Present Status of Clinical Application of Hyperbaric Medicine.

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It is one of the great honors of my professional life to have been invited by this distinguished group to deliver a lecture on the present status of hyperbaric oxygenation. It was in 1969 that I attended a meeting on this subject in Sapporo hosted by my long standing friend, Dr. Juro Wada. Dr. Iwa, who extended the present invitation to me, was working with Dr. Wada at that time and thus our friendship with him and his charming wife began.

I have given considerable thought to the organization of this talk and, of course, to what its content should be. Rather than presenting a review of the literature, I have assumed that this audience knows the subject and that my role is to give you my personal views as to where it stands with emphasis on principles of treatment. I apologize in advance for omissions and can only say that to summarize so large a field in one hour is a virtual impossibility. I shall do my best.

I have written on the history of hyperbaric medicine but regret that time will not permit showing you pictures of the many old chambers that are interesting in themselves. I would, however, be unforgivably remiss if I did not pay tribute to Dr. Iete Boerema. Without his pioneering work we would not be here today Once, when asked about the future of HBO, he told this story: Outside his front door was a brass sign that read "Prof. Boerema". One day a group of young boys rang his bell and asked "Does the sign mean professor or prophet?". Now, many years later, I feel much the same way.

The real importance of history is to help us avoid the repetition of past mistakes. There are a great many sound clinicians who feel that hyperbaric oxygen is of no benefit at all. It is most important to change this view if we are to avoid the fate of the nineteenth and early twentieth century chambers, namely to be melted down for scrap metal. Suffice to say by the mid-nineteenth century there were more than 50 chambers scattered through Europe. By and large their scientific merit was on a par with the use of the mineral water spa today. In fact, the patient in those days could choose whether to take mineral water or compressed air baths.

How can we put hyperbaric oxygenation on a more sound scientific footing? It is not enough to preach to the convinced! This is the day of the controlled clinical trial. While there are some diseases where the method is practical, by and large it is not, because of the relatively small clinical populations involved and the great diversity within each population. The experimental animal models of disease have been extremely useful and while some are not convinced by this approach, it is valid, but clinical observation and awareness of the natural history of the untre-

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ated diseases remain the mainstay. Lastly, I would like to say that there is nothing wrong with following the judgment of good common sense based upon theoretical consideration.

PRINCIPLES

There are two basic objectives achieved by the use of hyperbaric oxygenation. The first is to becrease the volume of undissolved gases and the second to get more oxygen to the tissues.

One must remember that at two atmospheres the volume of a bubble is decreased by 50% but that it requires 7 atmospheres to decrease the diameter by 50%.

In regard to getting more oxygen to the tissues, the truly important oxygen is that dissolved in the plasma. Only this oxygen supplies the tissues and creates the environment for wound healing. On the other hand, when dealing with surface infections, it is the actual $_pO^2$ to which the bacteria is exposed that is the important factor. I have had no personal experience with the use of fluosols under hyperbaric conditions but the combination has obvious and immediate applications.

Both the health of tissue and wound healing depend on the presence of adequate amounts of oxygen at the tissue level. Since the actual perfusion of oxygen, even at very high pressures, is less than 1 millimeter, perfusion of tissues by blood plasma is absolutely essential, hyperbaric oxygenation or not. The body has exquisitely sensitive self-regulating mechanisms that control the partial pressure of oxygen in tissues. The most useful is constriction or dilation of the arteries. Under HBO the arteries to an ischemic area remain dilated while adjacent well-perfused tissues exhibit vasoconstriction. Early in the modern development of hyperbaric oxygenation, this was not understood and there were endless arguments as to whether the oxygen at the cellular level was actually increased to ischemic tissue in view of decreased over all blood flow to the organ or area.

With these remarks as a background, I shall go into the specific uses to which hyperbaric oxygenation is being put at the present time and I shall also discuss areas that have not fulfilled the expectations we had. First, a word is in order about the chambers we use.

CHAMBERS

There is no question that the single place chamber is gaining in popularity and numerically is in wider use than the walk-in type. The reason for this is the lower cost of both the initial hardware and personnel. I believe this to be a pity because the walk-in chamber allows hands on care of the patient. It is also safer because it is compressed with air, and not oxygen, and is, of course, far easier to instrument than the single place chamber. The last survey done in the United States showed that more patients were still being treated in walk-in chambers than in monoplace chambers. However, this survey did not include many centers that, in my opinion, are established merely to make money with no regard for scientific validity. The treatment of senility is the most glaring example of this practice.

