A Research Framework for Evaluating the Promotion of Mental Health and Prevention of Mental Illness

LEON EISENBERG, MD

GIVEN THE RATHER BREATHTAKING SCOPE of this topic, it is evident that no comprehensive review of the literature is either possible or appropriate. Instead, I shall treat selected topics and present what I consider to be paradigmatic examples to provide a "research framework" for prevention. However, I propose to begin by stating some general principles to be kept in mind as we discuss specifics. I have tried to phrase them provocatively in order to highlight the controversy that surrounds them.

Some General Principles

1. Priority setting for health allocation, including those for prevention, is a political process.

As health professionals, we may have our views on the status of the evidence and the logic behind one or another program; the decisions will be made in the political arena. Note that this First Annual Conference on Prevention takes place not long after the peremptory discharge of Joseph Califano, Secretary of Health, Education, and Welfare, whose public pronouncements kept prevention high on the public agenda. According to the press, one factor in the Administration's decision was his "excessive zeal" in pursuing an anti-smoking campaign that threatened important constituencies in the tobaccofarming States. No less curious is the example of the People's Republic of China which, despite remarkable public health accomplishments in eliminating many parastic and infectious diseases, not only failed until very recently to campaign against smoking, but produces more tobacco tonnage than the United States via a highly profitable state monopoly.

Now, according to the New York Times of September 5, 1979, an anti-smoking campign was to begin; however, since its first announcement appeared in one of the less powerful newspapers, the reporter concluded that "there still was opposition to the move." The same concordance of financial interests and personal insistence on the right to health-injurious behavior has hampered other demonstrably effective preventive measures (seat belts, auto safety devices, motorcycle helmets, speed limits, and the like).

Dr. Eisenberg is Maude and Lillian Presley Professor of Social Medicine and Professor of Psychiatry, Department of Social Medicine, Harvard Medical School, 25 Shattuck St., Boston, Mass. 02115. Tearsheet requests to Dr. Eisenberg. The paper is based on his presentation at the First Annual Alcohol, Drug Abuse, and Mental Health Administration Conference on Prevention, held September 12–14, 1979, in Silver Spring, Md.

2. Enthusiasm for prevention makes strange bedfellows.

The aphorism: "An ounce of prevention is worth more than a pound of cure" is so thoroughly embodied in folk wisdom and so obviously on the side of the angels that those who urge caution before embarking on untested programs are likely to be regarded as churlish. Or they are likely to be accused of being part of an American Medical Association "restraint of trade" against prevention since, were disease to disappear, physicians would become redundant. That argument, to be sure, overlooks the likelihood that physicians will find a way to put themselves at the center of the prevention business; more than that, it betrays a childish faith in the possibility of returning to a Garden of Eden free of disease. To the contrary, were all known methods of prevention to be put into practice, the short-term result would almost certainly be an increase of health care expenditures as more of those who now suffer untimely deaths live to old age and to experience the chronic diseases for which we have, at best, palliative methods of care (1).

The dream of a Garden of Eden is an old one in human mythology; the concept of prevention was present in archaic medicine (2). Why, then, the current enthusiasm for so antique a notion? In part, it stems from the extraordinary success of vaccination programs, though it has been a bit tempered by the swine flu misadventure (3). When one considers that the entire investment in the basic and applied research and the field trials that made the poliomyelitis vaccine possible was less than the cost of the illness burden from a single epidemic year of the disease (4), pursuit of the same strategy for other disorders is immensely appealing; whether this strategy is an entirely appropriate model for other health problems is an issue insufficiently addressed (5). In part, the impetus for prevention stems from growing disillusionment with the increasing costs and limited success of medical treatment that employs high technology. Health care consumes more than 9 percent of the GNP, and it requires no economist to recognize that a limit is being approached. The putative economic benefits of prevention are heavily embodied in recent Federal policy statements in Canada (6) and the United States (7).

Those who favor prevention principally on humanitarian grounds and those who favor it chiefly on economic grounds are likely to come into conflict over decisions about programs capable of fostering health but at an expense greater than the cost of treatment. (An example is screening programs in infancy for rare metabolic disorders.) Furthermore, the appeal to future cost savings has its own political hazards when a decision must be made to invest more heavily (and hence tax

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more heavily) now to reap the benefits for future generations. Politicians are elected by today's constituents.

3. The current political support for prevention provides a singular opportunity to its proponents, but it also carries a considerable risk.

What must be avoided at all costs is the issuance of promissory notes that we cannot redeem. The history of American psychiatry provides grim warning that, however well meant, unfounded claims for psychoprophylaxis bring in their wake public disillusionment and subsequent greater resistance to the expenditure of tax monies for later and more cogent programs (8).

4. Measurement by distant outcome places a terrible burden of proof on childhood interventions; they must be powerful indeed to be able to show a clear effect despite the vicissitudes of subsequent life experience (9).

We have overwhelming evidence of the importance of infant nutrition, but the best fed baby will not become a healthy adolescent if it is starved in later childhood. Does that demonstrate that feeding infants is not worthwhile? It should be justification enough if it can be demonstrated that a program improves the quality of life for the children who participate in it. Providing the best care we know how to give now is no guarantee of a trouble-free future. Nothing is.

5. Realistic goals for medicine are modest: the diminution of suffering and the enhancement of the capacity to cope, not the biologically unattainable goal of the total elimination of disease and stress.

Programs for mental hygiene all too often imply the absurd objective of "preventing (all) mental illness." There is no entity "mental illness" to be prevented; there are, rather, a variety of psychiatric disorders, each with causes, mechanisms, and outcomes. The task for mental hygiene is to devise specific methods for preventing particular disorders. Certain of these may yield to public health measures at a given time.

Can all mental illness, any more than all disease, be eliminated in any foreseeable future? What we call disease is a result of a faulty adaptation of the human organism to its environment. When we alter the ecologic equilibrium to counteract disease, we set into action a train of interrelated events whose final steady state we can predict only imperfectly. Antibiotic treatment has been accompanied by the multiplication of resistant micro-organisms; medical salvage of the victims of hereditary disease increases the carrier rates for deleterious genes; prolongation of longevity adds to the burden

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of degenerative diseases and augments population pressures. Reduced mortality from prematurity portends morbidity in the form of brain syndromes among survivors. To the extent that schizophrenia is hereditary, effective treatment, by restoring patients to the community and thus enhancing the likelihood of marriage and procreation, adds to the gene pool for transmissible mental illness. When patients previously hospitalized are kept afloat in the community although psychologically impaired, there is a potential for damage to children reared by disturbed parents.

Yet, no reasonable assessment of the intended good (and unintended evil) effects of medical treatment warrants the conclusion that we should abandon medical progress. It has led to a considerable net gain in human lives and human happiness. These complexities remind us to be chaste in our promises, to be highly specific in the disease targets we choose and the methods we propose for attaining them, and to be tough-minded in the evaluation of outcomes of programs.

6. Poverty remains a decisive determinant of health status both in the developed and developing world.

Kitagawa and Hauser (10), on the basis of an extensive review of the epidemiology of disease in the United States, concluded that "the most important next gain in mortality reduction is to be achieved through improved socioeconomic conditions. . . ." Indeed, despite the increased use of medical services by the poor made possible by the Medicare and Medicaid programs, the gap in morbidity, disability, and mortality between poor and the nonpoor has widened in the past decade (11-13). McKeown has emphasized the extent to which the gains in longevity over the last century and a half are attributable to improvements in general living conditions rather than the new developments in medicine (14). Health services do make a difference (15), but they are ineffectual in the presence of gross environmental pathology (16).

Illness Burden

It is important to assess the illness burden of each category of disease by using indices sensitive to the varying consequences of illness: age-specific mortality (to provide a measure of years of potential life lost), hospital days, outpatient visits, days of disability, costs (direct health outlay plus lost income, and so on), as proposed by Black and Pole (17) and Rice and co-workers (18). Although the public health consequences of mental disorder are formidable even by present methods of reckoning (first in inpatient days, third among the causes of Social Security Assistance disability, sixth in total costs, and so forth), conventions of disease classification lead to an understatement of the total role of behavioral factors in disease. For example, morbidity and mortality from cirrhosis secondary to alcoholism is classified under digestive diseases; the consequences of violence (accidents, homicide, and suicide) are listed separately from mental disorders. Many disease categories represent, in greater or smaller part, the final common expression of behavioral aberrations. Thus, disorders of behavior, defined broadly, merit a high rank on the public health agenda. We may not have adequate means for prevention or treatment of many of these disorders, but the enormity of the burden identifies a major target for basic and applied research if there is to be hope of reducing the burden of illness.

Health Promotion

Theobald Smith early in the century drew attention to host factors in disease. Smith wrote: "To learn that any given microbe which produced a well defined disease in man is harmless to animals, that a disease germ, dangerous to one species, has no effect upon a closely related species, and that a human being may carry dangerous microbes which are held in check by unknown forces within him, are lessons which in themselves had a great influence in making medicine begin to appreciate the enormous complexity of the processes which protect us from disease or which lead to recovery" (19). Smith formulated a "law of disease" which set forth the proposition that the likelihood of disease is directly proportional to the virulence of the provocative agent and inversely proportional to the resistance of the host (20). This principle provides the medical rationale for health promotion; namely, the enhancement of host factors that contribute to resistance to disease, even when the disease agents are not known or beyond control.

