survived for six weeks from the beginning of treatment, went into remission. This is an encouraging experience, all the more so because other workers had reported failure mainly because the degree of toxicity was too great in the dose schedule they utilized. The study reported in this issue also emphasizes the possibility that if more patients had survived the initial period of six to eight weeks of risk from infection or hemorrhage, more remissions might have been achieved. Control of the bacterial flora of the patient may be of great importance in this group of patients.

It is to be anticipated that longer remissions and greater prolongation of life will be reported as a consequence of the many clinical investigations now under way through the Acute Leukemia Task Force of the National Cancer Institute, or in independent research programs, because of the concerted effort that is being made to take advantage of new pharmacologic data and clinical observations in making more effective use of the antileukemic agents presently available.

Further evidence for the suggestion that acute leukemia should be classified not only in terms of cell types but, more importantly, on the basis of response to therapy comes from the long survival of a small number of children and adults with acute leukemia. These comprise only 0.5 to 1.0 per cent of the total number of patients with this disorder. It is estimated that over 150 patients with acute leukemia have survived for more than five years. Ninety-three of these were collected from 49 investigators by Burchenal.[5] Of the 20 adults who survived for five years or more 7 have no evidence of disease, and 12 either are living, with some evidence of leukemia, or have died. Seventy-three are children, of whom 49 are living with no evidence of leukemia. Five of these are living at eight years, 6 at nine years, 1 at ten years, 2 at eleven years, and 1 each at twelve and fourteen years. A registry of these long-term survivors has been set up at the Acute Leukemia Task Force of the National Cancer Institute. These data are of great importance for the patients and families concerned. It must be emphasized that this experience does not apply to the more than 99 per cent of the patients with acute leukemia who do not respond in this manner. A precise explanation of this phenomenon could be of great importance for the control of acute leukemia.

It is possible, and even likely, that 2 different chemical agents may be required to solve the problem of acute leukemia in a given patient. The first must be designed to destroy all the leukemic tumor in all parts of the body. It is possible that the agents presently at hand will be employed successfully to this end. A second chemical must have as its target the destruction of the agent (? virus) responsible for the induction or the reinduction of the leukemic process in the same patient, or it must be capable of correcting the biochemical defect, no matter how produced, responsible for the chain of events leading to leukemia. And while these lines of investigation are continuing, the search for causative agents or mechanisms has increased markedly in the last few years. Success in this direction may make possible the production of a vaccine, or lead to the eradication of the leukemogen from the environment.

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HEMOLYSIS WITH VITAMIN E MALABSORPTION

Vitamin E actually comprises a number of closely related, lipid-soluble congeners, the most important of which is alpha tocopherol. Although ubiquitous in nature, occurring in unicellular organisms as well as in all animal tissues yet studied, its exact biochemical role remains enigmatic. The diversity of the pathologic conditions in which deficiency of the vitamin is thought to be involved confounds, rather than illuminates, its metabolic properties. Deficient chicks suffer muscular degeneration, exudative diatheses and encephalomalacia, and in rats reproductive incapacity, and in swine hepatic necrosis, result from prolonged dietary lack of the vitamin.

The best documented property of tocopherol is its ability to act as an anti-peroxide compound. If erythrocytes from tocopherol-deficient animals (including human beings) are exposed to hydrogen peroxide in vitro, rapid peroxidative denaturation of membrane lipids occurs, leading ultimately to striking hemolysis. These alterations are prevented by supplementation of vitamin E either in vivo or by addition of the vitamin directly to the in vitro system. This peroxide hemolysis test has become the simplest screening procedure for tocopherol deficiency.

In addition to its usefulness in the laboratory, the ability of peroxides to induce red-cell damage has generated interest in the possibility that such a phenomenon is operative in certain hemolytic disorders of human beings. Evidence has accumulated that Heinz-body anemias and possibly normal erythrocyte senescence and destruction may result from oxidative damage to the cell and its hemoglobin.[1] Recently two drugs, primaquine and pamaquine, which can produce Heinz-body hemolytic anemia in man, have been reported to generate hydrogen peroxide in vitro.[2,3] In addition the hemolytic anemia that follows the exposure of tocopherol-deficient animals to hyperbaric

oxygen is presumably related to the tissue peroxides produced during the exposure. A recent case of hemolytic anemia that followed a brief exposure to oxygen under high pressure has been described in a patient whose red cells demonstrated inordinate sensitivity to hydrogen peroxide in vitro.[4] This case may be the harbinger of others now that therapy with hyperbaric oxygen is utilized so widely.

