

Inside the Regs Outside the Envelope

Understanding the delayed effects of alcohol on pilot performance and flight safety

By David Curry

A STUDY OF FATAL U.S. AIRCRAFT ACCIDENTS between 1994 and 1998 revealed that significant blood alcohol concentration (BAC) levels (greater than 0.04) were present in a substantial percentage of the pilots involved (124 of the 1,683 or nearly 7.5 percent of all samples tested) (Canfield, et al). Federal Aviation Administration (FAA) information shows that data for the years 1987 to 1993 were almost the same in terms of percentage of alcohol involvement (306 out of 2,534 or 7.85 percent). Other

sources have asserted that anywhere between 10 and 30 percent of general aviation pilots involved in fatal accidents had measurable alcohol in their blood and tissue when post-accident medical analyses were conducted (Burton and Jaggars; Modell and Mountz; Rayman). These statistics may represent only the tip of an alcohol-related iceberg.

The Effects of Alcohol

In recent years, a great deal of research has been conducted regarding the effects of alcohol on human performance. It is now generally recognized that virtually all types of performance degrade to some degree as a result of any measurable quantity of alcohol in the bloodstream. In the flight regime, this has been widely demonstrated both in the air and in flight simulators (Billings, et al; Henry, et al). Alcohol at a BAC of 0.015 percent has

been shown to cause impairment of performance at tasks that require division of attention—such as monitoring two types of information simultaneously. One study placed pilots in a flight simulation that contained preexisting errors (e.g., brakes in the “off” position, landing gear in the “up” position, fuel select switch on the auxiliary tanks, flaps incorrectly set) (Wise). Results showed that although only 10 percent of the subjects missed one or more error after no alcohol consumption, that number increased to 89 percent within 30 minutes of reaching a peak BAC level of 0.10.

Use of alcohol and drugs by pilots is regulated by Federal Aviation Regulation (FAR) 91.17. Among other provisions, this regulation states that no person may operate or attempt to operate an aircraft:

- within eight hours of consuming any alcoholic beverage;
- while under the influence of alcohol;
- while using any drug that affects a person's faculties in any way contrary to safety;
- while having a 0.04 percent by weight or more alcohol in the blood.

The eight-hour time delay provides time for most individuals to metabolize the alcohol consumed, even after fairly significant consumption (normal metabolic rate will eliminate approximately one drink per hour from the body). However, this does not mean that a person is necessarily safe to fly an airplane. In the simulator study, researchers noted that as long as 14 hours after alcohol consumption error levels were still at the 68-percent level. A second flight simulator study also used a peak BAC

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level of 0.10 and investigated performance during a 14-hour post-ingestion time. Results showed decreased performance on almost all measures and significant differences on three of six variance measures and one of six overall performance measures (Yesavage and Leirer).

While the requirements in FAR 91.17 allow time for alcohol to be metabolized out of a person's system, the mandates are not necessarily adequate to allow recovery from other effects of drinking—such as disruption of the sleep cycle, suppression of rapid eye movement (REM) sleep, reduced G-tolerance, increased susceptibility to coriolis acceleration (acceleration that arises as a result of motion of an individual relative to a rotating system) and problems involving positional nystagmus (rapid, involuntary, oscillatory motions of the eye).

Sleep Apnea

Sleep apnea is a common disorder aggravated by alcohol consumption. Apnea is defined as a "cessation of air flow at the level of the nostrils and mouth lasting at least 10 seconds" (Guilleminault, et al). Although the number of people afflicted with this condition has never been accurately determined, snoring (a close associate) occurs in more than 20 percent of the population—and more than 60 percent of the older adult male population (Lugaresi, et al).

Sleep apneas fall into two general categories: 1) obstructive, which is the absence of respiratory airflow despite the presence of central respiratory drive and usually results from blockage of the upper airways; and 2) arrhythmic, which occurs as the result of the simple cessation of inspiratory effort.

