A monograph was published in 1956\textsuperscript{2} purporting to show that chronic low backache is in many instances the result of relaxation of the ligaments about the joints of the spine and pelvis. Treatment by the injection of an irritating solution consisting of the sodium salt of a vegetable-oil fatty acid, similar to that employed in the injection and treatment of varicose veins, was recommended. This treatment is alleged to produce proliferation of fibrous tissue and bone, thereby stabilizing the involved joints. Four thousand injections were reported, with “no unfavorable incident or sequelae.” Complete relief of symptoms was reported in 90 per cent of the patients treated.

There have been two recent reports of complications following this treatment. Schneider et al.\textsuperscript{3} reported the case of a 50-year-old woman with intermittent attacks of low-back pain and sciatica for 15 years. Severe pain and paraplegia developed at the time of injection of sclerosing solution into the lumbar region. The paraplegia improved, but severe hydrocephalus developed about 3 months later. At operation a “markedly adhesive arachnoiditis was found about the rim of the cisterna magna.” The patient expired a few hours after the procedure. At autopsy inflammatory changes were found at the base of the brain in the posterior fossa and the upper cervical spinal cord. There was perivascular infiltration about the major blood vessels and the choroid plexus. The brain was edematous. There was subependymal gliosis, with astroglial proliferation and nodular formation in the ventricles. The dura mater and leptomeninges in the lower spinal cord were fused together and frequently were indistinguishable. The authors felt that the chronic process of scarification was ascending, most pronounced around the lower segments of the spinal cord. They emphasized the extreme danger and inadvisability of injecting irritating solutions into areas where they may enter the subarachnoid space.

Keplinger and Bucy\textsuperscript{4} reported the case of a 53-year-old woman who had a transitory paraparesis following such an injection which subsided within 12 hours. She had recurrent headaches and stiff neck for about 2 weeks. About 7 weeks after the injection, a slowly progressive paraplegia developed, which rendered her unable to walk within 2 months. Five months after the injection, exploratory laminectomy showed severe arachnoiditis. Biopsy was reported to consist of dense collagenous fibrous tissue containing a few chronic inflammatory cells in the denser areas. The authors stated that the treatment of low-back pain by the injection of sclerosing solution is “inadvisable,” and that there is “no clear-cut evidence that this method of therapy is of any value.”

We have recently seen 3 patients with similar complications. In the first of these a mild paraparesis and communicating hydrocephalus developed. The hydrocephalus was controlled by ventriculojugular shunt. The second patient expired after a prolonged and unpleasant illness, with paraparesis and hydrocephalus caused by proliferative arachnoiditis. The third patient has had symptoms confined chiefly to the spinal cord.
CASE REPORTS

Case 1. E.F., a 59-year-old man, was seen in consultation at the request of Dr. Norman Rothermich at White Cross Hospital on Mar. 14, 1957. He had considered himself well except for some backache until Jan. 22, 1957, when he underwent injection of a sclerosing solution into his lumbar area. During the injection violent pain developed in his lower extremities with a sensation of numbness about the lower part of the trunk. He had an intense desire to urinate.

Headache and vomiting appeared later that day. In the succeeding weeks he became confused with marked deficit of memory and general dulling of mentality. He had intermittent visual hallucinosis with complete insight into the unreality of the peculiar objects that he was seeing. There were olfactory hallucinations and dysgeusia. He described symptoms suggestive of peduncular cerebellitis with hemorrhages and was readmitted to the hospital.

When first examined on March 14, 2 months after the injection, he was oriented as to person, but not as to place or time. He would become drowsy intermittently in the course of examination. He could flex the thighs slightly against gravity, but could not sit unassisted. There was good strength in the arms. Deep tendon reflexes were more active in the biceps on the right and the triceps on the left. Both knee jerks were absent. The ankle jerks were present, but more active on the right than on the left. There was bilateral Babinski’s sign.

Electroencephalography on Mar. 11, 1957 showed slowing of the basic patterns to 6–7 per sec. Leads anterior to the transaural plane reflected 2–4 per sec. waves of medium to moderate voltage, appearing in shifting fashion.

Pneumoencephalography was performed on Mar. 14, 1957 with 100 ml. of air. There was good filling of the entire ventricular system with virtually no filling of the subarachnoid spaces over the hemispheres and very poor, if any, filling of the basal cisterns except the cisterna pontis and cisterna magna. The ventricular system was dilated symmetrically throughout.

Spinal fluid contained 24 mg. per cent of protein, 119 mEq. per l. of chloride, 95 mg. per cent of glucose, and 831 fresh red blood cells. Colloidal gold curve was 11 100 0000. The Kahn test was nonreactive.

