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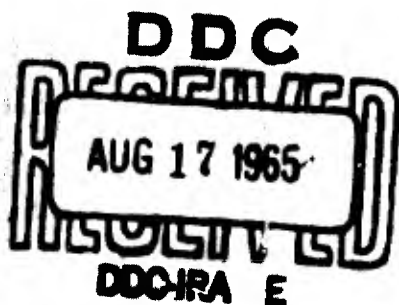
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AEROMEDICAL REVIEWS

THE ROLE OF THE ALTITUDE CHAMBER IN
THE DIAGNOSIS AND DISPOSITION OF PROBLEM
AEROMEDICAL CASES

Review 7-58



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**THE ROLE OF THE ALTITUDE CHAMBER IN THE DIAGNOSIS
AND DISPOSITION OF PROBLEM AEROMEDICAL CASES**

**CHARLES A. BERRY, Major, USAF (MC)
ARTHUR H. KING, Captain, USAF (MC)**

Department of Aviation Medicine

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THE ROLE OF THE ALTITUDE CHAMBER IN THE DIAGNOSIS AND DISPOSITION OF PROBLEM AEROMEDICAL CASES

The altitude chamber has been used in the training of airmen since Paul Bert trained Tissandier and his companions prior to their ill-fated balloon flight in 1875 (1). In some countries it has been utilized in the therapy of pertussis (3). Today it is used by the flight surgeon as a diagnostic aid in much the same way that the ECG, the EEG, and other laboratory procedures are used. It provides a medically controlled environment where the ills of the flier can be evaluated without subjecting him to the actual in-flight hazards and where accurate monitoring can be accomplished without difficulty. Heath (5) described the use of this device during World War II.

Evaluation by means of the altitude chamber may then be correlated with the history and physical examination of the patient in arriving at a proper diagnosis and disposition. This paper discusses the use of this diagnostic tool in relation to several cases seen in the Aviation Medicine Consultant Service at the School of Aviation Medicine, Randolph Air Force Base, Tex.

CHAMBER FLIGHT PLANNING

The "flight" must be carefully planned and individualized for each patient. It should attempt to accurately simulate the conditions producing the incident for which the patient was referred. Thus the rate of ascent or descent, peak altitude, and time at altitude vary for each "flight." In differentiating organic from functional symptoms it is frequently necessary to misrepresent the actual altitude to the patient. This is done by creating the noises usually associated with chamber ascent or descent and controlling an inside altimeter and rate-of-climb indicator. This arrangement allows the patient to remain at or near ground level and still have most of the impressions of being at altitude. Since a single uneventful chamber "flight" may not indicate that the patient will be symptom-free on future altitude exposure, multiple "flights" may thus be indicated. The conclusions reached on the basis of a single flight are tenuous, at best, for many conditions.

Presented at the annual meeting of the Aeromedical Association, Washington, D. C., March 1958.



The equipment necessary to conduct diagnostic chamber "flights" may include only the simple altitude chamber or the chamber combined with such specialized items as x-ray machines, electroencephalograph and electrocardiograph recorders, gas-sampling equipment, and respirometer, See figures 1-4. The chambers used by the Physiological Training Units on many bases will serve very well for diagnostic chambers. The equipment, as well as the flight plan, must be tailored to individual cases.

The personnel necessary to conduct diagnostic flights consist of the usual chamber operating crew and at least one flight surgeon for outside monitoring. Certain cases require the presence of an additional flight surgeon as inside observer.

The usual recovery room equipment and medications should be available.

CLINICAL CASES

The altitude chamber will be used more often to aid diagnosis as it becomes more readily available and as diagnosticians become familiar with its possibilities. During the three-year period 1955-1957 it was used to substantiate diagnoses in approximately 7 percent of the 1,000 consultation cases seen at the School of Aviation Medicine. Case histories are presented demonstrating its use in patients with ENT problems, head injuries, spontaneous pneumothorax or pulmonary blebs, pulmonary resection, hypoxia, hyperventilation, cardiac problems, and chamber reactions. All cases seen on the consultant service and reported in this paper were given a thorough medical work-up but only pertinent details are reported.

Ear, nose, and throat

The changing barometric pressure with altitude produces difficulty in ventilation of the middle ear and sinuses. This difficulty is one of the most frequent complaints among flying personnel. Our principal concern has been those individuals who are chronically unable to adequately ventilate their ears or sinuses and not those cases associated with an acute upper respiratory infection.

FIGURE 1

Patient in chamber instrumented for constant electroencephalogram, electrocardiogram, and respirometer tracings.

