pelvic girdle. These symptoms led to the performance of a spinogram which was interpreted as exhibiting no abnormality. Approximately six months following the onset of symptoms paralysis became almost complete and the patient experienced respiratory difficulty, dysphagia, dysphonia, and pooling of secretions of the pharynx. He was placed in a chest respirator which subsequently broke down necessitating his transfer to University Hospitals in December, 1951. On admission a bluish line was noted at the gingival margin. Neurologic findings included involvement of the lower cranial nerves as evidenced by dysphonia, dysphagia and excessive accumulation of salivary secretions. The deep tendon reflexes were absent in the upper extremities. The left ankle jerk was absent, but the remaining deep tendon reflexes of the lower extremities were hyperactive. Clonus was elicited from the right ankle and both knees. The Babinski reflex was extensor on the left and the abdominal reflexes were absent. The body musculature exhibited severe wasting and both coarse and fine muscle fasciculations were present throughout. Shortly following admission the urinary excretion of mercury was 340 micrograms in 24 hours as compared with normal values of 0.5 micrograms in the same period. Urinary co-proporphyrin excretion was 226 micrograms per liter.

Further questioning revealed that the patient had been employing Ceresan for approximately 7 years without regard to precautionary measures. This fungicide owes its activity to a mercury content of 7.7% in the form of phenyl mercury acetate.

BAL administration resulted in increasing the urinary mercury excretion to 740 micrograms in 24 hours. However, the patient failed to manifest clinical improvement, and he died after seven months of progressive bulbar involvement. The principal post mortem findings were in the central nervous system and consisted of pallor and vacuolization of the myelin in the pyramidal tracts in the brain stem

together with a moderate degree of demyelination and vacuolization of the lateral columns of the cord. The anterior horn cells were markedly decreased in number in some areas, and were absent in others. The remaining cells exhibited marked hyperchromaticity and shrinkage.

Comment

BAL therapy was effective in mobilization of the mercury and an increased urinary excretion of the metal. However, the clinical results were disappointing as often noted in far advanced cases of heavy metal intoxication. When involvement is less severe, BAL seems to be capable of arresting the progression of symptoms as was demonstrated in the remaining three cases of mercurial intoxication due to Ceresan.

Hypnotic and sedative drugs: These have been among the most common sources of intoxication for many years. Toxic symptoms have often resulted from the use of these drugs medically as a sedative or anticonvulsant. These substances have also been popular as suicidal agents and have occasionally been employed in attempted homicide. They have been included in patent medicines popular for a variety of complaints, and especially large amounts have been consumed by the addicted. In the past, intoxications resulted principally from the use of bromides or chloral hydrate. Bromides are excreted only slowly resulting in a marked cumulative effect of the drug. Bromide intoxications thus became particularly common in individuals under therapy for psychic disturbances, and in those consuming large quantities of patent medications such as Bromo-Seltzer, cold tablets, and the like.

Since the introduction of the barbiturates into therapy by Fisher in 1903, these drugs have largely replaced all others as the sedative of choice. They have been equally appealing to addicts and those attempting suicide. Their use is now of such proportion that over 400 deaths annually are attributed to excessive barbiturate consumption.[1] The fatal dose exhibits marked individual variation. Certain factors, including renal or hepatic disease with failure of detoxification or elimination of the drug, cardiovascular disease, respiratory infections and advanced age, tend to lower the tolerance. Renal or hepatic damage is of special import when these drugs are administered over an extended period. In such cases the diminished excretion or detoxification of the drug may allow toxic levels to appear in the body even though daily consumption has been held within supposedly safe limits. Ethyl alcohol is also reported to exert a synergistic effect with barbiturates and may thus lower the lethal dose.

Barbiturate intoxication may appear acutely as a result of overdosage or may appear chronically when the drug is ingested in smaller amounts over prolonged periods. Acute intoxication most commonly produces headache, drowsiness, confusion, and ataxia, which then proceeds into deep lethargy, and ultimately into coma. Convulsions or delerium may also occur at some time during the intoxication. The deep reflexes disappear and the Babinski reflexes become extensor. In fatal cases death may occur in respiratory failure, or when survival is of longer duration demise often results from secondary infection or respiratory obstruction.

