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Editorial

Commentary: Metabolic syndrome and the hepatorenal reflex

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EDITOR'S NOTE

Surgical Neurology International (SNI) is publishing a paper by Mike Wider, a physiologist, on the cause of the metabolic syndrome, one of the major disease syndromes affecting people around the world and associated with the world epidemic of obesity. His hypothesis is that the autonomic nervous system (ANS) is a central component of this disease. He has written a short summary of his paper below for our readers, who only have the time for the capsule summary of his idea.

The 21st century will see the development of research and neurosurgical specialties on the influence of the central nervous system (CNS) on diseases in many organ systems. As neurosurgeons, we see this relationship in the electrocardiogram (EKG) and cardiac changes after subarachnoid hemorrhage (SAH) and in neurogenic pulmonary edema associated with a sudden increase in posterior fossa pressure. The CNS and ANS play a role in addiction syndromes, and functional disorders that we read about. The ANS, which we all learned about in medical school and then forgot, plays a central role in many diseases. The paper below is one more example. With this new knowledge, neurosurgeons, and neurosurgery departments will develop relationships with many other departments. Stereotactic Neurosurgery will become central to the treatment of many of these diseases.

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Obesity is increasing rapidly on a global scale and is regarded as an epidemic due to the broad spectrum of associated comorbidities requiring costly medical care and lost work time. The article "Metabolic syndrome and the hepatorenal reflex" in this issue of SNI is a review of the physiology at work in normal gastrointestinal function and its role in producing the medical problems associated with obesity when put into overdrive.

Obesity is accompanied by a number of medical conditions that are limited or cured by weight loss including polycystic ovary syndrome, sleep apnea, hypertension, glucose intolerance, cardiovascular disease, and hypercholesterolemia, as well as shortened lifespan. Metabolic syndrome (MetS) is a clinical designation for those patients who are obese and have hypertension along with elevated "bad" cholesterol and high blood glucose. These patients have increased levels of insulin but are resistant to many of its effects including its actions in lowering blood sugar and metabolizing fat. While much is known about the impact of insulin resistance on the morbidities associated with obesity and the potential role of gastrointestinal and adipose tissue hormones, the

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underlying cause of MetS is still very unclear. What is known is that not all obese patients have MetS, and MetS is sometimes seen in lean individuals, indicating that the level of body fat is not the cause of this condition.

During the last two decades, work by research groups around the world has identified increased sympathetic nerve activity in the muscles and kidneys in MetS patients as well as in animal models. It has become clear that this elevated sympathetic tone can cause many of the comorbidities, but it is as yet unclear as to what could produce the sympathetic overdrive in MetS. The author cites the extensive evidence for immediate remission of MetS in a significant number of patients following a decrease in stimulation of the stomach and duodenum by food. A number of large studies are referenced following patients for years after placement of a plastic sleeve in the proximal small intestine or subsequent to bariatric surgery (removal of part of the stomach or diversion of the gastrointestinal flow from the proximal stomach to the mid small intestine). Many patients have remission of MetS in a matter of days following these procedures, all of which prevent the partially digested food from contacting the mucosa of the stomach and duodenum.

The comorbidities of MetS are also cured or reduced in many patients in a short timeframe following the start of a calorie restricted diet, long before any weight loss. The author argues that it is the reduction of nutrient contact with the gastrointestinal mucosa by bariatric surgery, plastic sleeve, or diet that is curative. MetS and type 2 diabetes patients have been shown to have low hepatic ATP levels that indicates limited oxygen delivery to the liver, and the paper makes the case that it is the chronically high intestinal oxygen demand from excessive eating that reduces oxygen delivery and lowers ATP production.

Reduced oxygen in the liver is known to cause a sympathetic reflex to the kidneys referred to as the "hepatorenal reflex," which results in constriction of the renal blood vessels and stimulation of the renin angiotensin system that raises blood pressure and further increases sympathetic tone. The author postulates that lowering the intestinal oxygen demand, by limiting contact of nutrient with the gastrointestinal mucosa, increases O_2 delivery and reduces the sympathetic tone, resulting in remission of the comorbidities.

The immediate elimination of the comorbidities in many patients, independent of weight loss, is presented as a strong argument for the role of eating rather than excess fat in causing MetS. Future research will be required to determine why many obese patients who also eat too much, do not develop MetS but are "metabolically healthy." Could it be an issue inherent in the liver vasculature or fat accumulation in liver cells restricting blood flow? Could it be a lower biologic threshold for the hepatorenal reflex or perhaps higher intestinal oxygen demand? Only clinical studies will eventually uncover the answer.