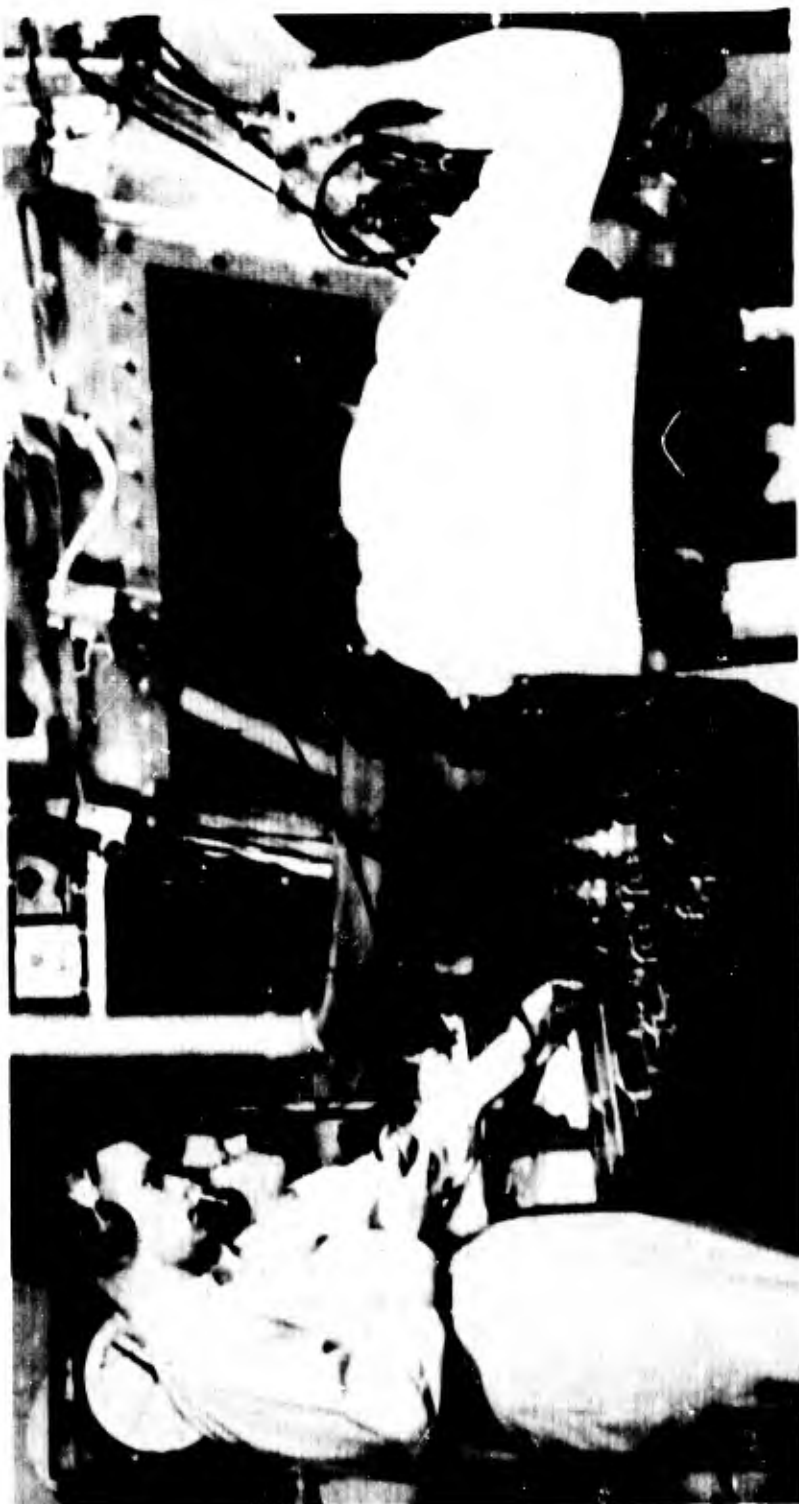


FIGURE 2

Flight surgeon monitoring electrocardiogram outside the chamber.

Case 1

This 24-year-old pilot with 290 hours of military flying time, 150 hours of which had been in the T-33, was referred for evaluation of repeated difficulty in clearing his ears on rapid jet penetration after transition to such aircraft one year before. He was politizerized on numerous occasions with good results. Five months preceding our examination he was treated by x-irradiation for hypertrophy of adenoid tissue around the eustachian tube orifices. He continued to have difficulty on rapid jet penetration.

Complete ear, nose, and throat examination was essentially negative except that both tympanic membranes showed limited movements on the Valsalva maneuver.

A chamber flight was first carried out in which the patient was supposedly taken to an altitude of 12,000 feet (although the chamber remained at ground level) and then dropped at a rate of 5,000 feet per minute. During this test, the noise was comparable to that experienced during a real chamber flight. He experienced no difficulty during this procedure although he was tense and repeatedly cleared his ears.

He was next actually taken to an altitude of 12,000 feet in the chamber and descent was accomplished at a rate of 5,000 feet per minute. This descent was interrupted on three occasions because the patient was unable to adequately ventilate his middle ear. His nasal mucous membrane was sprayed with $\frac{1}{4}$ percent neo-synephrine® on each occasion. After reaching ground level, examination showed both tympanic membranes to be dull, hyperemic, and retracted. He was politizerized with good results.

This patient was not returned to flying status as most people who develop chronic aero-otitis show evidence of this disease early in their career.

Case 2

This patient was a 24-year-old pilot with 555 hours of military flying time, 150 hours of which had been flown in the T-33. He was referred for a history of recurrent episodes of pain over both frontal sinus regions on descent from altitude. Nasal sprays and inhalers used before or during flight had no effect.

Ear, nose, and throat examination revealed only a hyperemic, boggy, and moist nasal mucosa. The nasal septum deviated to the left posteriorly with approximately 15 percent obstruction. Both middle turbinates were quite large. The patient was taken to an altitude of 26,000 feet in a low-pressure chamber. He developed no symptoms on ascent nor while the altitude was maintained at 26,000 feet for 25 minutes. On rapid descent, (5,000 feet per minute) he developed severe pain over the right



FIGURE 3

Flight surgeon monitoring the electroencephalogram outside the chamber.

frontal sinus region at 16,000 feet associated with definite lacrimation of the right eye. Descent was stopped and the patient's nose was sprayed with $\frac{1}{4}$ percent neo-syneprine® after which he was able to relieve the discomfort by performing the Valsalva maneuver. Descent to ground level was then uneventful. Sinus x-rays following this episode showed clouding of the right frontal sinus as compared with a preflight film.

