Asia-Pacific Chiropractic Journal

CLINICAL DISCUSSION

Chiropractic spinal adjustments in patients with heavy sympathomimetic drug use

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Background: Cervical spinal adjustment or manipulation (CeSAM) is widely used for musculoskeletal conditions and is generally considered safe and effective. However, rare neurovascular complications have been reported, particularly following CeSAM. The affected demographic commonly includes young adults who would otherwise be considered healthy. Several risk factors are linked to blood vessel integrity. One such risk factor is heavy sympathomimetic drug use, including cocaine use of approximately 1 g/day, which is strongly associated with vascular pathology, including vasospasm, thrombosis, and arterial dissection leading to strokes. Other sympathomimetic substances, including amphetamines and methylphenidate, have also been associated with adverse vascular effects.

Discussion: This narrative review examines the literature relating to sympathomimetic drug-associated vascular pathology and adverse vascular events reported in association with CeSAM. Heavy cocaine use has been shown to contribute to endothelial dysfunction, vasospasm, hypertension, hyper-coagulability and structural vascular pathology. Although direct evidence linking sympathomimetic drug use, CeSAM and cervical artery dissection is lacking, a biologically plausible mechanism exists whereby pre-existing vascular pathology may increase susceptibility to an adverse vascular event. Cervical manipulation could theoretically contribute to thrombus or embolus displacement in an already compromised cervical artery. Clinicians considering CeSAM should therefore recognise heavy sympathomimetic drug use as a potentially important vascular risk factor and, where clinically appropriate, regard active heavy use as a relative contraindication while prioritising conservative management approaches.

Conclusion: Heavy use of cocaine, amphetamines and methylphenidate has been associated with vascular pathology and arterial injury. These exposures should therefore be considered potentially modifiable risk factors when assessing patients before cervical spinal adjustment or manipulation. Appropriate history-taking, individualised risk assessment and consideration of conservative treatment alternatives are warranted when significant sympathomimetic drug use is identified.

Indexing terms: Chiropractic; Cocaine; methamphetamine; methylphenidate; cervical spinal manipulation; cervical artery dissection.

What is already known on this topic

Cervical artery dissection (CeAD), which can result in ischaemic intra-cerebral stroke, is an uncommon vascular pathology that affects approximately 1 to 3 individuals per 100,000 asymptomatic, otherwise healthy young adults annually. 2:1 female: male ratio with age <50

The literature has proposed a range of potential risk factors for cervical artery dissection (CeAD), informed by emerging epidemiological and clinical evidence.

Certain pharmacological agents have been implicated in vascular injury, leading to inflammation (vasculitis), luminal narrowing or constriction, and structural weakening of arterial walls. Notably, substances such as cocaine, methamphetamines and methylphenidate, collectively known as sympathomimetic drugs, are recognised for their capacity to induce pronounced vasoconstriction. Chronic exposure may result in irreversible vascular injury, thereby elevating the risk of hypertension, stroke, and other cardiovascular pathologies.

Introduction

The pathogenesis of cervical artery dissection (CeAD) is highly complex and not yet fully understood. Multiple risk factors have been shown to increase vulnerability to CeAD, as evidenced by independent associations that have been reported in the literature. (1) Among these, connective tissue disorders, particularly Ehlers-Danlos syndrome type IV, are well-documented to compromise arterial wall integrity, thereby substantially increasing the propensity for dissection. (2) Recent infection has been associated with an increased risk of CeAD, possibly through inflammatory or immune-mediated mechanisms that transiently weaken the arterial wall. (3)

'... Epidemiological evidence and case reports demonstrate that cocaine use is associated with both haemorrhagic and ischaemic stroke, while heavy or prolonged exposure has been linked to vascular dys-function...'

