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INFLIGHT LOSS OF CONSCIOUSNESS A CASE REPORT

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ABSTRACT

A case of inflight vertigo and loss of consciousness in a private pilot, flying alone, is presented. The differential diagnosis and the significance of the findings of 5-7 per second theta waves in his resting EEG and high voltage slow waves during caloric irrigation of his right ear are discussed.

Loss of consciousness can result in serious consequences when it involves a pilot in control of an aircraft. It is a manifestation of a variety of physiological and metabolic disorders.¹⁻⁴ Cardiovascular dysfunctions have long been under suspicion as a main cause and a number of investigations has been devoted to studying their relation to loss of consciousness.¹⁻⁶ At present the condition of the cardiovascular system of a pilot is under close surveillance both in military and civil aviation. Thus, syncope due to serious cardiovascular disturbances has become a decreasing threat to aviation safety. On the other hand, loss of consciousness due to nervous disorders is poorly understood and only recently have some studies been devoted to this subject.⁷⁻¹¹ To illustrate the importance of this etiology an unusual case is reported.

REPORT OF THE CASE

A 19 year old student pilot with aspirations of becoming a commercial pilot, and 105 hours of flying time, was completely asymptomatic until 11:00 a.m., April 13, 1962, at which time he experienced vertigo and subsequent loss of consciousness.

The evening before, being a "school night," was quiet and routine with no alcoholic beverages or dietary indiscretions. He did note some epigastric uneasiness with nausea before retiring. He slept well, as usual, from 1:00 p.m. to 6:55 a.m.; ate a usual breakfast of bacon, eggs, toast, and coffee and attended college classes from 8:00 a.m. to 10:50 a.m. There were no stressful events associated with these classes, in fact an exam was returned on which he made a better-than-expected grade. He denies taking any drugs during this time.

From school he drove immediately to the local airport, rented an Aeronca Champion which had recently landed,

started the engine and taxied out for take-off. At this time he was wearing tinted prescription spectacles and a sport shirt with the collar open, and had, to the best of his knowledge, his seat belt fastened.

The take-off was made to the SE on runway 12 at about 11:00 a.m. under clear skies. During the take-off roll the aircraft yawed and he noted that he could not reach the rudder pedals. He therefore leaned forward to adjust the seat but continued the roll. This adjustment was completed and a normal posture was assumed prior to lift-off.

He climbed at an unknown rate to 400 feet where, without a stall, the nose dropped abruptly to the horizon. He checked the altimeter, found his altitude in the 390's, looked out of the window to the left and back, then relaxed. At this point he noted lightheadedness, a sensation of back and forth movement of the control panel, and blurred vision. There was no diplopia, tingling, nausea, or tinnitus noted. This was followed by true vertigo felt in level flight which was less obvious with his eyes closed. He became quite anxious at this point and decided to land. He made two 180° turns and trimmed up the aircraft for final approach. His vertigo was gradually intensifying during this period. When about 10-12 feet off the ground, he reached to his left with his right hand (without turning his head) to cut the magneto switch. He remembers nothing else until two hours later when he was in an ambulance, receiving oxygen, en route to the local hospital. During this 2 hour period he remembers only a jolt.

The plane, at half throttle, landed level according to skid marks, then left the ground and turned 180° striking a fence at the end of the runway with the left wing and then the ground with the right wing. The landing gear was torn off, the cowlings damaged and the tail was bent. There were no witnesses to the accident. He was first missed after he failed to land at the end of one-half hour and the plane was first sighted 1 1/4 hours after the accident.

The first person on the scene, his instructor pilot, found him lying on soft ground with his feet still in the aircraft, appearing only asleep. The seat belt was open, not broken. The bridge of his glasses was broken. There was no pallor, sweating, vomitus, incontinence, or body movement (twitching or jerking). Respiration was described as slow and regular without associated sounds. The pulse was felt and described as strong. The rate was not counted but it

did not seem unusually fast or slow. There were no signs of injury but an ambulance was called.

He regained consciousness in the ambulance approximately two hours after the onset of unconsciousness. He had no vertigo, only a cold sensation of the nose and throat. (He was receiving O_2 by mask at the time.) The ambulance attendant noted the odor of vomitus but found no evidence of it.

He was taken to the emergency room of a local hospital where a B.P. of 160/70 and extreme nervousness were the only recorded findings. He received intramuscular injections of promethazine HCl, 50 mg., and meperidine HCl, 50 mg. He slept from 3:00 p.m., April 13, to 9:00 a.m., April 14, awaking with thirst, anorexia, nausea, and a headache (dull, bilateral, frontal without throbbing) which persisted until 5:00 p.m. that day. Symptoms remitted spontaneously and he has been asymptomatic to the present.

