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Manual Versus Mechanical Compression of the Radial Artery After Transradial Coronary Angiography

The MEMORY Multicenter Randomized Trial

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ABSTRACT

OBJECTIVES The aim of this study was to compare manual versus mechanical compression of the radial artery after coronary angiography via transradial access regarding radial artery occlusion (RAO), access-site bleeding complications, and duration of hemostasis.

BACKGROUND Hemostasis of the radial artery after sheath removal can be achieved either by manual compression at the puncture site or by using a mechanical hemostasis device. Because mechanical compression exerts a more stable, continuous pressure on the artery, it could be hypothesized that it is more effective compared with manual compression regarding hemostasis time, bleeding, and RAO risks.

METHODS A total of 589 patients undergoing diagnostic coronary angiography by transradial access with a 5-F sheath were randomized in a 1:1 ratio to receive either manual or mechanical patent hemostasis of the radial artery. Radial artery patency was evaluated by color duplex ultrasonography 24 h after the procedure. The primary endpoint was early RAO at 24 h. Secondary endpoints included access-site bleeding complications and duration of hemostasis.

RESULTS Thirty-six (12%) early RAOs occurred in the manual group, and 24 (8%) occurred in the mechanical group ($p = 0.176$). There were no significant differences between the 2 groups regarding access-site bleeding complications (hematoma, 52 [17%] vs. 50 [18%]; $p = 0.749$; bleedings, 8 [3%] vs. 9 [3%]; $p = 1.000$). Duration of hemostasis was significantly shorter in the manual group (22 $\pm$ 34 min vs. 119 $\pm$ 72 min with mechanical compression; $p < 0.001$).

CONCLUSIONS Manual and mechanical compression resulted in similar rates of early RAO, although the total duration of hemostasis was significantly shorter in the manual group. (J Am Coll Cardiol Intv 2018;11:1050–8)

Transradial access (TRA) for diagnostic or interventional coronary procedures has now been adopted as the preferred vascular site approach worldwide (1,2). This has been driven mainly by the lower access-site complication rate, shorter hospital stay, patient preference, and lower costs compared with standard transfemoral access (3-7).

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However, TRA is not devoid of complications (8,9). Potential access-site complications during procedures performed via the TRA approach include radial artery occlusion (RAO), radial artery spasm, persistent post-procedural pain, upper extremity loss of strength, hematoma, pseudoaneurysm, and rarely arteriovenous fistula, radial artery perforation or eversion during sheath removal, hand ischemia, and compartment syndrome (8,9). Although most complications are rare and managed without surgery, early and late RAO might occur with an estimated incidence of about 1% to 10% and has been described as the "Achilles' heel" of the transradial technique (9–12).

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RAO is usually asymptomatic because of the dual blood supply to the hand, and for this reason it is often overlooked (9,10). However, the complication is not benign, as hand ischemia resulting from RAO has been reported (13–15), and patients may experience transient pain at the site of occlusion, paresthesia, or reduced limb function (16). Furthermore, in case of persistent RAO, the artery cannot be used as an access site for repeat catheterization or as an arterial conduit for bypass surgery (9,10). RAO has also been regarded as a relative contraindication for ipsilateral transulnar approach as well, because possible future site complications regarding the ulnar artery would put the patient's hand at risk for ischemia (9,10).

Endothelial injury of the radial artery and decrease in blood flow after sheath insertion and catheter propagation may create an environment prone to thrombosis, leading to RAO (17). Several risk factors for RAO have been described: the diameter of the sheath and its relationship to the size of the radial artery, post-procedural compression time, the presence of anterograde flow in the artery during hemostasis (patent hemostasis), and the use of anticoagulation (10,18–21). Although routine use of patent hemostasis, avoidance of sheath-artery mismatch, and shorter compression time have been shown to reduce the risk for RAO, the application of preventive measures to avoid it remains challenging (9,10).

Because of the superficial nature of the radial artery, hemostasis after sheath removal is achieved by direct compression at the puncture site. This can be performed either manually (firm pressure applied on the radial artery by an operator's fingers, similar to femoral artery compression) or "mechanically" (use of a device, in the form of a bracelet device that wraps around the patient's wrist, pressing the puncture site) (22). Mechanical compression is more convenient and uses fewer human resources compared with manual compression. Furthermore, because mechanical compression exerts a more stable, continuous pressure on the artery, it could be hypothesized that it is superior to manual approach regarding post-procedural access site complications such as RAO.