Nutrition. Nutrition provides a prototype of the problems in conducting research on health promotion. Epidemiologic studies of infection in infants and children demonstrate the following propositions: malnutrition increases the likelihood that exposure to infection will result in infectious disease; malnutrition impairs the individual's inflammatory and immune reactions to infectious disease; malnourished persons show increased systemic manifestation of diseases which tend to be more limited in those who are well nourished; in communities where malnutrition is prevalent, intercurrent infection and associated complications are markedly more frequent among the malnourished than the better nourished (21).

In particular, gastrointestinal infection, made more likely by malnutrition, in itself increases the nutritional stresses on the host; fever increases caloric requirements at the same time that infection decreases food intake and reduces nutritional absorption. Health may be further worsened by local customs of managing children with infectious diseases. In some preindustrial societies, a diluted carbohydrate gruel (low in calories, proteins and electrolytes) is fed to sick children, and the reduced food intake may lead to profound metabolic and electrolyte disturbances in the infected child (21). Not only is immediate morbidity and mortality greater for malnourished infants and children, but longitudinal studies have demonstrated that the conjoining of chronic malnutrition with disadvantageous family circumstances results in retarded cognitive and social development (22). Although nutrition is defined as health promotion when the target is the reduction of the secondary complications associated with malnutrition, nutrition is a specific preventive for malnutrition defined per se as disease.

Because malnutrition and infection in infancy and childhood are major problems in the developing world and the infection-malnutrition complex impairs brain development, health promotion via nutrition (and sanitation) ranks high on the WHO agenda for the prevention of mental disorders (23).

The evidence for the deleterious effects of malnutrition is so straightforward and the logic of repair by nutritional supplementation is so compelling that nutrition might appear to have little relevance to research in prevention. However, it has been surprisingly difficult to document the efficacy of nutritional supplementation (24). Malnutrition almost inevitably occurs in a web of deleterious social circumstances. Longitudinal studies of retarded development in previously malnourished children indicate that it is the interacting and multiplicative effects of the simultaneous biological and social insults that produce the damage. The social patterns in a deprived community are not readily changed by health interventions that leave the basic political structure and power relationships unaltered. In such communities, food supplements are not necessarily used or are not used as intended. Outcome comparisons suffer from the differences in the avidity with which some families make use of nutritional supplements, thus raising the question of whether the better outcomes are to be attributed to the food per se or to the characteristics of the families enterprising enough to secure it.

In communities with endemic malnutrition, some children and families fare better than others. Prof. Stephen Richardson of the Department of Pediatrics, Albert Einstein College of Medicine, suggested to me (personal communication) that a useful research strategy would be to study successful families in order to identify the behavior patterns which permit coping under adversity so that the parenting skills tried and tested within the culture might be taught to the less effective parents. Grantham-McGregor and her colleagues (25, 26) have shown that a program of stimulating hospitalized children, continued by their parents who were educated by home visitors after the child was discharged, resulted in greater developmental gains for a group of children who had suffered protein-energy malnutrition than did a similar group renourished but not stimulated.

Despite the self-evident proposition that food protects against malnutrition, the developmental consequences of malnutrition in infancy and childhood occur in a larger context of social deprivation. Remediation, to be effective, must be targeted at the entire complex. Fortunately, such extreme degrees of malnutrition are infrequent in this country. Maternal and infant health programs have demonstrated that pregnancy outcomes can be favorably influenced by community-based programs that include social welfare as well as medical components (27).

Social networks. A second area where imaginative interventions and systematic research could yield important dividends might be termed, by analogy, "social malnutrition." Epidemiologic studies have documented increased rates of psychiatric disorder and of general medical morbidity and of mortality among persons who are socially isolated. Let me epitomize the evidence briefly.

Brown and his colleagues (28) have found that having an intimate and confiding (though not necessarily sexual) relationship with a husband or male friend significantly reduces the likelihood of depressive symptoms in women-despite the life stress ordinarily provocative of such symptoms. Miller and Ingham (29), in a survey of subjects sampled from a general practice, found that both psychological and physical symptoms were lower in women who reported having confidants and friends than among those who reported having few or none. In a study of a random sample of the general population of Canberra, Henderson and co-workers (30) found that the closer the social bonds reported by the respondent, the lower the score for symptoms on the General Health Questionnaire (GHQ). When the level of adversity (as measured by life events) was held constant, there was a statistically significant decline in GHQ scores with increasing levels of social bonds.

Most striking of all are the findings of Berkman and Symes (31) who conducted a 9-year mortality followup study on more than 4,700 adults between the ages of 30 and 69 years in Alameda County, Calif. At initial enrollment, data were collected on the subjects' selfreported health status, socioeconomic condition, health practices, and use of health services. In addition, study subjects were asked in detail about four sources of social contact: marriage, contacts with close friends and relatives, church membership, and informal and formal group associations. Age- and sex-specific mortality rates over the 9 years revealed a significantly lower mortality for subjects reporting each of these social ties. Relative mortality risks for those with the fewest social contacts, when contrasted with those with the most social contacts, were 2.3 for men and 2.8 for women! The association between social disconnectedness and mortality persisted after taking into account initial health status, social class, health practices, and utilization of health services, although each factor in turn influenced mortality.

A composite social network index correlated negatively not only with overall mortality but also with four separate causes of death: ischemic heart disease, cancer, and cerebrovascular and circulatory diseases. Because the Alameda County study employed mortality statistics, it is not possible to be certain whether social isolation leads to higher morbidity in the affected population (that is, a higher attack rate for disease) or to shorter survival time among those afflicted. Data internal to the study make it highly unlikely that the social isolation resulted from the presence of disease; first, the baseline health data argue against this; second, the deaths among the most isolated were not aggregated in the first few years of surveillance, as would have been expected if disability caused by disease decreased social connectedness. Although the intervening pathophysiological mechanisms have yet to be identified, the strength of the association between relative social isolation, on the one hand, and mortality, on the other, in these and other epidemiologic studies suggests a major opportunity for health promotion by community based activities designed to enhance social connectedness. It is, of course, not sufficient to assert this proposition; what is called for is a prospective controlled trial. Perhaps the most fruitful avenue for further exploration lies in efforts to mitigate the acute isolation resulting from bereavement.

Mitigating the effects of acute loss. Bereavement is a readily identifiable stress that has been shown to have negative physical and psychological consequences. Twenty years ago, Kraus and Lilienfeld (32) found agespecific mortality ratios for widowed males and females to be two- to fourfold higher than for those who were married, with the most marked differentials present in the younger age groups. In a British study (33) of 5,000 widowers ages 55 and older, there was a 40 percent excess of mortality compared to expectation in the 6 months following the death of the wife. In addition to these data on mortality from all causes, McMahon and Pugh (34) found suicide rates to be $2\frac{1}{2}$ times higher among the widowed population in the 6 months after bereavement. With the exception of a study by Clayton (35), whose negative findings remain unexplained, Klerman and Izen (36), in a comprehensive review of the medical literature, report general agreement on the deleterious health consequences of bereavement.

A number of hypotheses can be invoked to explain the association: homogamy, joint sharing of an unfavorable environment ,and psychophysiological effects of bereavement (37). Homogamy (the unfit marrying the unfit) is difficult to sustain as an explanation because most causes of death associated with bereavement are not likely to have been present at courtship and marriage. It is more difficult to rule out common exposure to an unsuitable environment, since the studies thus far available have not separated social class and social stress factors from physical factors. As to the third hypothesis, there is evidence for major neuroendocrine effects of loss (38). In addition, depressed affect may lead to an unfavorable change in health practices, resulting from self-neglect and from the absence of the care that had been provided by the spouse. Nonetheless, although the underlying mechanisms remain obscure, bereavement results in significant morbidity among the survivors.

Given the public nature of death in the family and the strong evidence of significant health consequences for the survivor, the design and testing of a public health intervention should be feasible. Although there have been a number of clinical and self-help programs (widow-to-widow counseling, for example) we lack a systematic controlled trial in which households experiencing the recent death of an adult have been assigned randomly to experimental and control conditions. It would be necessary to control for sex, age, and social class. Outcome indices should be for not less than 2 years to permit differences to become manifest, if they are indeed present.

The potential benefit would be the greatest in surviving spouses more than 60 years old who might also be facing other stresses from aging, impending retirement, and attenuation of family bonds—circumstances in which bereavement would constitute a major threat to coping capacity. The design for such a study should take into account the likelihood of a significant number of service refusers, whose outcomes must also be compared to the treatment and control groups. Access to such a population might be through a cooperating group of primary care physicians whose clinical records, appropriately protected for confidentiality, could provide outcome data on the group of refusers to be matched against those for experimental and control groups.

Schooling. Although American society has been characterized since its founding by an abiding faith in the ability of public education to improve the social and cognitive abilities of its citizens (39), today's conventional wisdom holds that schooling makes little difference in academic achievement or personal behavior. The massive Coleman study (40) has been interpreted as demonstrating few effects attributable to schooling in contrast to the major impact of home circumstance on attainment. Jencks and co-workers (41), in reanalyzing the Coleman data, stressed the importance of social class and cast doubt on the ability of public education to offset social inequality.

Rutter and his colleagues (42) at the Institute of Psychiatry in London have reopened the debate by employing a study design that is longitudinal, rather than crosssectional, so that change over time can be measured, and by using instruments sufficiently fine-grained and appropriate to school-related goals so that behavioral effects attributable to differences between schools as well as differences between pupils can be discerned. The investigators identified 12 comprehensive schools in London whose intake was drawn from a known population of students. The pupils who were to enter those schools were assessed at age 10 before entry, again 4 years later while attending, and finally at age 16, the minimal school-leaving age in Britain. Because the pupils had been assessed before they were enrolled, allowance could be made for the characteristics the children brought with them to the several schools in order to differentiate those aspects of their later performance that could be attributed to the school experience.

