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A DIVING CASUALTY SUGGESTING AN EPISODE OF THORACIC SQUEEZE:

A Case Report

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LT M. B. Strauss, MC, USNR and LCDR P. Wright, MC, USNR

Bureau of Medicine and Surgery, Navy Department
Research Work Unit MF12.524.006-9025.36

Released by:

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COMMANDING OFFICER
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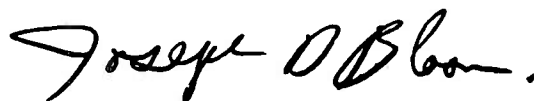
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SUMMARY PAGE

THE PROBLEM

To document an unusual diving accident which happened to an experienced diver while engaged in recreational diving. The report of this fatality, which was probably due to thoracic squeeze, is presented to inform physicians and diving supervisors of this potentially catastrophic diving problem, and to inform diving enthusiasts of this potential danger inherent in the aquatic environment.

FINDINGS

A diving accident characterized by pulmonary congestion and edema; interstitial and intra-alveolar hemorrhage is described. The clinical course was marked by transient improvement and incomplete recovery; then followed shortly thereafter by progression to a fatal outcome. The clinical and necropsy findings are discussed in relationship to the phenomenon of thoracic squeeze.

APPLICATIONS

The information presented in this report should be given wide dissemination among diving enthusiasts to make them aware of the potential danger of thoracic squeeze, as well as to physicians and diving supervisors who may be presented with responsibility for this type of diving casualty.

ADMINISTRATIVE INFORMATION

This case report was submitted to the Submarine Medical Center by the authors as qualified submarine medical officers wishing to record in the literature of submarine medicine a case report of thoracic squeeze. It was approved for publication as Report No. 36 on BuMed Research Work Unit—MF12.524.006-9025 and designated as Submarine Medical Research Laboratory Report No. 584, under date of 18 June 1969.

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ABSTRACT

With the rapid development of skin, scuba, and saturation diving, the medical profession and established diving organizations have made a concerted effort to inform diving enthusiasts of the potential dangers one may encounter in the aquatic environment. However, the phenomenon of thoracic squeeze is only cursorily discussed in recognized diving texts, and a review of recent diving literature failed to disclose any recorded cases of this medical emergency. It was therefore deemed valuable to present the following case report and discussion, with the idea of informing the physician and diving supervisor of this potentially catastrophic diving problem.

An unusual diving accident characterized by pulmonary congestion and edema, interstitial and intra-alveolar hemorrhage is described. The clinical course was marked by transient improvement and incomplete recovery; then, followed shortly thereafter by progression to a fatal outcome. The clinical and necropsy findings are discussed in relationship to the phenomenon of thoracic squeeze.

A DIVING CASUALTY SUGGESTING AN EPISODE OF THORACIC SQUEEZE:

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INTRODUCTION

With the rapid development of skin, scuba, and saturation diving, the medical profession and established diving organizations have made a concerted effort to inform diving enthusiasts of the potential dangers one may encounter in the aquatic environment. However, the phenomenon of thoracic squeeze is only cursorily discussed in recognized diving references.^{11,12} Review of recent diving literature has failed to disclose any recorded cases of this medical emergency.¹⁰ The following case report and discussion is presented with the idea of informing the physician and diving supervisor of this potentially catastrophic diving problem.

Case Report:

R. C. (28-year-old Lt. USN), an experienced underwater demolition team diver, was engaged in recreational diving at Subic Bay, Republic of the Philippines, with another UDT diver. They were performing deep breath-hold dives in water which had a maximum depth of eighty feet. They dove alternately so that each could observe the other. After a series of dives without incident, the companion diver first observed R. C. "floating" face up in a semiflexed position at a depth of forty feet, the estimated limit of visibility from the surface. R. C. appeared to be ascending slowly and was immediately brought to the surface by his partner. Total time of submersion was less than three minutes. Upon surfacing, R. C. was unconscious, apneic, and bleeding frothy bright red blood from the mouth. Five mouth-to-mouth respiratory exchanges were performed and the victim was carried to a small craft anchored fifty yards away. Examination by a physician with special training in diving medicine, within ten minutes of the accident, revealed a deeply cyanotic, unconscious patient with frothy red hemoptysis, irregular respirations,

