Chafetz Named Director of New National Institute on Alcohol Abuse and Alcoholism

The establishment of the National Institute on Alcohol Abuse and Alcoholism and the appointment of Morris E. Chafetz, M.D., as Director, were announced on May 6 by Secretary of Health, Education, and Welfare Elliot L. Richardson. The Institute is part of the National Institute of Mental Health. Also announced were the appointments of eleven members of the National Advisory Council on Alcohol Abuse and Alcoholism.

"Alcohol-related problems directly or indirectly affect the lives of an estimated 36 million persons," said the Secretary. The new Institute was authorized by the Comprehensive Alcohol Abuse and Alcoholism Prevention, Treatment and Rehabilitation Act, signed into law by President Nixon on December 31, 1970. Its principal sponsor was Senator Harold E. Hughes (D-Iowa).

In accepting the appointment as Director, Dr. Chafetz said, "Backed up by increased and strengthened programs of research and training, we intend to emphasize the building of a network of comprehensive community alcohol abuse and alcoholism treatment and rehabilitation services across the United States. In addition, we will develop special programs to meet the needs of several target groups which have particularly critical alcohol-related problems, such as drinking drivers, American Indians, chronic public intoxicants, and employed alcoholics. We need to remind ourselves again and again, however, that treating only the casualties of alcohol abuse and alcoholism amounts to no more than a holding action against this major public health problem. Therefore, a keystone element of our efforts must be, and assuredly will be, the development and execution of programs and nationwide activities to prevent alcohol-related problems."

Dr. Chafetz had headed the Division of Alcohol Abuse and Alcoholism at NIMH since September, 1970, and was formerly Director of Clinical Psychiatric Services at Massachusetts General Hospital and Associate Clinical Professor of Psychiatry at Harvard Medical School.

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Cirrhosis Develops in Healing Phase of Alcoholic Hepatitis

During the healing phase of alcoholic hepatitis, 5 of 9 patients developed cirrhosis and 4 healed without significant scarring, Dr. Harold J. Fallon, Professor of Medicine of the University of North Carolina School of Medicine, told a meeting of the American College of Physicians held in New York on February 17, 1971.

These patients who developed cirrhosis tended to have more severe necrosis, but no other significant differences were noted. Prednisone, which was administered to the patients, apparently had no effect on this process. Since alcoholic hepatitis occurs in only 10-12% of severe alcoholics, it is probable that this is the lesion which precedes the development of Laennec's cirrhosis in most patients. Once cirrhosis is established, repeated bouts of alcoholic hepatitis accounts for the continued deterioration in many patients who fail to abstain from alcohol.

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EDITORIAL

Alcoholism Research Must Continue

From the very limited funds available to Dr. Jack Mendelson as Chief of the National Center for the Prevention and Control of Alcoholism in 1969, 88% of the training grants were devoted to research, 69% of extramural research grants went to the development of centers and biological research, while only 7% was devoted to clinical research. Many of those interested in the treatment of alcoholic patients were concerned that research was being overemphasized.

In fact, the concentration of funds in research during this period may well prove to have been one of the significant factors in moving alcoholism toward scientific credibility, and in definitely unraveling essential clues to its management. As Dr. Mendelson said, "Although social and cultural factors contribute to the genesis and natural history of alcoholism, successful intervention for both the primary disorder and associated illnesses appears to require a better understanding of the biological factors that underlie the addictive state."

As a new era begins, with the establishment of the National Institute on Alcohol Abuse and Alcoholism and the appointment of Dr. Morris Chafetz as Director, the concern has shifted from the clinicians to the researchers. Aware of Dr. Chafetz' interests in treatment and prevention, researchers have worried that sources of funding would dry up, leaving many promising projects in midstream and undercutting the possibility of major breakthroughs.

At this juncture, Dr. Chafetz' unequivocal statement at the NCA meetings in Anaheim, California, in April, that the new institute would indeed continue strong support for basic research was more than welcome. It is to be hoped that the Congress, the Bureau of the Budget, and the Administration will provide enough funds so that while treatment and prevention interests become more visible, the essential progress of fundamental research will also keep up the momentum gained in the past few years.