The incidence of obstructive sleep apnea seems to be heightened by factors such as excessive weight, gender (males are more prone) and snoring. Those with sleep apnea experience no respiratory ill effects while awake; however, once asleep, the affliction manifests itself. The process has been described in this manner:

Suction collapse of the oropharyngeal airway occurs with the onset of sleep. Despite inspiratory efforts by the respiratory muscles, there is no airflow, and progressive asphyxia ensues. After 30 to 120 seconds, there is a transient arousal from sleep, with resumption of airflow through the upper airway. The cycle may be repeated 200 to 400 times each night, and profound hypoxaemia (oxygen desaturation of the blood) may occur (Issa and Sullivan).

In other words, airflow ceases and the individual must at least partially awaken to resume breathing. These arousals do not always result in a complete awakening, but may simply involve a lightening of the sleep state (a sort of a semicontinuous movement between the deeper, more restful stages of sleep to the lighter stages that must be transitioned to in order to resume breathing). These incidents are usually quite short, lasting one to four seconds, but result in sleep fragmentation, less total sleep time and drastically decreased sleep efficiency (Guilleminault and

Rosekind). Sleep efficiency is calculated on the basis of percentage of sleep stage changes per measured sleep epoch (usually 30 seconds) and the intrusion of alpha waves on the electroencephalographic (EEG) record during this period.

The accompanying decrease in arterial oxygen saturation is quite pronounced during the apneatic events, probably exceeding that which would occur if the individual were to hold his/her breath for a similar period of time (Remmers). Individuals with obstructive sleep apnea typically have a history of snoring. In addition, the condition is linked to heart failure, cardiac arrhythmia and many central nervous system disorders (such as memory loss, personality changes, daytime somnolence and dementia). This is believed to be the result of the fragmentation of the individual's sleep and the hypoxaemia engendered by the apneatic episodes (Issa and Sullivan).

Evidence indicates that the consumption of alcohol strongly influences the frequency and severity of apneatic events. Alcohol has also been shown to induce sleep apnea in subjects with no previous history of the problem. Plausible explanations for this are that ethanol either depresses the reticular activating system, activates a brain stem inhibitory system or some combination of both. In turn, this causes a depression in upper airway motoneuron activity, resulting in at least partial closure of the airway due to relaxation of the muscles required to hold it open. Alcohol also seems to increase the threshold of the stimulus required to arouse the sleeper, thus prolonging the apneatic episodes (Remmers; Guilleminault and Rosekind). Some researchers have postulated that asphyxia and arterial blood deoxygenation during sleep is the leading factor contributing to alcohol-related brain damage (Issa and Sullivan).

In one study using an uncontrolled amount of alcohol (subjects were asked to drink as much as they would maximally consume on social occasions), the researchers reported that even while awake, it appeared oxyhemoglobin saturation was reduced by an average of three to four percent when compared to control nights (Issa and Sullivan).

Alcohol appeared to have two main effects during sleep:

- 1) the duration of apneatic episodes was prolonged and arterial oxyhemoglobin desaturation increased to a much greater degree;
- 2) the incidence of sleep apneas increased significantly.

These changes were most pronounced during the first two hours of sleep and seemed to parallel the time-course of alcohol metabolism. The effects were also apparently highly dose dependent. Lower levels of oxyhemoglobin saturation and longer apneas were observed in those who drank the most in relation to their body weight. Oxygen saturation (SaO₂) levels of below 92 percent are considered hypoxic and those below 70 percent have been associated with objective evidence of brain malfunction (Issa and Sullivan).

These results have been confirmed by other researchers. Using dosages of approximately one

gram of alcohol per kilogram of body weight (g/kg), studies by Guillemainault, et al indicated that the consumption of alcohol results in longer apneatic episodes, an increase in their incidence (approximately 30-percent more episodes per hour), and a significantly lower degree of arterial oxygen saturation (from 83.25 percent to 73.75 percent during non-REM sleep, and from 78.25 percent to 71.25 percent during REM sleep).