As it was felt that the large middle turbinates might be obstructing the sinusoidal ostia, they were infracted under 4 percent topical cocaine anesthesia and another chamber flight was carried out the following day. The patient was taken to 16,000 feet and then descended at the rate of 5,000 feet per minute. The patient experienced no difficulties during this or repeated chamber flights.

Case 3

This patient was originally seen in January 1957 for evaluation following a right frontal sinusotomy performed in October 1956 for a mucocoele. A tantalum foil had been inserted connecting the sinus to the nasal cavity for drainage. In the immediate postoperative period this patient developed diplopia which was persistent at all times in all directions of gaze.

Examination revealed a marked paresis of the right superior oblique muscle and also signs of infection in the right frontal sinus. It was recommended that the patient be carefully observed until the ear, nose, or throat condition became static. In March 1957, the tantalum foil was removed.

Examination in January 1958 revealed no evidence of active ear, nose, or throat disease. Ophthalmologic examination was within normal limits. A diagnostic chamber flight was performed. The patient was taken to an altitude of 5,000 feet and returned to ground level without incident. He was then taken to an altitude of 43,000 feet and remained for approximately 3 minutes with positive pressure breathing. He experienced no difficulty on ascent, descent, or rapid decompression from 8,000 to 22,000 feet. This patient was returned to flying status.

Cardiac problems

Various reflexes activated by exposure to altitude may initiate cardiac arrhythmias. In patients where such symptomatology is suspected it is important to obtain an electrocardiogram while the patient is exposed to changes in barometric pressure. The following cases demonstrate the value of such a study.

Case 4

This 37-year-old senior pilot with 5,850 hours of military flying time was referred for evaluation of dizziness following a flight. Examination

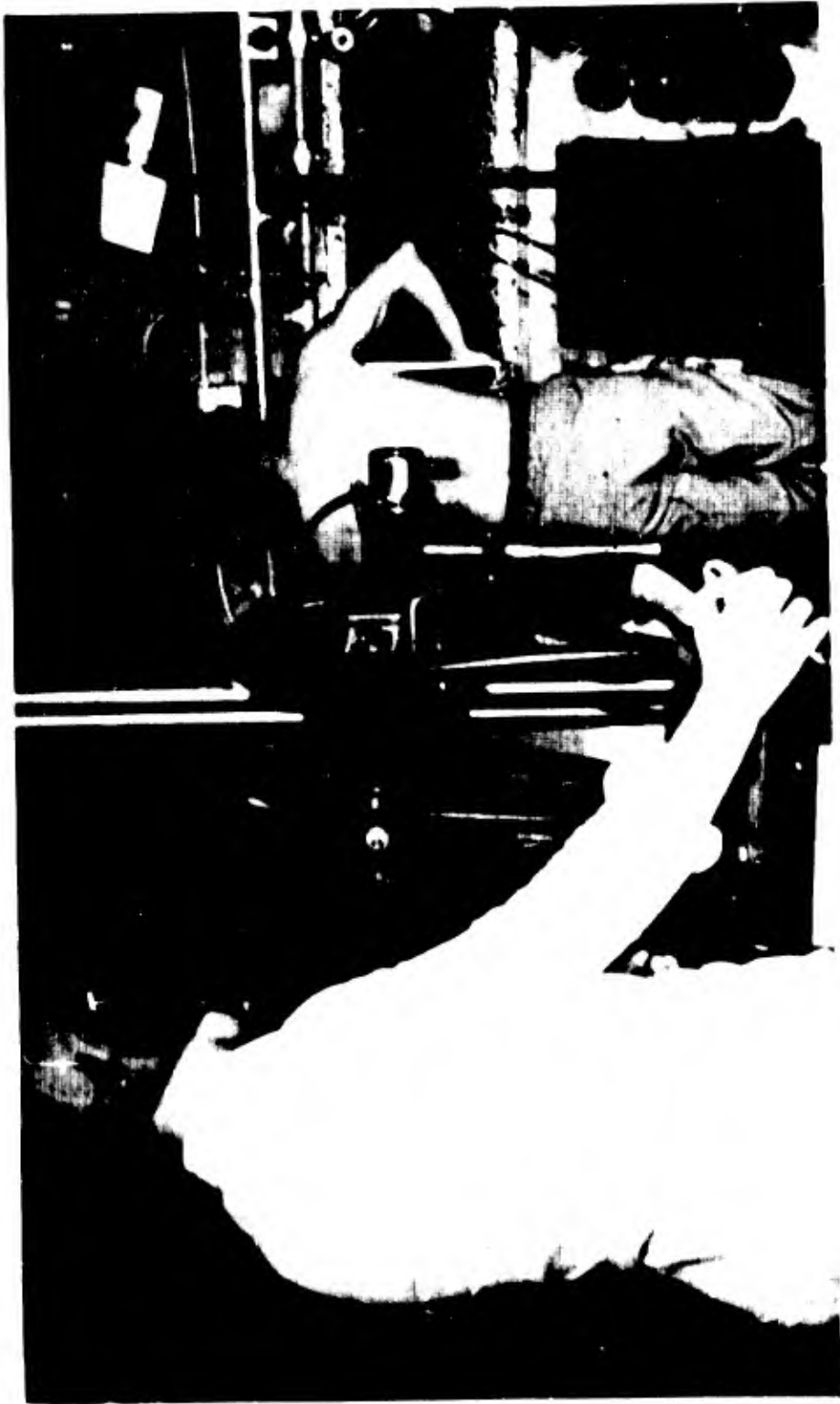


FIGURE 4

Patient in position for x-ray of chest taken at altitude.

at his base revealed an irregularity of the pulse, a hypersensitive carotid sinus on the right, evidenced by a near-syncopal episode during compression and a sino-auricular block on the ECG. The right carotid sinus area was irradiated in hopes of decreasing sensitivity.