Many interventional cardiologists continue to believe that manual compression for femoral access should be considered the gold standard for safety and effectiveness (23). Conversely, there are only scarce data comparing these 2 hemostatic methods in transradial procedures (19,24,25). The aim of this randomized trial was to compare manual versus mechanical compression of the radial artery (although manual is less widely used) after transradial coronary angiography regarding RAO, access-site bleeding, and hemostasis duration.

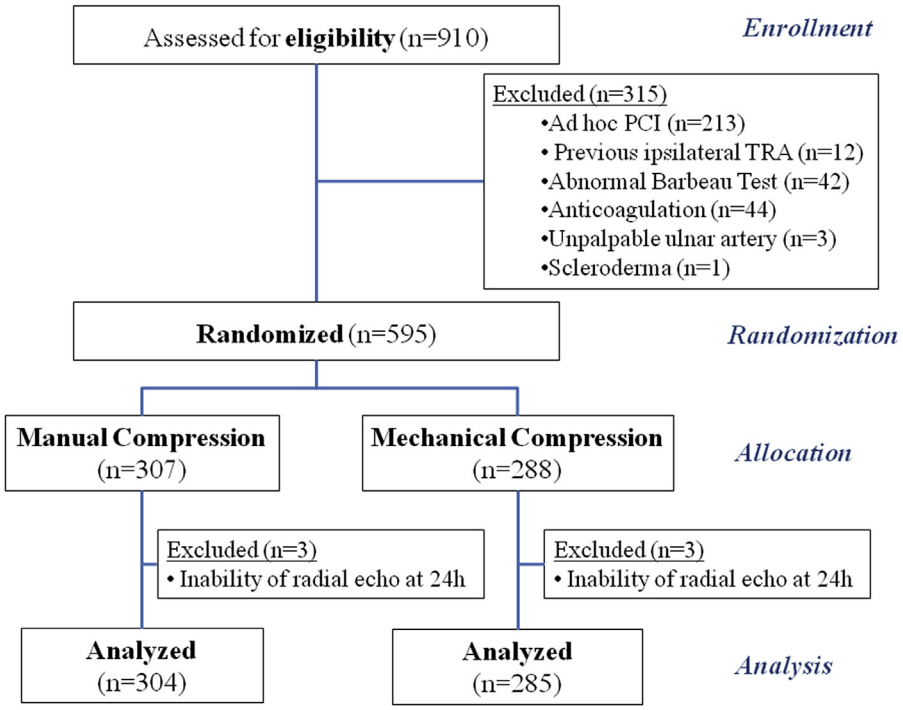
ABBREVIATIONS AND ACRONYMS

ACT = activated clotting time
BARC = Bleeding Academic Research Consortium
CI = confidence interval
OR = odds ratio
PCI = percutaneous coronary intervention
RAO = radial artery occlusion
TRA = transradial access

METHODS

STUDY DESIGN AND PATIENTS. The MEMORY (Manual vErsus Mechanical cOmpression of the Radial arterY after transradial coronary angiography) trial was designed as a prospective, randomized, open-label, multicenter study. Consecutive patients referred for coronary angiography for any reason using TRA were enrolled if they fulfilled the following inclusion criteria: 1) age ≥18 years; 2) ability to provide written, informed consent; 3) use of a 5-F sheath; and 4) normal Barbeau test results and palpable ulnar pulse at the distal forearm (26). The main exclusion criteria were ad hoc percutaneous coronary intervention, and high bleeding risk (previous use of anticoagulation medication, platelet count <100,000/μl, hepatic disease, and estimated glomerular filtration rate <30 ml/min/m^2). Patients in cardiogenic shock, patients with end-stage renal disease receiving renal replacement therapy, patients at risk for hand ischemia (previous ipsilateral TRA or unpalpable ipsilateral ulnar artery), and patients with scleroderma were also excluded. Finally, inability to perform radial artery color duplex ultrasonography within 24 h after diagnostic coronary angiography and inability to tolerate heparin (history of heparin-induced thrombocytopenia) were also regarded as exclusion criteria. Patients were randomized (1:1) to receive either manual or mechanical compression of the radial artery using a software-based automatic randomization program incorporating a random block size (of 2, 4, 6, or 8) for each study center. Randomization took place after diagnostic coronary angiography with a 5-F sheath, if ad hoc percutaneous

FIGURE 1 Study Flow Diagram



The MEMORY (Manual Versus Mechanical Compression of the Radial Artery After Transradial Coronary Angiography) study randomized 595 patients to manual (n = 307) and mechanical (n = 288) compression methods. In 6 patients, radial ultrasonography was not possible, and data on the primary study endpoint of 24-h radial artery occlusion were collected on a total of 589 patients. PCI = percutaneous coronary intervention; TRA = transradial access.