FAS

SUMMER COURSES

JUNE 27-JULY 16-1971 Summer School of Alcohol Studies, Rutgers University. Applications from Mrs. Marjorie Dreher, Rutgers University, New Brunswick, N.J. 08903.

JUNE 28-JULY 2-New York State Summer Institute on Alcohol Problems, New York University.

JULY 11-16-Eastern Pennsylvania Institute of Alcohol Studies, Ursinus College, Collegeville, Penn. Information from Miss Margaret Sutton, Chief, Community Organization, P.O. Box 90, Harrisburg, Penn. 17120.

JULY 11-16-Kentucky School of Alcohol Studies. Information from Gerald Globetti, Director, Center of Alcohol Education, Murray State University, Murray, Kentucky 42701.

JULY 18-23-International School of Alcohol Studies, Bismarck, N.D. Information from Bernard Larsen, Co-Director, Commission on Alcoholism, State Capitol, Bismarck, N.D. 58501.

JULY 25-29-14th Annual Institute of Alcohol Studies, Austin, Texas. Information from Commission on Alcoholism, 808 Sam Houston State Office Building, Austin, Texas 78701.

JULY 25-30-Training Course-Alcoholism Information and Referral Center, Madison, Wisconsin. Information from Wisconsin Extension, Department of Social Work, 609 State Street, Madison, Wisconsin 53706.

JULY 26-30—Nevada School of Alcohol and Drug Abuse. Information from G.A. Brotom, Director, Department of Physical Education, University of Nevada, Reno, Nevada 89507.

AUGUST 9-13—Western Institute on Drug Problems. Information from George C. Dimas, Director, Alcohol and Drug Section, Mental Health Division, 309 S. W. 4th Avenue, Portland, Oregon 97204.

AUGUST 15-20—Southeastern School of Alcohol Studies. Information from Charles E. Methvin, Director, 47 Trinity Avenue, S.W., Room 534, Atlanta, Georgia 30334.

AUGUST 29-SEPTEMBER 3-Florida Summer School on Alcoholism/Drug Abuse at Orlando. Information from Bureau of Alcoholic Rehabilitation, P.O. Box 1147, Avon Park, Florida 33825.


MEETINGS


JUNE 28-JULY 2-17th International Institute on the Prevention and Treatment of Alcoholism, West Berlin. Information from ICAA, Case Postale 148, 1000-Lausanne, Switzerland.

AUGUST 6-8—International Doctors in AA—Annual Meeting at Treadway Thousand Islands Club, Alexandria Bay, N.Y. 13607. Information from Secretary, IDAA, 1950 Volney Road, Youngstown, Ohio 44511.

SEPTEMBER 12-17—22nd Annual Conference of North America Association of Alcohol Programs. Hartford Hilton, Hartford, Conn. Concurrent Region I AMSA Meeting, September 12, 8-10 p.m.

OCTOBER 5-8—International Symposium on Alcoholism and Drug Dependence, Dublin, Ireland. Information from ICAA.

BOOKS

Professional Training on Alcoholism

Proceedings of a conference held April 1-2, 1970, this book presents speeches, workshops, and appendices on the present state of theory and technique in the teaching of alcoholism in professional schools, including paramedical instruction as well as medical teaching.

Actions of Alcohol: Biochemical, Physiological, and Psychological Aspects. 2 Volumes.

A comprehensive review of experimental work on the effects of alcohol on the living organism, based on an interdisciplinary approach. Volume 1 thoroughly reviews scientifically established data on the basis of biochemical, physiological, and behavioral changes caused by single doses of alcohol (acute exposure). Volume 2 treats effects of chronic alcohol consumption, interactions of alcohol with other drugs, and current views on the etiology of alcoholism and therapy of alcoholics.
Alcohol Treatment Needed in Tuberculosis Hospital

If a patient is both tuberculous and alcoholic, alcoholism must be considered the primary problem and tuberculosis the complicating, superimposed condition, not the other way around, Dr. Donald J. Ottenberg, M.D., of the Eagleville (Penn.) Hospital told the 1971 Spring Conference on Respiratory Disease held March 30 in New York City.