There is no evidence to support the statement by the attending physician that the airman had an upper respiratory infection with a middle ear obstruction creating dizziness and the black-out. The attending physician reported that symptoms cleared after antibiotic therapy which the patient denies receiving.

Past medical history reveals a brief loss of consciousness at age 10 due to trauma from a baseball bat, occasional external ear infections in the summer, and the usual childhood diseases without complications.

The family history is non-contributory.

The review of systems revealed only rare faint sensations on sudden change of position, an allergy to penicillin, spectacle correction of myopia with tinted lenses due to "hypersensitivity to light," mild dissatisfaction with present duties (part-time retail store assistant manager), occasional mild depression, and occasional fatigue upon awakening.

On physical examination on June 4, 1962, the airman weighed 113 pounds and was 64 1/2 inches tall. Distant vision was 20/40 in each eye with spectacle correction to 20/15 O.D. and 20/20 O.S. There was a 50 db loss in hearing by air conduction at 4000 and 6000 cps, A.S. and 6000 cps, A.D. determined by pure tone audiometry. The blood pressure was 104/50, recumbent. The resting pulse was 60 per minute which returned to 76 per minute after exercise. During an orthostatic tolerance test the blood pressure was 120/72 and the pulse rate 90 per minute after standing for three minutes. Bilateral caloric stimulation for 40 seconds with 28°C water produced strong nystagmus and dizziness with recovery in five minutes. Head movement during rotation produced symptoms of similar intensity and duration. The remainder of the examination, including the neurological examination, was within normal limits.

A neurological consultation was obtained on August 7, 1962, and no evidence of neurological disease was found.

Laboratory studies performed on June 5, 1962, were as follows: red blood count, 5,090,000 per cubic millimeter; hematocrit, 49 per cent; hemoglobin, 16.3 gm.; erythrocyte sedimentation rate, 3 mm. per hour; white blood count, 9,600 with normal differential; fasting glucose, 99 mg.%; two hour post-prandial glucose, 104 mg.%; and urinalysis, normal. During a five hour oral glucose tolerance test, performed on January 29, 1963, blood glucose levels were: fasting, 102 mg.%; 30 min., 135 mg.%; 1 hr., 99 mg.%; 2 hr., 105 mg.%; 3 hr., 73 mg.%; 4 hr., 43 mg.%; and 5 hr., 51 mg.%.

Radiographs of the skull and chest revealed no abnormalities.

Electrocardiographic studies during resting and working conditions were within normal limits.

During a cardio-respiratory evaluation test the resting blood pressure was 112/70; the pulse rate, 70 per minute; the maximum working blood pressure, 168/60 with a pulse rate of 180 per minute; and the maximum oxygen intake was 41.3 ml/kg/min. Passive tilting to 60° resulted in a minimum blood pressure of 96/80 with a pulse rate of 96 per minute. There was no indication of any tendency for orthostatic syncope.

On June 4, 1962, a flight test was given in a Cessna 172 by an instructor pilot accompanied by a flight surgeon. He was subjected to the following maneuvers without any admitted symptoms, pallor, sweating, or nystagmus: repeated duplications of the flight profile which preceded his onset of vertigo and unconsciousness; vertical reversements involving banks in both directions of approximately 90° with maximum pitch-up angles of 45°; lazy 8 maneuvers continuously for 4-5 minutes with speed changes from 70 to 120 mph, with roll angles and banks of 90°, and nose-up angles of 40-50°; and tight, steep turns (greater than 60°) with approximately 2 g for 2-3 minutes.

In the morning of June 5, 1962, an electroencephalogram was recorded. Disc electrodes were placed at FP_1 , FP_2 , F_7 , F_8 , C_3 , C_4 , T_3 , T_4 , O_1 , and O_2 . Clip-on disc electrodes were attached to the two ear lobes (A_1 and A_2). Fig. 1 shows the international nomenclature for the positions of the EEG electrodes. A_1 was used as reference electrode for all the left side electrodes and A_2 for right side electrodes. The subject was instructed to relax in a supine position in a semi-dark room. Recording was made with a Grass Model III electroencephalograph.

His resting record showed high amplitude occipital alpha waves, often above 100 μV and sometimes above 200 μV . The left occipital alpha waves (O_1 , A_1) were often slightly suppressed. A slight suppression was also noted on the right temporal records. However, the most significant finding was the appearance of theta waves (5-6 per sec.) shown in the FP_1 and FP_2 leads, often between 50 and 100 μV but sometimes above 100 μV . Less prominent theta waves were also noted in the C_3 and C_4 leads. These theta waves appeared while the subject was awake as indicated by the presence of continuous alpha waves. These abnormal waves appeared more often just before the subject became drowsy. However, when the subject was drowsy or asleep, theta waves were not observed.

