the smaller airways, smaller than 2 mm in diameter which have few muscle fibres, may not respond to the bronchodilators. It is always tempting to give in to these usually young, bright, enthusiastic individuals, but, the risk is too great. Once an asthmatic, always an asthmatic.

If the diagnosis of asthma is in doubt or if the patient is asymptomatic, a positive histamine and/or methacholine test substantiates its existence. In asthmatics, hyperreactivity to methacholine persists for years, even in the absence of active asthma. In proper hands, this test is a safe diagnostic tool.(7) A positive test contraindicates exposures to increased pressures which could lead to unacceptable risk for pulmonary overpressurization accidents.

REFERENCES

1. Sherman Wm B. Asthma. In: PB Beeson and W McDermott Eds. *Cecil-Loelb Textbook of Medicine* Eleventh Edition. Wm B Saunders Co, 1963.
2. Bouhuys, Arend. *Breathing. Physiology, Environment and Lung Disease* Greene and Stratton, 1974.
3. Wagner PD, Dantzker DR, Jacovoni VE, Tomlin WC and West JB. Ventilation-perfusion inequality in asymptomatic asthma. *Am Rev of Resp Dis* 1978; 118: 511-536.
4. Hornon DJ, Sunda WL, Kinsman RD, Souhrada J, and Spector SL. Broncho-constrictive suggestion in Asthma: A role for airways hyper-reactivity and emotions. *Am Rev of Resp Dis* 1978; 117: 1029-1038.
5. Zeballos RJ, Shturman-Ellstein R, McNally Jr JF, Hirsch JE and Souhrada JF. The role of hyperventilation in exercise induced bronchoconstriction. *Am Rev of Resp Dis* 1978; 118: 877-884.
6. Weng TR and Levison N. Pulmonary function in children with asthma at acute attack and symptom-free status. *Am Rev of Resp Dis* 1969; 99: 719-728
7. Townley RG, Ryo VY and Kang B. Bronchial sensitivity to methacholine in asthmatic subjects free of symptoms from one to twenty-one years. *J Allergy Clin Immunol* 1971; 47: 91-92.

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COMMENT

The case reported by Dr Clinton-Baker (page 17) suggests that asthmatics needing treatment to stay symptom free should not dive. Yet many do dive successfully. Should opinion or fact govern medical advice? It is to provide facts that the Royal Prince Alfred Hospital investigation has been started.

PATHO-PHYSIOLOGICAL CONSIDERATIONS OF HYPERTHERMIA IN HYPERBARIC ENVIRONMENTS

Ian M Calder

During the past decade hypothermia as a cause of death in the European sector of professional diving operations has become recognised. However, hyperthermia as a problem was reported by Cox et al.(1) in which six fatal accidents were cited, and undoubtedly a number of unexplained deaths could be attributed to this. The problem has been identified at present as occurring in the decompression chamber rather than in the working environment at depth.

HYPERTHERMIC SYNDROMES

Adolph (2) identified several heat disorders but for the purposes of the hyperbaric environment only three need be considered. These are:-

1. Water deficiency heat exhaustion, described by Black et al.,(3) and caused as a result of lack of drinking water intake in a hot environment.
2. Hypochloraemic (a salt deficiency) heat exhaustion due to inadequate replacement of salt loss described by Ladell et al.(4) and leading to dehydration and reduced blood volume.
3. Heat exhaustion due to physical exercise in a hot environment with sweating but without replenishment of salt or water.

PHYSICAL CONSIDERATIONS

To appreciate the dynamics of the induction of hyperthermia in a compression chamber it is necessary to examine the thermodynamics of the environment. The adiabatic mathematical formula gives the theoretical background to the induction of high temperature in a chamber. The equation for this is:-

$$T_2 = T_1 \quad \frac{(P_2)}{(P_1)} \frac{8 - 1}{8}$$

where T_1 is initial and T_2 is final temperature in degrees Kelvin (=°C + 273), P_1 is initial pressure and P_2 is final pressure. d is the ratio of specific heating; for air d - 1.4. Therefore:-

