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May is “Mental Health Month,” and thanks to a recent behavioral health event sponsored by TERROS, more than 600 healthcare professionals are better prepared to treat clients of diverse cultures.

By Jeffrey C. Friedman, MHS, LISAC

Paul Cox is a man of quiet reserve who is casual conversation gives the impression of a business executive just entering his peak earning years, rather than the hard working carpenter he was in his youth. But today, at 43, Paul has no career—unless you count the prison job he held each day in the New York penitentiary where he is serving double life for the 1988 murders of a couple he had served next door to the night before New Year's Eve in 1988, when, in an alcohol-induced blackout, Paul stabbed them to death.

Readers who recall the news events of the early 1990s might remember the case of Paul Cox, not for the fact that he had committed an unspeakably brutal crime while in an alcoholic blackout—crimes committed in alcohol-fueled blackouts are too way too common to make the front pages of newspapers. His case made headlines for an entirely different reason. Paul was arrested and charged with two counts of second-degree murder four long years after the night of the stabbings, because he had disclosed his involvement in the crimes at a meeting of Alcoholics Anonymous.

At his trial, Paul’s freedom depended on his lawyer’s assertion of two key points: point one was that his blackout on the night of the crimes constituted a state of temporary insanity that prevented Paul from appreciating the wrongfulness of his actions, and point two, that since Paul’s confession occurred in the context of an A.A. meeting, his statements should be considered privileged in the same way a disclosure to a priest or psychiatrist is. Because communication within an Alcoholics Anonymous meeting is privileged, his

The Strange State of Oblivion
At trial, the story told by the prosecutor about Paul’s actions on that night in December 1988 is one guaranteed to send a chill down the spine of anyone who has ever suffered an alcoholic blackout. After an evening of heavy drinking at a Larchmont, New York, bar, Paul was driving home with two drinking buddies. His last memory of that night was trying to negotiate a tight curve and one of his friends warning, “You're not going to make it.” Then nothing. The alcohol Paul had consumed that night had triggered a biological reaction deep within his frontal lobe that temporarily but effectively prevented Paul’s brain from forming new memories. Not even knowing it, Paul had entered the strange oblivion of an alcoholic blackout.

But somehow, he made it home that night and, too early the next morning a bleary-eyed and befuddled Paul was roused by a phone call from the local police who were particularly interested in why they had found his damaged, but drivable car abandoned along a Westchester County highway. Paul reappeared through a week and inexplicable explanation about why he had left his car by the side of the road the night before, in reality Paul had no clue about why his car was still on the highway. In fact, prior to hearing from the police, he had assumed the car was parked in its usual spot in his driveway. Eventually, one of his drinking buddies told Paul that he had hit a gaurd rail and, even though the car was still operable, he abandoned it and his bewildered friends and calmly walked off the road.

The same morning he had spoken to the police, Paul first heard an ominous beep circularing in the neighborhood about a husband and wife, both prominent local doctors, who had been stabbed to death the night before while asleep in their beds—in their Desert colonial home at 36 Lincoln St.—the same house in which the Coxes had lived when Paul was a child.

The police mounted a vigorous investigation of the murders but for a long time, despite the money and time they put into the case, the detectives hit a dead end. The police hadn’t found any viable suspects, they had no promising leads and they lacked even a plausible motive for the crime. At the same time that the Larchmont police were searching their heads over the murders, Paul began to feel a haunting unease and an inexplicable but increasingly intense sense of guilt whenever anyone talked about the crime. He also began to experience fragmented but persistent dream-like recollections of stabbing his own parents in their old family home. Those frightful quasi-memories combined with other alcohol-related life unmanageabilities and eventually Paul sought recovery in Alcoholics Anonymous. Two years after joining, A.A., and in the clarity of sobriety,

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The Alcoholic Blackout

By Jeffrey C. Friedman, MHS, LISAC

The 7th Annual Cesar E. Chavez Behavioral Health Conference on Friday, March 25th at the Arizona State University West Campus in Glendale featured 50 national and local presenters. Topics ranged from: teenage alcohol and drug abuse, how to better treat clients of Mormon faith, Arabic and Islamic and other diverse cultures; how healthcare professionals discriminate against overweight people; why queer youths are at a greater risk for suicide; how art can promote recovery in Latino populations; and others.

Dr. Karel Kumpfer, Ph.D. of the University of Utah, was one of the event’s distinguish speakers.

“In the last two or three years, we’ve had a significant surge in alcohol, ecstasy, prescription drugs and marijuana use among teens. And you notice that all are the party drugs,” said Dr. Kumpfer, a nationally recognized researcher and substance abuse treatment expert.

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He is a summa cum laude graduate of The School of Human Services at Lincoln University (Pa.), where he worked at Cottontown includes treating chemically dependent and disordered gambling patients, lecturing on the neurology of addictive and mood disorders, and presenting workshops on a range of behavioral health issues at counseling conferences throughout the U.S., Europe and Asia. His articles have appeared in Counselor Magazine and Addiction Professionals. For more information about Cottontown de Tucson visit www.cottontowntucson.com, call 800-877-6520. Email Jeff at jfriedman@ cottontowntucson.com
PAUL was beginning to connect the dots of that horrible night, and ultimately shared his fears and haunting recollections about the stabbings at an A.A. meeting. Another person at the meeting informed the police about what he had disclosed, and detectives soon matched Paul’s fingerprints with those left in the victim’s blood all over the gristy crime scene. Shortly months later, Paul was looking at the world through the bars of the upright New York prison where he would likely spend the rest of his life.