Mild hyperhomocysteinaemia has similarly been implicated through mechanisms involving endothelial dysfunction and vascular injury. Elevated plasma homocysteine concentrations have been reported as a recognised risk factor for cardiovascular disease, stroke and thrombotic events. (4) Reported estimates of the incidence of CeAD associated with cervical spinal adjustment or manipulation vary substantially within the literature, ranging from approximately one event per 400,000 treatments, as reported by Dvorak and Orelli in 1985, (5) to 6.39: 10,000,000 manipulations. (6)

These figures may be considered alongside the estimated spontaneous incidence of cervical artery dissection within the general population, reported by Béjot et al. (2014) (7) to be approximately 1 - 3 cases per 100,000 people annually. This has been updated to 2 - 9 cases per 100,000 by Griffin et al. (2024), (1) who found a 4-fold increase between 2002 - 2020. The considerable variability in these estimates reflects uncertainty about the incidence and causal interpretation of these events.

Chaibi & Russell (2019) (8) proposed three possible explanations for the temporal association between CeSAM and cervical artery dissection: the association may be coincidental; the event

may be directly iatrogenic; or the patient may already possess an underlying predisposition to arterial dissection, including vascular vulnerability associated with hypermobility syndromes or other conditions. In an experimental investigation, Symons et al. (2002) (9) subjected cadaveric vertebral arteries to mechanical strain and concluded that the forces typically generated during spinal manipulation were unlikely to directly damage an otherwise healthy vertebral artery. However, mechanical forces could theoretically displace a pre-existing thrombus or embolus. Thomas (2016) similarly concluded that manipulation could theoretically dislodge a pre-existing thrombus or embolus in a susceptible individual.

Common modifiable risk factors that may contribute to vascular damage include hypertension, hypercholesterolaemia, obesity, type 2 diabetes mellitus, oral contraceptive use, suboptimal dietary habits, excessive alcohol consumption and tobacco use; these have been extensively discussed by Triano and Kawchuk (2006) (11) Another modifiable risk factor that should be considered is the use of cocaine and cocaine-like substances. Cocaine is a potent sympathomimetic drug that increases concentrations of neurotransmitters including norepinephrine and serotonin and markedly increases sympathetic nervous system activity by inhibiting catecholamine re-uptake. (12) Its physiological effects include tachycardia, hypertension and widespread vasoconstriction.

Cocaine increases heart rate, blood pressure, and cardiac contractility, thereby raising myocardial oxygen demand. Second, vasoconstriction promotes platelet activity, facilitating thrombus formation, and triggers coronary spasm, collectively reducing oxygen supply. These effects are associated with substantial cardiovascular and cerebrovascular morbidity. Heavy or prolonged cocaine use (~1 g/day) has been associated with an increased risk of ischaemic stroke, arterial dissection and other serious cardiovascular complications. (13)

Other stimulant drugs warrant consideration. Hemphill et al. (2024) (14) reported an association between methamphetamine use and cerebrovascular disease in younger adults. Long-term methylphenidate therapy, commonly used in the treatment of attention-deficit/hyperactivity disorder (ADHD), has also been investigated in relation to cardiovascular risk, vascular function and cerebral blood flow. (15, 16) Although these substances differ pharmacologically from cocaine, they share sympathomimetic and vasoconstrictive properties that may contribute to vascular compromise in susceptible individuals. Vascular compromise may present with symptoms including numbness, altered sensation, or pain in the extremities, and may be mistaken for musculoskeletal symptoms. (16)

What this study adds

This study should provide insight into how sympathomimetic drugs may influence clinical practice.

Understanding the risk factors for certain sympathomimetic drugs can help identify individuals at risk for cerebrovascular and coronary artery disease and enable chiropractors to make better-informed clinical decisions, including when to refer patients for medical care.

Cervical spinal adjustment or manipulation is a recognised intervention within Chiropractic practice and is commonly used to manage cervical spine pain, dysfunction, and some headache disorders. Although most reported adverse effects following spinal manipulation are mild and transient, cervical artery dissection remains among the most serious vascular events reported in

temporal association with CeSAM. A systematic review by Pankrath et al. (2024) (17) and large retrospective cohort studies by Chu et al. (2023) (18) have not demonstrated convincing evidence of a direct causal relationship between CeSAM and CeAD. Findings reported by Brown (2024) (19) and Gorrell et al. (2023) (20) further illustrate the continuing uncertainty surrounding direct causation.