$$\frac{\gamma - 1}{\gamma} = 0.2857$$

Thus, if the initial temperature is 32°C

$$T_1 = 305°K \text{ (ie. } 32 + 273 \text{ degrees absolute)}$$

and if a fourfold pressure rise is induced

$$\frac{P_2}{P_1} = 4$$

$$T_2 = 305\ (4)\ 0.2857$$

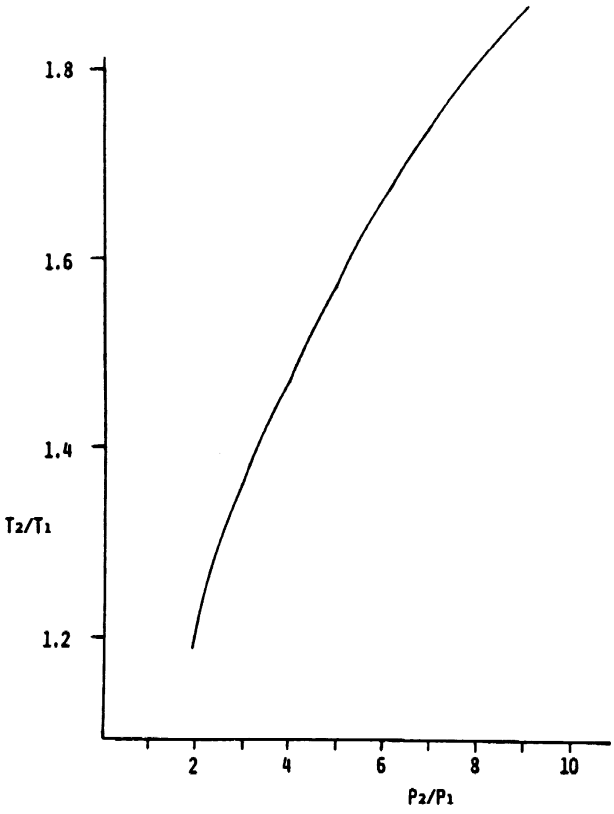
= 453K
= 180°C

When such changes are plotted on the graph, this shows the relationship between T_2/T_1 and P_2/P_1 which when expressed in actual terms shows theoretical temperature rise for example:-

Pressure Rise (in atmosphere)	Initial Temp. (°C)	Final Temp. (°C)
2	10	90
8	40	300

FIGURE 1

THE RELATIONSHIP BETWEEN INITIAL AND FINAL TEMPERATURES FOR MULTIPLES OF INITIAL PRESSURE



This calculation does not make any allowance for physical influencing factors which may cause considerable variations in the final temperature and environmental conditions reached. These are:-

1. The surface temperature of the chamber. In cold climes this will reduce the theoretical final temperature. However, a more serious aspect is the situation in which the chamber is exposed to tropical sun, thus giving an additional radiation heating effect.

2. The speed at which the chamber is compressed. This has a profound influence, in that a rapid increase leads to a large number of calories being introduced into the chamber with consequent environmental temperature rise, which cannot be reduced by radiation from the surface.

An additional dimension to be considered in connection with the physiological effects of these two factors, is that of relative humidity. When the environmental temperature reaches or exceeds 37°C this assumes greater importance. At and above this point, the control of the body temperature is dependant upon the loss of fluid from the body surface by evaporation and cooling is due to the mechanism of latent heat of evaporation. It necessarily follows that as the environment temperature increases above 37°C, together with that of the relative humidity approaching 80%, the cooling mechanism fails and body temperature rises which, outside narrow limits results in irreversible changes. Heat transfer to the body core is necessarily greater in an oxyhelium environment, which compounds the effective rise in body temperature.

CLINICAL ASPECTS

Hyperthermia may pass unrecognised and unappreciated unless it is considered in the differential diagnosis.

When considering the thermodynamics of the hyperthermic situation it is not necessary to have a high initial temperature to induce the unphysiological final temperature, as is shown by reference to the temperature pressure graph.

This has been shown by two deaths in the temperate climes of Northern Europe following rapid compression. However, mere emphasis has to be placed on the adverse effects of rapid compression in tropical and sub-tropical climates, both from personal experience and that of King (unpublished), which has resulted in the total of six deaths. The awareness of such a condition could in retrospect be an acceptable cause of two otherwise unexplained deaths during recompression.

All eight cases had shown signs which were clinically compatible with decompression sickness, and this was the rationale for recompression. The subsequent change and development of symptoms were not those of decompression sickness. These ranged from mental confusion and aggression, anxiety, absence of sweating (hot dry skin), to circulatory collapse. Although it is impossible to generalise from a small series of cases, in the three incidents where more than one person was involved, and there was a survivor, it is apparent that the obese have a smaller chance of survival. This would correlate with the reduction of weight to surface area ratio, with consequent lower heat loss.