The subject returned on August 7 (about four months after the accident) for a second study. The theta waves in the FP_1 , FP_2 , C_3 , and C_4 leads persisted and occasionally even appeared in the T_3 , T_4 , O_1 , and O_2 leads (Fig. 2). To determine the relative prominence of these waves on the scalp, the zero leads were employed. Figure 3 shows the appearance of prominent theta waves in F_7 and C_3 leads, using A_1 and A_2 as reference leads. Less prominent theta waves appeared in P_3 leads and almost none in occipital leads, indicating the more prominent frontal distribution of these waves.

To complete the study of geometrical distribution of the theta waves on the scalp, an EEG was recorded from F_7 , F_8 , F_9 , P_3 , P_4 , T_3 , and T_4 leads. The records are shown in Fig. 4. Theta waves appeared more prominently in leads F_7 , F_8 , P_3 , P_4 , T_3 , and T_4 and slightly less in F_9 and F_8 . There is some asymmetry in the prominence of the theta waves, the left side being more prominent.

A topographical distribution of the theta waves on the scalp is shown in Fig. 5. The area of more prominent theta waves is formed by linking the more prominent leads

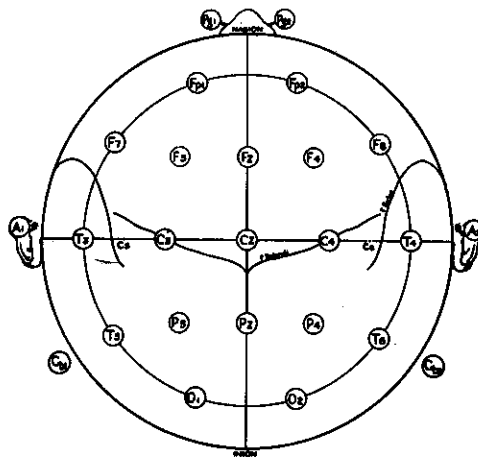


Figure 1. International nomenclature for EEG electrode positions.



Figure 2. Resting EEG showing the nearly sinusoidal theta waves, more prominent in Fp₁, Fp₂, C₃ and C₄ leads.



Figure 3. Resting EEG showing more prominent theta waves in F_z and C_z leads.

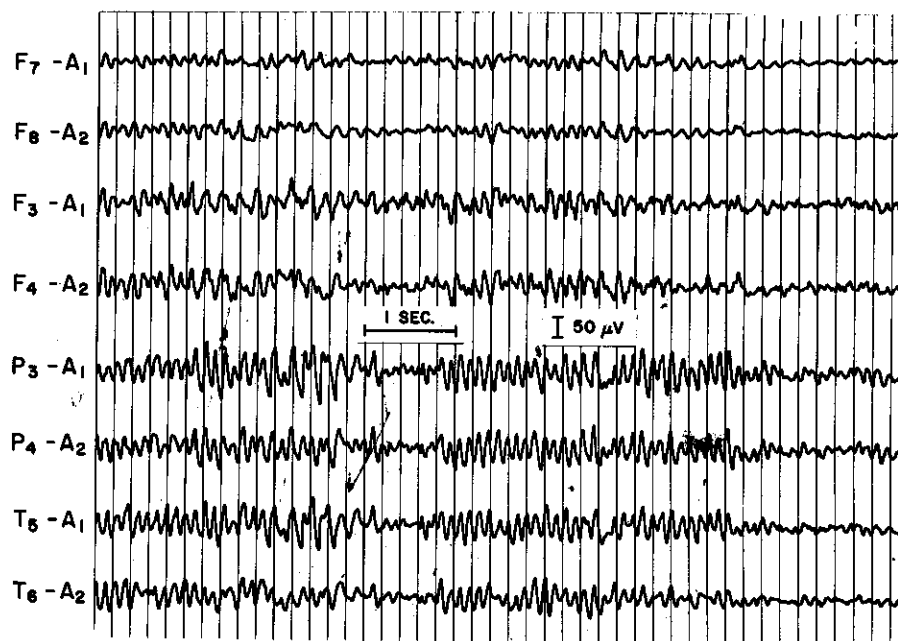


Figure 4. Resting EEG showing more prominent theta waves in F₃, F₄, P₃, P₄, T₃, and T₄ leads.

