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By Dr. John C. Lowe, for Thyroid-UK 2006.
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Air Hunger to Death : Breathing Problems of Hypothyroid Patients

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Air Hunger to Death : Breathing Problems of Hypothyroid Patients.

“Last week I consulted long distance with a young hypothyroid woman who had a distressing symptom-trouble getting a deep enough breath. She told me, I just can't seem to get in enough air. She said her doctor doesn't believe her hypothyroidism is the cause of her breathing problem”.

I referred the patient to several sections of my book, The Metabolic Treatment of Fibromyalgia [1]. In those sections, I explain that laboured breathing is a troubling symptom for which some hypothyroid patients seek medical care [2, 11, 12, 13, 15-24].

Medical journals contain many reports of some patients with hypothyroidism or thyroid hormone resistance having laboured breathing. The difficult breathing is called air hunger or dyspnoea. Air hunger, however, isn't the worst of the breathing troubles of some patients with too little thyroid hormone regulation.

In The Metabolic Treatment of Fibromyalgia [1], I explain various mechanisms of these patients air hunger, all of which result from too little thyroid hormone regulation. One mechanism is weakness of their respiratory muscles, including the diaphragm [22-23].

Researchers call this weakness of respiratory muscles hypothyroid myopathy.

Another mechanism is impairment of the phrenic nerves that regulate contractions of the diaphragm. When impaired, these nerves send too few signals to the diaphragm for it to contract normally [23].

A third mechanism is decreased central drive of respiration [34]. This means that the brain centers that regulate breathing are impaired by too little thyroid hormone regulation.

Still another mechanism is abnormal heart and lung function [14-15, 20].

Some patients suffer from air hunger only when they exert themselves; others do so even at rest.

The young hypothyroid woman I consulted with had been on T4-replacement for three months. She had been troubled with hypothyroid symptoms, including air hunger, for a year before her doctor diagnosed her hypothyroidism. The T4-replacement had slightly improved her fatigue and dry skin, but not her air hunger. Her dose of T4 was 100 mcg (0.1 mg) per day.

I was not surprised that this patient was still suffering from hypothyroid symptoms, including air hunger.

To illustrate, consider the study data reported by a group of endocrinologists in 1997 [30].

They measured nine hypothyroid patients TSH levels and their resting metabolic rates, and compared them to their doses of T4. I analysed their data from a perspective different from theirs.

At one point in the study, six of the endocrinologists nine patients were using 100 mcg (0.1 mg) of T4. For three of these patients, this dose provided normal metabolic rates, although the rates of two of them were low normal. These two patients metabolic rates were 7% and 9% below the calculated average rate. (We consider a rate abnormally low only if its

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10% or more below the calculated average rate). For the other three patients on 100 mcg of T4, the dose was woefully inadequate: One patient's metabolic rate was 18% below normal, another was 23% below, and a third patient's was 26% below normal.

The endocrinologists didn't report this finding. I found it simply by looking at their data from a different perspective. From this vantage point, their own data show that some hypothyroid patients are kept hypometabolic on 100 mcg of T4.

We find this regularly in our clinic when we measure the metabolic rates of patients on T4-replacement. And 100 mcg of T4 is obviously not working for this young woman, whose main troubling symptom is air hunger.

Doctor-Caused Lifelong Breathing Problem. Yesterday I consulted long distance with a woman in her seventies who has had breathing troubles far worse than those of the young woman I mentioned above.

In the early 1970s – before the Tyranny of the TSH[32] began – an endocrinologist treated her for hypothyroidism. Through trial-and-error, he found that her safe and effective dose of Armour Thyroid was 6 grains (360 mg).

She remained well on that dose until, years later, a second endocrinologist found her TSH level suppressed. The patient had no symptoms of overstimulation. Despite that, this endocrinologist insisted that she reduce her dose of Armour. She complied, lowering it to 4.5 grains (270 mg).

Soon, she told me, I simply was not functional. I was gasping for air and coughing so bad that I felt like I was damaging my lungs. To breathe, she had to sit upright on a sofa without leaning her head back to rest. This went on for eight weeks.

When her daughter, a nurse, phoned the endocrinologist, he denied that the patient's breathing problem was related to her lower dose of Armour. He advised her to consult an ear, nose, and throat specialist. She saw a lung specialist instead, and he prescribed 40 mg of cortisone and an inhaler. The inhaler enabled her to get through her days. But even with the help of the inhaler, she couldn't walk up a short flight of stairs without gasping for air. Her voice was

so badly affected that she could no longer sing, which she had always enjoyed. I could only croak, she said.

