

Management of Non-Tropical Sprue: Medical vs Osteopathic

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FROM THE ARCHIVES

Among the mysteries occasionally confronting a busy general practitioner, one of the most baffling is non-tropical sprue. It is also known as celiac disease, idiopathic steatorrhea, malabsorption syndrome and steatorrhea syndrome.

According to Cecil and Loeb: "The term sprue apparently derives from the Dutch word 'sprouw,' meaning aphthous stomatitis which was first used by Katelaer in 1669 to designate an illness characterized by sore mouth and voluminous stools. The first description of sprue in 1766 is credited to an Englishman, William Hillary, in the Barbados, but it was not until 1880 that the publications of Sir Patrick Manson in China and Van der Burg in Batavia clearly identified sprue and awakened widespread scientific interest in the disease. The extensive studies of Thaysen in 1932 led to more frequent recognition of sprue in temperate climates and pointed to the underlying identity of tropical sprue, non-tropical sprue and celiac disease."¹

According to Wollaeger and Green: "The essential features of this syndrome are the signs and symptoms of abnormal intestinal function, such as diarrhea, flatulence and loss of weight, plus the manifestations of associated nutritional-deficiency states that are usually present, such as anemia, hypoproteinemia with edema and osteomalacia. There is good evidence to show that non-tropical sprue in adult patients is the same as celiac disease in infants and children. Probably also tropical and non-tropical sprue are closely related. The term 'non-tropical sprue' seems preferable to 'idiopathic steatorrhea' or 'malabsorption syndrome' as a designation for this disease."²

But according to Comfort and Wollaeger: "The impairment of absorption in non-tropical sprue, contrary to early beliefs, affects all nutrients, including fat, protein, carbohydrates, vitamins, minerals and even water. Although steatorrhea is an easily recognized characteristic feature of this disease, the fecal content of nutrients other than fat is also increased. For this reason the terms 'idiopathic steatorrhea' and 'steatorrhea syndrome' are not satisfactory designations for non-tropical sprue."³

The essential features of non-tropical sprue are the signs and symptoms of abnormal intestinal function, such as diarrhea, flatulence,

Editor's note: We have found a great article to present "from the archives" in this issue. Dr Doran A. Farnum, DO, takes us through an extensive review of multiple diseases which cause signs and symptoms of intestinal malfunction. This is taken from the 1967 yearbook of the Academy of Applied Osteopathy, and although molecular biology has certainly advanced, the majority of the clinical symptoms, signs, and known findings have not. In addition, for several of these diseases, the treatments have advanced only minimally. Dr Farnum did an excellent job of using a case to review the osteopathic findings in regards to both structural and viscerosomatic changes and associated treatments as well as Chapman's reflexes, and he brings together multiple references to the research and thinking of the time. I was somewhat amused that the patient was not allowed to drink coffee or eat pork, but tea and Sanka were okay. Please enjoy Dr Farnum's article.

This article has been edited to conform to The AAO Journal's current style and to standardize spelling and punctuation.

When he retired in November 2013, Dr Farnum had been California's oldest physician with an active medical license and the oldest member of both the American Academy of Osteopathy (AAO) and the American Osteopathic Association still in practice.

Dr Farnum died April 30, 2015, at age 103. Learn more about Dr Farnum in the [May 2015 issue](#) of AAO Member News.

loss of weight and muscular weakness, plus the manifestations of associated nutritional-deficiency states that are usually present, such as microcytic or macrocytic anemia, hypoproteinemia with edema, glossitis and osteomalacia. This disease is not only of malnutrition but also a disease of malabsorption.

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Diagnosis

The standard medical diagnosis of non-tropical sprue is made by clinical signs and symptoms, x-ray examination of small intestines and stool examinations. In the x-ray examination, it is found that the small bowel is dilated; the mucosal markings are thickened; the contours of the lumen are smooth; the usual markings of the valvulae conniventes are obliterated; barium is clumped in elongated masses, and after the bulk of the opaque meal has passed, remnants of barium adhere to the walls, giving it a peculiar flecked appearance.

Non-tropical sprue must be differentiated from pancreatic steatorrhea, nonspecific granulomatous gastroenteritis (regional enteritis), Whipple disease, steatorrhea complicating diabetes mellitus with neuropathy, amyloidosis and scleroderma, lymphosarcoma and other tumors involving the small intestines and tropical sprue.

Pancreatic Steatorrhea

The steatorrhea usually develops after years of recurring attacks of abdominal pain characteristic of recurrent acute or subacute pancreatitis. It is a late development which indicates extensive destruction of the pancreas. Concomitantly with the development of steatorrhea, pancreatic calcification or diabetes, or both, appear and serve to locate the pathologic process in the pancreas.