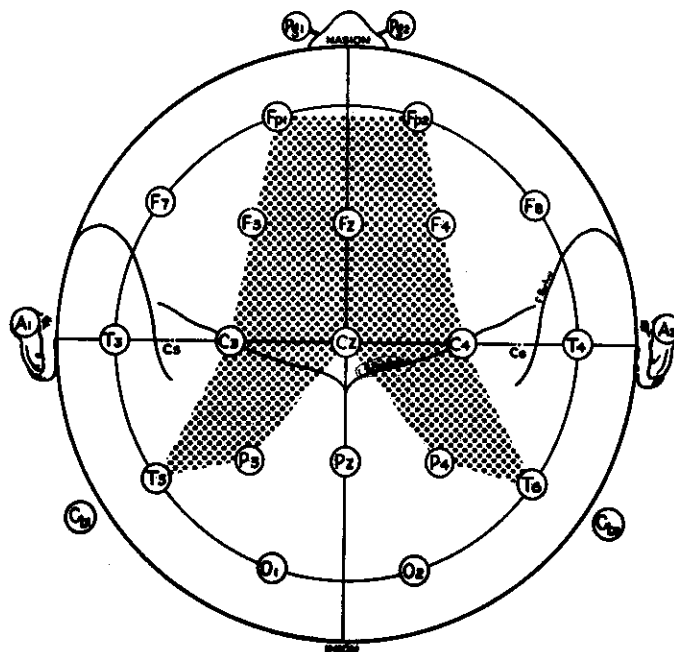


Figure 5. Topographical distribution of theta waves (stippled area).

as shown by the stippled area. This area is bilaterally situated and includes the midline leads F_z and C_z . Frontally the prominent area is more or less restricted to the medial portion of the cortex and caudally it spreads more laterally to the posterior parietal (P_3 and P_4) and temporal (T_3 and T_4) areas.

Flashes of 10 microseconds duration and approximately 50,000 candle power peak intensity at the eye level were provided with a Grass PS 2 Photostimulator. Frequencies of 3, 5, 7, 10, 20 and 30 per second were employed. On all three examinations (June 5 and August 7, 1962, and January 29, 1963) the subject, with eyes open, responded to all these frequencies with occipital lead synchronization. However, only occasional synchronizations were observed on 3, 20 and 30 per second stimulations. More faithful and prominent waves followed the 5, 7, and 10 per second stimulations. Figure 6 shows an EEG record taken on the second examination during photostimulation at the frequency of 7 per second. The flashes are marked by the sharp spikes in lead C_4 and synchronization is best seen in lead O_1 . The positive spikes shown in lead O_1 are presumably the lambda waves which occur in normal individuals when the illumination is strong and the subject gazes at the light with intention.¹²⁻¹⁶ Our subject showed these waves in O_1 lead in both the later examinations but not in the first one. Although the subject responded to photostimulation with faithful synchronization of occipital waves, there were neither abnormal waves or spikes, nor the appearance of seizure-like motor responses. Synchronization was limited to occipital leads and seldom spread to the C_3 and C_4 leads. Even with higher intensity of stimulation (2x and 4x), no abnormal brain waves or movements were observed.

In all three examinations, the subject was instructed to breathe as deeply as he could for 3 minutes and to keep each inspiration and expiration with the beats of an electric metronome which was set at the rate of 80 per minute. High voltage slow waves appeared in the F_{P_1} , F_{P_2} , C_3 , C_4 , O_1 , and O_2 leads and sometimes in the T_3 and T_4 leads during hyperventilation (Fig. 7). Return of brain waves to his resting level occurred within one minute in the first and third examinations. In the second examination, high voltage slow waves persisted until 160 seconds after cessation of hyperventilation. Valsalva and breath-holding also produced no significant EEG changes.

In the third examination external auditory canal irrigation for 30 seconds with water at 25°C was performed on the subject while he was lying supine with his head tilted to one side. Electroencephalograms from F_{P_1} , F_{P_2} , T_3 , T_4 , C_3 , C_4 , and O_1 were recorded on seven channels and, on the last channel, horizontal movements of the left eye were recorded with two silver-disc electrodes, one close to the lateral canthus and the other on the bridge of the nose. Three irrigations were performed, the first two on the left side and the last one on the right side. In the first test, nystagmic eye movements with the fast component towards the right side started 38 seconds following the beginning of left canal irrigation. The maximum amplitude of the electrical signal was about 250 μV and the frequency of the nystagmus was from 2-3 per second. The duration of high amplitude nystagmus was only 5 seconds. The amplitude of the remaining nystagmus potential was below 100 μV . The total duration of discernable nystagmus (above 50 μV) was 110 seconds. In the second test, the left ear was irrigated again while the subject performed an arithmetic task. The result was comparable to the first except