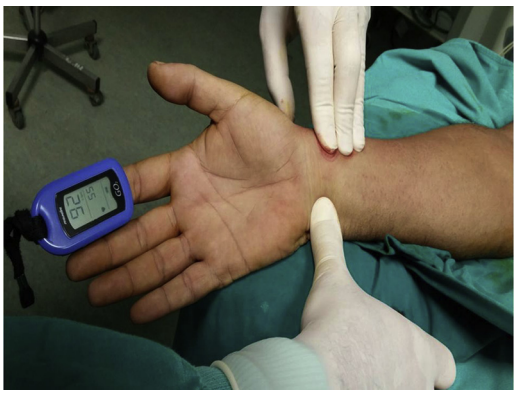
coronary intervention was not planned. Five different catheterization laboratories across Greece participated in the study (Athens, Thessaloniki, Patras, Heraklion, and Veroia) from January 2015 to May 2017 (Figure 1). The study protocol was approved by the institutional ethics committee of each hospital, and all subjects provided informed written consent.

According to power analysis, 600 patients would be needed to have at least 80% statistical power to detect a 2-tailed (superiority for either method) 6% difference in RAO incidence between the 2 groups with a type I error rate of 5% and assumed incidence rates of 12% in the control group and 6% in the intervention group.

DIAGNOSTIC CORONARY ANGIOGRAPHY USING TRA. After sterile preparation and 2% lidocaine infiltration, using a siliconized 21-gauge needle, the radial artery was accessed using the modified Seldinger (anterior wall single puncture) technique (27). An 11-cm, 5-F, hydrophilic-coated introducer sheath (Teleflex Medical, Research Triangle Park, North Carolina) was inserted into the radial artery lumen over a 0.018-inch guidewire. All patients received 50 IU/kg unfractionated heparin intravenously, whereas the intra-arterial use of vasodilators such as nitroglycerin or verapamil was left to the operator's preference. Diagnostic coronary angiography was performed using only 5-F catheters. Choice of catheter shape and number of catheters was also left to the operator's discretion.

STUDY ENDPOINTS. The primary endpoint of the study was the incidence of early (within 24 h of the diagnostic coronary angiographic procedure) RAO using radial artery color duplex ultrasonography. RAO was confirmed by absence of anterograde flow in the radial artery while compressing the ipsilateral ulnar artery, using an HP 11-3L ultraband (3 to 11 MHz) vascular transducer (Hewlett Packard/Philips Ultrasound, Andover, Massachusetts) with the Sonos 5500 ultrasound scanner (Hewlett Packard/Philips Ultrasound). Color duplex ultrasound studies were performed in all patients the day following the procedure by physicians who were blinded to the method of hemostasis applied. Radial artery diameter both

FIGURE 2 Hemostasis Method



For manual compression, pressure with 3 fingers over the puncture site and centrally was applied to establish hemostasis. The operator was trying to achieve patent hemostasis, and patency was checked every 1 to 2 min by transient manual compression of the ulnar artery and evaluation of the status of radial artery patency by plethysmography.

proximal and distal to the puncture site of the access forearm was also recorded. Secondary study endpoints included access-site bleeding using the Bleeding Academic Research Consortium (BARC) score (28), hematoma formation using the EASY (Early Discharge After Transradial Stenting of Coronary Arteries Study) hematoma scale (29) and time required for successful hemostasis.

Demographic data including age, sex, body mass index, details on patients' medical history, and indications for diagnostic coronary angiography were recorded on dedicated case-report forms. Procedural data including number of puncture attempts, procedural time, duration of sheath stay in radial artery (from the initiation of the procedure), occurrence of severe radial artery spasm, and confirmed patent hemostasis were also recorded.

HEMOSTASIS. After completion of the transradial procedure, hemostasis was achieved as follows: for manual compression, the introducer sheath was pulled out 4 to 5 cm, and then pressure with 3 fingers over the puncture site and centrally was applied. The sheath was pulled out completely until some bleeding was visible, to purge the prethrombotic and thrombotic material and establish radial artery flow as evidenced by mild bleeding at the site. Pressure with 3 fingers over the puncture site and centrally was reapplied to establish hemostasis. Manual compression was maintained for 10 min or longer if required to control bleeding. The operator was trying to achieve patent hemostasis, and patency was checked every 1 to 2 min by transient manual compression of the ulnar artery and evaluation of the status of radial artery patency by plethysmography. If anterograde flow in radial artery was absent (lack of signal), the pressure was decreased to attempt reestablishment of anterograde radial artery flow without sacrificing hemostasis (patent hemostasis) (Figure 2). When hemostasis was achieved, a light wound dressing was applied at the puncture site. Manual compression was performed by 1 or 2 operators at each study center.

