Chapter

Hypertension with a Focus on Comprehensive Magnetic Resonance Imaging

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Abstract

Arterial hypertension is a leading cause of mortality, affecting at least a quarter of the adult population, with its effects having devastating consequences to the global economy. Unfortunately, the underlying causes and pathophysiology of the disease often remain unclear. Ongoing research in this important field investigates the mechanisms involved in the genesis of hypertension. Magnetic resonance imaging is a well-established imaging technique that is widely used for anatomical organ and vascular evaluation. According to the latest European Society of Hypertension (ESC) guidelines, cardiovascular magnetic resonance can be used in the assessment of hypertensive patients. But the authors advocate a more comprehensive and multisystem use of the varied and novel sequences of MRI scanners to provide an even better understanding of the development of hypertension and its consequences. The extensive and detailed data that can be derived, with the additive focus on the concept of the 'selfish brain hypothesis', might further assist us in altering and providing a more individualised therapeutic approach to one of the greatest non-communicable causes of human mortality and morbidity.

Keywords: hypertension, cardiac magnetic resonance, electrocardiogram, aortic disease, pathophysiology

1. Introduction

Hypertension has been identified as a disease for more than 150 years [1] and is a major contributor to mortality affecting at least a quarter of the adult population [2] with devastating financial effects on national healthcare systems worldwide [3].

Several risk factors for the development of persistently elevated BP, including genetic variations, age, obesity, insulin resistance, high alcohol consumption, increased sodium intake and stress, have been identified and are well established. Unfortunately, despite the leading role of hypertension in global mortality, the detrimental effects, and the enormous economic burden it carries [3–5], the primary causes and predictive factors for the development of hypertension in >90% of patients remain elusive [4–6].

This could explain the fact that, despite various antihypertensive medications, there is poor blood pressure (BP) control in >50% of hypertensive patients [7].

Given that essential hypertension is one of the major modifiable cardiovascular risk factors, understanding the pathophysiology and uncovering its possible root causative mechanism(s) would have a tremendous public health impact. This, of course, could lead to more efficient and bespoke antihypertensive treatments, including prediction or even the prevention of the development of the disease and its detrimental consequences altogether.

Furthermore, according to recent statistics from the 2014 Health survey for England, hypertension is refractory to treatment in ≤20–30% of cases despite the availability of numerous classes of antihypertensives, and finding a primary cause could improve treatment of such refractory hypertension.

As per Hypertension European Society of Cardiology (ESC) guidelines, patients should be thoroughly screened for potentially treatable secondary causes, such as aortic coarctation, adrenal adenomas, phaeochromocytoma, renal artery stenosis, occult chronic renal disease, and evidence of hypertension-mediated organ damage (HMOD) [8].

This chapter has a focus on the use of magnetic resonance imaging (MRI) which can be introduced, currently in appropriately selected individuals, as a safe and effective screening modality for secondary hypertension and end-organ damage evaluation without the use of ionising radiation imaging. [9]

Cardiac MRI (CMR) is the gold standard non-invasive imaging technique for the assessment of cardiac anatomy and function [10], but MRI is also well established in cerebral and cerebrovascular imaging, aortic and abdominal visceral assessment. Aside from anatomical delineation, there are numerous sequences that are regularly employed for detailed tissue characterisation, tissue mapping, myocardial strain imaging, myocardial replacement fibrosis and infarction evaluation, contrast and non-contrast angiography, and phase contrast vessel flow imaging [11–13].

By combining these sequences, comprehensive imaging protocols can be established and the patient investigated in a single hospital episode; this may, therefore, alleviate the need for and higher cost of other multiple investigations, for example, echocardiography, abdominal/renal ultrasound, carotid and vertebral duplex sonography, computed tomography (CT) angiography, etc. [9].

MRI is not touted as a substitute for a detailed history, physical examination, and the usual necessary metabolic/biochemical investigations e.g. endocrine tumours may not always be in typical location.

2. Hypertension and cardiovascular magnetic resonance imaging

2.1 Hypertension and the ‘selfish brain hypothesis’

There is a well-documented relationship between increased sympathetic nerve activity and hypertension but the primary cause is still to be established [14]. Early on, one of the proposed pathologic hypotheses has been that an increase in BP is an essential response to thickened and narrowed blood vessels to provide more blood supply to organs. This line of pathologic investigation has been mired in the classic difficulty of determining if the arterial luminal narrowing is the cause or effect of hypertension.

The effect of hypertension on the cerebrovascular structure is often described through the pathophysiology of vessel remodelling and decrease in luminal diameter leading to decreased blood flow [15–17]. But is it possible that the reverse hypothesis could be true whereby cerebral vessel remodelling precedes and is even a cause of hypertension? The answer to this, we believe, is 'yes'.

The archaic term 'vasomotor center', currently known as the rostral ventrolateral medulla, is the area involved with the basal sympathetic activity and supplied by the vertebral artery; disruption of vertebral artery flow may increase systemic BP.

At the beginning of the twentieth century using canine models, Harvey Cushing indicated that reduced cerebral blood flow due to increased intracranial pressure led to increased blood pressure [17]. Later, animal and post-mortem human studies by Cates and Dickinson proposed that narrow vertebral arteries lead to brain stem hypoperfusion and subsequently to a vital increase of blood pressure as a protective mechanism to maintain cerebral blood flow to the brain stem in the case of stenotic vertebral arteries; this theory is known as Cushing's mechanism or the 'selfish brain hypothesis' of hypertension. This mechanism triggers sympathetic overdrive and hypertension to maintain brain stem perfusion [15, 16].

Using magnetic resonance (MR) angiography and MR phase-contrast imaging, our group has been investigating the association of the 'selfish brain hypothesis' with high BP in conscious adults. The data has shown, for the first time, that congenital variants in the cerebral circulation may be the trigger for hypertension rather than the result [18].

3-dimensional MR angiography can help to illustrate the cerebral anatomy of the circle of Willis and the vertebral arteries. **Figure 1** shows normal anatomy.

The higher prevalence of cerebrovascular variants whereby vertebral artery hypoplasia/absence (VAH) and incomplete posterior circle of Willis (ipCoW) is strongly associated with increased cerebrovascular resistance and reduced cerebral blood flow in hypertensive patients in comparison to healthy adults [18]. We have also shown this in a large cohort of young-onset hypertensive patients [19] with a possible link between the 'selfish brain' hypothesis (**Figure 2**). Cerebral blood flow can also be measured at mid-neck level prior to the common carotid artery bifurcation; in