Of the two conditions, alcoholism is the more deeply rooted, the less well understood, and the far more difficult to treat with any degree of predictable success. The attributes that make a patient easy to manage in a tuberculosis hospital—dependence and conformity—are usually not conducive to his total welfare.

NIAAA, National Advisory Council Appointees Named

(Continued from page 1)

approval authority on grant awards in this area, and collecting information on alcohol-related studies being carried out both in the United States and abroad.

The appointees to the Council are Harold W. Demore, Jr., Executive Director of United Community Services of Metropolitan Boston; G. Carlton Edmonds, Director and Vice President of the Alcoholism Research Council, Chapel Hill, N.C.; Dale H. Farabee, M.D., Commissioner of the Kentucky Department of Mental Health; George T. Herrmann III, Chairman of the Board of George T. Herrmann and Company, a national insurance brokerage firm; Peter B. Hutt, Esq., of the legal firm of Covington & Burling, Washington, D.C.; and Quinton C. James, M.D., Regional Chief of the South Central Mental Health Service of the Los Angeles County Department of Mental Health.

Also appointed were James S. Kemper, Jr., President of Lumbermens Mutual Casualty Company of Chicago and many other insurance firms; Mrs. Marty Mann, founder and former Executive Director of the National Council on Alcoholism; Thomas P. Pike, Vice Chairman of the Board and Director of the Fluor Corporation of Los Angeles, and recent chairman of the National Advisory Committee on Alcoholism; Herbert A. Raskin, M.D., a practicing psychiatrist in Southfield, Michigan, and Chairman of the AMA’s Committee on Alcoholism and Drug Dependence; and Marsha Varnicelli, Ph.D., a post-doctoral clinical psychology intern from Cambridge, Mass., who has specialized in alcoholism.

NCA Names Officers at Annual Meeting

Irvin E. Hendryson, M.D., was elected President of the National Council on Alcoholism at its annual meeting in Anaheim, California, in April. He succeeds Luther A. Cloud, M.D., Associate Chief Medical Officer for the Equitable Life Assurance Society of the United States, who served two consecutive one-year terms.

Dr. Hendryson, an orthopedic surgeon who is professor of surgery at the University of New Mexico School of Medicine, has been a member of NCA’s Board of Directors since 1960. He is a consultant to the U.S. Department of Health, Education and Welfare and to the U.S. Public Health Society. In New Mexico, Dr. Hendryson is Chairman of the Governor’s Advisory Council, Emergency Medical Services, and a consultant for the Alcohol Countermeasures Program, Department of Transportation, in Albuquerque.

Also at the annual meeting, John K. MacIver, an attorney and partner in the law firm of Michael, Best and Friedrich, Milwaukee, was selected Chairman of the Board of NCA. He succeeds F. Porsha Russell of Kansas City, Missouri, who served two consecutive one-year terms. Mr. MacIver is a past president of the Milwaukee Council on Alcoholism.

Other officers elected were: Thomas P. Pike, First Vice President; Los Angeles; William S. Simpson, M.D., Second Vice President, Topeka, Kansas; Thomas G. Terbell, Treasurer, Greenwich, Conn.; and Byron S. Miller, Secretary, New York City.
Alcoholism: Nature or Nuture? Reports From NCA Conference
(Continued from page 1)

Genetics Affects Preference for Alcohol in Rats

An experiment on a behavioral characteristic, free choice ethanol consumption, which varies widely among inbred strains of mice, was reported by Professor John L. Fuller of the State University of New York at Binghamton. The trait was studied in hybrids between a high-prefering strain and a low-prefering strain. The results indicate that two loci or closely linked blocks of genes control the major portion of the difference in alcohol preference between the two strains. However, much more experimentation will be required to prove this. It is possible that the "polygenes" controlling this preference may be separable.