When toxicity results from the chronic utilization of barbiturates, there is commonly ataxic gait, somnolence and dysarthria. Periods of agitation may occur as may tremors and occasional convulsions. The patient is often confused and thought processes are retarded. There may be emotional instability and dulling of the moral senses leading to obscenity and indecent exposure. Ocular manifestations are also common.

Therapy of mild intoxication requires little more than the withdrawal of the offending drug. In more severe cases, a variety of therapeutic measures has been utilized. The most popular therapy in the past has employed

analeptics such as picrotoxin, metrazol, amphetamine and caffeine with sodium benzoate in an effort to maintain life until the effects of the barbiturate have worn off.[1] Recently the efficacy of this treatment has been questioned[8], 9,10 and certain investigators have even considered this therapy to be detrimental.[8] Clemmesen[11] reduced the mortality of barbiturate poisoning in Copenhagen from 25% to 3.7% over a six year period, while avoiding the use of analeptics. His regime also included abolition of gastric lavage, the use of antishock measures consisting of positioning the patient and transfusion with blood or plasma when indicated, continuous oxygen therapy with constant attention to prevent obstruction of the pulmonary airway, the administration of antibiotics to prevent secondary infection, adequate fluid administration with particular attention to body electrolytes, and good nursing care including frequent turning of the patient. Despite these reports, clinical experience with barbiturate intoxication in this study indicates that judiciously employed analeptics still may be a valuable measure in the therapy of this toxicity. Robie[12] has advocated subconvulsant electroshock therapy, but corroboration of his efforts has not yet appeared in the literature.

The more rapid removal of barbiturates from the blood stream by means of hemodialysis employing the artificial kidney appears to offer promise in the therapy of this intoxication.[13,14] Studies in experimental animals have indicated that barbiturate coma in dogs given 40 milligrams of sodium pentobarbital per kilogram of body weight may be reduced from a 6 to 10 hour period to between 3 and 4 hours by hemodialysis.[15] Similar results have been noted in human beings. Hemodialysis has been thus employed in an attempt to shorten the period of unconsciousness and reduce mortality resulting from respiratory obstruction or secondary infections. An example of this therapy is afforded by the following case.

Case 3

A 27 year old housewife was found comatose after the ingestion of approximately 5 1/4 grains of phenobarbital and 13 1/4 grains of nembutal. Four hours later the patient was treated at a local hospital by picrotoxin and electrical stimulation. Continuing coma with ensuing atelectasis necessitated tracheotomy and the patient was then transferred to University Hospitals approximately 24 hours after ingestion of the drug. Administration of 1100 mg. of metrazol on admission failed to alter the course of the coma. A following dosage of 18 milligrams of picrotoxin was similarly unsuccessful. Hemodialysis was then initiated employing the artificial kidney. The blood barbiturate level at that time was spectrophotometrically determined to be 2.65 milligrams percent. Dialysis was carried out for six hours during which period the coma lightened, deep reflexes returned to the lower extremities, and the patient became restless, yawned and moved her extremities. At the termination of dialysis the blood barbiturate level was lowered to 1.58 milligrams percent and a total of 217 milligrams of barbiturate had been removed into the dialysis fluid. Twelve hours after cessation of dialysis the patient was oriented and sitting up in bed. The tracheotomy tube was removed the following day and within several days there was apparent recovery from all noticeable effects of the intoxication.

Comment

Slow renal excretion of barbiturates appears to be concerned with the prolongation of barbiturate coma. The more rapid removal of the drug by dialysis may then diminish the duration of the coma and thus decrease the possibility of respiratory and other complications. It has been suggested that hemodialysis may be of value in instances of 1) prolonged coma 2) pulmonary distress, 3) unresponsiveness to analeptics, 4) oliguria or hypotension tending to diminish the urinary excretion of the drug, and 5) concurrent renal or hepatic disease resulting in prolonged barbiturate retention.[13]

DISCUSSION

This series included 30 cases in which a specific toxin was indicated as responsible for nervous system symptoms, as well as 13 instances in which intoxication was strongly suggested despite inability to incriminate a specific agent. Nervous system symptoms occur relatively frequently as a result of an intoxicant. The increased utilization of potential toxins in industry, agriculture, medicine and the home leads to an expectation that even more such involvements can be expected in the future. Recognition of intoxication then becomes of increasing medical significance.