Nonspecific Granulomatous Gastroenteritis (Regional Enteritis)

Careful roentgenologic examination of the small bowel usually will establish the diagnosis and should be carried out in every case of sprue-like syndrome. Fever, abdominal pain, tender masses and fistulas also serve to distinguish regional enteritis from non-tropical sprue. Other types of chronic enteritis, particularly tuberculosis, may produce steatorrhea and must be considered in the diagnosis.

Whipple Disease

Whipple disease is a rare condition which produces a sprue-like syndrome. The principal findings are voluminous fatty stools, pigmentation, arthritis and loss of weight. It may be suspected as the cause of the syndrome when arthritis is associated and in some instances when an abdominal mass is present. The diagnosis of Whipple disease may be proved by demonstration of the characteristic microscopic pathologic findings on biopsy of the wall of the small intestine and mesenteric lymph nodes.

Steatorrhea Complicating Diabetes Mellitus With Neuropathy

Severe, long-standing diabetes mellitus associated with neuropathy sometimes produces a sprue-like syndrome presumably by involvement of the nervous mechanism of control of the intestinal tract. The simultaneous occurrence of lesions of the autonomic and

peripheral nerves in a case of long-standing severe diabetes mellitus with fatty diarrhea permits a ready diagnosis.

Amyloidosis and Scleroderma

Amyloidosis occasionally involves the small bowel and produces a sprue-like syndrome. It is suspected when the disease involves multiple organs (heart, liver, skin, intestinal tract, kidneys and others) and is proved by the Paunz test or biopsy of an involved organ with demonstration of amyloid by the appropriate staining procedures.

Scleroderma is another widespread disease which may occasionally involve the small intestine and cause steatorrhea. Because of the skin and joint manifestations as well as the evidence of anatomic and functional change in the esophagus, this disease usually is not difficult to recognize.

Lymphosarcoma and Other Tumors Involving the Small Intestine

Lesions of this type may produce a clinical picture closely simulating sprue and are not always detectable even on careful roentgenologic examination of the small bowel. The clinical course of the patients with such lesions is likely to be more progressive and the response to treatment not as satisfactory as that of most patients with non-tropical sprue.

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Tropical Sprue

It is possible, as some observers believe, that tropical and non-tropical sprue are the same disease occurring in different climatic belts of the world. Reports in the medical literature indicate that certain differences exist between them, however, among these are the following differences:

1. Tropical sprue in some parts of the world has a seasonal incidence and tends to occur in certain geographic regions in the tropics where it may affect many inhabitants at the same time. It often develops in a relatively abrupt manner and runs a rapid course. Non-tropical sprue, on the other hand, is less common, occurs sporadically, has no seasonal or geographic incidence, and is usually a chronic and insidious disease.
2. In most cases of tropical sprue reported, a macrocytic anemia indistinguishable from pernicious anemia is present and is associated with megaloblastic bone marrow. Although, macrocytic anemia is found commonly in non-tropical sprue, it only occasionally presents the characteristics of pernicious anemia and may not be associated with megaloblastic marrow.

3. Most patients with tropical sprue give a history of inadequate ingestion of food, particularly of protein. Among patients with non-tropical sprue such a history is the exception rather than the rule.
4. Evidences of vitamin D deficiency such as osteomalacia and tetany are rarely reported among patients with tropical sprue but are common in the non-tropical variety.

According to Comfort and Wollaeger, the complete medical treatment of non-tropical sprue consists of:

Treatment of non-tropical sprue over the years has proved difficult. Although some patients who adhere closely to a careful program of treatment may get along satisfactorily for many months or years, intake-excretion studies carried out when they are in remission seldom if ever disclose entirely normal absorption of fat. Also, any deviations from the regimen of treatment, particularly dietary indiscretions, are likely to cause a flare up of symptoms. ... Dependence should not be placed on a single therapeutic measure. Instead, in the absence of a specific remedy, a regimen should be chosen which is based on knowledge of the pathologic physiology of the disease and is designed to avoid irritating or overburdening the malfunctioning bowel, as well as to compensate for malabsorption and to overcome deficiency states. One general measure is avoidance of over expenditure of nervous and physical energy. A period of rest in bed during exacerbations of the disease is often helpful in bringing about a remission.

The objective of dietary management is to overcome and avoid malnutrition and the protein, mineral and vitamin deficiencies. The diet should contain calories, protein, minerals, and vitamins in quantities larger than needed by the normal person to allow for losses in the feces. ... It should be low in fat to avoid the irritating effects which even moderate amounts of fat produce on the gastrointestinal tracts of patients with this disease.

