ence over which the lecturer has substantial control. (Just imagine what the impact on lecture preparation would be if other faculty members were present at lectures—let alone if those lectures were to be made available in the library on video tape!) This issue will be faced because it must be. The faculty may be unlikely to face it on their own, but they can be led to face it by administrative influence. Responsible administrators, aware of widespread and often uninformed scrutiny of faculty workloads and productivity, will spur the effort.

The content of university programs will also change. That is, of course, an old story, since such programs have undergone steady evolution. But I see two respects in which the evolution will change character. The revision and modernization of content will continue, but a new emphasis on values, already emerging, will focus increased attention on the moral, esthetic, and social aspects of the traditional programs and disciplines. Moreover, the affective dimensions of student growth and maturation will receive an increasing amount of academic attention as students exercise greater influence in the decision-making forums of their universities. Both of these prospects hold dangers as well as opportunities, for the university is primarily, as Pake says, a context for the development of the intellectual powers of the student (as contrasted with his moral, political, social, or psychological attributes). Yet, just as sensitivity without competence is futile, so competence without sensitivity is empty, or worse. The task before universities, then, is to encompass within the limitations of an academic environment an effective approach to the evaluative and affective concerns of our students and our times.

Concluding Remarks

I cannot bring myself to believe that the problems of our universities are insoluble, partly because I see them as no worse than the problems of social injustice, environmental pollution, poverty, and war. Hence, abandoning the university's quest for well-being because of the complexity of its problems constitutes an affirmation of discouragement and withdrawal that we can ill afford. (I am not arguing that the university has any privileged claim on the efforts of the talented—only that, when they simply abandon it in despair, they provide an unhappy commentary on the solubility of all our complex, large-scale problems.) Yet neither can I imagine that the prospects at present are unmitigatedly bright. That George Pake, as well as some of the few others of such ability, has withdrawn from the battle is alone enough to give one pause.

I end, then, sharing Pake's inability to predict the future of our universities and sympathetic to his view that that future hangs in precarious balance. But the question is not one of survival. Our universities will endure. Rather, what is at issue is the form in which and the vitality with which they will survive. I retain an abiding confidence, based, I believe, on evidence, that what Pake calls "the most significant human institution for the future of free men" will rise—indeed, is rising—to the challenges that confront it and that the American university will survive and ultimately flourish as the lifeblood of our intellectual heritage.

References and Notes

1. G. Pake, *Science* **172**, 908 (1971).
2. 1 Dupont Circle, Washington, D.C., is the address of the national headquarters of the American Council on Education, as well as several other national educational organizations.
3. Specific examples of each of these points are described in the *Chronicle of Higher Education*; almost any sampling of several recent issues will do.

NEWS AND COMMENT

Cooley's Anemia: Special Treatment for Another Ethnic Disease

Last winter, a 30-year-old chemist named Michael Iovene picked up the telephone and called his congressman. Within a matter of days, Iovene and a friend of his named Dorothy Guiliotis met with Representative Robert N. Giaimo (D–Conn.) in his district office in downtown New Haven. Their meeting was on a Saturday morning—the 22nd of January. That day, the process of transforming an obscure ethnic disease known as Cooley's anemia into the target of a national program of research and screening began.

The success of that process has not been met with unbridled enthusiasm in all camps. Indeed, many observers of the health scene see it as the epitome of politicalization of disease. They see the campaign to combat Cooley's anemia as a symbol of all they consider irrational in biomedical research planning. Individuals who supported special legislation for Cooley's, however, call its successful passage "an example of the democratic process at its best."

Iovene and Mrs. Guiliotis talked to Giaimo about this grim, inherited blood disorder named after Thomas B. Cooley, a Detroit physician who described it in 1925, and also known as thalassemia, from the Greek word for "sea." Many of the victims of thalassemia are of Mediterranean ancestry. In the United States, the highest incidence of the disease appears to be among individuals of Italian or Greek or Spanish descent.

The real incidence of the disease in the United States is not known and estimates are little more than guesses. Many researchers agree that a figure ranging somewhere between 5,000 and 15,000 is plausible. Outside of the United States, in certain European and Asian countries, the incidence is much higher.