The results are striking. The average level of academic achievement of children in some schools was much better than that in others. Attendance at certain schools was associated with a marked reduction in the frequency of behavior problems as well as with academic gains; other schools generated a relative surplus of behavior problems and less academic gain. The effect of social class on achievement was not obliterated by the schools' effects. Class differences remained; the successful schools enhanced achievement for both middle and lower class children. In the better schools, both the middle and the lower class children performed at a higher level and displayed fewer behavior problems.

The outcomes measured included academic attainment, attendance, in-school behavior, and delinquency. The independent variables of the schools studied included academic emphasis, continuity, teacher activity, reward and punishment, and opportunity for student participation.

A school's academic emphasis (such matters as assigning homework and making sure it was completed, the care given to planning courses, the supervision of teachers by department heads and the like) demonstrated a strong positive relationship with attendance, as did high staff morale. The actual time spent teaching (as opposed to disciplinary or administrative activities) was significantly related to both attendance and attainment. Firm discipline, but without the use of corporal punishment, and liberal reward by encouragement characterized successful schools. Students' participation in planning and their feeling they could approach a teacher were strongly related to positive academic outcomes. An index based on combining the independent school-based variables showed a 0.8 correlation with behavioral outcomes. Clearly, schools do make a difference!

This demonstration of the powerful impact of schools on pupil behavior has important implications for mental health promotion via the upgrading of schooling. The decisive "controlled clinical trial" remains to be done: is it possible to intervene in the less successful schools to incorporate the desired school characteristics and to demonstrate that these lead to better pupil outcomes? Such interventions, with the potential for increasing students' attainment (which is itself highly correlated with later occupational success) and for diminishing delinquent behaviors, may be fruitful enterprises for research in prevention.

Family planning: contraception and abortion. The new developments in contraceptive technology, which have made available means that are effective, acceptable, and relatively safe, have resulted in a significant and steady decline in the U.S. birth rate. Nonetheless, Tietze (43) estimated that there have been more than 2 million unintended pregnancies in the United States per year since the early 1970s, a finding that correlates with the rise in abortions to the level of about 1.3 million in 1978. In recent years, concern about the hazards of oral contraception has increased. If this concern led all married users of the pill under age 40 to change to the diaphragm or other available measures, the additional contraceptive failures would lead to a demand for a million more abortions (44, 45) than there were in 1977.

Unwanted children and their parents face serious psychological and social hazards. Studies of children born to mothers who applied for abortion and were refused have been carried out in Sweden (46) and in Czechoslovakia (47). Psychological profiles and educational accomplishments of these unwanted children were compared with those of controls. In both studies, although many families coped, the children as a group were at greater risk for developmental failure. Unwanted children are more often the target of child abuse (48). All too many are abandoned to foster care and to State institutions and suffer a heavy toll of psychiatric disorder (49). Moreover, later-born children in large sibships, particularly when the interbirth interval is short, show larger decrements on tests of cognitive attainment (50).

Earlier menarche, earlier sexual experience, and inconsistent contraceptive practices have resulted in increasing pregnancy rates among unmarried teenagers. Between 1971 and 1976, the prevalence of premarital intercourse among never-married women in the United States aged 15 to 19 years increased by 30 percent, so that by 1976 the rates for 15-year-olds were 18 percent, for 17-year-olds 40 percent, and for 19-years-olds 55 percent. Yet one-third of sexually active teenagers used no contraception, and another half used it inconsistently (51). Despite this overwhelming evidence of a major health problem, most school jurisdictions in the United States provide no sex education; the few that do, do so in a cursory fashion.

Maternal mortality from pregnancy and childbirth is, depending upon the age of the mother, from two to eight times higher than that from legal abortion (52). Moreover, delays in abortion stemming from restrictions on access by recent legislation guarantee additional deaths, since there is a 50-fold greater maternal mortality from abortion after the 16th week than before the 9th week of pregnancy (53). The death toll from unwanted pregnancies carried to term will be highest among teenagers because of the higher rates of toxemia, abnormalities of labor, postpartum infection, and hemorrhage among this age group.

Maternal immaturity is associated with a much higher rate of low birth weight infants (54). Low birth weight is associated not only with a much higher perinatal mortality but also with a disproportionate number of developmentally impaired children (55). The impact of motherhood on the teenager can be equally devastating. Furstenberg has documented the marital instability, school disruption, economic problems, and difficulties in regulating family size and in child rearing among adolescent mothers when they were compared with classmates who did not become pregnant premaritally (56).

What of the hazards of abortion itself? As noted, maternal mortality rates are higher at all ages from pregnancy and childbirth than from legal abortion. Furthermore, in a careful analysis of the subsequent pregnancy experience of women undergoing abortion, Daling and Emanuel have demonstrated no adverse effects on pregnancy outcome in comparison with a matched control group without abortions (57). In addition, Harlap and co-workers (58), in a study of 32,000 women followed from their first prenatal visit, concluded that "there is little or no risk of spontaneous abortions after induced abortions when performed by current techniques." The contention that abortion is psychiatrically dangerous is refuted by Brewer's data showing that post-abortion psychosis is less than one-fifth as common as postpartum psychosis (59). Moreover, in a study of women who underwent first trimester abortions, Greer and co-workers (60) found their psychosocial adjustment to be considerably better 18 months after abortion than it was before the procedure.

The evidence is clear: reproductive freedom is not only a human right, it is an essential component of human health. It must be a central feature of a responsive program for preventing mental disorder to provide young Americans with knowledge about human sexuality, to make available family planning services including contraception and abortion, and to assure the pregnant mother and her infant the obstetrical, nutritional, and social services necessary for good health.

Specific Protection

The current possibilities for, and actual accomplishments in, specific protection against psychiatric disorder are far greater than are recognized within the medical community Unless one restricts the term "psychiatric" to mean "psychogenic disorders of obscure etiology," a wholly unwarranted though all too common practice, it can be demonstrated that distribution, transmission, and cause have been pinpointed for a number of major psychiatric disorders with the result that their incidence and prevalence have been markedly reduced. Is the dementia associated with general paresis no longer "psychiatric" because it has a known cause, pathogenesis, treatment, and prevention?

Progress in recent years has been greatest in developing methods to protect the integrity of the central nervous system against insult through genetic counseling, amniocentesis, and selective abortion, improved delivery of obstetrical care during pregnancy, metabolic screening for newborn infants, immunization of infants and children against infectious diseases, and measures to diminish childhood accidents and poisonings. Although many of these procedures are identified with the fields of obstetrics, pediatrics, preventive medicine, and public health, they are not any less appropriate as measures of preventive psychiatry, nor does it diminish the responsibility of the mental health disciplines for insuring that they are incorporated into a national program of health care.

What is intolerable is the extent to which methods known to be efficacious have not yet been made fully accessible to our population. For example, some 40 percent of American children 1 to 4 years of age in 1976 were incompletely immunized against measles, rubella, diphtheria, tetanus, pertussis, poliomyelitis, and mumps (61), each of which can have major central nervous system effects as well as effects on general development. The discussion that follows will not cover each of these topics, nor consider those selected in equal detail, but it is intended to survey representative developments.

Amniocentesis and abortion. Recent developments in techniques of amniocentesis and cell culture, together with safe methods for therapeutic abortion, permit specific preventive measures against some forms of severe mental subnormality that have a prevalence (among 10year-old children in the population) of approximately 4 to 5 cases per 10,000 (61). Of cases of severe subnormality, about one-third are caused by Down's syndrome, a disorder resulting from more than two copies of chromosome 21 in somatic cell nuclei. Ninety-five percent of the cases of Down's syndrome arise from nondisjunction during meiosis, which leads to trisomy of chromosome 21 and a total of 47 chromosomes; the remaining 5 percent of cases are characterized by translocation or mosaicism. In translocation, part of chromosome 21 is translated onto another chromosome (usually 15) so that, although the total number of chromosomes is 46, there is extra chromosomal material from the translocated part of 21 in addition to the two normal 21s. In mosaicism, nondisjunction occurs at an embryologic stage later than fertilization; thus, some cells have a normal chromosome complement and others have trisomy 21.

The likelihood of recurrence (that is, of having a second child with Down's syndrome) is greater when the index case results from translocation; in such families, one percent carries a chromosome with a translocation but has a normal phenotype because of a total chromosome number of 45 (that is, one chromosome 21 is missing). Although the theoretical risk for recurrence is one in three, the observed frequency is somewhat lower. For such families, genetic counseling can be of great assistance; as a public health measure, it would, however, have no detectable effect on the prevalence of Down's syndrome because of the relative rarity of translocation.

Although the incidence of Down's syndrome is declining as the result of changing birth patterns and the use of selective abortion, its prevalence is increasing. This paradox arises because improved care (antibiotics against infection, surgery to correct life-threatening congenital anomalies) has increased the longevity of such children at a rate that more than offsets the decline in incidence.

