Analysis

A Review of King HH and Lay EM, "Osteopathy in the Cranial Field," in *Foundations for Osteopathic Medicine*, 2nd ed.

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Cranial osteopathy (aka craniosacral therapy) is a palpation/manipulation-based medical practice conceived in the early 20th century. Supplementing our recent critique,[1] we review here the chapter titled "Osteopathy in the Cranial Field," by King and Lay[2] in the new *Foundations for Osteopathic Medicine* (2nd ed.). Many consider this volume the American Osteopathic Association–sanctioned textbook for osteopathic medicine. Much evidence presented in this chapter was either overinterpreted or misinterpreted, and pertinent literature was overlooked. In the interest of the osteopathic profession and our students—and coincident with the best scientific evidence—this chapter should be revised extensively or deleted altogether.

Cranial osteopathy and craniosacral therapy are variants of a treatment originating with Sutherland[3] and used by physicians (primarily osteopathic), physical therapists, occupational therapists, chiropractors, dentists, and others. It "has been a part of standard training . . . in all osteopathic medical schools."[2(p986)] Perhaps as many as 60 000 practitioners have been trained through a facility in Florida[4] and as many as 37–49% of chiropractors may use related techniques on some of their patients.[5]

The biological model usually called upon to explain and justify the various diagnostic and therapeutic ministrations performed by practitioners is the "primary respiratory mechanism."[3] This model, as endorsed by the American osteopathic community,[2] includes the following elements:

1. inherent rhythmic motility (the ability to move spontaneously, without external influence) of the brain and spinal cord,
2. rhythmic fluctuation of cerebrospinal fluid (independent of cardiac and respiratory influences),
3. articular mobility of cranial bones,
4. mobility of intracranial and intraspinal dural membranes, and
5. mobility of the sacrum between the ilia.

According to the model, intrinsic rhythmic movements of the brain (independent of respiratory and cardiovascular rhythms) cause pulsatile movements of cerebrospinal fluid and specific relational changes among dural membranes, cranial bones, and the sacrum. Practitioners believe that, through palpation alone, they are able to evaluate and modify numerous parameters of this system to a patient's health advantage. Unfortunately, the primary respiratory mechanism is invalid, interexaminer reliability is approximately zero, and there is no scientific evidence of clinical efficacy.[1]

Recently, the second edition of *Foundations for Osteopathic Medicine*[6] was published under the auspices of the American Osteopathic Association (AOA). This is, in effect, the AOA-sanctioned textbook for osteopathic medicine and an important source for the profession's own views on all topics osteopathic. Therefore, revisions

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to the chapter on cranial osteopathy[2] should have been more extensive. Many arguments advanced by King and Lay in support of their position are common to writings of cranial practitioners, are evidentially baseless, and should be abandoned. The following are only the more fundamental points at issue.

Inherent Motility of Brain and Spinal Cord (p. 988)

According to Sutherland's model, the motive force for the primary respiratory mechanism is the central nervous system (CNS); that is, the brain and spinal cord are thought to be motile (capable of self-generated motion). In the first of 2 paragraphs intended to corroborate this assertion, King and Lay began with: ". . . much research has confirmed the inherent motility of the brain and spinal cord." They then related findings from 4 groups of researchers,[7–10] all reporting brain movement but none demonstrating or mentioning brain motility. All explicitly concluded that brain motion was a secondary product of cardiovascular forces. In other words, the brain moves, but only consequent to the intracranial arterial pulse and intracranial changes in venous pressures coincident with breathing. We are aware of no data suggesting independent CNS motility in humans or any other mammal. In fact, to our knowledge, this notion has never arisen outside the community of cranial practitioners.

In the second paragraph, King and Lay drew indirect support for CNS motility from the fact that glial cells—support cells of the CNS—"contain actin and myosin, which are capable of contractile motility." That is, glial cells are motile. Actually, all mammalian cells contain actin and myosin filaments,[11(pp62,65)] and many mammalian cells are motile. Motility is a fundamental characteristic of life. However, none of this suggests that brains, as organs, might be capable of motility. Neither neurons nor glial cells contain the dense arrays of actin and myosin filaments or intercellular structures required for significant force generation and shortening. This element of the primary respiratory mechanism is not supportable.

