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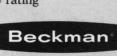
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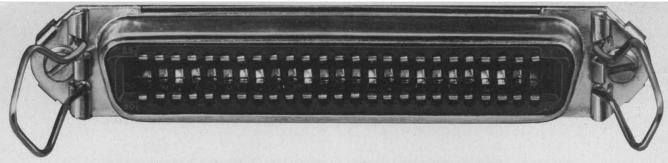
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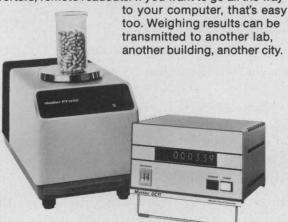




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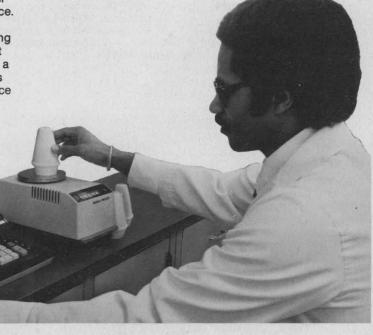
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Volume 194, No. 4270

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COVER

Moreno Glacier at Lago Argentino in the Andes of Argentina with its terminus advancing into a stand of southern beech (Nothofagus pumilio) [J. H. Mercer, Ohio State University, Columbus]. The orbital diagram [G. Kukla, Lamont-Doherty Geological Observatory, Palisades, New York] symbolizes conclusion of research that variations in the earth's orbit are the fundamental cause of the succession of late Pleistocene ice ages. See page 1121.

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Autoradiograph of $10\mu m$ zirconia spheres containing alpha-emitting plutonium. Nomarski differential interference contrast. Negative magnification: 800X.

Photomicrographs 1, 2 & 3: Julie Langham Grilly, Los Alamos Scientific Laboratory.

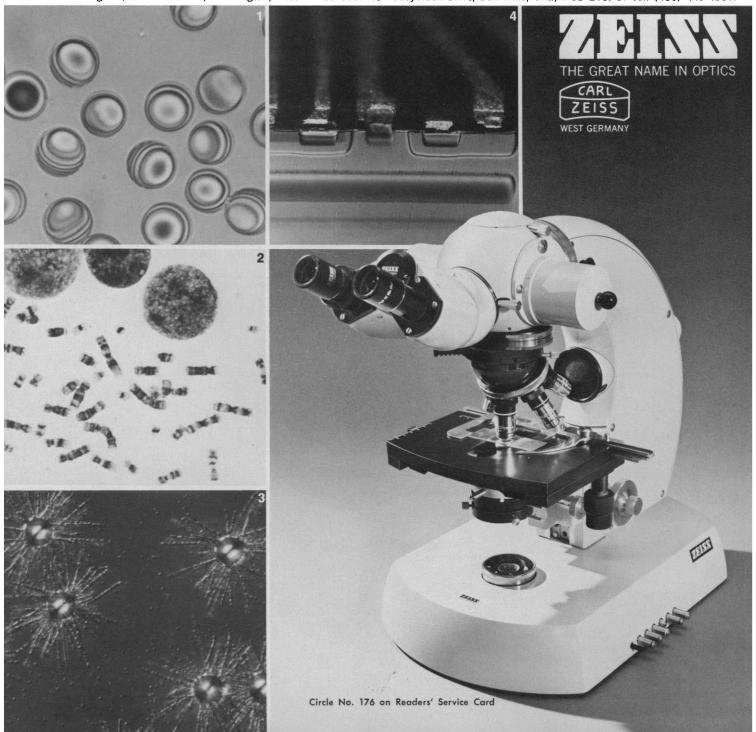
4. Semiconductor

Tapered cross section after etch, showing emitter base, buried layer, isolation and resistor diffusions. Nomarski differential interference contrast. Negative magnification: 528X.

Photomicrograph: Motorola Semiconductor Products Division.