Trisomic Down's syndrome occurs far more often among infants born to older than younger mothers. For example, the overall risk in New York City has been about 1 in 900 births but rises to 1 in 200 mothers aged 35 to 39, and 1 in 50 to mothers 40 and older. Mothers over 40 gave birth to only 1.5 percent of all infants, but they accounted for 16 percent of the Down's infants (62). Although the strong association with maternal age had been taken to implicate the mother as the source of the extra chromosome, the development of staining methods that permit the identification of unique features of chromosomes in each individual has revealed that, in about a quarter of the cases studied, the extra chromosome had come from the father (63). This finding should help offset the guilt experienced by many mothers who blame themselves for the child's abnormality. The special chromosome staining technique is of research interest but has no role in pediatric counseling, since pointing the finger of blame at either parent is counterproductive; rather, the new scientific finding enables the pediatrician to tell the parents honestly that the error could have arisen in the germ cells of either parent.

Because trisomy occurs at fertilization, there is no way to identify the mother or father at specific risk, since each has the normal karyotype. However, the affected fetus can be detected before birth by amniocentesis and cell culture of the fluid obtained at about the 15th week of pregnancy. This technique makes possible the alternative of aborting such a fetus, thus obviating the tragedy of severe mental handicap for the family. It has been calculated that, if all mothers 35 or older were screened by amniocentesis, about one-third of the cases of Down's syndrome would be prevented; if screening were restricted (because of limited capacity) to mothers 40 and over, about one-sixth could be prevented.

It should be noted that these projections are estimates based upon the rates of childbearing in relation to maternal age derived from public health data a decade ago. In more recent years, as the result of the wider availability and the more consistent use of contraception, the birth rate among women over 35 years has been declining more rapidly than that among younger women (64). In consequence, a greater proportion of the children with Down's syndrome now are born to younger mothers, even though the incidence among this age group remains the same; it is simply that young mothers contribute a larger proportion of all births. For example, from 1960 to 1976, the percentage of births among Massachusetts mothers under 35 years rose from 87.7 to 94.8 percent of the total births during a time span when all births declined by 43 percent. Thus, the effectiveness of an amniocentesis and abortion program for mothers 35 years and older to prevent Down's syndrome has declined from the projected 1 in 3 to about 1 in 5, still a considerable savings. Laboratory facilities for amniocentesis remain far short of the total that would be required to make this a routine procedure for all pregnancies. However, even were there not a limitation in facilities, routine amniocentesis for all pregnancies to prevent Down's syndrome would not be medically justified at the current level of technology. The best figures indicate a risk from the procedure of about 3 in 1,000 (65) which is 2 to 3 times greater than the risk for the disease itself.

Prenatal diagnosis by amniocentesis has been extended to a number of other severely handicapping conditions. One of the most recent-and still controversial-applications has been to the prevention of neural tube defects (anencephaly and spina bifida), which have an annual incidence in the United States of 8.8 per 10,000 births (66). Earlier work with pregnant women known to be at higher risk (because of the previous birth of a defective fetus) revealed that, when the neural tube defect is open, there is a significant increase in alpha-fetoprotein in the amniotic fluid. Subsequent investigations established that elevated levels of alpha-fetoprotein could be detected in maternal serum, and this finding is the basis for a routine screening procedure. Amniocentesis was done selectively whenever the serum level was more than two times the median value for the population (67).

There has now been substantial experience in the United Kingdom with this screening procedure and considerable success in avoiding the birth of abnormal infants. Although some have expressed concern that there is a disproportionate risk of fetal wastage when amniocentesis is carried out following the determination of elevated alpha-fetoprotein serum levels (68), the most recent data (69) indicate that the risk from the procedure is far outweighed by its benefits. Not only does selective abortion prevent the multiple severe handicaps (paralysis, incontinence, mental deficiency) found in the majority of infants with neural tube defects (70), but it removes a source of great distress for the family members responsible for the care of such children. In one study, the observed divorce rate among families with a surviving spina bifida child was nine times the rate among a comparison population (71).

Metabolic screening of newborns. Substantial progress has been made on another front: screening newborns to identify early and treat the metabolic abnormalities whose effects on the brain can be minimized by prompt correction of the biochemical defect. These disorders include phenylketonuria (PKU), galactosemia, and congenital hypothyroidism. On the basis of data from screening of newborns, PKU is found in approximately 1 in 11,500 live births (72), galactosemia in 1 in 40,000 to 60,000 (73), and congenital hypothyroidism in about 1 in 3,500 if all subtypes are summed (74). If untreated, each of these disorders will result in a severely defective child, handicapped by seizures, severe behavior disorder, and other manifestations of central nervous system damage. Yet, with prompt, effective treatment (a phenylalanine-low diet, a lactose-free diet, and extrinsic thyroxin, respectively) the child at risk can be enabled to develop in a normal or nearly normal fashion.

Whether one labels metabolic screening plus dietary management primary or secondary prevention is, to some extent, arbitrary. That is, the newborn with galactosemia does have an inherited metabolic disorder that we are unable to reverse at the molecular level. However, although the genotype and the cellular phenotype remain unchanged, a lactose-free diet fully obviates all the deleterious consequences of the metabolic error, and normal development ensues. Clinical disease never becomes manifest and, in this sense, dietary management can be considered primary prevention.

Prenatal and neonatal screening do have costs that must be assessed. Not only is the yield from tests for the extremely rare conditions small in relation to the investment, but the number of false positives will be disproportionately large. At the least, a false positive creates unwarranted anxiety; at the worst, treatment, which has its own risks, will have been wrongly begun. Thus, laboratory procedures for screening must be associated with a sophisticated diagnostic assessment of each case to make possible a firm biochemical diagnosis. The obverse danger is a false negative: failure to identify a case because of the insensitivity of the test, laboratory failure, or the use of the test at a suboptimal time in the infant's life. Further, it makes little sense to institute screening without an array of followup services, diagnostic facilities, and treatment programs (75). Despite excellent technical performance, a screening program can fall far short of its goal because of lack of funds and facilities or public education, case retrieval, continuing medical care, and counseling.

Legislation to mandate screening is passed with laudable intent; yet the law may err by specifying the diseases rather than establishing an expert commission that can alter the protocol from time to time according to the best evidence for expanding (or narrowing) the spectrum of diseases to be screened.

More than 150 known metabolic disorders are associated with defects in central nervous system function (76). For only a few do we have effective methods of intervention, but that number is certain to increase with scientific progress. In addition, the capability of detecting parents who are at increased risk (for Tay-Sachs disease, for example) is rapidly developing. When both parents are carriers, examination of the amniotic fluid or culturing fetal cells, or both, permit the identification and selective abortion of the homozygous fetus. The accomplishment is not only negative; if the defective fetus can be identified, so also can the normal one. Parents at risk of a recessive genetic disorder who might wish to have children but fear that they will be defective can be enabled to bear their own children by the assurance offered through amniocentesis that a particular fetus is normal (or a carrier). Although such assurance may rarely be fully certain, the probabilities are dramatically altered by the new methods.

The very success of newborn screening programs in identifying patients with phenylketonuria and the effectiveness of dietary management begun early and maintained carefully (77, 78) has resulted in a second generation of problems. How long must dietary management be maintained? The issue still has not been fully resolved, but information available suggests maintenance of dietary restriction as long as the family can adhere to it (79). The problem does not stop there. There is reason for concern about pregnancies of the female with treated PKU. Mothers with untreated PKU, whether they are of normal intelligence or not, appear to be at risk of giving birth to an infant with major defects, including microcephaly and cardiac malformations. No accurate statistics on the risk to mothers with PKU are available because, in most reported cases, the mother's disorder has been ascertained as the result of her having given birth to one or more children with microcephaly (80).

The public health challenge of maintaining low blood levels of phenylalanine during the pregnancy of mothers with PKU is daunting. Because of the critical nature of embryologic developmental events in the first 6 weeks of gestation, it would be logical to start a phenylalaninerestricted diet before conception. This feat has been accomplished with the birth of an apparently normal child (81). This approach is obviously not feasible for the mother with unrecognized PKU, a condition estimated to occur at a rate of 1: 80,000 in the general population (82). This rate might justify antenatal screening for maternal PKU or at least a screening program for all mothers with a family history, those of low intelligence of no known origin, and those with infants with microcephaly (83). Even such screening, of course, can lead to protection of the fetus only after dietary control is instituted (almost certainly later than the sixth to eighth week) when the damage may already have been done. The PKU mother should, in any event, not breast feed her infant since her milk will contain an abnormally high concentration of phenylalanine (84).

The recent recognition that PKU is a heterogeneous group of biochemical disorders creates further complexities. Some 3 percent of these infants are deficient in either dihydropteridine reductase or dihydrobiopterin synthetase rather than phenylalanine hydroxylase. Unrecognized, these disorders will result in progressive neurological damage despite a phenylalanine-low diet (85). It may be possible to treat these patients orally with tetrahydrobiopterin and supplements of neurotransmitter precursors (86). These successes will in turn pose new problems: how long should treatment be maintained? What is the proper management of women during their reproductive life?

Genetic counseling. Genetic counseling for carriers of genetic diseases enables them to be informed of the relative risk of bearing a child with the defect. Current estimates indicate that, of children in a pediatric hospital, 6 to 8 percent suffer from a single gene defect and 20 to 30 percent from genetically influenced disorders (87, 88). In many diseases, it is the interaction between genetic susceptibility and socially determined behavior that leads to clinical manifestations (89). The rationale for (90, 91) and the clinical problems associated with genetic counseling have been considered extensively elsewhere (92, 93). A partial list of the genetic diseases with central nervous system effects would include the cerebromacular degenerations, Wilson's disease, Lesch-Nyhan syndrome, maple-syrup disease, histidinemia, homocystinuria, tyrosinosis, Niemann-Pick disease, the glycogen storage diseases, and the mucopoly-saccharidoses.