There is a third type of "hyperbaric chamber" that has no right to the name. It is a chamber of a size suitable to placement around an arm or leg where it is sealed by a cuff. Oxygen at increased pressure is then circulated within the "chamber". Its proponents claim is to be effective against various circulatory and infections disorders. To me, it is quackery. A moment of thought will make this obvious. The physiology which makes it possible to expose patients to 2 and 3 atmospheres of pressure rests on the principle that the internal partial pressures of gas within the body are the same as those in the environment. The so called small hyperbaric chamber does nothing about the internal pressures, so as soon as the pressure exceeds venous pressure, it becomes, in effect, a tourniquet that does not allow blood to exit from the extremity. I stress this type of chamber only to condemn it, because a good deal of

money is being spent on its promotion and the public is being told it represents a mimaculous advance!

A word about level of pressurization is in order. The present rebirth of hyperbaric oxygenation began which started in the 1960's and early in that period the Amsterdam group worked at 3 atmospheres absolute while Mr. Illingworth's team in Glasgow used 2 atmospheres. Some sporadic attempts at using 7 ATA for the performance of cardiac surgery were made at Duke University.

As time has gone by this question has been resolved by sheer experience and practicality. Pressures in the 2 to 3 atmosphere range have been found to be the most desirable. Pressures higher than these present great problems in regard to decompression times as well as the ability of the operating team to function properly because of nitrogen narcosis. Pressures below 2 ATA are not really effective except in rare instances which will be mentioned later. From a practical point of view, 2.5 ATA is effective and decompression times are minimal. In our unit where more than 27,000 individuals have been exposed, we work at 2.5 atmospheres for most medical treatments but will treat the very sick gas gangrene patient at 3 ATA. For the performance of surgery which, again, will be mentioned later, 2 ATA is used for general support and 3 ATA when very maximal oxygenation is desired, as while repairing a carotid artery.

There have been instances in our experience where we have adjusted the chamber pressure depending on $_pO^2$ of the arterial blood. In these conditions the hyperbaricist is simply trying to keep the patient alive until some other factor results in improvement. Because of the inevitable alveolar oxygen toxicity which occurs when breathing pure oxygen even at ambient pressure and the potentially long time needed for treatment, the goal is to maintain minimal arterial $_pO^2$. This would be so in a case of autoimmune hemolytic anemia, until steroid therapy takes effect or compatible blood is found. In the case of respiratory failure where conventional respiratory support cannot maintain life, we need time until the lung gets somewhat better. The desired $_pO^2$ in this type of situation is generally in the range of 60 to 70 mm. Hg. For example, in this type of patient with a $_pO^2$ of 40, 1/3 rd ATA may be sufficient.

DECOMPRESSION ACCIDENTS

There can be no debate here. "Squeezing down the bubble" that is lodged in a blood vessel or joint is the most effective treatment available. Because of the actual hazards imposed and the sheer time involved, treatment at lesser pressures while breathing oxygen has proved more effective than relying on elevated pressures alone. One must also remember that the use of anticoagulants and agents such as low molecular weight Dextran serve a real purpose in decreasing blood clotting and keeping the blood circulating in low flow vascular beds.

Twenty four hours after a dive is the arbitrary cutoff time that is used at our facility for accepting a decompression accident for treatment. There are sporadic case reports of patients with air emboli during cardiac surgery being treated with success. We have treated a single case of explosive decompression not due to a diving accident but secondary to rapid ascent of a jet airplane in which the cockpit pressurization failed. The patient survived without sequelae, but he had been very ill on admission with a hematocrit of over 60.

Although not dealing with a decompression accident, I would like to mention a paper that we wrote on four cases of decompression of a Cantor tube balloon complication. I don't know whether a similar tube is used in Japan for intestinal decompression, but this tube has at its end a thin rubber bag which is sealed and contains a small amount of mercury. In rare cases, the balloon expands because of captured air and cannot be removed. Heretofore, it was necessary to perform a

laparotomy so that the balloon could be punctured. Now, one has only to pressurize the patient in a hyperbaric chamber. In practice, now with an experience of 6 cases, we simply place the patient in the chamber and start pressurization. At each atmospheric increment, we pull on the tube. If it does not dislodge, the pressure is increased another atmosphere until success is achieved. Of course, oxygen does not have to be administered.

