Effect of Aspirin on Brachial Artery Occlusion following Brachial Arteriotomy for Coronary Arteriography

By Kieran M. Hynes, M.D., Gerald T. Gau, M.D., Barry D. Rutherford, M.D., Francis J. Kazmier, M.D., and Robert L. Frye, M.D.

SUMMARY
The effect of oral aspirin in doses known to influence in vitro platelet aggregation was evaluated in patients undergoing brachial artery catheterization. Neither arterial thrombus formation nor pulse reduction was affected in patients given aspirin when compared to controls. Aspirin in small doses does not decrease the incidence of vascular occlusion after brachial artery catheterization.

Additional Indexing Words:
Antithrombotic agents  Arterial occlusion
Arteriotomy complications  Aspirin

VASCULAR occlusion, frequently with thrombus formation, is the primary complication of brachial arteriotomy after brachial artery catheterization, reaching an incidence as high as 24% in recent studies.1,2 The development of platelet antiaggregating agents (of which aspirin is one) and our previous experience with complications of brachial artery catheterization prompted us to undertake a prospective study to observe the effects of aspirin on arterial occlusion occurring with brachial artery catheterization for coronary arteriography.

Methods
A consecutive series of 150 patients (125 males, 25 females) referred for coronary angiography or angio- graphic visualization of aortocoronary bypass grafts formed the basis of this report. Patients were informed of the nature of the study. Patients were excluded from the study for the following reasons: refusal to participate in the study, symptoms suggestive of an active duodenal ulcer, history of a bleeding disorder, or history of allergy to aspirin.

All patients were seen in the hospital the night before catheterization, at which time their axillary, brachial, radial, and ulnar pulses were graded 0–4, 0 being an absent pulse and 4 a normal pulse. Each patient was then given a capsule containing either a lactose placebo (66 patients) or 325 mg or 650 mg of aspirin (84 patients), according to a randomized list. Forty-eight patients had taken aspirin during the 7 days before the study and thus were assigned preferentially to the group that received aspirin. Thirty-five patients were taking anticoagulants within the 10 days prior to catheterization.

Patients were catheterized in the fasting state, 12–15 hours after receiving the aspirin or placebo, and after having received 100 mg of sodium pentobarbital 15–30 min prior to the study. A diluted heparin solution (5–8 ml of a solution containing 30 ml of normal saline and 6250 units of porcine heparin) was injected intravenously prior to insertion of the catheter and prior to repair of the arteriotomy. At the completion of the catheterization, a Fogarty catheter was passed proximally and distally to the arteriotomy site. The artery was stabilized with angled vascular clamps and repaired with interrupted 6–0 silk sutures which were wiped with sterile mineral oil. After the repair, the pulses in the catheterized limb were evaluated and graded immediately, on the patient’s leaving the catheterization laboratory, and on the evening of the study. Reopening of the arteriotomy repair when necessary was performed, with thrombectomy and reclosure in the catheterization laboratory. After the procedure, the entire forearm was wrapped in a large bulky dressing, which was worn by the patient until the following morning.

The major end points of the study for the evaluation of the effect of aspirin were (1) the incidence of thrombus formation found at repair by the routine passage of a Fogarty catheter, and (2) reduction of the radial or ulnar arterial pulses after repair of the artery. Other factors analyzed included patient’s age and sex, whether a previous arterial study had been performed in that limb, the presence of local atheroma or arterial spasm, the number of catheters used in each patient, the length of time that the catheter was in the artery, the total time for repair of the arteriotomy, the use of anticoagulants in the 10 days prior to study, and selected hemodynamic parameters, including mean arterial pressure, cardiac index determined by left

From the Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

Address for reprints: Section of Publications, Mayo Clinic, Rochester, Minnesota 55901.

Received July 10, 1972; revision accepted for publication November 12, 1972.

Circulation, Volume XLVII, March 1973
ventricular dye curves, and the calculated systemic resistance. The presence of arterial spasm was determined by the resistance encountered in manipulating the catheter and a decrease in distal pulse if no thrombus was found on routine passage of a Fogarty catheter.

Results

Patients were grouped according to whether they received placebo (66 patients), 325 mg of aspirin (39 patients), or 650 mg of aspirin (45 patients). The groups did not differ significantly with respect to age, sex, the number of catheters used per patient, the time the catheter was in the artery, or the time for repair of the artery (table 1).