Given the strong evidence that both schizophrenias and manic depressive disorders are familial and that the risk among first degree relatives of probands is greater than that for the population at large, it is appropriate to consider if genetic counseling has a role in public health control measures for psychosis. Although the likelihood of bearing schizophrenic offspring is approximately 10 percent when one parent is affected and 40 percent when both parents have a history of schizophrenia (in contrast to a 1 percent risk in the general population), only some 4 percent of schizophrenics report having had parents known to be schizophrenic (94). The decision not to have children would have only a trivial effect on the prevalence of schizophrenia in the population, particularly so since only a fraction of the parental generation will have had the first schizophrenic episode before childbearing occurs. On the other hand, there is reason to believe that current medical advances permitting brief hospitalization and community care (without denying the substantial benefit in the quality of life this means for the patients themselves) are increasing the chances for fertility among schizophrenics and thus probably contributing an additional genetic load (95) as well as a stressful environment for their offspring (96).

Nevertheless, genetic counseling may make sense clinically for the family with a schizophrenic member. Parents must weigh the risk of having 1 child in 10 manifest the disease sometime during a lifetime (with no way of anticipating whether the episode would be mild or severe, single or recurrent, episodic or chronic) against the 90 percent likelihood of having children who will lead reasonably normal lives.

Early intervention for children at risk. Does the foreknowledge of risk permit other kinds of preventive intervention? Despite strong evidence for the efficacy of phenothiazines in treating acute episodes of schizophrenia (97), their "prophylactic use" in children in such families is unjustified, because of the hazards of the long-term use of powerful psychotropic agents during the time of maximum development of the central nervous system (98) as well as the impropriety of "treating" nine relatively normal children for every affected child. Far more appropriate are efforts to buffer the child of psychotic parents against the vicissitudes of familial stress. Such measures include the systematic provision of contraceptive services so that uncontrolled family size does not precipitate parental decompensation (99), day nursery and after school programs, and homemakers and social services during episodes of parental incapacitation (100). Although there is no evidence that psychiatric intervention for children in such families will "prevent" the onset of schizophrenia later in their lives, one might expect that sensitive and nonintrusive psychotherapy for children undergoing lives of turmoil would relieve distress and promote healthy personality development. We must await the results of current longitudinal studies of children in high risk families before we can know whether such interventions diminish psychiatric morbidity (101).

Minimizing environmental hazards to the brain. The potential for prevention with respect to mental subnormality includes all public health measures that can help preserve the integrity of the central nervous system: minimizing exposure of pregnant women to noxious substances that are dysgenetic or teratogenetic, controlling exposure to medical radiation during pregnancy, control of environmental chemicals (to prevent both occupational and consumer hazards), and reduction of exposure to medication during pregnancy. Similarly, brain damage through head trauma can be prevented by accident control measures (speed limits, infant and child car safety restraints, air bags, crash helmets for moped and skateboard users, better highway design, and the like).

Primary prevention for mental disorders of aging. The category of mental disorders that we can be certain will increase in the next century is composed of the psychiatric disorders associated with aging. According to the Bureau of the Census, the 24 million Americans over 65 years in 1979 will increase to 30 million by the year 2000 and to 50 million by 2030 (102). The population at risk for senile dementia will be doubled; however,

there is reason to expect that its prevalence will more than double, even with no change in incidence. Data of Gruenberg and Hagnell (103) from Sweden reveal that senile persons lived for more years after the onset of the senile disorder in the decade between 1957 and 1967 than they had a decade earlier. The difference in survivorship is largly attributable to the effectiveness of antibiotics in postponing death from intercurrent infection, particularly from pneumonia.

At the least, the available data emphasize the importance of planning to meet what will be a rapidly growing need for medical and social support services for the senile elderly in the decades to come (104). Whereas, not long ago, the primary site for care was the State mental hospital, the primary inpatient locus is now the nursing home. Nevertheless, even when the State mental hospital population was growing, for every hospital resident over 65, there were 4.5 nonhospitalized people who were at least as disabled in their mental functioning (105). Clearly the key question concerning prevention of this major illness burden is: is there evidence that intervention is efficacious?

There is presently great interest in the molecular structure of the neurofibrillary tangles of Alzheimer's presenile dementia and those of senile dementia (106) and active speculation that "slow" neurotropic viruses may be at work (107). These developments argue for greater support for research on the aging brain, but they provide no grounds for current action.

Far more relevant, if less dramatic, is the clinical and sociological evidence that diminution in self-esteem, decrease in social ties, forced abandonment of responsibility, and loss of familiar surroundings contribute in a major way to the decrease in effective mental function associated with aging in our society (108). An active program to maintain the "engagement" of the elderly and embed them in supportive social networks might make a considerable difference in the preservation of functional capacity. Moreover, mental decline is likely to be more rapid if lack of ready access to medical care allows other, concurrent, treatable medical disorders to persist and worsen, placing further physical and psychological stress on the organism. If the family of the elderly patient in need of extra care is supported in meeting the additional burden, that family will be less likely to press for institutional placement.

There are clinical grounds for arguing that optimal sociomedical care can successfully slow the aging process; what is needed is a controlled clinical trial. With health resources constrained and multiple health constituencies competing for them, the demonstration of health benefit and a diminished need for institutional care will greatly assist the press for community services for the elderly.

One note of caution should be added. It is important that the services offered respect personal preferences. Under the appropriate title of the "Diogenes Syndrome," Clark and his colleagues (109) described a group of 30 patients admitted to a geriatric unit with acute medical illness and extreme self-neglect. All but two had lived alone in extreme domestic squalor despite adequate income and savings (and even wealth in a few instances). Their homes were malodorous and in complete disarray, several were horders of useless objects, most dressed in multiple layers of unclean clothes. They and their homes were considered eyesores by the community. The medical problems which had precipitated admission included congestive heart failure, cerebrovascular disease, bronchopneumonia, and other serious ailments. What is striking is that the I.Q. of this group of patients was in the top quarter of the population for age (mean 115, range 97-134), and there was no gross deviation of personality although the patients were aloof, suspicious, and detached. The authors warn against the use of compulsory powers and argue for "care by consent. If they recover (from their medical ailments), their freedom to decide their future should be respected."

Preventing the evil effects of racism on mental health.

Health professionals should not need further documentation of the destructive effects of the racism prevalent in American society on physical and mental development. The report of the Bureau of the Census (110) entitled "The Social and Economic Status of the Black Population in the United States: An Historical View, 1790-1978" provides a ready summary, although the magnitude of the problem is underestimated because blacks are undernumerated in census counts and the conventions for estimating unemployment rates result in significant under-counts. A colleague and I (111) have elsewhere summarized the child development literature on the interlocked effects of poverty and race on child development. Thomas and Sillen (112) reviewed the relevant psychiatric literature and the National Institute of Mental Health has compiled a bibliography on racism (113).

Although the deleterious effects of racism on American blacks are pervasive and totally unacceptable, and although racism impairs psychological development in the white population (114), I object to the concept that racism is a "mental illness." As Frantz Fanon (115) emphasized: "The habit of considering racism as a mental quirk, as a psychological flaw, must be abandoned." Rather, as Comer points out: "A given society may promote and reward racism to enable members of the group in control to obtain a sense of personal adequacy and security at the expense of the group with less control" (116). To define racism as a mental illness implies a model of "treatment" by care and understanding just as one would want care and understanding for a psychotic or neurotic patient.

Psychodynamic theories of racism place its causes in the psychological sphere (rather than in the economic, social, and legal forces that maintain it). They thus divert attention from the economic, social, and legal remedies that alone will create the conditions under which the secondary psychological rationalizations for racism can be combated. For example, although mental health professionals can help school personnel to insure a more constructive outcome when schools are desegregated, sensitivity training sessions in the absence of social action to end residential and school segregation is not only ineffective but misdirects social action. We health professionals, predominantly white, by our failure to develop affirmative action programs to facilitate the entry of blacks into the professions, not only indict ourselves as racist but contribute to the perpetuation of the evil we profess to recognize in others.

Thus, because racism is a prime factor in generating excess rates of physical and mental ill health, combating racism must top the agenda of health promotion and disease prevention. Ten years ago, the National Advisory Commission on Civil Disorders (117) listed what needed to be done about jobs, housing, schooling, and health to begin to remedy the social injustices of a divided society. The Commission noted: "What white Americans have never fully understood-but what the Negro can never forget-is that white society is deeply implicated in the ghetto, white institutions created it, white institutions maintain it, and white society condones it" (page 2) ... "This nation will deserve neither safety [from riots] nor progress unless it can demonstrate the wisdom and the will to undertake decisive action against the root causes of racial disorder" (page 34). It is a bitter commentary that, 10 years later, the same issues are even more pressing. Many of the topics I have discussed in this paper (nutrition, schooling, contraception, and abortion) and specific protection (obstetrical care, vaccination, accident and poisoning prevention, genetic counseling, support for the aged) have particular applicability to the unmet needs of the black (and Hispanic) populations.

