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PERSPECTIVES

The Abdominal Brain and Enteric Nervous System

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ABSTRACT

Conventional medical treatment for neurologic disorders such as epilepsy, migraine, and autism focuses on the brain. Although standard medical treatment is often helpful, the underlying causes of these disorders are not well understood. Furthermore, some individuals respond poorly or not at all to regular medicine. Evidence is accumulating in the medical literature that the enteric nervous system (ENS)—that part of the nervous system associated with the alimentary canal—also plays a role in these disorders. Historically, the concept of an autonomous abdominal nervous system was advocated by Byron Robinson, Johannis Langley, and Edgar Cayce. The work of these three prominent historical figures is considered along with modern viewpoints on the abdominal nervous system. Complementary therapies that address the nervous system of the abdomen have potential as useful adjuncts to conventional treatment for certain neurologic disorders.

INTRODUCTION

It is evident from the historical and modern literature that the peripheral nervous system, and particularly that portion associated with the alimentary canal, is a prominent element in certain neurologic disorders associated with the cerebral brain. For example, abdominal epilepsy and abdominal migraine are well-established diagnostic entities in modern medicine in both children and adults (Babb and Eckman, 1972; Loar, 1979; Mitchell et al., 1983; Reimann, 1973; Santoro et al., 1990; Symon and Russell, 1986). Some researchers regard the presence of abdominal features in these illnesses as important and of possible etiologic

significance (Amery and Forget, 1989; Mavromichalis et al., 1995; Peppercorn and Herzog, 1989). Recently, autism has been added to the list of neurologic conditions with abdominal features (Horvath et al., 1998; Murch et al., 1998; Wakefield et al., 1998;).

A possible explanation of the relationship between abdominal symptoms and neurological syndromes can be found in the influence that the enteric nervous system (ENS) has on the central nervous system (CNS). The ENS is an extensive network of neurons widely dispersed throughout the gut that coordinate to regulate gastrointestinal events such as peristalsis, blood flow, secretion, and absorption (Costa and Brookes, 1994; Goyal and Hirano, 1996;

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Gershon et al., 1994). The ENS can influence the CNS both through nerve reflexes and the production of neuropeptides. It is estimated that 80% of vagal fibers are visceral afferents (Davenport, 1978). Recent work has also shown a vast overlap of neuropeptide activity in the gut and the brain (Pert et al., 1985). The ENS is an active area in physiologic research with more than 600 articles on MEDLINE since 1985.

The revival of interest in the ENS has strong historical roots. Nearly 100 years ago, American physician Byron Robinson, a medical doctor, did extensive research and writing culminating in his impressive work, *The Abdominal and Pelvic Brain* (Robinson, 1907). The premise of Robinson's book is that the abdominal viscera contain a vast and complex nervous system that influences, and to a great degree regulates, the vegetative process of the abdominal viscera.

Robinson was not alone in his fascination with the nervous system of the abdomen. At about the same time that Robinson was discovering the "abdominal brain," British physiologist Johannis Langley of Cambridge University recognized that, "the nervous system of the gut was capable of integrative functions independent of the central nervous system" (Gershon et al., 1994, p. 424). It is now known that the human gastrointestinal (GI) system, deprived of CNS innervation, is capable of coordinated digestion, mobility, secretion, and absorption (Davenport, 1978). Langley labeled the brain in the gut the enteric nervous system, the term now used for this system.

Edgar Cayce, who has been called the father of modern holistic medicine (Callan, 1979; Mein, 1989) was another strong advocate for the abdominal nervous system. Cayce believed that the idiopathic forms of certain neurologic syndromes (such as epilepsy and migraine) have an abdominal etiology. A wide variety of nonintrusive therapies were recommended by Cayce for the treatment of these syndromes.

The remainder of this article focuses on abdominal epilepsy, abdominal migraine, and autism with colonic features with the aim of understanding peripheral nervous system involvement in these disorders. Theoretical and clinical implications of the rediscovery of the abdominal nervous system are explored.

ABDOMINAL EPILEPSY

The association of abdominal symptoms with epilepsy has been recognized for many years. For example, "gastric and intestinal disturbances" were viewed as primary etiologic factors by medical doctors during the late 19th and early twentieth century (Dercum, 1912, p. 917). Osteopaths noted that, "... in cases where the exciting factor seems to be in the intestine and there is reverse peristalsis of the intestines, causing a reversion of the nerve current in the vagi, thorough rapid abdominal treatment will normalize peristalsis and aid in preventing an impending attack" (Hazzard, 1905, p. 275). Edgar Cayce insisted that idiopathic epilepsy is produced by lacteal duct adhesions along the right side of the abdomen that produce nerve reflexes to the brain. "From every condition that is of true [idiopathic] epileptic nature there will be found a cold spot or area between the lacteal duct and the caecum" (Cayce, 1934). The invention and clinical application of the electroencephalogram (EEG) during the 1920s shifted the focus of medical attention from the abdomen to the brain where, for the most part, it has remained to this day.

Another example of the abdominal connection in epilepsy is the aura that is common in certain types of epilepsy. For example, temporal lobe epileptic seizures frequently begin with an aura. In neurologic terms, an aura is actually a mild seizure that precedes the primary seizure. It can be thought of as a warning that a seizure is about to happen. Most often, auras manifest as an altered consciousness or peculiar sensation. "The most common aura is of vague gastric distress, ascending up into the chest" (Gordon, 1942, p. 610).