Fifteen patients had a decrease in pulse after catheterization (table 2): 12 of the males (9.6%) and three of the females (12%). These patients did not differ in age, number of catheters used, time of catheter in artery, mean arterial pressure, cardiac index, or systemic resistance from those with normal pulses after catheterization. However, arterial thrombus formation and spasm were seen more frequently in patients with decreased pulses. The incidence of decreased pulses (a decrease of at least two grades) immediately after catheterization or by the evening of the study did not differ between the placebo and the aspirin groups (table 3). There was no decrease in the incidence of arterial thrombus formation (5.7%) in the patients who received anticoagulants as compared to those who did not (26.1%) is significant. No hemorrhagic complications were encountered in the anticoagulated patients.

Of the 35 patients who were receiving anticoagulants, two of the 16 in the placebo group had arterial thrombus formation, while none in the aspirin groups had thrombus; these numbers are too small to derive statistical significance. No patient taking anticoagulants had arterial spasm. Of the patients receiving anticoagulants, one in the placebo group and one in the aspirin group had a decrease in pulse. The prothrombin times of the patients with anticoagulation and either placebo or aspirin were nearly identical—one and one-half the normal prothrombin time for our laboratory.

Eight patients had arteriotomy repairs reopened, thrombectomy with the Fogarty catheter, distal flush with dilute heparin, and reclosure of the arteriotomy. Five of these had distal pulses restored to precatheterization status, while three regained distal pulses only partially. Two patients had an absent pulse by the evening of the study, one with an absent radial pulse and an ulnar pulse of grade 4, and a second patient with an absent ulnar pulse (precatheterization only grade 1) and a radial pulse of grade 2. The third patient regained only a radial pulse of grade 1 and an ulnar pulse of grade 1. No patient developed ischemia of the hand or forearm.

Discussion

In recent years, the response of flowing blood to injury as well as the major role of the platelet have been clarified in both normal hemostasis and in thrombus formation. Platelet interaction with surfaces, platelet aggregation, and alteration of blood flow all have important roles in thrombosis. Initial arterial trauma results in platelet adhesion to subendothelial microfibrils and collagen and in

Table 1

<table>
<thead>
<tr>
<th>Clinical Data from Patients Given Placebo or Aspirin</th>
<th>Group</th>
<th>325 mg aspirin</th>
<th>650 mg aspirin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>55.2</td>
<td>51.8</td>
<td>54.4</td>
</tr>
<tr>
<td>(N = 55)</td>
<td></td>
<td>(N = 35)</td>
<td>(N = 35)</td>
</tr>
<tr>
<td>Females</td>
<td>55.5</td>
<td>46.8</td>
<td>52.3</td>
</tr>
<tr>
<td>(N = 11)</td>
<td></td>
<td>(N = 4)</td>
<td>(N = 10)</td>
</tr>
<tr>
<td>Catheters (mean ± sd)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No./pt</td>
<td>1.41 ± 0.68</td>
<td>1.59 ± 1.07</td>
<td>1.22 ± 0.56</td>
</tr>
<tr>
<td>Time in artery (min)</td>
<td>29.7 ± 11.7</td>
<td>30.1 ± 13.0</td>
<td>31.6 ± 11.6</td>
</tr>
<tr>
<td>Repair of artery (min)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mean ± sd)</td>
<td>11.1 ± 12.0</td>
<td>12.5 ± 7.6</td>
<td>12.2 ± 13.3</td>
</tr>
</tbody>
</table>

Circulation, Volume XLVII, March 1973
Table 2

Patients with Normal or Decreased Pulses

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pulse Decreased (N = 15)</th>
<th>Normal (N = 135)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>53.7</td>
<td>54.2</td>
</tr>
<tr>
<td>Males</td>
<td>12</td>
<td>113</td>
</tr>
<tr>
<td>Females</td>
<td>3</td>
<td>22</td>
</tr>
<tr>
<td>Arterial thrombus</td>
<td>7 (47%)</td>
<td>25 (18.5%)</td>
</tr>
<tr>
<td>Spasm</td>
<td>7 (47%)</td>
<td>1 (0.7%)</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>2 (13%)</td>
<td>33 (24.4%)</td>
</tr>
<tr>
<td>No. of catheters (mean ± sd)</td>
<td>1.53 ± 0.74</td>
<td>1.39 ± 0.78</td>
</tr>
<tr>
<td>Catheter time in artery (min)</td>
<td>33.7</td>
<td>30.0</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>91.6</td>
<td>97.1</td>
</tr>
<tr>
<td>Cardiac index (liters/min/m²)</td>
<td>2.73</td>
<td>2.87</td>
</tr>
<tr>
<td>Systemic resistance (units/m²)</td>
<td>34.1</td>
<td>36.7</td>
</tr>
</tbody>
</table>