Secondary Prevention

My assignment was restricted to primary prevention. Nonetheless, to place primary prevention in perspective, a few words on secondary and tertiary levels of prevention are necessary. For, with respect to psychotic disorders in particular, contemporary psychiatry has had its greatest success at these levels. There is by now strong evidence that appropriate treatment with psychotropic drugs at the outset of acute psychosis can markedly influence its duration and outcome (118, 119). The almost simultaneous development of two thrusts in contemporary treatment—the rediscovery of "moral treatment" under the label of social psychiatry and the development of powerful new drugs—has resulted in a marked foreshortening of acute episodes of psychosis and in greater likelihood for the resumption of a productive life (120). The chronicity associated with hospitalization has been recognized as more a function of the prolonged hospitalization and the attendant social breakdown syndrome than of the illness itself (121).

Further, there is strong evidence that drug therapy alone is insufficient without concurrent psychotherapy and social support. Weissman and colleagues (122) have demonstrated that, though antidepressant drugs are the agents of choice for symptom relief, depressed patients receiving simultaneous psychotherapy attain a higher level of social adjustment than a comparison group on drugs alone. Lithⁱum has been shown to be remarkably effective in the prophylaxis of recurrent episodes of mania or depression in bipolar affective disorders (123) and maintenance tricyclic therapy equally effective for preventing recurrence of depression in unipolar disorders (124).

Although a maintenance regimen of phenothiazines diminishes the likelihood of recurrence of schizophrenia in patients returning to families characterized by high emotional intensity, these drugs confer no additional benefit on patients returning to more tranquil families or those sheltered from family interaction by day care programs that diminish face-to-face contact (125). As a further indication of the important influence of social and cultural factors on the course and outcome of psychosis, the WHO nine-nation study has demonstrated that patients from "less developed" countries have a considerably better prognosis than those in the "developed" world (126). This observation stresses the importance of continuing to attend to the psychosocial environment rather than being dazzled by the illusory promise of combating illness by technical means alone (127).

The Biometrics Branch of the National Institute of Mental Health has analyzed office visit reports indicating that the major suppliers of mental health services for those patients whom primary care physicians recognize as having mental health problems are the primary care physicians themselves (128). Surveys of nonpsychiatrist physicians indicate that they are dissatisfied with their training and competence in managing psychiatric problems. Further, clinical experience demonstrates that

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depressive episodes, in particular, are all too often not recognized as such by physicians when the patients express primarily somatic complaints. Thus, a major opportunity for improving diagnosis and early treatment of mental health disorders lies in more effective undergraduate and graduate training of nonpsychiatrist physicians.

Tertiary Prevention

The resident State mental hospital population, which had been steadily increasing year by year through 1955 has declined dramatically by two-thirds in the last two decades. Over the same period, inpatient care episodes per 100,000 population have increased slightly from 795 to 847 (129). The locus of care for acute episodes has shifted significantly toward general hospital psychiatric units and V.A. hospitals. Inpatient care of the chronically ill continues to be given primarily in the public mental hospitals and nursing homes (129).

The one clear gain has been the shortened mental hospitalization, but this improvement has been somewhat offset by higher readmission rates and by the numbers of chronically mentally ill who live in welfare hotels and inhumane nursing homes under circumstances no better and probably worse than those of the much criticized State hospitals.

A system of accountability for the health status of the chronic patients within each specified population is much needed. The best contemporary care for acute psychotic episodes leaves a residual group of chronic patients whose illness defies complete resolution. A chronic schizophrenic is no less ill, no less in need of care, and no less "psychiatric" if he wastes away in a nursing home, lives in quiet misery in a single room, or sleeps in subways and is not tabulated in any count of "patients." Yet health authorities too often find it convenient to publicize the reduction in numbers of registered hospital patients as a measure of progress when the patient is discharged from the rolls without having improved.

If we are to be able to assess the impact of new methods of care and rehabilitation, we must track systematically the clinical course of chronic patients with periodic measures of their functional capacity and residual symptoms—from the time that they enter care, whatever the administrative category of the primary social or health agency, the labels used, or the place of residence.

Reflections on Public Policy Implications

Throughout this review, we have been confronted by the pervasive influence of socioeconomic factors on health status. Being poor, black, employed in certain industries, unemployed, or elderly and dependent, to name a few, is to be subject, not to unique diseases, but to increased rates of incidence and prevalence for most diseases and to higher mortality for many. At the beginning I noted the argument that improved living conditions over the past century and a half had had more to do with the reduction of morbidity and mortality than improvements in medical care (14). Some (130) carry the argument further to the doctrine of "medical nemesis:" technological medicine worsens health and further diminishes personal self-reliance. This doctrine may or may not make good theology, but it fails utterly to correspond with the realities of the social and health circumstances of the disadvantaged (131, 132).

Although all do not agree, let us assume for the sake of argument that unemployment results in increased rates of mental disorder (133). What are the policy implications for a mental health agency?

The agency does not have the hiring capacity to make a substantial direct impact on unemployment (although social programs have been used for job distribution and they do keep mental health professionals off the welfare roles, an element of professional self-service not irrelevant to public debate). Individual mental health practitioners can try to call to public attention the destructive effects of joblessness on personal identity, competence, and motivation (133, 134). This action has not been a notably effective strategy, although I continue to employ it. I suspect that this is so, not merely because mental health professionals are not charismatic orators, but because the health argument is largely irrelevant to the terms of public debate.

Few contend that unemployment is good for health; most agree that high levels of unemployment are undesirable, even though some conservative economists insist that some minimum percentage of unemployment is an essential structural feature of an "efficient" economy. Thus, the real debate is about what ought to be done to minimize unemployment and at what expense to whom.

A mental health agency staff that worked at reforming the economy would have little prospect for success, at the cost of what they might have achieved by applying resources and expertise to those health measures that have been shown to be efficacious under existing social conditions. The family that has been spared the birth of an infant with Tay-Sachs disease, the mother who, having been protected against rubella, has been able to deliver a normal infant, the schizophrenic man whose acute psychosis has been aborted by phenothiazines, the severely depressed woman whose suffering has been shortened by tricyclics (even though life stress may have precipitated the episode)-all are beneficiaries of health care. Each health professional as a citizen has a moral responsibility to fight for the eradication of social conditions that impair health.

Health agencies have neither been given the public

charge nor do they have the expertise and resources to be social change agents except (and this is a notable exception) within their domain of action; that is, by acting for equal opportunity, rectifying inequities in service delivery, targeting service to populations at risk, and the like. Since health agencies are subordinate to the executive and legislative branches of government, any attempt to make or carry out policy not in keeping with government directives must necessarily result in punitive action against the health agency, with consequent loss of its capacity to improve health within the constraints set by existing socioeconomic conditions.

In the most impoverished nations of the world those conditions reduce health measures to comparative futility. For example, in parts of Africa, mortality rates from measles in the first year of life are as high as 1 in 10 because of endemic malnutrition and infection; the measles vaccine is relatively ineffective in infancy; moreover, the necessary refrigeration of the vaccine is extremely difficult. Thus, measles remains a devastating disease for African infants (135) despite the enormous potential for prophylaxis by vaccine when nutrition and sanitation reach at least minimal standards. It would be a perversion of public health responsibility to ignore these realities.

On the other hand, in the United States, where the worst poverty is above that level of extremity, measles vaccination has markedly reduced morbidity and mortality (136) despite the persistence of social inequality. (It has been estimated that, in the first 10 years of its use in the United States, measles vaccination had obviated 24 million infections, 2,400 deaths, and 8,000 cases of mental subnormality.) What is inexcusable is our national failure to ensure that a comprehensive vaccination program is available to those children from 1 to 4, many of them poor and black, who would benefit from it, despite the other social hazards they face. What is inexcusable is public policy which makes health services such as abortion dependent upon income.

I do not suggest that health workers retire from the struggle against social injustice. To the contrary, it is my conviction that health workers can contribute greatly by working within the health field for equity, quality, and effectiveness in public services—and beyond it by fulfilling our political responsibilities as citizens to strive for a more just society.

References

- Gori, G. B., and Richter, B. J.: Macroeconomics of disease prevention in the United States. Science 200: 1124– 1130 (1979).
- 2. Sigerist, H. E.: A history of medicine: primitive and archaic medicine. Oxford University Press, New York, 1951.