and intermittent generalized clonic seizures. Conservative care, including postural drainage, gentle restraint, and maintenance of an oral airway was instituted while the patient was promptly transferred to a recompression facility. Soon after arriving at the recompression facility, approximately thirty minutes after the accident, R. C. improved substantially while breathing air at ambient pressure. It was elected not to pressurize the chamber. During this interval the patient gradually regained consciousness and became coherent in speech. His blood pressure and pulse rate were normal. The peripheral cyanosis reflected in lips and nail beds had cleared. Periodically, the patient would cough up and expectorate small amounts of clotted dark red blood. Intermittent breathing of one hundred percent oxygen was poorly tolerated, for it provoked episodes of coughing. Resonant rales were heard throughout the chest but were more accentuated in the right hemithorax. Ten minutes later, the patient's pulse rate gradually increased to 120 per minute. Breathing 100% oxygen, which was better tolerated at that time, lowered the rate to 90 per minute. The patient, while intermittently breathing oxygen, was transferred by ambulance to the naval hospital. During the latter portion of the ambulance trip, R. C. complained of increasing difficulty in breathing and said he was "feeling bad again." At hospital admission, approximately one hour after the accident, peripheral cyanosis and tachycardia were again present. The blood pressure was 110/70. One hundred percent oxygen was administered with positive pressure ventilation via a naso-tracheal tube. Ringer's lactate was infused intravenously. Moderate amounts of bright red frothy blood were aspirated from the naso-tracheal tube. Auscultation revealed loud resonant rales in all lung fields. The patient's condition continued to decline despite these measures and hypotension gradually developed. Eight hundred mil-

liliters of plasma and 1250 ml. of Ringer's lactate solution were transfused over a period of 90 minutes but failed to counteract the tachycardia and declining blood pressure. The patient gradually lost consciousness. Cyanosis continued to be unaffected by positive pressure ventilation with oxygen. Approximately three hours after the initial insult, cardiac arrest occurred. The arrest was refractory to all resuscitative measures and the patient expired.

Post Mortem Examination

Necropsy examination revealed no evidence of external injury or suggestion of bites or stings by venomous sea animals. Pertinent findings were confined to the lungs which weighed approximately three and one-half times their normal weight (right lung: 1250 grams; left lung: 1150 grams). On gross examination, the lungs were dark red and hemorrhagic throughout. Only slight crepitation was present. Cut sections revealed a consolidated purplish parenchyma which exuded large amounts of bloody fluid. Microscopic examination disclosed congested alveolar capillaries and multifocal areas of interstitial and intra-alveolar hemorrhage throughout the lungs. These findings were consistent with a diffuse bilateral vascular injury with intravascular congestion, interstitial edema, diffuse disruption of small vessels, and intra-alveolar hemorrhage. These findings were not consistent with the post mortem picture of drowning.

DISCUSSION

The clinical findings, necropsy examination, and pathophysiology in this case fit the syndrome thought to result from a thoracic squeeze. This disorder is an unusual medical problem associated with diving in that it requires a considerable degree of expertise before conditions can be achieved to precipitate it. The breath-hold diver must have sufficient breath-holding capabilities to remain submerged for over a minute as well as great facility in rapidly clearing his ears and sinuses with descent. Theoretically, thoracic squeeze occurs when the air comprising the total lung capacity (after maximal inspiration on the

surface) is compressed by the increasing ambient pressures while descending to an amount less than the residual lung volume. Such pressure volume relationships would respond in accordance with Boyle's Law, namely $P_1 \times V_1 = P_2 \times V_2$. Since few divers know their lung capacities and carefully observed world record setting breath-hold dives have far exceeded the so-called total lung capacity/residual volume threshold, calculation of the threshold, in general, is an exercise in theory only.⁸ It has been estimated that in the "average" sized man, this limit would occur at 80 to 100 feet.