The foundation diet provides 130 g of protein, 55 g of fat, and 370 g of carbohydrates with 2500 calories. It also contains 1780 mg of calcium, 18 mg of iron and meets the recommended daily allowance of the National Research Council for all vitamins except vitamin A. The full diet provides 130 g of protein, 70 g of fat, 400 g of carbohydrates and 2750 calories and meets the recommended allowances in every respect. In general, the diet is low in fat and high in protein, carbohydrates and calories, and the amount of residue is restricted.

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A large proportion of the carbohydrates used are those from fruits and simple sugar, fructose and glucose. The diet is varied with the person; in some cases even the foundation diet will require alteration. In others, the physician may order additions to increase the calories by increasing the fat and starch allowed, especially during remissions. In general, patients use the foundation diet during exacerbation of their disease, and additions to the diet can be made one at a time as clinical improvement occurs until a full sprue diet is being taken.

The observation ... in 1950, that children with celiac disease improved when wheat flour and rye flour were excluded from their diets, and ... that fecal fat decreased when these flours were excluded from the diet have altered the therapy of celiac disease. The clinical similarity, if not actual identity, of celiac disease and non-tropical sprue induced workers in 1954 to treat non-tropical sprue similarly. They reported that 5 of 12 patients with non-tropical sprue apparently returned to normal health on a wheat-free diet in two months. ... Encouraging results have been obtained in a few patients treated at the ... clinic, but there have also been failures. These experiences suggest the need for a critical trial of the gluten-free diet. In a disease like non-tropical sprue which has spontaneous remissions and which usually responds satisfactorily to the time-tested sprue diet outlined, it may not be easy to evaluate this new diet or to demonstrate its superiority over diets now commonly in use. Since the gluten-free diet imposes considerable restriction on the patient and entails the use of special recipes for wheat and rye substitutes, superiority must be clearly demonstrated before the diet can be prescribed routinely for all patients with non-tropical sprue. It may be found useful in the treatment of certain patients who do not respond favorably to the usual measures.

Each patient should receive at least one multiple vitamin capsule daily. If evidence of vitamin deficiencies exists, large additional doses of the several vitamins may be given.

Because of the frequency with which osteomalacia develops in non-tropical sprue, the oral administration of calcium lactate in doses of 4 g (1 dram) 3 times per day along with suitable prophylactic doses of vitamin D is recommended for all patients. For patients with known osteomalacia, larger doses of calcium lactate, as high as 12 g (3 drams) 3 times per day and even higher, and also larger doses of vitamin D are necessary. For the treatment of active tetany the intravenous use of calcium gluconate is helpful.

The frequency of potassium deficiency and the probability that hypokalemia is one of the common causes of death in non-tropical sprue emphasize the need for adequate replacement therapy by the oral, and if necessary, by the intravenous route during episodes of diarrhea. Intravenous infusions of 5% dextrose in isotonic saline solution are helpful in the management of patients with water and electrolyte imbalance associated with exacerbations of the disease.

Iron deficiency anemias when present can sometimes be corrected by the oral administration of ferrous sulfate or other iron-containing compounds. However, many patients do not tolerate such medication, and in those who do, the response of the anemia to their administration is often disappointing. Even when iron is administered parenterally, the anemia of some patients thought to be due to iron deficiency is not corrected. On occasions blood transfusions are helpful in controlling intractable anemia and severe hypoproteinemia or in rehabilitating seriously ill patients. Although the effects are often temporary, the administration of serum albumin in some instances has seemed to improve the patient's general condition and bring about a remission of symptoms which have persisted for long periods. This type of therapy has also been of use in the control of severe and persistent tetany which does not respond readily to the administration of large doses of calcium and vitamin D.

Cortisone, hydrocortisone, and their analogues, prednisone and prednisolone, as well as corticotropin (ACTH) exert a beneficial effect when administered to patients with non-tropical sprue in relapse. Both subjective and objective improvement are observed. Stools decrease in number and become more normal in consistency; appetite and strength improve, and symptoms of abdominal cramping and distention become less marked or even disappear. The amounts of solids, water, fat, and nitrogen in the feces decrease, and the prothrombin time returns to normal. Fecal loss of potassium is decreased, but its urinary excretion is increased, and the serum level may be further depressed. Sodium and chloride are retained at least initially during treatment with cortisone with increased positive balances and elevation of serum values. Rebound occurs when the use of cortisone is discontinued. Values for serum calcium may increase toward normal; fecal losses may decrease, and positive balance may be exhibited, but in some cases hypocalcemia has become more marked in spite of treatment with hormones, indicating the necessity of continuing administration of calcium and vitamin D as an adjunct to cortisone

therapy. Untoward symptoms, including hypertension and edema, occur when the hormone is given in large doses.