- 3. Neustadt, R. E., and Feinberg, H. V.: The swine flu affair: decision making on a slippery disease. U.S. Department of Health, Education, and Welfare, Washington, D.C., 1978.
- 4. Fudenberg, H. H.: Fiscal returns of biomedical research. J Invest Dermatol 61: 321-329 (1973).
- 5. Eisenberg, L.: The perils of prevention: a cautionary note. N Engl J Med 297: 1320-1232 (1977).
- Lalonde, M.: A new perspective on the health of Canadians. Department of National Health and Welfare, Ottawa, 1974.
- Public Health Service: Forward plan for health FY 1978-82. DHEW Publication No. (OS) 76-50046, U.S. Government Printing Office, Washington, D.C., August 1976.
- Deutsch, A.: The history of mental hygiene. In American psychiatry: 1844–1944, edited by J. K. Hall. Columbia University Press, New York, 1944, pp. 326–365.
- Eisenberg, L.: The human nature of human nature. Science 176: 123-128 (1972).
- Kitagawa, E. M., and Hauser, P. M.: Differential mortality in the United States: a study in socioeconomic epidemiology. Harvard University Press, Cambridge, Mass., 1973.
- Elinson, J.: Have we narrowed the gaps in health care between the poor and nonpoor? Med Care 15: 675-677 (1977).
- 12. Lerner, M., and Stutz, R. N.: Narrowing the gaps 1959–61 to 1969–71, mortality. Med Care 15: 620–635 (1977).
- 13. Wilson, R. W., and White, F. L.: Changes in morbidity, disability, and utilization differentials between the poor and nonpoor. Med Care 15: 636-646 (1977).
- 14. McKeown, T.: The role of medicine: dream, mirage or nemesis? Nuffield Provincial Trust, London, 1976.
- McDermott, W.: Medicine: the public good and one's own. Perspect Biol Med 21: 167-187 (1978).
- McDermott, W., Dueschle, K. W., and Barnett, C. R.: Health care experiment at Manyfarms. Science 175: 22-31 (1972).
- 17. Black, D. A. K., and Pole, J. D.: Priorities in biomedical research. Brit J Prev Soc Med 29: 222-227 (1975).
- Rice, D. P., Feldman, J. J., and White, K. L.: The current burden of illness in the United States. Institute of Medicine, Washington, D.C., 1976.
- 19. Smith, T.: Quoted in Beecher, H. K., and Altschule, M. D.: Medicine at Harvard: The first 300 years. University Press of New England, Hanover, N.H., 1977.
- 20. Gage, S. H.: Theobald Smith 1859-1934. Cornell Vet 25: 207-228 (1935).
- Birch, H. G., and Cravioto, J.: Infection, nutrition and environment in mental development. *In* The prevention of mental retardation through control of infectious diseases. PHS Publication No. 1692, Washington, D.C., 1966, pp. 227-248.
- 22. Richardson, S. A.: The relation of severe malnutrition in infancy to the intelligence of school children with different life histories. Pediatr Res 10: 57-61 (1976).
- World Health Organization Expert Committee Report: Child mental health and psychosocial development. Technical Report Series 613, World Health Organization, Geneva, 1977.
- 24. Pollit, E., and Thomson, C.: Protein-calorie malnutrition and behavior: a view from psychology. In Nutrition

and the brain, edited by R. Wurtman and J. Wurtman. Raven Press, New York, 1977, vol. 2.

- 25. Grantham-McGregor, S. M., Stewart, M. E., and Desai, P.: A new look at the assessment of mental development in young children recovering from severe malnutrition. Dev Med Child Neurol 20: 773-778 (1978).
- Grantham-McGregor, S., et al.: Effect of stimulation on mental development of malnourished child. Lancet 2: 200-201 (1979).
- Richmond, J. B.: The needs of children. Daedalus 106: 247-260 (1977).
- Brown, G. W., Bhrolchain, M. N., and Harris, T.: Social class and psychiatric disturbance among women in an urban population. Sociology 9: 225-259 (1975).
- 29. Miller, P. McC., and Ingham, J. G.: Friends, confidants and symptoms. Soc Psychiatry 11: 51-58 (1976).
- 30. Henderson, S., et al.: Social bonds, adversity and neurosis. In The social consequences of psychiatric illness, edited by P. J. Clayton, J. K. Wing, and L. N. Robins. Bruner/Mazel, New York, 1980.
- Berkman, L. F., and Symes, S. L.: Social networks, host resistance and mortality: a nine-year follow-up study of Alameda County residents. Am J Epidemiol 109: 186– 204 (1979).
- 32. Kraus, A. S., and Lilienfeld, A. M.: Some epidemiologic aspects of the high mortality rate in the young widowed group. J Chron Dis 10: 207-217 (1959).
- 33. Young, M., Bernard, F., and Wallis, G.: The mortality of widowers. Lancet 2: 454-456 (1963).
- 34. McMahon, B., and Pugh, T. F.: Suicide in the widowed. Am J Epidemiol 81: 23-31 (1965).
- 35. Clayton, P.: Mortality and morbidity in the first year of widowhood. Arch Gen Psychiatry 25: 745-750 (1974).
- 36. Klerman, G. L., and Izen, J. E.: The effects of bereavement and grief on physical health and general well-being. Adv Psychosom Med 9: 63-104 (1977).
- Jacobs, S., and Ostfeld, A.: An epidemiologic review of the mortality of bereavement. Psychosom Med 39: 344– 357 (1977).
- 38. Hamburg, D. A., Hamburg, B. A., and Barchas, J. D.: Anger and depression in the perspective of behavioral biology. In Emotions—their parameters and measurement, edited by L. Levi. Raven Press, New York, 1975, pp. 235-278.
- 39. Bremner, R. H.: Children and youth in America: A documentary history. Harvard University Press, Cambridge, Mass., 1970, pp. 185-261, vol. 1.
- Coleman, J. S., et al.: Equality of educational opportunity. U.S. Government Printing Office, Washington, D.C., 1966.
- 41. Jencks, C., et al.: Inequality: a reassessment of family and schooling in America. Basic Books, New York, 1972.
- 42. Rutter, M., et al.: Fifteen thousand hours: secondary schools and their effects on children. Harvard University Press, Cambridge, Mass., 1979.
- Tietze, C., et al.: Unintended pregnancies in the United States, 1970–1972. Fam Plann Perspect 11: 186–188 (1979).
- 44. Ryder, N. B.: Contraceptive failure in the United States. Fam Plann Perspect 5: 133-142 (1973).
- 45. Jaffe, F. S.: The pill: a perspective for assessing risks and benefits. N Engl J Med 297: 612-614 (1977).
- 46. Forssman, H., and Thuwe, I.: One hundred and twenty children born after application for therapeutic abortion

was refused. Acta Psychiat Scand 42: 71-88 (1966).

- Matejcek, Z., et al.: Children from unwanted pregnancies. Acta Psychiatr Scand 57: 67-90 (1978).
- Lauer, B., et al.: Battered child syndrome: review of 130 patients with controls. Pediatrics 54: 67-70 (1974).
- Eisenberg, L.: The sins of the fathers: urban decay and social pathology. Am J Orthopsychiatry 32: 5-17 (1962).
- 50. Zajonc, R. B.: Family configuration and intelligence. Science 192: 227-236 (1976).
- 51. Zelnik, M., and Kantner, J. F.: Sexual and contraceptive practices of young unmarried women in the United States, 1971 and 1976. Fam Plann Perspect 9: 55-71 (1977).
- 52. Cates, W., and Tietze, C.: Standardized mortality rates associated with legal abortion: United States, 1972-1975. Fam Plann Perspect 10: 109-112 (1978).
- Cates, W., et al.: Legal abortion mortality in the United States: epidemiologic surveillance, 1972–1974. JAMA 237: 452–455 (1977).
- Ventura, S. J.: Teenage childbearing: United States 1966-1975. Monthly Vital Stat Rep 26, No. 5, Supplement 1-15 (1977).
- 55. Hardy, J. B.: Birth weight and subsequent physical and intellectual development. N Engl J Med 289: 973-974 (1973).
- Furstenberg, F. F.: The social consequences of teenage parenthood. Fam Plann Perspect 8: 148-164 (1976).
- 57. Daling, J. R., and Emanuel, I.: Induced abortion and subsequent outcome of pregnancy in a series of American women. N Engl J Med 297: 1241-1245 (1977).
- Harlap, S., et al.: A prospective study of spontaneous fetal losses after induced abortions. N Engl J Med 301: 677-681 (1979).
- 59. Brewer, C.: Incidence of post-abortion psychosis: a prospective study. Br Med J 1: 476-477 (1977).
- Greer, R. H., et al.: Psychosocial consequences of therapeutic abortion: King's therapeutic study III. Br J Psychiatry 128: 74-79 (1976).
- Center for Disease Control: United States immunization survey 1976. DHEW Publication No. (CDC) 78-8821. U.S. Public Health Service, Atlanta, Ga., 1977.
- Stein, Z. S.: Strategies for the prevention of mental retardation. Bull NY Acad Med 51: 130-142 (1975).
- 63. Magenis, R. E., et al.: Parental origin of the extra chromosome in Down's syndrome. Hum Genet 37: 7-16 (1977).
- 64. Holmes, L. B.: Genetic counselling for the older pregnant woman: new data and questions. N Engl J Med 298: 1419-1421 (1978).
- 65. National Registry for Amniocentesis Study Group: Midtrimester amniocentesis for prenatal diagnosis: safety and accuracy. JAMA 236: 1471-1476 (1976).
- 66. Center for Disease Control: Congenital malformations: surveillance report: October 1976-September 1977. U.S. Public Health Service, Atlanta, Ga., March 1978.
- 67. Brock, D. J. H., et al.: Maternal plasma alpha-fetoprotein screening for fetal neural tube defects. Br J Obstet Gynecol 85: 575-581 (1978).
- Bennett, M. J., et al.: Some problems of alpha-fetoprotein screening. Lancet 2: 1296-1297 (1978).
- 69. Brock, D. J. H., and Gosden, C.: Serum alpha-fetoprotein screening and risk of amniocentesis. Lancet 2: 206 (1979).