Michael Iovene and Dorothy Guiliotis each has a special interest in Cooley's anemia. Iovene, a young man of Italian ancestry, is a Ph.D. candidate in chemistry. At 30, he is one of those rare individuals with this lethal blood disease who survive much past the age of 20. People say he has already outlived his time by a decade. He tries not to think about it, but it doesn't do much good.

Mrs. Guiliotis's family roots are in Greece. Her sister died of Cooley's a few years ago. Today, Mrs. Guiliotis is the executive director of the Connecticut chapter of the Cooley's Anemia Blood and Research Foundation for

Children. "Neither Michael nor I had really planned to go to see Congressman Giaimo," she recalls. "But one day we felt we had to do something, and Michael just called him. We really felt we were not prepared for the meeting."

When the two talked with Giaimo, they told him that there is no cure for Cooley's anemia. Its victims, who can be identified during the first few months of life, are unable to produce red blood cells with a sufficient amount of hemoglobin. In addition, the weak red cells they do make live no more than a few weeks. Normal red blood cells have a life span of 3 to 4 months. The only thing physicians can do for Cooley's patients is to give them frequent transfusions, sometimes as often as every 2 to 4 weeks, for their entire lives. The transfusions are painful, particularly after the patients' veins become tender and fragile. The transfusions themselves, which can take 3 or 4 hours or more, are both lifesaving and lethal. Debilitated by tissues that have been deprived of oxygen by hemoglobin-poor red blood cells, Cooley's patients need blood transfusions in order to live. But gradually an excess of iron builds up in their bodies from all that blood, depositing itself in heart, kidney, and other tissues. Eventually, many victims of Cooley's anemia die from what amounts to iron poisoning. Nobody can stop it.

Frequent blood transfusions are not only painful for the patient, they are time-consuming and emotionally rough on the patient's family. Parents of children with Cooley's say it is hell to have to take their kids into the hospital all the time for what they know is such a difficult thing.

In addition to the emotional strain frequent transfusions impose, they also create a financial burden. According to Fred Hyde, a physician who is now studying law at Yale University, Connecticut Blue Cross has agreed to a system under which families can be reimbursed for transfusion costs even when their child is treated as an outpatient. When outpatient treatment is medically satisfactory, Hyde says, it is better and cheaper for everyone. Blue Cross is trying the plan for a year.

In an interview on a program about Cooley's anemia produced by station WTIC-TV in Hartford, Connecticut, Iovene talked a little about his life. "My life is very dependent on the hospital," he said. He also said a bit about how it feels to wear the mask of a Cooley's patient. In victims of this disease, the bone marrow works overtime, and the excess marrow produced leads to enlargement of the bones, particularly facial bones. That feature, along with a darkish skin pigmentation, gives these individuals an Oriental look. "People tend to treat me as an Oriental, but I'm Italian," Iovene said. As a child it used to bother him, especially when other kids teased him. Now, he says, he's used to it.

Giaimo had never heard of Cooley's anemia until Iovene and Mrs. Guiliotis came to him. He was moved. A large number of his constituents are of Mediterranean ancestry and so is he. According to what the two Cooley's advocates told Giaimo, the federal government has been derelict in providing funds for research on this disease, which has certain similarities to sickle cell anemia. At the time of their conversation, sickle cell anemia had been singled out for special attention by President Nixon, and legislation to mount a national program to combat sickle cell was pending before the Congress. Giaimo said he would see what he could do for Cooley's anemia.

Cooley's Virtually Unknown

Back in Washington, Giaimo's first move was to try to amend the sickle cell anemia bill to include funds for Cooley's. However, he has said, leaders of the subcommittee that was steering the bill through the House (the Subcommittee on Public Health and Environment, headed by Democrat Paul G. Rogers of Florida) balked at the idea of an amendment. They had never heard of Cooley's anemia, just as many of them had been unfamiliar with sickle cell before that, and they did not want to jeopardize the bill's chances of passing.

