reference to the general safety technologist not completely familiar with the problems of laboratory safety. It fills a void of which every laboratory supervisor has been acutely aware at one time or another.

The publication represents yet another discipline in the series inaugurated by the publisher 60 years ago with the now-familiar Handbook of Chemistry and Physics. However, this modern compilation adopts a format enjoying current popularity in this era of technical specialization. Editor Steere has invited 40 contemporaries in the field of safety to contribute chapters on specific subjects in which they are particularly qualified. Unfortunately the contributions too frequently are articles previously written for a periodical, and the result is a decided lack of editorial uniformity which sometimes yields obvious redundancies. This is not objectionable to the casual reader, but is distracting to the student seeking an overall comprehension of laboratory safety.

Each of the chapters has a list of pertinent references appended, and there are numerous tables. The most unusual, from the standpoint of content, is the large one which covers 110 pages at the back of the book. It is a compilation of health- and fire-hazard and related properties for more than a thousand laboratory chemicals. This feature is reason enough in itself for having the handbook available in many laboratories. Let us hope that the editor is diligent in expanding and updating this table in future editions.

The volume is not without editorial oversights and typographic errors. Typographic errors in text are usually inoffensive, but when they appear in diagrams, as in figure 13, page 378, or result in the mislabeling of components as in figure 1, page 83, they become annoying. Negative feelings were also aroused by a chapter on deleterious effects of electric shock and another discussing the physical qualities and giving a complete description of all types of glass. In a handbook on laboratory safety guidelines, they create an impression of padding. In direct contrast, many of the chapters, notably the one on compressed gas cylinders and cylinder regulations, present exactly the safety information a laboratory man would be seeking in a handy reference.

In concept and content the handbook, from an overall viewpoint, is an exceptionally fine fulfillment of a long-standing need. Scanning the volume should be a revelation of unrecognized

or previously learned but forgotten hazards for the laboratory supervisor. The professional safety technician will find the reference source an excellent point of departure for more comprehensive study of laboratory safety problems.

C. L. BALDWIN

Dow Biohazards Department, Pitman-Moore Research Center, Zionsville, Indiana

Viral Oncology

Subviral Carcinogenesis. First International Symposium on Tumor Viruses, Nagoya, Japan, Oct.—Nov. 1966. Yohei Ito, Ed. Aichi Cancer Center, Chikusa-Ku, Nagoya, 1967. xvi + 441 pp., illus. \$18.

After the ninth International Cancer Congress held in Tokyo in October 1966, a group of virologists and scientists in related fields continued their discussion in Nagoya. This volume is a record of their conference. As is often the case in a rapidly moving field in which productive scientists are asked repeatedly to write reviews, many of the data had already appeared elsewhere. Although progress has been made since the conference, it is convenient to have the material assembled in a volume which accurately reflects the state of knowledge of viral oncology in the fall of 1966.

The reader of this volume will be exposed to the widely diverse experimental techniques and methodology devised and used by tumor virologists. The techniques encompass physical, chemical, and immunological methods. Various laboratory tumor-virus-host model systems were reported in the hopes of finding clues to the understanding of human malignancies.

Several participants emphasized that evidence for the persistence of virus genes in cells transformed to malignancy is conclusive; this view is based on the discovery of viral induced antigens and virus-specific messenger RNA in virus-induced tumors. Equally conclusive is the ability of defective virus genomes to effect transformation and to persist in the transformed cells. Transmission of the defective viral genome from cells transformed by RNA viruses to susceptible cells can apparently occur by cell-to-cell contact. There remains the question of how the viral genes impart the properties that lead to oncogenesis.

Cells doubly transformed by two

papovaviruses, SV40 and polyoma, contain both transplantation antigens; this report is similar to the finding that a murine leukemia cell which continually produces an RNA leukemia virus can be further transformed by polyoma virus and then contains additional transplantation antigens induced by the DNA virus

The RNA of Rous sarcoma virus and that of avian myeloblastosis virus were reported to have molecular weights of 12×10^6 daltons, similar to those of the murine leukemia and mammarytumor viruses. The continuous participation of cellular DNA was found to be required for the growth of Rous virus. In cells transformed by the Rous sarcoma virus, persistence of the virus genome was observed; particles from the nonproducing cells contain substances with the same high molecular weight as that of the virus RNA. However, infective RNA could not be isolated from the particles or from the tumor cell.

Studies on the adenovirus-SV40 hybrid population were reported. Laboratory-induced transcapsidants were obtained that contained an SV40 defective genome in a type 2 adenovirus capsid. This for the first time conferred oncogenic potential on the type 2 adenovirus.

Human tumor viruses and human leukemia virus remained elusive. Progress being made at the cellular and molecular level is heading towards a resolution of many of the complex problems concerned with the transformation of cells by viruses. Much hope is riding on the work in hybridization, specifically on the search for specific homology between transformed-cell messenger RNA (mRNA) and the DNA of the adenovirus that might have caused the tumor. This methodology, which works for virus-induced animal tumors, lends itself to exploitation in determining whether human cancers contain genetic information specified by known viruses. However, there must be a note of caution; even if virus-specific mRNA should be found in human cancer cells, this by itself would not be rigorous proof that the virus coding for the resident mRNA was the etiologic agent of human cancer.

In order that the reader profit most from this specialized volume he should have a background knowledge both of the nature of viruses and of the basic concepts of immunology.

Joseph L. Melnick

Department of Virology and
Epidemiology, Baylor University
College of Medicine, Houston, Texas