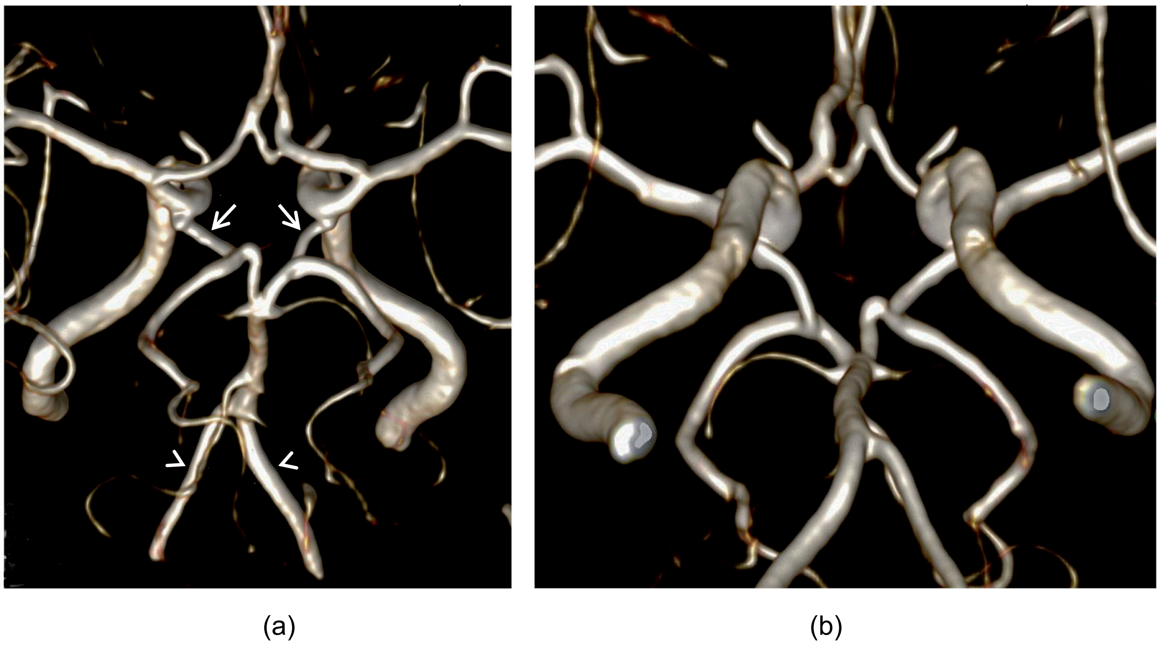


Figure 1.
Normal anatomy. (a) – superior view and (b) – inferior view, showing an intact circle of Willis with normal-sized posterior communicating arteries (white arrows) and normal-sized vertebral arteries (white arrowheads).

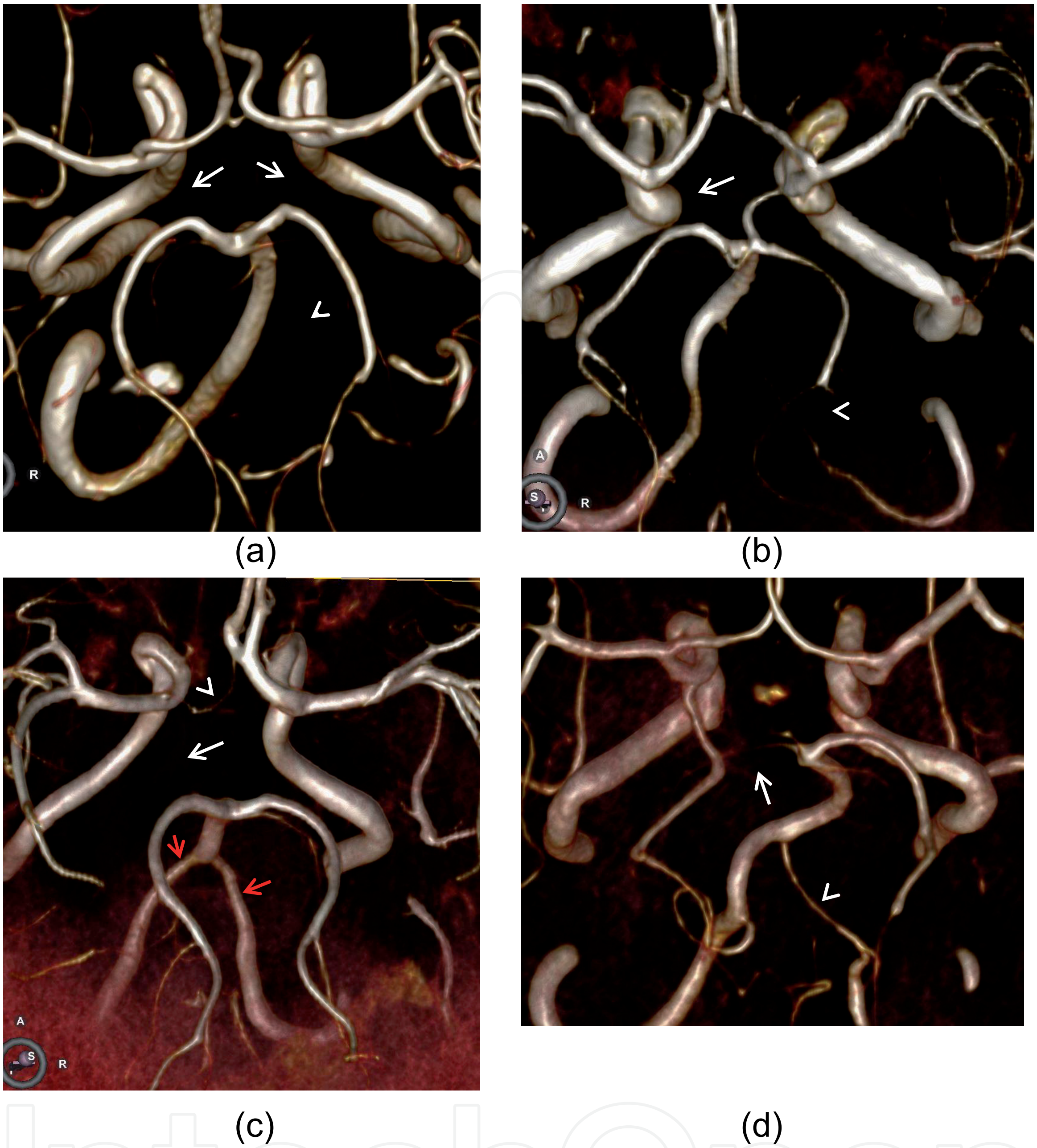


Figure 2.
Variant circle of Willis anatomy found in patients with young-onset hypertension. Superior projection 3-dimensional MRI angiography. (a) – Bilateral absent posterior communicating arteries (white arrows) and absent right vertebral artery (white arrowhead). (b) – Absent left posterior communicating artery (white arrow) and severely hypoplastic, interrupted right vertebral artery (white arrowhead); left vertebral artery stenosis in the V4 segment is also noted. (c) – Absent left posterior communicating artery (white arrow), bilateral small calibre distal vertebral arteries just prior to the basilar arterial confluence (red arrows); note also the severely hypoplastic left A1 segment of the left anterior cerebral artery (white arrowhead). (d) – Foetal-type left posterior cerebral artery (PCA) with absent left P1 segment of the PCA (white arrow), and a hypoplastic right vertebral artery (white arrowhead).

the presence of upstream anomalies of the posterior circulation, the split percentage vertebral arterial flows are significantly reduced.

We have found similar results in a cohort of persistently hypertensive patients post-coarctation repair which could guide subsequent treatment [20]. According to this study, cerebrovascular variants of the posterior circulation as manifested by VAH + ipCoW are more common in patients with repaired coarctation (CoA) and are independent predictors of hypertension or difficult to treat hypertension post-CoA repair in contrast with age and specific type of repair. This may, of course, affect interventional treatment strategy in the form of aortic arch stenting and whether to cover the subclavian artery or a variant arch origin of the vertebral artery in the context of upstream variants that might predispose to or cause hypertension; thus, an intervention intended to treat hypertension may in fact exacerbate the problem unless an appropriate alternative or hybrid arch vessel revascularization strategy is employed.

This is a strong indication that the increase in cerebrovascular resistance caused by congenital cerebrovascular variants is actually the trigger for the increase in sympathetic nerve activity and hypertension. This finding is in accordance with the theory proposed by Dickinson back in 1959. Additionally, this study interestingly showed that cerebral blood flow was normal in hypertensive patients without treatment but diminished in those on treatment with controlled blood pressure. This observation may have important implications in the treatment strategy for hypertensive patients and may change the diagnostic and therapeutic approach; indeed, some patients rendered 'normotensive' may present with soft occult neurological signs of poor concentration, impaired memory, or 'brain fog'. Further longitudinal studies are needed to confirm these results.

2.2 Hypertension and the electrocardiogram

Left ventricular hypertrophy (LVH) is a common finding in hypertensive patients due to the increased workload of the left ventricle [21]. The presence of LVH in hypertensive patients is known to have significant implications in prognosis and treatment strategies [22–25]. The 12-lead electrocardiogram (ECG) is widely used to detect LVH [8] and it is well validated against echocardiography [26].

As per European Society of Cardiology (ESC) guidelines, a 12-lead ECG is required for the assessment of hypertensive patients and specific ECG criteria for left ventricular hypertrophy are well established [8] and validated against echocardiography [26].

