

Comment

Spinal headache is an unfortunate and not infrequent sequela of lumbar puncture. Unfortunately, treatment modalities range from conservative to invasive, with no middle ground. The collective experience of this clinic has found caffeine sodium benzoate to be a very effective treatment for this problem. The literature, however, seems to overlook this modality, and few studies have examined CSB use in any detail. Standard textbooks offer little more.

Sechzer and Abel³ reported on 104 patients with spinal headache and the use of intravenous CSB (0.5 g/2 mL) in 41 of those patients who did not respond to the usual conservative measures. In a double-blind demand method they found a highly significant difference ($P < 0.0001$) when compared with control. Seventy-five percent of those patients initially receiving CSB had relief of the headache. With a second injection, headache relief increased to 85 percent. In 70 percent the headache did not return. Other than mild central nervous system stimulation, no other side effects were reported.

The causal mechanism is felt to be lowered cerebral blood volume and decreased cerebral spinal

fluid pressure, brought about by increased vascular resistance and thus decreased cerebral blood inflow. In short, caffeine sodium benzoate seems to reverse the abnormal cerebral dynamics present in precipitating a spinal headache.³

Summary

Caffeine is often used in the treatment of headaches. Parenteral caffeine sodium benzoate is a single and safe modality for the treatment of spinal headache. Its application is easy and it is approved for this use.⁴ Headache usually responds after one injection, but occasionally a second or third can be used at four- to eight-hour intervals when necessary. Other than mild central nervous system stimulation, side effects are rare.

References

1. Dripps RD, Eckenhoff JE, Vandam LD: *Introduction to Anesthesia*, ed 5. Philadelphia, WB Saunders, 1977, pp 273-277
2. Albright G: *Anesthesia in Obstetrics—Maternal, Fetal and Neonatal Aspects*. Reading, Mass, Addison-Wesley, 1978, pp 205-207
3. Sechzer PH, Abel L: Post-spinal anesthesia headache treated with caffeine: Evaluation with demand method: Part I. *Curr Ther Res* 24:307, 1978
4. *Physicians' Desk Reference*, ed 34. Oradell, NJ, Medical Economics, 1980, p 1019

Laxative Abuse Causing Gastrointestinal Bleeding

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Laxative ingestion for the purpose of weight reduction is occasionally seen in clinical practice. Numerous metabolic abnormalities and bowel motility disorders can occur, but the etiology of

these problems may be difficult to ascertain because such patients do not always admit they use laxatives. Many of these patients have underlying psychological problems, of which depression is the most common.

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Case Report

A 24-year-old white woman was first seen in the emergency department at the University of Ari-

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zona Health Sciences Center complaining of neck pain resulting from a minor motor vehicle accident one week previously. She was noted to be pale; her hemoglobin level was 7.9 g/100 mL.

She had been obese for many years. Her weight was reported to be 286 lb four years previously, when because of her husband's dissatisfaction with her appearance, she began a diet consisting almost exclusively of soups and salads. She later began taking an over-the-counter laxative containing 97.2 mg of yellow phenolphthalein (Feen-a-mint) and noticed that the laxatives increased her weight loss. She began to rely on this medication as the mainstay of her weight reduction program. After careful questioning, it was estimated that she was consuming more than 150 tablets per week. Most of these tablets were taken during weekends in an effort to improve her appearance for her husband, who weighed her every Monday morning.

She reported no significant past medical history. She had never been pregnant; her menstrual periods occurred monthly and were not excessively heavy. She had not been constipated, and there was no history of hematemesis, ulcer disease, melena, or hematochezia, nor was there any family history of anemia.

On physical examination she appeared extremely pale. She was 67.75 in tall and weighed 209 lb. Her temperature was 36° C, respirations 16 per minute, pulse 80 beats per minute, and blood pressure 120/85 mmHg. There was no orthostatic blood pressure drop. The thyroid gland was not enlarged. The lungs were clear and a grade 1/6 systolic ejection murmur was heard at the left lower sternal border. Abdominal examination revealed no masses or organomegaly. Rectal examination revealed a markedly positive test for occult blood (Hemoccult). The remainder of the examination was unremarkable.

Laboratory findings included a hematocrit of 26.6 percent and hemoglobin 7.9 g/100 mL. The mean corpuscular volume was 64 μ^3 , the mean corpuscular hemoglobin was 19.6 $\mu\mu\text{g}$, and the mean corpuscular hemoglobin concentration was 29.8 percent. The reticulocyte count was 2.3. The serum iron was 6 $\mu\text{g}/100\text{ mL}$ and the total iron binding capacity was 497 $\mu\text{g}/100\text{ mL}$. The erythrocyte sedimentation rate was 19 mm/h. The serum folate was 5.7 mg/mL (normal, 4 to 20 mg/mL). The total protein was 7.9 g/100 mL and the albumin 4.4 g/100 mL. SGOT was 60 IU/L (normal, 4

to 20 IU/L), the alkaline phosphatase 52 IU/L (normal, 30 to 100 IU/L), and the total bilirubin 0.4 mg/100 mL. Serum electrolytes were within normal limits: sodium, 140 mEq/L; potassium, 3.8 mEq/L; chloride, 104 mEq/L; and bicarbonate, 27 mEq/L. The creatinine was 0.7 mg/100 mL and glucose 100 mg/100 mL.

A chest radiograph was unremarkable. A barium enema demonstrated normal colonic motility and well-visualized reflux into the terminal ileum; no abnormalities were seen. An upper gastrointestinal tract series with small bowel follow-through was also normal. Anoscopy revealed no hemorrhoids.