A study of 20 asymptomatic male subjects found that doses of 2 ml/kg of body weight of 100-proof vodka had similar effects. Ingestion of alcohol significantly increased the number of arterial oxygen desaturation events (118 to 226, $p < 0.01$) and the number of apneatic events (20 to 110, $p < 0.01$). The results further indicated that incidence of oxygen desaturation events was also significantly higher ($p = 0.01$) on the second night following alcohol ingestion, even if no further alcohol was consumed (Taasan, et al).

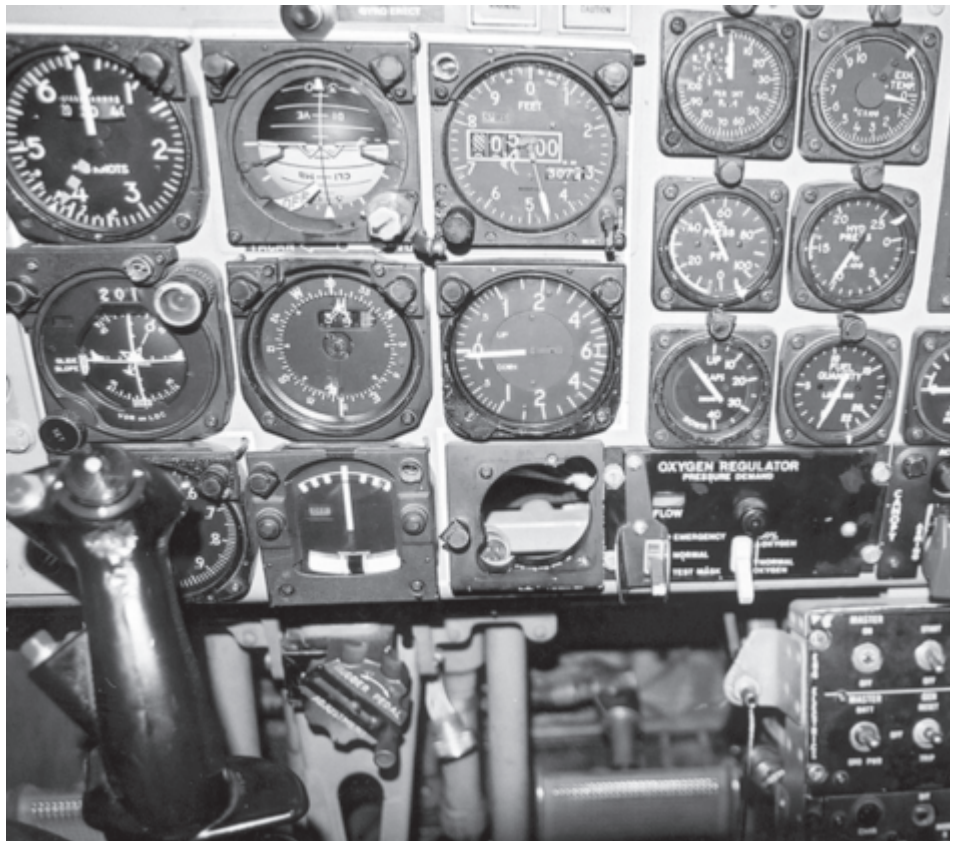
The primary result of apneatic episodes (other than those associated with oxygen deprivation) is the level of fatigue produced as a result of sleep fragmentation and lack of sleep efficiency. Such fatigue manifests itself in several ways including spatial disorientation, poor judgment, distraction, loss of G-tolerance, poorer hand/eye coordination, increased reaction time and a drop in voluntary movement of the eyes (scanning) (Tilton)—effects that can clearly degrade flight safety.

REM Deprivation

A second sleep-related effect of alcohol is a systematic, dose-dependent reduction of REM sleep. REM sleep has been highly correlated with incidents of inferred visual dreaming in laboratory subjects and appears to be a necessary component of restful sleep. In a study performed using laboratory rats, doses of 1, 2 and 4 g/kg of ethanol were administered. Results showed that REM sleep was totally blocked for periods of 90 minutes, 160 minutes and 315 minutes, respectively. In this case, onset of REM sleep corresponded well with the mean time needed for the rats to metabolize two-thirds of the blood ethanol concentration produced by each alcohol dose (Hatten and Eacho).