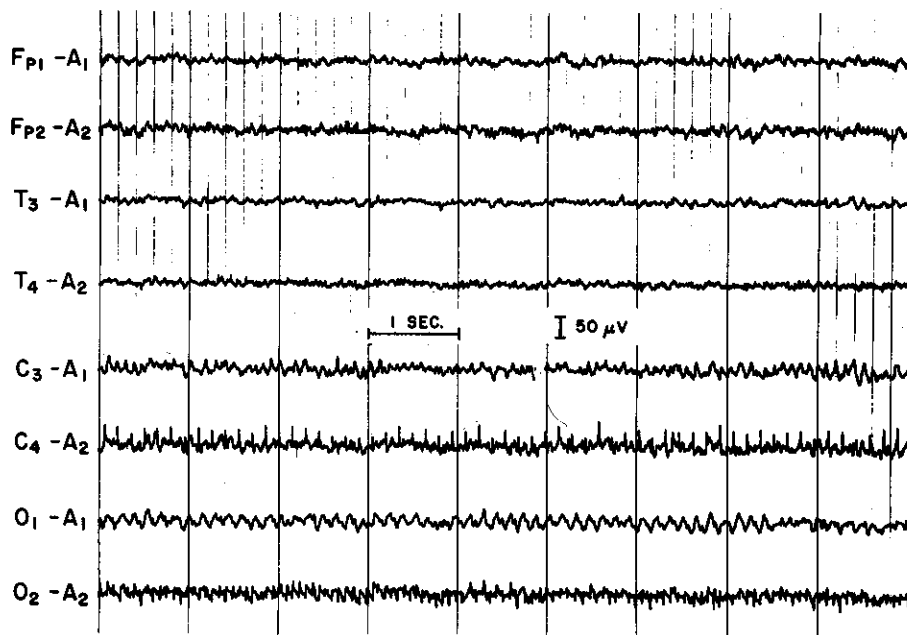


Figure 6. Photostimulation at 7 per second frequency. Stimulations marked by spikes in lead C₃. EEG synchronization best seen in O₁ lead. Lambda waves in O₂ lead.

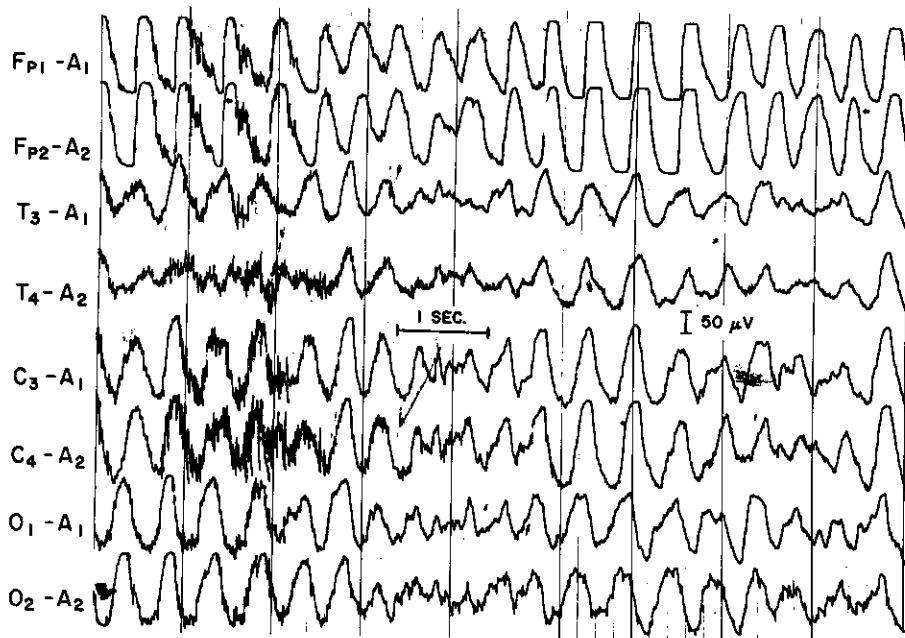


Figure 7. High voltage slow waves in all EEG leads at the end of three minute hyperventilation.

that the duration of high amplitude nystagmus was prolonged to about 100 seconds with a total duration of discernable nystagmus of more than 200 seconds. No other abnormal brain waves were noted in these two tests except some 5-7 per second waves similar to those appearing in his resting records. The subject reported that he experienced no unusual sensations or discomfort during these tests. The subject was then allowed to rest for more than 10 minutes with periodic EEG recordings made during the rest period. A large number of theta waves appeared during this period, much more frequently than in his previous resting records.