Gerald E. McClearn, Ph.D., of the University of Colorado, discussed sleeping time tests on strains of inbred mice. The results of his work showed that at least three different phenomena are based on genetic differences: (1) preference for alcohol; (2) level of liver alcohol dehydrogenase activity; and (3) sleeping time. The last phenomenon is due to brain sensitivity.

In a study of behavioral and physiological differences between rat strains specially selected for their alcohol consumption, Kalervo Eriksson of Helsinki, Finland, noted that the sex differences found in alcohol consumption can be partly explained by the weight difference. The females have much greater energy requirements because of their smaller size and also have a greater ability to eliminate alcohol. However, females showed the same level of drinking behavior than the males. In general, though, the heritability of drinking behavior is relatively low in mice and rats and less population-specific than is generally supposed. Dr. Eriksson also suggested that morphine addiction and alcohol addiction are interchangeable.

Operant Conditioning Theory of Alcohol and Opiate Addiction

Addicts endow their children with a biological legacy which will make them "good dope fiends or alcoholics under proper circumstances, but also make them brighter, more dynamic people under other circumstances," says John R. Nichols, Ph.D., Professor of Social Science and Psychology, Pennsylvania State University. Dr. Nichols has proposed a general theory of addiction in which chronic alcoholism and opiate addiction are expressions of the same basic process. In brief, the addict learns to interpret the stimuli from his environment and from his own body in a new way: he learns to evaluate these stimuli as signals of his need for his drug. The basic process is operant conditioning—a powerful method for changing behavior.

In his work with laboratory animals, Dr. Nichols and his co-workers have shown that the behavior of animals as well as humans could be induced to give themselves opiates, thus invoking the operant conditioning model. However, rats showed differences in susceptibility to relapse. To find out whether these differences in susceptibility were inheritable, high drinkers in each generation were mated to high drinkers; low drinkers were crossed with low drinkers. Selection continued through four generations, always within strains. Differences between strains were significant. Also differences increased significantly from generation to generation.

A subsequent experiment showed that addiction-prone rats remained addiction-prone, and addiction-resistant rats remained addiction-resistant, whether raised by their own mothers or by foster mothers. Furthermore, two strains bred for susceptibility and resistance to opiate addition showed a parallel susceptibility and resistance to alcohol addiction. Twin Study and Half-Sib Research Show Genetic Predisposition

An analysis of 13 alcohol-related questionnaire items from the National Merit Twin Study showed an interesting and probably dependable tendency toward high heritability for items suggestive of heavy drinking—but a tendency subject to some variety of possible interpretation," said John C. Loehlin, University of Texas, who with Robert C. Nichols designed the study and gathered the data. Approximately 850 pairs of like-sex twins were among the 600,000 high school juniors who took the National Merit Scholarship tests in 1962. These twins were diagnosed as identical or fraternal by means of a mail questionnaire, and filled out a battery of personality and interest questionnaires and self-ratings, again by mail.

In estimating the heritability of a trait from twin correlations, Loehlin found that with only minor exceptions, the items suggestive of excessive drinking have the highest estimates of genetic determination, from about a third to more than a half of the variance; the items concerning drinking customs show low heritabilities; and the items on attitudes toward drinking show no heritability—in fact, the identical twins are if anything slightly less alike than the fraternal.

In qualifying these results, Loehlin pointed out first that the items suggestive of heavy drinking tend also to be those infrequently endorsed by the twins, so that the actual number of concordant heavy-drinking pairs involved in some of the correlations is fairly small. Second, the heritability estimates for the heavy drinking items are in most cases higher than the corresponding identical twin correlations, which strongly suggest that the estimates are inflated, perhaps by a greater environmental similarity than for fraternal twin pairs.