The diagnostic performance of the above criteria against CMR, the gold standard, non-invasive technique for the assessment of left ventricular mass (LVM) can be evaluated.

It is also well documented that hypertension frequently coexists with obesity [27]. Due to discrepancy between previous echocardiography studies about the effect of obesity on the ECG capacity of detecting LVH, the ECG criteria for LVH can be recalibrated against CMR measurements by using the steady-state free precession sequence. This study also went through the impact of obesity, over the diagnostic performance of ECG in detecting LVH, thus creating new novel obesity-specific partition values to increase the diagnostic accuracy of ECG in this specific patient's category [28].

In hypertensive patients, the left ventricular ECG strain pattern (≥1 mm concave downsloping ST-segment depression and T–wave inversion in the lateral leads) [29] is associated with increased cardiovascular risk in hypertensive individuals [30] with the underlying mechanisms being unclear.

A great advantage of CMR is its unique ability for advanced myocardial tissue characterisation [11].

Myocardial fibrosis is an established histologic element of hypertensive heart disease; the presence is known to adversely affect prognosis [31]. Left ventricular systolic dysfunction in hypertension, characterised by myocardial strain impairment, has been documented in previous CMR studies [32].

Taking the above facts into consideration using these CMR techniques, a strong association between ECG strain pattern, increased interstitial fibrosis and myocardial systolic dysfunction [33] can be demonstrated; the ECG strain pattern in hypertensive patients is linked to significant LVH and interstitial fibrosis. There is also an association between ECG strain and significant impairment of circumferential strain, even with preserved left ventricular ejection fraction (LVEF) [34].

Obesity is also strongly associated with a left atrial enlargement (LAE), an index of diastolic dysfunction [35], and also a common finding in hypertensive patients, identifiable on ECG [36]. LAE is an index of diastolic dysfunction [35] with important prognostic and therapeutic implications; [37] the ECG may demonstrate LAE [36]. The diagnostic performance of the ECG in identifying LAE has also already been validated against echocardiography [38].

The diagnostic accuracy of ECG criteria of LAE against the CMR gold standard and the possible effect of obesity was explored; [39] all the ECG criteria are more specific than sensitive at identifying LAE and less specific when obesity is present. According to these findings, clinicians should not rely solely on ECG for the exclusion of LAE, and always take into consideration the patient's body mass index (BMI).

2.3 Hypertension and left ventricular hypertrophy

Systolic hypertension increases the LV afterload because the LV must work harder to eject blood into the aorta; the aortic valve will not open until the pressure generated in the left ventricle is higher than the elevated blood pressure in the aorta. In the presence of chronic afterload increase, the LV receives constant mechanical stress which results in hypertrophy [20].

Different phenotypes of hypertensive heart disease (HHD) are well-documented [40]. Even though asymmetric patterns of LVH have been previously investigated with 2-dimensional echocardiography, [41] CMR has shown the prevalence of asymmetric hypertrophic patterns in hypertension [42], a pattern previously described with aortic stenosis [43].

There is an increasingly well-recognised spectrum of hypertrophic LV patterns in hypertensive heart disease with variable cardiovascular prognosis and unknown pathophysiology [40]. Using multiparametric CMR, the significant differences between these phenotypes may explain the varying cardiovascular prognosis and change the therapeutic process [44].

There are three well-defined LV phenotypes—normal structure (normal LV mass and relative wall thickness), concentric remodelling (normal LV mass with increased relative wall thickness and elevated mass/volume ratio), and LVH (symmetric, asymmetric, and eccentric) [40]. Eccentric and concentric LVH is linked to significant intracellular and interstitial expansion and strain impairment. This could explain the varying cardiovascular prognosis following each hypertensive left ventricular phenotype and have an impact on hypertension treatment (**Figure 3**) [44].

The presence of LVH in hypertensive individuals can have a differential diagnosis of hypertensive heart disease (HHD) and hypertrophic cardiomyopathy (HCM); it can be difficult to distinguish between them. Novel predictors of HHD over HCM have been identified [45].

Hypertrophic cardiomyopathy (HCM) is defined by left ventricular end-diastolic thickness ≥ 15 mm, not solely explained by abnormal loading conditions [46]. As asymmetric LV phenotype is commonly seen in both HHD and HCM, it may be difficult to distinguish between the two pathologies; it is acknowledged that HCM and HHD can, of course, co-exist. The investigation has indicated that an acute aortoseptal angulation (angle between a line drawn along the RV side of the interventricular septum and

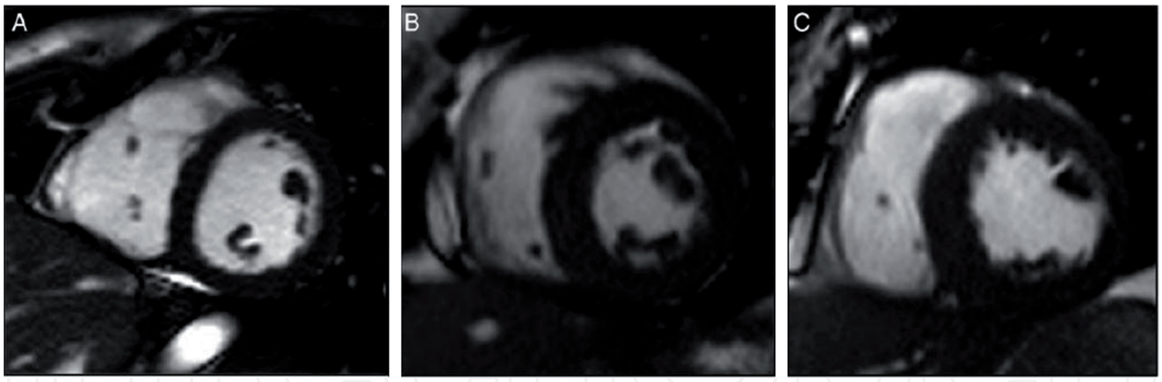


Figure 3.
Forms of hypertensive heart disease. (A) – Normal, (B) – concentric LV hypertrophy, and (C) – asymmetric LV hypertrophy.

a line drawn through the long axis of the aortic root; reflecting a reduced angle from ~130 degrees to 90 degrees or less) and reduced aortic distensibility advocate over HHD [45]. This reduction in aortoseptal angulation is interesting in that the altered LV outflow tract geometry may cause a greater effect on the basal interventricular septum in a region of increased wall stress during ventricular systole, thus contributing to the consequent asymmetric hypertrophy (**Figure 4**).

The reduction in the aortoseptal angle is a complex phenomenon; it likely occurs in part secondary to ageing, hypertensive, and/or atherosclerotic thoracic aortic/

