Impact of catheter insertion using the radial approach on vasodilatation in humans

Ellen A. DAWSON*,1, Sudhir RATHORE†1, N. Timothy CABLE*, D. Jay WRIGHT†, John L. MORRIS† and Daniel J. GREEN*‡
*Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Liverpool L3 2ET, U.K., †Liverpool Heart and Chest Hospital, Liverpool L14 3PE, U.K., and ‡School of Sport Science, Exercise and Health, The University of Western Australia, Western Australia, Australia

ABSTRACT

The aim of this study was to determine the impact of catheter sheath insertion, a model of endothelium disruption in humans, on the conventional FMD (flow-mediated dilatation) response in vivo. Seventeen subjects undergoing transradial catheterization were recruited and assessed prior to, the day after, and 3–4 months postcatheterization. The catheter sheath’s external diameter was 2.7 mm, and the average preprocedure internal radial artery diameter was 2.8 mm, indicating a high likelihood of endothelial denudation as a consequence of sheath placement. Radial artery flow-mediated and endothelium-derived NO (nitric oxide)-dependent function (FMD) was assessed within the region of sheath placement (sheath site) and also above the sheath (catheter site). GTN (glyceryl trinitrate) endothelium-independent NO-mediated function was also assessed distally. Measurements were made in both arms at all time points; the non-catheterized arm provided an internal control. Neither sheath (4.5 ± 0.9 %) nor catheter (4.4 ± 0.9 %) insertion abolished FMD, although both significantly decreased FMD from preintervention levels (9.0 ± 0.8 % sheath segment; 8.4 ± 0.8 % catheter segment; P < 0.05). The impact of sheath and catheter placement on FMD was no longer evident after ~3 months recovery (8.0 ± 1.5 and 8.1 ± 1.7 %, sheath and catheter, respectively). GTN responses also decreased from 14.8 ± 1.7 to 7.9 ± 1.0 % (P < 0.05) as a result of sheath placement, but values returned to baseline at ~3 months (13.0 ± 1.8 %). These results suggest that the presence of an intact, functional endothelial layer and consequent NO release may not be obligatory for some component of the FMD response. This raises the possibility of an endothelium-independent contribution to the flow-induced vasodilatation in humans.

INTRODUCTION

In recent years, FMD (flow-mediated dilatation) has become a popular technique in cardiovascular medicine and clinical physiology, as evidence has accrued that depressed FMD is an independent prognostic index of incident and recurrent cardiovascular events, which adds predictive value to established risk factor approaches [1–5]. The physiological rationale for the use of FMD, and a proposed reason for its prognostic relevance, is that it reflects endothelium- and (nitric oxide)-dependent vascular function.

When Celermajer, Deanfield and colleagues introduced the now conventional and widely adopted FMD approach in the Lancet in 1992 [6], no direct evidence was available that brachial and femoral dilatation, subsequent to the release of cuff-induced ischaemia, were endothelium- or NO-dependent in humans. The

Key words: catheterization, flow-mediated dilatation, endothelium, nitric oxide, radial approach.
Abbreviations: eNOS, endothelial NO synthase; FMD, flow-mediated dilatation; GTN, glyceryl trinitrate; HR, heart rate; l-NMMA, N\(^G\)-monomethyl-l-arginine; MAP, mean arterial pressure; SR\(_{\text{AUC}}\), shear rate area under the curve; TTP, time to peak.
1These authors are to be considered as joint first authors.
Correspondence: Dr Ellen A. Dawson (email e.dawson@ljmu.ac.uk).
approach was therefore based on evidence available at the time that the endothelium released a labile vasodilator substance \[7,8\], that increased luminal flow-induced vasodilatation in human conduit arteries \[9–12\] and, crucially, that FMD in animals was dependent upon the presence of an intact endothelial lining. In these latter studies, insertion of a catheter and balloon inflation was used to denude the endothelial layer, which abolished FMD. Very few studies of this nature have been undertaken in humans to examine FMD after endothelial damage \[13–15\]. While previous studies have reported a depression in the vasodilator function of the radial artery after it has been catheterized \[13,14\], the methods used to assess arterial vasodilator function were limited. In particular, discreet time measurements were used to assess the peak diameter, and as such, the ‘true’ peak is likely to have been missed. Furthermore, these studies assessed the peak diameter, and as such, the ‘true’ peak is likely to have been missed. Furthermore, these studies did not assess the stimulus the artery received during the FMD test \[\text{SR}_{\text{AUC}}\] (shear rate area under the curve). This has previously been demonstrated to be an important determinant of the FMD response and should be reported in order to elucidate whether any changes in FMD are due to alterations in the artery’s ability to dilate, as opposed to a reduction in the stimulus \[16\]. In addition, the time-course of recovery from endothelial damage remains largely unknown.