Three to five atmospheres of pressure have been necessary before the tubes could be withdrawn, an excellent example of the fact that the diametemr of a bubble decreases much less rapidly than its volume. In a few instances we have used a similar technique to decompress a severe toxic ileus. One must be prepared, in this instance, to keep the patient in the chamber for a long time because as soon as the chamber pressure is lowered the intestinal gas, if not gone, will increase in volume.

CARBON MONOXIDE POISONING

There can be absolutely no doubt that hyperbaric oxygenation can supply the oxygen needs of a patient whose hemoglobin oxygen binding system has been paralyzed by the greater affinity of carbon monoxide and also by the mass action effect of the very high oxygen which speeds up the unbinding of the carboxyhemoglobin. The response of the patient is often rapid and highly dramatic. The actual mechanism of improvement probably also involves decrease in elevated intracranial pressure. Obviously, if the brain has been damaged before treatment is started, the damage will not be reversed immediately. Although this is still a subject of some controversy, it is probably wise to continue to treat for some days because of carbon monoxide bound in the tissues which is released much more slowly. Fortunately rare is the very disturbing patient who has apparently madd a complete recovery but who suddenly dies 4 or 5 days later. This is due to hemorrhage into the basal ganglia. I have seen such a case noly once, but one does not forget it.

As the public becomes educated, carbon monoxide poisoning due to smoke inhalation at the time of a fire will probably end up being the single most common indication for the use of hyperbaric oxygenation. It is a great pity that this is not already the case.

To prevent paralysis of the system by too many candidates for treatment of smoke inhalation, emergency services should be instructed to send only those cases who are unconscious or incoherent, or who do not respond appropriately to verbal commands or painful stimuli. Once at the hyperbaric treatment center, only those patients with carboxyhemoglobin levels above 25% are treated and those, at any level, who exhibit central nervous system pathology. Steroids are given prophylactically.

To be complete, and because of the similarity of the intoxication mechanism, cyanide poisoning should be mentioned. I know of only one case. This occurred during a robbery in a chemical plant. As the police arrived one of the robbers hid in a vat of cyanide. He was found comatose and made a prompt recovery when treated in the Vancouver hyperbaric chamber.

INFECTIONS

The treatment of infection remains one of the most valuable uses of hyperbaric oxygenation. Before making comments on specific infections I wish to state some general principles concerning the effect of oxygen on bacterial growth.

(1) The growth of anaerobic organisms will be inhibited, or completely stopped by high oxygen tensions, but spores of the anaerobic organisms will be unaffected and can begin growth long after the primary infection has been eradicated. Recurrences of both gas gangrene and tetanus have been seen in instances of an infection which had apparently been completely eradicated at a remote time only to start again following local trauma

which presumably supplied an environment of necrotic tissue under anaerobic conditions.

(2) The growth of aerobic bacteria will be speeded up by increases in oxygen tension up to the range of 1.5 to 1.8 ATA. At higher pressures the multiplication will be effectively stopped. Since tissue levels of oxygen are far lower than 2 ATA, the use of HBO is contraindicated in deep seated aerobic infections Contrariwise, when a surface infection exists, as in an ulcer, HBO will effectively stop the growth of common organisms such as staphylococcus, streptococcus, and pyocyaneous.

(3) Mixed infections of aerobic and anaerobic organisms should not be treated with HBO because the overgrowth of the aerobes will cause tissue destruction and thus create an anaerobic environment which will favor the growth of the anaerobic organisms despite the administration of HBO.

(4) The decision to treat a specific infection must be based not only on the effect of oxygen on the bacteria but also on the newly created interstitial conditions which often allow the quiescent body defense and healing functions to operate. The treatment of chronic osteomyelitis is a perfect example.

With the above principles in mind, I shall discuss some specific infections.