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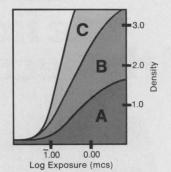
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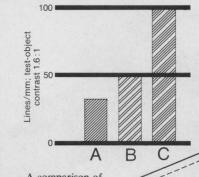
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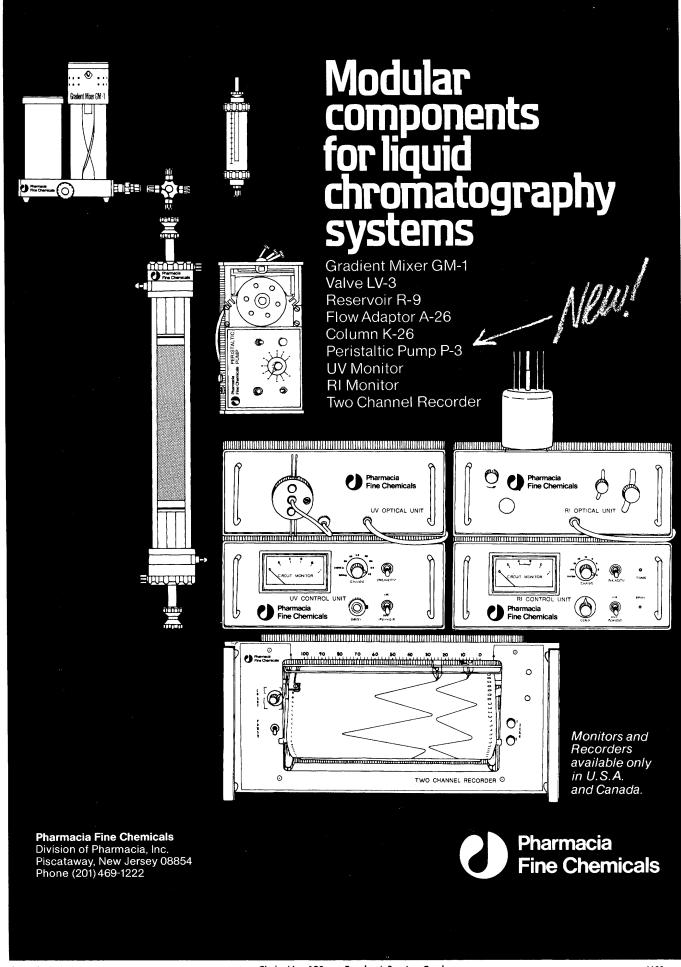
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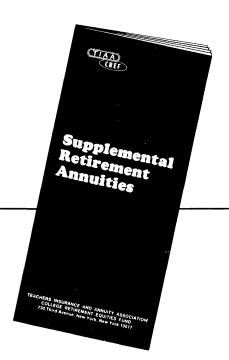
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LETTERS

Antitumor Drug Interactions: Additional Data

In response to the article "Cancer chemotherapy: An unexpected drug interaction" by Thomas H. Maugh II (Research News, 15 Oct., p. 310), we would like to call attention to published data (1) from our laboratory presented at the annual meeting of the American Association for Cancer Research in Toronto in March 1976. The data clearly show an unfavorable interaction between methotrexate and 5-fluorouracil with respect to their antimetabolic effect in de novo DNA synthesis. These studies were done in the L1210 cell system and more recently confirmed in the Friend leukemia system and in human bone marrow. We utilized the deoxyuridine suppression assay and tritiated deoxyuridine incorporation into DNA as sensitive indicators of de novo DNA synthesis which can measure the effects of 7.5×10^{-9} molar methotrexate and 0.2 microgram of 5fluorouracil per milliliter (2). The combination of 5-fluorouracil and methotrexate added to the cell lines or bone marrow did not significantly increase the de novo DNA defect as compared to the same amount of 5-fluorouracil or methotrexate alone (Table 1). The anti-DNA effect of methotrexate in this system could be significantly reduced by washing the cell lines after drug exposure, whereas the effect produced by 5-fluorouracil was not significantly reduced by washout. However, washing of the cells following exposure to the combination of the two drugs resulted in a 70 percent loss of the 5-fluorouracil effect. This decrease of 5-fluorouracil effect was directly related to the concentration of methotrexate and occurred even in sequential exposure. The addition of 5-formyltetrahydrofolic acid (folinic acid) to the drug combination appeared to prevent the washout of the 5-fluorouracil effect. We interpreted these results to mean that (i) some methotrexate-5-fluorouracil combinations are not additive or synergistic; (ii) methotrexate may diminish 5-fluorouracil effect during washout by preventing expression of 5-fluorouracil action mediated through a 5-fluorouracil high-affinity site; and (iii) the loss of 5fluorouracil effect in the presence of methotrexate probably resulted from a deficiency of the necessary folate coenzyme for the methylation of deoxyuridylate.