Examination at the School again demonstrated a sino-auricular block. Holding the breath on deep inspiration caused no abnormal rhythms. On standing, the pulse rate increased and the rhythm became normal with no episode of sino-auricular block.

After the right carotid sinus was stimulated for a short time, marked vagal inhibition occurred although the patient experienced no abnormal sensations.

A diagnostic chamber flight with constant electrocardiographic monitoring was done. A resting tracing prior to ascent showed episodes of sinus pause with nodal escape. Immediately on ascent, the ECG showed marked vagal inhibition with increased periods of sinus pause. These were followed in most instances by nodal escape. Examples of fusion beats with an impulse originating from the auricle and the A-V node during nodal escape were seen with increasing frequency. See figure 5. Sinus pause occurred every second impulse in certain instances. One aberrant nodal conduction was noted on ascent. After an altitude of 25,000 feet was reached, the rate was increased and there was less evidence of sino-auricular block. During pressure breathing the pulse rate was increased and sino-auricular block was abolished. On cessation of pressure breathing the pulse rate slowed markedly and sino-auricular block with nodal escape increased in frequency. On descent the pulse rate slowed markedly and increased frequency of sino-auricular block with nodal escape was again noted. The significant finding from the cardiac point of view was the increased frequency of sino-auricular block with nodal escape during ascent and descent.

Since this patient presented evidence of a complicated rhythm with sinus arrest and nodal escape, as well as fusion beats and all were increased during exposure to altitude, he represented a hazard to flying safety and was not returned to flying status.

Case 5

This 41-year-old command pilot with 3,100 hours of total flying time, including 200 hours in jet aircraft, was found to have an abnormal electrocardiogram at the time of his annual physical. The tracings revealed a first-degree A-V block with occasional atrial premature beats occurring in a bigeminal fashion. X-rays of the chest were negative and revealed no cardiac enlargement.

Special electrocardiographic studies including Double Master's Exercise Tolerance Test, respiratory maneuvers involving breath-holding,

