Hyperbaric Oxygen Therapy Research/Trial Proposal

Obesity, Metabolic Disorders and Type 2 Diabetes.

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HBOT Research Proposal – Obesity, Metabolic Disorders and Type 2 Diabetes.

Introduction

To diabetics, the subject of obesity and insulin resistance is of great importance and relevance. The inability to lose weight despite substantive efforts to follow low carbohydrate diets, and fairly active lifestyles, comes as a great disappointment to many millions of diabetics. This becomes a further frustration when medical staff disbelieve these diabetic sufferers who appear to “absorb” weight from the atmosphere, perpetuating a failing metabolic cycle that just seems unbreakable.

This paper applies previous learning in diving, diving medicine, and physiology, hyperbaric oxygen, and diving and decompression theory, as well as food and nutrition related physiology, to the problem. Such application is hypothesised within established and known parameters of physiology as a non-medical person. A parallel is drawn between the known symptoms, underlying causes and effects of diabetes, and with that which is known by hyperbaric doctors, and referred to as a ‘negative side effect’, and an uncanny inverse relationship between the two, and also to the physiology taught to professional divers, diving supervisors and hyperbaric chamber technicians and operators.

It is hypothesised that it is this negative side effect, and its relationship to metabolic consideration in diving, that can effectively be applied to treat obesity in the very overweight, insulin resistance in diabetics and other metabolic disorders, and the retraining or reconditioning of insulin sensitivity at a cellular level. This potentially offers not only effective management of the above conditions but also a potential permanent reversal of intra cellular failures responsible for high hBa1c levels, and unmanageable weight gain and retention, as well as low insulin response and ultimately cell, tissue and organ damage.

Hypothesis

This following hypothesis, which is supported, if not proven by current research, postulates that hyperbaric oxygen therapy can drastically increase insulin sensitivity while replacing those cells that have become ‘lazy’ in their response to insulin, with new stem cells that are then ‘programmed’ to a refreshed, renewed, and sustainable level of insulin sensitivity. The hypothesis further suggests that HBOT, as it is known to do, will increase VO2 rates and as a direct result promote optimised metabolic process in cell mitochondria, allowing the body to effectively manage sugar and fat by process of elevated gluconeogenesis combined with sustained, elevated insulin sensitivity, nullifying the need for drug therapy. The synergistic effect of the two approaches is theorised to manage uncontrollable obesity and insulin resistance and metabolic failures in one treatment regime and reverse the condition.

Hyperbaric Oxygen Therapy – Stating the Obvious

Hyperbaric oxygen therapy is described as the medical administration of pure oxygen or enriched breathing gas mixes at higher than atmospheric (Normobaric) pressure. [1 - Heyboer 2017]

A well-established body of knowledge describes the process and mechanism as one functioning on the principals of pressure differentials and gas tension differentials, inward and outward gradients being determined by tissue gas tension measured against breathing gas tension and the differential between them expressed as a gradient. [2 – USN Diving Manual Revision 7 – 2017]

Inward gradients are evident in the presence of a lower tissue saturation or gas tension when compared with that of a given gas or gas mix. (Nitrogen, Carbon Dioxide, Carbon Monoxide, Oxygen, medical air and so on). Both inert and non-inert gasses are subject to gas to liquid interchange and solvency in the presence of a tension/pressure differential as described by Daltons law of partial pressure [3 – Oxford Reference – 2016] and Henry's law [4 – Oxford Reference – 2016] as it pertains to Dalton's Law stating that the amount of dissolved gas in a liquid is proportional to its partial pressure in the gas phase. The proportionality factor is called the Henry's law constant.

Outward gradients are evident inversely and proportionately to inward gradients when the gas tension or pressure of a gas in a tissue is higher than that of the same gas in a breathing mix or gas.

In diving and decompression theory these are commonly referred to as in-gassing and out-gassing, and will continue in accordance with the above laws until equilibrium or saturation has been achieved or until factors change.

