Examination of the right upper extremity showed zero deltoid action and minimal triceps muscle action. The rotator cuff was good; elbow and wrist flexors and finger motion were normal. There was no sensory loss. A diagnosis of brachial plexus contusion involving C5 and C6 of the posterior cord was made. Therapy was conservative, the arm was put in a supporting sling. The patient was discharged on the seventh postoperative day with obvious improvement in shoulder function. Examination two years after operation showed good recovery, although the patient complained of some shoulder weakness. The impairment did not interfere with the patient's normal routine.

The pathogenesis of brachial-plexus injury during anesthesia has been considered by many. Stretch or compression of the brachial plexus associated with malposition of the body during anesthesia is responsible for many of the reported neuropathies. We can only speculate on the possible causes of brachial-plexus damage in this patient. It is possible that the intramuscular injection of the premedicating drugs in the right arm injured the axillary or radial neurons.

This appears improbable, as the patient had no pain or paresthesia. A congenital anomaly of the cervical vertebrae could produce compression of C5 and C6 cords with moderate hyperextension of the head. There is no support for this in the patient's history. Finally, a member of the surgical team could have rested on the patient's right shoulder. Continuous downward shoulder pressure in a paralyzed patient could produce posterior displacement of the humeral head or clavicle, with prolonged stretching of the brachial plexus and consequent damage. This suggestion is, of course, pure speculation.

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Prevention of Anaphylaxis from Contrast Media

To the Editor:—Drs. Millbern and Bell suggest that pretreatment with steroids and diphenhydramine should be considered prior to giving radiopaque contrast agents to patients with previously documented sensitivity to these agents to avoid anaphylactic responses. However, we have recently found that pretreatment with these agents failed to prevent an anaphylactic response to contrast medium. The patient was a 60-year-old white man who complained of increasing claudication in both legs, and for whom aortic angiography was planned. He had a well-documented history of anaphylactic responses, including cardiac arrests on two occasions when he had been given intravenous pyelogram dye. In preparation of the angiographic study, he was hospitalized and received a five-day course of prednisone, 20 mg, and diphenhydramine, 50 mg, orally, twice daily. On the morning of angiography, and with informed consent, he was premedicated with prednisone, 50 mg, and diphenhydramine, 50 mg, orally, and he was given methylprednisolone, 100 mg, and diphenhydramine, 25 mg, intravenously on arrival in the angiography suite. Monitors included an electrocardiogram, precordial stethoscope, blood pressure cuff, and transduced arterial waveform obtained from the femoral arterial catheter to be used for the angiography. The patient was sedated with diphenhydramine, 75 mg, morphine, 15 mg, and diazepam, 10 mg, intravenously, and was sleepy but easily rousable. Vital signs were pulse, 70 beats/min, blood pressure, 150/100 torr, respiration rate, 18/min, with spontaneous respirations. A test injection of Renografin-76® contrast material, 10 ml, resulted in no change in vital signs. Angiography of the abdominal aorta and both legs was then performed with a single mechanism injection of 75 ml of the same contrast agent. Immediately after the injection, the pulse decreased to 50 beats/min, blood pressure decreased to 60/20 torr, and the patient became very flushed. Marked bronchospasm, tachypnea, and dyspnea were present. The patient remained conscious and complained of severe generalized burning and pain. He was successfully resuscitated with intravenous fluids and epinephrine. Four hours after the incident he had completely recovered.

Pretreatment with methylprednisolone just prior to challenge failed to prevent the anaphylactic response in both our patient and Dr. Millbern's patient, even in conjunction with diphenhydramine therapy. For our patient the five-day course of orally administered prednisone also apparently had little or no effect. These experiences and other reports suggest that pretreat-
ment with very large doses of steroids, such as methyl- 
prednisolone, 1 g, intravenously, may be effective in 
preventing an anaphylactic response, whereas smaller 
doses may not be effective. These experiences also 
suggest that the optimal time for pretreatment is half 
an hour to several hours prior to challenge. Intra-
venous administration of diphenhydramine does not 
appear to be effective in preventing the anaphylactic 
response, although it may be helpful in decreasing 
the severity of the response. Of course, other 
unknown situational factors may be very important in 
preventing anaphylaxis, and it is not possible to de-
termined these factors from small numbers of anecdotal 
reports such as these. Both Drs. Millbern and Bell's 
report and our experience underscore the importance 
of having suitably trained personnel in attendance in 
situations where an anaphylactic response is likely or 
expected. Proper preparation for the eventuality and 
prompt, appropriate intervention can markedly affect 
the eventual outcome.

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Averaging pH vs. H+ Values

To the Editor:—In a recent letter to the editor, 
Giesecke1 criticized statistical methods used by Stoel-
ing2 in reporting gastric-fluid pH changes following 
several preanesthetic medication regimens. Stoelting 
measured pH in gastric aspirates and derived mean 
and standard deviation values. Giesecke claimed that 
[pH] must first be converted to a real number, then 
statistically manipulated, and finally reconverted to 
pH form. Although details of the transformation were 
not given, it would appear Giesecke meant one should 
convert the pH to a derived hydrogen ion concen-
tration ([H+]), average, take the negative logarithm, 
and call the result the average pH. He maintained 
that only a real number can be measured and that pH, 
being a logarithmic transformation of a real number is 
not real. (Parenthetically, a logarithmic transformation of a 
real number is most assuredly also a real number.) We believe that 
Giesecke is in error, and fear that acceptance of his 
letter by the editors of Anesthesiology might reflect 
a new standard for the review of statistical procedures 
involving pH.

Both Stoelting and Giesecke seem to implicitly 
accept pH as the expression of gastric-fluid acidity. We 
agree with them. Although many have called for the 
abolition of pH notation and for the use instead of 
a derived [H+] in describing acidity, a consider-
ation of thermodynamics applied to biologic systems 
confirms the superiority of pH over [H+] in relating 
acidity to physiologic function. Although pH was 
originally defined as pH = log 1/[H+], pH is now 
accepted as the measure of acidity without regard to 
that definition. pH is an independently determined 
variable; [H+] is a derived, dependent variable. Within 
certain tight constraints, it still remains true that 
pH = −log aH+, where aH+ = γ [H+] (aH+:hydrogen 
ion activity; γ: activity coefficient). It is likely that 
most physiologic processes affected by hydrogen ion 
respond in a manner proportional to the logarithm of 
the hydrogen ion activity.

A series of pH measurements can be summarized 
by a sample mean and sample standard deviation. It is 
erroneous to take the antilog of the pH, invert, 
average, take the negative logarithm of the average, 
and call this number the mean pH.7 Let us consider 
a simple example. Given two samples of gastric fluid 
of equal volumes with pH 1 and 6, the mean pH is 3.5. 
When Giesecke's method is used, the following cal-
culations have to be made. First, the pH values are con-
verted to [H+]; thus, pH 1 yields [H+] = 10^-1 mol/l 
and pH 6 gives [H+] = 10^-6 mol/l. Next, the average of