Our findings are supported by those of Santi and Martin, who have shown that methotrexate can interfere with the activ-

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Table 1. Methotrexate (MTX) and 5-fluorouracil (5-FU) interaction in L1210 leukemia. Cells were first incubated for 3 hours at 37°C in Hanks' balanced salt solution. Some were then washed three times with cold Hanks' solution. After the washout, [3H]deoxyuridine (0.1 μ c per tube) was added, and the incubation continued for 90 minutes. Additional details are given in (2).

Additions	[³ H]Deoxyuridine incorporated into DNA		
Additions	No washout (%)	Washout (%)	
Saline	100.0	100.0	
$MTX, 10^{-6}M$	17.4	95.6	
$MTX, 10^{-7} M$	90.0	105.1	
5-FU, 3.1 μ g/ml	6.9	13.9	
5-FU, 0.78 μ g/ml MTX, $10^{-6} M$ +	37.1	22.2	
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ity of 5-fluorodeoxyuridine when given in combination. Similarly, we and others have demonstrated that the cellular uptake of methotrexate and its antimetabolic effect may be reduced by 50 percent in the presence of certain corticosteroids commonly used in combination chemotherapy (2, 3). These studies emphasize the need for careful pharmacologic and biochemical assessments of the interaction of cancer chemotherapeutic agents when used in combination.

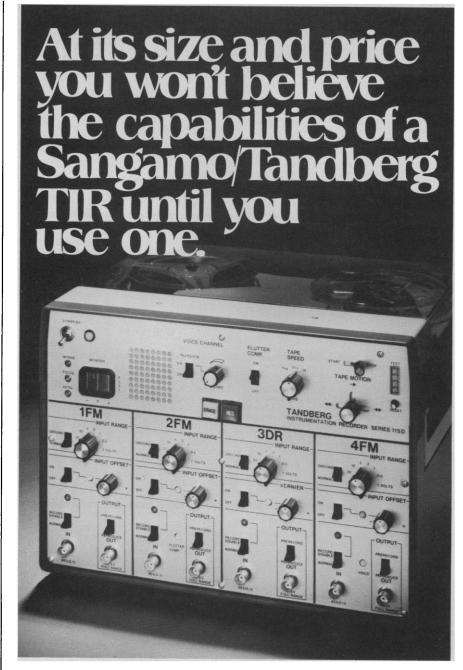
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We also have been concerned about the use of methotrexate and 5-fluorouracil in combination in the clinic and about the conflicting evidence as to whether this combination of drugs is synergistic. additive, or antagonistic (1). We hypothesized that the combination would give additive or synergistic antitumor effects if treatment with high doses of methotrexate preceded treatment with 5-fluorouracil, and that this combination would be antagonistic if the drugs were administered in the reverse sequence. The rationale for this theory was that pretreatment with methotrexate would result in high intracellular levels of this drug which would act as an analog of



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SCIENCE, VOL. 194

 N^5 , N^{10} -methylenetetrahydrofolate, the coenzyme for thymidylate biosynthesis, and as a result 5-fluorodeoxyuridine monophosphate would bind irreversibly in ternary complex with methotrexate to thymidylate synthetase (2). The reverse sequence would not result in irreversible inhibition, since 5-fluorodeoxyridine monophosphate levels would be falling at a time when methotrexate levels would be increasing. Use of high doses of methotrexate would also be important, since low intracellular levels of methotrexate would inhibit dihydrofolate reductase, but not thymidylate synthetase, thus leading to a decrease in N^5 , N^{10} -methylenetetrahydrofolate levels, and possible antagonism with 5-fluorouracil.

We have shown (3) that, in accord with this concept, treatment of mice bearing the sarcoma 180 with methotrexate 1 to 4 hours before treatment with fluorouracil enhances antitumor effects of fluorouracil; when the combination is given simultaneously, no additive effects are present; when treatment with 5-fluorouracil precedes treatment with methotrexate the effect is antagonism (less effect than either drug alone). Martin et al. (4) have also reported that methotrexate pretreatment (1 hour) followed by treatment with 5-fluorouracil resulted in therapeutic synergy against a spontaneous (CD8FI) mammary cancer.