The relevance of determining the degree to which FMD responses are endothelium-dependent is heightened by a recent debate over the extent to which FMD represents an endothelium-dependent and NO-mediated response \[18,19\]. NO blockers have been infused during FMD in humans, with differing effects. Joannides et al. \[20\] demonstrated that \(l\)-NMMA \(N^\text{G}l\)-monomethyl-\(l\)-arginine) infusion converted flow-mediated vasodilatation into constriction in the radial artery in healthy young humans, while Doshi et al. \[21\] reported abolition of FMD with \(N^\text{G}l\)-NMMA \[22\]. Somewhat in contrast, Mullen et al. \[22\] Lieberman et al. \[23\] and Koojiman et al. \[24\] all observed decreased FMD in the presence of \(N^\text{G}l\)-NMMA, but not FMD abolition by NO blockade in these studies. In addition, eNOS (endothelial NO synthase) knockout mice exhibit preserved FMD responses \[25\]. These studies suggest that compensatory mechanisms may play a role in the FMD response if the endothelial release of NO is dysfunctional or absent and/or that NO is not obligatory for a dilator response to ischaemia. They also raise the question of to what extent FMD responses are dependent upon the presence of an intact and functional endothelium.

The principal aim of the present study was to use a model of endothelial disruption in humans to determine the impact on FMD acutely. We also examined the impact of sheath compared with catheter placement, the impact of sheath placement on endothelium-independent GTN (glyceryl trinitrate) responses and the time-course of recovery of FMD and GTN responses. We hypothesized that sheath insertion would diminish or abolish the endothelium-dependent and -independent function within, but not above, the sheath site.

**MATERIAL AND METHODS**

**Patients**

Seventeen subjects (16 men, one woman) were recruited from the list of patients requiring radial artery catheterization for coronary angiography or coronary angioplasty. The following were excluded: patients who had previously undergone coronary artery bypass surgery or coronary intervention via the radial route or had myocardial infarction during the previous 3 months, valvular heart disease, a left ventricular ejection fraction < 40 %, chronic obstructive lung disease, or renal or hepatic dysfunction.

The study conformed to the standards set by the Declaration of Helsinki and ethical approval was obtained from the Liverpool Local Regional Ethics Committee. All patients provided informed written consent.

**Study design**

Patients were tested on three occasions: the day of the transradial procedure (immediately before the catheterization ‘Pre’), the day after catheterization (‘Post’) and ∼ 3 months after catheterization (‘Recov’). Volunteers were requested to abstain from alcohol or caffeinated beverages and cigarettes (if they were smokers) for 12 h prior to each testing session. Assessments were taken in a quiet, temperature-controlled room. Patients rested in the supine position for approximately 20 min to ensure that all haemodynamic variables had stabilized. The radial artery was assessed with the arm extended and supported at an angle of approximately 80° from the torso. A rapid inflation/deflation pneumatic cuff was positioned on the imaged arm around the wrist. A standard catheter sheath was then used to mark the length of the catheter on the surface of the arm, from the scaphoid process. Care was taken to image the same section of the artery during repeat measurements. We assessed both arms to determine whether changes as a consequence of catheterization were specific, or more generalized, throughout the vascular system. On each occasion, endothelial-dependent function (FMD) was assessed over a distal section of the radial artery (FMD sheath), within the zone containing the sheath and over a proximal section (FMD catheter), which lay above the sheathed region. A minimum of 20 min was observed between repeated FMD assessments in the same arm. The order in which the arms were tested was randomized for FMD and GTN. Endothelium-independent function was assessed as the vascular response to a sublingual dose of GTN with the scans taken in the distal section of the...
radial artery after the FMD assessments. A minimum of 30 min was given between repeat doses of GTN.

Radial artery access and procedural details
In this study, we recruited patients undergoing treatment for coronary artery disease. These patients have their coronary arteries either imaged (angiography) or have the narrow arteries widened (angioplasty). In order to gain access to the coronary circulation, a catheter is placed into a peripheral artery, in this case the radial artery, and the catheter is guided up to the heart. Introducer sheaths are used to gain access to the radial artery and to keep the artery open in order to facilitate the exchange of the guiding coronary catheter to the heart.