Human studies have concentrated on more-benign dose levels. One study found no effect on REM sleep with alcohol doses ranging from 0.16 to 0.64 g/kg of body weight (Stone). Results of another low-dose study indicated that a dosage of 0.25 g/kg produced significant reductions in REM sleep in nonalcohol-dependent adults (Rouhani, et al).

Studies with higher dosage levels have confirmed this result. One study employed doses that induced BAC levels of 80 and 150 mg percent (0.08 and 0.15



BAC, respectively). Results indicated that during at least the first half of the night, REM sleep decreased significantly from that experienced during no-alcohol control nights. For the lower dosage, an inverse relationship was indicated between the amount of REM sleep recorded during each half of the night.

In other words, REM sleep was depressed during the first half of the sleep period, but experienced a rebound effect during the second half of the night (Knowles, et al; Lobo and Tufik; Landolt, et al). This led to an overall level of REM sleep that did not significantly differ from control nights. The larger dosage appeared to prevent the occurrence of this REM rebound effect. These results were replicated by Rundell, et al, with the added caveat that repeated alcohol ingestion over longer periods (several nights in succession) leads to a resurgence of REM sleep back to near-normal levels even after alcohol ingestion.

The decrease in REM activity is interesting because of the observed consequences in laboratory studies of REM deprivation in both animals and humans. The earliest of these studies were performed using cats, with the primary effects of lack of REM sleep being increased restlessness, hypersexual behavior and a ravenous appetite (Dement (a), (b); Dement, et al; Dewson, et al). Research on humans has indicated that the same effects are present, usually accompanied by impaired concentration and increases in irritability, anxiety, aggressiveness, suspiciousness and overall tiredness [Dement(a), (b); Dement and Fisher; Cledes and Dement]. These last three effects are usually

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Beyond Aviation

The problem of delayed alcohol effects is not restricted to the aviation environment. Most research in the automotive environment has focused on the effects of low to moderate doses of alcohol, but several studies have noted that alcohol doses as low as 0.01 percent increase susceptibility to sleepiness and aggravate the effects of sleep deprivation. Subjects given low doses of alcohol after a night of reduced sleep have been shown to perform poorly in driving simulators, even when no detectable alcohol remains in their systems (Roehrs, et al; Krull, et al). According to Roehrs, "The results show that sleepiness and low-dose ethanol combine to impair simulated automobile driving, an impairment that extends beyond the point at which BEC (breath ethanol concentration) reaches zero."

Many other delayed effects noted for pilot performance (e.g., fatigue, REM deprivation, narrowing of the attentive field, difficulty performing procedural tasks) can have a pronounced effect on driving or other workplace tasks, particularly in situations that involve the detection of unexpected hazards or complex machine/process setup or monitoring. The potential contribution of positional alcohol nystagmus to falls and loss of balance is also an important issue that should be assessed, particularly with regard to elevated construction or worksites.

deprived subjects (Kopell, et al). This suggests that the number of cues within the "attentive field" has been reduced due to the higher state of arousal. Initially, this would appear to be beneficial. However, it leads to fixation on those aspects of the environment that the subject feels are germane to the task at hand; unexpected or secondary stimuli are easily ignored or not perceived. In the flight management environment, such performance degradation can be disastrous.

Studies have shown that procedural memory is also negatively affected by REM suppression (Smith). One manifestation of this would be an increase in the number of action slips. This means that in situations where judgment should be exercised or decisions made regarding particular courses of action, the individual has a tendency to either perform tasks by rote once started on a sequence of events, or fails to follow through on a "preprogrammed" series of actions and does not realize that s/he has done so.

ascribed to a heightened drive level in the subjects. Other researchers have shown that such an increase in drive level often results in a drastic reduction in cue utilization—a phenomenon often referred to as "a narrowing of the attentive field in aroused states" (Callaway and Dembo; Callaway and Thompson).