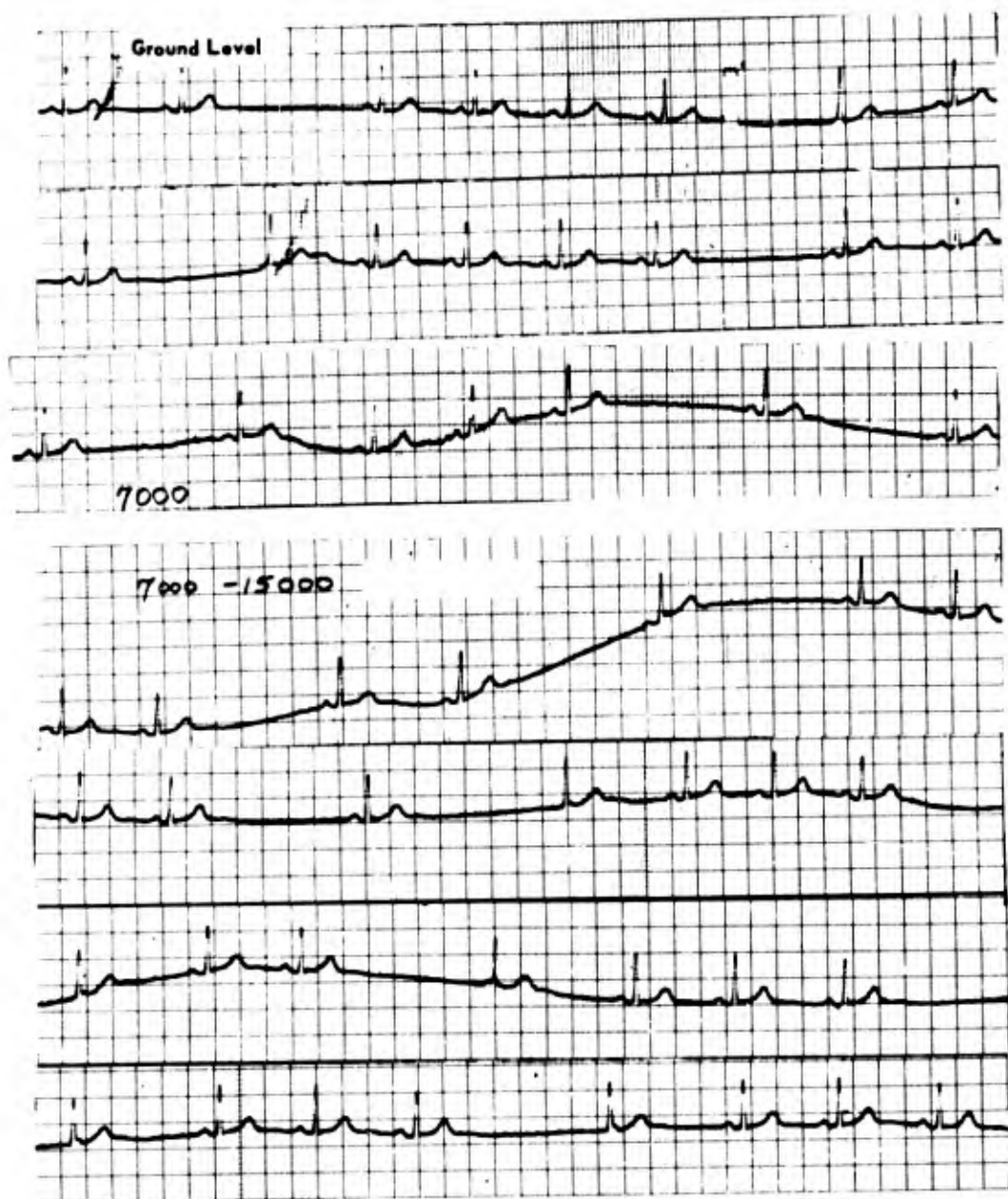
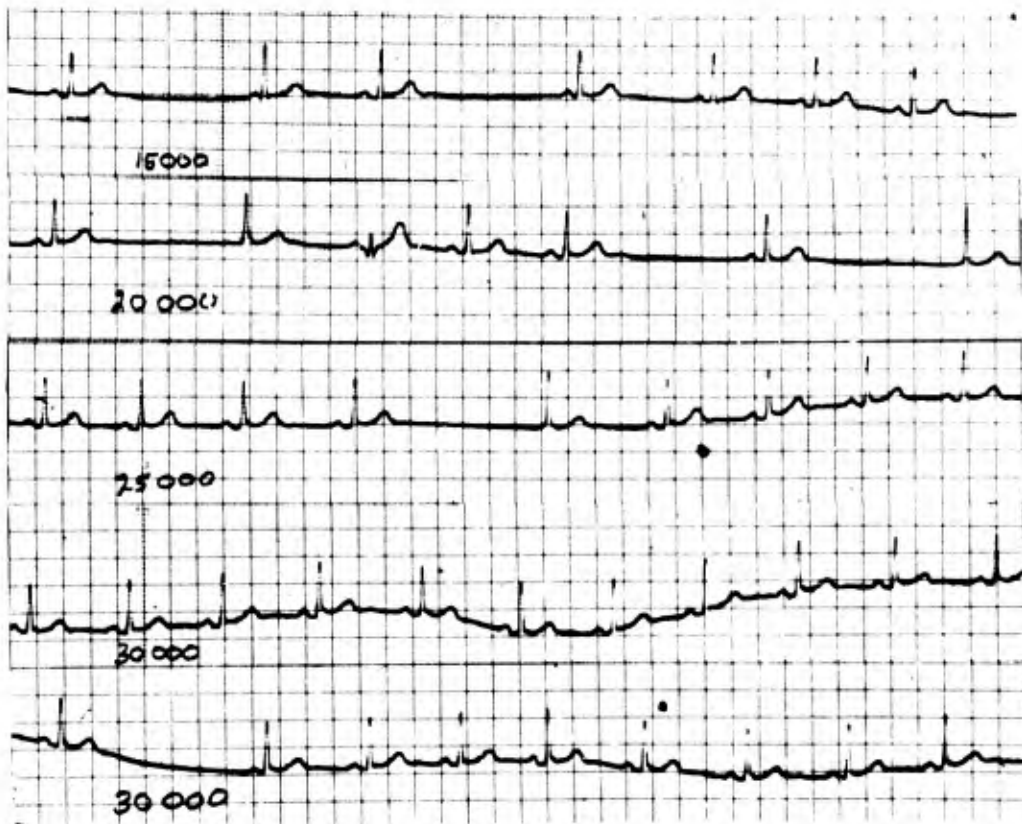


FIGURE 5

Electrocardiographic tracing showing sinus pauses with nodal escape prior to ascent and vagal inhibition with increased pauses and escape at altitude.

FIGURE 5 (Contd.)



deep breathing, and hyperventilation in the recumbent and standing positions and carotid massage and injections of gr. $\frac{1}{4}$, atropine I. V. were performed. The PR interval in these tracings was noted to be variable and became normal in duration with hyperventilation. No other significant findings appeared during these studies and no atrial premature beats occurred.

An electrocardiogram was obtained during an altitude chamber flight to 43,000 feet with pressure breathing. Prior to ascent an occasional atrial premature beat was noted. These prematurities became more frequent with ascent and at 30,000 feet occasional coupling first appeared. With further increase in altitude, transitory runs of bigeminy appeared and persisted intermittently thereafter through descent and at ground level. On the day following the chamber flight, there were persistent atrial premature contractions occurring in bigeminal fashion. The PR interval was prolonged for both the initial and premature complexes; the delay in A-V conduction was greater for the premature contraction than for the initial or normal contraction, indicating that the

initiating focus for the premature contraction was even farther from the A-V node than was the normal impulse focus or that the premature contraction impulses were traveling a different conduction pathway through the A-V node.

The presence of these conduction defects in the absence of organic disease or demonstrable hemodynamic embarrassment due to the arrhythmia did not constitute a medical contraindication for this patient's return to flying status.

