Louisa Burns, DO

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Louisa Burns (1868-1958) graduated as a Doctor of Osteopathy in 1903 at the age of 35. She went on to take a Masters Degree in science. For the next fifty years Burns was the dominant figure in Osteopathic research. The centre of her life's work was a study of the pathological changes that follow and accompany "Osteopathic Lesions." She studied animal models, that is, she studied animals, in her case rabbits, in which "Osteopathic Lesions" had been experimentally induced. Always, however, she attempted to correlate her work to clinical observations, to give it practical meaning. The breadth of her interest was wide. She was quite prolific. A woman, she gave emphasis to gynecological and obstetrical considerations. The depth of her academic, clinical, and educational commitment may be illustrated in the following description of a procedure not dissimilar to our *motion palpation* given in an article on "The Teaching of Osteopathic Skill" in 1946.[1]

. . . move the vertebrae and the ribs and note any limitation of motion, the speed of return to the normal relation after the release of pressure, and the bilateral symmetry of the spinal tissues and the vertebrae.

By so palpating, layer by layer, and by devoting constant attention to the information which is derived from palpation, a student can gain a very exact picture of the lesion and its surrounding tissue.

Burns' work was thorough and often insightful. So large is the body of work that it is difficult to encompass in a short review. Let me site a few examples:

Burns' observations of muscle texture are fascinating. Following a lesion she notes that –

. . . Muscular contraction usually subsides, but not always, until only the deep spinal muscles adjoining the lesion are palpably contracted . . .

Palpation of the deep spinal muscles around a lesion discloses several perplexing findings. A day or a few days after a lesion has been produced, the muscles are palpably uneven in texture. Small areas of contraction seem to be present in localized areas, but this contraction is not palpably identical with that of muscles normally stimulated. When the contracted areas are large enough to be outlined by palpation, they seem long and slender as if small isolated groups of fibers were affected. . . .

The lesion affects muscles in one of two ways; the muscle may be abnormally relaxed, with less than normal tone, apparently because it lacks some normal stimulus. In other cases the muscle is abnormally contracted in an atypical manner. Areas within the muscle substance may be palpably firmer, as if contracted, while other areas seem relaxed and quite often even more soft than ordinarily relaxed muscle tissue. This quality cannot be explained at this time (1947).† It is represented in microscopical sections by alternating areas of muscle which appear contracted, and those which appear relaxed. In both areas many fibres are found in which the cross striations are dim or irregular.[2]

The parallel between this description of muscle and Travell's discussion of myofascial trigger points is striking!

† nor can they be explained today.

Burns' description of changes in the joints themselves is noteworthy on account of the scant attention this crucial area has received. Burns found an initial increase in synovial fluid (for several weeks) followed by a deminision with changes in quality. She also observed that, "The capsular ligament thickens very slowly. Thickening is most marked on the side which is slightly shortened by the abnormal position of the bone."[2]

The following observation on joint afferentation is particularly interesting in the context of Wykes later work in this area:

The immediate effect produced by upper thoracic lesions appears to be due to disturbance of the sensory impulses reaching the corresponding spinal centres from the joint surfaces and other tissues whose relations are disturbed by the lesion. We found that direct stimulation of the joint surfaces is followed by at least temporary constriction of the meningeal and cerebral vessels, and it seems evident that a sudden change in the structural relations might have the same effect.[2]

With regard to the quality of Burns' work, Cole, in a 1970 critical review of her life and work, reproduced a diagram from a 1907 paper of Burns showing spinal sympathetic pathways which could scarcely be improved upon today so fine and accurate is the detail.[3]

We often hear call for the publication of case reports and analysis of clinical data with respect to SMT. There is great excitement when reports correlating "spinal lesions" with clinical findings are announced. Louisa Burns published such data across five decades. A clinical analysis of cardiac disorders published in 1953 draws on a population of over 12,000.[4]

Perhaps because she was an "Osteopathic" researcher, Burns' work has been questioned and maligned. Gevitz in his book "The D.O.s: Osteopathic Medicine in America"[5] does an acute hatchet job on Burns inventing abuse by misquoting his sources. Cole, in the review cited above[3], is kinder. He writes that, "Her work has been criticized – often without justification when it has been taken out of historical context. She has been faulted for lack of proper control, although she was adamant in maintaining proper controls. She has been criticized for using nonscientific methods when she was contributing to the development of such methods. She was called repetitious, but today repetition is considered necessary for substantiation of a theory by experimental methods."

One area in which she can be faulted is in "presentation." Some of her reports did not present a proper description of material and methods, and much of her work lacks proper referencing. Notwithstanding this problem we should realize that today's standardization of reportage is a convention and convenience – in so far as it has become a dogmatic ritual it runs the risk of being a straight-jacket. Burns has too much to offer us for us to just ignore her as primitive or eccentric.

For fifty years Burns studied the effects of spino-articular lesions on tissue. She found that with very minor exceptions there was a single consistant pattern of response in all tissues, and this was an ischemic response.

Immediately following a lesion, a transient blanching occurs in those visci affected by the lesion (see below). "The color

then returns almost or quite normal; then paling again may appear." Some minutes later the viscus flushes becoming "brighter scarlet than normal." At this time microscopic examination reveals a distention of arteries and capillaries. Blood cells migrate into the surrounding tissue. A condition of congestion and edema develops and evolves.