Modern medical science has rediscovered the abdominal connection in epilepsy. A few articles published in the medical journals during the 1960s called attention to the abdominal features in epilepsy (Berdichevskii, 1965; Takei and Nakajima, 1967, Juillard, 1967). Over the past 25 years, numerous researchers and clinicians have reported on various aspects of abdominal epilepsy (Agrawal et al., 1989; Babb and Eckman, 1972; Bondarenko et al., 1986; Douglas and White, 1971; Hotta and Fujimoto, 1973; Loar, 1979; Matsuo, 1984; Mitchell et al.,

1983; Moore, 1972; O'Donohoe, 1971; Peppercorn et al., 1978; Peppercorn and Herzog, 1989; Reimann, 1973; Singh and Kaur, 1988; Solana de Lope et al., 1994; Yingkun, 1980; Zarling, 1984).

Common clinical features of abdominal epilepsy include abdominal pain, nausea, bloating, and diarrhea with nervous system manifestations such as headache, confusion, and syncope (Peppercorn and Herzog, 1989). "Although its abdominal symptoms may be similar to those of the irritable bowel syndrome, it may be distinguished from the latter condition by the presence of altered consciousness during some of the attacks, a tendency toward tiredness after an attack, and by an abnormal EEG." (Zarling, 1984, p. 687). Mitchell et al. (1983) regard cyclic vomiting as a primary symptom of abdominal epilepsy manifesting as simple partial seizures. Although abdominal epilepsy is diagnosed most often in children, the research of Peppercorn and Herzog (1989) suggests that abdominal epilepsy may be much more common in adults than is generally recognized.

One of the primary problems in understanding abdominal epilepsy is clearly defining the relationship of the abdominal symptoms to the seizure activity in the brain. In other words, what is the pathophysiology of abdominal epilepsy? Is the essential pathology in certain areas of the brain that happen to be connected to the abdominal organs? Or is the primary pathology in the abdomen, which is conveyed through connecting nerve fibers to the brain, resulting in epileptic seizures? Peppercorn and Herzog (1989) noted both possibilities in their attempt to understand the cause of abdominal epilepsy:

The pathophysiology of abdominal epilepsy remains unclear. Temporal-lobe seizure activity usually arises in or involves the amygdala. It is not surprising, therefore, that patients who have seizures involving the temporal lobe have GI symptoms, since discharges arising in the amygdala can be transmitted to the gut via dense direct projections to the dorsal motor nucleus of the vagus. In addition, sympathetic pathways from the amygdala to

the GI tract can be activated via the hypothalamus.

On the other hand, it is not clear that the initial disturbance in abdominal epilepsy arises in the brain. There are direct sensory pathways from the bowel via the vagus nerve to the solitary nucleus of the medulla which is heavily connected to the amygdala. These can be activated during intestinal contractions (Peppercorn and Herzog, 1989, p. 1296).

The vagal link in epilepsy has also received attention with regard to a surgical procedure in which a pacemaker is implanted on the vagus nerve in the upper chest. Regular stimulation of the vagus has reduced or eliminated seizure activity in some treatment-resistant patients (Amar et al., 1998; Handforth et al., 1998; Lundgren et al., 1998). The therapeutic effect is thought to be produced by calming "hyperexcited" nerve cells and reverting brain activity to its normal patterns (Snively et al., 1998).

If stimulation of the peripheral nervous system, in this case the vagus, can reduce seizure activity in the brain, perhaps pathologic irritation of this or other peripheral nerves may also play a role in the etiology of certain forms of epilepsy. At this time, there is no definitive model of abdominal epilepsy that explains the association of brain seizures and abdominal symptoms. The clinical (therapeutic) implications of abdominal etiology in epilepsy are discussed below.

ABDOMINAL MIGRAINE

From a medical perspective, migraine presents as a complex systemic illness with various combinations of neurologic, GI and autonomic symptoms. Although the neurologic components are a primary focus in medical diagnosis and treatment, historical and contemporary viewpoints also attribute great significance to gastrointestinal features. "In the majority of migraine patients there is some fault in the gastro-duodenal-hepatic chain . . . " (Hare, 1912, p. 382). "Gastrointestinal disturbances including nausea, vomiting, abdominal

cramps, or diarrhea are almost universal." (Silberstein, 1995, p. 387).

Historical perspectives on syndromes such as migraine tend to take all of the symptoms into consideration in a more systemic interpretation of the illness. Thus, the significant GI aspects of migraine received much greater attention, both with regard to causation and treatment. The medical treatments prescribed for migraine in previous eras addressed the gastrointestinal features of the illness directly with a spectrum of relatively natural therapies intended to improve digestion, assimilation and elimination through the bowel.

Diet was frequently emphasized. For example, Spear (1916) cautioned that, "Heavy pastries, rich foods, and alcoholic drinks are best avoided." (Spear, 1916, p. 626). Therapies for intestinal cleansing were also commonly prescribed. "The bowels should be regulated, and under no condition should constipation be allowed to occur . . . if the lower bowel contains much fecal matter, a hot soapsuds enema should be given." (Spear, 1916, p. 626). Dercum (1912) relied on a mild saline laxative (such as Carlsbad salts) for intestinal cleansing, claiming that, "It is a not uncommon experience to find that a beginning migraine attack is frequently aborted by a saline, taken as soon as prodromal symptoms are noted." (Dercum, 1912, p. 906).

Modern medical science has acknowledged the rediscovery of the abdominal connection in migraine in various ways. The most obvious is the recognition of a diagnostic entity called "abdominal migraine" (Bentley et al., 1984; Mortimer and Good, 1990; Santoro et al., 1990; Symon and Russell, 1986). Abdominal migraine is diagnosed most often in children. For example Mavromichalis et al. (1995) studied a consecutive series of 31 children (median age 12 years) suffering from migraine. Endoscopic esophageal, gastric, and duodenal biopsy were used to determine whether the complaints were of GI origin. Of these 31 children, 13 (41.9%) showed esophagitis, 16 (51.6%) gastritis of corpus, 12 (38.7%) antral gastritis, and 27 (87.1%) duodenitis. Thus, 29 of 31 children studied had an underlying inflammatory lesion explaining their complaints. The researchers concluded, "Our findings provide further evidence that recurrent abdominal pain is an early expression of migraine and strongly support a causal link between recurrent abdominal pain and migraine." (Mavromichalis et al., 1995, p. 406).

