LETTERS TO THE EDITOR

The Journal welcomes Letters to the Editor. If found suitable, they will be published as space allows. Letters should be typed double-spaced, should not exceed 400 words, and are subject to abridgment and other editorial changes in accordance with Journal style.

CONCUSSION SEQUELAE

To the Editor:

Having recently read the article on the preparticipation sports examination by Fields and Delaney (*Fields KB, Delaney M: Focusing the preparticipation sports examination. J Fam Pract 1990; 30:304–310*), I decided to try to improve the yield on my "sports physicals." The morning after reading the article, I saw a 17-year-old young man for a preparticipation sports examination prior to football season. He had suffered a well-documented grade II concussion several months earlier, and his mother had specifically instructed him to "ask the doctor if that head injury could be a problem this football season." Armed with the article, I actually showed them where it said that "athletes with a previous concussion have a fourfold greater risk of intracerebral hemorrhage." They wanted to know what an intracerebral hemorrhage was, and we had a long discussion about that.

The next day, after lengthy telephone sessions with the boy's worried and confused parents and learning that the high school principal (with whom they had shared my statistic) seemed slightly perturbed with me, I decided to investigate further. After all, I knew that many high school football players suffered concussions, and in 16 years in medicine I had not yet personally seen any intracerebral bleed from a football injury. I called a local neurologist, a neurosurgeon, and the head of a sports medicine clinic. None of them had ever heard of the alleged fourfold risk of intracerebral hemorrhage after concussion. I pulled the article cited by Fields and Delaney,[1] and I was irritated to learn that they had made an error in citing the article. It actually states that "players with a prior history of loss of consciousness had a risk of loss of consciousness four times that of the player without a prior history," and it did not mention any relationship between concussion and intracerebral hemorrhage. I telephoned one of the authors of that article, who was surprised to learn of the misquotation. After apologizing to the family (they were very understanding), I became angry. I had misled a family because of an error in an article, and I felt that I could no longer believe anything in that article. Here I don't mean to single out Fields and Delaney, but authors have a responsibility to thoroughly check each and every "fact" that they present, and editors of medical journals also must ensure that "facts" and citations from other literature are accurate. Clinicians are lucky to find the time to read articles carefully, and they should not need to personally check out the veracity of seemingly authoritative writings.

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Reference

1. Gerberich SG, Priest JD, Boen JR, et al: Concussion incidence and severity in secondary school varsity football players. Am J Public Health 1983; 73:1370–1375

The preceding letter was referred to Dr Fields and Ms Delaney, who respond as follows:

Dr Soman is correct that in our paper "Focusing the Preparticipation Sports Examination"[1] we made an error in citing Gerberich et al [2] on concussion in high school football players. We are convinced, however, that athletes who have a history of head trauma or continuing postconcussive symptoms or who have had recurrent mild head injuries are a special risk group. To underscore our point accurately, we should have written the following: "Gerberich et al, using data from 3063 secondary school football players in Minnesota, emphasize that athletes with a previous loss of consciousness have a fourfold greater risk of subsequent loss of consciousness.[3] Based on recent CT and MRI research,[4,5] this suggests to us the possibility of more serious injury including intracerebral hemorrhage."

The following paragraphs more fully explain the reasons for our cautionary stance.

The section on neurological injury in our paper was intended to increase the awareness of primary care physicians about two areas of concern: the additive risk of recurrent concussions and the uncertainty about the diagnosis of concussion.

In an article entitled "The Cumulative Effect of Concussion,"[3] Gronwall and Wrightson state, "Doctors do have a duty to convince the controlling bodies and participants in sports where concussion is frequent, that the effects are cumulative, and that acceptance of concussion injury, though gallant, may be very dangerous." Similarly, in a round-table discussion on concussion in athletes, Rimel et al[6] make note of the increased risk of repeated concussions and state, "The consequences of repeated injury are often worse, even if the impact is the same." In a case report entitled "The Second Impact in Catastrophic Contact-Sports Head Trauma,"[7] Saunders and Harbaugh report a case in which a college football player returned to activity following the resolution of an apparently minor concussion only to experience a fatal intracerebral bleed. They make the following statement: "The common resumption of contact play soon after a concussion suggests that sequential minor impacts may occasionally lead to major cerebral pathologic conditions."