In light of these results we have instituted clinical studies using pretreatment with methotrexate in combination with treatment with 5-fluorouracil in patients with breast cancer and colon cancer. If the experimental tumor data are applicable to human cancer, this should lead to an increased therapeutic effect without an increase in drug toxicity.

J. R. BERTINO

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Maugh refers to an observation of Santi and Martin that methotrexate interferes with the activity of 5-fluorodeoxyuridine on tumor cells grown in culture when the drugs are given at the same time. He also indicates that methotrexate should interfere with the activity of 5fluorouracil, since the mechanism of action is similar to that of 5-fluorodeoxyuridine. In his article, Maugh states that Martin and Santi contacted the National Cancer Institute, and that from limited data it was concluded that no additive effects were observed when methotrexate and 5-fluorodeoxyuridine were employed in combination chemotherapy of tumor-bearing mice.

In view of this, we should like to call attention to studies involving combination chemotherapy with methotrexate plus 5-fluorouracil in the treatment of mice with advanced leukemia L1210 (1). In a series of five experiments, Kline et al. observed that concomitant daily treatment with methotrexate plus 5-fluorouracil was more effective in increasing the survival time of the mice than daily treatment with the drugs employed individually. In these experiments the drugs were combined in a number of methotrexate/5-fluorouracil dosage ratios, and a wide range of daily treatment levels was employed for each combination ratio. Daily methotrexate alone was consistently more effective than daily 5-fluorouracil alone in increasing survival time, and the optimal combination treatment was 11 to 57 percent more effective than the optimal daily dose of methotrexate alone. In these experiments the improved therapeutic effect was observed with a relatively low daily dose of 5fluorouracil combined with a dose of methotrexate which was optimal or higher than the optimal daily dose for methotrexate alone. The data suggest that, on the daily schedule, 5-fluorouracil contributed to the enhanced therapeutic effect without itself adding significantly to the toxicity for the host. Other schedules of therapy for which a therapeutic advantage was obtained with this drug combination include 5-fluorouracil administered as a single dose (day 6 after leukemic inoculation) plus methotrexate administered either every 4 days or daily (from day 6).

In another experiment (2), a comparison was made of concomitant daily treatment with methotrexate plus 5-fluorouracil and methotrexate plus 5-fluorodeoxyuridine in the treatment of mice with advanced leukemia L1210. Both drug combinations were approximately equally effective in enhancing therapy relative to the drugs employed individ-

The selection for combination chemotherapy of methotrexate plus 5-fluorouracil (and 5-fluorodeoxyuridine) was based on the biochemical rationale that therapeutic enhancement might result



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from a sequential blockade in thymidylate synthesis or as a form of concurrent blockade involving purine and thymidylate biosynthesis. However, although therapeutic enhancement was obtained with the combination, it was concluded that the fundamental nature of the enhanced therapeutic response remained to be determined. Thus, although in accordance with the rationale, an "expected drug interaction" occurred, the fundamental basis for the therapeutic enhancement may not have been related at all to this interaction.

A wide variety of combinations of drugs has been demonstrated, in animal tumor systems, to provide a therapeutic advantage over that observed with the drugs employed individually. The demonstration of such therapeutic synergism may be dependent upon the dosage ratios employed, the dosage levels, and the schedule of administration, including the interval between treatments, number of treatments, total duration of treatment. and the timing of administration of the drugs relative to one another. The degree of advancement of disease and extent of infiltration or metastasis, as well as a variety of factors pertaining to the host, may influence the extent of therapeutic effect observed. Any biochemical rationale pertaining to fundamental interactions of a drug combination must be reflected in increased antitumor specificity in the tumorous host in order to obtain an improved therapeutic response.

We agree with Maugh that detailed investigations of drug combinations should be conducted and that fundamental investigations of biochemical and pharmacologic action, both in vitro and in vivo should be pursued, both retrospectively and prospectively, in relation to the usefulness of drug combinations in the treatment of clinical neoplasia.

ABRAHAM GOLDIN

Division of Cancer Treatment, National Cancer Institute, Bethesda, Maryland 20014

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Social Science in the White House

For nearly two decades social scientists have been talking about the desirability of having a voice in the highest policy-making councils of the nation. Some have favored placing in the White House a Council of Social Advisors, which would function like the Council of Economic Advisors. Others would rather add social scientists to that council.