The gastrointestinal connection in migraine has also been rediscovered with regard to food allergies (Bentley et al., 1984; Dalton, 1975; Grant, 1979; Hanington, 1980; Hughes et al., 1985; Mansfield, 1987, 1988; Mansfield et al., 1985; Monro et al., 1984; Peatfield, 1995; Peatfield et al., 1984; Trotsky, 1994; Vaughan, 1994; Wilson et al., 1980). The designation of dietary migraine is sometimes used in such cases (Dalessio, 1972). The conceptualization of migraine as a gastrointestinal allergic response has historical precedent:

The allergists have much to say which warrants careful evaluation as to the nature of the migraine episode as well as its etiology. They believe that fatigue, nervous and emotional factors produce changes in the motor activities of the GI system which result in duodenal stasis. This promotes the absorption of the allergens to which the patient reacts in his inherent pattern of migraine. They report that accurate allergy diets result in complete relief in 30 per cent of migraine patients and partial relief in 45 per cent (Gordon, 1942, p. 556).

One of the major problems in understanding the etiology and pathophysiology of migraine is how to conceptualize both the nervous and vascular aspects of the syndrome. Traditionally, migraine has been regarded as a "vascular" headache due the obvious abnormalities in circulation to the head (Agnoli and DeMarinis, 1985; Thomsen and Olesen, 1995). More recently, nervous system involvement has been emphasized, with particular emphasis on the trigeminal or fifth cranial nerve (Buzzi et al., 1995). An integration of these two models has culminated in a trigeminovascular theory that integrates nerve and circulatory processes (Buzzi and Moskowitz, 1992).

Interestingly, Edgar Cayce, an intuitive diagnostician practicing during the first half of the 20th century, also recognized the abdomi-

nal connection in migraine. Cayce claimed that the pathophysiology of idiopathic migraine involves allergic processes in the intestinal tract that trigger nerve reflexes to the triggeminal (fifth cranial nerve) resulting in migraine headaches. Consistent with abdominal etiology, Cayce's therapeutic recommendations focused on the intestinal tract and peripheral nervous system (i.e., diet, colonic irrigation, manipulative therapy). As an example of Cayce's unique views on the association between the abdominal brain and neurologic disease, the following excerpt describes the pathophysiology of migraine in a child:

As we find, while the body is in the developing stages, the sources of the conditions to which the body becomes allergic in the digestive system should be looked for—that deal with all migraine headaches.

So, this information might be used universally as to the sources of such, if it would be accepted. For, here we find such in its beginnings, and it is in the digestive system, causing—through a state of circulation—an inflammation in the connections of the intestinal tract through [the] blood and nerve supply . . . (Cayce, 1943).

Thus, according to Cayce, the general pattern of pathology in idiopathic migraine involves an allergic irritation in the intestines which is transmitted to the trigeminal (fifth cranial nerve) and also triggers an imbalance of circulation to the head. Cayce's model of idiopathic migraine takes into consideration both the nerve and vascular aspects of migraine.

Although Robinson did not directly address the topic of abdominal migraine, he did devote a significant portion of a chapter of his book to the abdominal and pelvic influence on the trigeminal nerve. (Robinson, 1907) Thus, in addition to the well-known visceral connections of the vagus (tenth cranial nerve), the abdominal connections of the trigeminal provide another possible route for nerve reflexes to the head in migraine.

The possibility that the abdominal features of migraine may have etiological and therapeutic implications suggests that further re-

search is needed. The degree to which abdominal migraine exists as a subgroup within the broader classification of migraine must be determined. Abdominal migraine in children is well established. The prevalence of abdominal migraine in adults is less well known. The efficacy of traditional clinical interventions for migraine that focus directly on the gastrointestinal system (i.e., diet and colonic irrigation) deserves further study.

AUTISM WITH INTESTINAL FEATURES

People with classical autism show three types of symptoms: impaired social interaction, problems with verbal and nonverbal communication and imagination, and unusual or severely limited activities and interests. Symptoms of autism usually appear during the first 3 years of childhood and continue throughout life. Although there is no cure, appropriate management may foster relatively normal development and reduce undesirable behaviors.

Recent medical research may add autism to the growing list of neurological illnesses with abdominal features. Wakefield et al. (1998) investigated a consecutive series of children with chronic enterocolitis and regressive developmental disorder. The 12 children (mean age 6 years) had a history of normal development followed by loss of acquired skills, including language. The children also had diarrhea and abdominal pain. Murch et al. (1998) report that 47 of 50 autistic children they studied showed significant bowel pathology. When subjected to colon cleansing, these children showed notable improvement in their autism symptoms. The researchers conclude, "We reemphasize the fact that there is a consistent pattern of gut inflammation in a high proportion of children within the broad autistic spectrum. Understanding the link between the bowel and the brain in autism may allow new insights into this devastating illness." (Murch et al., 1998, p.

Further evidence of intestinal involvement in autism has surfaced when a substance called secretin has been surprisingly effective in the treatment of autism for some children. After Victoria and Gary Beck successfully treated

their autistic child with secretin and triggered interest in this substance, Horvath et al. (1998) studied the therapeutic effects of secretin on three autistic children and noted significant clinical improvement, both gastrointestinal and behavioral. Secretin is now being tested with more autistic children to determine its potential.