Before the advent of computed tomography (CT) and magnetic resonance imaging (MRI), many of the conclusions regarding the detrimental

continued on page 668

continued from page 588

effect of repeated "concussions" were based on serial neuropsychological testing and documentation of symptoms persisting for many months following injury. The case reported by Saunders and Harbaugh[7] effectively underscores the point that concussion has been used as a clinical term implying minor head injury. Without CT or MRI confirmation, however, an athlete who has been rendered unconscious even briefly cannot confidently be advised that he has only a minor condition.

This leads to our second point, the uncertainty about the diagnosis of concussion. Recent studies suggest that apparently mild head injuries, traditionally labeled "concussion," are accompanied by pathological changes on CT or MRI. Levin et al[8] found that patients with mild head injuries, as measured by the Glasgow Coma Scale, had numerous abnormal CT and MRI findings. Without radiologic studies, these cases could be misdiagnosed as simple concussions, (ie, brief alteration or loss of consciousness without focal neurological injury), since 11 of 12 patients were able to obey commands within 12 minutes of the injury. Lehman and Ravich[9] note, "Frequently, a serious acute closed head injury is produced without gross anatomic disruption, but with subtle but highly significant anatomic changes seen at a microscopic level." One specific type of injury identified by CT or MRI, cortical contusion, can be mistaken for a less serious problem, since small contusions do not severely impair initial consciousness. Approximately 70% of frontal lobe contusions[4] and over 50% of all cortical contusions, however, are hemorrhagic.[4,5]

Based on this information, we suggest careful evaluation of the acutely injured athlete with any but the most minor head injury. Fortunately, team physicians no longer have to rely strictly on clinical findings to assess a closed head injury. We agree with the recommendations of Bruno et al,[10] who state that "any athlete who suffered loss of consciousness from head injury for more than one minute, . . . or who has more than one episode of unconsciousness, however momentary, during any one playing season, should be referred for neurological examination and CT evaluation." For the athlete who comes for a preparticipation examination and relates a history of head injury during a previous season, we recommend careful questioning to detect any postconcussive symptoms and a full neurological examination. If he has persistent symptoms or an abnormal examination, neurological consultation and further evaluation are appropriate. If not, a return to full sports activity is allowed.

We apologize for our error in citation and hope that it will not dissuade physicians from carefully screening for a history of concussion. Careful evaluation of athletes with a positive history of head injury seems essential to us for the detection of rare but potentially serious problems.

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2. Gerberich SG, Priest JD, Boen JR, et al: Concussion incidence and severity in secondary school varsity football players. Am J Public Health 1983; 73:1370–1375
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7. Saunders RL, Harbaugh RE: The second impact in catastrophic contact-sports head trauma. JAMA 1984; 252:538–539
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9. Lehman LB, Ravich SJ: Closed head injuries in athletes. Clin Sports Med 1990; 9:247–261
10. Bruno LA, Gennarelli TA, Torq JS: Management guidelines for head injuries in athletics. Clin Sports Med 1987; 6(1):17–29

Editor's note:

Dr Soman's tale is the sort editors dread, all the more so because of its utter inevitability. Fields and Delaney's article was reviewed by two experts in sports medicine and by a substantial editorial staff; none caught the error.

The authors' lengthy response, however, suggests that a simple correction of the citation error falls short of conveying the potential seriousness of postconcussion sequelae. Readers should carefully evaluate their current practice in examining and counseling athletes with head injury.

Errors of attribution occur in the best journals.[1] We are individually responsible, not for perfection, but for writing, reviewing, and editing with meticulous professionalism. Perhaps the citation error should have been caught, but in this case it resulted in a more complete description of the issues, in my view just the sort of interchange that should characterize a good medical journal.—*A. O. Berg, MD, MPH, Interim Editor*

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1. Yankauer A: The accuracy of medical journal references. CBE Views 1990; 13(2): 38–42

ERYTHROMYCIN TOLERANCE

To the Editor:

I would like to take strong exception to the conclusion of a recently published study in the Journal,[1] which stated, "Enteric coating of erythromycin base offers little protection from the common dose-related gastrointestinal adverse effects of oral erythromycin." The authors compared the gastrointestinal side effects of a nonenteric preparation (Erythromycin Base Filmtab, Abbott) with those of an enteric-coated prepara-

tion (Eryc, Parke-Davis) at two dosage levels, under the condition that both preparations be taken on an empty stomach despite their recognition that studies "have documented unaltered bioavailability of enteric-coated pellets of erythromycin base in the presence of food and markedly decreased absorption of nonenteric-coated erythromycin base." Furthermore, they admitted that their methodology "may have added negative bias against the enteric-coated product." Although blood levels of erythromycin are above the minimum inhibitory concentrations required to be effective against most organisms for which their product is indicated, the manufacturers of Eryc still recommend that it be administered on an empty stomach because even higher levels may be obtained when the product is taken in that state (package insert, Eryc, Parke-Davis, February 1988). Erythromycin blood levels of other enteric-coated erythromycin bases, however, are not affected by food,[2] and the manufacturer of one such product recommends that it may be taken without regard to meals (package insert, Ery-Tab, Abbott, September 1987). Another enteric-coated erythromycin base that is equivalent to Ery-Tab (E-Mycin) may also be taken without regard to meals. My own clinical experience with various erythromycin preparations over more than 15 years is that to recommend taking any erythromycin preparation on an empty stomach is to ask for trouble.

Because of the foregoing considerations, the study of Ellsworth et al, although methodologically sound, draws conclusions that are overgeneralized, probably incorrect, and possibly detrimental to patient care. They proved that Eryc, given on an empty stomach, offers no advantage over Erythromycin Base Filmtab; to generalize this result to all other enteric-coated preparations is not only unjustified, but potentially harmful to patients, should their physicians, reading only the abstract of Ellsworth et al, be influenced to change their prescribing in favor of the nonenteric-coated preparations taken on an empty stomach. Before their conclusion can be taken seriously, the authors need to repeat their experiment to include an enteric-coated base such as Ery-Tab or E-Mycin taken with meals.

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1. Ellsworth AJ, Christensen DB, Volpone-McMahon MT: Prospective comparison of patient tolerance to enteric-coated vs nonenteric-coated erythromycin. J Fam Pract 1990; 31:265–270
2. Macrolides and lincosamides. In Drug Evaluations, ed 6. Chicago, American Medical Association, 1986, p 1399

The preceding letter was referred to Dr Ellsworth and colleagues, who respond as follows:

We appreciate the opportunity to comment on Dr Hahn's letter. Erythromycin is commonly used in clinical practice, and gastrointestinal side effects are frequently reported. Erythromycin stimulates smooth muscle and gastrointestinal motility. Indeed, Janssens et al[1] successfully utilized erythromycin's promotility properties to treat patients with diabetic gastroparesis. Pharmaceutical manufacturers promote various salts and enteric coatings in an effort to decrease side effects and preserve adequate blood levels (erythromycin base is quite acid labile).

Our study reconfirms that gastrointestinal adverse effects are common with erythromycin and that they are dose related. An enteric-coated erythromycin base capsule offered little protection from these adverse gastrointestinal effects in a prospective comparison of 368 patients utilizing data from two telephone interviews and a side effect diary.[2]

Many clinicians believe that enteric-coated products reduce gastrointestinal side effects despite evidence that dose-related gastrointestinal reactions occur even with intravenous administration of erythromycin.[3] These gastrointestinal adverse effects

are the result of systemic blood levels, not local irritation amenable to buffering with food or an enteric coating. We instructed patients to take both enteric-coated and nonenteric-coated erythromycin products four times daily on an empty stomach, 30 to 60 minutes before meals, based on manufacturer specifications at the time of study. Despite extensive verbal and printed instructions, 30% to 40% of our subjects took their erythromycin with food. Though there were no differences in this regard between groups, the prevalence of this noncompliance may have added a negative bias against the enteric-coated product. Enteric-coated erythromycin base provides unaltered bioavailability compared with nonenteric-coated erythromycin base in the presence of food, as pointed out by Dr Hahn. Hence, higher systemic blood levels may be expected with enteric-coated products if administered concurrently with food. In our study, side effects were prominent in both groups, we feel, minimizing the above-mentioned potential bias. Post-hoc analysis of subsets of patients taking each product and dosage level with and without food was not possible with the collected data.

