

Winston Churchill's cerebrovascular disease: small vessels with big implications

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Winston Churchill is among the most significant political figures in British, European and world politics in the twentieth century. Best known for his leadership of Britain in World War two, and for his extraordinary political and personal determination, Churchill faced some of his greatest challenges from the consequences of cerebral small vessel disease, between 1949 and his death in 1965.

In three articles in the Journal of the Royal Society of Medicine, Scadding and Vale provide clear and detailed accounts, taken from multiple contemporary sources, of Churchill's strokes in 1949 and 1953, and other manifestations of cerebrovascular disease from 1950-1952. The articles elegantly remind us of the characteristic clinical features of ischaemic strokes due to cerebral small vessel disease (where occlusion of a small perforating artery typically causes a distinct "lacunar" clinical syndrome, most commonly a "pure motor stroke" with hemiparesis affecting the face, arm and leg) but also highlight important gaps in our current knowledge and treatments for this form of cerebrovascular disease. Small vessel occlusion accounts for about one in four ischaemic strokes;¹ although the prognosis for recovery is generally good, a proportion of patients progress acutely in the first few days after onset, particularly in the domain of motor function.² This notable clinical feature of small vessel strokes remains a clinical and research challenge today.

Winston Churchill's behaviour and account at the time of onset of the first stroke in 1949 suggest that the symptoms progressed over a matter of hours (first removing the ring, then mentioning a cramping feeling, then having writing difficulty). The progressive onset is typical of small vessel occlusion,² and the clinical syndrome suggested involvement of the internal capsule and thalamus. The second stroke in 1953 also showed progressive and fluctuating onset in the first 24 hours, with the involvement of the face, arm and leg indicating a "pure motor

stroke" due to a lesion in the corticospinal projections within the posterior limb of the internal capsule. This characteristic progression and fluctuation in the first day or so from the onset of small vessel occlusion are still not at all well understood. Suggested mechanisms include propagation of thrombosis, haemodynamic factors, excitotoxicity, inflammation, oedema, or conduction block. Some evidence suggests that patients with branch artery atherosclerotic disease affecting the parent artery (e.g. the proximal middle cerebral artery from which the lenticulostriate perforators arise) with proximal occlusion at a perforator origin are more likely to clinically progress than those with more distal occlusion of a perforating artery;³ this is hypothesised to result from either thrombus propagation along the perforator (with sequential progressive occlusion of lateral smaller branches) or reduction in perfusion (causing ischaemia spreading from the distal to proximal tissue irrigated by the perforating artery). Some evidence from perfusion MRI supports a role for haemodynamic failure in progressive lacunar syndromes.⁴ Another hypothesis is that peri-infarct oedema might exert pressure on neighbouring perforating branches, causing them to occlude, thus extending the infarct.⁵

These mechanisms are not just of academic interest, because they highlight the persisting gaps in our understanding and a lack of specific treatments. If thrombus propagation is important, then acute anticoagulation or thrombolysis might be reasonable in progressive small vessel stroke. However, although intravenous thrombolysis is considered beneficial in all types of ischaemic stroke, there is limited evidence of specific efficacy in small vessel occlusion.⁶ Endothelial dysfunction and platelet aggregation might logically respond to antiplatelet therapy while haemodynamic failure might require measures to improve perfusion therapy. Antiplatelet therapy with GP IIb/IIIa inhibitors (which can reduce thrombus growth and prevent reformation by competitive inhibition with fibrinogen) has shown some promise in small studies of progressive small vessel occlusion.⁷ Finally, although mechanical thrombectomy has

revolutionised the treatment of ischaemic stroke due to large vessel occlusion in the anterior circulation, it is not feasible to apply this method to small vessel occlusion. Because there is still no specific treatment targeting small vessel occlusion, this is clearly an important topic for future clinical trials.

These accounts of diagnosis made by careful clinical observation also remind us of the advances in brain imaging that underpin modern stroke diagnosis and treatment. The authors describe accurate anatomical localisation based on careful clinical assessments, but a small haemorrhage could not be discounted entirely in either of these strokes, given Winston Churchill's heavy alcohol intake and hypertension which, though associated with all stroke types, are the strongest risk factors for intracerebral haemorrhage. It is well known that a small deep haemorrhage can exactly mimic a classical lacunar syndrome. Even computed tomography – introduced in the 1980s - has limited sensitivity to detect small vessel occlusions (especially in the first hours), while MRI- introduced in the 1990s – is extremely sensitive to acute ischaemia within minutes of arterial occlusion.⁸ Recent advances in high field MRI now potentially allow visualisation of individual small vessels and occlusions, while blood-sensitive sequences can detect even small and chronic areas of bleeding, including cerebral microbleeds.⁹

These accounts vividly show how occlusion of even tiny arteries can have profound personal and, in Winston Churchill's case, political, implications. Although the first stroke in 1949 was minor and effectively concealed, seemingly not affecting his re-election in 1951, the subsequent consequences of small vessel disease undoubtedly had a profound impact on him. The transient attacks and cognitive disturbances occurring between 1950 and 1952 were clearly noted by Churchill and those that knew him, suggesting progressive neurological impairment,

although he continued to function well in office. The later stroke of 1953 certainly endangered Churchill's premiership and indeed the viability of his political career. It seems very likely that in addition to the generally favourable prognosis of strokes due to small vessel occlusion, Churchill's unique determination and resilience were key factors in helping him to continue to function at the highest level within weeks of the stroke onset in 1953, despite the cumulative effects of cerebral small vessel disease over the preceding years.

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