Table 3

Incidence of Decreased Pulse and Arterial Thrombus Formation

<table>
<thead>
<tr>
<th>Group</th>
<th>Patients (no.)</th>
<th>Pulse immediately after catheterization</th>
<th>Arterial thrombus</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Decreased by 2 grades</td>
<td>Absent</td>
</tr>
<tr>
<td>Placebo</td>
<td>66</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>325 mg aspirin</td>
<td>39</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>650 mg aspirin</td>
<td>45</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>35</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>No anticoagulants</td>
<td>115</td>
<td>4</td>
<td>6</td>
</tr>
</tbody>
</table>
of aspirin would be necessary to settle this ques-
tion. Brachial arteriotomy, in addition to the platelet
adhesion and aggregation that results from exposure
to subendothelial material after arteriotomy repair,
involves initially injury to the entire vessel wall.
Local tissue-thromboplastin release with activation
of the clotting mechanism results and contributes to
thrombus formation. Small doses of aspirin may not
be sufficient to overcome all the thrombogenic
stimuli of the model. This is supported by the
decreased incidence of arterial thrombus formation
in the anticoagulated group, in which we would ex-
pect the anticoagulant to interfere with fibrin for-
mation.

Our study was designed to evaluate the anti-
thrombotic effect of small doses of aspirin in this
model. Due to the limitations of the model, no
general conclusions can be drawn about the use-
fulness of aspirin as an antithrombolic agent.

The incidental finding of decreased thrombus
formation and a lower incidence of decreased or
absent pulses in the anticoagulated group is of
interest. The number of observations is small but
support a protective effect of systemic anticoagula-
tion.

Careful repair of arteriotomy sites and the use of
the Fogarty catheter in experienced hands are im-
portant in decreasing the incidence of thrombotic
complications after brachial arteriotomy.14 Our data
suggest that, although small doses of aspirin do not
decrease thrombotic complications after brachial
arteriotomy, systemic anticoagulation does.

Acknowledgment

The authors gratefully acknowledge the consultation
statistical analysis provided by Lila R. Elveback, Ph.D., the
help of Sister Kathleen Van Groll, and the nursing staff at St.
Marys Hospital.

References

1. Ross RS: Arterial complications. In Cooperative Study
   on Cardiac Catheterization, edited by E Braunwald,
   HJC Swan. New York, American Heart Association,
   Inc, 1968, p 39
2. Machleder HI, Sweeney JP, Barker WF: Pulseless arm
   after brachial-artery catheterisation. Lancet 1:
   407, 1972
   Hemat 5: 91, 1968
4. Hirsh J, Doery JCG: Platelet foundation in health and
disease. Progr Hemat 7: 185, 1971
5. Mustard JF, Packham MA: Factors influencing platelet
   function: Adhesion, release, and aggregation.
   Pharmacol Rev 22: 97, 1970
6. Evans G, Packham MA, Nishizawa EE, Mustard JF,
   Murphy EA: The effect of acetylsalicylic acid on
7. Danese CA, Voleti CD, Weiss HJ: Protection by
   aspirin against experimentally induced arterial throm-
   bosis in dogs. Thromb Diath Haemorrh 25: 288,
   1971
   the editor: Two in-vivo studies comparing high and
   low aspirin dosage. Lancet 1: 399, 1971
9. Salzman EW, Harris WH, Desanctis RW: Reduction
   in venous thromboembolism by agents affecting
10. Sherry S: Prospects in antithrombotic therapy. Amer J
    Cardiol 29: 81, 1972
11. Campion BC, Frye RL, Pluth JR, Fairbairn JF II,
    Davis GD: Arterial complications of retrograde
    brachial arterial catheterization: A prospective study.
12. Stuart RK: Platelet function studies in human beings
    receiving 300 mg of aspirin per day. J Lab Clin Med
    75: 463, 1970
    monophosphate, aracaine, and anti-inflammatory
    agents on thrombosis and platelet function in rabbits.
14. Baker LD, Lessin SJ, Mathur VS, Messer JV:
    Routine Forgarty thrombectomy in arterial catheteri-
Effect of Aspirin on Brachial Artery Occlusion following Brachial Arteriotomy for Coronary Arteriography
KIERAN M. HYNES, GERALD T. GAU, BARRY D. RÜtherFORD, FRANCIS J. KAZMIER and ROBERT L. FRYE

Circulation. 1973;47:554-557
doi: 10.1161/01.CIR.47.3.554
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/47/3/554

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/