

*The*

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NATURAL HISTORY

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*of* ALCOHOLISM

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*REVISITED*

GEORGE E. VAILLANT



# *The Natural History of Alcoholism Revisited*

≈ George E. Vaillant

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# Acknowledgments

The title page of this book is misleading, for it suggests a single authorship. In truth, this book represents a vast collaborative effort that has continued for 45 years. The effort began in the late 1930s as two separate studies: a study of juvenile delinquency by Sheldon and Eleanor Glueck at Harvard Law School, and the Grant Study by Clark Heath and Arlie Bock at the Harvard University Health Services. In 1972 the two studies were brought together under the auspices of the Harvard Medical School as the Study of Adult Development. I have written this book as one member of a very large team, and I have played for the team for only a third of its 45 years. Many others can also claim authorship.

Clearly, I am most deeply indebted to the several hundred erstwhile college sophomores and Boston schoolboys who are the study's subjects. Since 1940, they have generously shared their time, their lives, and their experiences. This work is also indebted to the two independent teams of researchers who conceived, sustained, funded, and guided this longitudinal research for the first 30 years of its existence. The mere listing of names grossly understates the enormous devotion and painstaking hard work required to follow more than 600 individuals for 40 years.

Sheldon Glueck and Eleanor Glueck were responsible for the creation of the Core City sample, as a control group for their classic text, *Unravelling Juvenile Delinquency*. In their work many helped them—especially Mildred

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Not only has this work had many collaborators, it has enjoyed many patrons. The first explanation that I received for the name of the Grant Study was: "Well, it took an awful lot of grants to support the study for 30 years."

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Finally, anyone who has ever written an academic text knows that it is editors, research assistants, medical artists, and typists smarter than oneself who make such books possible and the reader's task bearable. Credit for authorship belongs to them as well as to the initial scribbler. In this regard, I render special thanks to Eric Wanner, Linda Son, and Camille Smith. Nancy Knysh, Kate Hughes, John Oberbeck-Friedlich, Liv Bjornard, and Lianne Carlin also played valuable roles.

I hope that the many others who have helped me to create this book but whose names do not appear will understand that the limiting factor is space and not gratitude.

G.E.V.  
1983

### ~ Acknowledgments Revisited

Almost 15 years have passed since the above acknowledgments were written and *The Natural History Of Alcoholism Revisited*, like its predecessor, has benefited from the help of many people. The most important contributors to the book, of course, have been the more than 600 surviving men of the College and the Core City samples who have collaborated with the Study of

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Finally, Angela von der Lippe and Camille Smith, editors at Harvard University Press, deserve my thanks for their creative guidance and help in transforming my typescript into a finished book.

G.E.V.  
1995

# Contents

The Problem	1
<i>The Problem Revisited</i> · 2	
The Seven Questions · 3	
<i>The Questions Revisited</i> · 11	
I. What Is Alcoholism?	
1. Is Alcoholism a Unitary Disorder?	17
Empirical Evidence · 23	
2. The Etiology of Alcoholism	46
Cultural Factors · 59	
Genetic Factors · 64	
<i>Genetic Factors Revisited</i> · 72	
Childhood Environment · 73	
Personality and Premorbid Emotional Stability · 75	
Anxiety and Alcohol Abuse · 77	
<i>Anxiety and Alcohol Abuse Revisited</i> · 79	
Depression and Alcohol Abuse · 80	
<i>Depression and Alcohol Abuse Revisited</i> · 82	
Sociopathy and Alcohol Abuse · 85	
<i>Sociopathy and Alcohol Abuse Revisited</i> · 96	
A Case Example · 104	
Other Etiological Factors · 109	
Conclusions · 115	



3. The Natural History of Alcoholism	120
<i>Alcoholism in Women Revisited</i> • 122    Patterns of Alcohol Use among the Core City and College Men • 124	
<i>Groups IV and V Revisited</i> • 127    Comparison of the College and Core City Samples to a U.S. Sample • 129	
<i>Prevalence of Alcohol Abuse Revisited</i> • 131    Patterns of Alcohol Abuse and Mental Health • 132    Patterns of Alcohol Abuse and Physical Health • 137	
<i>Coronary Heart Disease Revisited</i> • 139    The Safe Limits of Alcohol Consumption • 141    The Natural History of Treated and Untreated Alcoholism • 142	
<i>The Natural History of Alcoholism Revisited</i> • 150    The Core City Sample • 156	
<i>The Core City Sample Revisited</i> • 160    Is Alcoholism a “Progressive Disease”? • 163	
<i>Table 3.9 Revisited</i> • 170    A Multifaceted Disease • 174 <i>Progression Revisited</i> • 178	
The Problem of the Atypical Alcoholic • 184    The Natural History of Treated Alcoholism • 186    Alcoholism and Morbidity • 200	
<i>Mortality Revisited</i> • 205    Hypertension and Alcohol Abuse • 212    Prognosis • 215	
Habit, Addiction, and Relapse • 221	