The authors' experience with steroid therapy in non-tropical sprue has been confined almost exclusively to the use of cortisone acetate administered orally—a route which has been found more effective than parenteral injection. Cortisone is not used to replace the usual methods of treatment, such as diet, rest, and the administration of vitamins and minerals, but rather to supplement them and help establish a remission in patients who are severely ill and whose symptoms do not respond readily to ordinary measures.⁵

In my opinion, non-tropical sprue is more a disease of malabsorption rather than one of malnutrition. The body does not only fail to digest fats but also fails to digest proteins. Stool examinations show an excessive loss of protein as well as fat. Even though the patient has a normal ingestion of proteins, there is loss of muscle tone and muscle strength, as well as loss of weight. I feel that many cases of non-tropical sprue are subclinical in nature and are not diagnosed. One step further in their development and they could be diagnosed as such. I believe this disease exists in a pre-clinical state, asymptomatic except from occasional isolated symptoms not referable to a specific syndrome. The patient may have only the flatulence and loss of weight with an occasional diarrhea, but not of the voluminous, frothy type; or he may have weakness and loss of weight with an increase in the number of bowel movements per day without diarrhea. Osteopathic management of both classical and subclinical non-tropical sprue provides the physician with the only effective approach to these conditions. Classical medical management is, for the most part, symptomatic, palliative and temporary. To exemplify this opinion, I shall present a detailed case history which was treated both medically and osteopathically.

Case History

In April 1959, a white, asthenic, 44-year-old female consulted me because of a condition that has been diagnosed as non-tropical sprue. She was 5 feet, 2 inches tall, weighed 116 pounds, and looked worried and apprehensive. She had had this condition for about 4 years.

In February 1954, the patient had gone to a doctor complaining of voluminous, frothy stools once a day, but occasionally 2 to 4 times a day. She had constant pain across the lower abdomen, similar to intestinal flu. She had had a loss of weight from approximately 118 pounds to below 100. She was hospitalized for 3 days for a complete examination in March 1954. A complete blood and urine examination was made; various liver tests were done, as well as an

x-ray examination including GI, colon, and gall bladder series. These 2 studies were inconclusive.

In April 1954, the patient was hospitalized in a second hospital where a complete blood and urine examination, as well as a stool examination and liver tests were made. Another complete x-ray examination was also made. Her condition was diagnosed as non-tropical sprue.

Conventional Medical Treatment

The treatment prescribed consisted of liquid vitamin B complex, a vitamin-mineral tablet, high protein concentrate and vitamin B₁₂ injections.

A rigid diet was prescribed consisting of beef, chicken, lamb and liver, and baked potatoes (no skins, butter or gravy). Vegetables allowed were beets, carrots, peas, green beans, and squash. No fats, wheat products, cabbage or vegetables from the cabbage family, onions, melons, raw fruits—except bananas, strawberries and peaches—no pork and no coffee were permitted. Tea and Sanka were allowed.

The patient improved over a 2 1/2-year period, with some remissions. During this time, she had periodic examinations.

In March 1957, the patient suffered a severe flare-up of the bowel condition and was re-hospitalized, and another complete examination was made, including gastric analysis. She was found to have a non-functioning pancreas and achlorhydria. Pancreatin and hydrochloric acid were given. She was also instructed to stay on her other medication and diet. She was much improved for 1 week and then, suddenly, became much worse.

The patient was sent to Mayo Clinic for a complete examination and evaluation. They found that the pancreas was functioning normally and she had normal gastric acidity. From a very complete x-ray examination of the small bowel, where every inch of the small intestine was fluoroscoped and x-rayed, they found nothing except an extremely overactive digestive tract. She was told it was due entirely to nerves and was given a prescription of Belladonal and vitamin K. She improved to some extent but still had recurrences of the bowel condition, where the bowel movement was voluminous, frothy and liquid, and left her with extreme weakness.

In March 1958, the patient developed a swelling and rash of both legs. She was again hospitalized, and a diagnosis of drug allergy,

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*Information gained from lecture by Mitchell.

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probably due to the Belladenal, was made. The Belladenal was withdrawn, and she was given an antihistamine and cortisone. She improved slowly, and returned home after 4 days, but was instructed to stay off her feet for at least 2 weeks. The swelling and rash disappeared completely, with time, and she continued to improve slightly, but had flare-ups of the bowel symptoms. All this time she continued vitamin B complex, vitamin K and the prescribed diet. She complained of an empty, weak feeling, having to eat 5 times a day, and having to rest an hour and a half every afternoon in order to do the barest of her house work. Heart, lungs, blood pressure, pulse, CBC and urinalysis were normal.