In the subacute state congestion is the major feature. "The affected tissues remain slightly purplish in color, they become edematous in varying degree, and are palpably cooler than are control tissues."[2] Burns speaks of a condition of "petechial hemorrhages" as an important concomitant of the congestive reaction, and sees the organization and resolution of these minute hemorrhages as the genesis of fibrosis: "progressive fibrosis" being the final stage in the sequence of response. Note that all these stages are congestive and may be termed hyperemic, but nonetheless what we are looking at is an ischemic process.

In summary, tissue becomes hyperemic, congested and edemic, with petechial hemorrhages followed by coagulation and organization with fibrosis and ischemia, and this is a general pattern in all tissues. With some reservation about the "petechial hemorrhages" this pattern of response holds no surprise for the modern pathologist and appears to have been well observed.[6]

We should note that while much of the congestive ischemic response to lesions observed by Burns can be explained in terms of autonomic vasomotor reactions, sympathetic stimulation has a multitude of effects at other levels, and that these too may produce ischemic changes. Thus Raab notes that "Myocardial oxygen consumption is dominated by sympathetic nerve activity", that sustained sympathetic stimulation causes ischemic changes in the myocardium with "disseminated necrotic foci."[7] (Note also that corticosteroids potentiate these sympathetotoxic effects.)

Burns mapped the effects produced by lesions at different segments. She found a close correspondence in the pattern produced in experimental animals and the clinical patterns observed by herself and her colleagues. Interestingly she found a homogeneity in all upper cervical lesions (C0-C3). Her findings are listed below.

segmental level	*organs affected*
C0-C3	– all organ systems in the *head*
C0-C3	– all organ systems in the *head* e.g. pupils dilate – motor disturbances of the *heart* e.g. arrhythmias – motor disturbances to the *diaphragm* – motor disturbances of the *stomach* and increased gastric acidity – *behavioural* changes
C4-C6	– has effects on the *pulse* – shallow *breathing*, with occasional sighing or hiccough – rarely may duplicate effects of upper cervical or lower lesions
C7-T3 (T4,5)	– all organ systems of the *head* e.g. pupils dilated – *thyroid* – *esophagus, stomach, duodenum* – *arrhythmias*
T2-T5	– *heart* circulation – *lung* circulation
T3-T4	– arrhythmias, weak pulse, slow recovery after exertion
T3-T5	– *heart*, myocardial tissue changes – *gastric* mucosa – *bronchial* vessels (upper and mid-lung fields)
T6-T9,10	– decreased *blood pressure* – *gastric* and *intestinal* tissue, with hypochlorhydria, diarrhea – *splenomegaly* – edema and hyperemia in the *pancreas* (particularly T10) and including degeneration of the islets – gradual malnutrition
T10-L2	– tissue changes in the *uterus, ovaries, adrenals, kidneys, ileocecum* (no testicular hyperemia, but marked scrotal edema) – *sterility, abortions* – increased body weight due to water retention – abnormal contractions of psoas and lumbar musculature

Very little attention was directed to the lower lumbar region as lesions in this area proved difficult to produce experimentally. However, Burns noted that clinically lumbar and SI lesions are associated with problems in the kidneys, bladder, vagina, rectum (including hemorrhoids), ptoses of pelvic organs (sagging), lumbago, sciatica, weakness in the leg muscles, swelling in the lower limbs, and spasms of lumbar muscles and psoas. Reading her work one gains the impression that this mapping was carefully observed, and in so far as our own Chiropractic Meric equivalents correspond I think we may be proud.

There are two major criticisms that can be leveled against the work of Louisa Burns, the first being that her reportage does not conform to our later-day standards and format, and the second being that her work is now "classical" if not archaic. If all that is old and difficult to access were lost we would be the poorer. We should be aware that Burns existed and is a well from which we can all drink and gain sustenance.

Louisa Burns' work is a source of useful and pertinent information for the manipulative physician and researcher. I hope here that I may have reviewed several of the more interesting fragments.

References

1 Burns L. The teaching of osteopathic skill JAOA 1946; 46(4): 217–219.
2 Burns L. Pathogenesis of visceral disease following vertebral lesions, Chicago, The American Osteopathic Association, 1948.
3 Cole W.V. Louisa Burns Memorial Lecture, JAOA 1970; 69: 1005–1017.
4 Burns L. Incidence of certain etiologic factors in cardiac disorders JAOA 1953; 52(7): 369–372.
5 Gezitz N. The D.O.s: Osteopathic Medicine in America, Baltimore, John Hopkins University Press, 1982.
6 Resch A. Personal Communication, Dr. Resch is the neuropathologist at the Toronto Western Hospital and is associated with the University of Toronto.
7 Raab W. The nonvascular metabolic myocardial vulnerability factor in "Coronary Heart Disease," Am Heart J 1963; 66(5): 685–705.

Answers to Chiroquiz

1 FALSE
2 TRUE
3 TRUE
4 TRUE
5 TRUE
6 TRUE
7 TRUE
8 TRUE
9 TRUE
10 TRUE