Tissues, (specifically the fluids contained therein), which then consequently have a lower gas tension than blood plasma will in-gas accordingly. The downstream knock on effect is that as plasma achieves a higher oxygen tension, a differential will then exist between plasma and cells (specifically intra cellular fluid). Intracellular fluid will then strive to achieve equilibrium in accordance with the above laws and the gas tension will rise allowing optimal and even 'super charged' cellular functions. This is observed as accelerated healing, accelerated vascularisation (vasculogenesis), decrease of inflammation, increased rate of cell mitosis and angiogenesis. [5 – Thom – 2011] [6 – Boykin et al – 2007]

Hyperbaric Oxygen Therapy is the administration of pure oxygen at increased ambient pressure in a hyperbaric chamber. Commonly referred to as **HBOT** (**H**yper**B**aric **O**xygen **T**herapy).

It is currently used for a wide array of ailments and conditions including problem wounds, [6 – Boykin et al – 2007] [7 – Van Neck et al – 2017] fibromyalgia, [8 – Efrati et al – 2015] Multiple sclerosis, [9 Oxygen and The Brain – The Journey of Our Lifetime – James – 2014] carbon monoxide poisoning,[10 – Weaver – 2014] radiotherapy injuries, [Moen et al – 2012] neural injury [9- James – 2014] [12 – Tel Aviv University – 2013} among others but not the extent that it should be in the opinion of many researchers. HBOT has applications far broader and far more beneficial than currently in use by the medical profession including obesity, type 2 diabetes reversal, cancer cell death, [11 – Moen et al – 2012] conditions of the macular and kidneys etc. Basically, anything requiring improved vascularisation and higher than normal oxygen saturation in blood plasma and consequently tissues, can benefit from

HBOT as a primary or complimentary therapy, including even immune response in the treatment of HIV and other immune deficiency conditions.

While the mechanisms are a bit more complicated than presented above, the purpose of this article is to rather suggest that the very same mechanisms in play, can be directed to benefit individuals rather than be dismissed as side effects when employing HBOT in the few limited applications it is currently indicated for.

Below, accelerated healing and metabolism will be discussed, and at the risk of stating the obvious, highlight a link between metabolic function, exercise, the glucose cycle and volume of oxygen as it relates to basal metabolic rate. Also, how increased oxygen saturation can and does improve all these functions simultaneously along with greater adenosine triphosphate (ATP) function. These functions can, collectively, certainly benefit those suffering from metabolic disorders such as type 2 diabetes, morbid obesity and a general weight retention despite efforts, activity and diet regimes. Speculation will also be made on gene expression, and how it relates to cellular mitosis and the function of stem cells in this process.

There will also be discussion on less considered factors such as air pollution, oxygen transport and how that reduces metabolic function further and how it can be remedied using HBOT.

Volume of oxygen and VO2 Rates

Definition:
VO2 (or oxygen consumption) is a measure of the maximum volume of oxygen that is used by your body [13 – Bruno et al – 2013] to convert the energy from the food you eat into the energy molecules, called adenosine triphosphate (ATP), that your body uses at the cellular level. VO2max (or maximal oxygen consumption) is simply the maximum possible VO2 that a given person can achieve. VO2 and VO2max are important in the context of exercise, because they are a measure of your body's ability to generate ATP, and ATP is the energy source that allows your muscles to continue working while you are exercising. [14 – Knowles – 1980 - quoted]

Divers from both commercial and recreational disciplines are taught extensively about what they know as the ‘Volume of Oxygen’ calculation (VO2). Such teaching is done to demonstrate that consumption of breathing oxygen under pressure, (whether pure or as a component gas in a mix), increases proportionately with an increase in ambient pressure. In other words, the deeper one goes, the more oxygen the body will burn proportionate to the depth. Oxygen is not consumed by metabolism at a constant fixed rate when we add increased ambient pressure to the equation, and we can indeed exceed “VO2 Max” in a hyperbaric environment. From a practical standpoint, this is taught to enable student’s skills for calculating the rate at which oxygen in a closed-circuit rebreather type system will be depleted, allowing for more accuracy in dive planning and profiles based on available oxygen. [2 – USN 2017]