Osteopathic Treatment

Osteopathic structural examination revealed pelvic imbalance, a combination of symphyseal, sacroiliac and iliosacral lesions.

In this case, the pelvis was balanced first, using Mitchell's technique as described in the Academy of Applied Osteopathy Yearbook 1958. Care was taken to correct the lesions in their proper order: symphyseal, sacroiliac, and iliosacral. The imbalance of all 6 groups of the thigh muscles was equalized—adductors, abductors, hamstrings, external and internal rotators, and quadriceps. Intermittent muscle resistance, according to Ruddy, was used to reestablish them to a state of bilateral balance so the muscles were of equal length and strength. Since the iliosacral lesion is caused by forces from below upward into the sacroiliac joint, it is quite logical that when the leg muscles are not equal in length they do not exert equal pull on both sides of the pelvis to which they attach. I've found this lesion to occur more frequently than the others.

Attention was given next to the lumbar curve which was a lateral curve to the left with the bodies of the vertebrae rotating into the convexity. The bottom of the curve began to break over at the level of L4 and L5. In examining this part of the problem, the body of L4 was found both laterally flexed and rotated to the left. Correction was made with the patient sitting in forward bending and the spine was both laterally flexed and rotated to the right, carrying L4 into the corrective position until the restrictive barrier was reached. The patient was then instructed to push back and up for 3 intermittent efforts; each time lateral flexion and rotation were increased as the tissues released. Fryette's principles were used for the easy-normal correction. However, the method of application was one of guiding with the use of muscular energy as the initiating force. The lower lumbar lesion is important, as it is at the level of the posterior colon reflex (3-5L).

The accommodative scoliosis apexed at T9, T5 and C7. Derotation of these curves was accomplished with the same type of technique as was used in the lumbar curve apex.

In the lower cervical area, C7 was rotated posteriorly to the right in easy normal. There was involvement in the upper cervical spine with lesions of C1, C2, and C3 which I felt could possibly have been interfering with the vagus distribution to the small intestines. The vagus arises from the medulla oblongata and passes down through this area. Contraction and congestion of tissues in the upper cervical area could conceivably interfere with its normal function. I feel that it did, as the small intestine function improved with cervical functional improvement.

The paravertebral tissues along with the entire spine were contracted. These structures were given attention, bilaterally, with direct fascial technique as described by Ida P. Rolf.*

The 4th, 10th, 11th and 12th ribs on the left were lesioned in inspiration, as was the 1st rib on the right. These were corrected with a guiding type of technique using respiration as the intrinsic force.

The viscerosomatic reflexes of the colon were palpable, as were those of the small intestines. The colon reflex centers are on the

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anterolateral aspect of both thighs, and extend from the greater trochanter down to approximately 1½ inches above the knee. An interesting point is that these reflexes are located in inverse position. The ascending colon reflex center starts at the right greater trochanter, and, as you go down the thigh, the reflex influence moves up toward the hepatic flexure and the right transverse colon. On the left side, the same is true of the descending colon moving up to the splenic flexure and the left side of the transverse colon as you go down the left thigh. Shotty plaques were felt along the anterolateral border of each thigh. These plaques vary in size and shape. They may be small, the size of a pea, or they may be stringy masses 1 to 5 or 6 inches long. These plaques were extremely tender and painful to touch. Of interest was the fact that as symptomatic changes of improvement occurred (fewer stools and less discomfort) there was notably palpatory change in the anterior colon reflexes. They decreased in size and tenderness.

The anterior reflex centers of the small intestines are located at the anterior end of the 9th, 10th, and 11th intercostal spaces. The posterior reflex centers of the small intestines are between the tip of the spinous process and the transverse process on both sides of the 9th, 10th and 11th vertebrae. The anterior small intestine reflexes were palpable bilaterally, and were extremely tender. Posteriorly there seemed to be a thickening of the tissues in the reflex area, which were very sensitive. By steady pressure and slow rotary motion of the index or middle finger, I was able to break through the somatic manifestations in the tissues and interrupt the flow of sensory impulse. Usually the anterior reflex centers are used to diagnose as well as treat; while the posterior centers are for treating only. As improvement occurred, similar change was noted in the small intestine reflexes, as in the colon reflexes. Due to the extreme tenderness in both the small intestine and colon reflexes, very light pressure was used. As tolerance allowed, deeper pressure was exerted, until all palpable and tender reflexes were obliterated. There was steady improvement throughout the treatment program.

Complete understanding of the osteopathic lesion complex has not been comprehended, and the possibility that it will be understood soon is doubtful. We do know that the closer we can come to establishing total functional integrity, the greater the chance of achieving the goal of health. We must evaluate and treat the patient as a whole if we are to return it to normal function.