In March of 2004, she located the original endocrinologist who had put her on 6 grains of Armour in the early 1970s. Her put her back on that dose. It took me the rest of the year to get off the cortisone and get well, she told me. Before the second endocrinologist lowered my Armour dose, I'd never had any asthma or other breathing problems. Now I'm left with a lifelong breathing problem. She's largely over the breathing problems that began on the lower dose, but she still has to use an inhaler one or two times each day.

I admire this woman's strength of character in wresting control of her health away from her doctors. Some of them have recently tried to persuade her to lower her dose again. They simply don't listen when I tell them what happened to me on the lower dose. I feel that I'm up against a wall of idiocy, she said. I don't have too many years left to fool around with their book theories, and I'm not going to let them kill me. The tragic fact is, of course, that many doctors today would sacrifice her health and, indeed, even her life. Like most fanatics, to these doctors any price the patient might pay is worth achieving their obsessive goal — keeping the patient's TSH in range.

Asthma. Some inhalant patients use to relieve their asthmatic symptoms work by stimulating beta2-adrenergic receptors in the bronchial tubes.[28] Stimulating the receptors dilates the tubes and constricts blood vessels in their lining, which dries mucous secretions. These effects of the inhalants relieve shortness of breath, coughing, and wheezing respiration.[25,p.207]

An alternative to using the inhalers is to increase the number of beta2-adrenergic receptors in the bronchial tubes by taking an effective daily dose of thyroid hormone.[33]

T3 is especially useful for this purpose. In 1991, Egyptian researchers treated 23 asthmatic children with T3.[29]

The children weren't hypothyroid. During the 30 days of treatment, they continued to use their usual anti-asthma drugs as needed, but they reduced their doses as low as they could.

The researchers wrote, They [the children] all

reported at the end of the 30 days an obvious subjective improvement of their asthmatic conditions with a decrease in the number of exacerbations. Seven patients stopped their usual anti-asthmatic medicines, being maintained on T3 only and 3 have decreased the amount of bronchodilators needed. A significant improvement of pulmonary function tests was noted in all patients.

According to the researchers, All patients tolerated well the T3 regimen without any adverse effect. They concluded that T3 induced beneficial effects: T3 proves to be a useful adjuvant to classic anti-asthma therapy, and may reduce the amount of bronchodilators needed.

Other researchers reported that patients with asthma became hypothyroid from using iodine-containing expectorants. After becoming hypothyroid, their asthma worsened. The worsening of the asthma was relieved, however, when the patients stopped using the expectorants and their thyroid function became normal again [27].

Another group of researchers took hypothyroid children off thyroid hormone for a month. By the end of the month, the children's bronchial tubes became more reactive to antigens. (Heightened reactivity of the bronchial tubes to antigens, of course, is the basis of allergy-induced asthma.) The children then resumed their thyroid hormone therapy. Their bronchial tubes, however, remained more reactive to antigens for two more months. Finally, after twenty more days, their bronchial tubes became normally reactive to antigens [26].

Respiratory Failure and Other Breathing Problems. In 2004, doctors described a 36-year-old male admitted to the hospital for progressive respiratory failure.[3] His chest X-ray and CT scan were normal, but he had a severely slow heart rate (bradycardia) and his cognitive function was slow. His TSH was over 150 microU/ml (in the US, the current upper limit for a normal TSH is 2.5 or 3.03, depending on which authority one consults). After the man began thyroid hormone treatment, his respiratory function steadily improved.

The doctors who reported this patients case listed five other respiratory disorders common among hypothyroid patients: dysfunction of the

diaphragm, poor respiration from low central nervous system drive, obstruction of the airways, pleural effusion, and sleep apnea. They advised doctors to evaluate patients with respiratory problems for hypothyroidism.

Anaesthesiologists reported the case of a 58-year old man who was severely hypothyroid. They wrote that his hypothyroidism was the cause of ventilatory failure. They described his 3-to-4 breaths per minute after surgery as an exceedingly low respiratory rate.

The volume of air he took in each minute was low. Despite his low oxygen intake overall, he developed primary acute respiratory alkalosis (carbon dioxide loss from hyperventilation) and associated hypocarbia (abnormally low carbon dioxide in arteries).

