days the collective imagination of the American public has been dominated by John Glenn's orbital flight. Television did a remarkably competent job of presenting the actual flight and the festivities that followed its successful completion. But during this whole period not so much as a single half-hour segment of television time on any station or network was devoted to an explanation of the scientific background of this exploit. Presentation of a few basic principles—such as the concept of an orbit, weightlessness, physical conditions in space, and the physiology of space flight—could have lent meaning and substance to this great technological achievement.

Is it not the duty of the AAAS, as the spokesman for American science, to see that another such opportunity does not pass unheeded?

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Cell Growth

A preoccupation with Mendelsohn's concluding remarks in his report on chronic infusion of tritiated thymidine into mice with tumors [*Science* **135**, 213 (1962)] to the effect that a tumor "literally doubles before one's eyes" should not deflect attention from the real significance of the finding that an appreciable number of tumor cells do not give evidence of DNA accretion during a 3- to 7-day period and are therefore not proliferating. If this conclusion can be stretched to embrace a corollary hypothesis—that cancer is not necessarily a wildly proliferative, exuberantly growing, racing reduplication of cells, that it may, in fact be just the reverse—it will then be found to fit in with a welter of ancillary evidence, emerging from all medical subdisciplines, that calls for a reassessment and readjudication of common (descriptively borne) notions of the nature of cancer.

If the question had arisen in connection with the usual experimental tumor —a transplant—I would not have bothered to comment. Arising as it does from studies of tritiated thymidine uptake (studies whose validity I feel to be established) by an autochthonous tumor which is as close as anyone can get to spontaneous human carcinoma, the factual data from Mendelsohn's Fig. 2,

showing that in an infusion period of up to 4 days three out of seven tumors show about 50 percent labeling, must be taken seriously. Compared with normal acinar cells, the rate of proliferation must be considered markedly *diminished* in the neoplasm.

Why, then, tumor formation? The answer in this instance applies to carcinoma in any situation. Normal epithelial cells are always contiguous to a lumen, where their proliferation products are discharged. Allow clones of these normal epithelial cells to be displaced beneath muscularis mucosae where such desquamation is impossible, and intumescence is inevitable if proliferation occurs at all, even if it occurs at only 1/100 the rate of proliferation of the homologous cells of origin (Barnard and Oppenheim, *Brit. Med. J.*, **1**, 943 (1962). The "doubling" in tumor size in a period of 1 week can be put into proper perspective when one considers the quadrupling in size, in a 6-hour period, of the normal lactating breast: although the volume may be fluid, all of it represents a prior accretion of acinar cells.

The growing realization that some experienced pathologists have been right for years in calling malignant neoplasm a frozen senescence rather than an exemplification of youthful proliferative overactivity arises now from a diversity of promptings. One of these that deserves mention, in light of Mendelsohn's doubts about the rationale of administering the halogenated pyrimidines as cancer chemotherapeutic agents, is the notorious failure of any of the antimetabolite therapies to do anything for the patient other than retard the function of the bone marrow and the intestinal mucosa where a high proliferation rate *is* extant.

From our present knowledge that all popular cancer chemotherapeutic agents (with the exception of steroids and certain antibiotics)—whether of the ureide-base analog, folic acid antagonist, alkylating agent, or radiomimetic class—have in common the characteristics of (i) curbing ureide-bases synthesis, (ii) preventing nucleoside and nucleotide formation, and (iii) curtailing nucleotide polymerization and incorporation into the polynucleotide chain, we can say that Mendelsohn's argument applies to all these as well as to definitive irradiation. And well it might, if, as we are beginning to suspect, the nature of cancer is the opposite of what we always thought it to be—if a cancerous cell is a cell finding difficulty in organizing its information through adequate DNA synthesis.

To further increase this informational digestive disability by exhibiting noxious potions is a therapeutic fallacy which I believe is pointed up by Mendelsohn's results.

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There are three ways in which the growth of a cell population can increase: (i) through prolongation of the survival time of nonproliferating cells, (ii) through shortening of the generation time of proliferating cells, and (iii) through increase in the fraction of proliferating cells in the population. Methods for measuring these parameters in cell populations are only just becoming available. Until we have the appropriate information about the growth properties of normal and tumorous breast tissue, I cannot support the statement that breast tumor cells are proliferating at a diminished rate. Actually, there are few if any cases where enough data are available for an adequate comparison between comparable normal and tumorous tissues, but this situation should soon be rectified. For

example, A. B. Reiskin and I are studying normal basal cells and carcinogenically induced epidermoid tumors in the hamster pouch. As is often the case, these tumors have a higher mitotic index than their normal counterparts. In addition, the tumors have a shorter duration of DNA synthesis, and labeling is more intensive (there are more grains per labeled cell) than in normal cells. Since the fraction of cells that become labeled after a single injection of tritiated thymidine is higher in the tumor than in the normal cell, it is clear that in this case the tumor cells have the shorter generation time, or the larger fraction of proliferating cells, or both.

In any case, these arguments and the infusion experiment are significant only for those therapeutic situations where effectiveness hinges on mitotic activity of the target cells during exposure to the agent. Barnard may be justified in his skepticism about most cancer chemotherapy, but I tend to be more optimistic in view of our lack of complete understanding of the mechanism of action of current therapeutic agents (including ionizing radiation).

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Desert Vegetation in Nevada

"Succession in desert vegetation on streets of a Nevada ghost town" [*Science* **134**, 670 (1961)] is a commanding and iconoclastic title, and an article on this subject deserves very careful reading. Upon such reading, I feel sure the paper deserves commendation—and comment. In this study, Philip V. Wells (New Mexico Highlands University, Las Vegas) compared the density and frequency of the most abundant species on the 33-year-abandoned streets of an ephemeral ghost town in Nye County, Nevada, with conditions on "undisturbed" adjacent bajada. He shows that "several shrubs of dry washes can become established in abundance on . . . an alluvial fan," and states, "Obviously, these pioneer plants of the desert play a role similar to that of successional plants of more humid regions."

As a statement of what Wells is trying to topple, I quote Wells: "Shreve . . . concluded: 'Each habitat in each subdivision of a desert area has its