Discussion

The research work being carried on at Kirksville, and reported by Korr, sheds some light on the relationship between the somatic and autonomic nervous system. Correlation has been shown between what is clinically known as the “osteopathic lesion” and demon-

strable “physiologic lesions.” The basic triad of nervous function (sensory, motor, and autonomic) has been under study. Related patterns of variation in sensory, motor, and autonomic activity were discernible in various segmental levels. Some understanding of the nature of the process by which organized related patterns of the sensory, motor, and autonomic systems was developed. In order to further understand mechanisms, patterns and pathway processes, and pathways of interchange between somatic and autonomic, anatomic studies have been set up. That the eventual finding will prove the somatovisceral reflex, I have no doubt; for clinically it has been proven thousands of times.

An excellent article by Allan Eggleston entitled “Differential Diagnosis of the Cervical and Upper Thoracic Area” brings out in its preliminary remarks the futility of thinking of disease by eponyms, and the many other names that in no way indicate the physiological disturbance. Eggleston quotes Korr frequently, such as these shining examples: non-tropical sprue, arthritis, “flu,” lumbago, slipped disc, labyrinthitis, et cetera, et cetera, to emphasize his points, both as to diagnosis and effectiveness of the biomechanical approach.

“Familiarity with the segmental relationships and their variations provides a tool for accurate differential diagnosis on a remarkably broader basis than that provided through the concept of the single etiologic factor.

“The great splanchnic nerve is formed by the preganglionic fibers of the fifth, sixth, seventh, eighth, and ninth white rami, which in conjunction with the vagus forms the celiac plexus. The balance of influence of the sympathetic and parasympathetic effects through the celiac plexus determines the activity of the gastrointestinal tract, the liver, pancreas, spleen, kidney and bladder. The adrenal medulla receives innervation through a preganglionic sympathetic fiber.

“Lesioning of a spinal segment causes excessive activity of the sweat glands supplied by the sympathetics of that segment. This was clearly demonstrated in the Kirksville Laboratory when a subject under climate-controlled conditions, at physiologic and psychologic [*sic*] rest was shown to have over ten times as many active sweat glands in a dermatone supplied from a lesioned segment as in a dermatone supplied from a non-lesioned segment. Any stimulation of the subject by pain, pressure, thermal or psychic stimuli caused an explosive response in the facilitated dermatone while

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*Owens’ terminology for tissue change at reflex center.

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the non-facilitated paralleled area showed but minimal increase of activity.

“It is logical to assume that the same excessive stimulation or excessive inhibition (depending on the sympathetic function in the involved tissue) will follow in the viscera and endocrines in the presence of biochemical stress. If this be so, a careful analysis of the spinal pattern, evaluated by knowledge of the sympathetic distribution and effect, provides a means of differential diagnosis peculiar to this profession alone.”⁸

At the level of T9 and T10 through the celiac ganglion comes the sympathetic nerve supply to the small intestine. From the floor of the 4th ventricle comes the 10th cranial nerve which also enters the celiac plexus to supply the parasympathetic nerves to the small intestines. From the sacral division come the parasympathetic nerve supply to the colon. With disturbance of the small intestines, how else can you approach the management but by treating the whole patient?

From these anatomical considerations it becomes obvious that pelvic imbalance related to faulty lower extremity mechanics not only acts as a primary source of disturbed autonomic function, but also provides the basis for an accommodative scoliosis (apexing, for example, at T9 or T10, again at T5, C6 or C7, and eventually influencing articular function at the base of the skull or higher).

As the spinal biomechanical state returns to normal, the following was observed by Louisa Burns: “Normal tone and elasticity return after the lesions have been corrected. ... As the normal spinal relations are maintained, the digestive tracts return more and more rapidly to normal.”⁹

The treatment of Chapman's Reflexes proved to be an important factor in speeding the recovery of the patient with non-tropical sprue. Their use would be an important factor in treating the whole patient in many conditions. To better understand a viscerosomatic reflex, I will quote from a lecture by Mitchell on this subject:

“Viscerosomatic reflexes were first described by Chapman in the early 1920s and later by Owens, who wrote the book *An Endocrine Interpretation of Chapman's Reflexes* in 1927.

“Viscerosomatic reflexes deal with the circuit movements in the autonomic nervous system. There are definite areas of the body that influence the viscera of the body. These areas are not only used in making a diagnosis but are also used in treatment.

“A reflex arc is still one of the marvels of biology. It is a two-way street and may operate in one direction on one occasion and in the opposite direction, or both directions at another time. The purpose of viscerosomatic reflex treatment is the breaking of the reflex arc circuit movement. This is one of the major premises of medicine today through drugs. We are trying to do the same thing through manipulation rather than by medication.