## II. Patterns of Recovery

4. Paths into Abstinence	231
Definition of Abstinence • 231    “Abstinence” <i>Revisited</i> • 233    Etiology of Abstinence • 235	
<i>Stable Abstinence Revisited</i> • 246    The Relationship of Alcoholics Anonymous to Abstinence • 254	
<i>Alcoholics Anonymous Revisited</i> • 265    The Consequences of Abstinence • 269	
<i>The Consequences of Abstinence Revisited</i> • 276	
5. Return to Asymptomatic Drinking	278
<i>Asymptomatic Drinking Revisited</i> • 284    Case Histories • 286	
<i>Return to Controlled Drinking Revisited</i> • 294    Social Environment • 298	
<i>Behavioral Training Revisited</i> • 302    Conclusion • 303	



## III. Methodology

6. The Sample	307
The College Sample · 307    The Core City Sample · 311	
Comparison of the Two Samples · 315	
7. The Measures	318
Childhood Premorbid Variables · 318    Midlife Outcome	
Variables · 329    Attrition · 337    Limitations · 343	

## IV. Lessons for Treatment

8. The Doctor's Dilemma	347
The Clinic Sample as an Illustration of the Dilemma · 348	
Natural Healing Forces in Alcoholism · 352    Resolution	
of the Dilemma · 359 <i>Alcoholism Treatment Revisited</i> · 360	
9. Suggestions for Would-Be Helpers	362
<i>Pharmacotherapy and Psychotherapy Revisited</i> · 369	
Additional Guidelines · 371	
A Summing Up	375
Alcoholism: Symptom or Disease? · 376 <i>Symptom or</i>	
<i>Disease Revisited</i> · 377    Is Alcoholism a Progressive	
Disease? · 378    Are Alcoholics Premorbidly Different? · 380	
Is the Cure Worse Than the Disease? · 382    Can	
"Real" Alcoholics Ever Safely Drink Again? · 383	
<i>Abstinence versus Controlled Drinking Revisited</i> · 383	
Which Clinic Treatments Help? · 384 <i>Clinic Treatment</i>	
<i>Revisited</i> · 385 <i>Behavioral Approaches Revisited</i> · 387	
Is Recovery through AA the Exception or the Rule? · 388	
A Final Reminder · 388	
Appendix: Measurement Scales	393
References	407
Index	439



## ≈ *The Problem*

Alcoholism is a disorder of great destructive power. Depending on how one defines alcoholism, it will afflict, at some time in their lives, between 3 and 10 percent of all Americans. In the United States alcoholism is involved in a quarter of all admissions to general hospitals, and it plays a major role in the four most common causes of death in males aged 20 to 40: suicide, accidents, homicide, and cirrhosis of the liver. The damage it causes falls not only on alcoholics themselves but on their families and friends as well—and this damage touches one American family out of three.

Such a serious and widespread problem demands to be studied, yet our lack of knowledge about alcoholism is astonishing. If to the casual bystander the disorder is obvious, some experts who have studied alcohol abuse for years doubt that any such entity as alcoholism exists. The reason is that alcoholism has an unstable, chameleon-like quality that makes it difficult to pin down at any given time.

Thus, the professional literature on alcohol abounds in controversy; and controversy, if unresolved, may add to uncertainty and actually detract from knowledge. For example, is alcoholism caused by heredity or by environment? Is it a cause or a result of mental illness? Is it a sin or a sickness? Some experts contend that calling alcoholism a disease is merely a semantic trick to counter the lingering belief that it is a vice. Others view alcoholism as an insidious disease that makes itself known with the first drink. Until we know the answers to such questions, we will be unable to devise rational ways to treat individuals with alcoholism.

