Brit. J. Psychiat. (1970), 117, 705–6

Alcohol and Narcolepsy

By DONALD W. GOODWIN, FRANK FREEMON, BENJAMIN M. IANZITO, and EKKEHARD OTHMER

Narcolepsy is a syndrome characterized by recurrent sleep attacks and one or more of the following symptoms: cataplexy (transient loss of muscle tone), sleep paralysis (inability to move in the transition between sleep and arousal), and hypnagogic hallucinations (Sours, 1963). Polygraphic sleep studies indicate that narcoleptics have an abnormal sleep record. Normally, rapid eye movement (REM) sleep is preceded by 90 to 100 minutes of non-REM sleep, whereas narcoleptics often have an REM-period at the onset of sleep (Hishikawa and Kaneki, 1965; Rechtschaffen *et al.*, 1963).

This report describes an individual with symptoms of narcolepsy whose sleep was monitored polygraphically before and after the administration of alcohol.

A 24-year-old Negro male electrician was admitted to Renard Hospital in St. Louis with a nine-year history of recurrent sleep attacks, cataplexy, sleep paralysis, and hypnagogic hallucinations. At age 21 he received a diagnosis of narcolepsy and was treated with amphetamines. Subsequently he developed persecutory delusions persisting intermittently to the present time. He also gave a history of low tolerance to alcohol, stating that 'a few drinks' invariably precipitated deep, prolonged sleep, sometimes preceded by assaultive behaviour about which he was later amnesic. He was hospitalized to review the diagnosis of narcolepsy and rule out amphetamine psychosis and schizophrenia.

During the hospitalization the patient was co-operative, sociable, and manifested neither unusual sleepiness nor definite symptoms of a psychiatric disorder. After ten days of medication-free observation, he was transferred to the Renard sleep laboratory for continuous polygraphic monitoring over a 40-hour period. His sleep record was normal during the first night of monitoring, but after breakfast on the following morning, he fell promptly asleep with immediate appearance of an REM period. During the next 24 hours he had three other periods of sleep characterized by immediate appearance of REM, one occurring at the onset of the second night's sleep. He reported sleep paralysis and hypnagogic hallucinations following early-onset-REM periods, failing to report such phenomena after polygraphically normal sleep.

On the following morning, because of the history of sensitivity to alcohol, it was decided to give the patient alcohol while continuing to collect polygraphic data. Over a one and a half hour period he consumed 3 drinks, each containing 60 ml. of 86-proof bourbon diluted in 60 ml. of water, sipped slowly over 15-minute periods. During the drinking he became somewhat exhilarated and talkative but otherwise behaved normally. Within a few minutes after finishing the third drink, however, the patient became drowsy and fell into a deep sleep. Attempts to arouse him were unsuccessful in producing complete wakefulness. He could be supported in a sitting position but never opened his eyes completely and was unresponsive to questioning. He was unable to co-operate with neurological testing, but there was a definite lateral nystagmus and suggestion of dysconjugate eye movements. Vital signs were normal. He slept for about three hours before returning to the ward, where he remained drowsy for another four or five hours. Electroencephalographic tracings obtained during the sleep period showed predominantly delta waves (less than 4 cycles per second). No REM periods were observed. At the end of the drinking period his blood alcohol concentration, assayed by gas chromatography, was 110 mg./100 ml. On the following day he had no recollection of the vigorous attempts to arouse him, nor of the venipuncture.

Discussion

The long history of narcoleptic symptoms and a sleep record compatible with narcolepsy indicate the patient has this disorder. The purpose of reporting the case is to describe the patient's highly unusual response to a moderate amount of alcohol. His coma-like condition was accompanied by a slow-wave EEG pattern, so clearly was not feigned. Nor did it resemble a typical narcoleptic sleep attack. Ordinarily narcoleptics can be awakened easily during a sleep attack; attempts to awaken this patient fully were unsuccessful over a three-hour period. Furthermore, typical sleep attacks usually begin with a full-blown REM period accompanied by hypotonia and a desynchronized EEG record. Slow waves typical of Stage 4 sleep dominated this patient's

https://doi.org/10.1192/bjp.117.541.705 Published online by Cambridge University Press

EEG pattern during the coma-like reaction to alcohol.

In connection with various studies we have administered alcohol to more than 60 subjects ranging from teetotalers to alcoholics and have never observed a similar reaction to this quantity of alcohol. Whether the patient was also idiosyncratically sensitive to other drugs was not evaluated, but nothing in his history (other than a possible paranoid reaction to amphetamines) indicated this was likely. It cannot be concluded from a single case whether narcolepsy and bizarre reactions to alcohol are related. A review of the narcolepsy literature reveals no reference to the effects of alcohol on narcoleptic patients. In Pond's paper (1952) on narcolepsy, however, it is noted that half of his patients had alcoholic fathers. The father of our patient also was alcoholic. A possible relationship of narcolepsy to abnormal responses to alcohol warrants further inquiry.

Summary

A moderate amount of alcohol produced a coma-like state in a 24-year-old man with narcolepsy. This response differed from a typical narcoleptic sleep attack. The patient could not be fully awakened for a several hour period and electrophysiological data obtained during the episode were uncharacteristic of narcolepsy, showing slow-wave sleep and absence of rapid eye movements. It is unknown whether this represents a common response of narcoleptics to alcohol.

Acknowledgements

Mary P. Hayden, Margie M. Casinger, Alexis Hafken, and Eva Lee Roeder assisted with this study; which was supported in part by United States Public Health Service grant MH-09247.

References

Hishikawa, Y., and Kaneki, A. (1965). 'Electroencephalographic study on narcolepsy.' *Electroenceph. clin. Neurophysiol.*, **18**, 249–59.

Pond, D. A. (1952). 'Narcolepsy: A brief critical review and study of eight cases.' *J. ment. Sci.*, **98**, 595–604.

Rechtschaffen, A., Wolpert, E. A., Dement, W. C., Mitchell, S. A., and Fisher, C. (1963). 'Nocturnal sleep of narcoleptics.' *Electroenceph. clin. Neurophysiol.*, **15**, 599–609.

Sours, J. A. (1963). 'Narcolepsy and other disturbances in the sleep-walking rhythm: A study of 115 cases with review of the literature.' *Journal of Nervous and Mental Diseases*, **137**, 525–42.

Donald W. Goodwin, M.D., *Assistant Professor of Psychiatry, Department of Psychiatry, Washington University School of Medicine, 4940 Audubon Avenue, St. Louis, Missouri, 63110, U.S.A.*

Frank Freemon, M.D., *Special Fellow in Psychiatry*

Benjamin M. Ianzito, M.D., *Assistant Resident in Psychiatry*

Ekkehard Othmer, M.D., Ph.D., *Assistant Professor of Experimental Psychiatry*

(Please address requests for reprints to Dr. Goodwin.)

(*Received 23 March 1970*)

https://doi.org/10.1192/bjp.117.541.705 Published online by Cambridge University Press