Finally, Loehlin noted, "showing that genes are involved doesn't tell you how they are involved." Identical twins of a pair were more likely to both have had a hangover during the past year than were fraternal twins of a pair. This is probably due in some sense to the fact the identical twins were genetically more alike. However, it cannot be established whether the genetic similarity had its principal effect in a similar physiological after-response to alcohol, or in a tendency to enjoy the same parties.

A study of twins in Finland reported by Juha Partanen of the Finnish Foundation of Alcohol Studies showed that there was a hereditary component in such traits related to drinking as amount consumed per drinking occasion, frequency of drinking, and abstinence. Similar indications were observed in connection with coffee drinking and smoking. On the other hand, none of the variables describing symptoms of alcoholism, such as ability to control one's drinking, social complications resulting from drinking, or arrests for drunkenness, showed a significant degree of heritability. The collection and analysis of longitudinal data would help clarify the relationships of intrafamily differences and other variables.

Genetic research may not only explain the occurrence of some of the complications of alcoholism but may also help to recognize different subspecies of the disease, reported Dr. Lennart Kaj of Lund, Sweden. His own study of twins showed a higher concordance rate between loss of control and physical dependence in identical than in fraternal twins, while Partanen and his coworkers found a low degree of hereditability for loss of control. The data, however, are not truly comparable, and environmental factors were only partly controlled for both studies since the twins were reared together.

Dr. Kaj also proposed that a model for alcohol preference parallel to that suggested for peptic ulcer by George Engel be developed. According to this model, the pepsinogen level is largely genetically controlled. A high pepsinogen level is reflected in or linked with a low (Continued on page 5)
threshold for infantile oral deprivation. Individuals with a high pepsinogen level who were exposed to early oral deprivation thus constitute a high-risk group for ulcer in adult life. The virtue of this model, says Dr. Kaij, is that it integrates genetic, biochemical, and psychodynamic observations of the disease. An analogous model for the explanation of alcohol preference is within the realm of possibility.

A child is more likely to become an alcoholic if he himself had an alcoholic biologic parent than if he lived with an alcoholic parent figure. Research on half-sibling data, reported by Marc Alan Schuckit, M.D. of the Washington University School of Medicine, indicates that when environmental and genetic factors are compared, the genetic factors are more important. The conclusions reached in family history research appear to support the same conclusion, he said.

**Color-Blindness Link to Alcoholism not Proved**

Color-blindness does not appear to be genetically linked to alcoholism, two researchers reported, refuting evidence cited in 1964 by Cruz-Coke in Chile. Dr. James W. Smith, Director of Schick's Shadel Hospital, reported that red-green color-blindness tests on 173 male and 33 female alcoholic patients both before and after treatment showed that the rate of defectiveness was essentially the same as in the general population. Yellow-blue color-blindness tests showed similar results.

However, deficiencies in overall color discrimination ability were correlated with abnormal liver tests and with inferior performances on psychological tests, suggesting that color vision defects are like other partially reversible biochemical and behavioral disorders seen in alcoholics. They result from chronic alcohol abuse.

Dr. H. C. Thuline, Director of Laboratories at the Rainier School, Buckley, Washington, suggested that Cruz-Coke's evidence, on which he based the hypothesis for association between alcoholism and color-blindness of known genetic type, used inappropriate methods and improperly interpreted results. However, Dr. Thuline stressed that because of the clinical importance attached to the question of genetic predisposition to alcoholism, the true nature of reported color vision test results should be determined.

**Chromosomal Irregularities Found in Alcoholics**

Defective chromosomal patterns were found in the blood cells of a group of alcoholics studied by Denes de Torek, Ph.D. of the Carnegie-Mellon University and Mayview State Hospital. These defects may inhibit the production of enzymes which enable the body to handle alcohol in the normal way.