But obtaining the answers is not easy. To be trusted, information should come from meticulously conducted long-term prospective studies—studies in which individuals are selected for study *before* they develop problems with



alcohol and then followed for many years. Hundreds of retrospective papers have been written about the genesis of alcoholism, but there are almost no prospective studies of its development in a normal population. Most of the existing studies have a cross-sectional design—a design that captures the characteristics of alcoholics at a certain point in their lives but reveals little about how they got to that point or what will happen to them thereafter. Alcoholism often lasts a lifetime, and it is hard to believe that virtually no studies have followed alcoholics for more than five years.

In the search for answers about alcoholism, longitudinal study offers many advantages. For one thing, since alcoholism is a chronic affliction, both its victims and the nature of their disability change over time. Thus, a cross-sectional view of an alcoholic's life will never adequately capture the nature of the disorder. Second, alcoholism is a malady about which there are no black and white answers, and longitudinal study is far better suited than cross-sectional study to elucidate clinical "grays." Third, unlike most habits and conditions, alcoholism has direct, as well as indirect, effects upon the central nervous system. Alcoholism affects personality and perceptions about the past so markedly that the true facts of an alcoholic's life can often be discovered only by prospective study.

The insights about alcoholism that I present in this book come from such a prospective study, the Harvard Medical School's Study of Adult Development. This project has followed 660 men from 1940 to 1980, from adolescence into late middle life. Information has been collected about many aspects of their lives, including their use of alcohol. The 660 subjects fall into two groups: 204 in the upper-middle-class College sample, chosen for study when they were sophomores at an elite college; and 456 in the less privileged Core City sample, chosen when they were inner-city boys of junior high school age. The data about these men's lives are supplemented by information from a third, very different group of subjects, the Clinic sample: 100 alcohol-dependent men and women followed for eight years after being admitted to a clinic for detoxification. Taken together, these three diverse samples yield a fund of information about alcohol use and abuse that no other published study can match.

## The Problem Revisited

Fifteen years have passed since the above was written and the data were collected for the earlier version of this book; 12 years have passed since the literature was reviewed to provide comment on, challenge to, and confirma-



tion of its data. Much has changed; much has stayed the same. Rather than alter the original text, I have chosen to add data collected and literature reviewed since 1980 in new sections, under headings marked with the symbol ∞ and the word *Revisited*. (Additional brief new passages appear in occasional footnotes.) One purpose of using separate sections for new material is to underscore the relativity that time imposes upon "truth." A second purpose is to highlight the power of further long-term follow-up to add to our understanding of complex social problems.

## The Seven Questions

There are at least seven controversial questions that longitudinal study of alcoholism might help to resolve: (1) Is alcoholism a symptom or a disease? (2) Does alcoholism usually get progressively worse? (3) Are alcoholics, *before* they begin to abuse alcohol, different from nonalcoholics? (4) Is abstinence a necessary goal of treatment, or can insisting on abstinence sometimes be counterproductive? (5) Is returning to safe social drinking possible for some alcoholics? (6) Does treatment alter the natural history of alcoholism? (7) How helpful is Alcoholics Anonymous in the treatment of alcoholism?

Let me pose these seven questions in greater detail. First, is alcoholism a symptom, a social problem, or a disease? As long ago as 1804 Thomas Trotter wrote unambiguously: "In medical language, I consider drunkenness, strictly speaking, to be a disease produced by a remote cause in giving birth to actions and movements in a living body that disorder the functions of health" (p. 2). Yet in 1882, in a pamphlet entitled "Drunkenness a Vice, Not a Disease," J. E. Todd wrote: "Every human soul is worth saving; but what I mean is, that if a choice is to be made, drunkards are about the last class to be taken hold of." And a century and a half after Trotter, McGoldrick (1954) could still write: "Alcoholism is no more a disease than thieving or lynching. Like these, it is the product of a distortion of outlook, a way of life bred of ignorance and frustration." Perhaps it was more from charity than conviction that the World Health Organization in 1951 decreed that "Alcoholism (or rather certain forms of it) is a disease process."

Since the WHO report, writers like Robinson (1972) have suggested that the term "alcoholism" is too vague to have meaning. Others (Roman and Trice 1968) have reviewed the multiple dangers of the medical model and the labeling of alcoholics. Some writers have even suggested that the disease label can provide alcohol abusers with a means of avoiding responsibility.

Gitlow has argued from authority: "The American Medical Association,



American Psychiatric Association, American Public Health Association, American Hospital Association, American Psychological Association, National Association of Social Workers, World Health Organization, and the American College of Physicians have now each and all officially pronounced alcoholism as a disease. The rest of us can do no less" (1973, p. 8).

