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 pathogeneses 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 premedicant 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.[1] 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 mechanized 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[2,3] suggest that pretreat-

ment with very large doses of steroids, such as methylprednisolone, 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. Intravenous administration of diphenhydramine does not appear to be effective in preventing the anaphylactic response,[4-6] although it may be helpful in decreasing the severity of the response.[7] Of course, other unknown situational factors may be very important in preventing anaphylaxis, and it is not possible to determine 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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REFERENCES

1. Millbern SM, Bell SD: Prevention of anaphylaxis to contrast media. ANESTHESIOLOGY 50:56-57, 1979
2. Miller WL, Doppman JL, Kaplan AP: Renal arteriography following systemic reaction to contrast material. J Allergy Clin Immunol 56:291-295, 1975
3. Zweiman B, Mishkin MM, Hildreth EA: An approach to the performance of contrast studies in contrast material-reactive persons. Ann Intern Med 83:159-162, 1975
4. Patterson R, Schatz M: Administration of radiographic contrast medium after a prior adverse reaction. Ann Intern Med 83: 277, 1975
5. Kelly JF, Patterson R: Anaphylaxis: JAMA 227:1431-1436, 1974
6. Schatz, M, Patterson R, O'Rourke J, et al: The administration of radiographic contrast media to patients with a history of a previous reaction. J Allergy Clin Immunol 55:357-366, 1975
7. Morrow DH, Luther RR: Anaphylaxis: Etiology and guidelines for management. Anesth Analg (Cleve) 55:493-499, 1976

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Averaging *p*H *vs.* H^+ Values

To the Editor:—In a recent letter to the editor, Giesecke[1] criticized statistical methods used by Stoelting[2] in reporting gastric-fluid *p*H changes following several preanesthetic medication regimens. Stoelting measured *p*H in gastric aspirates and derived mean and standard deviation values. Giesecke claimed that *p*H must first be converted to a real number, then statistically manipulated, and finally reconverted to *p*H form. Although details of the transformation were not given, it would appear Giesecke meant one should convert the *p*H to a derived hydrogen ion concentration ($[H^+]$), average, take the negative logarithm, and call the result the average *p*H. He maintained that only a real number can be meaned and that *p*H, being a logarithm, 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 *p*H.

Both Stoelting and Giesecke seem to implicitly accept *p*H as the expression of gastric-fluid acidity. We agree with them. Although many have called for the abolition of *p*H notation and for the use instead of a derived $[H^+]$ in describing acidity,[3-5] a consideration of thermodynamics applied to biologic systems confirms the superiority of *p*H over $[H^+]$ in relating acidity to physiologic function.[6,7] Although *p*H was originally defined as $pH = \log 1/[H^+]$, *p*H is now accepted as the measure of acidity without regard to that definition.[7] *p*H is an independently determined variable; $[H^+]$ is a derived, dependent variable. Within certain tight constraints, it still remains true that $pH = -\log a_{H^+}$, where $a_{H^+} = \gamma\ [H^+] \cdot (a_{H^+}$: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.[6]

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