Chronology of separate techniques for endoscopic carpal tunnel release

To the Editor:
I read the article "Endoscopic Carpal Tunnel Release" (*JAOA* 1994;94:295-298) by Dr Payne and colleagues with interest. On page 295, the authors state that the "endoscopic management of carpal tunnel syndrome was first reported by Okutsu and coauthors in 1989. Refinement of the technique and development of specialized instruments was [sic] undertaken by Chow." This statement is incorrect. I think that Dr Payne and his coauthors need to understand that Dr Okutsu's technique differs completely from mine.

As noted in the reference section of this article, Dr Okutsu's article and my article were published in the same issue of *Arthroscopy*. In fact, our articles appear only 8 pages apart. Both of our techniques were developed at the same time, but using completely different instrumentation and different concepts. The aforementioned statement *should* read: "Endoscopic management of the carpal tunnel syndrome was first reported by Okutsu and Chow." Basically, we reported our results simultaneously.

James Chow, MD
Mt Vernon, Ill

Response

To the Editor:
My colleagues and I appreciate Dr Chow's writing to correct any misconceptions that may have been inferred by reading our article on endoscopic carpal tunnel release. It was not our intent to imply that Dr Chow's technique was an extension of work initially done by Dr Okutsu. It is well recognized that the two-portal technique of endoscopic carpal tunnel release that Dr Chow originally described was developed independently from and is distinctly different from that described by Dr Okutsu.

We thank Dr Chow for this added historical perspective, and we regret any confusion that may have been created by our review of the history of his technique.

John C. Payne, DO
Sandusky, Ohio

Somatic dysfunction revisited

To the Editor:
Gregory A. Dott, DO, and his colleagues are to be commended on their research and discussion of the relationship between sacral base unlevelness and iliac crest measurements as they relate to radiographic screening of short leg syndrome and the prescription of lift therapy (*JAOA* 1994;94:383-390). Their conclusions correlate with my clinical experience and my thoughts concerning low-back dysfunction. The study demonstrates an unreliable correlation between these two measurements and reflects the independence of the ilium and the sacrum as each responds to acute and chronic stress.

Historically, researchers have searched for such a correlation; therefore, the current research is worthwhile. However, if *somatic dysfunction* is defined as restriction of motion and *leg* (as it applies to short leg syndrome) is defined as everything from the calcaneus to the sacral base during a standing examination, why should we still try to define the motion restriction with a graphic of the hard structures in one plane? Furthermore, without defining the restriction, why would we add further restriction in the form of a heel lift? Too many joints and too many planes of soft tissue stress—including proprioceptor accommodation and fibrosis—are intervening for further motion restriction. Such a situation would leave too much for the operator to explore and to resolve manually.

Besides the multiple limb joints, which can confuse the diagnostic picture, the transverse planes of fascia are found above the femoral head. With their visceral contents, these planes have too many soft tissue stressors, including proprioceptive accommodation and fibrosis. As a result, the operator has too much to explore and to resolve manually.

Our credibility depends on our ability to describe what we do and to predict results. In our quest to do just that, we have, in the past, fallen into a reductionist approach. Such is the case in searching for radiographic definitions and evidence of dysfunction. Rather, I am convinced that we as osteopathic physicians need to proceed with the most sensitive "instrument" at our disposal—our hands, guided by our mind's comprehension of functional anatomy.

Dysfunction is more than the disruption of the relationship

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between solid structural components; it is a 360-degree alteration of freedom of motion around an anatomic locus. This point or portion of a fascial plane can be attributed first to a proprioceptor response to inflammation, which is the result of some injury. It can also be attributed to fibrosis due to stagnation of unresolvable tissue congestion. This congestion develops when free lymphatic and venous drainage is disrupted. In other words, motion restriction is a dynamic function of multiple physiologic responses to injury.

A summation of force vectors are translated along fascial planes and act on particular bony structures, such as the sacrum. However, this relationship fits into patterns and syndromes.

Regarding the question of functional short leg syndrome, clinically, I find a predominant pattern with a small variant in each dysfunctional leg, just as Dr Dott has documented. The dominant pattern is for the patient lying in a supine position to demonstrate iliac elevation and anterior superior iliac spine away from the short leg, with the sacrum following as a unit. In a small number of cases, an independent torsion or extension of the sacrum exists. The frequency of such varies, and the symptoms do not suggest the difference. It is only with a palpatory examination and by making a sequential assessment and treatment of the interrelating areas that the nature of the dysfunction is elucidated and the resolution is achieved.

If osteopathic manipulative treatment could be reduced to tests and simple algorithms, not only would osteopathic physicians be

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out of work, but osteopathy as a science and as an art would never have been established.

Zachary Comeaux, DO
Laurelville, Ohio

Response

To the Editor:

We appreciate Dr Comeaux's confidence in our work and findings. We are happy to see our findings correlate with his clinical observations. The purpose of our article was to identify potential problems with palpatory examinations for short leg syndrome. The stimulus came from the treatment of patients who had previously received inappropriate heel-lift therapy from paraprofessionals.

We documented in our article the inadequate method of using iliac crest heights (which are reportedly the best palpatory method of evaluating short leg syndrome) when defining and measuring anatomic short leg syndrome in patients with less than 0.5 inch of unlevelness. X-ray film evaluation has been pioneered to be a reliable measure of postural imbalance. Using this scientific method, we evaluated one aspect of a complex medical syndrome. Our study defines a method of reliable measurements for those physicians who wish to help their patients compensate for short leg syndrome through heel-lift prescription.

However, not all instances of leg length discrepancy produce somatic dysfunction, because the body can compensate. The definition of somatic dysfunction is not limited to restriction of motion. Restriction of motion is but one of four characteristics of somatic dysfunction. The other three are tissue texture change; asymmetry of motion and relative position; and tenderness. *Somatic dysfunction* is defined as the impaired or altered function of related components of the somatic system, which are the skeletal, arthrodial, and myofascial structures, as well as the related vascular, lymphatic, and neural elements.

Our article does not address common compensatory patterns. However Gordon J. Zinc, DO, addressed these patterns in the 1960s.[1] Drs Irvin and Kuchera[2] have documented the need to consider the sagittal plane when treating postural problems. Our article in no way intentionally or unintentionally was meant to contradict these earlier findings. As we are asked to produce more data that support our way of examining our patients, we must balance our traditional way of reporting our findings with the currently accepted scientific methods of investigation.

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