Giaimo, therefore, decided to introduce a new bill. "Thus started the long, tortuous but orthodox route through the committee system," he wrote in the New Haven *Register*. Letter-writing campaigns were organized, and promoters of the legislation began working with the modest coalition of Cooley's groups around the country. "They constituted a very unimpressive lobby," said one of Giaimo's aides, "but we all thought it was appropriate to get legislation parallel to that passed for sickle cell anemia, so we got things going." Apparently, it is only recently that voluntary Cooley's groups have really begun to think in political terms. The first such group, which is headquartered in New York, began as an outfit designed to help individual patients and their families. Now, although that is still a main objective of most Cooley's chapters, some are paying more attention to benefits that can accrue through the political process.

Giaimo did what he calls "the necessary horse-trading and bartering to make sure that a hearing—the all-important first step for a new bill—would be held." Representative Ella Grasso (D–Conn.) became interested in Cooley's, reportedly after she saw the WTIC special when she was home in Connecticut one night. She, too, joined the ranks of Cooley's supporters, which were filled by a number of congressmen from northeastern states, where there are clusters of communities of people of Mediterranean descent.

Rogers scheduled hearings before his subcommittee for last May. As had been the case at the sickle cell hearings earlier, there was testimony pro and con. Opposition to special Cooley's legislation came primarily from men within the Nixon Administration who argued that legal authority to deploy funds for Cooley's research already existed, that research was already being supported, and that a new bill would merely duplicate existing authority.

They lost. In both the House and the Senate, where the bill was guided primarily by Senators Abraham Ribicoff (D–Conn.) and Edward M. Kennedy (D–Mass.), the act to control Cooley's anemia passed overwhelmingly. The President signed it in August, just before he headed for Hawaii for talks with the prime minister of Japan. Cooley's anemia thus became the second ethnic disease to rate special national attention. It is supposed to get $11.1 million during the next 3 years.

Many of the problems and issues associated with a concerted effort to do something about Cooley's anemia are similar to those raised by the sickle cell anemia program (*Science*, 13 and 20 October). Each disease afflicts a definable ethnic group. Each is a killer. Each has been somewhat neglected by the research community. Politically, it would be very difficult for any legislator to vote against a program aimed at helping the victims of these genetic disorders.

Nevertheless, a number of high-ranking individuals in the Department of Health, Education, and Welfare and, specifically, in the National Institutes of Health (NIH) think that the apparent trend toward categorical attacks on

genetic disease is a mistake. Many see a particularly dangerous precedent in the Cooley's anemia bill. "The President should have vetoed it," one NIH official declares. Even on Capitol Hill there are indications that some congressmen want to keep the potential proliferation of bills concerning ethnic diseases in check. (Dysautonomia and Tay-Sachs diseases, which strike Jews, and cystic fibrosis, which primarily afflicts whites of western European ancestry, have been cited as candidates for future legislative action.) Comments by some of Rogers' staff aides indicate that he is sympathetic to the idea of putting on the brakes. And some Hill staffers are beginning to refer to Cooley's anemia as the "ethnic disease of the month," a macabre way of hinting that they think things may be getting out of hand.

When it comes to supporting basic research in Cooley's anemia, there seems to be some disagreement between investigators, on the one hand, and congressmen and Cooley's groups, on the other, about what constitutes relevant work. Studies by W. French Anderson at the National Heart and Lung Institute are a case in point. Anderson is an M.D. who for many years has concentrated on studies of the molecular basis of genetic blood disorders, of which thalassemia is one. Highly regarded by his colleagues, he has won acclaim recently for elegant studies of hemoglobin synthesis. During the congressional hearings on the legislation, Anderson was cited as one of the few people at NIH whose research is directed at dealing with thalassemia. He is spending, Congress was told, about $125,000 a year on thalassemia research. The congressmen and the backers of Cooley's bill said they hardly considered that a very major federal effort, certainly not sufficient, even by way of example, to defend the position that adequate research is already being conducted.