The anaesthesiologists cautioned that severely hypothyroid patients are at risk for adverse events around the time of surgery. They are more sensitive to anesthetics, have a higher incidence of surgery-related cardiovascular disease, and have an increased risk for ventilatory failure following surgery [34].

Hypothyroidism doesn't have to be severe to lead to weak breathing responses to increased carbon dioxide in the body. Researchers published a report on ten patients with hypothyroidism less severe than that of the patient in the above paragraph. Two of these patients had blunted ventilatory responses to hypercapnea. This means that high carbon dioxide levels didn't provoke a normal increase in breathing to lower the carbon dioxide levels. The patients breathing responses to increased carbon dioxide improved when they underwent thyroid hormone therapy [35].

Hypothyroidism and Obstructive Sleep Apnea. Hudgel recommended thyroid hormone as a ventilatory stimulant for hypothyroid patients with sleep-disordered breathing [5]. He explained that in 1964, Massumi and Winnacker reported the association of sleep-disordered breathing with hypothyroidism [6]. Their report was about two hypothyroid patients who had obstructive sleep apnea. In 1981, Orr reported the results of polysomnograms (recordings of physiology during sleep) of three hypothyroid patients [7]. The tests showed repetitive obstructive apneas during sleep. Further testing

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by Rajagopal tightly linked hypothyroidism to obstructive sleep apnea [8]. Nine of his 11 consecutive hypothyroid patients had apnea.

Not all hypothyroid patients, of course, have obstructive sleep apnea. Lin found that among 25 hypothyroid patients, only 25% had sleep apnea [9]. The patients who had apnea were older and more obese than the patients without apnea. And not all patients with obstructive sleep apnea are hypothyroid. Lin found that only 3% of patients with apnea were hypothyroid [9].

But for most hypothyroid patients who do have sleep apnea, the consequences can be troubling. [10] For example, because they don't sleep deeply long enough, they can be severely fatigued throughout the day. And as the section below shows, for at least one hypothyroid patient, apnea wasn't just troubling; it was fatal.

First Reported Death From Hypothyroidism and Sleep Apnea. In 1999, doctors reported the first case of a hypothyroid patient who suddenly died from sleep apnea [4]. The patient was a 48-year-old man who was short and slightly overweight. He had never been treated for his hypothyroidism.

For several years, the patient suffered from air hunger at night. In the previous few weeks, his air hunger had worsened. In the hospital, doctors noted that he was lucid, but his mental and physical functions were slow. They found that he had a multinodular goiter that had moved his windpipe (trachea) from its normal position. His tongue was enlarged, and his pharynx (the cavity that connects the mouth and nasal passages to the esophagus) was swollen with edema. His blood was adequately saturated with oxygen at 97%.

Doctors treated the man with T3 and hydrocortisone. They doubted that he had sleep apnea syndrome (SAS), but they applied pulse oximetry. (Pulse oximetry is a monitor, usually with a finger sensor, used during anesthesia and critical care. It measures oxygen saturation of

arterial blood.) The man suddenly died seven hours later.

The oximetry record showed that over the seven-hour period, the patient had prolonged episodes of sleep apnea. These episodes had caused deep drops in oxygen saturation of his blood. The doctors who reported the man's death urged other doctors to promptly diagnose hypothyroidism that's associated with obstructive sleep apnea. They noted that the condition can become serious and require intensive care with continuous nasal airway positive pressure. They wrote that some patients may need tracheal intubation with assisted ventilation. They cautioned, Continuous cardiac monitoring should also be carried out, given the risk for acute coronary complications and ventricular arrhythmias in the early phases of substitutive therapy with thyroid hormone.

Conclusion:

The studies I've cited in this report show that too little thyroid hormone regulation can cause a variety of breathing problems. These range from mere frustrating air hunger to death from sleep apnea.

If you have a breathing problem and your doctor can't find the cause, ask him or her to evaluate you for hypothyroidism or thyroid hormone resistance. If you're being treated for hypothyroidism with T4-replacement, ask your doctor to consider that this thyroid hormone therapy leaves almost 50% of patients suffering from chronic hypothyroid symptoms [31].

Ask your doctor to also consider that you may need to switch to a more effective therapy such as a T4/T3 combination product or T3 alone. At minimum, if your doctor isn't aware that too little thyroid hormone regulation can cause breathing problems, share this special report with him or her.

The doctor may use the information not only to relieve your breathing problem, but to help other patients, too.

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