Articular Mobility of Cranial Bones (p. 989–990)

King and Lay claimed that "cranial sutures are constructed in such a way to allow for motion" and implied that palpable cranial interbone movements are common in humans of all ages. However, ages at ossification for human cranial sutures render this belief untenable.[12–15]

They began by quoting Pritchard et al., work prominent in practitioners' claims of lifelong sutural patency in humans: ". . . in man and most laboratory animals sutures may never completely close."[16(p84)] Although practitioners frequently quote this observation, rarely do they mention that Pritchard et al. attributed it to Bolk. Examination of Bolk shows that he reached conclusions similar to those of others.[12–15] For example, in most humans, vault sutures have begun to ossify by about age 30. That is, most adult calvarial sutures are partly or completely ossified and Pritchard et al. did not suggest otherwise.

King and Lay also repeated frequently cited claims drawn from the work of Retzlaff and colleagues.[18–20] Quoting Retzlaff et al.,[20] they stated: "Examination of the parieto-parietal and parietotemporal cranial sutures obtained by autopsy from 17 human cadavers with age range of 7 to 78 years shows that these sutures remain as clearly identifiable structures even in the oldest samples." By 1980 Retzlaff had examined 4 additional specimens in that age range and concluded that none of the 21 "show[ed] evidence of sutural fusion due to ossification."[19] Neither of these abstracts provided information regarding ages at death, how the sample was drawn, or other essential details. Furthermore, none of the more comprehensive examinations of ages of sutural ossification in humans has corroborated Retzlaff's findings.[12–15] Studying hundreds of skulls from around the world, these other researchers have established early ossification times for some calvarial sutures, including the parieto-parietal (sagittal). Retzlaff's reports—based on a small, ill-defined sample—have not been replicated and cannot be extended generally to humans.

Some research[18,19] apparently has demonstrated that cranial sutures, when present, may contain vessels and nerves.[2(p989)] King and Lay implied that this was evidence of functional mobility or even long-term sutural patency in humans. Regarding the latter, the finding that sutures, when present, may contain vascular and neural elements is irrelevant to considerations of long-term patency. Likewise, it suggests nothing about long-term mobility because, in many adult humans, many vault sutures no longer exist.[12–15]

King and Lay cited 4 reports purporting to demonstrate movement between left and right parietal bones in monkeys and cats.[2(p989)] However, that slight movement might occur at sagittal or other sutures in felines or primates, including humans, is irrelevant to the case for ubiquitous, long-term cranial interbone mobility. Adult humans often do not have sagittal, coronal, or lambdoidal sutures. Movement cannot occur at these locations because the sutures have been replaced by bone. The same observation

renders findings cited by King and Lay, that slight movement may occur at specific sutures in humans,[21,22] irrelevant in the context of cranial practitioners' general claims. Moskalenko et al., observed that deformation of bone itself (e.g., in locations where sutures have ossified) would "demand application of considerable effort" and is therefore "hardly possible."[21,23] Movement is conceivable only at "flexible regions in places of cranial bone junctions [sutures]."[21] In other words: no sutures, no movement.

Under "Mechanics of Physiologic Motion," King and Lay cited *Gray's Anatomy*[24] in support of their assertion that the "key articulation at the sphenobasilar symphysis . . . in the base of the skull . . . is a cartilaginous union up to the age of 25 years. . . ."[2(p990)] More recent and comprehensive research has been done on large samples of living tissue (by CT scan) and nonembalmed cadaveric tissue. It has shown that these 2 bones almost always undergo complete bony fusion at their bases between the ages of 12 and 19.[25–28] According to King and Lay (still apparently referencing *Gray's Anatomy*), even after ossification, this bony union "has the resiliency of cancellous bone." In the context of Sutherland's model, the implication that movement occurs here after ossification suggests that the heavily mineralized matrix of 2 cm or more of bone can be palpably deformed by minute forces inside the cranium. There is no scientific support for this idea.

Although movement between the bases of the sphenoid and occipital bones "is an essential part of Sutherland's functioning model [the primary respiratory mechanism],"[29] and although King and Lay and others[1] have claimed that movement is possible here throughout life, this view does not withstand scientific scrutiny. More broadly, palpable interbone movements required by Sutherland's mechanism cannot occur in most adults, so this element of the cranial mechanism is invalid for a large percentage of humans.

