MERCURY NEPHRITIS *

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The instances of renal disease in which exclusively one portion of the anatomical structure of the kidney has received injury are so excessively infrequent that they at once excite interest as means of testing our conceptions of physiological functions of this organ and the mode of origin of some signs of disease. These conditions are, patently, those of successful experimentation. That the occasional cases of toxic nephritis in man have not been more fruitful in yielding information applicable to broader studies is due chiefly to the great rapidity and severity of the symptoms, culminating after a few days in death. Instances of fatal poisoning with mercuric chlorid are common, and there is a well recognized type of lesion found in the kidney in these cases; but as the period of life after ingestion of the poison is relatively brief, the signs and symptoms of renal injury are obscured and massed in those occasioned by a corrosive poison.

In the case of mercuric poisoning reported here the patient lived forty-one days, so that many of the features ordinarily seen in these cases, while present during the first week or more, were not sources of confusion at later periods in the disease.¹

CASE REPORT

History.—Mrs. A. R., aged 26, was brought to New York Hospital, May 26, 1914. She stated that she had that morning (8:30) taken six tablets for a headache. Shortly after, discovering that the tablets were mercuric chlorid, she sent for a physician. About half an hour after taking the tablets the patient vomited a large amount of material. On admission to the hospital at 10:15 a.m. the patient did not appear acutely ill. There were no excoriated areas in the mouth, though the pharynx was considerably congested. Other than some sensitiveness in the epigastrium the admission examination was negative. Lavage water containing gastric contents gave positive tests for mercury. A catheterized specimen of urine contained considerable albumin, numerous granular casts, but no blood. This specimen was not tested for mercury, but the metal was detected a number of times in the urine subsequently.

Treatment and Course.—Treatment was at once begun by frequent lavage of the stomach and bowel and an endeavor made to increase the fluids taken by the patient, supplemented by the Murphy drip. During the first day the

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¹ The longest period of life recorded after swallowing poisonous amounts of mercuric chlorid is twenty-one days.
patient's mouth became sore, which interfered with the ingestion of anything. This symptom became worse and for two weeks there persisted an extreme degree of stomatitis and ptyalism which was an obstacle to the efforts made to spare the kidneys. The irritated condition of the gut was evidenced for ten days by frequent discharges, containing much mucus and blood. This state of affairs not only made the retention of the rectal tube painful, but seemed to decrease the absorption of water from the intestine.

There was fever up to 101 F. and a leukocytosis of 20,000 during the first week, after which both temperature and leukocyte counts were normal; the systolic blood-pressure varied between 100 and 140 mm. of mercury during the whole course of the sickness.

Due to the conditions mentioned the amount of water taken by mouth and absorbed from the intestine was not as large as desired; on May 30, 900 c.c. were retained and on June 4, the tenth day of the sickness, there were noted convulsive tremors involving all the muscles. Later there was a mild general convulsion and the patient was in a stuporous state the remainder of the day. This was repeated June 7, the thirteenth day of the illness, with more severity, although the urine volume had risen to 2,000 c.c. A state of semi-coma persisted for two days. As the patient became more conscious it was evident that there existed a mild toxic psychosis. On the whole, the general condition was regarded as good during the following two weeks, and a favorable prognosis was considered because of the improving state of renal activity. Vomiting had been a troublesome symptom from the start, but during the latter part of June this symptom came more into prominence, until scarcely anything was retained in the stomach. Any fluid or food was rejected. The patient said, however, that she felt quite comfortable. Hypodermoclysis was carried on during this period at intervals. On July 2, 8 liters of saline solution were thus given. Glucose was given by enema but evidently not absorbed.

The patient's mental condition gradually became more cloudy and stuporous. For twenty-four hours before death the respiratory movements were of quite characteristic air-hunger type. The patient died on the forty-first day after taking the mercury.

Retinal examinations were made on several occasions but no abnormality noted.

The Urine: The amount varied considerably, depending chiefly on fluid intake. The average was about a liter a day, with something over two liters as the high extreme. There was not evident at any time an inability to excrete water by the kidney. There was no edema and in the last week the response to hypodermoclysis was prompt and adequate. The specific gravity of the urine was generally low, rising above 1.015 on two days, and on several being below 1.005. After the first week the amount of albumin was slight and was precipitated almost completely by acetic acid in the cold (nucleo-albumin). The urine contained considerable pus from a cystitis, and usually erythrocytes; but casts of any sort were seldom noted after the first week of the sickness. Mercury was found in all specimens of urine examined until June 21, when the tests were negative.

