

of cancer. We do not understand how locally secreted autacoids, with only a brief half-life, can exert a therapeutic effect on malignant cells (e.g., colon, head and neck, and bone metastases). For example, about 95 percent of infused prostaglandin E₂ is metabolized during one passage through the pulmonary circulation. Is the action of NSAIDs in cancer due to an effect on classical eicosanoids, or are there pathways of essential fatty acid metabolism yet to be identified?

As discussed in the articles by Solomon et al. and Bresalier et al. in this issue of the *Journal*, trials of COX-2 inhibitors as prophylaxis against colonic polyps and colon cancer have been suspended because of an increased incidence of cardiovascular events. An example is the Adenoma Prevention with Celecoxib trial. In this study, patients ingesting 400 mg of celecoxib daily had an increased risk of myocardial infarction and stroke in comparison with patients who received placebo. These complications are driven by activated platelets; celecoxib cancels the platelet-inhibiting action of prostacyclin. However, this action cannot be the only reason for an increased risk of vascular events among patients who took celecoxib, perhaps because many more patients in clinical trials involving more than 1000 subjects should have had a cerebrovascular event. An alternative explanation is that the affected patients represented a subgroup with a low threshold for platelet activation that surfaced only after at least 18 months of treatment with the COX-2 inhibitor. Testing of platelet function in at least a fraction of the patients, to measure dose responses to a variety of agonists, has not been performed in these or other pioneering studies such as the aspirin component of the Physicians' Health Study. Such testing might reveal subgroups of patients with symptoms that could be described as resistance to aspirin or COX-2 inhibitors. Investigators with either a basic or a clinical interest in the metabolism of essential fatty acids and eicosanoid production have all such information at hand in this excellent compendium.

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CORRECTIONS

Multiple Myeloma (October 28, 2004;351:1860-73). On page 1869, in the second full paragraph in the left-hand column, lines 11 through 12 should have read, "a decrease in the levels of the proteins that inhibit apoptosis," rather than "the levels of the proteins that promote apoptosis," as printed.

Acute Pericarditis (November 18, 2004;351:2195-202). On page 2198, in the right-hand column, lines 8 through 13 should have read, "ST-segment elevation also may be seen in patients with an early repolarizing pattern. The most reliable feature in distinguishing pericarditis from early repolarization may be the ratio of ST-segment elevation to T-wave height in lead V₆; when this ratio exceeds 0.24, acute pericarditis is almost always present," rather than, "The most reliable distinguishing feature may be the ratio of ST-segment elevation (in millimeters) to T-wave amplitude (height in millimeters) in lead V₆. When this ratio exceeds 0.24, acute pericarditis is almost always present," as printed.

Rofecoxib, Merck, and the FDA (December 30, 2004;351:2875-8). In the letter by Topol, in Table 1 on page 2878, the 95 percent confidence interval for the number of total deaths and cardiovascular events in study group 090 should have been "1.2–46.3," rather than "1.2–146.3," as printed.

Oral Erythromycin and the Risk of Sudden Death (January 20, 2005;352:301-4). In the letter by Schoenholtz, lines 1 through 4 of the second full paragraph on page 302 should have read, "Between thioridazine and haloperidol are the 'atypical' drugs, which are far more potentiating of cytochrome P-450 inhibitors than haloperidol," rather than "which are far more potent blockers of cytochrome P-450 inhibitors than haloperidol," as printed.

NOTICES

Notices submitted for publication should contain a mailing address and telephone number of a contact person or department. We regret that we are unable to publish all notices received. Notices also appear on the Journal's Web site (www.nejm.org/meetings). The listings can be viewed in their entirety or searched by location, month, or key word.

LEROY D. VANDAM, M.D.: AN ANESTHESIA JOURNEY

Leroy Vandam was a universal man: surgeon, artist, scientist, writer, and anesthesiologist. The Department of Anesthesiology at Boston University Medical Center and Anaesthesia Associates of Massachusetts have published a DVD featuring Dr. Leroy Vandam's narration of his illustrious career and the evolution of anesthesia equipment. Dr. Vandam provides an eloquent and intimate account of his professional experiences and the very nature of anesthesiology in the 20th century. Many historical photographs and film clips of equipment, people, and places accompany the program. The DVD directed by Rafael Ortega, M.D., honors Dr. Vandam's memory and is distributed free of charge. You may e-mail maureen.omalley@bmc.org to request a complimentary copy.

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