Aspirin and Catheter Size

To the Editor:—Bedford and Ashford\(^1\) recently demonstrated that aspirin pretreatment significantly decreased the incidence of vascular occlusion following cannulation of a radial artery with an 18-gauge catheter. Previously, Bedford\(^2\) had documented that a similar decrease was afforded by the substitution of a 20-gauge for an 18-gauge cannula in patients receiving aspirin. Despite the findings that "the incidence of post-cannulation radial arterial occlusion can be decreased significantly by using 20-gauge cannulas instead of 18-gauge cannulas,"\(^3\) Bedford and Ashford chose to employ the potentially more hazardous larger catheter because "it was necessary to examine the efficacy of aspirin pretreatment specifically in the situation where cannulas occupy a high proportion of the vessel lumen."

I believe this experimental design raises important issues. The authors documented no advantage to using the 18-gauge catheter. Indeed, it would appear that it was used solely to produce a sufficiently high incidence of thrombosis to facilitate statistical comparison of the placebo and aspirin groups. Thus, it is not surprising that both studies\(^4,5\) showed equally high incidences of arterial occlusion when patients monitored with an 18-gauge cannula did not receive aspirin (table 1, Groups II and III). Were the patients (as well as the Committee for the Protection of Human Subjects in Research) informed specifically that one of the authors had already shown that 50 per cent of the subjects would receive a treatment (18-gauge catheter without aspirin) that was associated with a potentially high risk of complications? Further concern arises from the observation that there is no significant difference in the incidences of arterial occlusion when the 20-gauge (no aspirin) and 18-gauge (aspirin) groups are compared (table 1, Groups I and IV).

Bedford has already demonstrated that the incidence of arterial occlusion is more than 30 per cent when more than 50 per cent of the vessel lumen is occupied by a 20-gauge catheter.\(^2\) Although these individuals comprise only a fraction of the total population, they are the ones at risk when a 20-gauge cannula is used. Obviously, they are also the individuals in whom the use of aspirin might have been expected to have produced a statistically significant beneficial effect. Because the authors obtained arteriograms prior to decannulation, these patients could have been readily identified.

In summary, I believe that a far more appropriate experimental design would have substituted a 20-gauge cannula for the one the authors used. This would have avoided the increased risk (with no possible benefit) to patients with radial arteries of large diameter. Patients with small vessels receiving the placebo would have sustained no additional increase in risk related to the study. Finally, patients with small vessels receiving aspirin might actually have benefited from the investigation.

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REFERENCES


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In reply:—Dr. Cohen's letter raises three specific issues: Were the patients informed of a high risk of complications? Is there a benefit to using 18-gauge radial-artery catheters? Is patient safety increased by using 20-gauge catheters?

Both the local committee for the protection of hu-
man subjects in research and all patients in the study were informed specifically that the technique of radial-artery cannulation used often resulted in vascular occlusion, and that there were ways to prevent this from occurring (i.e., small catheters, short duration of cannulation). The patients also knew that they could choose one of these options if they so desired. In fact, however, since we began documenting ulnar artery blood supply to the hand by the presence of retrograde pulsatile Doppler signals in the radial artery to be cannulated, we have not seen any instance of distal ischemic complications resulting from 338 elective cannulations performed with Teflon® 18-gauge catheters. In addition, despite the difference between incidence of asymptomatic radial-artery thrombosis with 18- and 20-gauge catheters, we have found no difference in the incidences of symptomatic complications (i.e., necrosis of skin over the catheter). From the patients' point of view, the advantage of using 18-gauge catheters is that they do not kink in situ and become dysfunctional during major operative procedures, as do 20 per cent of 20-gauge catheters. When 18-gauge catheters become dysfunctional due to accumulation of thrombotic material, it usually occurs hours after the completion of the surgical procedure. We felt that the greatest risk to the patients in this study was not related to radial-artery cannulation, but rather was the 1.7 per cent incidence of anaphylactoid reactions associated with injection of radiographic contrast medium. If we had pursued Dr. Cohen's suggested experimental design using 20-gauge catheters, approximately three times as many patients would have been exposed to arteriography to achieve the same result.

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References
5 Bedford RF: Radial arterial function following percutaneous cannulation with 18- and 20-gauge catheters. Anesthesiology 47:37–39, 1977
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Anesthetic Mortality

To the Editor:—I read with great interest the article of Dr. Arthur Keats, and the editorial of Dr. William Hamilton. Dr. Keats supported the case for "unavoidable" adverse actions of drugs as an important cause of anesthetic mortality, while Dr. Hamilton believes that 90 per cent of anesthetic deaths may be attributed to management errors.

Is management error a bias or a fact? In a trial to answer this important question, 36 cases of cardiac arrest in our hospital attributable to general anesthesia were analyzed (table 1). The analysis revealed three groups of patients: in Group I (14 cases), management error was considered the primary cause of cardiac arrest; in Group II (12 cases), an adverse reaction to a drug or a technique was a contributing factor, although we could not entirely exclude management error; in Group III (10 cases), cardiac arrest was attributed to the patient's poor condition, and we believed that both management errors and adverse reactions could be excluded as causes.

Table 1. Causes of Cardiac Arrest in 36 Cases

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<th>Group I, management errors (14 cases)</th>
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<tr>
<td>1. Technical failure, e.g., esophageal intubation</td>
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<td>2. Machine failure, e.g., N₂O instead of O₂, leaking flowmeters</td>
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<tr>
<td>3. Drug overdosage (anesthetic and nonanesthetic)</td>
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<tr>
<th>Group II, adverse reaction to a drug or a technique with or without management errors (12 cases)</th>
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<tr>
<td>1. Adverse reaction to a drug e.g., hypersensitivity reactions, interaction of a drug with the disease of the patient</td>
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<tr>
<td>2. Complications of a technique e.g., regurgitation and aspiration during crash induction, hypertensive technique</td>
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<th>Group III, poor condition of the patient (10 cases)</th>
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<td>Exsanguination, amniotic fluid embolism, uncontrolled heart failure, etc.</td>
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From this analysis I agree with Dr. Keats that we must rid ourselves of the "error bias," but unfortunately, a management error is still a common and preventable cause of anesthetic cardiac arrest.