Others, however, identify this kind of statement for what it is, a sociopolitical generalization (Pattison et al. 1977; Blane 1978). They remind us that in delineating the disease concept of alcoholism, Jellinek (1960) was far more cautious, and they suggest that there is no single entity which can be defined as alcoholism. Their point is that alcoholism cannot be reified but reflects a collection of various symptoms and episodic behaviors that collectively make up perhaps as many alcoholisms as there are alcohol abusers.

The debate goes on not only between individuals, but also within individuals. For example, a few years ago Hodgson and his colleagues wrote: "Whenever alcoholics are said to be characterized by a particular attribute then we can be sure some are and some are not" (1978, p. 339). The next year the same authors suggested: "the syndrome of alcohol dependence is given expression and in various ways . . . but remains nevertheless, a unitary syndrome" (1979, p. 9). In other words, alcoholism is and is not a disease.

But the debate over whether alcoholism is a disease is far more than just a semantic argument. Is alcohol abuse the cart or the horse? Is it the underlying cause or the sometime result of the patient's disordered personality, culture, or lifestyle? Our answer to this question will define our approach to treatment.

The most compelling empirical evidence against the existence of a sharp distinction between alcohol use and the disorder, alcoholism, has been Cahalan's (1970) study of a national panel of alcohol users, which suggests that drinkers cannot be divided into social drinkers and alcoholics, but that the categories of alcohol users and alcohol abusers merge with each other depending upon one's definition of abuse. Alcohol abuse is not black and white; it is gray. One of my purposes in this book, therefore, in following 600 men for four decades, is to watch individual lives unfold and to examine the different shades of gray expressed through their drinking behavior. I shall apply several different definitions of alcoholism and try to determine the circumstances under which the disease model seems legitimate.

In this context a paradox must be acknowledged. For purposes of conceptualization, I shall examine alcoholism within the medical model. But I must concede at the outset that however dexterously alcoholism may be shoe-



horned into a medical model, both its etiology and its treatment are largely social. Indeed, in modern medicine there may be no other instance of sociology's contributing so much to our understanding of a so-called disease. Thus, a major focus of this book will be to contrast social and medical models of alcoholism to see if they are congruent.

Related to whether alcoholism is a disease is the second unanswered question: Is alcoholism inevitably progressive? Once a regular pattern of alcohol abuse is established, once dependence, whether psychological or physiological, seems clear, does the disorder take on a life of its own? Does alcoholism, like Huntington's chorea, multiple sclerosis, and diabetes, manifest the statistical tendency to get worse without treatment? One side of the debate is set forth in Jellinek's model of phases in the drinking history of alcoholics (1952); this model represents alcoholism as an insidious, progressive disease that if not arrested ends eventually in death. This model is also a basic tenet of *Alcoholics Anonymous*. The other side of the debate is set forth in a paper by Drew (1968) who found that after age 50 there was a progressive decline in the number of alcoholics presenting themselves for treatment. By minimizing the contribution of death to this decline, Drew suggests that spontaneous return to normal drinking and spontaneous abstinence account for the improvement.

Because we lack longitudinal studies of both treated and untreated alcoholics, the current student of alcoholism can go no further than to agree with Cahalan (1970), who pointed out that with passage of time some alcoholics will die, some will become abstinent, some will return to social drinking, and some will be unchanged. The proportion of alcoholics following any single route is unknown. Positions taken on the progressive nature of alcoholism often depend more upon the treatment orientation of the observers than upon the adequacy of their data. Whether or not alcoholism is inevitably progressive can only be determined by following large numbers of alcoholics for long periods of time without significant attrition and without the bias that results from selecting a clinic population.

The third question about which there is sustained controversy is whether alcoholics are premorbidly different from nonalcoholics. Is their biochemistry different? Is their heredity different? Is their childhood environment different? Is their premorbid personality different? Recent years have seen most of the hypothesized biochemical differences between alcoholics and nonalcoholics put to rest (Jellinek 1960; Pattison et al. 1977; Mello and Mendelson 1978). There is no good evidence that alcoholism is caused by hypogly-



cemia, vitamin deficiency, disordered metabolic pathways, or “allergy” to alcohol in other than the most metaphorical sense. But the other etiological questions are not as easily answered.

