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Q1 You've spoken movingly in the past about growing up in Jerusalem and Bethlehem, training in Bristol and London, and being shaped by powerful mentors at Imperial College London, UK. Looking back, what were the defining moments that steered you toward interventional cardiology?

There were several defining moments, but perhaps the deepest influence was growing up between Jerusalem and Bethlehem, where one becomes aware very early that vulnerability is never only biological. Illness, access, instability, duty, and human dignity all intersect in ways that stay with you for life. My early education, followed by my move to the UK and subsequent medical training in Bristol, helped shape my discipline, curiosity, and strong sense of responsibility. Bristol, in particular, was formative for me, both personally and academically.

My later training in London, especially at St Bartholomew's Hospital, London, and later at Imperial College London, sharpened that further. I was fortunate to be surrounded by outstanding mentors who taught me not only the craft of cardiology, but also the importance of scientific depth. They impressed on me that one should never be satisfied with treating the visible consequence of disease without trying to understand the underlying biology.

Interventional cardiology drew me because it sits at that very powerful intersection between thought and action. It is intellectually demanding, technically exacting, and yet capable of making an immediate difference to a patient in front of you. I found that combination deeply compelling. Over time, it became clear to me that the catheter laboratory was not the end point of my interest, but rather one part of a much larger journey into atherosclerosis, inflammation, imaging, prevention, and ultimately the broader question of how we reduce vulnerability in all its forms.

Q2 You frame your work around the concept of 'vulnerability', from plaque to patient to society to civilisation. How did that framework evolve for you, and how does it shape the way you practice medicine today, both in the cath lab and beyond it?

The framework evolved gradually, but quite deliberately.

My scientific starting point was the vulnerable plaque, the atherosclerotic lesion that is biologically active, prone to rupture, and capable of precipitating myocardial infarction. That remains a central scientific question for me, because if we can identify vulnerability more precisely, we have a better chance of preventing catastrophe rather than simply reacting to it.

But over time, it became clear that plaque biology alone is not enough. Events occur not simply because a lesion exists, but because plaque biology interacts with thrombosis, inflammation, systemic risk, and the broader condition of the patient. That led naturally to the idea of the vulnerable patient, the person whose risk is often greater and more complex than conventional measures alone may suggest.

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From there, the concept widened further. In medicine, one sees very quickly that vulnerability also resides in systems and societies. Delayed access to care, structural disadvantage, conflict, fragmentation of services, interrupted education, and inequality all shape outcomes. So, for me, the idea expanded from vulnerable plaque to vulnerable patient, to vulnerable society, and, indeed, to vulnerable civilisation.

That framework now shapes how I think about science and medicine. The real question is why this patient is vulnerable, and how that vulnerability can be reduced in a sustained and humane way. In the catheter laboratory, it is simply not enough to ask whether I can treat a stenosis. It's a perspective that extends well beyond the cath lab, shaping my work in prevention, cardioimmunology, remote monitoring, service development, and education. Ultimately, medicine should not only rescue patients from acute events; it should also help build resilience at biological, clinical, and societal levels.

Q3 You were an early advocate of the idea that atherosclerosis is not just about cholesterol accumulation but about immune and inflammatory processes. What do you think clinicians still underestimate about the immune system's role in coronary disease, and how might that change prevention strategies over the next decade?

I think the most important point is that this is not a matter of cholesterol versus inflammation. Cholesterol remains fundamental. However, low-density lipoprotein (LDL) only becomes truly dangerous in a biologically meaningful sense when it undergoes modification and becomes antigenic.

At that point, one is no longer dealing simply with lipid deposition, but with an immune and inflammatory process that shapes plaque evolution, instability, and clinical events.

What clinicians and some guidelines may still underestimate is how deeply the immune system is woven into the disease. Atherosclerosis is not a passive storage disorder; it is a disorder of maladaptive homeostasis in which immune pathways are constantly involved. That has implications not only for pathogenesis, but for risk prediction and treatment.

Over the next decade, I think prevention will become more nuanced. We will continue to lower apolipoprotein B (apoB)-containing lipoproteins aggressively, quite rightly, but we will also become better at identifying residual inflammatory and immune-mediated risk. Biomarkers, immune phenotyping, and mechanistic understanding will increasingly help distinguish which patients remain biologically vulnerable despite otherwise acceptable conventional control. The future, I think, lies in integrated prevention: rigorous lipid lowering, better biological characterisation, and more tailored strategies for those in whom inflammation remains active.

Q4 Your group has pioneered molecular imaging approaches from near-infrared fluorescence and PET imaging to nanoparticle-based targeting of oxidised LDL to identify biologically high-risk plaques. How close are we to routinely identifying vulnerable plaque in clinical practice, and what barriers still need to be overcome?

We are undoubtedly closer than we were even a few years ago, but I do not think we are yet at the point of routine clinical identification of biologically vulnerable plaque at scale. Current intravascular techniques are extremely valuable for defining structure and morphology, but the key limitation is that most do not directly interrogate biology. To use the analogy I employed in my lecture, if one wants to know whether a volcano will erupt, it is not enough simply to study its shape; one needs to understand the activity of the lava beneath it.

That is the gap molecular imaging seeks to fill. If we can identify biologically dangerous plaques, those enriched for oxidative modification, inflammation, and active instability, we move much closer to truly preventive intervention. Our work with antibodies to oxidised LDL, imaging probes, nanoparticles, and now newer translational platforms is all directed towards that goal.

The barriers are substantial but surmountable. We need reproducibility, workflow feasibility, cost effectiveness, and, above all, evidence that acting on this information improves outcomes.