Blood group determinations, cross karyotypic determinations and separation, correlation, and identification of the electrophoretic patterns of serum proteins were carried out on groups of both alcoholics and nonalcoholic subjects, divided into race, sex, and age groups. Subjects were further divided into transient alcoholic patients and to those suffering from permanent organic brain damage (OBS). Strikingly, 43.7% of chromosomal complements obtained from all the counted metaphase plates of alcoholism-connected OBS patients revealed the 2n-1 situation, and thus the number 45 is overwhelming among the cells of these patients. Furthermore, only 4.4% of the counted cells carried the normal diploid number of 46. These random peculiarities in chromosome numbers lend support to the theory of aneuploidy as a result rather than as the cause of alcoholism.

Although these studies show clearly that the genetic material is involved with alcoholism, it remains to be learned whether such chromosomal anomalies exist before the alcoholic stage or whether they are a partial or total result of high consumption of alcoholic beverages.

**Alcoholism Detrimental to Fetal Development**

Chronic alcoholism creates an unhealthy intrauterine environment for the developing fetus, the consequences of which may be life-long, according to Dr. Christy Ullehand of Seattle, Washington. In a group of infants observed over an 18-month period, intrauterine growth failure occurred in 10 of the 12 offspring (83%) of women whose alcoholism was readily recognized, while only 2.3% of the infants of nonalcoholic mothers were undergrown.

The infants also failed to thrive, having weight and head circumferences below the 3rd percentile on follow-up. Of 10 such infants evaluated by Gesell and/or Denver developmental scales, 5 were retarded and 3 borderline.

**Birth Order and Alcoholism**

Last-born males in families of from 4 to 10 children have a significantly higher incidence of alcoholism, as well as several other psychiatric illnesses, said Herbert Barry III of the University of Pittsburgh School of Pharmacy and Herbert Barry, Jr., of the Department of Psychiatry of Massachusetts General Hospital.

**Differences Between Families of Alcoholics and Criminals**

The love and affection of even one parent may deter the development of alcoholism, said Joan McCord, Ph.D., of Drexel University. Using an operational definition of alcoholism which depended upon publicly recognizable behavior, she matched the backgrounds of individuals who had received treatment as alcoholics or had been convicted twice for drunkenness with the backgrounds of nondeviant men matched to them on socio-economic variables, family size, and ordinal position, and with criminals from similar circumstances. Among the three groups, the alcoholics had unique exposure to both nonpunitive parental rejection and to homes in which the sex roles were confusing.

Dr. McCord also differentiated between the rejecting, passive father of the alcoholic and the rejecting, punitive father of the criminal.

**Alcoholics Make Poor Parents**

Children of alcoholics learn from their parents complex behavior patterns that provide a series of responses available to them in times of stress when they themselves become adults, says E. David Burk, M.D., Assistant Professor of Psychiatry, of the Stanford University School of Medicine, and Director of the Peninsula Children's Center. These children are vulnerable to the influences of poor or inadequate models—alcoholics who happen to be their parents. They see adults coping with stress by using alcohol at the same time they are exposed to mass media that encourage alcohol use as a necessary accomplishment of pleasure and socialization. When the child is younger, excessive alcohol use may appear as an effective social stimulant, and as a means to reduce anxiety. The stage is set for similar behaviors in later life. By the time the social or physical decay of chronic alcoholism is apparent in the parent, the child may be so reinforced and patterned toward excessive alcohol use that the outcome is certain. However, many children of alcoholics do achieve adulthood without these attitudes and behaviors, indicating that there may be other factors involved. Our best strategy for reducing the problem of alcoholism in the next generation, Dr. Burk believes, may be to provide the children of alcoholic parents with an opportunity to observe mature models with healthy attitudes and behaviors toward alcohol.
Alcohol Enhances Hepatic Collagen Accumulation

Feeding alcohol with adequate or protein-deficient diets results in hepatic collagen accumulation, which is associated with enhanced collagen formation and increased hepatic collagen proline hydroxylase activity. Lawrence Feinman and Charles S. Lieber of the Mount Sinai School of Medicine and the Bronx VA Hospital told the meetings of the American Society for Clinical Investigation, held May 1-4 in Atlantic City.