The radial artery was approached with the arm extended and supported with the wrist in mild hyperextension. Local anaesthesia was achieved with 2 % lignocaine after disinfection at the puncture site. The radial artery was punctured with a 21-gauge arterial needle through which a 0.118-in platinum-tipped nitinol guidewire was introduced. Following this, the needle was withdrawn, and a small skin incision was made. A 6 F introducer sheath (13 cm in length, external diameter 2.7 mm; Cook Medical) with a dilator tip length of 2.5 cm was inserted. A weight-adjusted dose of heparin was introduced into the central circulation following the introduction of the first catheter. All introducer sheaths were removed at the end of the procedure and haemostasis was achieved in the catheterization laboratory by a compression device. The patients were mobilized immediately, and the compression device was removed after 2–4 h.

Experimental procedures
Ultrasound assessment of conduit artery function
HR (heart rate) and MAP (mean arterial pressure) were determined from an automated sphygmomanometer (Dinamap; GE Pro 300V2) on the contralateral arm. A 12-MHz multifrequency linear array probe attached to a high-resolution ultrasound machine (T3000; Terson) was used to assess radial artery function.

FMD (endothelium-dependent NO-mediated function)
Baseline scans assessing resting vessel diameter and flow were recorded in the final minute of the initial rest period. The occluding cuff was then inflated to >200 mmHg for 5 min. Diameter and flow recordings resumed 30 s prior to cuff deflation and continued for 5 min thereafter. Blood pressure and HR were recorded during the resting period and once the cuff had been released.

GTN dilatation (endothelial-independent NO-mediated function)
Vessel diameter was recorded 1-min before and, continuously for 10 min after, sublingual administration of GTN (400 μg). Blood pressure and HR were recorded during the rest period and 5 min after the GTN had been administered.

Data analysis
Posttest analysis was carried out using custom-designed edge detection and wall tracking software [26,27]. Our previous detailed analysis of power requirements using this software indicates that, at an alpha level of 0.05, seven subjects are required to ensure 90 % power to detect a 2 % change in FMD [27]. FMD or GTN were calculated as the percentage rise from preceding baseline diameters. The TTP (time to peak) diameter (in seconds) was calculated from the point of cuff deflation to the time of peak postdeflation diameter. Postdeflation shear rate data, derived from simultaneously acquired velocity and diameter measures at 30 Hz, were exported to a spreadsheet SR_{AUC} was calculated for data up to the point of maximal postdeflation diameter (FMD) using the Riemann sum technique for each individual. SR_{AUC} represents the stimulus for FMD [17].

Statistics
The responses were assessed using a two-way repeated measures ANOVA. Where significant interaction was observed, a one-way ANOVA was carried out on each arm separately to identify differences. Results are expressed as means ± S.E.M. A P value <0.05 was considered significant.

RESULTS
The clinical characteristics of the patients are described in Table 1. The majority of the patients were on aspirin, clopidogrel, a statin, an ACEI (angiotensin-converting enzyme inhibitor) and a β-blocker. Efforts were made to avoid changes to the patients’ drug regimes throughout the study. In any event, the within-subjects design, with contemporaneous contralateral limb measures at each time point effectively controlled for any drug effects.

Seventeen subjects completed the pre- and post-scans for the FMD sheath site, and 11 subjects completed all three testing points. For the FMD catheter site (proximal) data, 13 subjects completed the pre- and post-scans, and nine subjects completed all three testing sessions. For the GTN protocol, 15 subjects were tested pre- and post-catheterization, and 11 completed all three testing protocols. The average age of the subjects was 64 ± 10 years, mean blood pressure was 95 ± 15 mmHg and HR was 59 ± 7 beats/min, and the baseline clinical characteristics are shown in Table 1.
There was no significant difference in resting radial artery diameter between arms at the sheath site (distal) prior to catheterization, but diameter was significantly larger ($P<0.05$) in the catheter (proximal) section of the catheterized arm compared to the control (non-catheterized) arm (Table 2). This may relate to the fact that the catheterized arm was typically the dominant right arm. There was no significant difference in $SR_{AUC}$ or TTP between arms or across time.