“The anatomical basis of Chapman's Reflexes is that the cells of origin are connected with the white rami communicantes and go to the lateral chain ganglia which are at the sides of the vertebrae and are distributed to all smooth muscle, to all tissue, all cardiac muscle and go via the main nerves, such as the cardiac, splanchnic, etc. They also go over the gray rami communicantes and then through the central division of the autonomic nervous system. It is at termination of these nerves, at the intercostal spaces and

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other areas, where we pick up the ganglioform contractions. Chapman's Reflex is called a receptor organ in the lymphoid tissue.* The conclusion reached is that the visceral pathology sets off a flow of sensory impulses via the sensory autonomic to the related segments of the spinal cord. The synapse to the lateral horn cells have motor impulses to both the viscus and somatic part relayed by the gray rami communicantes."¹⁰

Conclusion

Since this paper is a discussion of the treatment of a case of non-tropical sprue, both medically and osteopathically, I feel it should be repeated that the patient recovered completely under osteopathic care and received only temporary symptomatic relief under medical care.

There are many and varied ideas as to exactly what non-tropical sprue is, what causes it, and how it should be treated. I feel that non-tropical sprue is a separate and distinct entity with definite clinical signs and symptoms except as set forth earlier in this paper. As non-tropical sprue, in my opinion, is a disease of malabsorption of proteins and fats, and probably carbohydrates, vitamins, minerals, and water as well, inquiry must be made into the causes of such disordered intestinal function. The means whereby intestinal homeostasis was disturbed is clearly indicated by the researches of Korr, and others, which we have previously considered in this paper. The manifestations of the disease follow logically, once the basic pathology is grasped. Thus, the stresses of daily physical fatigue, emotional affective states, and generalized inanition took their toll on the neurological segments which were "facilitated" by the postural strain pattern which the patient manifested.

This idea is substantiated by investigations at the Kirksville College and reported by Korr. "Investigations conducted at the Kirksville College have indicated that the musculoskeletal stress initiates, or is associated with, unbalanced streams of impulses entering the central nervous system, and that these have the effect of upsetting the delicate balance of that part of the nervous system with which the lesioned part is most directly connected. This was first demonstrated for the muscular or motor component by Denslow and his colleagues in the early 1940s. He demonstrated that the segments which are in lesion, as determined by subjective clinical criteria commonly utilized by him and many other osteopathic physicians (tissue-texture abnormality and deep hyperalgesia), were objectively distinguished by physiologic criteria of motor activity.

"Segmental motor reflex thresholds were determined by measuring, in kilograms, the amount of pressure applied to the spinous process of each segment which just evokes contraction of the para-verte-

bral muscles at that segmental level. Muscular contractions were detected and evaluated by electromyographic recording. Lesioned segments invariably required weaker stimuli than did non-lesioned segments. The lesioned segment was therefore said to be characterized by lowered motor reflex thresholds—the more severe the lesion the lower the threshold.

"In a later study, Denslow, Korr and Krems demonstrated the diffuse and remote stimuli, including those from the higher centers, and stimuli that occur in normal life, preferentially excited the pathways to para-vertebral muscles of the lesioned segments. Responses occurred in these segments, to impulses from many sources, while at the same time non-lesioned segments remained quiescent. Under conditions in which there was generalized muscular contraction, the activity in the lesioned segments was relatively exaggerated. The easier opening of the motor pathways in lesioned segments suggested that this was a sustained form of the phenomenon of facilitation under study in numerous neurophysiologic laboratories and that, like the experimentally induced form, it too had its origin in a sustained afferent bombardment by impulses from some segmentally located source."¹¹

Non-tropical sprue is treated medically by a low fat, high protein diet, as well as by the use of a general vitamin-mineral product, natural B complex and vitamin B12. While this is considered a medical treatment, it is in fact only an attempt at incomplete nutritional management and not truly medicinal care. Comprehensive osteopathic management includes nutrition, both supportive and corrective, manipulative therapy to restore biomechanical integrity, and correction of the viscerosomatic component with Chapman's Reflexes.

To summarize: Non-tropical sprue is in fact a disease of tissue starvation, secondary to the body's reaction to disturbed biomechanics; a clinical manifestation demonstrating tissue breakdown or malfunction; a part of the greater osteopathic lesion complex.

Management is primarily one of re-establishing total body mechanics by manipulative methods, including the use of viscerosomatic (Chapman's) reflexes to re-balance the sympathetic and parasympathetic influence on the gastrointestinal tract. Adjunctive measures are correction of dietary habits with addition of therapeutic B complex, B12 and a balanced vitamin-mineral formulation.