Such Divers and supervisors are also taught this to effectively manage required volumes of oxygen for the treatment of divers, in a hyperbaric Chamber, (usually a DDC - Deck Decompression Chamber), for the treatment of decompression sickness by means of therapeutic treatments, (Rx tables), or the preventative and routine decompression method known as surface decompression or SurdO2. It is also, and more importantly, taught to enable supervisors and chamber technicians to calculate the rate of CO2 production, as a by-product of metabolism, which can potentially render a chamber atmosphere deadly to its occupants in minutes. [15 – Naval Sea Systems Command – 2004]

The spin off from this is, it becomes obvious that the very same oxygen depletion rate, (measurements of CO2 production), in terms of volume is also an indication of increased metabolism. In fact, it could be said that the prime metabolic indicator is the production of CO2. The depletion is not a measure of depletion as it would be calculated in an open circuit situation, where gas density is the primary reason for increased usage, but rather a calculation of how oxygen is used in metabolism, and then breathed out and scrubbed as carbon dioxide, and replaced with a measured amount of new oxygen from the breathing circuit. It must go somewhere, and that somewhere is the metabolic process, since it is not an inert gas and does not simply dissolve into solution to come back out later, as is the case with say, nitrogen for example. Divers are always taught that basal metabolic rate increases when diving. Anecdotes of 'A dive is as good as a run without the exertion' are common. Mostly dismissed as wishful thinking, these claims turn out to be true in fact. A dive is indeed as good as run to some measure, in metabolic terms at least, as will be visited later under the heading 'Exercise'.

By measuring how much oxygen a divers body consumes in a closed-circuit/system environment in terms of flow, allows one to then also measure its volume or weight. The higher consumption rate indicates a higher oxidative function in the body which essentially is an increase in metabolic rate. This is evidenced by the functioning of carbon dioxide scrubbers, which remove the by-product of metabolism – CO2, and an observable increase in atmosphere CO2 content, expressed as a percentage on flow/content meters, and as calculated below by the University of Utah.

Metabolic Rate:

CALCULATIONS The volume (V) of oxygen consumed per unit time is calculated as V = flow rate × (FIO2 - FEO2) where FIO2 is the fractional volume of O2 in the ambient air (assumed to equal 0.2095) and FEO2 is the fractional volume of O2 in the effluent air coming out of the metabolic chamber.

Note that the oxygen analyser records %O2 of the effluent air. Therefore, the specific equation for V in this case is

V = flow rate × (20.95 - %O2) ÷ 100 Flow rate in these experiments is 500 ml/min.

Volumes need to be standardized to 0°C (i.e., 273°K) and 760 mmHg of pressure. The volume measurements used in these experiments are made across a range of different temperatures, but the pressure at which the measurements are made is always approximately 640 mmHg because of the altitude at U of U. Therefore, standardized volumes can be calculated as VSTPD = V × [229.9 ÷ (273 + °C)] where °C is the temperature at which the %O2 measurement is made

The above calculations are for all intents and purposes the same as diving calculations used in saturation diving, air diving, rebreather diving and consumption in chambers and closed systems, with the exception of accounting for increased pressure. The fact that pressure is

accounted for though, indicates that VO2 is subject to ambient pressure. It is simply a method of measuring how much CO2 is produced from oxygen metabolism.

Since the oxygen depletion is calculable, and can be measured, it is concluded that metabolic function increase is a direct result of breathing oxygen at higher than Normobaric conditions.

Metabolism

Basal metabolic rate is an estimate of how many calories one would burn, or rate of energy expenditure if doing nothing but rest per unit time. [17 – McNab – 1997] It represents the minimum amount of energy needed to keep the body functioning, including breathing and keeping the heart beating.

Basal metabolic rate is dependent on oxidative metabolism which in turn is driven by inward gradients of oxygen. The more oxygen being driven into cells the higher the metabolic rate of that cell as established above. Conversely the less pressure driving oxygen into cells reduces metabolic function as is observed in altitude sickness and hypoxia. [17 – Coote – 1995]

Consciousness is maintained above partial pressures of about 0,16 ATA partial pressure of oxygen (ppO2). An individual remains conscious due to the pressure of the gas not the percentage. The pressure determines the amount of oxygen transported in blood. Evident when flying and in other altitude related activities. Above a certain height, an individual will fall into unconsciousness because the pressure of oxygen (ppO2) drops below a point required to maintain blood saturation (SaO2) of around 90% or more limiting brain function.