There was a significant increase in baseline diameter (Table 2) postprocedure in the catheterized arm ($16 \pm 5\%$) in the sheath section only ($P<0.05$). There was no significant change in baseline diameter in the control arm at either the catheter or sheath sites. The recovery (15 ± 1 week) resting baseline diameters were not significantly different from preprocedure in either the catheterized or non-catheterized control arm.

There was no significant difference in FMD or GTN between arms precatheterization (Figure 1). There was a significant reduction in FMD at both the sheath ($P<0.01$) and catheter sites ($P<0.01$) in the catheterized arm (Figure 1), but FMD was not completely abolished at either site. Similarly, there was a significant decrease ($P<0.05$), but not abolition, of the artery response to the GTN postprocedure in the sheath segment of the catheterized arm. These decreases had returned towards preprocedure values by 3–4 months after the catheterization (Figure 2). In contrast, there was no change in FMD or GTN across any of the time-points in the control arm (Figure 1). One patient had an occluded artery postprocedure that remained occluded at the 3-month recovery period.

There was no change in MAP across time (Table 3). There was a decrease in MAP with administration of GTN, which was significant for both arms at the preand posttesting sessions ($P<0.01$).

**DISCUSSION**

The aim of the present study was to determine the impact of radial artery sheath and catheter placement on vascular function in vivo. There are several novel

### Table 1 Clinical characteristics of the study patients ($n=17$)

Values are number of patients (percentages). PCI, percutaneous coronary intervention.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Male 16 (94%)</th>
<th>PCI 17 (100%)</th>
<th>Current smoker 3 (18%)</th>
<th>Ex-smoker 11 (65%)</th>
<th>Diabetes mellitus 3 (18%)</th>
<th>Hypertension on medication 13 (77%)</th>
<th>Current smoker 3 (18%)</th>
<th>Ex-smoker 3 (18%)</th>
<th>Diabetes mellitus 3 (18%)</th>
<th>PCI 17 (100%)</th>
<th>Male 16 (94%)</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Baseline diameter (mm)</th>
<th>Time to peak (s)</th>
<th>$SR_{AUC}$ ($s^{-1} 10^3$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FMD sheath site</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sheath</td>
<td>$2.8 \pm 0.1$</td>
<td>$3.2 \pm 0.1^*$</td>
<td>$2.7 \pm 0.2$</td>
</tr>
<tr>
<td>Control</td>
<td>$2.6 \pm 0.1$</td>
<td>$2.8 \pm 0.1$</td>
<td>$2.9 \pm 0.2$</td>
</tr>
<tr>
<td><strong>FMD catheter site</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sheath</td>
<td>$3.1 \pm 0.1$</td>
<td>$3.4 \pm 0.1$</td>
<td>$2.8 \pm 0.2$</td>
</tr>
<tr>
<td>Control</td>
<td>$2.8 \pm 0.2^*$</td>
<td>$2.8 \pm 0.1$</td>
<td>$2.8 \pm 0.2$</td>
</tr>
<tr>
<td><strong>GTN sheath site</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sheath</td>
<td>$2.8 \pm 0.1$</td>
<td>$3.2 \pm 0.1$</td>
<td>$2.6 \pm 0.2$</td>
</tr>
<tr>
<td>Control</td>
<td>$3.0 \pm 0.2$</td>
<td>$2.9 \pm 0.1$</td>
<td>$3.2 \pm 0.2$</td>
</tr>
</tbody>
</table>

© The Authors Journal compilation © 2010 Biochemical Society
Table 3  MAP during baseline measurements and postcuff release or post-GTN administration preprocedure (Pre), the day following the procedure (Post) and ∼ 3 months postprocedure (Recover) in the catheterized (cath) and non-catheterized (control) arms

Results are presented as means ± S.E.M. Mean arterial pressure (FMD) was assessed in the contralateral arm. Measurements were taken during the baseline period and ∼ 1 min after cuff release or ∼ 3 min post-GTN administration. *Significantly different from baseline (P < 0.05); #Significantly different from Pre.