Secretin is a natural substance, produced in the intestinal tract by all mammals. While it is not a drug, and not harmful, the Food and Drug Administration (FDA) nevertheless requires that it be sold only by prescription. Secretin is usually given by slow injection (infusion), but other methods of administration are being considered. The only FDA-approved use for secretin is in the diagnosis of gastrointestinal problems, not as a treatment for any disorder.

THE ABDOMINAL NERVOUS SYSTEM

Although many of the researchers cited above allow for the possibility that abdominal factors have etiologic significance in neurologic conditions, the anatomic and physiologic basis for such a connection is uncertain. What is there about the abdomen that could possibly be linked to neurologic conditions such as epilepsy, migraine, and autism? To answer this important question, it is helpful to review the medical literature of the early decades of the twentieth century. For example, the work of Robinson exemplifies the position that the abdomen contains a secondary brain.

In mammals there exist two brains of almost equal importance to the individual and race. One is the cranial brain, the instrument of volitions, of mental progress and physical protection. The other is the abdominal brain, the instrument of vascular and visceral function. It is the automatic, vegetative, the subconscious brain of physical existence. In the cranial brain resides the consciousness of right and wrong. Here is the seat of all progress, mental and moral . . . However, in the abdomen there exists a brain of wonderful power maintaining eternal, restless vigilance over its viscera. It presides over or-

ganic life. It dominates the rhythmical function of viscera. . . . The abdominal brain is a receiver, a reorganizer, an emitter of nerve forces. It has the power of a brain. It is a reflex center in health and disease. . . .

The abdominal brain is not a mere agent of the [cerebral] brain and cord; it receives and generates nerve forces itself; it presides over nutrition. It is the center of life itself. In it are repeated all the physiologic and pathologic manifestations of visceral function (rhythm, absorption, secretion, and nutrition) (Robinson, 1907, pp. 123–126).

For Robinson, the abdominal brain is centered in the solar plexus (Fig. 1). The abdominal brain is the primary control center of an extensive peripheral nervous system containing a number of "little brains." Anatomically, this peripheral system is roughly equivalent to the autonomic nervous system. Physiologically, the comparison breaks down because Robinson's perspective of the abdominal nervous system is much more autonomous than modern opinions about this system.

Working separately from Robinson, British Physiologist Johannis Langley also recognized the relative independence of the abdominal nervous system. Focusing on the ganglia of the gut, he believed that they do more than simply relay and distribute information from the cerebral brain. He was unable to reconcile conceptually the great disparity between the enormous numbers of neurons $[2 \times 10^8]$ in the gut and the few hundred vagus fibers from the cerebral brain, other than to suggest that the nervous system of the gut was capable of integrative functions independent of the central nervous system (Wood, 1994). Langley labeled the brain in the gut the enteric nervous system (ENS).

Although for several decades Robinson and Langley's work was ignored, modern medical research has finally rediscovered the abdominal brain with its enteric nervous system. In fact, research on the nerve connections in the abdomen represents one of the exciting areas of physiological research:

To a considerable extent, the new interest in exploring the ENS has come from the

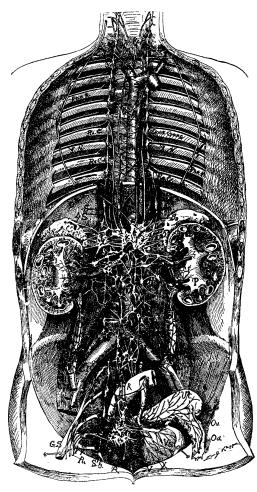


FIG. 1. Figure 33 from *The Abdominal and Pelvic Brain* by Byron Robinson, M.D. (1907). Robinson's massive work on the peripheral nervous system consists of 670 pages and 207 detailed drawings such as the one shown above.

realization that both the ENS and the remainder of the autonomic nervous system are richly endowed with neurotransmitters and neuromodulators. Many substances are found in both the bowel and the brain, a coincidence that strikes most observers as intrinsically interesting, if not immediately explicable (Gershon et al., 1994, p. 386).

The similarity between the structure of the ENS and that of the brain, combined with the ability of the ENS to mediate relatively simple behaviors, suggest that general principles can be derived from studies of the ENS that will eventually be applicable to the CNS. Given the unique position of the ENS as the only peripheral system capable of autonomous function, it seems more likely that such principles will emerge from investigations of the ENS than from studies of other aggregates of peripheral ganglia. The parallel between the bowel and the brain also suggests that newly discovered principles of central neural function may find applicability in studies of the ENS, in a sort of reverse form of reductionism whereby the brain serves as a model for the gut (Gershon et al., 1994, p. 414).

In addition to the biochemical and structural similarities between the cerebral brain and the abdominal brain, contemporary researchers are drawing computer analogies and using information processing models to describe the relationship between the cephalic and enteric brains.

The cephalic brain communicates with the smaller brain in the gut in a manner analogous to that of interactive communication between networked computers. Primary sensory afferents and extensions of intramural neurons in the gut carry information to the central nervous system. Information is transmitted from the brain to the enteric nervous system over sympathetic and parasympathetic pathways. . . . The current concept of the enteric nervous system is that of a minibrain placed in close proximity to the effector systems it controls. Rather than crowding the hundred million neurons required for control of the gut into the cranial cavity as part of the cephalic brain, and transmitting signals over long, unreliable pathways, natural selection placed the integrative microcircuits at the site of the effectors (Wood, 1994, p. 424).

Langley regarded the ENS as a third division of the autonomic nervous system (ANS) (Gershon et al., 1994). Considering the modern view of the ANS, this classification does not ade-

quately convey the scope and independence of the ENS. In fact, if Langley and Robinson are to be taken seriously, the modern concept of the ANS must be reconsidered. Currently, the entire ANS is taken to be little more than a handmaiden to the CNS. Langely and Robinson held the role of the ANS in much higher esteem than their modern counterparts.

