POST DECOMPRESSION SHOCK*

(A Case Report)

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I will briefly review the existing knowledge of the Syndrome called "Neuro-circulatory collapse at altitude" and place before you the clinical history of such a case.

Collapse resulting from exposure to altitude refers to the clinical syndrome occurring during or after exposure to altitude and is characterised by symptoms which may be chiefly neurological, chiefly circulatory or a mixed type which is called neuro-circulatory.

There are a variety of factors which can lead to collapse at altitude. The most common is that due to hypoxia resulting from decreased partial pressure of oxygen in the inspired air. Collapse can also result from hyperventilation caused by anxiety or apprehension. Coronary insufficiency or spasm, vasovagal syncope, hypersensitive carotid sinus and hypoglycaemia are other causes of collapse. At altitudes above 28 to 30,000 feet, dysbarism is the most common cause, next to hypoxia. Dysbarism refers to the expansion of gases in the enclosed body spaces and liberation of gases, chiefly nitrogen, in the blood and tissue fluids. Several cases of collapse at high altitude due to dysbarism are on record.

Collapse due to dysbarism is associated with other symptoms of this syndrome. These may be in the nature of severe abdominal pain due to expansion of gases in the gastro-intestinal tract, "bends", disturbances of sensations, skin reactions, disturbances of neuro-muscular mechanism and of special senses; all of which fall under the category of decompression sickness and result from liberation of nitrogen bubbles in the body.

The exact mechanism of collapse due to dysbarism is not known. Primary shock is considered to be reflexly produced by painful impulses from abdomen, limbs or chest. Liberation of nitrogen bubbles in the central nervous system, which has a large fat content, is believed to be responsible for the variety of symptoms of decompression sickness, and the associated spasm of vessels particularly of the medullary centres may be the factor producing the state of collapse. Blocking of the smaller branches of the pulmonary artery is known to produce reflex vasoconstriction of the coronary vessels and generalised vasodilation of the systemic vessels. The latter is designated "the pulmonary relief reflex". It is therefore possible that collapse may result from the obstruction of pulmonary vessels by nitrogen emboli.

Results of autopsy on seven fatal cases after decompression have been described in the U.S. Air Force. Most of the cases revealed varying degrees of haemoconcentration,

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pulmonary oedema, hydrothorax, hydropericardium, cardiac dilatation, congestio viscera and changes in the brain suggestive of acute ischaemia.

Case History

SUBJECT: Flt. Lt. A. B. C. G. D. (P)
Age: —24½ years
Ht.: —71½ inches.
Wt: —196 lbs.

Total flying hours:—1,500
Liberator:—1,000 hrs.
Vampire : —44 hours.

The officer reported to Station Sick Quarters, Air Force Station, New Delhi, at 08 hrs. on 5.6.57 for a Decompression Chamber Test prior to proceeding to U.K. for conversi training in Canberra Jet bombers.

Pre-run - Medical examination was carried out at 0830 hours.

Personal History - An obese individual, weighing 2 stones more than norma non-vegetarian, smokes about 10 cigarettes a day, takes liquor occasionally, does not pla any particular game. For the past few months he was trying to reduce weight though il advisedly i.e. by periodic starvation. Married and has no domestic or financial worries Happy in squadron life and likes his job.

Previous history - Officer did not give a history of any relevant illness in the past His medical documents, which arrived later revealed the following:—

(a) He was involved in a motor cycle accident in November 1953, and was admitted to hospital for cerebral concussion for a period of 2 weeks. He was boarded at Air Force CMB after a month and was declared fit for flying duties. Investigations carried out during this period included a psychiatric interview and an EEG recording, both of which were normal.

(b) Suffers from occasional attacks of headache mainly frontal in nature and feels relieved with one codopyrin tablet each time.

He actually took one codopyrin tablet without the knowledge of the medical officer for a very mild headache before getting inside the chamber.

Results of Medical Examination

Temp :—97 °F.
Pulse :—72/mt. Vol and tension normal. Rhythm regular
Blood Pressure:—S/D-120/82 mm Hg.
Heart:—Size and sounds - normal
Lungs:—Clinically normal
Ears:—Normal
C.N.S:—N.A.D.
Found fit to undertake a chamber flight.