For mechanical compression, an inflatable air filled wrist bracelet (Vitatech Pressure Bandage, KDL Medical Group, Shanghai, China) was applied at the arterial puncture site, the bladder was inflated, and the introducer sheath was removed from under the band. The bladder was then decompressed to lower the compression pressure until some bleeding was visible, to purge the prethrombotic and thrombotic material and establish radial artery flow as evidenced by mild bleeding at the site. The bladder was then reinflated with 1 to 2 ml of additional air to reestablish hemostasis. Transient manual compression of the ulnar artery was performed to evaluate the status of radial artery patency by plethysmography, and if anterograde flow in radial artery was absent (lack of signal), the process of deflation and reinflation was repeated to attempt re-establishment of anterograde radial artery flow without sacrificing hemostasis (patent hemostasis). The bladder was gradually deflated with removal of 3 ml of air after 30 min and thereafter with removal of 3 ml of air every 15 min. In case of bleeding, reinsertion of the removed air was performed to achieve hemostasis. When empty of air, the device was removed, and a light wound dressing was applied at the puncture site. For both methods, radial artery patency was also assessed by digital plethysmography using the reverse Barbeau test immediately after placement of the wound dressing, and patients were monitored for at least 30 min after application of the light wound dressing.

EVALUATION OF RADIAL ARTERY PATENCY. As previously described, color duplex ultrasound studies were performed in all patients the day following the procedure by physicians who were blinded to the method of hemostasis applied. Radial artery patency was also evaluated during compression, at the time of removal of the radial compression band or bracelet and 24 h after the procedure (30) using digital plethysmography and the reverse Barbeau test.

STATISTICAL ANALYSIS. Results are presented as mean ± SD for continuous variables and as percentages for categorical data. Normality was tested by

TABLE 1 Baseline Characteristics

	Manual Compression (n = 304)	Mechanical Compression (n = 285)	p Value
Age, yrs	64 ± 11	65 ± 11	0.434
Male/female	218/86	198/87	0.587
BMI, kg/m^2	29 ± 5	29 ± 5	0.598
BSA, m^2	1.93 ± 0.2	1.95 ± 0.2	0.225
Indication for coronary angiography			0.633
CAD	185 (61)	180 (63)	
Valvular/other	119 (39)	105 (37)	
History of CAD	61 (20)	66 (23)	0.410
Hypertension	198 (65)	178 (62)	0.485
Diabetes mellitus	97 (32)	88 (31)	0.786
Dyslipidemia	146 (48)	131 (46)	0.801
Smoking	109 (36)	100 (35)	0.863
Family history of CAD	46 (15)	40 (14)	0.722
Hemoglobin, g/dl	13.9 ± 3.0	13.8 ± 2.0	0.668
Creatinine, mg/dl	0.9 ± 0.2	1.0 ± 0.3	0.224
GFR, ml/min/m^2	92 ± 41	95 ± 41	0.533
Periprocedural treatment characteristics			
Antiplatelet agents	222 (73)	205 (72)	0.918
Aspirin	192 (63)	171 (60)	0.569
Clopidogrel	116 (38)	103 (36)	0.704
Ticagrelor	9 (3)	17 (6)	0.275
Prasugrel	3 (1)	0 (0)	0.500
Triflusal	4 (1)	2 (1)	0.684
Vasodilators			0.343
Nitrates	191 (63)	162 (57)	
Verapamil	109 (36)	123 (43)	
None	3 (1)	1 (0.5)	
Heparin, U	4,098 ± 794	4,314 ± 2,003	0.082*
Periprocedural characteristics			
Arm (right/left)	286/18	276/9	0.221
Radial artery spasm	18 (6)	11 (4)	0.341
Puncture attempts	1.3 ± 0.8	1.4 ± 0.8	0.278
Sheath length, mm	10.8 ± 0.8	10.8 ± 0.9	0.563
Sheath time, min	22.1 ± 14.1	22.5 ± 14.0	0.779
Radiation time, min	6.1 ± 12	7.7 ± 16.0	0.194
ACT, s	155 ± 55	147 ± 61	0.325
RA diameter distal, mm	2.8 ± 0.6	2.8 ± 0.7	0.380
RA diameter proximal, mm	3.5 ± 0.7	3.5 ± 0.6	0.861
Contrast volume, ml	74 ± 27	84 ± 42	0.002*

Values are mean ± SD or n (%). *p value refers to comparisons using the unpaired Student's *t*-test.