Involuntary Mobility of Sacrum Between Ilia (p. 987)

King and Lay assert or imply that inherent rhythmic motility of the CNS (element 1 of the cranial mechanism) produces movement of the sacrum (element 5), synchronous with cranial movements (element 3), via dural attachments through the vertebral canal (element 4). Perhaps little need be observed here except that, when cranial rhythms were claimed to be palpated at the cranium and sacrum simultaneously, there was no indication of synchronicity.[30–32] In fact, perceived rates in the two locations often were negatively correlated.[30,31]

The Cranial Rhythm and Traube-Hering Waves (p. 990)

Nelson et al. reported that cranial rhythms palpated by one practitioner were temporally coincident with subjects' Traube–Hering/Mayer (THM) waves.[33] Although King and Lay provided only an outline sketch of this work, this concept will be addressed here.

THM waves are pulselike microvariations in blood pressure. Perhaps what some cranial practitioners perceive as a cranial rhythm is, or is influenced by, TH or M waves. However, it is unlikely that THM waves manifested in intracranial arteries could produce palpable interbone and dural movements. Such an assertion is as inconsistent with well-established physiological knowledge as the suggestion that rhythmic secretion of CSF from choroid plexuses in the cerebral ventricles could be the source of numerous features of the primary respiratory mechanism.[29] If it should develop that THM waves or something like them contribute to what is perceived as the cranial rhythm, then it would have to be THM waves palpated in the microvasculature of tissues outside of the skull, and all elements of Sutherland's elaborate mechanism would be superfluous. Whatever the cranial rhythm represents, numerous independent research groups have shown that it cannot be reliably measured. In fact, in tests of interexaminer reliability, measured rates seem to be a property of practitioners, not subjects.[1] This suggests that, if the cranial rhythm exists at all, many examiners have been perceiving something other than subjects' THM waves.

Finally, although it is conceivable that a TH or M wave could be a palpable subcomponent of an arterial pulse, there is no evidence that the rate, amplitude, or other parameters of such waves are related to health. They would best be considered epiphenomena associated with respiratory and cardiac rhythms that are themselves unlikely to influence health. Even if a direct relationship between health and qualities of THM waves were assumed, there is no evidence or reason to believe that they could be modified, through palpation, to a patient's advantage. King and Lay implied that further study of the relationship between the cranial rhythm and Traube-Hering and Mayer phenomena may one day legitimize cranial osteopathy, and this seems unlikely.

Diagnostic Reliability

Conspicuous in its absence from King and Lay's summary was any mention of interexaminer diagnostic reliability. Work performed by numerous independent groups has shown that interexaminer reliability associated with cra-

nial osteopathy is approximately zero.[1,32] These reports give no evidence that any 2 practitioners palpate the same phenomena or even that the perceived phenomena exist.

Clinical Experience

King and Lay's description of cranial osteopathy made it apparent that cranial diagnostic and treatment procedures are based on assumptions explicit or implicit in Sutherland's—or a closely related—mechanistic model. Because this model is unsupportable,[1] there is no reason to believe that clinical ministrations could make biomedical sense. Therefore, if a real supraplacebo effect on patient health followed cranial manipulation, it could only be by coincidence. This makes King and Lay's concluding proclamation also untenable: ". . . [M]ore than 50 years of clinical experience has indicated that the use of [cranial osteopathy] has given relief to many patients in whom no other treatment was effective."[2(p1000)]

A clinical encounter can be an empty experiential slate upon which both patients and practitioners may paint a picture of clinical success, even when the method is ineffective. Most maladies improve without treatment, placebo effects and regression to the mean may lead to improvements not directly caused by the treatment, and subjective validation may lead to imagined improvements where none exists.[34,35] Before King and Lay can justify claims of clinical effectiveness, clinical trials controlled to exclude these and other biasing influences must be shown to lead to replicable, positive outcomes.

Conclusions

Over time, science-based disciplines expand their bases of understanding and utility. Cranial osteopathy has not done so. Its advocates still proffer: (1) the same biologically untenable mechanism proposed by Sutherland 65 years ago, (2) no indication of diagnostic reliability, and (3) no properly controlled research showing efficacy. After many years, practitioners of cranial osteopathy, including King and Lay, have provided little evidential support for their many claims. These facts should lead to an extensive revision of this chapter or to its removal from the next edition of the *Foundations for Osteopathic Medicine*.

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