The diagnosis of nervous system toxicity is often difficult with means now at our disposal. An easily elicitable history of exposure or a clinical syndrome characteristic of a known toxic agent may alert the physician to the proper diagnosis. However, intoxication too commonly produces vague and uncharacteristic symptoms. The only clues may be diffuse or multi-focal, often bizarre symptoms, which may improve when hospitalization removes the patient from the source of the toxin. Even this improvement may be negated in barbiturate habituates, narcotic addicts and others who somehow arrange to obtain the toxic preparation although hospitalized. Hospitalization may also fail to provide adequate protection against the continued administration of some poison in homicidal attempts.

Relatively few laboratory procedures are available as aids in the detection of the majority of nerve toxins. Arsenic or heavy metals may often be demonstrable in the urine of intoxicated individuals. This determination, however, may fail in its purpose, since urinary excretion of the toxin may not be elevated even during profound intoxication. Such a situation may result from binding of the toxic material in the body tissues. Under certain circumstances, therapy with BAL or a chelating agent may mobilize the toxin in these individuals, thereby increasing urinary levels. Barbiturates may be spectrophotometrically identified in body fluids and bromides are also subject to quantitation. However, the great majority of nervous toxins cannot be conveniently identified once they have entered the body. This holds particularly true for the organic solvents which are now responsible for ever increasing numbers of clinical intoxications. Certain intoxications may be suspected because of evidence of accompanying hepatic or renal damage. However, such involvement is by no means necessarily associated with nervous symptoms during clinical toxicity. Other studies such as quantitation of urinary coproporphyrin, carbon dioxide combining power, and electrolyte quantitation may be of value, but again are too commonly non-contributory to the diagnosis of intoxication.

Even the historical evidence of exposure to a toxin, or the identification of toxic materials in body fluids may not conclusively demonstrate that toxin to be responsible for neurologic symptoms. The only criteria which can be employed in such circumstances is the relationship of toxic exposure to clinical symptoms and the remission of symptoms with appropriate therapy and cessation of exposure. These criteria are similarly not foolproof since multiple sclerosis and other neurologic conditions may be characterized by spontaneous remission.

The considerations presented lead to the suggestion that a strong index of suspicion of intoxication be maintained in all cases exhibiting nervous symptoms not adequately explained by another distinct pathologic process. Evidence of intoxication is also to be particularly sought in neurologic syndromes such as amyotrophic lateral sclerosis and others in which the prognosis is otherwise unfavorable.

SUMMARY AND CONCLUSIONS

1. Exposure to toxic materials in industry, agriculture, medical therapy and the home is an increasingly

common cause of nervous system symptoms.

2. A diagnosis of nervous system intoxication was warranted in 43 cases observed over a five and one half year period in the Division of Neurology of the University of Minnesota Hospitals.

3. Clinical toxicity may mimic virtually any nervous system disorder. Consequently, the physician must maintain a high index of suspicion, particularly in the presence of diffuse or multifocal symptoms.

4. Selected cases of organic solvent, mercury, and barbiturate intoxication are presented.

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II. MEDICAL SCHOOL NEWS

Coming Events

February 14 - 16	Continuation Course in Internal Medicine for Internists
February 17	Special Lecture; "The Effects of Disease on History;" Professor John Fulton, Yale University; Weyerhaeuser Room, Minnesota Historical Society Building, St. Paul; 2:00 p.m. (Tea hour following lecture)
February 17 - 19	Continuation Course in Cancer Detection for General Physicians
Feb. 28 - Mar. 2	Continuation Course in Clinical Hematology for General Physicians and Internists
March 21 - 23	Continuation Course in Cardiovascular Diseases for General Physicians

* * *

Continuation Course

A continuation course in Clinical Hematology for physicians will be presented by the University of Minnesota under the auspices of the Center for Continuation Study from February 28 to March 2, 1955. Registrants will be provided with slides for study during the course showing examples of various blood cell types and of the more frequent hematological diseases. Lectures will include discussions of the anemias, the leukemias, and coagulation defects and a clinical correlation conference will include a brief discussion of recent advances in therapy. Registration will be limited to 12.