In conclusion, an effort has been made in this article to differentiate between non-tropical sprue and other nutritional diseases. The different methods used in diagnosing this disease have been used to show that treating only clinical signs and symptoms of non-tropical

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sprue, or any disease, cannot fully eradicate the ailment. With osteopathic care, the body is treated in its entirety to fully eliminate the condition. There is no other treatment for non-tropical sprue except by osteopathic means. Eggleston states most appropriately my feelings in the following quote: "I repeat my belief that there is no field of therapy in which the intelligent application of the science of osteopathy will not improve the results obtained by any other method of treatment."¹²

References

1. Cecil and Loeb.
2. No. 294648, p. 1. Consulting Bureau Service. Wollaeger, Eric E. and Green, Paul A. "Idiopathic Non-tropical Sprue (Malabsorption Syndrome)," *American Journal of Gastroenterology*, 35:569, June 1961.
3. No. 265671, p. 1. Consulting Bureau Service. Comfort, Mandred W. and Wollaeger, Eric E. "Nontropical Sprue: Pathologic Physiology, Diagnosis and Therapy," *A.M.A. Archives of Internal Medicine*, 98:807, December 1950.
4. Refer to No. 3, pp. 10-14.
5. Refer to No. 3, pp. 14-18.
6. Eggleston, Allan A. "Differential Diagnosis of the Cervical and Upper Thoracic Area," *Academy of Applied Osteopathy, 1961 Yearbook*, p. 21.
7. Refer to No. 6, p. 22.
8. Refer to No. 6, pp. 22-23.
9. Castlio, Y. "Principles of Osteopathy," *K. C. O. S., Kansas City, Missouri*, 1932, p. 120/
10. Mitchell, Fred L. Lecture, Badger Academy of Applied Osteopathy, April 1962.
11. Korr, Irvin M. "Symposium on the Functional Implications of Segmental Facilitation, Part I, The Concept of Facilitation and its Origins," *Academy of Applied Osteopathy 1960 Yearbook*, pp. 70-71.

12. Eggleston, Allan A. "Progressive Osteopathy," *Academy of Applied Osteopathy, 1950 Yearbook*, p. 10.

Additional Resources

13. Spies, Tom D., Suarez, R. M., Garcia Lopez, G., Milanes, F., Stone, R. E., Lopez Toca, R., Aramburu, T., Kartus, S.: *J. Am. Med. Assn.*, 139, 521, 1949.
14. Thaysen, T. E. Hess: *Non-tropical Sprue, A study in Idiopathic Steatorrhea*, New York, Oxford University Press, 1932.
15. Buchan, D. J., Marko, A. M., and Gerrard, J. W.: "Malabsorption Syndrome: Its Treatment with Gluten Free Diet," *Canad. M.A. J.*, 79:227-230, 1958.
16. Sleisenger, M. H.: "Clinical and metabolic studies in non-tropical sprue." *New Engl. J. Med.*, 265:49, 1961.
17. Rubin, C. E., Brandborg, L. L., Phelps, P. C., Taylor, H. C., Jr.: "Studies of celiac disease. I. Apparent identical and specific nature of duodenal and proximal jejunal lesion in celiac disease and idiopathic sprue." *Gastroenterology*, 38:28, 1960.
18. Wang, C. I., Bossak, E. T.; "Hemorrhagic manifestations in idiopathic sprue." *J. Mount Sinai Hosp.*, N. Y., 24:317, 1957.
19. Moore, M. J., Strickland, W. H., Prichard, R. W.: "Sprue with bleeding from hypoprothrombinemia." *Arch. Intern. Med.* 97:814, 1956.
20. Hartley, J.: "Osseous changes and fractures in the malabsorption syndrome." *J. Mount Sinai Hosp.*, N. Y., 24:346, 1957.
21. Juergens, J. L.: "Severe osteomalacia associated with occult steatorrhea due to non-tropical sprue." *Arch. Intern. Med.*, 98:744, 1956.
22. Spitzer, R., Ryan, J. A.: "Belated diagnosis of steatorrhea." *Canad. Med. Ass. J.*, 85:656, 1961.
23. Carter, D., Shelden, W., Walker, C.: "Inheritance of celiac disease." *Ann. Hum. Genet.*, 23:266, 1959.
24. Spies, Tom D., Stone, R. E., Suarez, R. M., Garcia Lopez, G., Lopez Toca, R., Reboredo, A.: "Antianemic properties of reaction products of vitamin B12 and the intrinsic factor." *J. A. M.A.*, 151:1264-1266, 1953. ■