GAS GANGRENE

Although the treatment of gas gangrene with HBO is still controversial because of the number of variables involved, there should be no debate: hyperbaric oxygenation can save life and limb. Only a controlled clinical trial will ever really settle the controversy. This will probably never be done because I can not imagine an experienced hyperbaric center withholding treatment from any of these critically ill patients. My strong opinion is based on personal observation of hundreds of cases referred from major medical centers in which the downhill clinical course was promptly reversed when the only added treatment modality was HBO. While the HBO physician is on the defensive about this, one need only point out innumerable areas in clinical medicine where such controversy exists. Not until general computerized registries are established, with common definitions of the individual variables, will a convincing answer be obtained. This is no reason for not proceeding with HBO treatment of gas gangrene.

The diagnosis of gas gangrene must be based on purely clinical findings. It must be stressed that the mere presence of Clostridium perfringens, or a similar organism, does not constitute an infection requiring HBO therapy. Isolated clostridial cellulitis is probably treated quite adequately by antibiotics alone, although if a chamber is readily available the employment of HBO cannot be criticized. The clostridial antitoxins have no place in the therapy of gas gangrene both because of ineffectiveness and a high incidence of serum sickness inherent in their use. Penicilldn in the dose range of 30 million units per day remains the most effective antibiotic regimen. In the presence of penicillin allergy, the tetracyclines are the next most effective. The Amsterdam regimen of three treatments in the first day and two treatments the second and third days seems to have withstood the test of time in most centers. Our bias is to treat at 3 ATA until obvious improvement has taken place and then to switch to 2.5 ATA.

Every hour can be crucial when HBO therapy is to be instituted. If the patient arrives for treatment alive and speaking, the therapy will almost always succeed; if comatose, mortality will almost always ensue. The single exception to this broad statement is in cases of gas gangrene occurring in patients in whom the basic body immune mechanisms have been paralyzed by disease or previous therapy. Chemotherapy in advanced malignancy is the most common example. These cases are uniformly fatal no matter what is done.

Three treatments in the first 24 hours have been given, delaying surgical intervention until the initial response can be evaluat-

ed. The policy results in far less radical surgery, with the amputation avoided or performed at a lower level than would have been the case if the surgery had been done earlier.

NECROTIZING FASCIITIS

These cases are referred because they are thought to be gas gangrene. They consist of very extensive subcutaneous crepitant infections covering large areas, usually involving the thighs and perineum secondary to anal or urinary tract problems. Multiple pathogenic bacteria are cultured, one of them being an anaerobic bacterial organism other than Clostridia.

We have recently written up our rather large experience. The conclusion reached is that immediate radical surgery is the treatment of choice. The mortality is very high even when treated in this way. I suspect that the reason hyperbaric oxygenation does not help is that the aerobic organisms may well have their growth rate increased and they are as deadly here as the anaerobe.

TETANUS

Hyperbaric oxygenation serves no useful role in the treatment of tetanus. The reason is that the Clostridium tetanus' organism has largely disappeared by the time symptoms appear. These symptoms are due to a toxin which is unaffected by oxygen.

CHRONIC OSTEOMYELITIS AND RADIATION NECROSIS

Treatment of these conditions is rapidly becoming the single most common entity for which HBO is now used. When one sees success in repeated cases against which treatment has been ineffective over a period of 15 to 20 years, there is no reason to doubt its efficacy. Experimental animal models confirm the clinical impression.

The mechanism is almost certainly the conversion of relatively anoxic tissues to a state of normal oxygenation in which the normal healing mechanisms become operative. When, and if, a sinus or ulcerated area is present where oxygen, at least at 2 ATA, can come into direct contact with the infected surface area, then the bacteriostatic mechanism described above becomes operative but, overall, this is thought to be a minor factor. It is of interest that oxygenation will actually take place for a short distance through the skin in a hyperbaric oxygen environment. Many years ago we were doing experimental work in a monoplace chamber on long tube pedicle grafts having a length to width ratio of 5 to 1. There was less necrosis of tissue in the center of the pedicle in those animals treated with hyperbaric oxygen. However, the point of interest here is that some of the animals died and we did not recognize it because the skin color remained pink.

When using a walk-in chamber surface oxygen is applied to the wound by placing around the area a plastic bag which has humidified oxygen circulating through it. It is prudent to sponge away surface fluids and clean out sinus tracts because oxygen will diffuse only a short distance even at hyperbaric pressures.