HBOT works on the premise that an increase in pressure increases the inward gradient of oxygen and in turn increases the tension of oxygen in blood plasma and in a knock effect, also the tissues that blood feeds. This creates a differential between oxygen tension in blood plasma and cellular fluid, or an ‘inward gradient’, resulting in a desire for equilibrium and inward gassing and solvency of oxygen into cellular fluid and tissue, (Henry’s Law).

As the tissues saturate with oxygen to higher tension levels, metabolic process increases facilitating improved cellular function allowing for what is known as accelerated healing, optimal mitosis, improved vasculogenesis and improved angiogenesis, etc. [6 – Boykin et al – 2007] [7 – Van Neck et al – 2017]

Metabolism doesn’t only provide the body with a means to heal however. It is also part of the glucose cycle which is where type 2 diabetes comes into it. By stimulating the metabolism, one also stimulates the glucose cycle which is compromised in metabolic disorders such as type 2 diabetes and obesity.

The glucose cycle including glycogenesis and gluconeogenesis is included in this.

As discussed above an increase in Vo2 rate is indicative of an increase in metabolic rate. Even if it is only basal rate that increases the body still functions at higher than normal rates. Much as it does during exercise.

Exercise

Sports medicine determines that aerobic exercise increases metabolism by increasing muscle function and strength as well as mitochondrial use of oxygen. The mitochondrial need for more oxygen during periods of exertion stimulates a higher than normal oxygen intake by means of increased heart rate and respiration. The higher the oxygen consumption the higher the metabolic rate.

Exercise does not however allow for further saturation of plasma. This much is governed by pressure gradients as discussed. In the absence of any pressure or tension differential plasma will only become as saturated as ambient pressure allows. Henry's law dictates that gas solubility in a liquid is determined by the ambient pressure applied to that gas's partial pressure as it compares to the tension of that gas in the liquid. Haemoglobin will saturate to the full extent however, which is not far of its normal SAo2 condition in healthy people. (more on this later) Haemoglobin can only carry a finite number of oxygen molecules however. Increased breathing rate and blood flow facilitate a higher VO2 consumption and in turn a higher metabolism during and shortly after exercise. Sound familiar?

Hyperbaric oxygen has been established as a metabolic stimulator during and shortly after treatment session. It's the reason it works. [18 – Fujita et al – 2012] While most treatments are seeking optimised vascularisation or improved mitosis for accelerated healing of damaged capillary structures and tissue, it also stimulates metabolism, which is overlooked when considering the glucose cycle and obesity as well as metabolic disorders such as type 2 diabetes. It is in fact treated as a side effect to be cautious of. Granted the number of patients suffering from type 2 diabetes in the UK alone is far too high to treat exclusively by means of HBOT but it should not be overlooked for people who are unable to take glucose regulators such as metformin and those unable to exercise adequately for other medical reasons.

In both HBOT and exercise, the body returns to normal function relatively quickly after treatment or exertion. The effect is not permanent. Consequently, it can be said that a single workout is not going to change much but rather a regular regime will maintain overall higher average metabolic function allowing the body's own glucose management to operate more optimally.

The same can be suggested for HBOT treatments. A regular regime of treatments should indeed have a similar result as regular exercise and potentially even more so given that plasma saturates in HBOT treatments whereas it does not during exercise. Exercise relies on increased respiration and heart rate to deliver more oxygen to cells.

It is suggested that HBOT treatments could be a suitable and even superior replacement for physical exertion. This is not to suggest that the population in general should have chamber sessions rather than exercising, but rather to suggest that those individuals unable to exercise can benefit immensely, and in the same manner other people benefit from exercise, from a similar metabolic result with regular HBOT treatments. Restoration of normal glucose metabolism usually normalizes glycogen metabolism, as well.