Pulmonary problems

Normal pulmonary function is the best known and most obvious prerequisite to adequate physiologic adjustment to altitude. Many medical conditions which infringe on pulmonary function create no problem at ground level but must be thoroughly evaluated before airmen having them are exposed to the rigors of altitude. This evaluation includes pulmonary function studies at ground level and medical monitoring of a chamber flight with pressure breathing and rapid decompression. Patients referred to us because of spontaneous pneumothoraces and pulmonary resection but who show normal pulmonary function at ground level and at altitude (chamber) are recommended for return to flying duties. One problem encountered concerns pulmonary blebs. Patients with blebs or bullae may have normal pulmonary function and x-rays at ground level, but x-rays at altitude may demonstrate the blebs or bullae in a hyperinflated state. The basic physiology and anatomy of pleural blebs has been well described by Kjaergaard (6).

Case 6

This patient was referred for evaluation of a spontaneous pneumothorax. He was a 34-year-old pilot with 1,780 hours of military flying time, 400 hours of which had been flown in the B-47. Our evaluation revealed no significant findings except for x-rays taken in the altitude chamber which demonstrated blebs in the right upper lobe which expanded between ground level and 30,000 feet. The patient then had an apical wedge resection of the right upper lobe. Three months later he was again referred for evaluation. Pulmonary function studies were normal. Chest x-rays revealed tenting of the right leaf of the diaphragm and fibrotic tissue strands in the right upper lobe which were attributed to postsurgical changes.

A diagnostic chamber flight was then performed. After ascent to an altitude of 43,000 feet, positive pressure breathing was sustained for approximately 3 minutes. The chamber was then lowered to 27,000 feet where the time of useful consciousness was within normal limits. A

rapid decompression from 8,000 to 22,000 feet was accomplished without difficulty. X-rays of the patient's chest were taken at 10,000, 20,000, 30,000, and 43,000 feet. There was no evidence of pulmonary pathology.

Head injuries

In the interest of obtaining valuable information and assuring that such cases have been thoroughly evaluated in the flying environment, head injury cases are all given a diagnostic chamber flight. The details of instrumentation and procedure have been reported by Sarnoff and Haberer (8).

Case 7

This 34-year-old pilot skidded off a curve and fell down a 70-foot embankment in his automobile. He was unconscious for at least an hour. He had no clear-cut memories for the 2 or 3 days following the accident, although he remembers the accident clearly. He was evacuated to Fitzsimmons General Hospital where it was noted that he had a comminuted fracture of the right acetabulum as well as a depressed compound skull fracture. No neurologic abnormalities were noted. He underwent an open reduction of the acetabular fracture with internal fixation. He also had a débridement of the scalp and elevation of the depressed skull fragments of the right frontal bone. Six months later a cranioplasty was performed and an acrylic plate placed in the right frontal area. He was returned to flying status 1 year after the automobile accident. Approximately 1½ years later it was noted that the patient's span of attention was quite limited and mentation appeared somewhat slow. He was removed from flying status for this reason.

Physical and neurologic examinations were essentially negative. The patient was observed to be interested and cooperative. He hesitated before answering many of the questions asked of him and then spoke slowly, with little change in affect. On subtracting 13 from 300 he made several mistakes initially and had to begin again. At the second attempt he was very slow in his responses and began to make repeated mistakes and became confused.

The routine electroencephalogram was normal. The electroencephalogram recorded at altitude in the chamber did not reveal any abnormal cerebral reactions as long as the patient was receiving supplementary oxygen. However, when supplementary oxygen was discontinued at 27,000 feet the electroencephalogram almost instantaneously showed slow activity of 6-8 per second activity at greatly enhanced voltage as well as a few bursts of 10 per second. The patient began to hyperventilate and there was a rapid subsiding of the slow activity.

The patient's response to a chamber flight to 43,000 feet was not satisfactory. He tended to hyperventilate even when warned not to do so. This is often seen in patients with postcranial trauma.

The inability to concentrate over sustained periods plus the abnormal EEG responses to acute hypoxia were interpreted as representing permanent residua of cranial trauma.

Hyperventilation

Hyperventilation has long been known in ordinary medical practice as a factor complicating anxiety. Recent studies have shown its importance in the flying situation (2). The following case demonstrates the use of the altitude chamber in therapy and training as well as in diagnosis.

Case 8

This 23-year-old student pilot was flying at 9,000 feet in a T-28 and performing an instrument maneuver. He noted difficulty in concentrating on the instruments. He became confused and was unable to interpret or cross-check his instruments because he could not remember the instrument readings from one moment to the next. Noting the man's difficulties, the instructor called to him on the interphone. The patient later said that he could hear the instructor and understand his commands, but he was unable to carry them out. The instructor took over the controls and landed the plane.

Upon landing, the patient was noted to be "perspiring heavily." He was seen by the flight surgeon while still seated in the cockpit and it was noted that his back was arched and his eyes were fixed. His extremities and hands were in spasm and his respiratory rate was approximately 40 per minute. He was unable to speak and had difficulty moving but he was able to understand questions as evidenced by slight nods of the head. After 5 minutes of rebreathing into a cap, the patient was able to leave the cockpit without assistance.

A complete physical and neurologic examination was negative. Electroencephalogram was interpreted to be within normal limits, but demonstrated a labile response to extreme hyperventilation. The patient was evaluated in the altitude chamber with EEG, ECG, and respiratory rates being recorded simultaneously. At no time during the ascent to 43,000 feet or with pressure breathing did the patient breathe rapidly. It was apparent that the patient was aware of his respiratory rate and was controlling it voluntarily. After descent to 9,000 feet he was seated at a multiple-coordinator machine in order to simulate the conditions surrounding his hyperventilation episode. In the initial stages of mastering the operation of the machine his respiratory rate was increased

from 12 to 24 per minute. As he relaxed, the rate was stabilized and the depth of respiration was reduced.