Case 1

A medical assistant and a doctor entered a recompression chamber to attend a diver with pulmonary barotrauma. They were compressed to 60 m (equivalent depth sea water) on a 23/77 mixture of "Heliox". The ambient temperature was approximately 35°C and the humidity 85%. The medical assistant was an obese West African, 153 cm tall; the doctor was lean and 180 cm tall. The medical assistant remained lightly clothed, the doctor

removed all his clothing and drank copious quantities of fluid while the medical assistant declined to drink. After 26 minutes the medical assistant was so distressed that he entered the outer lock to decompress, but he still drank nothing. He became increasingly distressed, and was observed picking at his clothes in a confused state. When he left the chamber he collapsed. After about 15 minutes he was recompressed to 42 m, at which time he was stuposose with an irregular almost impalpable pulse of about 120/minute and a respiratory rate of 50/minute. He was given 500 ml 15% dextrose with 20 mg dexamethasone but he died some 5 and a half hours later. At necropsy the body and tissues were noted to be very dry with no interstitial fluid. The CSF sodium was 174 mmol/l (nominal mean 127 in Naumann's series and 131 in a personal series of ten cases). There were no other gross abnormalities.

UNLIKE HYPOTHERMIA, HYPERTHERMIA IS AN IRREVERSIBLE AND RAPID PROCESS, AND ONCE PROTEIN COAGULATION OF VITAL CENTRES HAS OCCURRED, DEATH IS INEVITABLE.

POST MORTEM CONSIDERATIONS

Specific tissue changes developing as a result of acute hyperthermia have not been established, although degeneration of crypt epithelium of the small intestine has been suggested as an indicator. The time sequence is, however, usually too short for the presence of any changes to be established. However, the effects are essentially biochemical, which would not produce morphological changes.

Blood chemistry is an ideal parameter to measure during life, but rapid and variable changes occur after death. Electrolyte measurements of vitreous humor by Hughes (5) and cerebro-spinal fluid by Naumann (6) have shown that these levels are relatively unaffected during the first few hours after death. Taken in the proper context, examination of these parameters may give some indication of the electrolyte status immediately preceding death. However, Cooper et al.(7) make the important point that the least affected is the sodium content, but potassium is subject to some variation.

The use of this technique has been usefully applied in six cases of suspected death caused by hyperthermia. The normal figure of 127 mg.% quoted by Naumann (6) closely agrees with the mean figure of 131 mg.% obtained from a personal random autopsy series of ten cases. All samples were by needle aspiration of ventricular fluid and preservation in lithium heparin tubes before analysis.

The environment results essentially in a pure water depletion with a consequent rise in CSF sodium ion content. From the proven series of six deaths from hyperthermia levels of the sodium ion ranged from 141 mg.% to 181 mg.%. With this range it is apparent that such measurements cannot be regarded as definitive scientific diagnosis of hyperthermia. Such results have to be considered in the overall findings of the case, and must be regarded as a useful adjunct. During dissection, the impression may be formed of some desiccation of body tissues, which taken with the clinical history can lead to a constructive clinico-pathological conclusion.

DISCUSSION

Hyperthermia in a hyperbaric environment is a condition easily produced even in the more temperate climates. However, without an awareness of the speed and ease which such a condition may be produced, this may be omitted from the clinical differential diagnosis. As a result the condition may be worsened by the very nature of the treatment, in which the bizarre symptoms may be judged as being due to decompression sickness rather than to electrolyte disturbance from hyperthermia. It is certain in one case that such misinterpretation has resulted in a further recompression resulting in further deterioration of the patient's condition.

Treatment, once diagnosis has been made, must be symptomatic. The object is to reduce the environmental, and consequently core temperature, and restore the electrolyte balance with appropriate intravenous therapy.

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REFERENCES

1. Cox RAF, McIver NKI, King JD and Calder IM. Hyperthermia in Hyperbaric Activities *Lancet* 1980; ii: 1303-1304.

2. Adolph EF. Water Metabolism. *Ann Rev Physiol* 1947; 9: 381-408.

3. Black DAK, McCanee RA and Young WF. A Study of Dehydration by Balance Experiments. *J Physiol* London, 1944; 102: 406-414.

4. Ladell WSS, Waterlaw JC and Hudson MF. Desert Climate. Physiological and Clinical Observations. *Lancet* 1944; ii: 491-497.

5. Hughes W. Levels of Potassium in the Vitreous Humor After Death. *Med Sci Law* 1965;5: 150.

6. Naumann HN. Cerebro-Spinal Fluid Electrolytes After Death. Proc Soc Biol Med 1958; 58: 16-19.

7. Cooper EJ, Lechner E and Bellet S. Electrolyte Measurements in Blood and Cerebro-Spinal Fluid. *Am J Med* 1955; 18: 613-621.