In 1938 Karl Menninger could make the bold statement: “the older psychiatrists . . . considered alcoholism to be an hereditary trait. Of course, scarcely any scientist believes so today, although it’s still a popular theory. Alcoholism cannot possibly be an hereditary trait, but for a father to be an alcoholic is an easy way for a son to learn *how* to effect the retaliation he later feels compelled to inflict” (p. 177). Modern evidence unseats Menninger’s certainty. While it is unlikely that alcoholism represents a genetic disorder caused by a single aberrant allele, there is increasing evidence that genetic factors play a significant role (Goodwin 1976; Wolff 1972). Studies of adopted children (Goodwin 1976) suggest that alcohol abuse by the adoptee’s *biological* parents plays a greater role in alcohol abuse in the adoptee than does alcohol abuse in his *environment*.

But if in recent years it has become increasingly clear that environmental patterns of alcohol abuse are relatively unimportant as a cause of alcoholism, cultural patterns of alcohol *use* are very important. The attitudes toward drinking and the socially sanctioned drinking practices surrounded by which a child learns to drink play an important role in the development of subsequent alcoholism (Jellinek 1960; Pittman and Snyder 1962; Heath 1975). Economic factors and patterns of legislation appear to be equally important (Bruun et al. 1975).

If genes and society both play a role in alcoholism, what is the effect of childhood environment? Retrospective studies (Blum 1966) speculate that childhood factors are critical to the genesis of alcoholism. The much better designed prospective studies by the Gluecks (1968), the McCords (1960), and Robins (1966) certainly suggest that childhood environment contributes to antisocial behavior; and in delinquent populations *premorbid* antisocial behavior is associated with *subsequent* alcohol abuse. However, in the past there have been no prospective studies of middle-class or nondelinquent blue-collar families that have produced enough alcoholics to answer the question: do most alcoholics or only premorbidly sociopathic alcoholics have disturbed childhoods? In the present follow-up of 600 nondelinquent adolescents with well-characterized childhoods, it should be possible to answer this question.

An equally important area of disagreement in the alcohol literature is whether the alcoholic is premorbidly mentally ill or at least premorbidly manifests a specific personality style. Jellinek wrote: “In a large proportion



of alcoholics—the predominant species of alcoholism on the North American continent—prealcoholic, high psychological vulnerability is essential” (1960, p. 153). Wallerstein expressed the view that “alcoholism is a *symptomatic* expression of a deep-seated emotional difficulty” (1956, p. 228); and in a retrospective study of 161 alcoholics, Sherfey (1955) maintained that in every one of them, drinking was secondary to an abnormal psychiatric condition. Finally, as recently as 1973, in his widely used textbook on clinical psychiatry, Kolb wrote: “In spite of the conviction of most alcoholics that they would be quite normal if they ceased drinking, psychologically well-adapted personalities are seldom found during periods of sobriety” (1973, p. 205).

But opponents of this view are equally emphatic. In an often quoted review, Syme wrote: “it is rather clear that, on the basis of the evidence (all available relevant literature published from 1936 to 1956), there is no warrant for concluding that persons of one type are more likely to become alcoholics than persons of another type” (1957, p. 301). Syme did quote one MMPI (Minnesota Multiphasic Personality Inventory) study where the author wrote that the more maladjusted the individual the more need he seemed to have for alcohol as a crutch. However, in a prospective study of the MMPI, Kammeier and colleagues (1973) demonstrated that after the development of alcoholism previously normal MMPI’s are distorted into the very patterns thought typical of alcoholism. In an undocumented editorial on alcoholism for the *Annals of Internal Medicine*, Enoch Gordis wrote: “Changes in personality or mood are now recognized to be largely the consequence of alcoholism, not its cause” (1976, p. 823). Obviously, if these different viewpoints are to be reconciled, prospective studies of premorbidly well-defined populations are needed.

Jellinek warns us: “the idea that presents itself to an omnivorous reader of the alcohol literature is usually that alcoholism is either an economic, a psychological, a physiological or a sociological problem to the exclusion of other aspects” (1960, p. 13). What is needed is not an argument that one or another factor is the most important cause of the development of alcoholism, but rather an effort to understand the relative etiological contributions of each variable to the total clinical picture. Only a longitudinal design allows both an individual’s alcoholism and the relevant premorbid variables to be conceptualized as independent continua.

But which premorbid variables are relevant? An important strength of the long-term prospective nature of the Study of Adult Development is that it enables us to distinguish premorbid variables (such as ethnicity, strengths or