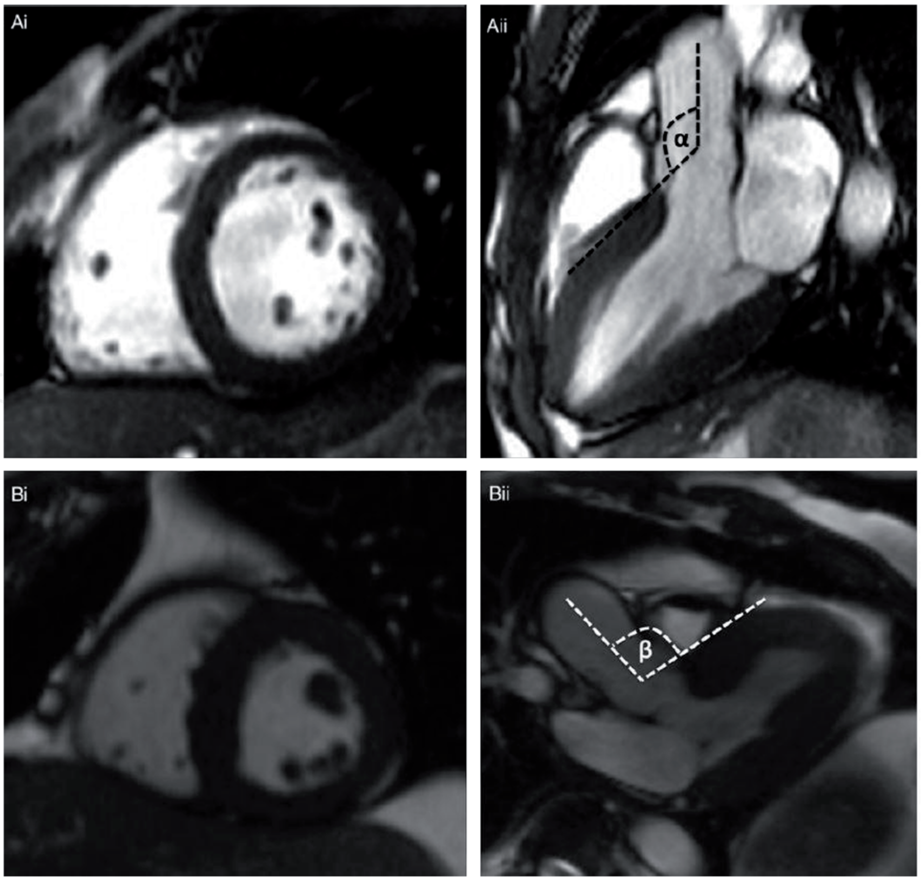


Figure 4.
Aortoseptal angulation. Aortoseptal angle is measured from the 3-chamber steady-state free precession cine at end-systole. Ai – hypertensive patient with no LV asymmetry, Aii – aortoseptal angle = 123 degrees. Bi = hypertensive patient with LV asymmetry, Bii – aortoseptal angle = 91 degrees.

aortic root dilatation and even in the context of obesity with raised hemidiaphragms secondary to increased visceral fat [41]. According to these findings, the diagnosis of HCM when HTN is present should not be based only on wall thickness [42].

With the use of CMR's unique tissue characterisation properties, one can introduce new discriminators for the assessment of hypertensive patients with HCM phenotypes—increased indexed LV mass, absence of systolic anterior motion of the anterior mitral valve leaflet/apparatus (SAM), and absence of mid-wall fibrosis are in favour to HHD [45].

The role of LVH on LVEF and myocardial shortening can be explored by using CMR strain measurements (**Figure 5**), with findings suggesting that LVEF is a poor index of systolic function in hypertensive patients in the setting of LVH [34].

End-diastolic and end-systolic endocardial and epicardial contours are drawn and propagated over the cardiac cycle commonly performed from two long-axis cines to calculate global longitudinal strain and strain rate or from the two chambers' short-axis cines to calculate radial strain. Feature tracking software extracts 3-d LV coordinates to allow measurement of LV strain and strain rate curves.

Hypertensive heart disease is usually considered a diastolic disorder since it often develops with preserved LVEF. Investigating the pathophysiology of LVH and the association with LV function using segmental engineering strain measurements derived from CMR indicate that end-diastolic wall thickness (EDWT), long axis shortening (LAS), and mid-wall circumferential fractional shortening (mFS) are linked to LVEF independently and significantly; this study supports findings from a previous CMR

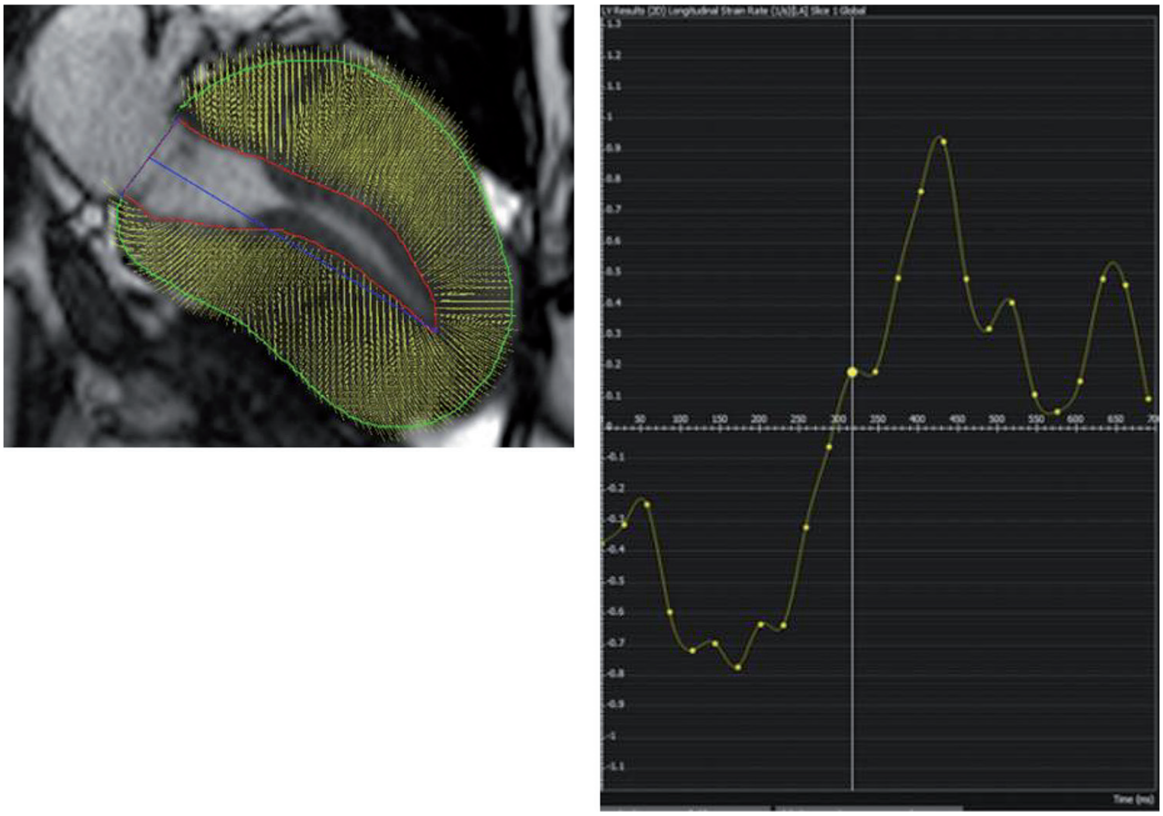


Figure 5.
Strain imaging in cardiac MRI. End-diastolic and end-systolic endocardial and epicardial contours are drawn and propagated over the cardiac cycle. Feature tracking extracts 3d LV coordinates to allow measurement of LV strain and strain rate curves.

study [32]. Increased EDWT and reduced myocardial fractional shortening lead to maintenance of absolute wall thickness (AWT) and thus preserved LVEF [34].

2.4 Hypertension and aortic disease

Aortic diseases are a major cause of cardiovascular morbidity and mortality [47]. Arterial stiffness is considered a major risk factor affecting the prognosis of hypertensive patients [48]. MRI can readily assess markers of aortic stiffness, pathological dilatation, vessel compliance, volume, and velocity of flow. Aortic stiffness is assessed by the measurement of distensibility using cross-sectional aortic area and diameter changes with the cardiac cycle and the simultaneous assessment of thoracic aortic pulse wave velocity (PWV) in the aortic arch; an increase in PWV may reflect increased aortic stiffness (**Figure 6**).

There are differences in myocardial intracellular and extracellular structure and a possible association with aortic function; there are differences in the prevalence of interstitial myocardial fibrosis, aortic distensibility and compliance, and myocardial circumferential strain between the varied hypertensive patterns, for example, concentric remodelling is linked to increased aortic stiffness [44].

Hypertension is often the common denominator in acute aortic syndrome (AAS) which encompasses a spectrum of entities from intramural hematoma, aortic dissection, and penetrating ulceration [49]. The treatment of AAS might include thoracic aortic stent grafting [50].