This provides a potential pathway to breaking the obesity / type 2 diabetes recurring circle of mutual support of each other. Less adipose tissue will result in less insulin absorption and consequently better insulin response, and effectively better fat management by the body. At that point the body should begin to manage its own glucose cycle and effective use of glycogen.

When diagnosed, many diabetics are informed that a diabetic body is great at making fat. We are excellent fat makes, but very bad at eradicating it. As suggested above, and explained further below, breaking the seemingly one-way fat making cycle for many diabetics is key to improving the reversal of glycogenesis (creation of glycogen and ultimate consequential increase in fat mass) and how it relates to the Cori cycle.

It is suggested, and supported by Fujita et al 2012, in their paper "Effects of Hyperbaric Oxygen on Metabolic Capacity of the Skeletal Muscle in Type 2 Diabetic Rats with Obesity ", [18] that HBOT can do this at the root of the cause. Imagine even then exercising under pressure or shortly afterward as many athletes do.

Glycogenesis, Gluconeogenesis and Glyceroneogenisis

Borrowing from past college nutrition lectures, where microbiology and physiology were also studied, and from online content for wording, I present brief definitions below. These are presented as a layman with quoted secondary citation.

Glycogenesis [19 – Encyclopaedia Britannica] - can be described as the process of glycogen creation, in which glucose molecules are added to chains of glycogen for storage. This process is activated during rest periods following the Cori Cycle in the liver and also activated by insulin in response to high glucose levels, for example after a carbohydrate-containing meal. IE Placing polysaccharide into cells which forms glucose on hydrolysis. The making of glycogen from sugar.

Gluconeogenesis (**GNG**) [20 – Silva et al – 2009] is a metabolic pathway that results in the generation of glucose from non-carbohydrate carbon substrates. From breakdown of proteins, these substrates include glucogenic amino acids from breakdown of lipids (such as triglycerides), they include glycerol (The backbone of lipids known as triglycerides); and from other steps in metabolism they include pyruvate and lactate.

Gluconeogenesis is one of several main mechanisms used by humans and many other animals to maintain blood glucose levels, avoiding low levels (hypoglycaemia). Other means include the degradation of glycogen (glycogenolysis),[1] fatty acid breakdown and fatty acid catabolism.

Glyceroneogenesis [21 – Nye et al 2008] is a metabolic pathway which synthesizes glycerol 3-phosphate or triglyceride from precursors other than glucose. Usually glycerol 3-phosphate is generated from glucose by glycolysis, but when glucose concentration drops in the cytoso (The cytosol or cytoplasmic matrix is the liquid found inside cells) it is generated by another pathway called glyceroneogenisis. Glyceroneogenesis uses pyruvate, alanine, glutamine (Alpha keto and amino acids). Glyceroneogenesis can be observed in adipose tissue and also the liver. It is a significant biochemical pathway which regulates cytosolic lipid levels. Intense suppression of glyceroneogenisis may lead to metabolic disorder such as type 2 diabetes

As mentioned, from a non-medical perspective, any understanding of the above 3 concepts is layman in nature and comments are as such.

In short and in layman's terms,

Glycogenesis is the creation of glycogen which is a stored form of polysaccharide glucose in the muscle cells and liver. This is the inward journey of nutrients on their way through the liver and being converted into usable sugar in cells containing mitochondria, as a secondary storage means for energy in the body.

Gluconeogenesis is the internal process by which the body creates useable glucose from stored energy sources including proteins and fats (triglycerides) It produces glucose in the blood stream which, in heathy individuals with normal insulin response, is then available to cytoso.

Glyceroneogenisis is the process similar to gluconeogenesis but Instead of producing fructose 1,6- bisphosphate as gluconeogenesis does, Glyceroneogenisis converts dihydroxyacetone phosphate to glycerol 3-phosphate. This happens with the aid of Alpha Keto and Amino acids in the liver and adipose tissue. It's the similar early stages of both Glyceroneogenisis and gluconeogenesis that are relevant to this discussion. HBOT is speculated to be an up regulator of Glyceroneogenis in adipose tissue as opposed to treating with down regulation of the process in the liver to reduce the triglyceride being released in to the bloodstream from the liver.