ACT = activated clotting time; BMI = body mass index; BSA = body surface area; CAD = coronary artery disease; GFR = glomerular filtration rate; RA = radial artery.

using the Kolmogorov-Smirnov test. Comparisons between categorical variables were performed using the chi-square or Fisher exact test when required. Differences in continuous variables between 2 groups were assessed using the unpaired Student's *t*-test.

The association of method of hemostasis with RAO incidence was evaluated in univariate logistic regression analysis models. Independence was assessed in multivariate logistic regression analysis models using variables that could act as possible cofounders for either RAO or study group allocation. Variables retained in final models were chosen with a backward stepwise selection method. For all logistic regression analysis models, odds ratios (ORs) with 95% confidence intervals (CIs) were calculated.

A p value <0.05 was considered to indicate statistical significance. However, to identify possible confounders or subgroups of patients with possible interaction effect, a p value of <0.100 was considered to indicate significance; all tests were 2-sided. IBM SPSS Statistics version 20.0 (SPSS, Chicago, Illinois) was used for all calculations.

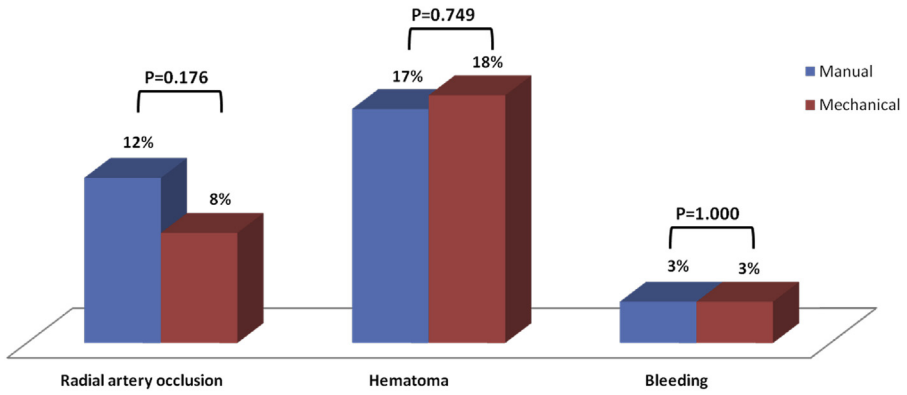
RESULTS

STUDY ENDPOINTS. From a total of 910 patients, 595 were randomized, and the final analysis included 304 patients in the manual and 285 patients in the mechanical compression group (Figure 1). Allocation of treatment (manual vs. mechanical) was not different among the 5 cardiac catheterization laboratories (p = 0.636). Baseline demographic, medical history, treatment, and procedural characteristics were similar in both groups (Table 1) with the exception of contrast volume used and heparin dose (borderline significance). The primary endpoint, incidence of RAO 24 h post-procedure was similar (p = 0.176) between the group of patients with manual compression (n = 36 [12%]) and the group of patients with mechanical compression (n = 24 [8%]) (Figure 3). Time-stratum analysis using a block of 100 patients for each stratum (on the basis of time of randomization) showed that there were no differences either on RAO incidence in the whole study population or in the distribution of RAO between the 2 groups as the study progressed.

Unadjusted logistic regression analysis showed that method of hemostasis was not significantly associated with incidence of RAO at 24 h post-procedure (mechanical vs. manual hemostasis; OR: 0.69; 95% CI: 0.40 to 1.18; p = 0.172). After adjustment for contrast volume used and heparin dose in regression analysis, method of hemostasis continued not to be significantly associated with the study primary endpoint (mechanical vs. manual hemostasis; OR: 0.68; 95% CI: 0.39 to 1.19; p = 0.179).

Failure of hemostasis (during and immediately after compression combined) was similar between the 2 groups (n = 2 [0.7%] vs. n = 1 [0.4%], p = 1.000). However, patent hemostasis during

FIGURE 3 Incidence of Efficacy (Radial Artery Occlusion) and Safety (Hematoma and Bleeding) Study Endpoints



Incidence of radial artery occlusion, hematoma, and bleeding at 24 h post-procedure in patients with mechanical **(red bars)** and manual **(blue bars)** hemostasis.

compression occurred significantly more often ($p = 0.002$) in the manual (n = 295 [97%]) compared with the mechanical compression group (n = 257 [90%]). Furthermore, hemostasis duration was shorter with manual (22 ± 34 min) than with mechanical (119 ± 72 min) compression ($p < 0.001$). Regarding access-site bleeding complications, no significant difference between groups was noted in hematoma formation or hemorrhage at access site (Table 2, Figure 3).