Psychiatric evaluation revealed no marked abnormalities. The psychiatrist however, described the patient as having an obsessive compulsive personality pattern and as one who insists upon control of every situation. When faced with a situation which he cannot control he responds by hyperventilation. In this individual rapid respiration is apparently an anxiety equivalent.

As actual flights are the only true test of his ability to control himself, he was referred for a trial of flying under the close supervision of his instructors and flight surgeon.

Hypoxia

One of the most frequently reported in-flight incidents is hypoxia. During World War II this was a constant problem, and even though we now have excellent oxygen equipment and training programs, hypoxia still occurs with discouraging frequency. Little definitive information is available concerning the amount of brain damage following acute hypoxia episodes and even less concerning long-term residua. Chamber evaluations are valuable in determining susceptibility to hypoxia.

Case 9

This 26-year-old navigator was flying as an observer on a B-66 photoreconnaissance mission. One magazine of film had been used and it was necessary for someone to change the film. Ordinarily the change is done at 10,000 feet but, fuel being low, it was attempted at the flight altitude of 23,000 feet. The oxygen walk-around bottle was too large for the patient to carry through the crawl-way to the camera compartment. The pilot dumped the pressure allowing the patient to open the hatch leading to the crawl-way and to begin his journey to the camera compartment. About 5 minutes later, the pilot decided that something might well be wrong as he had heard nothing from the navigator. He brought the plane to a lower altitude and asked one of the crew members to make his way to the camera compartment. The patient was found to be unconscious. The rescuer attached the patient's mask and turned on the oxygen regulator. Shortly after the patient began to receive oxygen, he began to convulse and thrash about madly. The rescuer had to restrain him during the entire trip back to the base which was approximately 1 hour. He was immediately hospitalized where he was described as trashing about in a maniacal manner and screaming with a wild, primitive, very distressing cry. The remainder of the physical examination was negative except for an equivocal bilateral Babinski reflex. Two days later the patient became aware of his surroundings. He noticed no

difficulty with his mentation except for an inability to recall the names of close friends. This cleared within a few days. An EEG done 7 days after the incident was reported as having a basic rhythm of 8-9 per second low amplitude waves of irregular form. There were large amounts of low amplitude 6 per second activity most prominent in the parietal area bilaterally and occurring at times in bursts of a few seconds' duration. No focal abnormal asymmetry was seen. An ECG showed minor T-wave changes.

There was rapid improvement in the patient's mental status after the first 2 days. At the end of a month, he was able to speak in a normal fashion. There was definite ataxia of the upper and lower extremities which improved in time and he was able to write legibly. He complained of some dimness of vision early in the course of the disease. When he was first ambulated, his gait was hesitant and unsteady, but quickly improved to a point where it appeared to be normal. An EEG at the end of his hospitalization (approximately one month) was within normal limits. Within 1½ months all neurologic findings had disappeared except for a residual inability to perform fine coordination movements with his fingers and hands. The neurologist described him as being obviously nervous and restless and unable to sit still during the examination. Three months later he was no longer nervous and neurologic examination was normal. Electroencephalographic and electrocardiographic tracings were normal. Psychologic testing revealed no indications of organic deficit.

An altitude chamber flight to 43,000 feet was performed. It included pressure breathing for approximately 3 minutes, exposure to ambient air at 27,000 feet, and a rapid decompression. No abnormalities were noted on the EEG, ECG, and respirogram. Exposure of the patient to ambient air at 27,000 feet brought on marked increase in respiratory and cardiac rate. The electroencephalogram revealed some 6 per second activity which occurred after 1 minute. There was a rapid enhancement of the 2-3 per second slow wave dominating after 2 minutes. At this point, there was interference with useful consciousness as determined by failure in his ability to write consecutive numbers backward. This time of useful consciousness was within normal limits. After return to 100 percent oxygen the patient's ECG, EEG, and respiratory rate rapidly returned to normal.

It was recommended that this patient be returned to flying status and that he have psychologic testing every 3 years.

Another man referred for blurring of peripheral vision while stationed in LaPaz, Bolivia (12,500 feet), required a special chamber flight to determine his susceptibility to prolonged mild

hypoxia. He was taken to an altitude of 12,500 feet in the low-pressure chamber and maintained at this altitude for 5 ½ hours without oxygen. A flight surgeon served as observer and control. Both patient and flight surgeon walked the length of the chamber 10 times and performed 10 deep knee bends at hourly intervals and smoked five cigarettes during the "flight." Blood pressure, pulse, visual acuity, and peripheral visual fields were measured. An EEG was also done. No significant variations from the control values were noted.

Chamber reactors

Increased operational flying at altitudes in excess of 30,000 feet has produced a need for training larger numbers of flying personnel in the physiology of high altitude flight. This increase in exposure along with more knowledge and conservatism on the part of chamber operators has led to the reporting of more chamber reactors in recent years (4). It is necessary to differentiate the various types of chamber reactions and avoid classifying them all as decompression sickness, for this diagnosis has certain connotations as to disposition. This has been especially evident in the partial pressure suit reactors where an original diagnosis of "chamber reactor" implying decompression sickness was changed to "poor suit fit" after study.