It should also be noted that Langley focused on the nerves lining the gut in defining the ENS, whereas Robinson was interested in the entire abdominal nervous system. Robinson's perspective includes the solar plexus (abdominal brain) and its extensive network of plexuses and connecting fibers. In Robinson's book consisting of 40 chapters, the ENS is covered in one chapter. If Robinson is correct in his view of the peripheral nervous system, the rediscovery of the ENS is only the beginning of a new appreciation of the nervous system of the abdomen.

Although this article has focused on neurologic conditions with intestinal features, the reverse relationship has also been observed. For example, irritable bowel syndrome (IBS), a common intestinal disorder involving abdominal pain, disturbed defecation, and bloating, often presents with significant neurologic and psychiatric features. Watson et al. (1978) and Jones and Lydeard (1997) documented a significant comorbidity of IBS and migraine. Fent et al. (1999) described associations between colonic sensitivity in IBS and hemispheric preference and cognitive style. Whorwell et al. (1986) and Maxton et al. (1991) have noted the numerous noncolonic features of IBS, such as headache, which may be indicative of a much more diffuse disorder than has previously been appreciated.

Numerous authors have described a link between IBS and psychiatric illness, particularly anxiety and depression. The review by Walker et al. (1990) is noteworthy, as it provides a pathophysiological model linking ENS dysfunction in IBS to the locus ceruleus (LC), a portion of the brain that regulates vigilance and attention to fear provoking stimuli. Commenting on the ENS/LC model, Lydiard (1997) observed:

This model suggests that a potentially vicious positive feedback loop may be ini-

tiated and maintained by pathologic anxiety and arousal. Like IBS patients, individuals suffering from anxiety or depression experience excessive autonomic symptoms, suggesting some common pathophysiology, perhaps in part at the level of the LC....GI distress could theoretically cause or worsen psychiatric symptoms such as anxiety (Lydiard, 1997, p. 55).

Thus, the gut brain/cerebral brain interaction can manifest in a variety of signs, symptoms, and diagnostic categories. The abdominal nervous system provides a plausible link between GI and central nervous system functioning regardless of the classification of the dysfunction.

CLINICAL IMPLICATIONS

The possibility that neurologic syndromes such as epilepsy, migraine, and autism may be caused by pathology in the gastrointestinal system raises some intriguing questions with regard to clinical practice and basic research. What is the nature of the pathology? Can it be measured? If pathology is shown (or assumed) to exist, what type of treatment regimen is most effective? Is there any evidence to support therapies that focus on abdominal pathology? Can these illnesses be cured?

As noted above, from an historical perspective, the medical treatment of epilepsy and migraine often included therapy for the abdominal aspects of these diseases. Robinson's work in particular was very influential with some of the developing "alternative medicine" practitioners of the late 19th and early 20th centuries. For example, the early osteopaths held Robinson's discoveries in high esteem, citing his findings as supportive of the premise of manual therapy for the treatment of a wide range of somatic and visceral disease. Spinal and visceral manipulation techniques were used to treat almost every illness including neurological disorders such as migraine and epilepsy (American College of Mechano-Therapy, 1910; Barber, 1898; Downing, 1935; Hazzard, 1905; Murray, 1925). Figure 2 illustrates a traditional osteo-

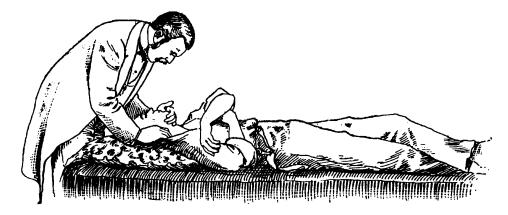


FIG. 2. Cut 18 from Osteopathy Complete by E.D. Barber, D.O., published in 1898. This illustration titled "Freeing and Stimulating the Pneumogastric Nerve" shows a typical osteopathic treatment intended to influence the 10th cranial nerve (pneumogastric/vagus). Regulation of physiologic processes (and particularly visceral physiology) was a major emphasis of early osteopathic treatment for a wide range of disorders. The vagus nerve is an important route for information between the cerebral brain and the abdominal nervous system. Osteopaths believed that they could influence both the peripheral and central nervous system by techniques aimed at nerve centers throughout the body.

pathic technique for regulating the pneumogastric nerve (vagus), a primary neurological connection between the cerebral and abdominal brains. To provide easy access to these historic works, McMillin (1998) has created a Web site containing the text of Robinson's book and several of the early osteopathic texts. A comparison of Robinson's and Cayce's views of the peripheral nervous system is also available (McMillin, 1997).

Although osteopathy has become well integrated into mainstream medical practice, the principles and techniques utilized by traditional osteopaths (e.g., manipulative therapy, diet and nutrition, hydrotherapy) have also been used by various alternative practitioners such as chiropractors and naturopaths. These forms of therapy have received increasing interest as complementary to conventional medicine. Manipulative therapy has been used in the treatment of migraine (Parker et al., 1978) and epilepsy (McGarey, 1968; Swink et al., 1997). Dietary therapy is used for migraine (Diamond et al., 1986; Mansfield et al., 1985; Vaughan, 1994). The ketogenic diet has seen increasing use for epilepsy (Kinsman et al., 1992; Swink et al., 1997). Hydrotherapy and abdominal castor oil packs have been used for epilepsy and migraine (McGarey, 1968; Mein et al., 1999).