Following the air study he became worse. He was agitated intermittently and somnolent. When responsive, he was euphoric and out of control. There was gross nystagmus in all directions. Malnutrition became a problem and feeding jejunostomy at one time was considered seriously. He was treated with Dramamine and cortisone, and slowly improved until he was able to get about with the aid of a walker. He remained euphoric, incontinent, and confused, but was noted to be “quite presentable on first impression.”

On Apr. 17, 1957, he was sent home with a diagnosis of arachnoiditis, vascular insufficiency of brain stem, and encephalomyelitis, secondary to unknown toxin.

He was followed as an out-patient over the succeeding 11 weeks during which time there was some general improvement in his ability to walk. He remained curiously euphoric, intermittently confused, and had almost no recent memory. He was often incontinent of bladder and bowel. He vomited almost every morning. On July 8, 1957, he was noted to have bilateral papilledema with hemorrhages.

On examination he showed a picture similar to that noted on his discharge in April. The abdominal reflexes and the right knee jerk had returned, but again he was unable to sit unassisted and showed marked defect of memory and confusion. An electroencephalogram on July 15, 1957, was reported as grossly abnormal because of high-voltage, 1–2 per sec. slow waves over all areas, and diffuse 5–6 and 25–18 per sec. waves. Lumbar puncture released normal spinal fluid under a pressure of 210 mm. of fluid. On July 15, 1957, a ventriculojugular shunt was performed on the right side with a Holter valve.

On his 4th postoperative day his wife reported excitedly that he was “normal again.” His mental dullness and euphoria had disappeared completely. He seemed to be totally amnestic for all events from the 3rd or 4th day following the injection until the 3rd or 4th day following the ventriculojugular shunt.

Since that time his legs have continued to improve. He still walks with the aid of two canes because of severe proprioceptive deficit, but motor power is good. The Babinski’s responses have disappeared. Both knee jerks have returned and all deep tendon reflexes are now symmetrical. There is still occasional vertigo upon arising in the morning, but vomiting is rare. An electroencephalogram on Jan. 6, 1960, was reported as mildly abnormal because of the right temporal accentuation of diffuse 7–10 per sec. sharp-wave activity. As far as can be ascertained, his intellectual functioning is at a very high level, apparently not distinguishable from that prior to his illness. The Holter valve appears to be patent. The fundi are normal. His health otherwise is good.

Case 2. W.H., a 56-year-old man, had suffered from low-back pain without significant radiation into the legs for many years. On July 12, 1957, an
unknown substance, presumably a sclerosing solution, was injected into his lumbar region. He experienced immediate pain and nausea. While driving home there developed headache, urinary urgency, and incontinence. On the same evening a generalized convolution occurred, and he was admitted to The Akron City Hospital. He was seen by one of us (W.C.B.) on July 17, 1957. At this time he was able to walk and seemed to be improving generally. He was discharged on that date.

Over the succeeding weeks, he complained of pain in the legs and headache. He was readmitted to The Akron City Hospital because of progressive mental deterioration, confusion and paranoid ideation. There was gross loss of memory. He was nauseated and vomited frequently. His condition became steadily worse and he expired on Aug. 29, 1957.

At autopsy there was extensive chronic productive inflammation in the dura mater and arachnoid membranes of the spinal cord, with obliteration of the subdural and subarachnoid spaces, and moderate dilatation of the ventricles. Focal nonspecific degenerative changes were seen in sections of the spinal cord. The histological picture (Fig. 1) was that of a chronic lepto- and pachymeningitis. Proliferative changes were marked and the subdural space was obliterated completely in many areas. The leptomeninges were thickened with proliferated granulation tissue and increased vascularity. There were scattered small foci of active chronic inflammation consisting mainly of accumulation of fibroblasts, lymphocytes, and plasma cells. The vascular pathology was that of peri- and endarteritis, but vascular occlusions were not observed in the sections examined.

Case 3. J.E.R., a 40-year-old salesman, had had periodic backache with radiation of pain into the left groin for some 15 years. On Jan. 17, 1957,
he was subjected to the injection of a sclerosing solution into the lumbar region of the spine, presumably for purposes of "fibro-osseous proliferation." He recalled no great discomfort at the time of injection, but an hour or two later there was onset of sensations of "pins and needles" and burning in the lower part of his back. That afternoon, he had a very severe throbbing headache. Tingling developed in the left great toe, and he had a feeling that he had to urinate but could not. He was confined to bed for 3 weeks because he was quite uncomfortable in the upright position. Since that time there had been slow progression of his weakness, especially in the left leg. He felt that there had been some progress even in the 3 months prior to his examination on June 21, 1960. He continued to be troubled with a low-grade burning dysesthesia in the feet. He was able to continue to work because he drove from place to place and rarely was on his feet for more than 10 minutes at a time.

In March of 1960 he was examined at the New York Hospital by Dr. Fletcher McDowell and Dr. Harold Wolff. Multiple attempts at lumbar puncture failed because of inability to obtain spinal fluid. Myelography was performed by the cisternal route and normal spinal fluid was obtained. The subarachnoid space was distorted from the T9 to the T12 level in a manner considered consistent with adhesive arachnoiditis, and the spinal canal was completely blocked at T12.