Chamber Record

On completion of briefing by the MO he was taken inside the chamber. Personal protective equipment and the chamber oxygen assembly were checked and found fully serviceable.

The medical observer took the first pulse reading at ground level. It was 74/mt. The chamber was then taken to 5,000 ft. altitude and brought down to the ground level at 3,000 ft/mt. The ear clearance test was normal. Oxygen drill was performed and the supply was then switched to "High Flow".

The chamber started ascending from ground to 20,000 ft. at 3,000 ft/mt. and from 20,000 ft to 30,000 ft. at 2,000 ft/mt. The last 7,000 ft. was made at 1,000 ft/mt. and chamber levelled off at 37,000 ft. at 11.30 hrs.

The subject showed no abnormal reactions during the first 50 minutes stay at altitude and the pulse recordings taken at 10 minute intervals showed little or no fluctuation.

At the 51st minute he experienced slight giddiness, ticklish sensation in the throat followed by few bouts of short dry cough.

Emergency oxygen was immediately turned on and the oxygen equipment was checked by the observer. There was no relief of symptoms and the subject complained of of a fainting feeling and slight blurring of the vision. He was bathed in cold sweat. Pulse was 50/mt. V.T. - Poor.

The chamber was immediately brought down to 22,000 ft. at 6,000 ft./mt. At this altitude most of the symptoms disappeared and he felt almost normal. Pulse recorded was 74/mt. The chamber was then brought down to ground level at 3,000 ft/mt. Emergency oxygen was given during the whole period.

On examination:—

Pulse:— 84/mt. V.T. normal. Rhythm regular.

Resp:—18/mt.

Blood Pressure:—S/D - 120/80 mm. Hg.

Heart, Lungs and Central Nervous System examinations revealed no abnormality. Ophthalmoscopy was not done.

Patient was normal except for a complaint of slight headache. Oxygen administration was continued.

At 14.30 hrs after about 1½ hrs, a sudden fall in blood pressure was noticed.

S/D:—80/60 mm. Hg.

Pulse:—104/mt. Volume and tension - Poor.

Pt. appeared to be slightly restless. There was sweating and complaints of slight headache and a feeling of tiredness. An occasional short dry cough was noticed. There was no pallor or cyanosis.

Heart, Lungs and the C.N.S. were examined. No abnormality was detected.

In view of the hypotension and headache it was decided to remove him to hospita
He was taken there within half an hour. The following observations were made in the hos
pital and treatment given.

At 16.30 hours - Complaints same as above.

Temp 98 °F. Pulse:—104/mt. V.T.:—Low B.P.:—S/D - 90/70 mm. Hg.
Heart, Lungs and C.N.S. - N.A.D.

Treatment Instituted:—

(a) Continuous inhalation of 100% oxygen.
(b) Coramine 1.7 c.c. I.M.
(c) I.V. Glucose saline - 40 drops/minute.

18.15 hours

Patient was conscious and talked rationally. Hands and feet cold.

Pulse:—90/mt. Resp:—20/mt. B.P.:—S/D - 100/80 mm Hg.
Heart Sounds:—Regular, though distant. No bruit.
Respiratory System:— Dry, irritating, cough - Clinically the chest was clear.
Cranial Nerves:—Normal.
Treatment:—Noradrenaline - 4 mgm/pint. Other treatment to continue.

18.45 hrs

B.P:—S/D - 110/80 mm Hg.
General condition was unchanged. Heart sounds regular, though distant.

20.00 hrs

Patient still restless.
Pulse - 100 /mt. V.T. Poor. B.P:—S/D - 106/70 mm. Hg.
Treatment:—Noradrenaline in glucose saline. 40 drops/minute.
Oxygen inhalation continued.

23.00 hrs

Patient quiet. Temp:—97 °F. Pulse:—110/mt. V.T. improved. B.P:—110/72mm. Hg.
Treatment continued.

6 Jun. '57 10.00 hrs -

Patient asymptomatic (22 hrs after the onset)
General condition:—good
Pulse:—88/mt.
Afebrile.
B.P.:—S/D - 130/80 mm. Hg.
Heart, Lungs, Abd. and C.N.S:—N.A.D.