As noted, Edgar Cayce emphasized the role of the abdominal nervous system with regard to causation and treatment of epilepsy and migraine. Consistent with the growing body of medical information on the "abdominal brain" and enteric nervous system, Cayce referred to the abdominal brain as the "solar plexus brain" (Cayce, 1926, 1944), the "secondary brain" (Cayce, 1944), and the "central brain in the solar plexus" (Cayce, 1927). Of particular interest are Cayce's therapeutic recommendations for epilepsy and migraine, which included special diets, abdominal castor oil packs, colonic irrigations, spinal manipulation and massage, and herbal teas to heal the intestinal tract. All these therapies were directed to improving digestive system functioning, and thereby decreasing nervous system incoordination between the abdominal and cerebral brains. Detailed analysis of Cayce's approach to epilepsy (Pahnke, 1968) and migraine (Bjork, 1968) have been published. Reilly and Brod (1975), McGarey (1983), and Mein (1989) have described Cayce's approach to the digestive system as it relates to systemic functioning.

Interestingly, one reading given for a child with mild autistic features recommended a digestive supplement similar to secretin (Cayce, 1937). A pilot study of 16 children with minimal brain dysfunction (including autism)

based on Cayce's concepts of intestinal etiology, showed promising results (Pecci, 1977). The Meridian Institute has investigated Cayce's therapeutic recommendations for migraine in a small pilot study (n = 5). Participants who followed Cayce's suggestions showed notable improvement (Meridian Institute, 1997).

CONCLUSION

Neurologic diseases with systemic features (particularly with significant GI symptoms) may be approached from a complementary medicine model that recognizes the role of the abdominal nervous system with regard to etiology and treatment. By linking the historical, systems-oriented clinical approaches to the modern research literature on the enteric nervous system, a complementary approach may be created that integrates the best of standard medical practice with traditional and alternative modalities and systems that are consistent with established anatomy and physiology.

Although epilepsy and migraine are common illnesses, the abdominal form of each is generally regarded as rare. Based on the sources described in this article, we are suggesting that idiopathic epilepsy and migraine may be understood better if the abdominal features were more thoroughly investigated. Abdominal epilepsy and migraine may not actually be rare. Modern medicine considers them rare because little attention has been given to the meaning of abdominal symptoms associated with these conditions. Perhaps the idiopathic forms of both illnesses involve intestinal etiology. Similarly, the intestinal aspects of autism may be clues to an important subgroup of this disorder.

Further research into the etiology and treatment of these conditions should consider possible abdominal nervous system involvement. Clinically, the presence of significant abdominal features may indicate that the treatment plan include traditional features (i.e., diet, colon hydrotherapy, and manipulative therapy) that may favorably influence the abdominal brain and ENS.

REFERENCES

- American College of Mechano-Therapy. Textbook of Osteopathy. Chicago: American College of Mechanotherapy, 1919.
- Agnoli A, De Marinis M. Vascular headaches and cerebral circulation: An overview. Cephalalgia 1985;5(suppl 2):9–15.
- Agrawal P, Dhar NK, Bhatia MS, Malik SC. Abdominal epilepsy. Ind J Pediatr 1989;56:539–541.
- Amar AP, Heck CN, Levy ML, Smith T, DeGiorgio CM, Oviedo S, Apuzzo ML. An institutional experience with cervical vagus nerve trunk stimulation for medically refractory epilepsy: Rationale, technique, and outcome. Neurosurgery 1998;43:1265–1276.
- Amery WK, Forget PP. The role of the gut in migraine: The oral 51-Cr EDTA test in recurrent abdominal pain. Cephalalgia 1989;9:227–229.
- Babb RR, Eckman PB. Abdominal epilepsy. JAMA 1972;222:65–66.
- Barber ED. Osteopathy Complete. 4th ed. Kansas City, MO: Hudson-Kimberly Publishing Company, 1898.
- Bentley D, Katchburian A, Brostoff J. Abdominal migraine and food sensitivity in children. Clin Allergy 1984;14: 499–500.
- Berdichevskii M. Meso-diencephalic epilepsy after abdominal injury. Vopr Psikhiatr Nevropatol 1965;11:374–376. Bjork RO. Migraine headaches. In McGarey WA, ed.
- Physicians Reference Notebook. Virginia Beach, VA: A.R.E. Press, 1968:258–263.
- Bondarenko ES, Shiretorova DC, Miron VA. Abdominal syndrome in the structure of cerebral paroxysms in children and adolescents. Soviet Med 1986;2:39–44.
- Buzzi MG, Moskowitz MA. The trigemino-vascular system and migraine. Pathol Biol 1992;40:313–317.
- Buzzi MG, Bonamini M, Moskowitz MA. Neurogenic model of migraine. Cephalalgia 1995;15:277–280.
- Callan JP. Editorial. JAMA 1979;241:1156.
- Cayce E. Edgar Cayce reading 1800-15; 1926. In: The Complete Edgar Cayce Readings on CD-ROM. Virginia Beach, VA: A.R.E. Press, 1993.
- Cayce E. Edgar Cayce reading 4613-1; 1927. In: The Complete Edgar Cayce Readings on CD-ROM. Virginia Beach, VA: A.R.E. Press, 1993.
- Cayce E. Edgar Cayce reading 1179-4; 1937. In: The Complete Edgar Cayce Readings on CD-ROM. Virginia Beach, VA: A.R.E. Press, 1993.
- Cayce E. Edgar Cayce reading 3326-1; 1943. In: The Complete Edgar Cayce Readings on CD-ROM. Virginia Beach, VA: A.R.E. Press, 1993.
- Cayce E. Edgar Cayce reading 294-212; 1944. In: The Complete Edgar Cayce Readings on CD-ROM. Virginia Beach, VA: A.R.E. Press, 1993.
- Costa M, Brookes SJ. The enteric nervous system. Am J Gastroenterol 1994;89:S29–137.
- Dalessio DJ. Dietary migraine. Am Fam Physician 1972; 6:60-65
- Dalton K. Food intake prior to a migraine attack—Study of 2,313 spontaneous attacks. Headache 1975;15: 188–192.