The patient was instructed to discontinue use of the laxative and was begun on ferrous sulfate, 300 mg three times per day. She received dietary counseling regarding a 1,200 kcal, high-fiber diet.

One month later, the hemoglobin was 10.7 g/100 mL, the hematocrit was 33.8 percent, and the mean corpuscular volume was 74 μ^3 . Stool tests for phenolphthalein and occult blood were both negative on numerous subsequent examinations. The patient had no difficulty with constipation; she reported daily, easily passed movements.

Further discussion revealed that there was a long history of marital difficulties, which were currently being made worse by her husband's threat to leave her if she did not lose weight. She had become significantly depressed, exhibiting sleep disturbance and episodes of spontaneous crying. After several weeks of counseling, she made the decision to separate from her husband. She moved out of state and was lost to further follow-up.

Discussion

Phenolphthalein is a stimulant or irritant cathartic similar in structure and activity to bisacodyl (Dulcolax). Although the precise mechanism of action is unknown, these drugs have a direct stimulatory effect on the bowel, which can be inhibited by the topical application of cocaine.¹ Phenolphthalein is relatively nontoxic, and acute ingestions of large doses have been tolerated without untoward effects.²

With long-term usage, however, adverse effects do occur. The most common of these is fluid and

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electrolyte disturbance as a result of chronic diarrhea causing excessive fecal excretion of sodium and potassium.³ Malabsorption syndromes frequently occur in patients who abuse laxatives, apparently as a result of pancreatic dysfunction. Steatorrhea, hypocalcemia,⁴ abnormal pancreatic function tests,⁵ osteomalacia,⁶ and protein-losing enteropathy⁷ have all been reported.

Anemia resulting from phenolphthalein use occurs infrequently; only one case has been reported in the literature.⁵ In that case, as in the present one, the evidence was suggestive that the anemia was due to rectal bleeding.

The negative barium studies in this patient tend to exclude structural lesions of the gastrointestinal tract. Although the possibility exists that a small, unidentified lesion could have been the source of the blood loss, it is unlikely that such a lesion would stop bleeding merely as a result of discontinuing the laxative.

It is also possible that the anemia was nutritional in nature rather than being due to blood loss. The patient's diet consisted only of soups and salads, which would not be expected to provide an adequate intake of iron. However, a dietary etiology would leave the bleeding unexplained, and its cessation after discontinuing the laxative strongly suggests that it was drug related.

The most serious adverse effect of chronic abuse of irritant cathartics is "cathartic colon," which is associated with chronic constipation, bloating, and lower abdominal pain resulting from marked dysfunction of intestinal motility. Characteristic radiographic changes are seen on barium enema examination in 10 to 30 percent of cases.⁸ In severe cases, the colon may become atonic, and surgical resection may be required.

Psychiatric disorders are common in patients who abuse laxatives. Depression occurs commonly, as illustrated in this case. Laxative abuse is also seen in anorexia nervosa, although these patients tend more commonly to attempt weight loss with diuretics.⁹ Ninety percent of patients who abuse laxatives are women, and not all will acknowledge that the problem exists. Many patients will conceal their laxative use from physicians, who undertake extensive and costly workups to evaluate presenting symptoms such as steatorrhea, hypokalemia, diarrhea, or abdominal pain.

A simple stool analysis can be done to detect phenolphthalein. A specimen of stool is placed in a test tube. It is alkalized with several drops of 0.1 N sodium hydroxide until a pink color appears. Confirmation of this positive test may be obtained by the addition of 2.0 N hydrochloric acid, which will result in the disappearance of the color. The same reaction occurs in vivo when a patient using phenolphthalein cathartics is given a soap suds enema; the stools are made alkaline by the enema and they turn red.¹

There is no evidence in the literature to suggest that there are any adverse effects from the abrupt discontinuation of phenolphthalein cathartics. This is well illustrated by the patient in this case, who reported normal bowel movements after cessation of laxative use. A reasonable program for the management of phenolphthalein laxative abuse would include the following:

1. The laxative must be discontinued.
2. A diet high in fiber and bulk (bran, fresh fruits and vegetables) should be initiated.
3. If constipation occurs, it may be treated by enema, bulk laxative such as psyllium hydrophilic mucilloid (Metamucil) or a stool softener (dioctyl sodium sulfosuccinate). There is no evidence that long-term use of bulk laxatives or stool softeners is associated with serious complications.
4. The emotional components of laxative abuse must be investigated, since underlying psychological problems requiring long-term care are common.

References

1. Goodman L, Gilman A (eds): *The Pharmacological Basis of Therapeutics*. New York, Macmillan, 1970, pp 1021-1022
2. Blatt ML, Steigman F: Phenolphthalein tolerance in childhood: An analysis of four cases. *J Pediatr* 19:344, 1952
3. Fordtran JS, Ingelfinger FJ: *Handbook of Physiology*. Washington, DC, American Physiological Society, 1968, pp 1457-1490
4. Rawson MD: Cathartic colon. *Lancet* 1:1121, 1966
5. Cummings JH, Sladen GE, James OF, et al: Laxative-induced diarrhea: A continuing clinical problem. *Br Med J* 1:537, 1974
6. Frame B, Gulang HL, Frost HM, Reynolds WA: Osteomalacia induced by laxative (phenolphthalein) injection. *Arch Intern Med* 128:794, 1971
7. Heizer WB: Protein losing gastroenteropathy and malabsorption associated with factitious diarrhea. *Ann Intern Med* 68:839, 1968
8. Todd IP: Cathartic colon: Surgical aspects. *Proc R Soc Med* 66:244, 1973
9. Cummings JH: Progress report—Laxative abuse. *Gut* 15:758, 1974