In the third test, the right ear was irrigated with the same procedure. High amplitude nystagmus (about 210 μ V) with 2-3 per second frequency appeared 40 seconds after the start of irrigation. However, this high amplitude nystagmus lasted for more than 200 seconds. At about 43 seconds after the onset of nystagmus, high voltage slow waves (2-3 and 4-5 per second) appeared in all the recorded leads and lasted 46 seconds (Figs. 8 and 9). These waves are not unlike those appearing at the end of 3 minute hyperventilation (Fig. 7). Although some of the slow waves appear to be synchronous with the nystagmic eye movements (Fig. 8), suggesting that they could be the result of artifacts, the facts that (1) these slow waves did not appear after left ear irrigation which produced similar amplitude and frequency of nystagmus, (2) they did not appear in the rest of the over 200 second records after right ear irrigation with the same amount of nystagmus present, and (3) many slow waves (Figs. 8 and 9) did not appear to be synchronous with the eye movements, indicate that these slow waves probably were true synchronized discharges from the cortex. The subject reported that during this last test he experienced the same sensation of light-headedness and dizziness as he did just prior to his loss of consciousness in the airplane.

TABLE I

Position	Heart Rate per minute
Supine	58
60° feet down	69
1st minute	69
2nd minute	69
3rd minute	74
4th minute	68
5th minute	71
6th minute	70
7th minute	71
8th minute	71
9th minute	68
10th minute	64
11th minute	71

The subject was placed on a tilt table and an EEG was recorded from the FP_1 , FP_2 , T_3 , T_4 , C_3 , C_4 , and O_1 leads in the top seven channels of the electroencephalograph. The last channel recorded the subject's ECG from his two arms. His heart rates before and during the 10 minute 60° feet down tilt from a supine position are shown in Table 1. No cardiac arrhythmia was noted during this period and the subject reported no unusual feelings. At the end of the tenth minute, firm pressure was applied to his eyes with thumbs for fifteen seconds. His heart rate at the end of ocular compression was 65 per minute and again no cardiac arrhythmia was noted. The patient again reported no unusual feelings. At the end of the eleventh minute, the heart rate was 71 per minute and the subject was tilted back to his supine position.

Electroencephalogram was periodically recorded at half hour intervals throughout five hour glucose tolerance test. No abnormal waves other than his usual theta waves were observed throughout the test even though at one time his blood glucose was as low as 43 mg/100 cc.

DISCUSSION

The occurrence in this case of inflight true vertigo followed by almost two hours of unconsciousness presents a most difficult diagnostic problem; one that is not yet completely resolved.

A great deal of interest exists in the field of aviation medicine in the vertiginous effect of flicker produced by light passing intermittently through rotating helicopter blades. Similar effects may be noted by pilots who view the sun through propellers, or see sunlight reflected off the propellers, of aircraft, particularly single engine models with tandem seating. Despite the possibility of appropriate flight conditions and the EEG finding of photic synchronization, flicker vertigo and a grand mal seizure can probably be ruled out in this case by the absence of subjective discomfort during photostimulation and the absence of epileptic EEG discharges even during photostimulation.

The spontaneous statement by this airman that the sensory experience with caloric stimulation was identical to that of the original episode confirms our suspicion that true vestibular vertigo occurred. The vertigo produced during the examination by the caloric test and by rotation in a Barany turning chair was a normal physiologic response of the labyrinth. While considerable motion of the head preceded the initial episode of vertigo, neither the flight path of the aircraft nor the reported subjective sensations are consistent with Coriolis reaction.