* * *

In Memoriam

A.L. Searle

Members of the Minnesota Medical Foundation will be saddened to learn of the recent death of Augustus L. Searle, Patron Member and enthusiastic supporter of the Foundation and its objectives. President of the Searle Grain Company, Mr. Searle was also widely known as a sportsman and for his life-long interest in the arts and sciences. To his family we extend sincere sympathy on behalf of the Minnesota Medical Foundation.

* * *

Faculty News

Dean H. S. Diehl, Dr. William F. Maloney, Assistant Dean, and Dr. Robert B. Howard, Director, Department of Continuation Medical Education, attended the Annual Congress on Medical Education and Licensure which was held in Chicago from February 5 to 8. Subjects of particular interest which were discussed at this meeting included the use of television in medical education, the teaching of legal medicine, and the problem of graduates of foreign medical schools.

* * *

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

February 14 - 19, 1955

Monday, February 14

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - 12:30 Physical Medicine and Rehabilitation Staff Seminar; Film, "Improving the Functional Capacity of Severely Involved Upper Extremities," (Warm Springs, Ga.); Heart Hospital Theater.

11:30 - Tumor Conference; Doctors Hitchcock, Zimmermann, and Stenstrom; Todd Amphitheater, U. H.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:30 Physiology Seminar; Plasma K Concentrations During Inhalation of High Concentration of CO_2; E. B. Brown; 214 Millard Hall.

1:00 - 2:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U.H.

1:30 - 3:30 Dermatology Hospital Rounds; H. E. Michelson and Staff; Dermatology-Histopathology Room, C-394 Mayo Memorial.

4:00 - 6:00 Anesthesiology Conference; F. H. Van Bergen and Staff; Todd Amphitheater, U. H.

4:30 - Public Health Seminar; Subject to be announced; Edward A. Hoebel; Room 100, Mayo Memorial.

4:30 - Pediatric-Medicine Infectious Disease Rounds; Station 33, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Ancker Hospital

8:00 - 9:00 Pediatrics Contagion Rounds; Richard Lein; Contagion 5.

8:30 - 10:30 Medical and Surgical Chest Conference; Dr. Gehlen and Staff; Auditorium.

9:30 - 12:00 Visiting Staff Rounds.

10:00 - 12:00 Surgery Grand Ward Rounds; Begin Floor E4.

11:00 - 12:00 Pediatric Rounds; Harry Orme; Contagion 1.

12:30 - 2:30 Surgery Out-Patient Clinic; Room 8.

Monday, February 14 (Cont.)

Ancker Hospital (Cont.)

2:00 - 3:00 Routine EKG Interpretation; Dr. Sommers and House Staff; Medical Record Library.

2:30 - 3:00 Discussion of Problem Case; Auditorium.

3:00 - 4:00 Surgery Journal Club; Classroom.

3:00 - 4:00 Lectures on Electrocardiography; Ben Sommers; Auditorium.

4:00 - 5:00 Medical Clerk Journal Club; Auditorium.

Minneapolis General Hospital

10:30 - 12:00 Medicine Rounds; Thomas Lowry; Station 31.

11:00 - Pediatric Case Discussions; Erling Platou; Station 4.

11:00 - Orthopedic and Fracture Rounds; Drs. John Moe and Arthur Zierold; Station 20.

12:30 - Surgery Grand Rounds; Dr. Zierold, Station 21.

1:30 - 2:30 Tuberculosis Conference; J. A. Myers; Station 8.

2:00 - Pediatrics Rounds; William Krivit; Stations 4, 5, & 6.

Veterans Administration Hospital

9:30 - Infectious Disease Rounds; Drs. Hall, Zinnemann, and J. Brown.

1:30 - Cardiac Conference; Drs. Smith, Berman, Hoseth, Simonson, Tamlyn, and Farquhar; Conference Room, Bldg. I; Rounds immediately following conference.

Tuesday, February 15

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; Samuel Feinberg, John A. Anderson and Staffs; Eustis Amphitheater, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 104 Jackson Hall.

12:30 - Physiological Chemistry Seminar; Appearance of Specific Proteins During Embryonic Development; R. G. McKinnell; 214 Millard Hall.