Anderson's view of the dimension of his effort is a little different. In his opinion, it is entirely reasonable—indeed, definitely appropriate—to say that virtually all of the work in his laboratory is directly related to thalassemia. According to Anderson, the only way one could arrive at the low $125,000 figure is to include only those projects with the words thalassemia or Cooley's anemia in the title. To do that, he thinks, is to miss the point. In fact, Anderson maintains, his laboratory is spending close to $500,000 a year on the problem and is making very definite headway in understanding the molecular nature of the defect and in laying the foundations for the ultimate "cure" or successful therapy of Cooley's patients. Like most investigators at NIH and elsewhere, Anderson says he could use more laboratory space. Unlike many, however, he is satisfied with the level of financial backing he is getting. By next spring, he expects his section to be elevated to branch status at the heart and lung institute and estimates that they will be able to expand their human studies from research with about 15 patients to a series of about 100 individuals. In short, things are going rather well. Anderson's principal word of caution is that the work which has been so exciting and successful lately is very basic stuff. It would be misleading to assume that it will lead to therapeutic measures shortly.

Hemaglobin Studies Promising

Anderson considers some of his most promising research at the moment to be work that has to do with the behavior of hemoglobin in sheep and goats. A recent paper is titled "Hemoglobin switching in sheep and goats: change in functional globin messenger RNA in reticulocytes and bone marrow cells." It is not the sort of thing a layman would think has anything to do with Cooley's anemia. But, Anderson says, it does.

To make his point, he touches on what is known about the genetic defect involved in thalassemia from work in his laboratory and those of a number of other researchers, including David Nathan at the Children's Hospital Medical Center in Boston and Paul Marks at Columbia University. In sickle cell anemia, with which Cooley's is often compared, the red blood cell is defective because of a structural abnormality. Sickle hemoglobin is qualitatively different from normal hemoglobin. In Cooley's, the distinction between normal and defective cells is quantitative. Red cells have too little hemoglobin, but what they do have is perfectly OK. Anderson thinks that he and other investigators are close to knowing why this is.

The problem, he believes, lies with a faulty gene product; that is, in persons with thalassemia, the gene that makes a product called messenger RNA, which directs the synthesis of part of the hemoglobin molecule, is awry. For some as yet undefined reason, the messenger RNA does not do its job the way it should.

This is where the sheep and goats come in and where some people find it hard to make the jump between the fundamental work and the clinical disease. Human beings have two major forms of normal hemoglobin—fetal and adult. Sometime during the first few months of life, infants stop making fetal hemoglobin and, by some genetically controlled switch, turn on production of adult hemoglobin. In sickle cell patients, it is the adult hemoglobin that is faultily constructed; in Cooley's victims, it is also the adult hemoglobin that is causing trouble. Sheep and goats make three forms of hemoglobin—fetal, adult, and an intermediate form called hemoglobin C. Goats and certain sheep that Anderson is studying have a neat mechanism for protecting themselves against anemia. "If you make an adult sheep of this special breed anemic," says Anderson, "he just stops making hemoglobin A [adult] and reverts to making hemoglobin C."

In theory, if one could elucidate that mechanism and, ultimately, apply the knowledge to man, one might be able to effectively treat people with sickle cell anemia, Cooley's anemia, and other hemoglobin defects by keeping them from making adult hemoglobin in the first place. It is perfectly possible, Anderson says, to live a normal life with fetal hemoglobin, as evidenced by cases of individuals who never switched to the adult form. The reason they fail to make adult hemoglobin is unclear.

While such fundamental work is being patiently pursued, there is much that could be done to aid Cooley's patients in the more immediate future, Anderson and others believe. One area that deserves greater research attention in that regard is the problem of iron poisoning from a lifetime of massive blood transfusions. Once iron is deposited in the heart or kidneys, for example, there is no natural physiological process by which it can be removed. And the drugs that are available for this purpose are generally highly toxic agents whose usefulness is, therefore, severely limited. A new and safe drug for iron removal is badly needed. There are, according to NIH scientists, a few laboratories that are apparently willing and able to undertake this research if funds become available.

Another area in which money can be spent in the hope of providing some relatively immediate assistance to people at risk of carrying the gene for Cooley's is screening. (In order to have

Cooley's anemia itself, or what is also known as thalassemia major, a child must inherit the gene from each of his parents. Trait carriers, who have thalassemia minor, are unlikely to have any physical manifestations of that condition.)