Typically, in a patient with osteoradionecrosis, the constant pain experienced will often disappear at first in the chamber and then entirely during the first week of treatments. In those patients with osteomyelitis of the jaw, most often secondary to tooth extraction, a useful and easily quantitated guide is how far the mouth can be opened. Progress can be judged by the observation of wound healing as well as by the relief of pain. The radiological appearance of the bone has not proven to be very useful as a guide because it occurs so slowly although at times a sequestrum will become apparent and must be removed surgically.

MELENEY ULCER

Dr. Frank Meleney, with whom I had the privilege of working, deserves credit for calling the attention of medicine to this entity and first treating it successfully. It is known

by a host of different names. Among these are synergistic gangrene and phagodenic ulcer. Bacteriologically, the one common organism is a microaerophylic streptococcus. This organism is in combination with a number of other non-specific organisms. The specific streptococcal organism is extremely difficult to culture and the fact that it is not evident should not preclude the diagnosis if the appearance of the wound is typical. The ulcer most often occurs in patients already debilitated by some other chronic disease such as ulcerative colitis or in patients recovering from major intraabdominal surgery.

This type of ulcer is readily recognized by its characteristic undermining of the skin at the ulcer's edge. The reason for this is that the microaerophylic organism can grow only in a narrow range of low oxygen tensions. The main problem in treating an ulcer of this pype is that it is like an octopus with many tentacles. If all the tentacles are not destroyed, the infection will recur. Dr. Meleney's treatment consisted of surgical excision of the ulcer followed by repeated packing of the residual defect with zinc peroxide in order to release oxygen at the wound edges, there by preventing the offending streptococcus from growing.

In our hands these ulcers have been cured quite dramatically by excision of the ulcer followed by constant exposure of the wound surface to humidified oxygen at 2 ATA for 24 to 48 hours with the patient breathing oxygen three times a day for 90 minutes at a time. This is one of the first diseases, other than decompression illnesses, where we have ever found it necessary to keep a patient in the chamber for prolonged periods. Such treatment presents a real challenge with regard to proper staffing both inside and outside the chamber.

ACTINOMYCOSIS

This anaerobic fungus has been treated only twice in our hands but since the standard, and often unsuccessful, treatment is intravenous penicillin in high dosage for a 6 week period, it is worth mentioning. One case was a perirectal abscess and the other was a liver abscess. The latter seemed to disappear only to recur later but it responded favorably the second time as well. Strangely, no cases of so called "lumpy jaw" have been referred. Treatment has consisted of 2 sessions per day at 3 ATA.

SICKLE CELL ANEMIA

In this condition the red blood cell changes shape at low oxygen tensions and the value of hyperbaric oxygenation seems apparent. In actuality, a subject in sickle cell crisis has not been referred to us in several years although there was no doubt that these crises were reversed by HBO. Leg ulcers, so common in these patients, have responded favorably. It is worthy of mention that one of the complications, priapism, is easily reversed.

There has, however, been one application that has proven extremely valuable, namely the ability to operate on the eye in these patients. Heretofore, this type of surgery has failed miserably; as soon as the eye was manipulated, the oxygen tension decreased and the red blood cells sickled with disastrous consequences. My colleague, Dr. Dennis Freilich, has operated, with complete success, on about 15 of these patients for retinal detachment.

ISCHEMIA AND WOUND HEALING

If the blood supply to an area is compromised severely, there will be an area of normal perfusion at the border of the ischemic area, an intermediate zone of little blood supply where the tissue is alive but probably not functioning and, often, a completely ischemic area where tissue death will occur in amounts of time varying with the nature of the tissue. Hyperbaric oxygenation is effective only in promoting the survival or healing of marginally perfused tissue. It will not bring dead tissue back to life. Agents that

increase perfusion by preventing sludging, or decreasing the viscosity, of the blood will increase the effectiveness of HBO. Attempts have been made to infuse intraarterial vasodilators during HBO therapy to augment its effects but they have been unsuccessful and were abandoned. I believe this is due to the complications of arterial injection and that flow cannot be increased appreciably in the ischemic vascular bed which is already maximally vasodilated.