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- Davenport WW. A Digest of Digestion. Chicago: Year Book Medical Publishers, 1978.
- Dercum FX. Treatment of the so-called functional disorders. In Musser JH, Kelly AO, eds. A Handbook of Practical Treatment. Philadelphia, PA: W.B. Saunders Co., 1912:857–949.
- Diamond S, Prager J, Freitag FG. Diet and headache. Is there a link: Postgrad Med 1986;79:279–286.
- Douglas EF, White PT. Abdominal epilepsy—A reappraisal. J Pediatr 1971;78:59–67.
- Downing CH. Osteopathic Principles in Disease. San Francisco: Ricardo J. Orozco, 1935.
- Fent J, Balazs L, Buzas G, Erasmus LP, Holzl R, Kovacs A, Weisz J, Adam G. Colonic sensitivity in irritable bowel syndrome and normal subjects according to their hemispheric preference and cognitive style. Integr Physiol Behav Sci 1999;34:54–62.
- Gershon MD, Kirchgessner AL, Wade PR. Functional anatomy of the enteric nervous system. In: Johnson LR, ed. Physiology of the Gastrointestinal Tract. 3rd ed. (Vol. 1). New York: Raven Press, 1994:381–416.
- Gordon B, ed. Hughes' Practice of Medicine. 16th ed. Philadelphia: The Blakiston Company, 1942.
- Goyal RK, Hirano I. The enteric nervous system. N Engl J Med 1996;334:1106–1115.
- Grant EC. Food allergies and migraine. Lancet 1979;1: 966–969.
- Handforth A, DeGiorgio CM, Schachter SC, Uthman BM, Naaritoku DK, Tecoma ES, Henry TR, Collins SD, Vaughn BV, Gilmartin RC, Labar DR, Morris GL, Salinsky MC, Osorio I, Ristanovic RK, Labiner DM, Jones JC, Murphy JV, Ney GC, Wheless JW. Vagus nerve stimulation therapy for partial-onset seizures: a randomized active-control trial. Neurology 1998;51:48–55.
- Hanington E. Diet and migraine. J Hum Nutr 1980; 34:175–180.
- Hare HA. A Text-Book of Practical Therapeutics, with Especial Reference to the Application of Remedial Measures to Disease and Their Employment upon a Rational Basis. 14th ed. Philadelphia: Lea & Febiger, 1912:805–806.
- Hazzard C. The Practice and Applied Therapeutics of Osteopathy. 3rd ed. Kirksville, MO: Journal Printing Company, 1905.
- Horvath K, Stefanatos G, Sokolski KN, Wachtel R, Nabors L, Tildon JT. Improved social and language skills after secretin administration in patients with autistic spectrum disorders. J. Assoc Acad Minor Phys 1998;9:9–15.
- Hotta T, Fujimoto Y. A study on abdominal epilepsy. Yonago Acta Medica 1973;17:231–239.
- Hughes EC, Gott PS, Weinstein RC, Binggeli R. Migraine: A diagnostic test for etiology of food sensitivity by a nutritionally supported fast and confirmed by longterm report. Ann Allergy 1985;55:28–32.
- Jones R, Lydeard S. Irritable bowel syndrome in the general population. BMJ 1992;304:87–90.
- Juillard E. Abdominal pains and epilepsy. Praxis 1967;56:
- Kinsman SL, Vining EP, Quaskey SA, Mellitis D, Freeman JM. Efficacy of the ketogenic diet for intractable seizure

- disorders: Review of 58 cases. Epilepsia 1992;33:1132–1136.
- Loar CR. Abdominal epilepsy. JAMA 1979;241:1327.
- Lundgren J, Amark P, Blennow G, Stromblad LG, Wallstedt L. Vagus nerve stimulation in 16 children with refractory epilepsy. Epilepsia 1998;39:809–813.
- Lydiard RB. Anxiety and the irritable bowel syndrome: Psychiatric, medical, or both? J Clin Psychiatry 1997;58(suppl 3):8;59–61.
- Mansfield LE. The role of food allergy in migraine: A review. Ann Allergy 1987;58:313–317.
- Mansfield LE. Food allergy and headache. Whom to evaluate and how to treat. Postgrad Med 1988;83:46–51.
- Mansfield LE, Vaughan TR, Waller SF, Haverly RW, Ting S. Food allergy and adult migraine: double-blind and mediator confirmation of an allergic etiology. Ann Allergy 1985;55:126–129.
- Matsuo F. Partial epileptic seizures beginning in the truncal muscles. Acta Neurol Scand 1984;69:264–269.
- Mavromichalis I, Zaramboukas T, Giala MM. Migraine of gastrointestinal origin. Eur J Pediatr 1995;154:406–410.
- Maxton DG, Morris J, Whorwell PJ. More accurate diagnosis of irritable bowel syndrome by the use of "noncolonic" symptomatology. Gut 1991;32:784–786.
- McCarey WA. The Edgar Cayce Remedies. New York: Bantam Books, 1983.
- McGarey WM. Physician's Reference Notebook. Virginia Beach, VA: A.R.E. Press, 1968.
- McMillin DL. The Early American Manual Therapy Web site located at: http://members.visi.net/~mcmillin1998.
- McMillin DL. Selections from the Abdominal and Pelvic Brain with commentary by David McMillin. Virginia Beach, VA: Lifeline Press, 1997.
- Mein EA. Keys to health: The Promise and Challenge of Holism. San Francisco, CA: Harper & Row Publishers, 1989.
- Meridian Institute. Migraine report documents improvement. Meridian Institute News, 1997:1:1–2.
- Mitchell WG, Greenwood RS, Messenheimer JA. Abdominal epilepsy. Cyclic vomiting as the major symptom of simple partial seizures. Arch Neurol 1983;40:251–252.
- Monro J, Carini C, Brostoff J. Migraine is a food allergic disease. Lancet 1984;2:719–721.
- Moore MT. Abdominal epilepsy. JAMA 1972;222:1426.
- Mortimer, MJ, Good PA. The VERG as a diagnostic marker for childhood abdominal migraine. Headache 1990;30:642–645.
- Murch SH, Thomson MA, Walker-Smith JA. Author's reply. Lancet 1998;351:908.
- Murray CH. Practice of Osteopathy: Its Practical Application to the Various Diseases of the Human Body. 6th Ed. Elgin, IL: Charles H. Murray, 1925.
- O'Donohoe NV. Abdominal epilepsy. Dev Med Child Neurol 1971;13:798–800.
- Pahnke WN. Epilepsy. In McGarey WA, ed. Physicians Reference Notebook. Virginia Beach, VA: A.R.E. Press, 1968:132–145.
- Parker GB, Tupling H, Pryor DS. A controlled trial of cervical manipulation of migraine. Aust N Z J Med 1978;8:589–593.

