lar injection. Latterly she had been eating very little food. It seemed likely that these factors, which tend to decrease the chloride content of body fluids, would lead to retention of bromide. A clinical diagnosis of chronic bromism was confirmed by a serum bromide estimation of 175 mg. per 100 ml. (17 mEq).

The bromide mixture was discontinued on October 14, and sodium chloride, 1 g. daily, was given by mouth. The signs of congestive cardiac failure increased and the sodium chloride was discontinued six days later. On October 19 there was some improvement in speech, but she was still drowsy, querulous, and ataxic; tremor was still present. By October 26 she was more alert and taking an interest in herself and her surroundings. On November 2 the serum bromide level was 100 mg. per 100 ml. (9.7 mEq). At this time she was much improved, although still drowsy and unable to walk unaided. Five weeks after discontinuing the bromide mixture she had recovered and was transferred to a convalescent hospital. Her personality had reverted to normal; she was alert and co-operative, and no longer had tremor, ataxia, or slurring of speech. On December 2 the serum bromide level was 10 mg. per 100 ml. (0.97 mEq). Subsequently a mitral valvotomy was performed, resulting in improvement in exercise tolerance.

COMMENT

Several aspects of the diagnosis of chronic bromism mentioned by Barbour *et al.* (1936) can usefully be re-emphasized. Of their five cases, ataxia occurred in three, and tremor, speech difficulty, and depression in two. Memory impairment and hallucinations were common symptoms. None of the five had a skin rash. Patients with atherosclerosis, anaemia, impaired renal function, and organic heart disease are more susceptible to bromide intoxication. There is a wide individual variation in the level at which symptoms appear—for example, a patient suffering from one of the above conditions may develop symptoms of bromism with a serum bromide level of 150 mg. per 100 ml. (14.5 mEq), whereas a healthy young man is able to tolerate the same level with no symptoms at all.

Barbour *et al.* point out that bromide acts by replacing chloride in the body and that the rate of substitution depends on the quantity of bromide given, the fluid and electrolyte intake, and the renal efficiency of the patient. Therefore a patient on a low-fluid and low-chloride regime will develop a higher level of bromide in the serum more quickly than a patient on a normal diet receiving the same amount of bromide. Rosenheim (1951) and Holt and Litchfield (1951) point out the dangers of giving a low-salt diet to patients with hypertension receiving hexamethonium bromide. A high ratio of bromide to chloride in the body fluids will also develop in a patient taking bromide and little or no food (Nabarro, 1954).

Chronic bromism of slight or moderate degree is easily overlooked because of the insidious development of the clinical picture. Vague symptoms such as fatigue, depression, and memory impairment may be attributed to senility, mental disorder, cerebral atherosclerosis, or the cerebral anoxaemia of congestive heart failure. The dangers of giving bromides to patients taking a diet low in sodium chloride or undergoing treatment with mercurial diuretics cannot be too strongly emphasized.

I thank Dr. Ian Gordon for permission to publish this case report.

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Lactosuria following Burns

Baar and Bull (1952) reported lactosuria in severely burned patients treated with a local application of penicillin lactose powder. The following report of a similar case illustrates again the absorptive capacity of "raw" cutaneous areas and indicates the danger of applying toxic substances to such damaged surfaces. In this case, however, the absorbed substance was harmless.

CASE REPORT

A woman aged 56 was admitted with burns of the back, buttocks, and thighs—approximately 2% of the surface area of the body. Her general condition was found to be satisfactory and the burns were dressed. The following day routine ward-testing of the urine with Benedict's reagent showed the presence of a reducing substance. In addition, it was noticed that both Rothera's test and Gerhardt's (ferric chloride) reaction were positive.

The presence of glycosuria in the first 36 hours after burning is not an uncommon occurrence, but, in order to eliminate the possibility of a coincident diabetes mellitus, a glucose-tolerance test was undertaken, the blood-sugar method of Kingsley and Reinhold (1949), with tungstic acid filtrates, being used.

Glucose-tolerance Test

Specimen	Venous Blood Blood-sugar Level (mg./100 ml.)	Urine		
		Reducing Substance (Approx. % as Glucose)	Rothera	Gerhardt
Fasting	105	0.5	Positive	Positive
1 hour after 50 g. glucose	150	0.5	"	"
2 hours after 50 g. glucose	105	0.5	"	"

The test showed no impairment of glucose tolerance, so the condition was obviously not diabetes mellitus. The positive Gerhardt (ferric chloride) reaction resulted from the administration of tab. codein. co. The positive Rothera reaction was explained by the mild ketosis that might be expected from the patient's condition.

The presence of a reducing substance in approximately constant concentration in all the urine samples prompted investigation into the nature of this substance.

Paper chromatography of the urine showed the following: fasting, lactose; at 1 hour, lactose and glucose; at 2 hours, lactose. The solvent used was *n*-butanol 60%, acetic acid 15%, distilled water 25%; the spray reagent was 3:5 dinitrosalicylic acid.

The lactosuria was found to be associated with the use of lactose as a bland dressing for the burned areas. It was thought, at first, that the urine samples had been contaminated with lactose at the time of collection, but a catheter specimen of the urine again showed the presence of lactose. Lactose was absent from a specimen of urine obtained one week after cessation of the lactose dressings.

There is no doubt that the lactose had been absorbed from the burned areas and excreted by the kidney. I am unaware of any evidence suggesting that such a temporary lactosuria is at all harmful.

My thanks are due to Mr. D. H. Patey for permission to publish this case.

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