The authors said that after 7 months, in primates fed alcohol, hydroxyproline concentration, indicative of collagen accumulation, doubled; in rats it increased by 69%. Rat and primate livers were assayed for collagen proline hydroxylase activity, which is necessary for collagen synthesis and represents the earliest indicator of hepatic fibrosis. Enzyme activity rose 50% in rats fed ethanol one month and 100% after 6 months; it increased 44% in primates after 7 months.

Ethanol Vapor Produces Dependence in Mice

Mice housed for several days in a transparent plastic box to which ethanol vapor was continuously delivered in the air inflow, maintained intoxicating blood alcohol levels, report Dora B. Goldstein and Nandita Pal of the Department of Pharmacology, Stanford University School of Medicine. It is difficult to maintain high blood alcohol levels in mice by injections, because they eliminate alcohol very rapidly. On removal from the alcohol all the mice developed withdrawal signs, which can be graded to indicate the time course and intensity of the withdrawal reaction. Physical dependence can develop in as short a time as 2 days.

These techniques were used by Avram Goldstein and Barbara A. Judson of the Department of Pharmacology, Stanford University, to test the hypothesis that physical dependence upon alcohol is due to the formation of an endogenous opiate. They tested whether ethanol-dependent mice would show typical opiate-dependent behavior (withdrawal jumping syndrome) when challenged with the opiate antagonist naloxone. At doses up to the convulsive range, naloxone caused no opiate-withdrawal jumping in any of the animals; nor did naloxone modify the course of the alcohol withdrawal syndrome. Since even a mild degree of opiate dependence can be detected by the naloxone test, they concluded that alcohol dependence is not a manifestation of dependence upon any endogenous opiate. (Science, Vol. 172, April 16, 171, pp. 288-292).

Staff Underestimates Alcoholism in Municipal Hospital

In an investigation of the extent of alcoholism at Harlem Hospital, the house staff diagnosed as alcoholic only 50% of the patients whom the investigators—Jane McCusker, M.D., Charles E. Cherubin, M.D., and Sheldon Zimberg, M.D.—had found to have moderate or greater degree of alcoholic abuse. The staff made only one false positive diagnosis. The total prevalence rate was 47%: 60% for males and 34% for females.

This high prevalence of alcoholism places a burden on the already overcrowded hospital system. This study, say the authors, indicates the need for wider appreciation of the alcoholism problem by the house staff, and for the provision of suitable therapeutic programs aimed at reducing the stay of alcoholics in the hospital. (New York State Journal of Medicine, April 1, 1971, pp. 751-754).

Adrenal Function and Alcoholism

Two studies by a team led by Dr. Jack H. Mendelson, of the Department of Psychiatry, Boston City Hospital, indicate that chronic alcohol ingestion and its concomitant effects are associated with adrenal cortical activation, stimulator of adrenergic activity, and alteration in pathways of catecholamine catabolism. (Psychosomatic Medicine, Vol. 33, No. 2, March-April 1971, pp. 145-180).

Ethanol Changes Metabolism of NE in Alcoholic

Ethanol not only changes the catabolic pathways of norepinephrine (NE), but also its synthesis, pool size, and turnover rate, report Drs. S. E. Gitlow, L. M. Bertrani, S. W. Dziedzic, and B. L. Wong of the Mount Sinai School of Medicine. They studied the long-term effects on NE metabolism of ethanol ingestion in an alcoholic at three stages; during ethanol ingestion, in withdrawal, and three months after his return to normal activities. During these three periods, total NE synthesis decreased. The labeled pool size was increased during ethanol ingestion and withdrawal. (Clinical Research, Vol. 19, 1971, p. 349).

San Francisco Alcoholism

The alcoholism rate in San Francisco may be the highest in the nation, the mental health advisory board of that city recently reported to the board of supervisors and mayor. About 48,000 persons have drinking problems sufficient to interfere with functioning. The cirrhosis rate and the suicide rate are almost four times the national rate.