<table>
<thead>
<tr>
<th></th>
<th>MAP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FMD sheath site</td>
</tr>
<tr>
<td></td>
<td>Baseline FMD</td>
</tr>
<tr>
<td><strong>Cath</strong></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>95 ± 4</td>
</tr>
<tr>
<td>Post</td>
<td>94 ± 3</td>
</tr>
<tr>
<td>Recover</td>
<td>96 ± 4</td>
</tr>
<tr>
<td><strong>Control</strong></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>95 ± 4</td>
</tr>
<tr>
<td>Post</td>
<td>89 ± 4</td>
</tr>
<tr>
<td>Recover</td>
<td>95 ± 5</td>
</tr>
</tbody>
</table>

Figure 2  Changes in FMD (%) within the area of the sheath (sheath site, n = 11) and above the area of the sheath (catheter site, n = 9) and changes in GTN (%) within the area of the sheath (n = 11) in the catheterized arm Pre, Post and ∼ 3 months (Recovery) postprocedure

Results are presented as means ± S.E.M. *Significantly different from Pre (P < 0.05).

findings. This is the only study, to our knowledge, which has assessed both distal and proximal sections of the radial artery in order to determine whether function was affected within the region of the catheter sheath alone or whether catheterization has more generalized effects on vasodilator function. Importantly, continuous measurement of artery diameter and flow allowed us to record the ‘true’ peak in vasodilatation in addition to the SRAUC, which represents the stimulus the artery receives during the FMD test. We also repeated measures ∼ 3 months postcatheterization to determine whether arterial function recovered, and we included within-subjects control measures on the contralateral non-catheterized limb. Our findings indicate that endothelium-dependent function is decreased acutely as a result of catheterization, both within and above the site of sheath, while no changes in arterial function were observed in the non-catheterized control arm at any time-point. This is similar to the work of Heiss et al. [15] who also reported a reduction in brachial as well as radial artery function postcatheterization. The lack of a significant change in the SRAUC, an estimate of the vasodilatory stimulus, supports the notion that the reduction in the FMD was due to an impaired ability of the artery to dilate. Impairment in FMD function was not complete, however, as substantial dilatation occurred despite sheath and catheter placement. Responses largely resolved, i.e. returned to near-baseline levels, following ∼ 3 months recovery.

The external diameter of the catheter sheaths used in this study was 2.7 mm and the mean preprocedure internal radial artery diameter above the insertion site was 2.8 mm (range 2.2–3.5 mm). While we cannot say for certain that the artery was denuded, given the fragile nature of the endothelial monolayer, it is highly likely that placement of the sheath disrupted and/or denuded the endothelial layer, in a similar manner to the induction of endothelial denudation by balloon inflation in animal models [28,29]. The suggestion that we induced endothelial disruption or denudation is supported by the finding that baseline arterial diameter in the sheathed radial segment was larger following the procedure (mean 3.2 mm). Studies of the impact of catheterization and subsequent denudation on radial arterial function are scant. Burstein et al. [13] reported that transradial catheterization impaired FMD immediately postprocedure. Recently, Heiss et al. [15] also observed a reduction in FMD in the radial artery 6 h postcatheterization. In contrast to our findings, values recovered after 12 h in non-smokers. Heiss and
colleagues did not assess GTN function, so they were unable to determine whether smooth muscle function was affected. In contrast, Sanmartin et al. reported no change in postischaemic radial artery dilatation immediately after the procedure, although this was probably due to an initial impairment in function [14]. Interestingly, despite a significant reduction in FMD in this study, there was still some vasodilatation in response to the hyperaemic challenge. This suggests that placement of a large sheath and consequent disruption or denudation of the endothelial layer decreases, but does not abolish, FMD. This is in agreement with some previous studies which have blocked NO function and found only partial reductions in FMD [22–24]. Likewise, eNOS knockout mice have been shown to have preserved FMD responses [25]. In these studies, it was assumed that some compensatory mechanisms, including the release of other endothelium-dependent vasodilators such as prostoglandins [25], may come into play if the endothelial release of NO is impaired or absent. However, our data raise the possibility that there may be a non-endothelium-dependent vasodilatation in response to increased blood flow and shear stress in humans. Future studies will be required to elucidate the contribution of both the endothelium-dependent and independent vasodilators to the conventionally used FMD technique.

In addition to the assessment of endothelial-dependent function, we measured endothelial-independent NO-mediated responses to GTN. As a NO donor, GTN provides a measure of the smooth muscle component of the vascular NO-dilator system. It is possible that insertion of the relatively large sheath into the radial artery results in disruption of the smooth muscle layer of the vessel wall. Decreased GTN responses in the catheterized, but not the non-catheterized, arm supports the contention that sheath-related impacts may not be limited to the intima and is in agreement with the previous literature [13,14]. As with the FMD data, it is notable that sheath placement did not completely abolish GTN-mediated vasodilatation, suggesting that some smooth muscle cell function remains intact despite the placement of the sheath, and that sheath placement impairs but does not eradicate smooth muscle responsiveness to an NO donor. These observations have implications for the use of nitrovasodilators in the clinical setting soon after percutaneous interventions are carried out.