More recently, high-level representation has been in disfavor among social scientists because they believe that such "high visibility" would make them into whipping boys of Congress. This is feared because social science tends to deal with value-laden issues and to have a relatively weak basis for many of its positions. Hiding behind the economists or natural scientists is hence considered prudent. Thus, it is said, social science does fare well within the National Science Foundation, and the best place for a social science adviser in the White House is as one of the deputies of the science adviser to the President.

Putting aside the question of how social science's voice is to be heard in national policy-making, it seems that despite its fledgling status, social science has significant contributions to make.

First, its representatives would provide an institutionalized source of basic social facts policy-makers are quick to ignore—for example, that many welfare clients are not able-bodied males, but old or disabled Americans, or mothers of several young children.

Similarly, if consulted before the recent rush to "de-institutionalize" mental patients, retarded children, and juvenile delinquents, most experienced social scientists would have pointed out that while many of those now in institutions can and should be released, (i) some cannot function on their own and have no families or community to return to, and (ii) some who are not now institutionalized need the kind of around-the-clock service only institutions provide. Therefore, closing institutions, as several states recently did, is premature to say the least.

There are literally thousands of such social facts, many summarized in Berelson and Steiner's *Human Behavior* and Rothman's *Planning and Organizing for Social Change*.* Unfortunately, books do not speak, and institutionalized occasions are needed to call attention to their content and to spell out their implications.

In addition to facts, social science perspectives ought to be represented in councils, which often contain only persons whose background is politics, law, or natural science. Thus, politicians typically tend to believe in the potency of the "Madison Avenue" approach. However, social scientists will point out that the view of human nature as subject to manipulation through advertising is probably erroneous. Ads may work well for products people already have a preference for, and are effective in switching people around among nearly identical products, but to overcome addictions or prejudices ads tend to be ineffectual. This has been established by studies on attitudes toward everything from campaigns against smoking to drives against prejudice. Can one, for example, expect an addict to heed such an emotionally shallow and brief input as a 60-second ad, compared to all the social, psychological, and physiological forces that bind him? Or, to put it more technically, can formal communication fight values and peer relations, community and social structure, personality, and biology?

True, social scientists will often not agree on what advice to give, but advisers from other specialities also disagree. And out of the heat of give-and-take a light does arise. Policy-makers should certainly not base their decisions solely on social science, but they might well be better off if they formed them after the social scientists' voices have been heard.—AMITAI ETZIONI, Columbia University, New York 10027, and Center for Policy Research, Inc., 475 Riverside Drive, New York 10027

^{*}B. Berelson and G. A. Steiner, Human Behavior: An Inventory of Scientific Findings (Harcourt Brace, New York, 1967); J. Rothman, Planning and Organizing for Social Change (Columbia University Press, New York, 1974).



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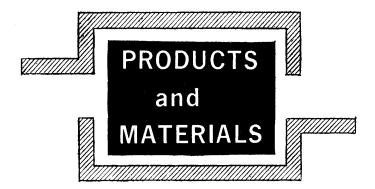
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Literature

Fluorescence News for October 1976 features an article on Protoporphyrin and Protoporphyrin Measurement. American Instrument. Circle 706.

Cell Biology is the latest data book from FASEB. It is available from the Federation at \$45. FASEB. Circle 707.

Laboratory Equipment and Supplies includes a complete line of scientific apparatus for researchers in chromatography, electrophoresis, and spectrophotometry. Manalco Supply. Circle 708.

Atomizer for Atomic Absorption Spectroscopy is devoted to the IL 555 Controlled Temperature Furnace. Instrumentation Laboratory. Circle 709.

Trace Oxygen Analyzer describes a device for the detection of oxygen in carbon dioxide. Thermco Instrument. Circle 710.

Small Computer features the Sol-20, which includes a microprocessor, a 1024-character video display circuit, a memory system, and an 85-key keyboard plus software and other peripherals. Processor Technology. Circle 711.

Immersion Circulators is a 4-page color brochure devoted to two new liquid heat-control devices. Techne. Circle 712.

Antisera is a 24-page catalog of chemicals for radioimmunoassay, affinity chromatography, immunoelectrophoresis, and immunodiffusion. Research Products International. Circle 695.

Cybergraph is an eight-page brochure that describes a system to convert graphics to computer language. Talos Systems. Circle 713.

Cycloaliphatic Epoxides includes structural formulas, applications, and cure chemistry. Union Carbide. Circle 714.

