Sclerotherapy for Baker’s Cyst

To the Editor:

Centeno and coworkers (Pain Physician 2008: 11: 257-262) brought to light a very important subject (1).

Historically, installation of irritating substances into a pathologic, fluid filled cavity has been known since Aurelius Cornelius Celsus (25 B.C.– 50 A.D.) described the injection of saltpeter (potassium nitrate), to cure hydrocele (2,3).

Contemporary work with sodium morrhuate (SM) began in 1930 after Higgins and Kittel (4) wrote an article about therapeutic applications of this agent. A year later in 1931 Poritt (5) described the treatment of chronic olecranon and pre-patellar bursitis. He drained the fluid from the sac and injected 5% of SM. In persistent cases, he injected a 5% phenol solution.

Subsequently, in 1933 Biegeleisen (6) published clinical and experimental research of SM in the United States. In 1936 he coined the term Sclerotherapy as a name for the injection clinic at the Stuyvesant Poly clinic in New York City, and very soon this term was adopted in the U.S. as well as abroad. Among the many applications for SM, he used it for bursitis in 5% strength, approximately 1 mL for each 4 – 5 mL of drained fluid. For ganglion cysts he preferred stronger solutions. He also introduced provocative sclerotherapy in chronic cases of “dry” bursitis injecting them with 1.25% or 2.5% of SM, converting them into an acute, fluid filled phase and then treated them as an acute stage.

There is a plethora of literature on experimental and clinical histologic findings on various sclerosing solutions in the treatment of varicose veins, hernias, and synovial joints but none describe the post-injection findings in a cyst or a ganglion.

There is however a case report from Ewell (7) describing intra-operative and histologic findings one month after 3 injections with quinine hydrochloride and urethane for hydrocele (this solution is allegedly less painful upon injection than SM. A small amount of dark amber fluid was in the sac, and the testicle and epididymis were found to be normal; some attached bands of organized fibrin were present. Microscopically; endothelium was intact and subserosa was thickened and infiltrated with organized fibrous tissue, which indicated that the solution did not obliterate the sac (7). So what has caused the therapeutic effect?

In 1937, Schultz (8), searching for a better way to treat painful subluxations of temporomandibular joint disorder, conducted animal experiments with intraarticular injections of several solutions including SM. Among those, Synasol (sodium psylliate — a 5% extract of psyllium seed oil produced by Searle Pharmaceutical and discontinued in 1960s) provided the best outcomes and therefore was chosen for clinical trials. All experimental joints demonstrated smooth and glistering cartilage surfaces and normal synovial membranes with capsules that were 5 –7 mm thicker in experimental animals versus control. Thus, there were no alterations in joint cavities but fibrosis was evident in the ligaments of injected joints. There were no gross changes in the ligaments other than thickening. A clinical study of 30 human subjects after biweekly injections of 0.25 to 0.5 mL Synasol demonstrated “entire patient satisfaction.” Schultz concluded that the principle of induced hypertrophy of the articular capsule by induced fibrosis may be applied to other joints capable of subluxations or recurrent dislocations. Injections restored normal joint function and the method was within the scope of treatment of a general practitioner (8).

Thus the therapeutic action of injectates was different in each condition. In hernias, the proliferation and subsequent regenerative/reparative response led to fibrotic closure of the defect. In hydroceles, hypertrophied sub-serous connective tissue reinforced the capillary walls of serous membrane and prevented further exudate formation (9-12). This latter mode of
Anatomy, Imaging, Treatment Options for Baker’s Cyst

To the Editor:

We read with interest the case report by Centeno et al (1) describing “Sclerotherapy of Baker’s Cyst with Imaging Confirmation of Resolution.” We wish to comment on aspects relating to the anatomy, imaging, and treatment options.

Adams (2) originally described the popliteal cyst in 1840 and in 1877 Baker (3) detailed it further as being caused by trapping of fluid in a bursa related to the semimembranosus tendon.

A Baker’s cyst is a synovial cyst that usually communicates with the knee joint by way of a slit-like opening, lined with synovium. Rausching and Lindgren (4) in their study suggested 2 mechanisms for cyst formation. The primary cyst has an unilateral valvular connection and the secondary cyst communicates freely with the knee joint and contains synovial fluid of normal viscosity. Intrinsic intraarticular disorders causing excessive joint effusion and cyst may serve as a protective mechanism by reducing destructive pressure in the joint space (5).

Centeno et al (1) describe a large Baker’s cyst that freely communicates with the knee joint based on MRI imaging. However the benefit of using MRI is the ability to use the axial plane images to establish positive identification of high signal intensity, at the fluid filled neck of the cyst that connects to the joint.