PREDICTORS OF RAO. Univariate predictors of 24-h RAO (with p values <0.100), which included number of puncture attempts, activated clotting time, radiation time, duration of sheath time in radial artery, study center, sex, diabetes mellitus, presence of hematoma, radial artery spasm, age, and patency during hemostasis are shown in Table 3. Multivariate analysis using the backward deletion method identified the number of puncture attempts (OR: 2.69; 95% CI: 1.42 to 5.14; $p = 0.003$), activated clotting time (OR: 0.98; 95% CI: 0.96 to 1.01; $p = 0.068$), radiation time (OR: 0.79; 95% CI: 0.63 to 0.99; $p = 0.046$), and presence of patency during hemostasis (OR: 0.08; 95% CI: 0.02 to 0.34; $p < 0.001$) as independent predictors of 24-h RAO. In a second multivariate model, when method of hemostasis was entered as an independent variable in addition to the previously mentioned variables, method of hemostasis was not associated with 24-h RAO occurrence (mechanical vs. manual; OR: 0.45; 95% CI: 0.13 to 1.58; $p = 0.215$).

TABLE 2 Access-Site Bleeding Complications

	Manual Compression (n = 304)	Mechanical Compression (n = 285)	p Value
Hematoma	52 (17)	50 (18)	0.749
EASY hematoma scale			0.510
Stage 1	46 (88)	48 (96)	
Stage 2	5 (10)	2 (4)	
Stage 3	1 (2)	0 (0)	
Hemorrhage	8 (3)	9 (3)	1.000
BARC staging of hemorrhages			0.892
Type 1	5 (63)	5 (56)	
Type 2	3 (37)	4 (46)	

Values are n (%).

BARC = Bleeding Academic Research Consortium; EASY = Early Discharge After Transradial Stenting of Coronary Arteries Study.

SENSITIVITY ANALYSIS. To identify possible subgroups of patients among which the use of 1 method over another could have an impact regarding occurrence of RAO, we investigated possible interaction effects between various subgroups of patients and incidence of RAO in logistic regression models. Sensitivity analysis (Table 4) identified age <70 years, male sex, no patency during hemostasis, coronary artery disease as an indication for coronary angiography, and prolonged time (>20 min) of sheath in radial artery as subgroups in which the use of mechanical compression could be statistically significantly associated with lower incidence of RAO compared with manual compression.

TABLE 3 Univariate Predictors of 24-h Radial Artery Occlusion by Ultrasonography

	Patients Without RAO (n = 529)	Patients With RAO (n = 60)	p Value
Age, yrs	65 ± 11	62 ± 12	0.100*
Male/female	380/149	36/24	0.072†
BMI, kg/m^2	29 ± 5	29 ± 5	0.470
BSA, m^2	1.9 ± 0.2	1.9 ± 0.2	0.670
Indication for coronary angiography			0.283
CAD	254 (48)	33 (55)	
Valvular/other	275 (52)	27 (45)	
History of previous CAD	111 (21)	17 (28)	0.184
Hypertension	333 (63)	44 (73)	0.119
Diabetes mellitus	159 (30)	25 (42)	0.076†
Dyslipidemia	249 (47)	29 (48)	0.891
Smoking	185 (35)	24 (40)	0.397
Family history of CAD	74 (14)	10 (17)	0.560
Hemoglobin, g/dl	13.8 ± 2.0	13.9 ± 4.0	0.767
Creatinine, mg/dl	0.9 ± 0.2	0.9 ± 0.2	0.770
GFR, ml/min/m^2	93 ± 41	96 ± 34	0.723
Periprocedural treatment characteristics			
Antiplatelet agents	386 (73)	42 (70)	0.729
Aspirin	328 (62)	38 (63)	0.875
Clopidogrel	196 (37)	26 (44)	0.423
Ticagrelor	26 (5)	1 (2)	0.712
Prasugrel	2 (1)	0 (0)	1.000
Triflusal	5 (1)	0 (0)	1.000
Vasodilators			0.844
Nitrates	317 (60)	37 (61)	
Verapamil	206 (39)	23 (39)	
None	6 (1)	0 (0)	
Heparin, U	4,215 ± 1,566	4,096 ± 858	0.563
Periprocedural characteristics			
Arm (right/left)	508/21	58/2	1.000
Radial artery spasm	21 (4)	10 (16)	0.007†
Puncture attempts	1.3 ± 0.7	1.7 ± 0.9	0.003*
Sheath time, min	21.8 ± 13.5	26.2 ± 17.9	0.077*
Radiation time, min	7.1 ± 14.8	4.7 ± 3.5	0.004*
ACT, s	150 ± 61	161 ± 23	0.091*
RA diameter distal, mm	2.8 ± 0.7	2.7 ± 0.5	0.446
RA diameter proximal, mm	3.5 ± 0.7	3.5 ± 0.5	0.937
Contrast volume, ml	79 ± 35	82 ± 36	0.498
Hemostasis duration, min	69 ± 73	63 ± 84	0.577
Patency during hemostasis	503 (95)	51 (85)	<0.001†
Presence of hematoma	90 (17)	23 (38)	<0.001†
Presence of bleeding	21 (4)	0 (0)	0.244
Failure during hemostasis	2 (1)	1 (2)	0.291
Study center			0.008†
Center 1	195 (86)	31 (14)	
Center 2	80 (94)	5 (6)	
Center 3	115 (89)	14 (11)	
Center 4	54 (86)	9 (14)	
Center 5	85 (99)	1 (1)	