Case 10

This 34-year-old senior pilot with 2,800 hours of military flying time, 2,050 hours of which had been flown in jet aircraft was referred because of a "chamber reaction."

This patient was being indoctrinated in the partial pressure suit. After 20 minutes at a maximum altitude the patient complained that he was having difficulty seeing the TV picture and hearing the sound. An alarm signal was given and the chamber was returned to a lower altitude. While descending, the patient described the sensation of faintness or sleepiness and the loss of sensation in his hands. At no time did he lose consciousness. An ECG, done before termination of the flight, revealed no abnormalities. He noticed return of all normal sensation after the suit was deflated. Special cardiovascular studies, electroencephalogram, and psychiatric evaluation were all normal. He was then evaluated by experts in assessing proper partial pressure suit fit. It was immediately apparent that the neck seal was too tight. The circumference of the neck seal was 12 inches, while the actual circumference of the patient's neck was nearly 15 inches. The suit also restricted a maximum thoracic inspiratory circumference by 30 percent. The patient

was redressed in the unaltered pressure suit, helmet, and gloves. He was placed in a chamber in the unaltered suit and taken to a maximal altitude where he rapidly developed duskiness and mild cyanosis of the face. He was returned to ground level after 10 minutes' exposure to altitude. He was immediately refitted in another suit of proper size, was placed in the chamber and taken to maximum mission profile altitude and stayed there maximum time with no complaints. No abnormalities were noted.

It was apparent that the combination of mild hypoxia at that altitude in the original run, with the added insult of marked stagnation of retinal and cerebral venous blood produced the visual disturbance and light-headedness he experienced.

Case 11

This 38-year-old senior pilot with 6,200 hours of military flying time was referred for evaluation 1 year after head injury received in an automobile accident. He did not lose consciousness but had retrograde amnesia for the 48 hours prior to the accident. A diagnosis of cerebral contusion was made. Neuropsychiatric consultation including psychologic testing was completely negative. His EEG was normal.

A diagnostic chamber flight, monitored throughout by EEG, ECG, and respirograms was then performed. The patient denitrogenated 20 minutes and ascended to 30,000 feet. Positive pressure breathing was started and he then ascended to 43,000 feet. It was noted on the ECG that the patient's cardiac rate was slowing and then atrial premature systoles appeared as intermittent coupling. Just before these extra beats occurred the patient noted a tingling sensation in his toes and finger tips. He thought that he might be hyperventilating and thus took a deep breath and held it. Then he heard the observing doctor request that the chamber be taken down.

The patient stated he felt "funny" at this time. It was during descent that the inside observer first noted that the patient was an ashen color and was perspiring profusely.

A thorough evaluation of the patient's records revealed that the cardiac rate diminished a significant time before any changes were noted in the EEG. The EEG changes which occurred reflected central nervous system hypoxia thought to be secondary to diminished cardiac output. Pressure breathing had probably overstimulated the pulmonary stretch receptors initiating cardiac slowing. An added insult was the further activation of these reflexes by taking a deep breath and holding it.

A second flight was performed after the patient was trained in the control of his respiration. This flight was uneventful.

This case demonstrates a point elucidated by Lamb et al. (7). Flying personnel who believe they are hyperventilating should not take a deep breath and hold it. In order to avoid potentiating this stretch reflex and producing the attendant vagal response, care should be taken to slowly reduce the respiratory rate.

Case 12

This 34-year-old flight engineer was referred for repeated episodes of pain in his right knee. His history was somewhat unusual in that he had no pain when decompressed unless his knee had been bothering him prior to a flight.

Physical examination was negative. Orthopedic consultation and x-rays were completely negative.

A diagnostic chamber flight was then performed. A sham chamber run was first obtained to evaluate any possible psychogenic factors involved in this problem. A sham altitude of 35,000 feet was attained while the actual altitude remained at only 12,000 feet. The patient even after exercise experienced no difficulties. The patient was then taken to an actual chamber altitude of 40,000 feet. Ten deep knee bends were done. Two minutes later the patient developed a Grade I pain in the right knee which progressed to a Grade II within 1 minute. The chamber was then lowered to 30,000 feet and the pain persisted as Grade I. On descent to 24,000 feet, he was completely free of pain. Denitrogenation had not been performed prior to this flight and a second flight was then attempted with adequate denitrogenation. The patient on the following day was denitrogenated for 1 hour prior to the flight. At 30,000 feet, 10 deep knee bends were performed. The patient was observed for approximately 3 minutes at this altitude. The chamber then ascended to 40,000 feet and 10 deep knee bends again were repeated. In one minute Grade I pain occurred in the right knee progressed to severe Grade III pain. A free-fall to 30,000 feet gave some relief. The chamber then descended to 24,000 feet where the pain completely disappeared. This patient experienced bends in the knee, which apparently were brought on by exercise. The patient's knee had been causing him no difficulty prior to either flight.

SUMMARY

The use of the altitude (low-pressure) chamber as a diagnostic tool in problem aeromedical cases was discussed. It provides a controlled altitude environment at ground level, thus providing greater safety and easier monitoring. Each "flight" must be tailored to the individual case and should closely simulate the actual conditions surrounding the referral incident. Case histories

of consultation cases from the School of Aviation Medicine have been presented. The chamber was an aid to diagnosis in the following types of cases: ENT problems, cardiac problems, pulmonary problems, hypoxia, hyperventilation, and head injuries.

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