It's that mechanism or process of Glyceroneogenisis, and its shared processes with gluconeogenesis, that causes type 2 diabetes when it fails or become supressed in the adipose tissue. And it fails because there is no signalling to trigger the process when blood sugar levels remain high because of poor insulin response. Without a reduction in blood sugar levels this process is not triggered. [18]

HBOT stimulates this process in adipose tissue, providing relief from suppression of the cycle allowing cells to respond better to natural insulin, cell gateways to open, allowing blood sugar levels to come down after moving sugars into the intra cellular fluid. Thus, lowering blood sugar levels and triggering glyceroneogenisis and gluconeogenesis. The pathway for converting fat back to sugar and using it up as energy at the intracellular level via means of better Adenosine Triphosphate (ATP) function and better regulated intra cellular energy use.

Insulin response is key to breaking the obesity cycle. Without effective insulin response, glyceroneogenisis is not triggered in adipose tissue and it stalls. In some cases, the body burns fat directly leaving behind ketone acids, (not to be confused with healthy ketosis), which can cause further damage to the kidneys. Artificially increasing insulin levels works as a short-term measure, but also further desensitises cells and further inhibits glyceroneogenisis in adipose tissue. A reasonable argument in favour of HBOT as a treatment method. Lower blood sugar results in lower insulin levels in type 2 diabetics, allowing for a better balance between glyceroneogenisis in the liver and adipose tissue.

Consideration must be given, for that which has been considered a potentially dangerous side effect for hypoglycaemic patients, is actually a positive treatment for hyperglycaemic ones.

Dive supervisor and chamber operator teaching dictates that one should watch out for blood sugar drop outs and unconsciousness. Why not put this to use for people who don't want to take Metformin or who are unable to, or indeed for those who have become desensitised to its effects over time and also as a replacement for either naturally, (vine leaf) or artificially increasing insulin in the blood stream as a form of treatment.

The effect will be twofold at least. A loss of weight, and also effective insulin response management.

An interesting study to look at was conducted and published by the Rubicon Foundation which states;

[abstract] A SIGN OF RAPID BODY WEIGHT LOSS UNDER HYPERBARIC OXYGEN TREATMENT WAS OBSERVED IN MICE WITH NON-ALCOHOLIC FATTY LIVER DISEASE [**22 -** Tsuneyamaa et al – 2009]

This would suggest an improved use of glycogen as energy, resulting in rapid weight loss, following replacement of that glycogen from adipose tissue, following treatment.

Stem Cells, Genetics and Mitosis

As a diver, it starts to get beyond my scope of study when we reach topics such as genetics. It is proposed that such a connection nonetheless exists. Owing to various life experiences, I have encountered the subject of genetics before and have had to develop my own understanding of them to a level where I could at least have a conversation.

Understanding lies in the knowledge that the body cells are regularly replaced by new cells every so often and in differing time scales depending on which cells we are talking about. The new cells copy the old cells and so on. For the purposes of this discussion neurons will be left out for now. While I have my own thoughts and beliefs on the regeneration of neurons they are somewhat unsubstantiated save fringe science studies into epigenetics and neuroplasticity, although modern research does claim to be able to convert glial cells into neurons but that's another discussion. **(Since this writing, further studies have confirmed this.)** [23 Boldrini et al – 2018]

There are two sides to this section.

The known fact that our bodies replace cells from time to time, and also the known fact that those cells are 'copied' in accordance with the DNA or RNA present in the cell being copied. This is why scar tissue remains scar tissue for life. Whether that is regular scar tissue or, say for example, scaring to the myelin sheath in multiple sclerosis patients. Hence the medical standpoint that MS is only manageable and not reversible. We also know that HBOT stimulates the release of stem cells which are essentially brand new, 'un-programmed' cells likened with a foetal state cell.

It is also theorised that HBOT treatments effect genetic expression and effect the protein inside a cell. In the time leading up to becoming a diabetic the genetic expression of cells is to respond to insulin. Over time and excessive exposure to very high levels of insulin, and an imbalance in glyceroneogenisis between the liver and adipose tissue, those cells become less sensitive to insulin following a change in genetic expression brought about by a desensitisation to insulin. The result is a metabolic disorder.