Values are mean ± SD or n (%). *p value refers to comparisons using the unpaired Student's *t*-test. †p value refers to comparisons using the chi-square or Fisher exact test.

RAO = radial artery occlusion; other abbreviations as in Table 1.

DISCUSSION

In the present study we compared mechanical and manual compression of the radial artery after transradial coronary angiography regarding RAO, access-site bleeding, and hemostasis duration. At 24 h post-procedure, the incidence of RAO was similar between manual (12%) and mechanical (8%) hemostasis. Furthermore, no significant differences were observed regarding hematoma or bleeding rates between the 2 groups. Finally, patent hemostasis was more common, and the duration of hemostasis was shorter in patients with manual compression. Adjustment for possible confounding variables relative to RAO incidence and combined assessment of efficacy and safety endpoints yielded similar results. Because systemic anticoagulation is a very important factor regarding the occurrence of RAO, and in our study heparin dose was statistically borderline higher in the mechanical compression group, we must underscore that adjusting for heparin dose in multivariate modeling yielded similar results regarding RAO incidence rate. Our observation is in line with the findings from the PHARAOH study, in which heparin administration, although it was a univariate predicter for RAO, in multivariate modeling lost its significance (20).

To our knowledge, our study is among the first to compare the efficacy and safety of these 2 different methods of hemostasis in a randomized, controlled manner (18,19,22,24,25). However, we must underscore that the manual hemostasis protocol may have limited operational adoption in catheterization laboratories. This will probably be driven by the need for larger involvement of post-procedural care team with repeated point-of-care evaluation of radial flow, making a rather simplistic and inexpensive process of hemostatic compression significantly more complex (30). On the contrary, as hemostasis duration is shorter with manual compared with mechanical compression, a possible advantage of the former method is earlier patient discharge, which may be important in terms of both patient preference and efficient management of patient flow. Moreover, as time-stratum analysis of our results showed that there was no difference in RAO incidence as the study progressed, is evident that there is no learning curve for manual compression regarding RAO occurrence.

Finally, RAO incidence in our study was rather high, especially compared with the RAP and BEAT (Radial Artery Patency and Bleeding, Efficacy, Adverse Event) (12) and PROPHET (Prevention of Radial Artery Occlusion-Patent Hemostasis Evaluation Trial) II trials (30); a possible explanation for this is that a slender