No study of the chlorid or nitrogen metabolism could be carried out since the ingest was made uncertain by vomiting and the patient was incontinent of urine. Two twenty-four-hour collections of urine secured by means of a retention catheter were analyzed; the sodium chlorid was just under 3 grams and the nitrogen about 6 grams. While these results are probably too low, as some urine was doubtless lost, they do not in themselves suggest a plus balance. This is supported in respect to the chlorid by absence of detectable edema. Considerable interest attaches itself to renal tests in cases of this nature, as the condition presents the essential features of an experimental nephritis in a human subject. A number of phenolsulphonephthalein tests were made and in none was there recovered a detectable amount of the test-substance.
Non-protein nitrogen of the blood was estimated three times; on June 10 it was 225 mg., on the 15th, 209 mg., and on the 26th, 238 mg. Of the last figure, 71 per cent. (176 mg.) was urea.

This history presents some interesting departures from the common course of events in cases of this sort where the duration of life is brief. Anuria was at no time a symptom, and when the amount of urine fell below normal this was adequately accounted for by a low ingestion of fluids. When the water intake was forced there was apparently a normal response in output. In contrast again with the usual picture are the convulsive seizures, muscular twitchings and the toxic psychosis. The stupor and coma that accompanies anuria induced by mercury poison in common with anuria from other causes, is so uniform in its manifestations that Ascoli separated the syndrome from that of uremia under the caption of "urinary poisoning." The salient characteristics in this clinical picture are the progressive lassitude and somnolence, gradually deepening into coma as death approaches. These are the manifestations notable in dogs after the ligation of the renal arteries, and they describe also the nervous symptoms usually evident in cases poisoned with mercuric chlorid. Convulsions are seldom observed and only in the latter hours of life. Of those symptoms which are associated with uremia, there were observed in this case, besides the evidences of renal disease, epileptiform convulsions, muscular twitching and a psychosis of the usual toxic type. Whether this constitutes uremia must be left undetermined.

Further consideration must be based on an anatomical study.

The autopsy disclosed, besides an eroded condition of the lower bowel and degenerative changes in the liver, a remarkable nephritis which was studied with care from many sections. The following note is Dr. Elser's comment on the condition observed.

Examination of Kidneys.—Microscopic examination of the kidneys shows dilatation of the tubules of the cortex with extensive degeneration, necrosis and desquamation of the lining epithelium. Many of the convoluted tubules are more or less completely filled with necrotic material which, in places, is the seat of calcific deposits. Some of these masses show small colonies of bacteria. The interstitial structures are edematous and show, in places, small areas of round cell infiltration. Occasional masses consisting entirely of bacteria are found in the vessels and between the convoluted tubules. The fact that the surrounding structures reveal no evidences of reaction suggests that this represents in a large measure a post mortem development of bacteria. The glomeruli are remarkably free from pathologic changes. Slight dilata-

2. It is of interest that Brauer, in his experimental study of the effects induced in the nervous system through poisoning with mercury, mentions that the symptoms he observed might be explained as uremia. The effects he remarked were increased reflexes, ataxia, and paralyses with convulsions as a terminal phenomenon. The worst cases of poisoning showed no nerve changes. Deutsch. Ztschr. f. Nervenh., 1897, xii, 1.
tion of the capsule is seen in places and here and there the epithelial lining is swollen and separated from the subjacent structures. Sudan III preparations show only traces of fat in the epithelial cells of some of the convoluted tubules.

The lesions enumerated above are those usually encountered in the kidney in cases of bichlorid (mercuric chloride) poisoning.

The lesions noted are remarkably similar to those induced in animals by such poisons as uranium and tartaric acid, in that the damage done is borne chiefly by the tubular epithelium. In this case the evidence of injury was confined more strictly to one structural element — tubules — than in any sections showing experimentally induced renal lesions that have come to my observation. On this account the large non-protein nitrogen of the blood in this case of mercury poisoning is of an especial interest, recollecting that it is with uranium nephritis in animals that this phenomenon also becomes pronounced.

The other consideration that arrests attention is the absence of tangible evidence pointing toward chlorid and water retention. The data available does not permit of conclusion, although the chlorid excretion was as large as the estimated ingest. Edema was not manifest. Considerable water retention may occur, however, before edema can be demonstrated, except by weight, and a "dry" chlorid retention is well recognized. These reservations seem advisable in view of Heinecke's observation that disturbances of chlorid excretion are associated with those poisons which injure the renal tubules.3

It is not permissible to build deductions from this scanty material, but better, perhaps, to regard this case as a straw in the wind of evidence.

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3. Relevant to the discussion is a case of mild mercuric chloride poisoning reported by Monakow since the above was written. Except during the first days of illness, there was no evidence of retention of water or nitrogen. There was, however, chlorid retention and later a minus chlorin balance. Edema was manifest. The poisoning was slight, as the patient recovered without special flushing treatment. Deutsch. Arch. f. klin. Med., 1914, cxv, 227.