In type 2 diabetics, there is generally plenty of insulin around, as generally the pancreas still functions. The body just doesn't respond to it like it did before. This can also explain sudden hypoglycaemia and even unconsciousness in some patients whose blood glucose drops by as much as 10 mmol/l upon pressurisation. (referenced above). In fact, it is a described side effect and technicians and attendants are warned to keep an eye out for it even in non-diabetic patients. Sudden improvement in insulin sensitivity makes available an adequate supply of insulin in the blood to the cells that weren't responding until pressurisation. [1 – Heyboer et al 2017]

Is the perception of an irreversible condition, perhaps owing to the gradual change in genetic expression over time, to a point where the condition is seemingly irreversible because any new cells are simply copies made of the faulty ones they are replacing. What if they weren't? What if the cells being replaced were at optimal function at the time of replacement? Surely, they would be copied with a different genetic expression reversing at least that one cells insulin resistance?

Could the result of long term treatment be, that as cells are replaced with new ones, from an increased release of stem cells cascading as a result of HBOT, [24 – Thom et al – 2018] , those new cells are replaced at least at a higher level of function as it pertains to insulin response, thus

providing a permanent, and if not complete, then partial and sustained reversal of the condition?

Treatment would then be multi-faceted. It would treat the immediate issue of poor insulin response, to reduce blood sugar in the immediate, thus preserving existing capillary structures and halting damage cause by high levels of glycated haemoglobin. While also treating consequential conditions like foot ulcers, problematic wounds, retinopathy, kidney disease and chronic kidney disease (CKD) by means of revascularisation of the kidneys and also potentially treat cells at a genetic level, leaving new cells in a better state of function and expression than their predecessors. They would then be able to signal the metabolic process better than before on their own without treatment. Thus undoing the cellular damage caused in the first instance.

Have any studies been conducted of the long-term effect of HBOT on the ongoing change in cellular response to insulin and the associated metabolic functions that go with it? Existing research suggests that more data are needed, but also suggests this is indeed the case.

Following long term treatment, perhaps 3 to 4 months, it could then prove valuable to maintain a program of regular home treatments in the form of Mild HBOT and soft shell chambers to maintain an ongoing effect.

Dare I say… Conclusion

I conclude, at least to my own sensibilities and thoughts, that hyperbaric oxygen therapy would indeed improve ATP function, up regulate glyceroneogenisis in adipose tissue, support metabolism in the long term and re-write the genetic expression of damaged cells, providing a long-term reversal of metabolic challenges such as type 2 diabetes, brought about by insulin resistance and uncontrollable obesity. Making life a lot more pleasant for many.

The referenced study at [25] here would seem to support these ideas. [25 – Wilkinson et al – 2015]

Thoughts on Pollution

As an aside and addition to these comments on localised pollution.

Some time ago I contacted the European Air Quality Commission as well as Public Health England to no avail.

This is rather an anecdotal aside but worth a comment since medical professionals I have discussed it with agree.

In 2016 I travelled to New Zealand where the air was cleaner than where I live in the UK. I had no problem losing weight. I lost around a kilogram a week for about 10 weeks before my return to the UK.

Nothing else had changed. I remained on the same low carb diet I had been, my activity was stable. The only change was location and consequently air quality.

Doctors and I speculated that owing to factory effluent in the area I live in, in the UK, and NHS Drager measurements of high carbon monoxide in my blood (18ppm), that what was an

already challenged metabolism, was pushed just that little bit further and compromised beyond the point of being able to self-regulate the whole metabolic process and glucose cycle.

Consequently, I have put a lot of it back on in the 8 months since my return. As we know carbon monoxide bonds with haemoglobin 100 times more efficiently than oxygen does, (Hence the only good treatment for CO poisoning is HBOT). Lower oxygen transport to cells leads to lower metabolic function and while it may only be a few percentage points on TPo2 levels it could, in some cases, be the difference between losing weight gradually and not losing weight at all and perpetuating the problem.

This further supports, that even if only a small measure of difference, HBOT treatments could well move patients just over that line between downward spiral; and upward improvement in their lives.

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