- Laurence, K. M.: Effect of early surgery for spina bifida cystica on survival and quality of life. Lancet 1: 301-304 (1974).
- 71. Tew, B. J., et al.: Marital stability following the birth of a child with spina bifida. Br J Psychiatry 131: 79-82 (1977).
- 72. National Academy of Sciences, Committee for the Study of Inborn Errors of Metabolism: Genetic screening: programs, principles and research. Washington, D.C., 1975.
- 73. Levy, H. L., and Hammersen, G.: Newborn screening for galactosemia. J Pediatr 92: 871-877 (1978).
- Dussault, J. H., et al.: Modification of a screening program for neonatal hypothyroidism. J Pediatr 92: 274– 277 (1978).
- Grover, R., et al.: Evaluation of the expanded newborn screening program in New York City. Pediatrics 61: 740-749 (1978).
- 76. Brady, R. O.: Inherited metabolic diseases of the nervous system. Science 193: 733-739 (1976).
- Williamson, M. L., Dobson, J. C., and Koch, R.: Collaborative study of children treated for phenylketonuria: study design. Pediatrics 60: 815-821 (1977).
- Dobson, J. C., et al.: Intellectual assessment of 111 fouryear-old children with phenylketonuria. Pediatrics 60: 822-827 (1977).
- Smith, I., et al.: Effects of stopping low-phenylalanine diet on intellectual progress of children with phenylketonuria. Br Med J 2: 723-726 (1978).
- Hansen, H.: Variability of reproductive casualty in maternal phenylalanemia. Early Hum Dev 2: 51-71 (1978).
- 81. Nielsen, K. B., Wamberg E., and Weber, J.: Successful outcome of pregnancy in a phenylektonuric woman after a low phenalanine diet introduced before conception. Lancet 1: 1245 (1979).
- Levy, H. L., et al.: Screening of the normal population in Massachusetts for phenylketonuria. N Engl J Med 282: 1455-1458 (1970).
- Pitt, D., and Gooch, J.: The problem of maternal phenylketonuria. Aust Paediatr J 10: 337-342 (1974).
- Fisch, R. O., et al.: The effect of excess L-phenylalanine on mothers and their breast-fed babies. J Pediatr 71: 176-180 (1967).
- Kaufman, S., et al.: Phenylketonuria due to a deficiency of dihydropteridine reductase. N Engl J Med 293: 785– 790 (1975).
- Niederwieser, A., et al.: Phenylketonuria variants. Lancet 1: 550 (1979).
- Scriver, C. R., et al.: The frequency of genetic disease and congenital malformations among patients in a pediatric hospital. Can Med Assoc J 108: 1111-1115 (1973).
- 88. Childs, B., Miller, M., and Bearn, A. G.: Gene mutation as a cause of human disease. *In* Mutagenic effects of environmental contaminants, edited by H. E. Sutton and M. I. Harris. Academic Press, New York, 1972, pp. 3–14.
- 89. Childs, B.: Prospects for genetic screening. J Pediatr 87: 1125-1132 (1975).
- 90. Murphy, E. A.: The rationale of genetic counseling. J Pediatr 72: 121-130 (1968).
- Motulsky, A. G., and Hecht, F.: Genetic prognosis and counseling. Am J Obstet Gynecol 90: 1227–1241 (1964).
- 92. Leonard, C. O., Chase, G. A., and Childs, B.: Genetic counseling: a consumer's view. N Engl J Med 287: 443-449 (1972).

- Pearn, J. H.: Patients' subjective interpretations of risks offered in genetic counseling. J Med Genet 10: 129-134 (1973).
- 94. Slater, E.: A review of earlier evidence on genetic factors in schizophrenia. In The transmission of schizophrenia, edited by D. Rosenthal and S. S. Kety. Pergamon Press, London, 1968, pp. 15-26.
- 95. Erlenmeyer-Kimling, L., Nicol, S., Rainer, J. D., and Deming, W. E.: Changes in fertility rates of schizophrenia patients in New York State. Am J Psychiatry 125: 916-927 (1969).
- 96. Anthony, E. J.: A risk vulnerability intervention model for children of psychotic parents. In The child in his family. Vol. 3. Children at psychiatric risk, edited by E. J. Anthony and C. Koupernick. John Wiley & Sons, Inc., New York, 1974, pp. 99-121.
- 97. May, P. R. A.: Schizophrenia: evaluation of treatment methods. In Comprehensive textbook of psychiatry II, edited by A. M. Freedman, H. I. Kaplan, and B. J. Sadock. Williams & Wilkins, Baltimore, 1975, vol. 1, pp. 955-981.
- Eisenberg, L.: Principles of drug therapy in child psychiatry. Am J Orthopsychiatry 41: 371-379 (1971).
- 99. Abernathy, V., and Grunebaum, H.: Toward a family planning program in psychiatric hospitals. Am J Public Health 62: 1638-1646 (1972).
- 100. Rice, E. P., Ekdahl, M. C., and Miller, L.: Children of mentally ill parents: problems in child care. Behavioral Publications, New York, 1971.
- 101. Garmezy, N.: Children at risk: the search for the antecedents of schizophrenia. Schizophr Bull 9: 55-125 (1974).
- 102. U.S. Bureau of the Census: Demographic aspects of aging and the older population in the United States. Current Population Reports. Special Studies Series D-23, No. 59. U.S. Government Printing Office, Washington, D.C., 1976.
- 103. Gruenberg, E. M., and Hagnell, O.: The rising prevalence of chronic brain syndrome in the elderly. In Society, stress, and disease: aging and old age, edited by L. Levi and A. R. Kagan. Oxford University Press, Oxford, 1979. In press.
- 104. Gruenberg, E.: The failures of success. Milbank Mem Fund Q 53: 3-24 (1977).
- 105. New York State Department of Mental Hygiene: Report of the Mental Health Research Unit. Psychiatric Q (suppl.) 34: 34-75 (1960).
- 106. Wells, C. E., editor: Dementia. Ed. 2. F. A. Davis Co., Philadelphia, 1977.
- 107. Gajdusek, D. C., and Gibbs, C. J.: Slow virus infections of the nervous system: In The nervous system, edited by D. B. Tower. Raven Press, New York 1975, vol. 2, pp. 113-135.
- 108. Cumming, E., and Henry, W. E.: Growing old: the process of disengagement. Basic Books, New York, 1961.
- 109. Clark, A. N. G., Manikar, G. D., and Gray, I.: Diogenes syndrome: a clinical study of gross neglect in old age. Lancet 1: 366-368 (1975).
- 110. U.S. Bureau of the Census: The social and economic status of the black population in the United States: an historical view, 1790-1978. Current Population Reports, Special Studies, Series P-23, No. 80. U.S. Government Printing Office, Washington, D.C., 1979.
- 111. Eisenberg, L., and Earls, F. J.: Poverty, social deprecia-

tion and child development. In American handbook of psychiatry, edited by D. A. Hamburg. Basic Books, New York, 1975, vol. 6, pp. 275–291.

- 112. Thomas, A., and Sillen, S.: Racism and psychiatry. Brunner/Mazel, New York, 1972.
- National Clearing House for Mental Information: Bibliography on racism. DHEW Publication No. (HSM) 73-9012, U.S. Government Printing Office, Washington, D.C., 1972.
- 114. Eisenberg, L.: Racism, the family, and society: a crisis in values. Ment Hyg 52: 512-520 (1968).
- 115. Fanon, F.: Toward the African revolution. Grove Press, New York, 1967.
- Comer, J.: White racism: its root, form, and function. Am J Psychiatry 126: 802-806 (1969).
- 117. Kerner, O.: Report of the National Advisory Commission on Civil Disorders. E. P. Dutton, New York, 1968.
- 118. Klerman, G. L.: Pharmacotherapy of schizophrenia. Ann Rev Med 25: 199-217 (1974).
- 119. Klerman, G. L.: The affective disorders: depressive illness and manias. Medicine 12: 536-550 (1975).
- 120. Eisenberg, L.: Psychiatric intervention. Sci Am 229: 117-127 (1973).
- 121. Gruenberg, E. M.: The social breakdown syndrome: some origins. Am J Psychiatry 123: 1481-1489 (1967).
- 122. Weissman, M. W., et al.: Treatment effects on the social adjustment of depressed patients. Arch Gen Psychiatry 30: 771-778 (1974).
- 123. Baastrup, P. C., et al.: Prophylactic lithium. Lancet 2: 826-830 (1970).
- 124. Davis, J. M.: Maintenance treatment in psychiatry II: affective disorders. Am J Psychiatry 133: 1-13 (1976).
- 125. Wing, T. K.: The social context of schizophrenia. Am J Psychiatry 135: 1333-1339 (1978).
- 126. Sartorius, N., Jablensky, A., and Shapiro, R.: Two year follow-up of the patients included in the WHO International Pilot Study of Schizophrenia. Psychol Med 7: 529-541 (1977).
- 127. Eisenberg, L.: Interfaces between medicine and psychiatry. Compr Psychiatry 20: 1-14 (1979).
- 128. Regier, D., Goldberg, I. D., and Taube, C. A.: The de facto U.S. mental health services system. Arch Gen Psychiatry 35: 685-693 (1978).
- 129. National Center for Health Statistics, National Center for Health Services Research: Health: United States, 1978. DHEW Publication No. (PHS) 79–1232, Hyattsville, Md., 1979, pp. 69–87, 330–331.
- 130. Illich, I.: Medical nemesis: the expropriation of health. Calder and Boyars, London, 1975.
- 131. Eisenberg, L.: The social imperatives of medical research. Science 198: 1105-1110 (1977).
- Fox, R. C.: The medicalization and demedicalization of American society. Daedalus 106: 9-22 (1977).
- 133. Brenner, H.: Estimating the social costs of national economic policy: implications for mental and physical health and criminal aggression. Joint Economic Committee, Congress of the United States, U.S. Government Printing Office, Washington, D.C., 1976.
- 134. Eisenberg, L.: Equity, quality and constraints. Hosp Comm Psychiatry 29: 781-787 (1978).
- Vaccination against measles [Editorial]. Lancet 2: 132– 134 (1976).
- 136. Bass, J. W., et al.: Booster vaccination with further live attenuated measles vaccine. JAMA 235: 31-34 (1976).