Mechanisms of Cocaine-Induced Vascular Injury

Long-term cocaine use has been reported to damage the vascular endothelium, promote a prothrombotic state and accelerate atherosclerosis. Several interrelated mechanisms may contribute to these vascular effects.

Endothelial Dysfunction

The vascular endothelium plays a central role in maintaining vascular homeostasis by regulating vascular tone, platelet activity, and inflammatory responses. Cocaine exposure has been associated with endothelial dysfunction, impairing these protective mechanisms and potentially contributing to progressive vascular pathology. Chronic cocaine use may also contribute to vascular fibrosis through excessive deposition of connective tissue within the vessel wall.

This process can reduce arterial elasticity and compliance, impairing blood vessels' ability to adapt to blood pressure changes. Progressive vascular stiffening may contribute to sustained hypertension and increase susceptibility to adverse cardiovascular events, including myocardial infarction and stroke. (21)

Prothrombotic effects

Cocaine promotes a prothrombotic state through mechanisms including enhanced platelet aggregation and alterations in circulating fibrinogen and von Willebrand factor concentrations. Together, these effects may facilitate intravascular clot formation and increase the risk of arterial thrombosis. (22) Arterial thrombosis may restrict or completely obstruct blood flow, potentially causing serious cardiovascular or cerebrovascular complications depending upon the vessel involved.

Vasospasm

Cocaine is a potent vasoconstrictor and may cause significant vasospasm through sustained contraction of vascular smooth muscle. The resulting reduction in arterial diameter limits tissue perfusion and the delivery of oxygen and nutrients, predisposing affected tissues to ischaemia. (23) Persistent ischaemia may subsequently result in infarction, defined as tissue death resulting from prolonged interruption of an adequate oxygen supply. Depending on the affected vascular territory, these pathophysiological processes can cause myocardial infarction, cerebral infarction, or gastrointestinal ischaemia and infarction. (23)

Structural degeneration of the arterial wall

Experimental and clinical evidence indicates that cocaine may compromise the structural integrity of the arterial wall. Clare et al. (23) described important neurovascular effects associated with cocaine exposure, including effects on vascular cellular function. Cocaine has also been associated with processes involving apoptosis of vascular smooth muscle cells. These cells are important in maintaining the structural integrity and contractile function of arterial walls.

Cocaine use has additionally been associated with pathological changes affecting the medial layer of blood vessels. Loss of smooth muscle integrity and degeneration within the arterial wall may reduce vascular resilience and potentially increase vulnerability to rupture or dissection, particularly when combined with substantial fluctuations in blood pressure. An arterial dissection occurs when disruption of the vessel wall allows blood to enter between arterial wall layers, potentially separating those layers and compromising blood flow to downstream tissues.

Accelerated atherosclerosis

Chronic cocaine use is associated with accelerated atherosclerotic disease, including premature atherosclerotic changes within the coronary circulation. Importantly, these pathological changes may occur in younger individuals who do not necessarily possess conventional cardiovascular risk factors such as hypercholesterolaemia or longstanding hypertension. (24) Cocaine exposure may promote the accumulation of lipids, inflammatory cells and fibrous tissue within arterial walls.

Over time, atherosclerotic plaques may progressively narrow arteries and restrict blood flow. Plaque disruption may additionally promote thrombus formation and acute arterial occlusion, potentially resulting in myocardial infarction or other ischaemic events. Accelerated vascular ageing therefore represents a clinically important potential consequence of chronic cocaine exposure. (24)

Evidence of vascular injury outcomes

Cocaine-associated myocardial infarction may occur through mechanisms that differ from those typically associated with chronic coronary atherosclerosis. One important mechanism is intra-coronary thrombosis, in which thrombus formation within a coronary artery obstructs myocardial blood flow. Importantly, this may occur even in the absence of extensive underlying atherosclerosis. (25)