Given the above observations with respect to the 'selfish brain hypothesis', one might thus reflect on the stent graft positioning and the subsequent clinical follow-up of these patients who presented with a major vascular clinical consequence of chronic hypertension; if the left subclavian artery and thence left vertebral artery are covered by the stent-graft deployed to improve thoracic aortic dissection haemodynamics and/or prevent progressive aortic dilatation, this might have adverse consequences to hypertension control in the context of potential upstream deficiencies in the posterior cerebral circulation as described previously; thus, such positioning may potentially augment the very disease process that caused the AAS in the first place. This should be a subject of future work.

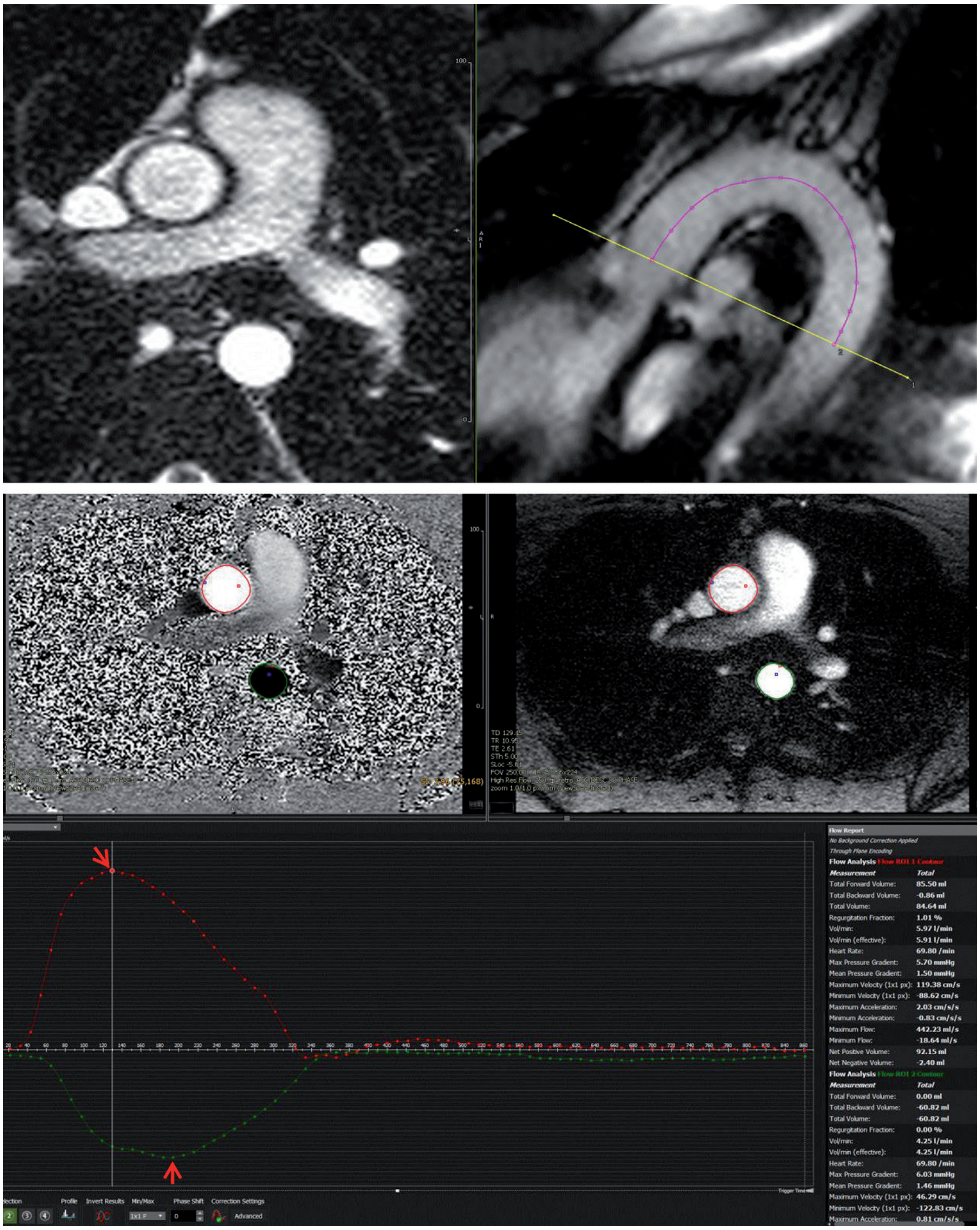
The prevalence of aortic coarctation (CoA) is ~4/10,000 live births [51] with arterial hypertension is considered a common complication (**Figure 7**) even post successful operative repair [52].

In both the above clinical entities, one might pay additional consideration to the cerebrovascular anatomy when the repair is planned for the reasons stated.

Furthermore, MRI can readily follow-up patients with chronic aortic dissection and also has the added advantage of being able to assess dissection flap dynamics which can adversely affect branch vessel perfusion. The MRI assessment of post-stent grafted aortas can be markedly affected by the nature and material of the graft material itself; some metallic artefacts can be catastrophic and therefore non-diagnostic in terms of image quality.

2.5 Concluding remarks and future perspectives

A thorough screening for secondary causes and asymptomatic organ damage is recommended for hypertensive patients according to hypertension ESC guidelines (**Figures 8-12**).



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Flow Report
No Background Correction Applied
Through Plane Encoding
Flow Analysis Flow ROI 1 Contour
Measurement                    Total
Total Forward Volume:          85.50 ml
Total Backward Volume:         -0.86 ml
Total Volume:                  84.64 ml
Regurgitation Fraction:        1.01 %
Vol/min:                       5.97 l/min
Vol/min (effective):           5.91 l/min
Heart Rate:                    69.80 /min
Max Pressure Gradient:         5.70 mmHg
Mean Pressure Gradient:        1.50 mmHg
Maximum Velocity (1x1 px):     119.38 cm/s
Minimum Velocity (1x1 px):     -88.62 cm/s
Maximum Acceleration:          2.03 cm/s/s
Minimum Acceleration:          -0.83 cm/s/s
Maximum Flow:                  442.23 ml/s
Minimum Flow:                  -18.64 ml/s
Net Positive Volume:           92.15 ml
Net Negative Volume:           -2.40 ml
Flow Analysis Flow ROI 2 Contour
Measurement                    Total
Total Forward Volume:          0.00 ml
Total Backward Volume:         -60.82 ml
Total Volume:                  -60.82 ml
Regurgitation Fraction:        0.00 %
Vol/min:                       4.25 l/min
Vol/min (effective):           4.25 l/min
Heart Rate:                    69.80 /min
Max Pressure Gradient:         6.03 mmHg
Mean Pressure Gradient:        1.46 mmHg
Maximum Velocity (1x1 px):     46.29 cm/s
Minimum Velocity (1x1 px):     -122.83 cm/s
Maximum Acceleration:          0.81 cm/s/s
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Figure 6.
Aortic arch assessment. Ascending and descending aortic areas are determined in end-diastole and end-systole (top left); this can give an indication of pathological dilatation and also of wall compliance. At the same cross-sectional level, the sampled aortic arch length is determined (top right) and breath-hold high temporal resolution phase-contrast CMR flow assessment is performed (middle); this enables generation of time - velocity/flow curves (bottom) from which can be derived additional data, such as the aortic pulse wave velocity. Red arrows point to the time (milliseconds) at peak velocity in the ascending aorta (red curve above) and the descending aorta (green curve below); then velocity (m/s) is the distance (mm)/time (ms).

As previously mentioned, echocardiography and abdominal/peripheral arterial ultrasound are used widely as first-line imaging tools even though the value of CMR is well-recognised [8].

CMR is known to be the gold standard non-invasive imaging technique for the assessment of cardiac anatomy and function, [10] with unique tissue characterisation properties, [11] and it does not require subjecting the patient to ionising radiation, is capable of imaging multiple organs and the vasculature with high diagnostic accuracy in a single session [9].