TABLE 4 Subgroup Sensitivity Analysis

	OR (95% CI)	p Value for Interaction
Overall crude	0.69 (0.40–1.18)	0.172
Overall adjusted*	0.68 (0.39–1.19)	0.179
Male	0.45 (0.22–0.95)	0.094
Female	1.2 (0.50–2.84)	
Age <70 yrs	0.51 (0.26–0.98)	0.088
Age ≥70 yrs	1.45 (0.53–3.96)	
Low GFR (<90 ml/min/1.73 m^2)	1.11 (0.43–2.86)	0.158
High GFR (≥90 ml/min/1.73 m^2)	0.43 (0.17–1.09)	
No patency during hemostasis	0.17 (0.03–0.89)	0.019
Patency during hemostasis	0.67 (0.33–1.37)	
Low BMI (<29.9 kg/m^2)	0.62 (0.31–1.24)	0.557
High BMI (≥30 kg/m^2)	0.85 (0.35–2.07)	
CAD as an indication	0.38 (0.18–0.84)	0.029
No CAD as an indication	1.69 (0.58–4.99)	
No diabetes mellitus	0.53 (0.26–1.09)	0.225
Diabetes mellitus	1.05 (0.45–2.46)	
No antiplatelet agents	0.70 (0.23–2.14)	0.601
Antiplatelet agents	0.49 (0.23–1.05)	
1 arterial puncture	0.63 (0.31–1.28)	0.718
>1 arterial puncture	0.77 (0.32–1.85)	
No radial arterial spasm	0.46 (0.22–0.99)	0.519
Radial arterial spasm	0.20 (0.02–2.28)	
Short sheath time (<20 min)	0.96 (0.44–2.09)	0.100
Long sheath time (≥20 min)	0.38 (0.17–0.87)	
Low ACT (<160 s)	0.37 (0.07–1.88)	0.176
High ACT (≥160 s)	0.79 (0.24–2.69)	
Short radiation time (<10 min)	0.66 (0.35–1.23)	0.704
Long radiation time (≥10 min)	0.47 (0.09–2.31)	

Sheath length and arm of puncture variables had too few events to perform subgroup analysis. *Adjustment for contrast volume used and heparin dose.

CI = confidence interval; OR = odds ratio; other abbreviations as in Table 1.

sheath and adjunctive to patent hemostasis ulnar compression were not used. However, both our RAO incidence (approximately 10%) and possible predictors of RAO (number of puncture attempts, activated clotting time, procedural duration, sheath length, duration of sheath time in radial artery, diabetes mellitus, presence of hematoma, radial artery, radial artery spasm, and patency during hemostasis) (10,18–21) were in line with published data (9–12). Furthermore, the high RAO incidence in our study suggests that we need to be more vigilant in our efforts to ensure patent hemostasis; a possible approach is to take special precaution in those patients with multiple puncture attempts, prolonged duration of sheath time in the radial artery, and other factors.

Of importance, using sensitivity analysis, our study identified possible subgroups of patients for whom mechanical hemostasis could result in a reduction in RAO incidence. These subgroups of patients include those age <70 years, male patients, those without patency during hemostasis, those with coronary artery disease as an indication for coronary angiography, and those with prolonged time (>20 min) of sheath in the radial artery. Of clinical importance, we may postulate that if we start hemostasis with manual compression and patent hemostasis cannot be achieved, switching to mechanical compression might decrease the chance of RAO. However, sensitivity analysis results are only of an exploratory nature and hypothesis generating and cannot be used to draw certain conclusions.

STUDY LIMITATIONS. First, in our study, novel methods to reduce RAO incidence with mechanical compression, such as ulnar artery compression or pneumatic control device compression, were not applied (19,30). Second, biochemical evaluation of hand ischemia was not performed. More important, according to a post hoc power analysis, our study was underpowered to suggest statistical significance of the observed 4% reduced rate in RAO incidence with mechanical compression. However, even in the case of extrapolating our findings to a study population achieving statistical power (90% in a study population with more than 2,500 patients) the number needed to treat to avoid 1 case of RAO would be 30, thus minimizing the clinical significance of the observed statistical superiority of mechanical hemostasis. Furthermore, because there was wide variation in the incidence of RAO among centers, we cannot exclude that some human or technical factors played a role in our results. Finally, our study did not include patients with abnormal Barbeau test results, with nonpalpable ulnar artery, undergoing ad hoc percutaneous interventions, and procedures in which sheaths of smaller or larger than 5-F in diameter were used. Hence, our findings cannot be extrapolated to those patients or procedures.

CONCLUSIONS

Manual compression was an effective, safe, and less time consuming hemostasis method compared with mechanical compression using devices (inflatable air-filled wrist bracelet) that exert stable and continuous pressure on the radial artery. However, mechanical compression may be considered a better alternative in certain subgroups of patients and, thus, larger studies are needed to corroborate our results.

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PERSPECTIVES

WHAT IS KNOWN? Mechanical pneumatic devices are widely used for radial compression after TRA for coronary procedures, because of their convenience.

WHAT IS NEW? Manual compression of the radial artery has never been robustly compared with mechanical compression before. The MEMORY study shows that manual compression is at least as effective and safe as mechanical compression and significantly shorter in duration.

WHAT IS NEXT? Larger studies are needed to corroborate these results, although manual compression may be considered if earlier patient discharge is desired.

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