A number of defense mechanisms are available to the red cell for the detoxification of hydrogen peroxide. Catalase, present in large quantities in human red cells, degrades hydrogen peroxide to water and oxygen, and even in its absence, as recently shown in studies in hereditary acatalasia,[3] hydrogen peroxide is dissipated in red cells through peroxidation of the sulfhydryl compound, reduced glutathione. If, as suggested by the studies of Binder and his colleagues, published elsewhere in this issue of the *Journal*, the survival of red cells is diminished in patients with steatorrhea and concomitant tocopherol deficiency, the combined mechanisms of glutathione peroxidation and catalasic decomposition of hydrogen peroxide are evidently not always sufficient to prevent damage to red cells by peroxides. The unique solubility of tocopherol in lipids may underlie this phenomenon. Thus, sulfhydryl groups in the lipid milieu of membranes may evidently be oxidized by hydrogen peroxide if tocopherol is absent despite the presence of adequate peroxide antagonists in the cytoplasm. The resulting destruction of reduced sulfhydryl groups in red-cell membranes would sensitize the cells to early destruction in the reticuloendothelial system.[5] Reports of hemolytic disease in patients with malabsorption syndromes who are exposed to oxidizing drugs, hyperbaric oxygen and the like will therefore be awaited with interest.

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89TH CONGRESS — FIRST SESSION

LIKE his medical team that left no stone unturned, President Johnson was equally thorough and supervised during the first session of the 89th Congress the passage of an unprecedented volume of welfare legislation designed to take the nation further along the road to the Great Society.

The Senate Republican Policy Committee has estimated that 50 of the Great Society bills enacted this year entail a cumulative cost of $112,000,000,000 — a figure beyond the immediate grasp and understanding of the average taxpayer, who, with his progeny, will pay for the Great Society. Almost as incomprehensible, to those mindful of the limitations of time and human capacity, is the fact that when the first session of the 89th Congress was adjourned, 16,882 bills had been introduced, including 950 that affected medicine, medical care and public health.

Two of the enacted bills, because of their scope and still undetermined effect on a nation's people, medical and allied professions, and institutions, would have satisfied a Congressman's constituents, if they had been the only bills enacted during the recent session, that their man in Washington was doing something.

These two bills — Medicare (P. L. 89-97; $6,500,000,000) and the Heart Disease, Cancer and Stroke Amendments of 1965 (P. L. 89-239; $340,000,000) when added to the $2,300,000,000 aid-to-higher-education bill, the $1,300,000,000 Elementary-Secondary Education Act, the $1,800,000,000 war on poverty program, the $7,800,000,000 Highway Act, and others, make quite a package. If anything, the first session of the 89th Congress did about everything. One hopes that when the second session convenes on January 10, its members will rest on their laurels and study the effects of the implementation of the various programs on the economy, health and welfare and integrity of the nation and its people.

Indications are, however, that, particularly in the health-care field, Congress may pick up where it left off when it adjourned at 12:51 a.m. on Saturday, October 23. The recent White House Conference on Health, conducted by White House Assistants and the United States Public Health Service, augurs more health-care legislation in 1966.

Furthermore, Senator Russell Long, of Louisiana, who confesses that it would be difficult for him to report on "just 70 per cent of what happened in the Finance Committee where I am a ranking member," predicted that "Next year they are going to extend and perhaps make unlimited the amount of hospital care a person can get under Social Security. One of these days the program will be expanded to include people below 65."

The *Journal* took the liberty of suggesting in an editorial, "Medical Visions and Horizons in the Great Society" (February 18, 1965), that "the lawmakers and the physicians both realize that the safest way up the medical ladder to the Great Society is, despite pressures and urgencies, one rung at a time."

The suggestion is worth repeating. Or — if a changed metaphor would help — it is suggested that the second session of the 89th Congress pause along the road to the Great Society for refreshment and sober contemplation.

— IN A COUNTRY CHURCH-YARD

As one leaves Killarney and its highland lakes and takes the Tralee road to the north, the first left turn