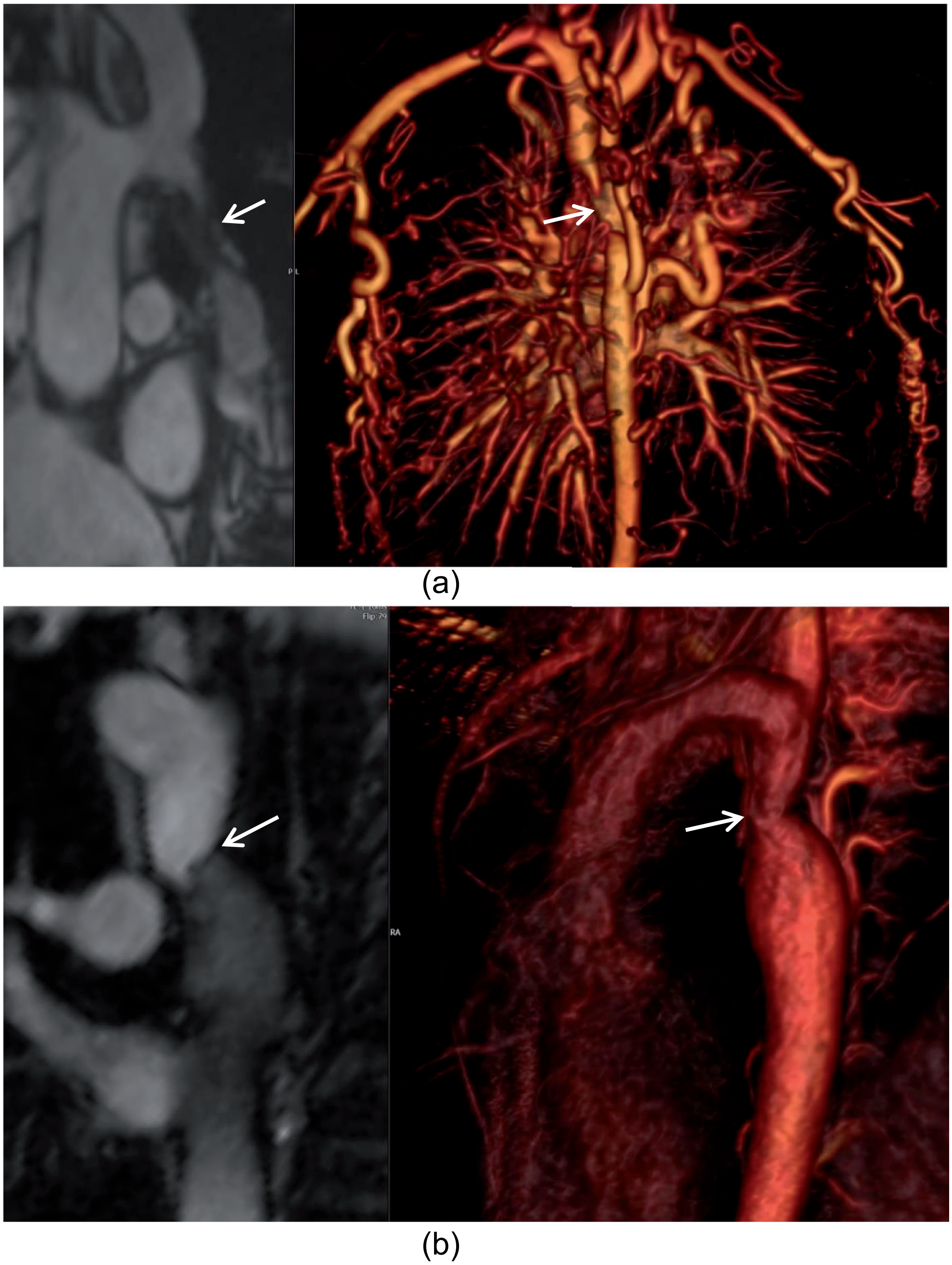

Figure 7.
Aortic interruption and severe coarctation. (a) – TrueFISP (left) and posterior view 3-dimensional MR angiography (right) showing a short aortic interruption (white arrows) with extensive lateral thoracic, internal mammary, and intercostal arterial collateralisation. (b) - TrueFISP (left) and left anterior oblique view 3-dimensional MR angiography (right) showing a focal tight aortic coarctation (white arrows) with a more notable membranous component at the level of the aortic isthmus with associated dilated intercostal arterial collateralisation.

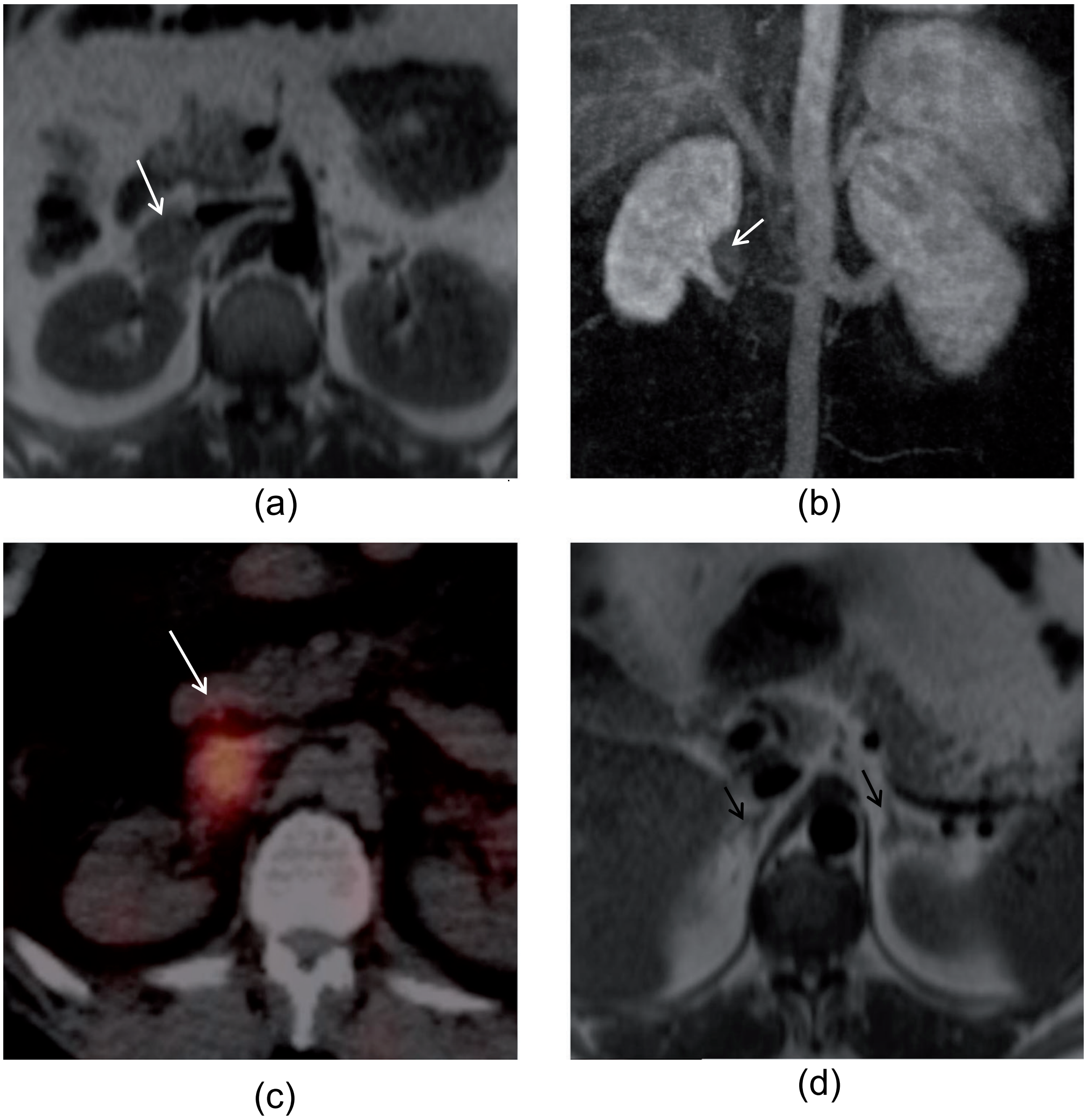

Figure 8.
Right-sided extra-adrenal phaeochromocytoma. (a) – Axial HASTE (Siemens). Half-Fourier Acquisition Single-shot Turbo spin Echo imaging. Well-defined, heterogenous soft tissue mass anterosuperior to the right renal hilum (white arrow). (b) – Multiphase gadolinium-enhanced angiography demonstrates homogenous enhancement (white arrow). (c) – MIBG (meta-iodo-benzyl-guanidine)-CT fusion imaging shows increased uptake within an extra-adrenal phaeochromocytoma (white arrow). (d) – Axial HASTE imaging shows bilateral normal adrenal glands (black arrows).