It was not the purpose of our study to compare erythromycin enteric coatings, but rather to compare enteric-coated versus nonenteric-coated erythromycin while controlling for dose. Enteric coating allows a preferential, delayed release of drug in the intestine where the pH is neutral, rather than in the stomach where the pH is acidic. A number of variables affect dissolution. Certainly differences may exist between enteric-coated products. This research is left to others. Investigators at the University of North Carolina have completed a pilot study using PCE Dispertab (Abbott), another enteric-coated product. Perhaps their data will help generalization of our results. We approached both Abbott and Upjohn unsuccessfully regarding study participation before receiving support from Parke-Davis.

We feel our results support our conclusions that enteric coating of erythromycin offers little protection from the common, dose-related gastrointestinal adverse effects of erythromycin.

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2. Ellsworth AJ, Christensen DB, Volpone-McMahon MR: Prospective comparison of patient tolerance to enteric-coated vs nonenteric-coated erythromycin. J Fam Pract 1990; 31:265–270
3. Putzi R, Blaser J, Luthy R, et al: Side-effects due to the intravenous infusion of erythromycin lactobionate. Infection 1983; 11:161–163

CLINICAL PHARMACY IN FAMILY PRACTICE

To the Editor:

In their September 1990 article Shaughnessy and Hume have presented descriptive data on the current state of clinical pharmacist activity in family practice residency programs.[1] While the authors conclude that pharmacy involvement in family practice is "alive and well," there are many plausible explanations for this phenomenon. The readiness of the authors to cite as a reason the "acknowledgment by medical educators in family practice of the value of clinical pharmacists in residency programs" conveys a sense of self-advocacy that is troubling. In fact, acknowledgment by medical educators of the impact of clinical pharmacists on family practice education has not been addressed in the literature in an unbiased, comprehensive manner. Published articles cite modification of reductionistic educational endpoints,[2–4] and also cite generally positive perceptions[5–7] of educational impact. One example of this genre has been thoughtfully criticized in a published editorial,[8] but the serious reservations raised have been largely ignored within the family practice literature. The existing descriptive and experimental work simply does not adequately describe the whole phenomenon of placing a pharmaceutical-limited specialist within a family practice program.

Any placement of a limited specialist within a family practice program requires critical appraisal. It is simplistic to believe that the lessons learned from the specialist consist only of their spoken word. The most powerful lessons may derive from the very selection of a specialist to be the assigned educator. The ability to prescribe medications in a rational and cost-effective manner is an essential skill for all family physicians. The use of a limited specialist to train the resident carries a limiting meta-message. In the case of the clinical pharmacist, the message is that a family physician cannot expect to achieve the skill of appropriate medication prescribing without specialization. Conversely, the use of a family physician to teach prescribing skills will teach the resident both the skill and the conviction that the skill is attainable. Use of a physician in this role was advocated in Melmon and Blaschke's editorial on grounds of economics as well adequacy of background in effective medical judgment.[8]

The interaction of the unique limitations of residents in training and of clinical pharmacists must be addressed. Residents have limitations in knowledge, experience, and confidence—and yet are able to prescribe medications, diagnose ailments, and initiate therapy. Clinical pharmacists in academics are knowledgeable, experienced in their field, and confident—and *must never* prescribe medications, diagnose ailments, or initiate therapy. It has not been addressed in the literature that when clinical pharmacists act as preceptors to residents early in their training, the pharmacists indeed do initiate treatments and prescribe medications by proxy. This phenomenon has not been addressed

in practice to any greater degree than it has been addressed in print. It is in fact nearly impossible to prevent prescription by proxy from occurring, even when interactions of clinical pharmacist and resident are also supervised by an attending physician.

The ability of clinical pharmacists to obtain funding from external sources has certainly aided the rapid growth in their representation within family practice residency programs. This rapid growth should not be interpreted as resulting from comprehensive assessment of the true impact of clinical pharmacists within these programs. Family practice programs should assess, in an unbiased and comprehensive fashion, the impact of having this particular limited specialty present in house. Clinical pharmacists should address the legal and ethical constraints on their activities as they work with residents in training, both in the literature and directly with the residents whom they help to train.