Zones of injury, as in a bone fracture or an ulcer, are normally at low oxygen tension. HBO will speed up the formation of granulation tissue by increasing the oxygen tension. Indolent non-healing wounds will frequently heal. If the wound is an ulcer, surface application of HBO will accelerate the healing by its direct effect on the wound surface. It will also prevent multiplication of aerobic bacteria. Therefore such wounds should be treated with a combination of systemic and surface oxygen. Because of systemic oxygen toxicity, severe limitations exist for the periods of time that oxygen can safely be administered via the respiratory tract but no limitation seem to exist for surface oxygenation. However, it is important to humidify the surface oxygen to prevent drying of the tissues.

The clinical documentation of the these beneficial effects has been difficult but the literature is replete with experimental animal models, whether they are ulcers, healing of skin grafts, or ligation of the blood supply to a specific area. The clinical problem has been that the benefits apply to only the marginally perfused tissues. One need only observe the immediate relief of ischemic rest pain in the patients with peripheral vascular disease to understand this last point. The marginally perfused pedicle skin graft will quickly demarcate during the course of HBO.

Fractures of the navicular heal notoriously poorly. I hope the lecture will stimulate a member of the audience to carry out a controlled study of this particular fracture. It has not been done and if the efficacy of HBO therapy can be demonstrated, it would bring about the treatment of many bone injuries.

INCREASED INTRACRANIAL PRESSURE

Our laboratory demonstrated many years ago that increased intracranial pressure can be decreased rapidly by hyperbaric oxygenation. The effect is due to vasoconstriction with concomitant maintenance of adequate oxygenation. This has been documented clinically many times and probably has application in acute spinal cord injuries as well. This effect has never been widely utilized because steroids and hyperosmolar agents are also effective. A comparative study is needed here as will.

SENILITY

Work emanating from Buffalo purported to show that mental function in senile patients exposed to HBO would improve and that this improvement would last beyond a series of treatments. Early on, we visited their facilities together with a geriatric psychiatrist. He examined the patients and the controls who were under treatment. It was his opinion that the improvement noted was not due to the oxygen therapy, but because they were severely depressed people. Any time depressed patients are given attention, as they were when they were transported to the chamber twice a day and observed closely, their depression will diminish and they will score better on tests to measure cognitive function.

Under any circumstances, our unit, and other centers as well, repeated the work and could demonstrate no benefit. It is my very firm opinion that HBO is of no value in the treatment of senility.

STROKE

The treatment of stroke by means of hyperbaric oxygenation has been extremely disappointing. Surely, about 30% of the

patients given HBO within six hours of onset of a stroke will exhibit partial or complete remission of their stroke symptoms. However, they cannot be left in the chamber for more than a few hours because of oxygen toxicity, and the symptoms return as soon as the HBO therapy is stopped. During a second treatment 6 hours later, only one half repeat the favorable response, and by the third treatment there is no benefit.

The favorable response during the early treatment is most certainly because increased oxygenation has restored function in marginally ischemic areas of the brain which eventually become so injured that they cannot respond during subequent treatments. Unhappily, nothing therapeutically useful has come from all the biochemical work on administration of various agents to combat oxygen toxicity. Were there a way to do this, it is likely that leaving the patient under HBO for days might very well protect the brain long enough to allow collateral circulation to develop.

Here I want to note, however, that the vascular surgeon can now easily repair a stenotic carotid artery in the neck, from which the majority of strokes emanate. Utilizing microsurgical techniques, a temporo-middle cerebral bypass is also possible. These operations are capable of restoring normal blood flow to the brain. Sadly, when these operations have been performed in instances of acute stroke, some patients have benefitted but many have died because the restoration of blood flow has converted an ischemic to a hemorrhagic infarct.

Even though HBO and surgery have failed individually, it seems likely that a combination may yet reverse many strokes. Thus, the hyperbaric exposure would be used to determine which strokes were reversible and therefore suitable for revascularization. The CAT scan can be used for initial screening in order to demonstrate infarcts at the very beginning. I have embarked on such a study but the problems are very real. The chamber, the CAT scanner, the angiology suite must be ready on an around the clock basis since a stroke does not respect working hours. Even more difficult is educating the local physician to send the patient to the chamber as soon as the diagnosis is made; there must be no delay in getting the patient to the hospital. We have treated a few patients thus far but since many acute stroke patients recover spontaneously, it will take many years before a conclusion can be reached.