What is an Alcoholic Blackout? Paul Cox’s journey to prison began in an alcoholic blackout—a phenomenon known to anyone with a working knowledge of alcohol and alcoholism. Actually, an alcoholic blackout is a peculiar form of retrograde amnesia caused by the presence of an insufficient amount of alcohol in a drinker’s bloodstream. The term retrograde amnesia refers to memory loss in which the affected person is unable to form new memories after an acting event (in this case, alcohol intoxication) but can remember everything—including procedural or how-to-memory, known before the onset of the amnesia. Antegrade amnesia is distinct from another kind of memory loss called retrograde amnesia. In retrograde amnesia, a person can remember present and ongoing events but cannot recall anything that happened prior to the acting event (typically a traumatic brain injury) that triggered the amnesia.

“A drunker who is in the netherworld of an alcoholic blackout can appear fairly normal; they are usually able to carry on conversations and even negotiate their way through difficult or complex tasks. There are even documented cases of pilots flying airplanes, surgeons performing complex operations and lawyers trying cases—all while in full-blown alcoholic blackouts. Both men and women drinkers seem to experience blackouts in about equal numbers and, surprisingly, blackouts are as common among social drinkers as they are in alcoholics (White, 2003). Some drinkers, particularly those with a history of blackouts, are at a higher risk for blacking out whenever they drink heavily. There might also be a link between paternal exposure to alcohol and a vulnerability toward blackouts. Some researchers believe there may even be a specific genetic predisposition to having blackouts (Harrer & Firestone, 2003).

And, while the alcoholic blackout is something that has long been recognised by medical science, the neurobiological process by which alcohol robs the brain of its ability to remember has only more recently been discovered.

Early Research

The noted alcoholism researcher E. M. Jellinek (1946) studied alcoholic blackouts in the 1940s by surveying recovering alcoholic members of Alcoholics Anonymous. Because of the high prevalence of blackouts in the drinking histories of A. A. members, Jellinek concluded that alcohol-induced blackouts, especially blackouts occurring early in one’s drinking career, were an accurate biological marker of alcoholism. In 1969, and based on interviews with 100 hospitalized alcoholics, Goodwin and his colleagues concluded that there were two distinct forms of alcoholic blackout: an acute and fragmentary. According to Goodwin, an acute blackout is a complete loss of memory, characterized by an inability—despite all efforts by the drinker or others to cue recall—to remember any events that occurred while intoxicated. Fragmentary blackouts, as the term implies, involve only a partial inability to remember events that occurred while the person was drunk. In fragmentary blackouts, forgotten events can sometimes be recalled with persistent coercing. These data and their clinical implications were the result of careful observations of blackout drinkers. Alcoholism researchers had to use a pain this kind of observed clinical data because they lacked the kind of neuroimaging technology that would allow them to look deep inside the brains of blacked-out subjects. In the late 1960s though, innovative neuromaging systems allowed a new generation of brain scientists to unravel the neurobiological interrelations of the alcoholic blackout.

The Neurobiology of an Alcoholic Blackout

In order to make sense of how alcohol can disrupt the brain’s ability to make memories, researchers first had to identify a model of memory formation that could be used as a contextual reference for the process of re-acquiring and, also one that was adaptable to the rigors of neuroscientific investigation. A number of the early studies into the neurobiology of alcoholic blackouts used a principle of memory formation first described by Eccles and others in 1968 and called the Model of Memory. In the Model of Memory, the formation of memories involves a number of distinct but coordinated actions that begin with sensory input forming an immediate, or sensory memory (the kind of memory that lasts only a few seconds). If a person attends to the sensory memory and if information encoded in the sensory memory is important enough or if the information related to the memory is rehearsed, the immediate memory may then proceed to long-term storage—a place where it can be retrieved whenever it is needed. Each of these processes is handled by a different part of the brain (Squire and Miller, 1977). The most critical of which is the hippocampus, a fleshy, pear-shaped structure deep in the forebrain. The job of the hippocampus is to coordinate the process of memory formation by routing raw sensory data from a variety of sites in the neocortex (the outer, wrinkled surface layer of the brain) and sorting this often-jumbled information into a coherent autobiographical memory. The hippocampus then sends the memory tiny structures on its surface called pyramidal

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cells—to the brain’s frontal cortex, where it can be used to reason, plan and guide behavior (Zola-Morgan et al., 1986).

In sufficient amounts, alcohol can prevent memory formation by disrupting the normal function of the hippocampus and paralyzing the pyramidal cells. When affected by alcohol, the hippocampus loses its ability to sort the random sensory data sent to it by various areas of the neocortex. Hippocampal impairment is then compounded by alcohol-induced pyramidal cells that are now incapable of sending memories, jumbled or otherwise, to the frontal cortex, by causing a dysfunction in the hippocampus and pyramidal cells, alcohol ensures that the drinker’s brain cannot form new usable memories. A person in an alcoholic blackout lives only in the present, lacking any kind of immediate or recent memory to guide their speech or actions. Memory loss is why intoxicated people often repeat themselves in conversation—going over the same conversational ground again and again.

The last twenty years have been a time when tremendous advances have been made in our understanding of the neurology of alcohol-related memory loss. In the near future, a new generation of electrophysiological recording devices—vehicles that will allow scientists to gather data from many parts of the brain simultaneously, will likely yield new and more detailed information and possibly a more nuanced understanding of how alcohol can impact a wide range of brain functioning, including memory formation. But these new insights may end up being of more interest to the neuroscientific community than to the typical problem drinker.

Most problem drinkers are already familiar with a much more common reality: Horrible things can happen to people when alcohol shuts down their brain’s ability to remember. Just ask Paul Cox.

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