An increase in drive level produces several negative effects. Subjects' perceptions become more influenced by internal clues than by situational demands (particularly in ambiguous situations) and the ability to perform critical data analysis is systematically reduced (Clemes and Dement).

Another major effect is a change in the attention differential on average evoked responses to visual stimuli (Kopell, et al). Selective attention is often described as the difference between the degree of responsiveness to a relevant versus an irrelevant stimulus. The average evoked potential is larger when a subject attends primarily to a single stimulus rather than when s/he is attending to the entire visual field.

Research has indicated that the differential between relevant and irrelevant stimuli is far higher in REM-

Nystagmus

The sense of balance or equilibrium in humans is a function of three different interacting systems: vision, vestibular system (the semicircular canals of the inner ear) and kinethesis ("seat of the pants" knowledge of awareness of body and body segment position) in a roughly 80:10:10 proportion. Nystagmus is the involuntary bouncing or jerking of the eye caused by any number of vestibular, neurological or physiological disturbances, any of which may drastically disrupt the sense of equilibrium to a greater or lesser degree. Positional nystagmus occurs when a foreign fluid is in unequal concentrations between the blood and the fluid in the semicircular canals.

Disruption of the vestibular system as a result of alcohol consumption (positional alcohol nystagmus or PAN) was first demonstrated in the laboratory in 1842 (Flourens). Research has demonstrated that this phenomenon has two phases. The first (PAN I) occurs approximately 30 minutes after drinking the initial dose of alcohol and is the result of the alcohol concentration in the blood being at a higher level than that in the vestibular system (the permeability of the canals' outer lining is different than that of most body tissues). The second phase (PAN II) occurs five to six hours later, after the individual has stopped drinking and the body has begun to metabolize the alcohol out of the bloodstream; it is the result of the blood's alcohol concentration being lower than that of the vestibular system. The PAN II phase lasts for at least five to 10 hours—long after the alcohol has been metabolized out of the bloodstream (Goldberg; Murphee, et al).

Nystagmic responses are also observed when the body undergoes coriolis acceleration (which occurs whenever the head is tilted with respect to the plane of the body's acceleration); most pilots develop a resistance to this effect due to repeated exposure (Dowd, et al). Experiments have shown that for the majority of subjects, coriolis nystagmus is accentuated and prolonged following the ingestion of alcohol, and that the normal habituation to repeated stimuli exhibited by most pilots does not occur (Ryback and Dowd).

During the course of a plane's flight, a number of angular accelerations, in one or two geometric planes, are imposed upon the pilot. Changing the angular relationships between the head and the axis of rotation during certain maneuvers results in false perceptions of position and movement, as well as visceral disturbances, especially if terrain references are absent. . . . This produces, among other responses, a vertical nystagmus which outlasts the stimulus for as long as 30 seconds in some individuals (Moore).

This effect has been shown to persist for up to 34 hours after the consumption of the alcohol and was accompanied by reports of disorientation and subjective tumbling on the part of the pilots involved. Other studies have reported that increased G-forces may provoke this effect for up to 48 hours after drinking (Oosterveld). It has been theorized that this effect may

be another function of the REM suppression attendant on alcohol consumption. This position is supported by studies which have shown that nystagmic fast-phase frequency increased significantly at higher accelerations when subjects were deprived of only moderate amounts of sleep (Wolfe and Brown).

Vision

Research regarding the effects of alcohol on vision has focused primarily on either acute or chronic consumption rather than on delayed effects. Low-dose alcohol studies have demonstrated visual impairments in the acuity of the eye itself as well as in the effectiveness of the visual perception process (much of which occurs in the brain rather than the eye itself).