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- Peatfield RC, Glover V, Littlewood JT, Sandler M, Clifford RF. The prevalence of diet induced migraine. Cephalalgia 1984;4:179–183.
- Peatfield RC. Relationships between food, wine, and beer precipitated migrainous headaches. Headache 1995; 35:355–357.
- Pecci EF. Minimal brain dysfunction in children. Lecture delivered at the 10th Annual A.R.E. Clinic Medical Symposium. Scottsdale, Arizona, January 1977.
- Peppercorn MA, Herzog AG, Dichter MA, Mayman CI. Abdominal epilepsy: A cause of abdominal pain in adults. JAMA 1978;40:2450–2451.
- Peppercorn MA, Herzog AG. The spectrum of abdominal epilepsy in adults. Am J Gastroenterol 1989;84:1294– 1296.
- Pert CB, Ruff MR, Weber RJ, Herkenham M. Neuropeptides and their receptors: A psychosomatic network. J Immunol 1985:135:820S–826S.
- Reilly H, Brod R. The Edgar Cayce Handbook for Health Through Drugless Therapy. Virginia Beach, VA: A.R.E. Press 1975
- Reimann HA. Abdominal epilepsy and migraine. JAMA 1973;224:128.
- Robinson B. The Abdominal and Pelvic Brain. Hammond, IN: Frank S. Betz, 1907.
- Santoro G, Curzio M, Venco A. Abdominal migraine in adults. Case reports. Funct Neurol 1990;5:61–64.
- Silberstein SD. Migraine symptoms: results of a survey of self-reported migraineurs. Headache 1995;35:387–396.
- Singh PD, Kaur S. Abdominal epilepsy misdiagnosed as psychogenic pain. Postgrad Med J 1988;64:281–282.
- Snively C, Counsell C, Lilly D. Vagus nerve stimulator as a treatment for intractable epilepsy. J Neurosci Nurs 1998;30:286–289.
- Solana de Lope J, Alarcon FO, Aguilar MJ, Beltran CJ, Barinagarrementeria F, Perez MJ. Abdominal epilepsy in the adult. Rev Gastroenterol 1994;59:297–300.
- Spear IJ. A Manual of Nervous Diseases. Philadelphia, PA: W.B. Saunders Company, 1916:620–627.
- Swink TD, Vining EP, Freeman JM. The ketogenic diet: 1997. Adv Pediatr 1997;44:297–329.
- Symon DN, Russell G. Abdominal migraine: A childhood syndrome defined. Cephalalgia 1986;6:223–228.
- Takei T, Nakajima K. Autonomic abdominal epilepsy-

- Clinico-encephalographic evaluation of 24 cases. Nippon Shonika Gakkai Zasshi 1967;71:543–551.
- Thomsen LL, Olesen J. The autonomic nervous system and the regulation of arterial tone in migraine. Clin Auton Res 1995;5:243–250.
- Trotsky MB. Neurogenic vascular headaches, food and chemical triggers. Ear Nose Throat J 1994;73:228–236.
- Vaughan TR. The role of food in the pathogenesis of migraine headache. Clin Rev Allergy 1994;12:167–180.
- Wakefield AJ, Murch SH, Anthony A, Linnell J, Casson DM, Malik M, Berelowitz M, Dhillon AP, Thomson MA, Harvey P, Valentine A, Davies SE, Walker-Smith JA. Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. Lancet 1998;351:637–641.
- Walker, EA, Roy-Byrne PP, Katon WJ. Irritable bowel syndrome and psychiatric illness. Am J Psychiatry 1990; 147:565–572.
- Watson WC, Sullivan SN, Corke M, Rush D. Globus and headache: Common symptoms of the irritable bowel syndrome. Can Med Assoc J 1978;118:387–388.
- Whorwell PJ, McCallum M, Creed FH, Roberts CT. Non-colonic features of irritable bowel syndrome. Gut 1986:27:37–40.
- Wilson CW, Kirker JG, Warnes H, O'Malley M. The clinical features of migraine as a manifestation of allergic disease. Postgrad Med J 1980;56:617–621.
- Wood JD. Physiology of the enteric nervous system. In: Johnson LR, ed. Physiology of the Gastrointestinal Tract. 3rd ed. (Vol. 1). New York: Raven Press, 1994.
- Yingkun F. Abdominal epilepsy. Chin Med J 1980;93: 135–148.
- Zarling EJ. Abdominal epilepsy: An unusual cause of recurrent abdominal pain. Am J Gastroenterol 1984;79: 687–688.

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