We advocate MRI as a safe and effective non-invasive screening imaging technique for hypertensive patients, using a comprehensive cardiac and non-cardiac protocol.

2.5.1 Implications for research and clinical practice

Ongoing studies are encouraged to investigate a) the relationship between the selfish brain hypothesis and hypertension (both childhood and young-onset hypertension), b) the relationship of these findings to the development of premature cerebrovascular disease or dementia especially in those who have their hypertension 'controlled' and in whom this may be too aggressive for them as individuals, c) the impact of hypertension on thoracic pathology, predicting adverse aortic remodelling

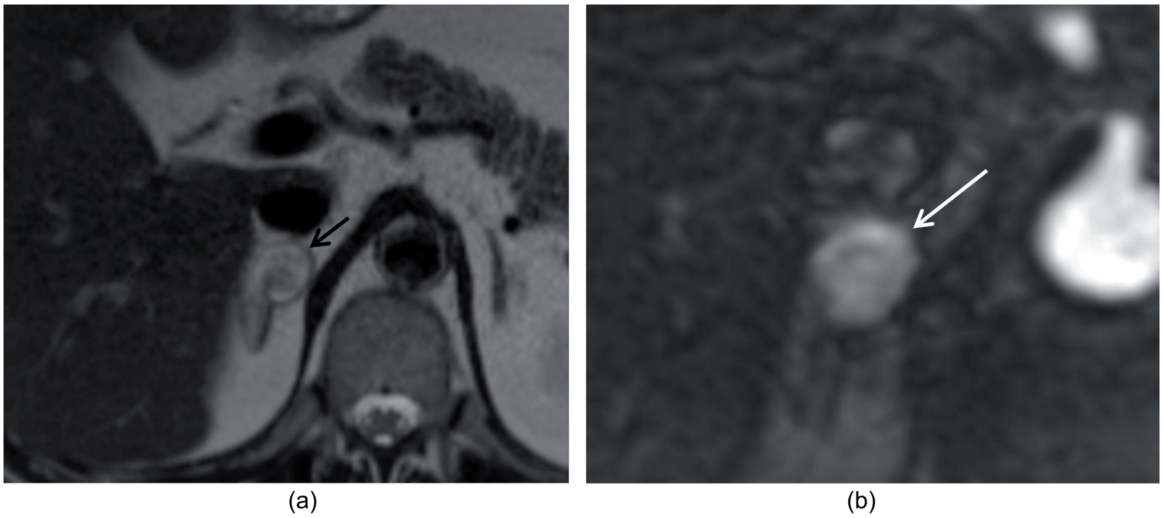

Figure 9.
Right adrenal phaeochromocytoma. (a) – Axial HASTE through the upper abdomen showing a heterogeneous, predominantly increased signal, well-defined soft tissue mass arising from the right adrenal gland (black arrow). (b) – Early phase axial angiography shows avid gadolinium enhancement of the mass (white arrow).

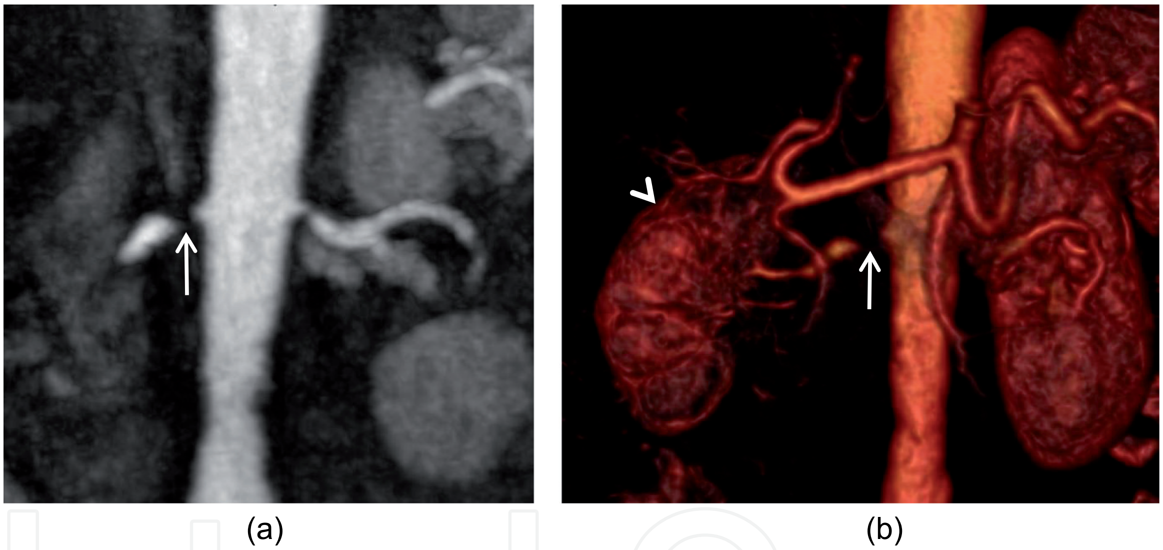

Figure 10.
Bilateral renal artery stenosis. Oblique coronal arterial phase gadolinium angiography showing a tight left renal artery stenosis and subtotal occlusion (white arrow) of the right renal artery (a) secondary to atherosclerosis, with associated right renal atrophy (white arrowhead) on 3-dimensional angiography (b). Note that the infrarenal abdominal aorta is atheromatous.

and the effect of the endovascular intervention on hypertensive patients with potentially important cerebrovascular variations, and d) the complex LV phenotype assessment by CMR and the effect of medical therapy on patterns of LV remodelling, LVH, and interstitial fibrosis, and thus the ultimate effect on patient prognosis.

If these findings were proven to be true, caution could be placed to the degree of BP reduction or nature of medication used in the hypertensive patient with VAH and incomplete posterior CoW and possibly avoid paradoxical adverse effects with treatment. From an investigational standpoint, the role of these cerebrovascular variants in the development of vascular dementia in hypertensive patients may need to be studied further. The future screening of cerebrovascular structure may require the screening of cerebrovascular anomalies in hypertensive patients, either via a Doppler