It is possible that the mechanical stretch induced by the sheath insertion played a role in the depressed vascular function, as diameter is an important determinant of the dilator-response magnitude, at least when large differences exist in the resting diameter. In the present study, artery diameter increased modestly as a result of artery catheterization (0.4 mm). Our previous data suggests that this magnitude of change in resting diameter is unlikely to fully explain the change in FMD observed in the present study, with an expected FMD of 10 % in healthy subjects for a radial artery of 2.8 mm and 8 % for a radial artery of 3.2 mm [30].

In addition to measuring endothelial function within the region of the catheter sheath, we also examined endothelium-dependent function in the proximal section of the artery, which was exposed to the catheter but not the catheter sheath. As with the distal section of the artery, we observed a reduction in FMD in the section of the artery only exposed to the catheter. From these data, we can suggest that the impact of catheterization extends beyond the site of sheath placement. This is in agreement with Heiss et al. [15] who reported a reduction in brachial artery function 6 h postradial artery cannulation, although this depression recovered after 12 h in non-smokers and remained depressed in smokers. It is feasible that the catheter itself may damage the endothelium as it is advanced. However, we cannot rule out the possibility that the catheter is associated with localized inflammation or irritation as a mechanism for reduced function in the catheterized arm [31]. Reduced function above the sheath site raises questions about injury to the rest of the arterial tree through which the catheter passes, including perhaps the coronary arteries. Since FMD in the contralateral arm was not depressed, a global impact of catheterization, mediated via inflammation or oxidative stress, seems unlikely.

It is interesting to note that both endothelial and smooth muscle responses return to near-baseline levels following a 3-month recovery period. This is in contrast to the study of Burstein et al. who previously reported that the impairment in FMD following transradial catheterization was still evident 6 weeks after surgery [13]. Our findings that vascular function had recovered ~3 months postprocedure supports that this period may be the minimum required for arterial function to normalize after catheterization and sheath insertion.

**Limitations**

This is the first study of its type to use automated-edge detection and wall-tracking software to derive operator-independent measures of arterial diameter and FMD. The study of two sites within and above the catheter sheath site along the radial artery also provides novel information, as does the repeated measurement following a recovery period. However, the present study also has some limitations. There were a relatively small number of subjects in each group, and we could not get all patients back for the 3-month recovery period. This limitation is somewhat mitigated by our within-subjects design and analysis. We did not control for age, pre-existing vascular disease, history of smoking or drug treatment. However, the use of contralateral arm as an internal control helped to negate this limitation.

**Conclusions**

In conclusion, transradial catheterization results in reversible depression in NO-mediated endothelial and
smooth muscle function in the catheterized arm. This effect is evident in the region of the sheath and also above the site of the catheter sheath, suggesting that both sheath and catheter insertion impact upon the vasculature, possibly via a localized inflammatory or irritant response. Although FMD responses were impaired by sheath placement, they were not abolished, suggesting that some endothelium-independent vasodilator mechanisms may contribute to the vasodilation response to an FMD test in humans. This hypothesis will require further investigation. From a clinical perspective, it is possible that optimizing the function and size of the artery prior to its cannulation may improve the outcome and recovery of the artery. To this end, exercise training has been shown to improve arterial function and induce outward remodelling [32–36], both of which might limit the impact of transradial catheterization and improve the health and recovery of the artery postprocedure.

ACKNOWLEDGEMENTS

We thank Chris Reed for his assistance with the development of the edge detection and wall-tracking software.

FUNDING

D.J.G. received funding support from the National Heart Foundation of Australia [grant number G 08P 3666].

REFERENCES


17 Pyke, K. E. and Tschakovsky, M. E. (2007) Peak vs. total reactive hyperemia: which determines the magnitude of flow-mediated dilation? J. Appl. Physiol. 102, 1510–1519


© The Authors Journal compilation © 2010 Biochemical Society

Received 29 October 2009/22 December 2009; accepted 8 January 2010
Published as Immediate Publication 8 January 2010, doi:10.1042/CS20090548

© The Authors Journal compilation © 2010 Biochemical Society