Historically, this condition was noted long before the self-contained breathing apparatus (scuba) was invented. In 1823, S. N. Smith stated, "... if the depth be considerable, the water on the breast and organs is so great that it occasions the eyes to become bloodshot and produces spitting of blood, and if the practice is persisted in, it most likely proves fatal." One may think of the thoracic squeeze injury as the converse of the air embolism disorder. In the latter disorder, lung tissue is contused and rent by overexpansion of lung tissue due to failure to exhale sufficiently while ascending. In thoracic squeeze, the alveoli have been compressed to their smallest non-collapsible volume (i.e., residual volume). As the pressure gradient increases, the "low pressure" alveolar capillaries become distended. Transudation, diapedesis, and finally rupture with hemorrhage into the interstitium and alveoli occur. Theoretically, if a large gradient is created rapidly enough, bronchioles and blood vessels could rupture simultaneously and air embolism could result. However, since one is dealing with a compressed volume of air rather than an expanding volume of air as in embolism, intravascular air emboli is much less likely to manifest itself. Production of explosive gradients, e.g., rupture of a submarine hull while "at depth" could cause a crushed chest, rupture of the diaphragm, and/or displacement of the viscera into the thorax.

The presented case raises four significant questions. First, how does one explain the appearance of a condition most likely to be a thoracic squeeze, occurring at a depth well

below the most conservative estimates of the victim's breath-hold diving threshold? Several explanations are apparent. His total lung volume could have been substantially reduced by failure to inhale fully before descending, expenditures of large amounts of air while descending because of difficulty clearing ears, sinuses, or face mask, or accidentally losing tidal volume due to coughing. A fifty percent reduction in the total lung capacity at the surface would reduce the depth threshold by one-half. While Linaweaver states, "considerable injury can occur in the absence of pain," Schaefer and Dougherty, in a personal communication, have noted in their observations that pain associated with thoracic squeeze appears before serious signs and symptoms are manifested.^{6,9} The explanation of shallow water blackout and loss of tidal volume while on the bottom is not adequate, for the unconscious patient could never passively exhale to a point greater than the residual volume of air in the lungs. If this happened after the diver successfully reached the bottom, the pressure gradient would become essentially zero and no lung injury should occur. On the other hand, if the patient lost consciousness, for example, while ascending, passively lost his tidal volume and then began to descend due to negative buoyancy, conditions to precipitate a thoracic squeeze are present. Judgment as to which explanation best accounts for the course of events that occurred in the case reported herein, is reserved. Since the victim was apparently ascending when rescued, loss of consciousness, passive loss of tidal volume, and descent to a point below the new depth threshold seems the less likely explanation.

The second question that arises is how does one account for the initial unconsciousness, hemoptysis, and clonic seizures? Clonic seizures are often associated with hypoxia, but usually the hypoxic insult is greater than three minutes duration. The possibility of traumatic rupture of air conducting structures as well as blood vessels and resultant cerebral air embolism is entertained. This was the rationale underlying the immediate transfer of the victim to a recompression facility. However, the spontaneous improve-