SURGICAL SUPPORT

We have used the hyperbaric chamber as an operating room and it functions like any other with a few additions to detail. One great advantage is that the anesthetist can't wander out of the operating room. Fluothane is largely used as the anesthetic agent but at lower concentration so that its partial pressure is not increased. Endotracheal and bladder catheter balloons are inflated with saline instead of air. Intravenous solutions are vented. Bipolar cautery is employed. Electrical apparatus is surrounded by a bag purged with nitrogen. This latter is a new procedure foisted upon us by fire regulations and probably totally unnecessary since the chamber is pressurized with air. Only the patient gets oxygen and that by endotracheal tube. In years gone by routine myringotomies were done but this has been stopped. The rare patient complains about "water in the ear" and this disappears in a week or two. Carotid surgery is done at 3 ATA, most other operations at 2 ATA. In major be cross clamped and decompression started as soon as the clamps are removed and the patient is stable. To date 845 operations have been done under hyperbaric oxygenation. At the present time we do about 100 operations per year in the chamber.

The indications for doing surgery under hyperbaric oxygenation fall into three general catagories but multiple indications are very often present in the same patient. They are

as follows:

(1) Poor Surgical Risk

A history of recent heart attack or stroke, minimum cardiac reserve with episodes of cardiac failure, decreased pulmonary function, decreased renal function, or malnutrition tend to lead us to do a case under HBO. An internist is involved and when asked whether he thinks the patient will tolerate the operation, the reply is quite standard and I quote, "Yes, I think he will tolerate the surgery but you must be very careful to avoid hypotension and keep him well oxygenated." This is most obvious to all surgeons but hyperbaric oxygenation fits the need. The real danger of hypotension is anoxia which means that if hypotension is encountered, the surgeon and anesthestist have a longer period of safety to correct the underlying problem. The patient is very well oxygenated, indeed.

(2) Major Blood Loss

Aortic reconstructive surgery falls into this group. While the surgery is usually done with tolerable blood loss, periodically one encounters horrendous losses during which blood replacement falls behind. With the patient hyperoxygenated there is more time to catch up with blood replacement and colloid or plasma serve almost as well as blood if the blood bank lags in getting blood to you.

(3) Specific Regional Support

Here, a specific area of the body is being deprived of circulation due to temporary obstruction of the artery supplying it. If the cessation of blood flow were total HBO would serve no purpose but fortunately, there are always collateral channels supplying the obstructed areas which means that the extra oxygen is getting there.

All carotid surgery that I perform is done under HBO. I have shown hundred of times that at 3 ATA the venous return from the brain is arterialized. Typically, the arterial $_pO^2$ is in the range of 1800 while the venous return, after ligating the facial vein, is 6 to 800. After 5 to 10 minutes of cross clamping the carotid artery during which the reconstruction is done, the venous $_pO^2$ will be in the 2 to 300 range. In more than 300 carotid endarterectomies we have not used a shunt and have had no reason to regret it. Many of our cases have bilateral carotid stenosis with complete obstruction on one side with vertebral artery pathology as well.

We have shown that during resection of an aortic aneurysm under HBO, the venous return from the legs has a higher $_pO^2$ after an hour of aortic cross clamping than is the case with normal circulation at ambient pressure. This serves to prevent anaerobic metabolism below the site of the clamp. As a rule in such surgery done under ambient pressure there is a rather profound drop in blood pressure when the clamp is removed. This is caused by the sudden flush of waste products of anaerobic metabolism into the general circulation. When such a procedure is done under HBO, these drops are minimal or do not occur. Experimentally we have shown that excess lactate production is avoided and presumably anaerobic metabolism minimised.

CONCLUSION

In conclusion, I wish to apologize for the length of this talk and for certain omissions in subject material that I'm certain have occurred to you.

Finally, thank you all for the gracious hospitality that is being extended to my wife and me. It has been far beyond what anyone could reasonably expect and in greatly appreciated.

（本稿は，昭和57年10月２日，金沢市にて開催された第17回日本高気圧環境医学会総会きおける特別講演を再録したものである。）