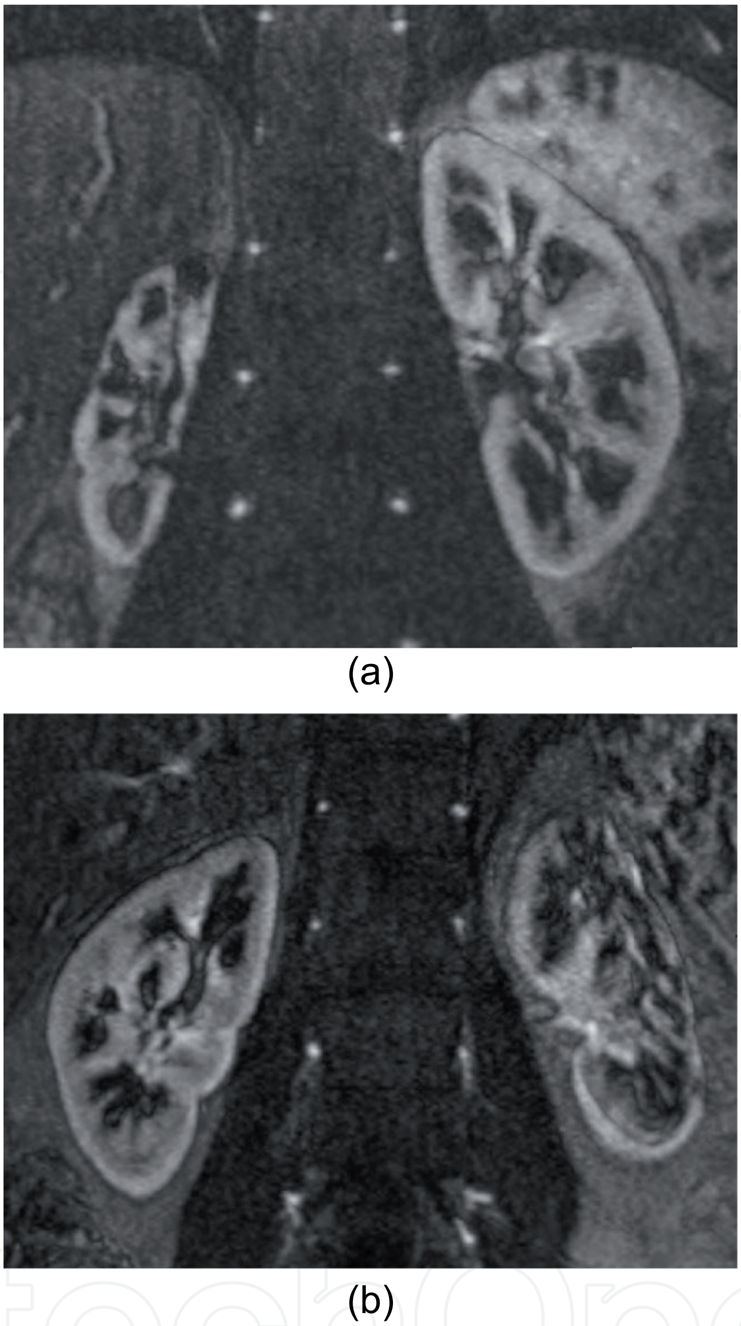

Figure 11.
Chronic unilateral renal atrophy with contralateral compensatory renal hypertrophy. Coronal early phase angiography shows severe right renal atrophy secondary to childhood reflux nephropathy (a) and severe left renal atrophy secondary to chronic pyelonephritis (b).

ultrasound or non-invasive angiographic imaging studies, such as computed tomographic angiography or magnetic resonance angiography.

Treatment of prehypertensive or borderline hypertensive patients with such anomalies may be directed towards using agents that mitigate sympathetic nerve activity such as angiotensin receptor blockers. This finding may also extrapolate to the treatment of acquired stenosis of the vertebrobasilar system from underlying atherosclerosis for which endovascular treatment is controversial.

Vertebral artery stenosis can be treated with stenting with good technical results, but whether it results in improved clinical outcomes is uncertain [53]. This study showed that stenting for vertebral stenosis has a much higher risk for intracranial

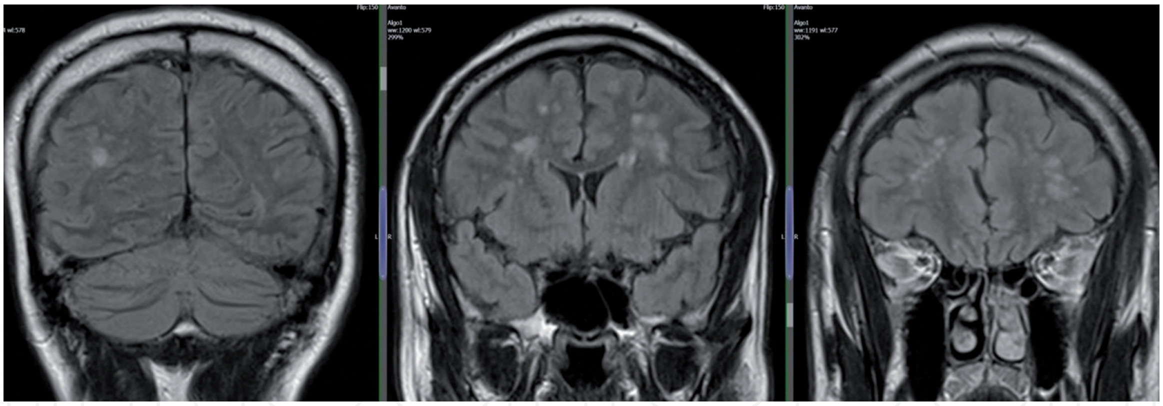

Figure 12.
Hypertensive cerebral microangiopathy. Coronal FLAIR imaging (Fluid-Attenuated Inversion Recovery is an inversion recovery sequence with a long inversion time; this removes the signal from the cerebrospinal fluid in the resulting images); shows patchy periventricular deep white matter high signal bilaterally in the frontal, parietal and occipital lobes in a patient with young-onset hypertension and cerebrovascular variation of the posterior circulation.

stenosis compared with extracranial stenosis; this pooled analysis did not show evidence of a benefit for stroke prevention for either treatment. In addition, the VIST trial compared the risks and benefits of vertebral angioplasty and stenting with best medical treatment alone for symptomatic vertebral artery stenosis; [54] this study also concluded that stenting in extracranial stenosis appears safe with low complication rates but that large phase 3 trials are required to determine whether stenting reduces stroke risk.

However, if the acquired stenosis or indeed congenital hypoplasia of the vertebral artery was found to be the cause of hypertension, vertebral stenting/angioplasty could be investigated for the possible benefit of an antihypertensive intervention.

Whether this pathologic mechanism is one of the unknown causes of essential hypertension and is more common than other previously proposed causes or a certain combination of pathological mechanisms underlie essential hypertension, remains to be definitively proven with longitudinal studies.

3. Summary

This chapter illustrates how the unique role of MRI can be used to comprehensively and non-invasively image patients with hypertension. In our institution, we have used such a protocol in those patients seen via a tertiary referral hypertension clinic for almost 10 years; such patients are generally difficult to treat/resistant hypertensives and young-onset hypertensive patients.

Patients who have sustained the consequences of a hypertensive insult, such as myocardial infarction, acute aortic dissection, cerebrovascular events, acute malignant hypertension, or pre-eclampsia, can also be assessed with MRI. Distinguishing between hypertensive heart disease and hypertrophic cardiomyopathy can be a particular challenge.

Importantly, we have also indicated how the ‘selfish brain hypothesis’ may play a vital role in the aetiology of ‘essential hypertension’. Future studies are clearly needed and we suggest that MRI can play an increasingly pivotal role to help guide an efficient and bespoke therapeutic approach to patient management.

From a non-invasive imaging perspective, the hypertensive cohort is particularly interesting and challenging; a vast amount of important detail can be obtained in a single study. Nevertheless, we can take a step closer to uncovering the causes of the greatest non-communicable cause of human mortality and morbidity.

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Conflict of interest

The authors declare no conflict of interest.

Appendices and Nomenclature

2D	two-dimensional
AAS	Acute aortic syndrome
AWT	Absolute wall thickness
BMI	Body mass index
BP	Blood pressure
CMR	Cardiovascular magnetic resonance imaging
CoA	Coarctation of the aorta
CT	Computed tomography
ECG	Electrocardiogram
EDWT	End -diastolic wall thickness
ESC	European Society of Cardiology
HCM	Hypertrophic cardiomyopathy
HHD	Hypertensive heart disease
HMOD	Hypertension mediated organ damage
HTN	Hypertension
ipCoW	Incomplete posterior circle of Willis
LAE	Left atrial enlargement
LAS	Long axis shortening
LV	Left ventricle
LVEF	Left ventricular ejection fraction
LVH	Left ventricular hypertrophy
LVM	Left ventricular mass
mFS	mid-wall circumferential fractional shortening
MR	Magnetic resonance
MRI	Magnetic resonance imaging
PWV	Pulse wave velocity
SAM	systolic anterior motion of mitral valve
VAH	Vertebral artery hypoplasia
VIST	Vertebral Artery Ischaemia Stenting Trial

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