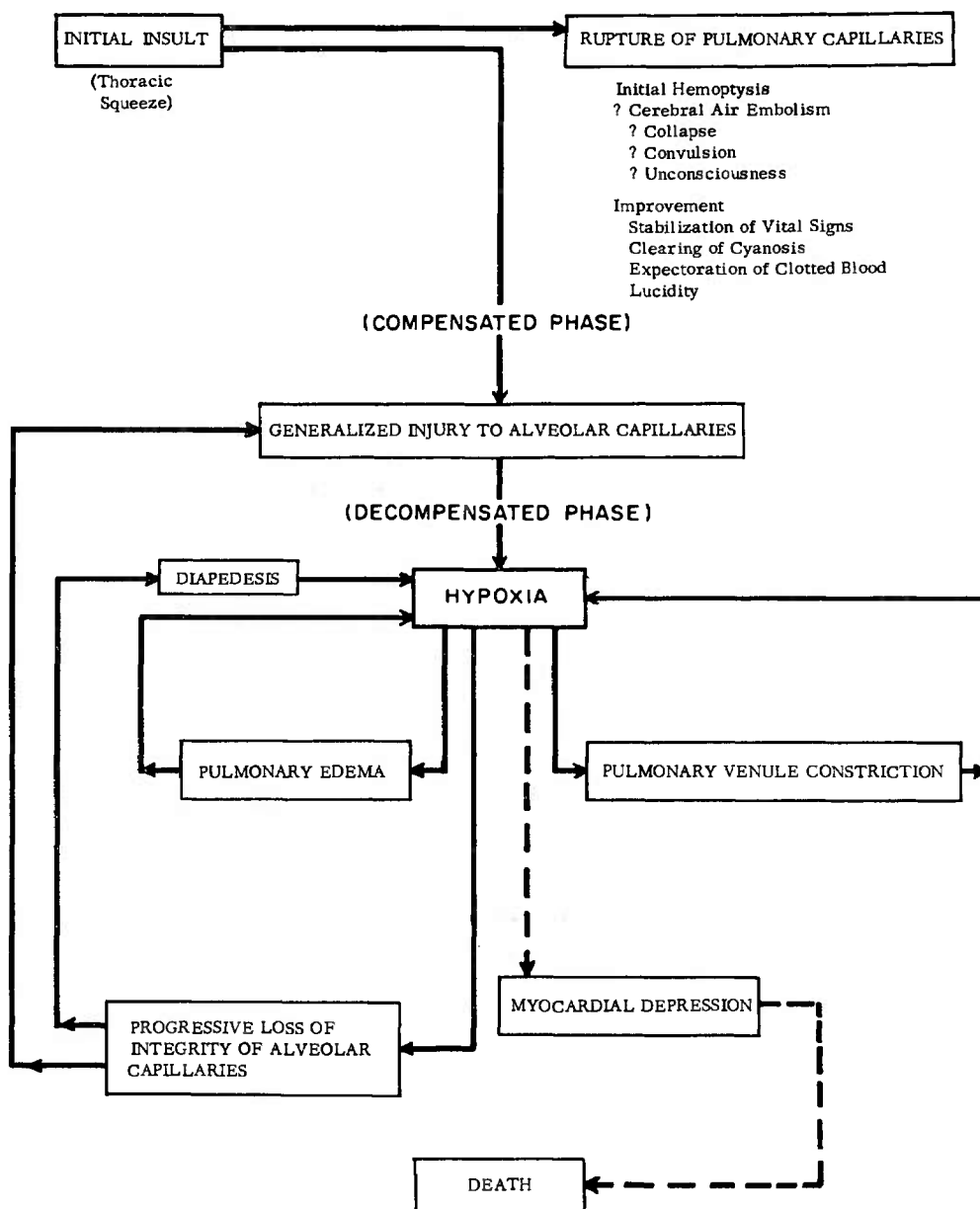
ment and absence of air emboli in the cerebral vasculature on sectioning the brain, makes this supposition difficult to prove.

Third, how can the initial transient improve while breathing air and receiving conservative treatment thereafter followed by a continuous gradual deterioration to a hypoxic death be explained? This course appears to reflect a progressive, alveolo-capillary diffusion block. After recovery from the "shock" of the initial insult, ventilation was adequate to supply the body's oxygen requirements and was characterized by the remarkable improvement. Subsequent decline occurred when the alveolo-capillary block became great enough to interfere with adequate respiratory exchange. When this became manifest a "vicious cycle" resulted. The deficient ventilation caused further hypoxia to the already damaged alveolar capillaries. The next step in the chain of events is pulmonary edema and diapedesis from the alveolar capillaries. These two sequelae cause further hypoxic insult. The deterioration after seeming improvement is not unlike the thirty to sixty minute latency periods noted before rapid fluid losses appear in the severely burned patient or the swelling response manifests itself in the newly traumatized joint. Diagram I is a graphic explanation of the proposed sequence of events reported in this case.