12:30 - Bacteriology and Immunology Seminar; "Extra-Mendelian Mechanisms:" Insects and Protozoa. (CO_2 Sensitivity in Drosophila; Kappa Factor in Paramoecium; Cytoplasmic Segregation - Alternative Cytoplasmic Steady State - in Paramoecium); James Crawford; 1050 Mayo Memorial.

12:30 - Anatomy Seminar; Experimental Alteration of Cellular Ultrastructure; J. F. Hartmann; 226 Jackson Hall.

3:30 - General Physiology Seminar; 323 Zoology Building.

3:30 - Pediatric Seminar; Subject to be announced; Eleanor Colle; 1450 Mayo Memorial.

4:00 - 5:00 Pediatric Rounds on Wards; John A. Anderson and Staff; U. H.

Tuesday, February 15 (Cont.)

Medical School and University Hospitals (Cont.)

4:00 - 5:00	Physiology-Surgery Conference; Todd Amphitheater, U. H.
4:30 - 5:30	Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.
5:00 - 6:00	X-ray Conference; Presentation of Cases from Ancker Hospital; Drs. Aurelius and Clemett; Eustis Amphitheater, U. H.
*8:00 p.m.	Minnesota Pathological Society Lecture; "The Pineal Gland;" Dr. Mark D. Altschule, Associate Professor of Medicine, Harvard University Medical School, Boston; Mayo Memorial Auditorium.

Ancker Hospital

8:00 - 9:00	Pediatric Rounds; Dale Cumming; Contagion 1.
9:00 - 10:30	Visiting Staff Rounds.
9:00 - 12:00	Practical Diagnostic Clinic; Harry Orme; Out-Patient Department.
11:00 - 12:00	Medical X-ray Conference; J. R. Aurelius; Auditorium.
2:30 - 4:00	Routine EKG Interpretations; Resident Staff.
4:00 - 5:00	Medical-Pathological Conference; W. F. Mazzitello, Auditorium.

Minneapolis General Hospital

9:30 - 10:30	Obstetrics and Gynecology Staff Rounds; William P. Sadler and Staff; 301 Harrington Hall.
9:30 -	Pediatric Rounds; Elizabeth Lowry and A. Bridge; Station 5.
10:00 -	Psychiatry Grand Rounds; R. W. Anderson, Station 3.
11:30 - 12:30	Neurology-Neurosurgery Conference; Classroom, Station 8.
12:30 - 2:30	Dermatology Rounds on Clinic; Carl W. Laymon and Staff.
12:30 -	ECG Conference; Boyd Thomes and Staff; 302 Harrington Hall.
1:00 -	Tumor Clinic; Drs. Eder, Coe, and Lipschultz; Classroom.
3:30 -	Pediatric-Psychiatry Rounds; Jack Wallinga; Station 4.

Veterans Administration Hospital

7:30 -	Anesthesiology Conference; Surgical Conference Room, Bldg. 43.
8:30 -	Hematology Rounds; Drs. Hagen and Wexler.
8:30 -	Surgery Journal Club; Conference Room, Bldg. I.
9:30 -	Surgery-Pathology Conference; Conference Room, Bldg. I.
10:30 -	Surgery-Tumor Conference; D. Ferguson and J. Jorgens.
1:00 -	Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.

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* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.

Tuesday, February 15 (Cont.)

Veterans Administration Hospital (Cont.)

1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

4:00 - Thoracic Surgical Problems; Conference Room, Bldg. I.

5:00 - Fluid Balance Conference; Conference Room, Bldg. I.

5:30 - Physiology Seminar; Surgical Conference Room, Bldg. 43.

Wednesday, February 16

Medical School and University Hospitals

11:00 - 12:00 Pathology-Medicine-Surgery-Pediatrics Conference; Todd Amphitheater, U. H.

12:30 - 1:20 Radio-Isotope Seminar; Betatron Room in Cobalt Underground Section, U. H.

1:00 - 2:00 Dermatology Clinical Seminar; F. W. Lynch; 300 North Clinic.

1:30 - 3:00 Pediatrics Allergy Clinic; Albert V. Stoesser and Lloyd Nelson; W-211, U. H.

3:30 - 4:30 Dermatology-Pharmacology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:30 - 5:50 Dermatology-Infectious Disease Seminar; 3rd Floor, Conference Room, Heart Hospital.

