ME AND MY NEUROLOGICAL ILLNESS

recurrent migraine aura triggered by coronary angiography

A coronary angiogram, but why and how does it cause migraine aura without headache?

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Fifteen minutes after having a coronary angiogram, as a preoperative test before replacement of my ascending aorta in 1998, at 41 years of age (Hankey 1999), I was lying on a trolley in the recovery room of the catheter suite, looking at the ceiling, and noticed something moving in the inferior temporal crescent of my right visual field. It was quite clear, like water, swirling about, and painless. I thought it was a problem with my right eye, and closed each eye in turn and could not convince myself that I could see it with my left eye. Over the next few minutes, the image gradually built up, and spread superiorly and medially toward the centre of my visual field. Again I closed each eye in turn and the image was now visible in the right visual field of both eyes. After about 10 min, I began to go blind in the extreme outer crescent of the right visual field

of each eye where the image of swirling water had originated from. Meanwhile, this image had continued to migrate more centrally across the right visual fields of both eyes, with the scotoma following behind it. I held my hands out in front of me (towards the ceiling), and moved the index finger of my right hand whilst bringing my hand from the periphery to the centre of my right visual field. There was definite loss of a large outer crescent of the right visual field of both eyes.

I wondered if the symptoms were caused by ischaemia or infarction in the anterior striate cortex of the left occipital lobe, perhaps due to thromboembolism from the catheter tip, or an intimal flap in the aortic arch that may have been dissected from the media by the catheter. Or could this be a peculiar adverse reaction to the contrast agent, triggering the visual hallucination and scotoma of a migraine aura, as has since been described after cerebral angiography (Beckman *et al.* 2001)?

A nurse noticed my antics (of testing my visual fields) and asked me 'what on earth was I doing!' I explained that I couldn't see clearly to one side. She said 'that sometimes happens' and called the cardiologist. He, however, said he had never heard of such symptoms after coronary angiography. As a relatively new young consultant, I was embarrassed to be mentioning to the hospital's senior cardiologist, and a colleague, and good friend of my father, a possible neurological complication of his procedure. I suggested to him that my symptoms were those of a coincidental first-ever migraine aura, emphasizing that my sister was a migraineur, and that I was probably genetically predisposed (Ducros *et al.* 2002). I insisted that my vision would recover, and that I would be appropriately 'rewarded' with a thumping headache for creating such a scene. He seemed satisfied with the diagnosis and said that I 'would be alright'.

Indeed I was. After about 25 min, my vision slowly returned to normal, but there was no headache. I was relieved and grateful. However, my neurotic neurological nature provoked wild speculation of an underlying structural lesion in the left occipital lobe, such as an arteriovenous malformation, that may have been linked somehow to the vascular abnormality of my aorta [hereditary aorto-annular ectasia, causing an ascending aortic aneurysm, and functional aortic incompetence (Hankey 1999)]. Rather than request further assessment by a neurologist, or brain imaging, I was more preoccupied with the

forthcoming potential risks of surgery on my ascending aorta, and preferred to await nature's fate.

The operation went well.

Ever since, I have continued to experience recurrent, stereotyped, short-lived, episodes of a similar visual disturbance, without headache. They occur irregularly, on average about once a week, and most commonly (but not always) about 20 min after I have swum about 1.5–2.0 kilometres. Although I have never undergone any brain imaging, my fears of a left occipital lobe lesion were dispelled with the subsequent occasional occurrence of scintillating scotomas affecting my left visual field, instead of my right.

The frequent association of the onset of the episodes soon after exercise (swimming) temporarily stimulated further wild speculation that I could have a metabolic disorder such as mitochondrial encephalopathy with lactic acidosis and stroke-like episodes (MELAS) (Montagna *et al.* 1988) or even some cerebral arteriopathy such as cerebral autosomal dominant arteriopathy with subcortical infarcts and leucoencephalopathy (CADASIL) (Verin *et al.* 1995). However, the failure of any other features of these rare entities to evolve over the past 5 years, and the benign prognosis of my syndrome, have extinguished any further flights of imagination.

In the meantime, I have not needed to take any migraine prophylactic drugs, because the symptoms are so short-lasting, and when they do occur I just ensure I am not driving a car or playing ball sports (e.g. tennis) for pride or fiscal gain, and wait for them to go away.

I am comfortable that the episodes are migraine aura without headache (Kunkel 1986), but I still don't understand how the coronary angiogram 'kick-started' it all. Was it a coincidence, and simply another manifestation of getting older (Fisher 1980)? Or was there some mechanical, chemical, immunological, or haemodynamic factor, or combination of factors, such as catheter-induced or contrast-induced release of vasoactive substances, such as serotonin or nitrous oxide, that precipitated them (Beckman *et al.* 2001)? And if so, why wasn't this a single, 'one-off' migraine, and why does it continue to recur? Was the angiogram enough to lower my threshold for migraine forever? I have never sought a neurological consultation to explain any of this. Perhaps it is now time to do so. Any thoughts?

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