Finally, how can one rule out a case of drowning secondary to losing consciousness in the water? The short duration of submersion, immediate onset of hemoptysis and clonic convulsions is not typical of the drowning victim. The rapid recovery initially is consistent with near drowning, but one would expect sustained improvement (barring pneumotonic complications) until normal, rather than the deterioration observed in this case. Gross and microscopic necropsy findings of diffuse bilateral pulmonary congestion, interstitial edema and interstitial and intra-alveolar hemorrhage suggest a thoracic squeeze injury. Pulmonary edema is the predominant finding in the near drowning victim.⁴

It is interesting to note that post mortem intracardiac electrolyte studies revealed a mild hemoconcentration in the left side of the

DIAGRAM I: PROPOSED SEQUENCE OF EVENTS OCCURRING IN R.C.'s
INJURY, INITIAL IMPROVEMENT, DETERIORATION, AND
DEMISE



heart as compared to the right side. This is thought to be consistent with drowning.

Appropriate treatment for this condition would include cardiovascular support as indicated and supportive oxygen ventilation. Pneumonitis should be anticipated and treated appropriately. Hyperbaric oxygen could be beneficial in counteracting, to a limited degree, the alveolo-capillary diffusion block. The extent of alveolo-capillary block should be monitored by arterial gas studies. In theory, a heart-lung machine could provide adequate gas transfer to and from the blood, but is virtually impractical.

SUMMARY

An unusual diving accident characterized by pulmonary congestion and edema; interstitial and intra-alveolar hemorrhage is described. The clinical course was marked by transient improvement and incomplete recovery; then followed shortly thereafter by progression to a fatal outcome. The clinical and necropsy findings are discussed in relationship to the phenomenon of thoracic squeeze.

Addendum

In light of the previous discussion, it is interesting to compare the breath-hold diving abilities of humans and the "diving" mammals. Descents to 3,300 feet have been recorded in the sperm whale while harbor seals, which have approximately the same lung capacities as humans, have dived to 825 feet.² The record human breath-hold dive is two hundred and forty feet.⁸

Several adaptations have been observed in the diving mammal which make the extraordinary depth excursions possible. First, the lungs of seals and whales can collapse to a point where they become atelectatic and yet not separate from the chest wall.¹ Compensation for the decreased lung volume is made possible by the marked compressibility of their thoracic cages. This would ameliorate the intrathoracic pressure gradients between the chest wall, alveoli, and pulmonary vasculature which would otherwise result from descending. Another factor which may be important is the shunting of blood from the ex-

trémities to the central vasculature. This has been well documented in the oxygen conserving (diving) reflex. Seals are noted to have tortuous vena cavae and the diaphragm is thought to act like a sphincter on these vessels while diving.⁵ The blood from the dilated vena cavae could, in theory, compensate for the void produced by the collapsed lungs. Since blood is noncompressible and would directly transmit the hydrostatic pressure while descending, the pressure gradients which precipitate thoracic squeeze would not be produced.

These adaptations have not been observed in the human breath-hold diver. However, certain adjustments have been noted. First, a significant increment in the tidal lung capacity and a relative decrease in the residual lung volume is observed after repeated breath-hold diving.⁷ The greater the changes, the greater the depth threshold for thoracic squeeze. Large discrepancies between the calculated depth threshold based on the total lung capacity, residual volume ratio for world record breath-hold divers R. A. Croft and Jacques Mayol are apparent. Schaefer, et al., have demonstrated a shift in blood volume to the thorax with descents in water.⁹ If this blood displaced a corresponding amount of air in the lungs, a degree of protection from thoracic squeeze would be realized and, in part, accounts for the discrepancy between Croft's record descent to 240 feet and his predicted thoracic squeeze depth threshold of 197 feet.⁹ The factor of chest wall compressibility has not been measured in the human, but may afford a measure of protection analogous to that predicted in the diving manual.

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