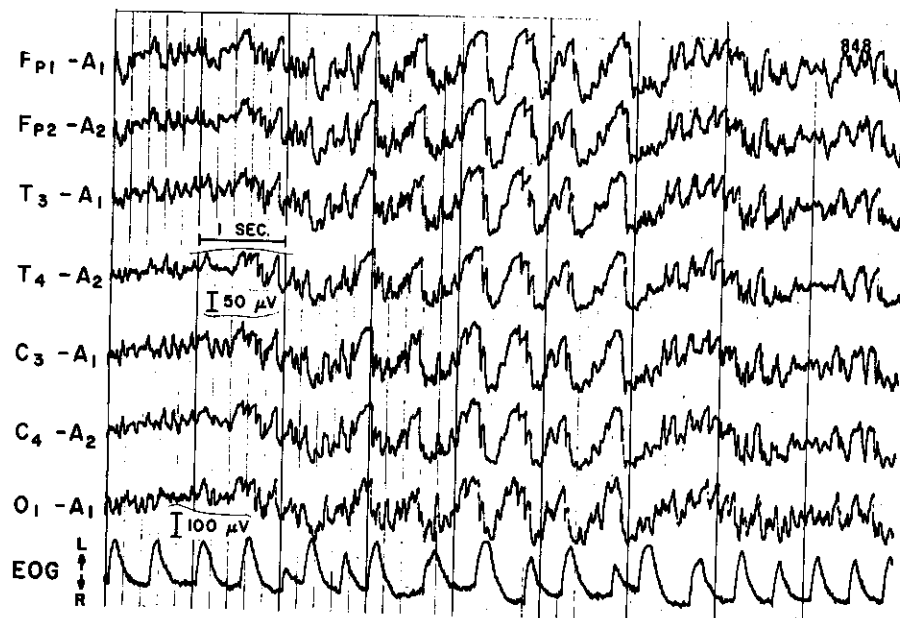


Figure 8. High voltage slow waves in all EEG leads 95 seconds after irrigation of right ear. Bottom channel shows the record of nystagmic movements.

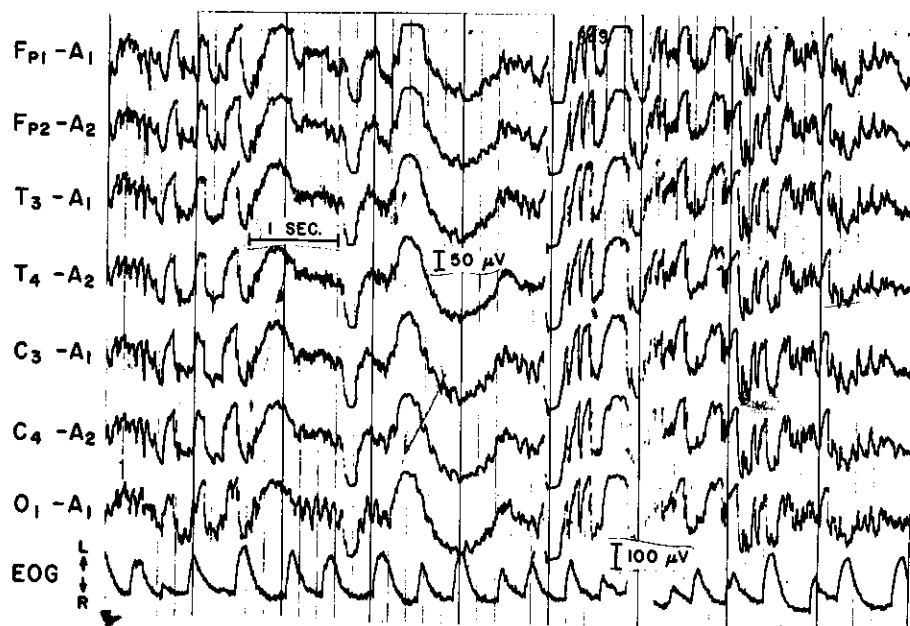


Figure 9. High voltage slow waves in all EEG leads 108 seconds after irrigation of right ear. Bottom channel shows the record of nystagmic eye movements.

Other causes of vertigo have been considered but none was confirmed by our findings. Sudden alteration of the air pressure in the middle ear can cause acute transient attacks of vertigo in airmen but should not occur between ground level and 400 feet during ascent. Endolymphatic hydrops is common and is a leading cause of abrupt attacks of vertigo lasting minutes or hours. Absent in this case, however, were the commonly associated stress, endocrine imbalance, tinnitus, hearing loss, reduced response to caloric test, sensation of aural pressure, nausea, and vomiting. A vasomotor disturbance involving endarterial branch vessels of the labyrinthine artery can cause postural vertigo and similar involvement of other vessels could explain the prolonged period of unconsciousness, but there is no family history, reported stress or recurrence to support this explanation.

Duration of the vertigo was inadequate for an acute toxic labyrinthitis or for a vestibular neuronitis. Because of the EEG abnormalities, consideration was given to disorders of the cerebellum, cerebral cortex and eighth nerve which can cause vertigo but there was no conclusive evidence to support a diagnosis of involvement of any of these areas.

The history of an allergy, the associated gastrointestinal symptoms, and the acoustic dip at 4000 and 6000 cps are not unusual findings in airmen with vertigo but, unfortunately, they do not help establish a diagnosis in this case.

While incapacitation occurs with severe vertigo, associated loss of consciousness, which occurred here, is seldom reported.

