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Massage Today

CranioSacrally Speaking: An Obscure Side-Effect of Obesity

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The sad passing in December of future NFL hall-of-famer Reggie White illuminates an obscure side-effect of obesity that also gives us some fascinating insight into the human body. White, who died at 43, topped out at weights exceeding 290 in the course of his career. While the results of his death weren't conclusive at the time of this writing, the coroner cited sleep apnea as having played a possible role.

Not to be confused with central sleep apnea secondary to brain dysfunction, White's type of sleep apnea is most common among men of large body mass. Like snoring, this sleep apnea is often secondary to the fatty enlargement of tissues in the nasal air passages at the back and upper areas of the throat. These patterns are generally characterized by gasping inhalations followed by long pauses during which there is little or no exchange of air via the airways into the bronchi and lungs.

Let's dig into the subject a little deeper and see what's behind all this. Involuntary respiration is controlled by nerve cells/neurons in the medulla oblongata located in the skull just above the upper end of the spinal cord. These nerve cells get their instructions from the pons, which is higher in the brain. The pons gets its information from several other brain centers then sorts out all the little details to develop regulations for breathing. I suspect some of the pons' incoming messages originate in the fat-enlarged tissues of the nose and mouth airways. These messages may then cause the pons to periodically hold back normal rhythmical inhalations.

Taken to the extreme, respiratory arrhythmias secondary to abnormally fatty tissues can take sleep apnea to the point that increased back pressure in the lungs can produce some degree of right-sided heart failure. The result is cyanosis, a bluish discoloration of the skin and mucous membranes that first appears in nail beds and lips. The discoloration comes from a reduced level of oxygen in the blood secondary to the compromised breathing that began with snoring and sleep apnea.

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Now let's look at a molecule called "nitric oxide" (NO). This

gaseous substance is attracted to lipid (fat) molecules. It is moderately reactive compared to inert gases such as helium, neon and argon, which do not react with other atoms, ions or molecules. What nitric oxide does within our bodies is remarkable. It has a great deal to do with the flow of blood through our vascular systems. With every heartbeat, a puff of nitric oxide gas is released from the endothelial cells where a great deal of it is stored. Endothelial cells line all our blood vessels, including arteries, veins, arterioles, venules and capillaries. So nitric oxide is released in some amount in every blood vessel.

The process by which nitric oxide causes blood vessel relaxation and dilation is somewhat complicated. It goes something like this: The puff of nitric oxide that's released from the endothelial cells of the blood vessels goes directly to the red blood cells (RBCs) where the nitric oxide molecules become attached to hemoglobin (Hb). The nitric oxide remains attached to the hemoglobin as assessments are made regarding how much oxygen is available to the body for its cellular needs.

Low oxygen levels cause more nitric oxide to be released from the hemoglobin. Higher oxygen levels cause hemoglobin to retain a higher percentage of nitric oxide. That makes good sense. When oxygen is low, nitric oxide causes blood vessels to dilate and deliver more blood to the tissues to increase the amount of oxygen getting to the cells. Incidentally, as cells take in available oxygen, most of it goes to intracellular mitochondria. There are thousands of mitochondrial organelles in the cytoplasm of each cell. The mitochondria make the energy that's required by every cell.

Back to our nitric oxide molecule travels. As nitric oxide is released by the hemoglobin, it emerges from the RBC after combining with the amino acid "cysteine" to form S-nitrosothial. In this form, nitric oxide will not be reattached by the hemoglobin as it travels through RBC cytoplasm. The nitric oxide is ushered out of the cell by specific proteins attached to the RBC membrane. It then enters the blood serum and the endothelial cells where the molecules are stored as nitric oxide. When it's time for the blood vessel to dilate, the nitric oxide goes to the smooth muscles in the blood vessel walls and causes the muscles to relax. This relaxation allows the blood vessels to dilate and pass more blood at a lowered blood pressure.

What does all this have to do with obesity? It was recently discovered that our paranasal sinuses produce a lot of nitric oxide. When nitric oxide is inhaled through the nasal airway, it gets into the lungs and increases the amount of oxygen that gets into the blood that is circulating through the lungs. The clearer the nasal passages, the more nitric oxide will be inhaled into the lung tissue. Hence, the more efficiently the oxygen will be absorbed via the lungs into the body vasculature, which then delivers the oxygen to

all body cells.

Obesity often causes sleep apnea and snoring, which indicates a blockage of nasal airways. The nitric oxide delivery to the lungs then is reduced, as is oxygen absorbed through the lungs. Lowered oxygen levels in the body signal that the tissues need more blood to supply the oxygen. The physiological response is to raise the blood pressure to increase blood flow and improve oxygen supplies to tissues. Hence, high blood pressure occurs because nitric oxide isn't getting into the lungs effectively.

Hopefully, the passing of Reggie White will wake others up to a lesser-known but potentially deadly side effect of obesity.

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