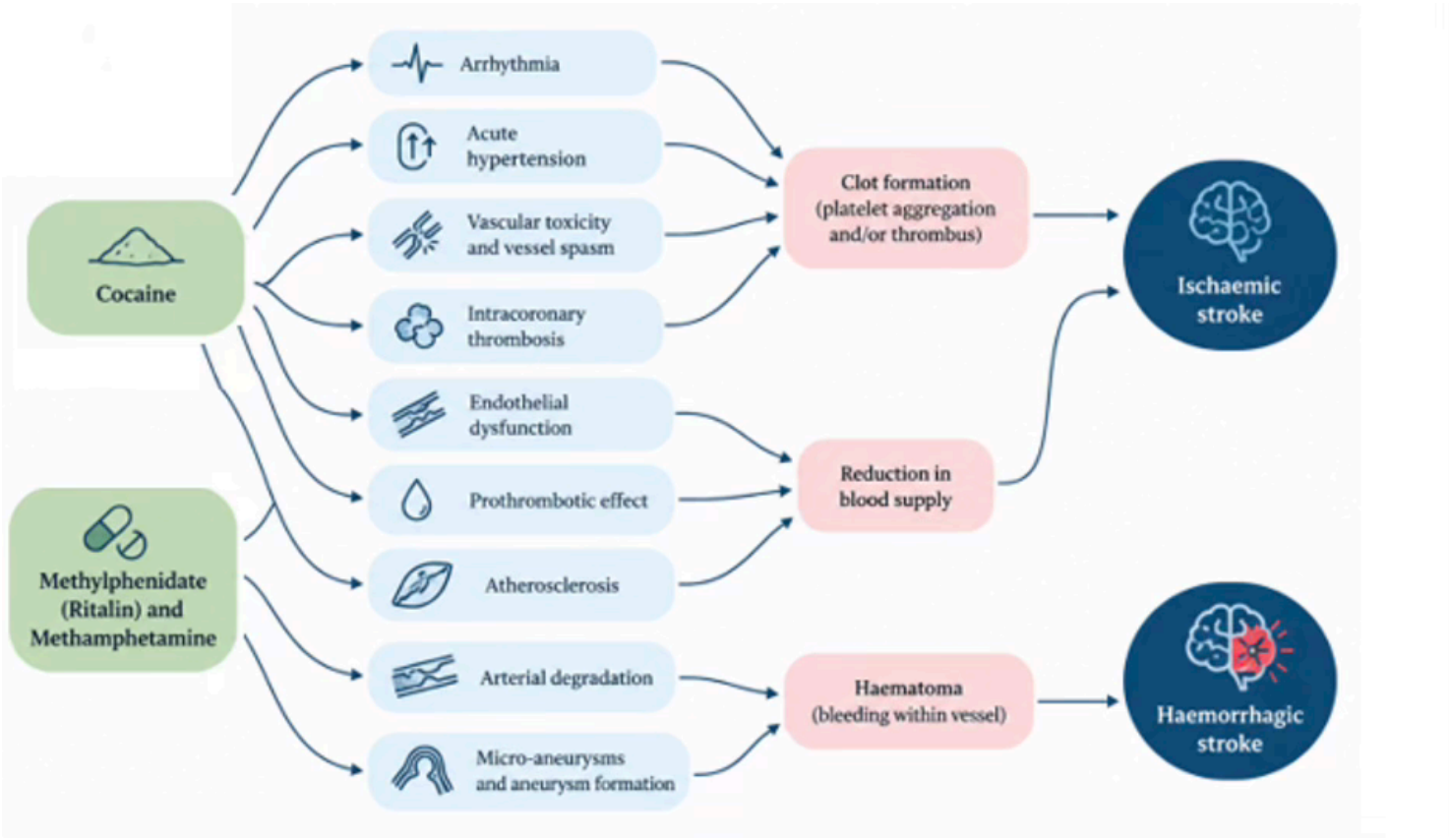
A second important mechanism is pronounced coronary vasospasm. Cocaine-induced constriction of the coronary arteries may substantially reduce or temporarily interrupt myocardial blood supply, resulting in ischaemia and potentially infarction. Both intra-coronary thrombosis and severe coronary vasospasm therefore contribute to cocaine-associated acute myocardial infarction, including in relatively young individuals who may otherwise appear healthy. (25, 26)