5:00 - 6:00 Radiology Residents Lectures; Wilms Tumor and Related Conditions; Milton Reiser; Todd Amphitheater, U. H.

5:00 - 5:50 Urological-Pathological Conference; C. D. Creevy and Staff; A503, Mayo Memorial.

5:10 - 6:10 Endocrine Seminar; The Fine Structure of the Pituitary as seen by Electron Microscopy; Dr. Farquhar; 271 Lyon Laboratories.

5:30 - 7:30 Dermatology Journal Club and Discussion Group; Hospital Dining Room.

7:30 - 9:30 Dermatology Seminar; Review of Interesting Slides of the Week; Robert W. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; J. Noble; Auditorium.

11:00 - 12:00 Pediatric and Contagion Rounds; Harry Orme; Contagion 1.

11:00 - 12:00 Medicine Resident Rounds; W. F. Mazzitello.

3:30 - 4:30 Pediatric Surgery Conference; Harry Orme and I. D. Baronofsky; Auditorium.

Minneapolis General Hospital

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station 11.

11:00 - Pediatric Rounds; Erling Platou and Richard Raile; Station 6.

Wednesday, February 16 (Cont.)

Minneapolis General Hospital (Cont.)

12:00 - Surgery-Physiology Conference; Arthur Zierold and E. B. Brown; Classroom.

12:30 - Pediatrics Staff Meeting; Classroom, Station 4.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Surgical Conference Room, Bldg. 43.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

9:00 - Gastro-Intestinal Rounds; Drs. Wilson, Zieve, Ferguson, Brakel, Swenson, Nesbitt and Sadoff.

10:30 - Psychosomatic Conference; C. K. Aldrich; 7th Floor, Bldg. 43.

12:30 - Medical Journal Club; Doctors' Dining Room.

12:30 - X-ray Conference; J. Jorgens; Conference Room, Bldg.I.

1:30 - 3:00 Metabolic Disease Conference; Drs. Flink and Williams.

3:30 - Urology Pathology Slide Conference; Dr. Gleason; Conference Room, Bldg. I.

7:00 - Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, February 17

Medical School and University Hospitals

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Room 3.148 Mayo Memorial.

11:00 - 12:00 Cancer Clinic; K. Stenstrom, B. Zimmermann; Todd Amphitheater, U. H.

12:30 - 1:55 Physiology Seminar 210; Transport; Selected Topics in Permeability; Nathan Lifson; 214 Millard Hall.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

4:00 - 5:00 Anesthesiology Seminar; F. H. Van Bergen and Staff; Room 100, Mayo Memorial.

5:00 - 6:00 Radiology Seminar; Thoracic Surgery Conference; Thomas J. Kinsella; Eustis Amphitheater, U. H.

7:30 - 9:30 Physiology 211 Seminar; Selected Topics in Heart and Circulation: Hemodynamics; M. B. Visscher and Robert Evans; 271 Lyon Laboratories.

Ancker Hospital

8:00 - 9:00 Pediatric Clinical Staff Conference; Contagion Classroom.

9:00 - 10:00 Pediatric Contagion Rounds; Alexander Stewart, Contagion 5.

9:30 - 10:30 Medical Grand Rounds; Auditorium; Visiting Staff Rounds immediately following Grand Rounds.

11:00 - 12:00 Pediatric X-ray Conference.

Thursday, February 17 (Cont.)

Ancker Hospital (Cont.)

11:00 - 12:00 Medicine Resident Rounds; W. F. Mazzitello.

2:00 - 3:00 Routine ECG Interpretation; Ben Sommers; Medical Record Library.

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station 4.

9:30 - Pediatric Contagion Rounds; R. B. Raile; Station 4.

10:00 - Psychiatry Grand Rounds; R. W. Anderson and Staff; Station 3.

11:30 - 12:30 Clinical Pathological Conference; John I. Coe; Classroom.

12:30 - 2:30 Dermatology Rounds and Clinic; Carl W. Laymon and Staff.

1:00 - Fracture X-ray Conference; Drs. Zierold and Moe; Classroom.

1:00 - House Staff Conference; Station 4.

Veterans Administration Hospital

8:00 - Experimental Surgery Laboratory Meeting; Conference Room, Bldg. I.

8:30 - Hematology Rounds; Drs. Hagen and Doe.

9:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

9:00 - Surgery Ward Rounds; D. Ferguson and Staff; Ward 11.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

1:00 - Infectious Disease Conference; Conference Room, Bldg. I. (Rounds immediately following conference.)

4:00 - 5:00 Medical-Surgical Conference; Medical Conference Room, Bldg. I.

Friday, February 18

Medical School and University Hospitals

8:00 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

11:00 - 12:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Eustis Amphitheater, U. H.

11:45 - 12:50 University of Minnesota Hospitals Medical Staff Meeting; Epidemiology of Cold Injury; Leonard M. Schuman; Powell Hall Amphitheater,

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

1:00 - 2:00 Physiology Seminar 212; Selected Topics in Respiration: Respiratory and Circulatory Effects of Hypothermia; E. B. Brown; 214 Millard Hall.

1:30 - 2:30 Dermatology Grand Rounds; Presentation of Cases from Grouped Hospitals (University, Ancker, General and Veterans) and Private Offices; H. E. Michelson and Staff; Eustis Amphitheater, U. H.

Friday, February 18 (Cont.)

Medical School and University Hospitals (Cont.)

2:30 - 4:00 Dermatology Hospital Rounds; H. E. Michelson and Staff; Begin at: Dermatological Histopathology Room, C-394 Mayo Memorial.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

3:30 - 4:30 Dermatology-Physiology Seminar; 3rd Floor Conference Room, Heart Hospital.

4:00 - 5:00 Physiology Seminar 213; Selected Topics in Advanced Neurophysiology; Role of the Vestibular Apparatus and the Cerebellum in the Extrapyramidal Motor Activity; Werner Koella; 129 Millard Hall.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hanson and Staff; E-534, U. H.

5:00 - Urological Seminar and X-ray Conference; A503, Mayo Memorial.

Ancker Hospital

8:00 - 9:00 Pediatric Rounds; Charles Steinberg; Contagion 1.

10:30 - 11:30 Pediatric Contagion Rounds; Richard Smith; Contagion 1.

11:00 - 12:00 Contagion Rounds; Harry Orme; Contagion 5.

2:00 - 3:00 Routine EKG Interpretation; Resident Staff.

3:00 - 4:00 Medical-Surgical-Pathological Conference; Auditorium.

4:00 - 5:00 Medical Journal Club; Conference Room, E5.

4:00 - 5:00 X-ray Surgery Conference; Auditorium.

Minneapolis General Hospital

10:00 - Otolaryngology Conference; Robert A. Priest; Large Classroom.

10:30 - Pediatric Surgical Conference; Tague Chisholm and B. Spencer; Classroom, Station 4.

12:00 - Surgery-Pathology Conference; Drs. Zierold and Coe; Classroom.

1:00 - 3:00 Clinical-Medical Conference; Thomas Lowry; Classroom, Station 8.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

11:00 - 12:30 Psychiatry Case Conference; Werner Simon; Psychiatry Department, VA Hospital Annex.

12:30 - Urology X-ray Conference; X-ray Department.

1:00 - CPC Conference; Conference Room, Bldg. I.

2:00 - Pathology Slide Conference; E. T. Bell; Conference Room, Bldg. I.

Saturday, February 19

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 9:30 Pediatric Grand Rounds; Eustis Amphitheater, U. H.

Saturday, February 19 (Cont.)

Medical School and University Hospitals (Cont.)

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; Alexander R. Margulis, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Rounds; J. L. McKelvey and Staff; Station 44. U. H.

10:00 - 12:00 Otolaryngology Seminar on Current Literature; L. R. Boies and Staff; Todd Memorial Room, A-675, Mayo Memorial.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

9:30 - 11:00 Medicine Grand Ward Rounds; W. F. Mazzitello.

11:00 - 12:00 Medical Clerk Case Conference; W. F. Mazzitello.

Minneapolis General Hospital

8:00 - Urology Staff Conference; T. H. Sweetser; Main Classroom.

9:00 - Psychiatry Grand Rounds; R. W. Anderson; Station 3.

9:30 - Pediatrics Rounds on all Stations; R. B. Raile.

11:00 - 12:00 Medical X-ray Conference; O. Lipschultz, Thomas Lowry and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Medical X-ray Conference; Conference Room, Bldg. I.