# New Zealand

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*He has disclosed that he is an investigator for a number of device trials on treating femoropopliteal disease but has no financial interests in these devices.*
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## What is the prevalence of endovascular SFA therapy as compared to surgical?

There is a high prevalence of endovascular SFA therapy in New Zealand, with all TASC A and B lesions and most TASC C lesions treated with an endovascular-first approach. It is rare for patients with these lesions to be offered open surgery, and that really hasn't changed in recent years. With the success of new minimally invasive endovascular techniques, some patients with TASC A to C disease, who would have traditionally been treated conservatively, are now being offered endovascular therapy. This is particularly true for calcified and restenotic lesions.

TASC D lesions are more challenging. In patients who have significant symptoms, a good venous conduit, and reasonable life expectancy, surgical bypass is offered. Other patients are treated via endovascular means.

## How would you describe device availability in your country, both in types of devices and different vendors within each class?

We are very fortunate to have access to a wide range of devices and vendors—almost unsurpassed by any other region. We have been using both drug-coated balloons (DCBs) and stents for many years and atherectomy for selective pathology and arterial locations. In addition, at our institution, we have access to novel devices as part of first-in-man trials, including bioresorbable drug-eluting stents (DES) and Lithoplasty balloon catheters (Shockwave Medical Inc.).

## In what ways does reimbursement (both government and private if applicable) affect device use? Which device classes are most affected?

The vast majority of advanced arterial interventions in New Zealand are performed in government-funded public hospital settings. In this environment, "workhorse" equipment is subject to multiregional product tender, and pricing is highly competitive among vendors. This includes, for example, angioplasty balloons and stents, including drug-eluting versions. However, we can still get access to directed therapies, if clinically indicated, without reimbursement issues causing concern.

## Are there any historic or cultural forces unique to your country that have affected the penetration of endovascular options?

Indigenous cultures in many countries have poorer health statistics and outcomes than other populations. In New Zealand, the indigenous Maori and Pacific Islanders also have higher rates of peripheral vascular disease. However, a well-funded public health system means that there are no major barriers to excellent endovascular interventional treatment outside of any cultural issues.

## How do most physicians receive training in endovascular therapies in your country?

Most physicians performing peripheral endovascular interventions are fellowship-trained interventional radiologists and vascular surgeons, although some interventional cardiologists are also involved. Traditionally, many physicians have followed the pathway I did and spent several years in overseas fellowships, usually in Europe or the United States. More recently, many interventional fellowships are available locally. For example, at Auckland Hospital, we offer two fellowships each year that cover the full range of endovascular interventions.

## What is your personal strategy or algorithm for treating:

- **Short, focal lesions:** Plain old balloon angioplasty (POBA) followed by DCB angioplasty if there is no significant residual stenosis or dissection. In those instances, I use a DES.
- **Long lesions:** In long lesions that are not complex (ie, a long CTO component or calcification), I again start with POBA, followed by DCB angioplasty if

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there is no significant residual stenosis or dissection. Any focal residual lesion after DCB use is then managed with a bare-metal stent (BMS). For more complex, long lesions, I'll primarily stent the CTO component with a DES.

- **Calcified lesions:** I will often use a biomimetic stent (Supera, Abbott Vascular) in this setting, as long as the vessel is well prepared with adequate angioplasty predilatation. In the popliteal artery (especially P2, P3), I prefer atherectomy and DCB use, as long as the calcification is not too severe. In the trial setting, we are using Lithoplasty devices for these lesions.
- **CTOs:** The challenge with CTOs is crossing and reentry, and we have a number of devices to facilitate this when crossing with a hydrophilic wire is unsuccessful. As previously mentioned, long CTOs are often associated with residual stenosis after angioplasty, particularly at the reentry points, so I have a low threshold for DES in this situation.
- **In-stent restenosis:** Drug-eluting technology is the primary strategy. For most cases, this involves a DCB, with a limited role for DES in refractory cases. I do not use atherectomy extensively in this setting, although this may have merit in association with DCB use.
- **Claudicants:** The key issue I bear in mind in claudicants is that this is a lifestyle-limiting symptom, and intervention is not likely to have a major impact on mortality. For that reason, I need to be confident that my intervention is safe and the treatment strategy is associated with good durability. ■