Cocaine use is also associated with both ischaemic and haemorrhagic stroke. Severe hypertension, vasospasm, thrombosis and other vascular abnormalities may contribute to these events. Cocaine-induced vasoconstriction can cause arterial narrowing and reduced cerebral perfusion, thereby increasing the risk of ischaemic stroke. Conversely, marked elevations in blood

pressure may increase the risk of vascular rupture and haemorrhagic stroke. Some cocaine-associated vascular changes may remain clinically silent until a major neurological or cardiovascular event occurs. Early recognition of significant cocaine exposure and associated vascular risk may therefore be important in appropriate clinical assessment and prevention. (See table 1 and figure 1)

Figure 1: Proposed Pathophysiological Pathways Linking Cocaine, Methylphenidate (Ritalin), and Methamphetamine Use to Ischaemic and Haemorrhagic Stroke

Note. This figure illustrates the potential cardiovascular and cerebrovascular effects of cocaine, methylphenidate (Ritalin), and methamphetamine, highlighting the proposed mechanisms through which these substances may contribute to ischaemic and haemorrhagic stroke



OpenAI. (2026). ChatGPT [Large language model]. https://chatgpt.com/

Table 1: Effects of cocaine across multiple anatomical structures and physiological systems.

Effect of Cocaine ($C_{17}H_{21}NO_4$) on the heart (28)

Cardiomyopathy: This condition causes the heart muscle to become stiffer and reduces its efficiency in pumping blood.

Myocardial dysfunction and heart failure: Progressive myocardial injury may impair cardiac output and cause fatigue, dyspnoea and reduced exercise tolerance.

Cardiac arrhythmias: Cocaine may disrupt cardiac electrical conduction and precipitate potentially life-threatening arrhythmias or cardiac arrest.

Spikes vital signs: These effects may include a rapid increase in heart rate, blood pressure, and body temperature.

Increased cardiovascular activity: Sympathetic stimulation may acutely increase heart rate, blood pressure, myocardial oxygen demand and body temperature.

Effect of Cocaine ($C_{17}H_{21}NO_4$) on blood vessels (28)

Increases vasoconstriction: Cocaine induces vasoconstriction by stimulating α-adrenergic receptors on vascular smooth muscle and inhibiting norepinephrine reuptake. These mechanisms cause acute, significant narrowing of arteries, including the coronary arteries that supply the myocardium.

Atherosclerosis: Accelerated atherosclerosis.

Coronary vasospasm: Rare cases of coronary vasospasm and myocardial ischaemia have been reported; these are not routine effects of prescribed use.

Damages blood vessels: Aortic injury.

Blood Clotting and Thrombosis: Platelet activation and thrombosis.

This impairment increases the risk of intravascular micro-thrombus formation within stenotic arteries.

Table 2: Effects of methylphenidate across multiple anatomical structures and physiological systems .

Effect of Methylphenidate ($C_{14}H_{19}NO_2$) on the heart (37; 38)

Increases Heart Rate: Heart rate can increase up to 10 beats per minute.

Rare Vascular Complications: Misuse, including crushing and injecting pills, may result in physical obstruction of blood vessels. Rare medical reports have linked stimulant use to severe conditions such as cerebral vasculitis and reversible cerebral vasoconstriction syndrome (RCVS).

Increased Cardiac Output: The heart beats slightly faster and with greater force.

Increased Blood Pressure: The majority of individuals exhibit modest increases in blood pressure, typically ranging from 1 to 5 mmHg.

Palpitations or Arrhythmias: Some individuals may have rapid, irregular, or forceful heartbeats.

Effect of Methylphenidate ($C_{14}H_{19}NO_2$) on blood vessels (39; 40)

Increases vasoconstriction: Methylphenidate (Ritalin) acts as a central nervous system stimulant. It increases norepinephrine and dopamine concentrations, resulting in blood vessel constriction.

Atherosclerosis: Accelerated atherosclerosis.

Coronary artery disease: Vasoconstriction, coronary artery vasospasm, intra-coronary thrombosis.