It is one thing to generate elegant images; it is quite another to show that these images change clinical decisions in a way that benefits patients. My view is that the first real clinical applications will likely be in selected high-risk populations and specialised centres. That is often how translation begins, but I do believe the field is moving in the right direction.

Q5 Your work on natural antibodies against oxidised LDL suggests some immune responses may actually be protective. Do you see a future where cardiovascular prevention includes immunisation strategies or immune modulation, and what would responsible translation of that science look like?

Yes, I do think there is a credible future for immune modulation in cardiovascular prevention, provided we proceed with scientific discipline. One of the most exciting aspects of this field is precisely that not all immune responses are harmful. Some appear to be protective. Our work, and that of others, suggests that certain antibodies to oxidised LDL may help clear harmful material and may be associated with reduced plaque vulnerability and lower event risk.

That opens the door to a different way of thinking about prevention. Rather than merely suppressing disease, one might strengthen or harness naturally protective mechanisms. But responsible translation is crucial. It would be unwise to make exaggerated claims or to imply that an immunisation strategy for coronary disease is imminent in any simplistic sense.

The path forward must be incremental: mechanistic validation, reliable biomarker work, clear identification of the relevant patient groups, rigorous safety assessment, and then carefully designed clinical trials. In the shorter term, the most realistic applications may lie in immune-informed risk stratification and targeted adjunctive therapies. In the longer term, there may indeed be scope for preventive immunological strategies. But if that happens, it will have to rest on very strong science and a great deal of caution.

Q6 In your recent work on residual risk following myocardial revascularisation, you explored adjunctive pharmacological strategies after percutaneous coronary intervention (PCI). Are we entering an era where the procedure itself becomes just one component of a broader biologically targeted strategy, and how should interventional cardiologists adapt to that shift?

I think we are already in that era. PCI remains a powerful and often indispensable treatment, but it is increasingly clear that it addresses only one dimension of the problem. It treats anatomy and relieves ischaemia, but it does not abolish the underlying biological drivers of risk. Residual lipid risk, inflammatory risk, thrombotic risk, and metabolic risk all remain relevant after a technically successful procedure.

For that reason, revascularisation should no longer be viewed as an isolated act, but as one component of a broader and more biologically informed strategy. The interventional cardiologist of the future will need to be just as comfortable thinking about inflammation, biomarkers, adjunctive pharmacology, and long-term risk modification as about wires, stents, and angiographic appearances.

To my mind, that does not diminish the importance of intervention; it places it in context. The best interventional cardiology has never been merely procedural. It has always combined technical excellence with judgement. What is changing now is that the biological depth of that judgement must increase.

Q7 Your team has demonstrated dramatic reductions in readmissions using home monitoring and AI-supported remote cardiac care. How do you see digital cardiology reshaping interventional practice, particularly for high-risk patients waiting for procedures or recovering afterwards?

Digital cardiology has the potential to transform precisely those periods in which patients are most vulnerable: after acute coronary syndromes, around major procedures, and during the transition from hospital to home. What our work has shown is that intelligently designed remote monitoring can reduce readmissions substantially while preserving safety. That matters enormously both to patients and to health systems.

Digital tools are not a replacement for clinical medicine, but an extension of it. They enable us to maintain continuity, identify deterioration earlier, provide more appropriate reassurance, and intervene before problems escalate into crises. This is especially relevant for

interventional practice in high-risk pathways, whether following myocardial infarction, after PCI, or increasingly in structural interventions.

Advanced monitoring systems may well have an important role within that ecosystem, particularly in signal detection and prioritisation, but I would emphasise that they should remain clinician-supervised and clinically accountable. Technology should support judgement, not replace it. Equally, one must remain attentive to equity. Digital innovation is only truly worthwhile if it narrows vulnerability rather than widening it. So, the challenge is to design pathways that are robust, humane, scalable, and fair.

Q8 You've built cardiac services in Palestine, integrated cardio-immunology clinics, and emphasised that 'work is love made visible'. For younger cardiologists entering a highly technical, high-pressure field, how can they retain a sense of purpose and moral clarity while pushing scientific boundaries, and what do you hope the next generation will do differently from your own?

I think the first thing is to understand that technical mastery, however important, is not enough on its own. Medicine is too demanding a vocation to be sustained by status, speed, or intellectual vanity. One has to remain anchored in purpose.

For me, that purpose has always come back to duty, to patients, and to the idea that one's worth is measured not simply by achievement, but by what one does for others.

Helping build cardiac services abroad and running our Cardiac Directorate at Imperial in the context of the post-COVID NHS, for 5 years, reinforced that in a very direct way. In settings where access is fragile and systems are under strain, one quickly learns that medicine is fundamentally about dignity, solidarity, teamwork, and continuity of care. Service building, training others, and creating structures that outlast you can be as meaningful as any individual scientific paper or procedure. It's what our mentors have told us since medical school: training good people is what matters, and everything else follows.

The phrase 'work is love made visible' matters so much to me, because it expresses something fundamental: Our daily labour, if it is to be worthwhile, must be animated by care. That does not mean sentimentality. On the contrary, it means discipline, honesty, rigour, and a refusal to become indifferent.

What I hope the next generation will do better than ours is to think more integratively. I hope they will be more collaborative across disciplines, more globally minded than we have been, more attentive to prevention, and more willing to engage constructively with global inequity in health and education.

With solid basics, translational science and a fearless appetite to innovate beyond the obvious, the future of medicine will be secure in the hands of this moral and responsible generation that I already look up to in so many different ways.