In a study commissioned by the U.S. Coast Guard, blood alcohol levels as low as 0.01 to 0.04 were associated with decreased ability to discern objects or faint lights at night (reduced contrast sensitivity), ability to notice objects located just outside the direct line of sight, ability to respond to constantly changing stimuli (pursuit eye tracking), and ability to select the correct response based on the nature of the perceived stimulus (McKnight, et al). Glare effects have also been shown to be significantly magnified by blood alcohol levels as low as 0.01, with greater impairment resulting from higher concentration levels. Depth perception, peripheral and color vision, as well as general night vision, have also been shown to be negatively affected by BACs at or below the level allowed by FAR 91.17.

These effects could potentially manifest themselves in an increase in the amount of time necessary to correctly identify objects in the visual field—or cause the individual to misidentify objects detected. Coupled with the noted reduction in the attentive field, this may well result in objects being initially miscategorized by the viewer—objects that are then never again focused on to verify the accuracy of the initial decision. The potential negative effects of such visual problems are many (e.g., misidentifying other air traffic as a larger aircraft further away, misperceiving altitude when making a visual approach, misperceiving a shoreline as the horizon when flying under a cloud deck at night).

Discussion

The effects and aftereffects of alcohol vary by individual depending on a variety of psychological and physiological factors. Careful consideration of the evidence presented in this article suggests that the current eight-hour minimum separation between drinking and flying may be inadequate. For individuals susceptible to alcohol-induced or alcohol-exacerbated sleep apneas, the consumption of even small quantities of alcohol can result in severe sleep fragmentation and a drastic decrease in sleep efficiency. After eight hours of “drying out,” a pilot may still be far too mentally fatigued to safely operate an aircraft.

Consumption of moderate to large quantities of

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It is now generally recognized that virtually all types of performance degrade to some degree as a result of any measurable quantity of alcohol in the bloodstream.

alcohol can easily cause an individual to be extremely tired, irritable, hyperaggressive and functionally fixated on only a few aspects of the environment due to REM deprivation and/or apneatic episodes. This creates a potentially dangerous situation even if only in terms of simple errors in control operation or situational awareness.

In the airline industry, examples of this would be a pilot's failure to notice the plane's flap settings before attempting take off or not performing instrument cross-checks due to fixation on certain aspects of the cockpit environment. A study performed for the Air Force in 1980 found that 46 percent of all crashes resulting from pilot error could be attributed to channelized attention and 20 percent to "excessive motivation" (Hartman). While not suggesting that these incidents were all (or even mostly) alcohol-related, the numbers are indicative that the same aftereffects of alcohol-disturbed slumber are those often cited as the cause of aircraft accidents involving "pilot error." When accidents involving spatial disorientation, possibly aggravated by positional alcohol nystagmus, are factored into the equation, the situation becomes acute.

As has been suggested, the best way to avoid these alcohol-induced problems is to increase the time between alcohol consumption and flying in order to allow for both an adequate degree of rest and the time necessary to overcome the time-delayed aspects of alcohol consumption (Gibbons).

Since the effects of alcohol on obstructive sleep apnea do not seem to extend beyond the first night following consumption, it is possible that extending the "bottle-to-throttle" rule to 24 hours would solve this problem in that it would provide a second, non-fragmented, relatively efficient night of sleep between alcohol consumption and flying. The REM rebound effect seems to extend over several nights (assuming the deprivation level was sufficient to require this), so this time extension could alleviate that problem as well. This number also corresponds relatively well with the maximum extent of the nystagmic effect, at least in a relatively benign flight environment.

Conclusion

More research is needed on the delayed effects of alcohol consumption. At a minimum, both the flying population and accident investigators should be aware of the potential danger of assuming that following the current regulations ensures immunity from alcohol-related flying mishaps. Many aircraft accident investigators conclude that if post-mortem BAC is less than the 0.04 as specified by FAR 91.17, then alcohol was not a factor and no further information regarding the pilot's physiological status is pursued. Such an assumption reflects a lack of understanding with regard to both the effects of low doses of alcohol on flying performance, as well as of the facts regarding alcohol-related "carryover" effects that may affect pilot performance for hours or days after drinking. ■

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