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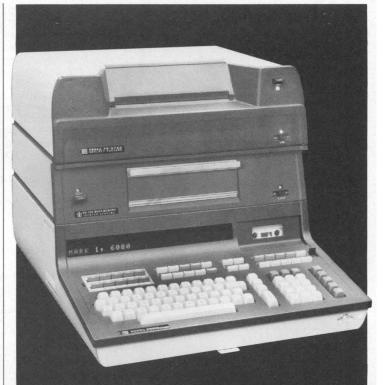


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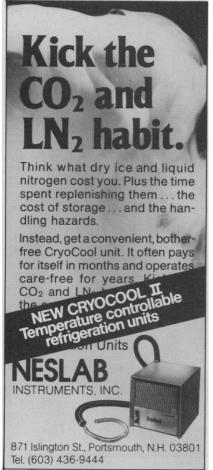
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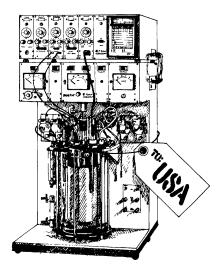


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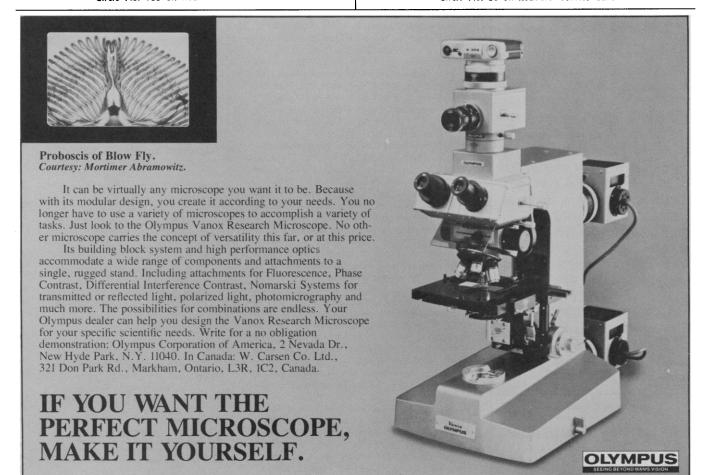


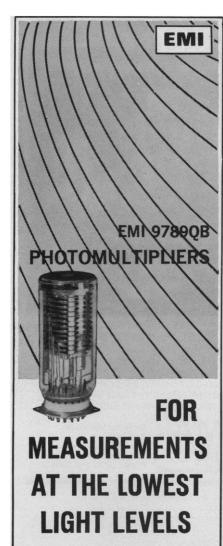
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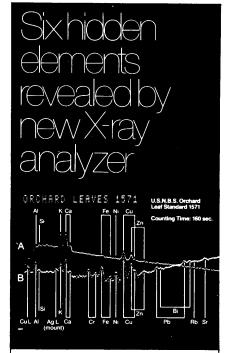
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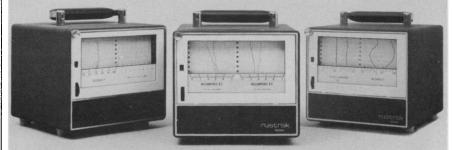
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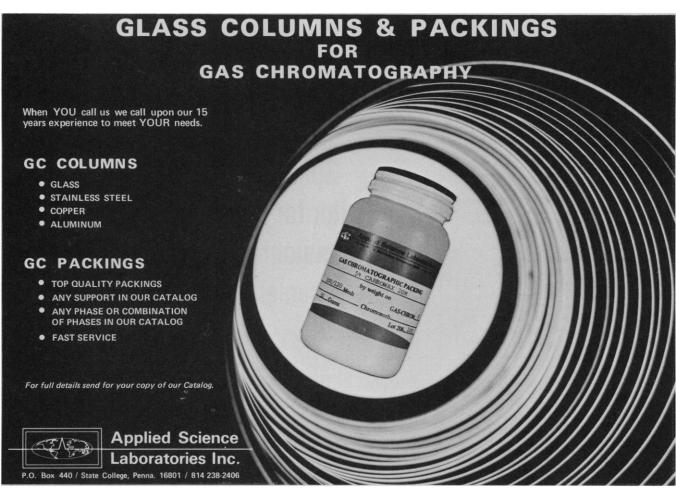
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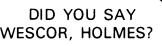


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