8 Jun. '57

Investigations

Fasting Blood Sugar:— 55 mgm %.

E.C.G:—N.A.D.

X Ray chest:—The Rt. Heart was more prominent than normal. No evidence of posterior enlargement of the chambers after Ba meal swallow.

Urine:—Urine chloride 6 gms/litre

T.L.C:— 5,600 c.m.m. D.L.C:—65/30/2/3/. No MP. Hb:—15.5 gm.%

B.S.R:—25 mm/1st. hour (Wintrobe)

Patient was asymptomatic for the next five days and was discharged on 12.6.57 as a case of decompression sickness.

Discussion.

There seems to be little doubt that the circulatory collapse in this case was a manifestation of altitude dysbarism. This is strongly suggested by the appearance of symptoms in an obese individual after about one hour at a simulated altitude of 37,000 ft. and their reappearance after a lucid interval of about 1½ hrs. It was however necessary to exclude other causes of circulatory collapse, not only because the effectiveness of emergency measures depends upon the correct diagnosis but also for assessing the individual's suitability for flying duties at altitude.

There was no evidence that hypoxia had contributed in any way to the production of symptoms. The oxygen system had been checked before, during and after the run. Moreover, the persistence of symptoms after the oxygen was turned on to emergency, excludes the possibiltiy of hypoxia. There was also no impairment of consciousness, which is always associated with severe hypoxia.

Hyperventilation can also be ruled out. No increase in the rate or depth of his breathing was observed and the collapse was not preceded by the usual symptoms of abnormal sensory phenomena and muscular spasms. Further the post-decompression fall in blood pressure cannot be explained on this basis.

Coronary insufficiency, hyper-sensitive carotid sinus and hypoglycaemia were excluded on subsequent examination.

As regards the physiological mechanism underlying the collapse in this particular case, it is difficult to say much except that the liberation of nitrogen bubbles in some part of the body had a possible role to play. The reflex effects of pain and 'bends' or abdominal distension are often manifested in collapse but in this case there was no pain apart from slight abdominal discomfort. Formation of nitrogen bubbles in the central nervous tissue and associated spasm of the vessels in or near the medullary centres has been considered as a cause of collapse but in this case localisation of such affects to the vasomotor centres only, is hard to postulate.

It is, however, probable that air embolism of the pulmonary vessels may have resulted in generalised vasodilation through the pulmonary relief reflex.

Conclusions

This is the first case of severe neurocirculatory collapse at altitude in the I.A.F. With the introduction of flights, actual and simulated, at high altitudes, reactions of this nature may be more commonly experienced. All cases experiencing severe 'bends', 'chokes', skin symptoms, C.N.S. involvement, circulatory collapse etc. should be considered serious. Immediate hospitalisation and early institution of treatment improves chances of recovery.

Observation for 2-4 hours after flight or decompression chamber runs is important in early detection of delayed reactions. Severe neurological reactions have been known to occur within 15 minutes to many hours after descent.

A brief outline of suggested treatment is: —

Descent

Chamber should be brought down to ground level as soon as possible.

Observation

In mild cases 2-4 hours observation at SSQ/Resuscitation Room is indicated. In serious cases immediate hospitalisation is advised.

Bed Rest

½ hourly recordings of pulse, blood pressure and respiration.

Oxygen

Continous 100% oxygen.

Use of drugs

Serious cases are therapeutic problems. For example, if vasospasm is thought to be responsible for ischaemia of the brain and the cause of neurological symptoms then it would seem logical to use vasodilators such as I.V. papaverine and aminophylline. The danger of using vasodilators is that a circulatory failure may be precipitated.

Barbiturates may be used particularly for patients with neurological symptoms.

There is some evidence to the effect that spinal tap is beneficial in these cases.

Ice and alcohol sponging in addition to routine treatment may be necessary for pyrexia.

Summary

I have presented the clinical history of a case of post-decompression shock and have reviewed briefly the existing knowledge of neuro-circulatory collapse at altitude.

The physiological training programme of all aircrew and the adoption of standard methods for pre-selection prior to conversion to high speed and high altitude flying have brought such reactions into focus.