Despite the absence of any external markings about the head, head impact during the crash must be seriously considered as the cause of prolonging the unconsciousness to almost two hours. His position at the time of discovery suggests that the seat belt was never fastened, that his head impacted soft ground during the crash, and that recovery from syncope would have been prompt in his head-down position.

During the EEG tests, various activating methods were employed to provoke syncope or seizure attack. As the subject was sensitive to bright light, photostimulation with a moderate intensity was attempted. His EEG showed synchronization to all the test frequencies (3, 5, 7, 10, 20, and 30 per second), indicating

sensitivity to photostimulation. However, no epileptic discharge was observed. His response to photostimulation probably cannot be considered abnormal as Pantelakis *et al.*¹⁷ showed that photostimulation produced epileptic EEG discharges in 13 out of 14 photo-sensitive children. Nor can the appearance of positive spikes from the occipital region (lambda waves) during photostimulation be considered abnormal as most normal persons show lambda waves in response to photostimulation under certain conditions.¹²⁻¹⁶

Other activating methods failing to provoke EEG abnormalities are the tilt table orthostatic tolerance test used by Dermksian and Lamb,¹² the valsalva maneuver used by Duvoisin *et al.*,⁸ and the 10 second ocular compression method used by Gastaut and his colleagues.^{3,3} Hyperventilation for three minutes did produce high voltage slow waves but except on one occasion the abnormal waves subsided within one minute. Although hyperventilation is routinely used clinically for EEG activation, its value for diagnosis is always questionable. Balke *et al.*¹⁸ showed that the response to hypocapnia can be modified by training or acclimatization.

Seipel and Wentz¹⁹ observed aggravated abnormal waves on their subject at the fourth and fifth hour of the glucose tolerance test when the blood sugar level was 65 and 80 mg. per cent. In our case, no aggravated abnormal waves were observed during the entire five hours of glucose tolerance test although the blood sugar level reached 73, 43, and 51 mg. per cent at the third, fourth, and fifth hour after the ingestion of 100 g. of glucose.

The only positive findings in our study are the presence of 5-7 per second theta waves in his resting EEG records and the appearance of high voltage slow waves during irrigation of his right ear. His resting theta waves were of moderate amplitude (50-100 μ V) and often nearly sinusoidal in shape. Cobb¹⁰ considered that monorhythmicity, i.e., simple sinusoidal waves not mixed with other frequencies, is more significant than mixed waves. Most investigators²⁰⁻²⁵ consider bilateral theta as an indication of lesions involving various subcortical areas including thalamus, hypothalamus, mid-brain, pons or medulla. One author,²⁵ however, considered mid-line theta discharge as an indication of temporal lobe epilepsy. According to

Cobb¹⁹ and Walter and Dovey,²¹ bilateral theta is the most frequently encountered EEG abnormality. It is also interesting to note that both our subject and the subject reported by Seipel and Wentz showed this type of abnormal waves. This leads to the question of whether or not loss of consciousness may often be associated with bilateral theta rhythm. Although most studies were made on tumors, other causes can also produce subcortical lesions. In our case, we cannot rule out the possibility that the abnormal EEG activity is the result of traumatic head injury since the subject had sustained head injury in his boyhood and also might have had concussion during his crash after landing. However, Cobb¹⁹ found that rhythmic EEG activity due to head injury usually disappeared by the end of three weeks, and our subject showed persistent bilateral theta rhythm two, four and nine months after the incident indicating that the rhythm is more likely a permanent feature and not the temporary effect of head trauma. The appearance of high voltage slow waves during the caloric test was unexpected. However, the fact that abnormal EEG waves can be provoked by vestibular stimulation appears to be in perfect

agreement with the contention that the lesion, if there is any, must be subcortical and very likely in the brain stem.

Recently Henry and Helfer²⁶ reported a case of sudden complete incapacitation of an apparently healthy ATR pilot. He was found to have a constricted middle cerebral artery on the right side as revealed by arteriogram. The authors raised the question of whether the existence of the lesion should have been detected during one of the 15 physical examinations made during six years preceding the episode. If an electroencephalogram had been performed during a physical examination of their pilot, it could have led to diagnostic studies before his sudden incapacitation.

Because of the possibility of unpredictable recurrences with serious consequences, it would be hazardous for this airman to resume flying until the cause (or causes) and relationship of his vertigo, period of unconsciousness, and EEG abnormality are better understood. Pursuit of a career in commercial aviation would be particularly unwise at this time. Periodic assessments of his interval history and physical condition are planned to determine when, or if, he may safely return to flying.

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