When examined on June 21, 1960, he appeared to be in good health. He walked on a wide base, placing his feet firmly in a manner suggestive of the gait in tabes dorsalis. The Romberg sign was strongly positive. The lower extremities were moderately weak, especially the flexors of the hip. Deep tendon reflexes were hyperactive, more on the left than the right, with abortive ankle clonus on the right and sustained ankle clonus on the left. There was a weak Babinski’s response on the right and a strong one on the left. Vibratory sense was absent on the ankles and weak at the knees. There was no sense of position in the toes. Light touch in the lower extremity was appreciated as abnormal with poor localization. The sense of sharpness from pin prick was diminished but present in all areas. A band of rather marked superficial hypesthesia with hyperalgesia encircled his trunk from D9 through D10.

**COMMENT**

On the basis of these cases, and those reported by Schneider et al.⁶ and Keplinger and Bucy,⁴ it is clear that the injection of sclerosing solutions into the region of the spine is a highly dangerous procedure.

In the 5 cases reviewed here, there seems little doubt that the irritating substance gained access to the subarachnoid space in varying quantities. In each instance, pain and neurological deficit occurred early, partially or completely subsided, and then recurred after a latent period of several weeks. In the initial reaction pain is prominent. There is acute malfunction of the nervous system evidenced by paraparesis, dysesthesia, and in 2 of the 5 instances, cerebral disturbances (confusion, convulsions). Complete or partial resolution of deficit may be followed by symptoms of acute meningeal reaction, also transitory. As the proliferative reaction develops, a number of weeks later deficit of the spinal cord may reappear, with or without communicating hydrocephalus and recurrent mental and brain-stem symptoms.

The clinical pattern coincides closely with the phases of experimental arachnoiditis that have been demonstrated by several investigators.¹³⁵ Keplinger and Bucy emphasized the long latent period between initial insult and the appearance of symptoms caused by chronic proliferative arachnoiditis.

This process may be confined to the spinal canal, producing myelomalacia, or may extend to the basal meninges, producing hydrocephalus and signs of cerebral and brain-stem disturbance. It is worth noting, however, that the dementia and brain-stem malfunction are not necessarily proportional to the degree of ventricular dilatation.

Similar disproportion has been observed in older people with low-grade hydrocephalus caused by tumor, and may simply represent the reaction of the old brain to a slowly progressive hydrocephalus. On the other hand, the autopsy findings in Case 2 indicate that ischemic lesions of the neuraxis occur secondary to the arachnoiditis. These lesions may be responsible for some of the disturbances of consciousness, peduncular hallucinosis, vertigo and other symptoms. The chronic symptoms, then, can be attributed to four factors:

1. Communicating hydrocephalus caused by obliteration of the absorptive apparatus for cerebrospinal fluid.
COMPLICATIONS AFTER INJECTING SCLerosING AGENT

Plastic leptomeningitis, with direct pressure on the superficial structures of the spinal cord.

Compression of the nerve roots at their place of exit through the leptomeninges, the larger fibers suffering the most.

Arteritis with impairment of blood supply and venous obstruction with passive congestion, resulting in ischemic lesions within the neuraxis.

These changes are illustrated in Fig. 1. The tracts of the white matter show softening and demyelination. There is also rarefaction of the ganglion cells in the anterior horn (Fig. 1A). The distinct focal pattern of degeneration of white matter is shown in Fig. 1B. The areas of focal encephalomalacia that are of recent origin are filled with gutter cells; the older areas show loss of myelin, axonal degeneration in forms of Cajal’s bulbs and gliosis. The obliteration of the subarachnoid space is also seen in Fig. 1B. The degeneration of an anterior root caused by pressure on its passage through the leptomeninges is shown in Fig. 1C. The myelin sheaths and axis cylinders apparently are destroyed. Arteritis was found in meningeal vessels only, and was represented by adventitial and endothelial proliferation with some reduction of the lumen (Fig. 1D).

Treatment is difficult. We have used corticosteroids for their anti-inflammatory and antiproliferative effect. In one instance we were able to control the communicating hydrocephalus by a ventriculojugular shunt, and effect a satisfactory remission of cerebral symptoms.

SUMMARY

1. Three cases of serious neurologic complications following the inadvertent injection of a sclerosing solution into the subarachnoid space are presented.

2. The resultant lesion is a severe adhesive arachnoiditis which produces a progressive paraplegia, hydrocephalus, or both.

3. The hydrocephalus may be controlled by ventriculojugular shunt.

4. Attempts to produce “fibro-osseous proliferation” by injections of sclerosing solution about the spine are of dubious value and unquestionably are hazardous.

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REFERENCES