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7. Johnston TS, Heffron WA: Clinical pharmacy in family practice residency programs: J Fam Pract 1981; 13:91–94
8. Melmon KL, Blaschke TF: The undereducated physician's therapeutic decisions: N Engl J Med 1983; 308:1473–1474

The preceding letter was referred to Drs Shaughnessy and Hume, who respond as follows:

Dr Corey is correct in stating that the literature evaluating the usefulness of clinical pharmacists in family practice residencies is limited. Although many aspects of clinical pharmacy practice have been addressed, the actual impact on residency education over the long term and on patient outcomes in the short term has not been investigated adequately. We are very interested in this question and currently are seeking funding for a comprehensive project that would evaluate the clinical pharmacist's influence on residency training and on actual patient outcomes such as blood pressure control. Unlike previously published studies, however, we plan to address these issues in a "real world" setting in which only one clinical pharmacist is available.

We would, however, disagree with Dr Corey in his premise that clinical pharmacists convey the message "that a family physician cannot expect to achieve the skill of appropriate medication prescribing without specialization." In actuality this statement addresses the broader issue of specialists teaching in family medicine residency programs and is no more true for clinical pharmacists than it is for any other "limited specialists" (sic) teaching their specialty within the family practice curriculum. Pediatricians, psychologists, obstetricians, cardiologists, nutritionists, and other specialists are involved in teaching their specialty knowledge and approach to patient care to family practice residents. The ethnocentric view (ie, only family physicians can teach family physicians) advanced by Dr Corey is held by members in all disciplines. Fortunately, it is specialty teaching that permits "cross-fertilization" and prevents a profession from succumbing to the danger associated with this type of intellectual "inbreeding."

We also disagree with Dr Corey's

second premise that clinical pharmacists initiate treatment and prescribe by proxy. This authoritative approach to teaching does little to teach the decision-making skills necessary for appropriate use of medications but does allow the learner to learn without "knowing." For many years pharmacists who wish to "prescribe" have had opportunities to do so in settings such as Veterans Administration hospital ambulatory care clinics,[1] the University of Southern California Pharmacist-Prescriber Project,[2] private family practice offices,[3] the Indian Health Service,[4] or the states of Florida,[5] Washington,[6] California,[7] and Mississippi.[8] In the setting of a residency program, however, clinical pharmacists, as well as all other clinical teachers, derive satisfaction from providing the appropriate information so as to permit the student (eg, resident) to problem-solve the initial and future patient-oriented questions. This end, after all, is the ultimate goal of clinical teaching.

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Anne L. Hume, PharmD
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3. Davis RE, Crigler WH, Martin H: Pharmacy and family practice: Concept, roles and fees. Drug Intell Clin Pharm 1977; 11:616–621
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5. Fla Stat § 465, 186 (1985)
6. Wash Rev Code § 18 64.011 (11)
7. Cal Bus & Prof Code § 4046 (c) (4) (West)
8. Miss Code Ann § 73-21-73 (m), § 73-21-73 (r)

PSEUDOCYESIS

To the Editor:

I read with interest the article entitled "Pseudocyesis" in the May 1990 issue of the Journal (*Paulman PM, Sadat A: Pseudocyesis. J Fam Pract 1990; 30:575–576*). The epidemiology is of particular interest.

I recently encountered a case in the family practice setting.[1] Zuber and Kelly, also family physicians, noted three cases in a 1-month period in 1984,[2] and there have been other cases cited in recent literature.[3–5] It is my contention that the increases in the incidence of pseudocyesis reflect mounting societal pressures on women to become pregnant. Some of these pressures being guilt following repeated abortion or miscarriage, sexual abuse, and problems stemming from infertility, especially in the perimenopausal woman who has delayed pregnancy.

Family physicians must be alert for this diagnosis in the differential diagnosis of secondary amenorrhea. It warrants diligent follow-up and counseling to prevent recurrence and further distortion of reality or severe depression.

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2. Zuber T, Kelly J: Pseudocyesis. Am Fam Physician 1984; 30(5):131
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