Testifying on the Cooley's bill before the House subcommittee, HEW and NIH officials declared that there is no reliable test that can be used in mass screening for thalassemia trait. Subsequently, they and investigators in the field have reiterated this opinion, adding that it would be wise to earmark some of the new money for research to develop an accurate and inexpensive screening technique.

Howard Pearson, a pediatric hematologist at Yale University, contends that a suitable test is already near at hand. Pearson, who has been deeply involved in both sickle cell and Cooley's programs in the New Haven area and who was one of the leading expert witnesses at the Cooley's hearings, reported then that his own studies of approximately 350 individuals point to a system, which automatically detects quantity and size of red cells, that can be used for screening on a large scale. He uses a Coulter electronic cell counter, which costs in the neighborhood of $50,000. Expensive as that may sound for anything involved in a mass screening program, Pearson points out that it is a piece of equipment that is standard in a large majority of hospitals.

Screening for thalassemia trait, he says, is important for two reasons. As is the case with sickle cell trait, the information obtained by screening can provide the basis for genetic counseling of couples who carry the gene for thalassemia. (Admittedly, it also raises certain psychological and social problems and is something that, ideally, must be done with the utmost concern and care that the people being screened be educated about what being a carrier means.) Just as important, if not more important, carriers of thalassemia trait need to be identified for their own physical good. It is possible, Pearson comments, for an individual with thalassemia trait to be diagnosed as having mild iron deficiency anemia and given iron to correct the deficiency. "Iron," he says, "is contraindicated in thalassemia trait. It can be bad for the patient." Therefore, anyone who carries the trait stands to gain by knowing it.

It is, of course, hard to say just what effect the national effort to combat Cooley's anemia will have. Certainly from the point of view of patients and their families, it can only help. Whether it will provide the extra measure of support that will lead to some discernible progress and whether it will, in fact, set a precedent for similar targeted attacks against other clearly ethnic genetic diseases is anything but clear.—BARBARA J. CULLITON

Environmental Legislation: Last Word from Congress

In a presidential election year, the last few weeks of congressional business are conducted in an atmosphere like the one that must have prevailed on the boat deck of the *Titanic*. When different parties control the White House and Congress, the contest to allocate credit and blame is likely to be particularly intense, and the last days of the 92nd Congress—adjournment came on 18 October—ran true to tradition. This year, with inflation a factor and a big deficit looming, the main issue was money, and the argument centered on which party was responsible for bloating the budget.

As the political witching hour approached for Congress, the usual bout of last-minute legislative bargaining occurred, and, for better or worse, a clutch of environmental measures received final action, including major water pollution control and pesticide regulation bills.

On the last day of the session, both the Senate and House voted to override a presidential veto of the Federal Water Pollution Control Amendments (S.2770). The bill authorizes some $24.7 billion over 3 years, including more than $18 billion for grants to the states for water treatment plants. The President said he had vetoed the measure on the grounds that the funds provided exceeded his request and might lead to higher taxes.

The new bill is not simply the most expensive environmental bill in history, it also changes the basis of pollution control. As the Senate Public Works Committee report puts it, the major effect of the legislation is "a change in the enforcement mechanism of the water pollution control program from water quality standards to effluent limitations."

Under the 1965 Water Act, states were to set water quality standards for their own waters; these standards had to meet federal requirements. Water was to be classified in different categories for different uses. Water used for drinking and swimming, for example, had to meet much higher standards regarding oxygen levels and bacteria content than water used for boating or, obviously, for receiving industrial effluents.

The program has not succeeded brilliantly. Many states have not established water quality standards acceptable to the federal government, and there have been serious technical difficulties in determining the relationships between specific pollutants and particular levels of water quality.

The new bill brings about a shift in criteria to effluent limitations. The timetable in the bill requires that by 1977 industry use the "best practicable" technology for treating wastes discharged into U.S. waters. By July 1983, industry must have installed the "best available" treatment equipment, and that same year is set for the goal of making all waters safe for fish, other wildlife, and people. The year 1985 is the target year for achieving the national goal of eliminating all polluting discharges.

Although most environmentalist organizations endorsed the bill, their enthusiasm was not unalloyed. The measure is an authorization, not an