ostasis gained clinical attention. Since bronchodilata-
tion and hypokalemia are mediated through the same
type of receptors, one wonders whether any future
adrenergic compound with systemic action could
promise to deliver one without the other. For injecta-
ble brands of terbutaline, the 39th edition of the PDR
(1985) lists transient hypokalemia under the heading,
“Usage in Labor and Delivery.” Hypokalemia is not
cited as an “adverse effect” in the sections on ter-
butaline or epinephrine. Using close cardiac monitor-
ing, one should not worry too much, and let these
bronchodilators do what they do best: dilate bronchi.
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Meet the Press—and Ignore Peer Review

The medical community has long been suspicious of
single-source press interviews. We have learned
that these frequently offer a national forum for self-
aggrandizement of medical cultists. However, a new
public relations phenomenon has become all too com-
mon in recent years. Legitimate investigators, some-
times willingly and sometimes under duress, have
become the primary participants in nationally pub-
lished press conferences. Resultant publicity has pro-
duced confusion and sometimes considerable harm.
False hopes are easily aroused by press releases
which misrepresent the clinical status of unproved
therapy. A new experimental treatment for Alzheimer’s
disease was recently described in the New England
Journal of Medicine. Only four patients were studied
and obviously the study was not a double-blind clinical
trial. The authors concluded that it would be an
appropriate time to do a double-blind placebo-con-
trolled crossover trial. They emphasized that their
study was a preliminary feasibility trial. The medical
center held a press conference and a patient in the
study was made available for an on-camera testimonial.
A national barrage of announcements was followed by
reports on NBC Nightly News, the NBC Today Show;
CBS Morning Reports, ABC's Good Morning Amer-
ica, the MacNeil-Lehrer Report, Cable News Net-
work, Family Circle, People magazine and other
periodicals. Newspaper headlines trumpeted:
“Alzheimer's Treatment Found Successful” and “Scien-
tists Find First Breakthrough Against Alzheimer's.”
Why did the press raise hopes before statistically
meaningful studies were undertaken to evaluate the
new therapy? Perhaps the problem was related to the
fact that there is a dearth of well-trained science
writers. However, the medical center itself bears some
of the responsibility, for it scheduled a formal news
conference to announce the “breakthrough.” An impor-
tant medical center gave a somewhat false impression
about the clinical applicability of research in their
institution.

As economic rivalry among hospitals intensifies,
many have begun to compete aggressively for pub-
licity.1 What the institutions seek to advertise fre-
quently may bear no relation to what is important in
science. The field of organ transplantation and use of
artificial organs has been associated with a torrent of
press conferences throughout the world. The enor-
mous amount of publicity from those conferences may
have caused a shift in research priorities and resultant
neglect of other modes of therapy. Sensationalism is
what sells newspapers and increases the viewing
ratings on television, but these temptations should be
resisted by reputable medical institutions and sci-
centists.

Fortunately, the press as well as the academic
scientific community is becoming more critical of
these practices. A trio of French investigators recently
held a news conference to announce a “breakthrough”
in the treatment of AIDS. They described dramatic
improvement after treatment with cyclosporine in only
two patients and only after one week of therapy! I
applaud the comments of reporters in Newsweek2 who
noted, “Even and his team also ignored a hallowed
scientific cannon—the requirement that experimental
results be reported first to one's peers, not to jour-
nalists. The history of quackery is a history of public
advertising,” said George J. Anas, professor of health

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law at Boston University.”

We recall with sadness and horror episodes such as the staged press conference by a distinguished academician who enthusiastically endorsed the “wonder drug,” krebiozen. We knew that this famed scientist had lost his objectivity when he bypassed traditional methods for determining the validity and clinical significance of new forms of therapy. It was easy to identify the dangers of these misguided efforts and it is not difficult to recognize the publicity ploys of the pseudoscientists or the medical huckster. However, it is tragic that we must now be concerned about legitimate and often distinguished physicians who have been beguiled into circumvention of essential medical peer review. This practice should be criticized by all segments of society. Perhaps it will then be a transient phenomenon.

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Sensitivity of Methacholine Testing in Occupational Asthma

Physicians use the methacholine inhalation test to diagnose asthma in patients with atypical symptoms. But, perhaps because physicians don’t agree among themselves exactly what asthma is, heightened bronchial responsiveness to methacholine is not a specific test for asthma. Townley and his co-workers found that persons with hay fever and those with asthma overlapped when their responses to methacholine were plotted. Patients with chronic bronchitis and emphysema can also react to relatively low doses of methacholine.

There has been general agreement, however, that a person who fails to respond to methacholine does not have asthma. Townley et al., for example, reported positive tests in all their asthmatic subjects. Even so, as reported in this issue of Chest (see page 389) evidence that methacholine testing is not a 100 percent sensitive test for asthma is growing. Chan-Yeung and co-workers have studied asthmatic workers exposed to western red cedar and discovered that, among 16 workers who left the industry and became asymptomatic, eight lost their hypersensitivity to methacholine. Hargrave and co-workers have reported a worker with asthma secondary to exposure to toluene diisocyanate (TDI) who, after two months off work, became asymptomatic, and lost his reactivity to methacholine. With return to work, his responsiveness returned, weeks after delayed asthmatic attacks began. Smith and co-workers reported a similar worker, who had an immediate-type reaction to TDI. Butcher and colleagues, in a publication describing longitudinal study of TDI workers which did not relate testing to current exposures, found three of 11 symptomatic workers did not respond to 25 mg/ml of methacholine.

Now the same researchers who have been following the TDI workers have reported a worker who failed to respond to methacholine despite a positive methylene diphenylisocyanate challenge test. This worker, too, had sporadic exposure to isocyanates. The likelihood of a positive methacholine test in isocyanate asthma, as the authors conjecture, seems to relate to the intensity and duration of work exposure.

Since the precise mechanism by which methacholine produces hyperreactivity is unknown, an explanation of why the test produces variable results in asthmatic workers can only be speculation. Did the described workers receive a sufficient test dose of methacholine? The worker reported by Banks et al inhaled 64 mg/ml of methacholine. Hypersensitivity is generally regarded as a response to less than 8 mg/ml. Can differences in baseline airway caliper or distribution of inhaled methacholine explain the results? Normal pulmonary function prior to methacholine testing and positive specific agent challenges in the reported workers make these explanations unlikely. Do pathologic changes in airway smooth muscle or permeability gradually develop or disappear, depending on work exposures? And does the response to methacholine mirror these changes? Does the type of asthma—immediate, delayed, or dual—reflect the degree of airway injury and likelihood of a positive methacholine test? A longitudinal study, with methacholine testing at frequent intervals and quantification of exposures, is needed to define correlations among worker exposures, symptoms, and methacholine test results.

Because methacholine testing has limited application, symptomatic workers who appeal to their employers for protection from exposures or who apply for compensation due to work-related illness should not be arbitrarily dismissed because of a negative methacholine test. Occupational medicine physicians and others who treat or evaluate workers with airways complaints may need to perform specific agent challenges in workers whose exposure history and symptoms suggest the methacholine test is falsely negative.

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