Peripheral Circulation: Reduced blood flow to the fingers and toes may result in sensations of coldness, numbness, or pain.

Raynaud's Phenomenon: Methylphenidate (Ritalin) has been linked to the development of peripheral vasculopathy. This includes Raynaud's syndrome, a disorder characterised by temporary colour changes in the fingers in response to specific stimuli.

Table 3: Effects of methamphetamine across multiple anatomical structures and physiological systems. **Note:** Note: Methamphetamine is a type of amphetamine, but they are not the same; methamphetamine is more potent and has a higher potential for abuse and addiction. While amphetamines are often prescribed for medical conditions, methamphetamine is primarily known as an illicit street drug with limited medical use

Effect of Amphetamine ($C_9H_{13}N$) on the heart (41)

Catecholaminergic stimulation: Amphetamine increases catecholaminergic signalling, particularly involving norepinephrine and dopamine, resulting in enhanced sympathetic activity.

Increased heart rate and contractility: Sympathetic stimulation may increase heart rate and myocardial contractility, raising cardiac workload and oxygen demand.

Elevated blood pressure: Increased cardiac activity and peripheral vascular resistance can raise blood pressure, sometimes markedly at high doses.

The effect of Amphetamine ($C_9H_{13}N$) on blood vessels (42)

Increases vasoconstriction: Amphetamine drugs elevate norepinephrine levels, which leads to vasoconstriction and a subsequent reduction in blood flow to specific tissues.

Vasospasm: Sudden, severe contractions of blood vessel walls can occur, restricting blood flow to the extremities, as seen in conditions such as Raynaud's syndrome.

Atherosclerosis: Accelerated atherosclerosis in young.

Coronary artery disease: Coronary vascular complications.

Clotting Risk: Thrombosis and coagulation.

Increased Blood Pressure: Blood pressure often increases markedly because of concurrent vasoconstriction and an elevated heart rate.

Vascular Permeability: High doses or prolonged exposure may alter the endothelium, the inner lining of blood vessels, increasing permeability and susceptibility to inflammation or the formation of small intercellular gaps.

Experimental studies suggest that the mechanical forces generated during cervical spinal manipulation are unlikely to routinely damage an otherwise healthy vertebral artery. Symons et al. (2002) found no evidence of routine mechanical disruption of healthy vertebral arteries under forces comparable to those associated with manipulation. (9) However, an artery already compromised by vascular pathology associated with cocaine or other sympathomimetic drug use may possess reduced structural resilience. This creates a biologically plausible hypothesis that chronic stimulant-related vascular injury could increase susceptibility to an adverse vascular event in a predisposed individual undergoing CeSAM. (43)

This distinction is important because it remains consistent with literature that has not established a direct causal relationship between cervical spinal manipulation and cervical artery

dissection, while acknowledging that individual patient characteristics may materially influence clinical risk. The question becomes particularly relevant when vasoactive substances such as cocaine, amphetamines and methylphenidate are involved. These substances can affect vascular integrity through vasoconstriction, elevated blood pressure, endothelial dysfunction, and altered platelet aggregation.

Individually or collectively, these mechanisms may contribute to arterial wall injury or thrombus formation. When such pharmacological factors are present, clinical risk assessment before cervical manipulation should therefore consider the possibility of underlying vascular vulnerability, particularly when additional vascular risk factors or suspicious symptoms are present. (43)

Clinical implications

> From a clinical perspective, active heavy use of cocaine, amphetamines or methylphenidate should be regarded as a potentially significant vascular risk factor. Where substantial stimulant exposure is identified, it may be appropriate to consider this a relative contraindication to cervical spinal adjustment or manipulation until an appropriate clinical assessment has been completed. (44)

Comprehensive history-taking should include appropriate enquiry regarding recreational and prescribed stimulant use, particularly cocaine, methamphetamine and other sympathomimetic substances. This information may materially affect assessment of vascular risk and subsequent clinical decision-making. Where heavy stimulant use is identified, conservative management strategies should be considered. Mobilisation, appropriate exercise therapy and soft-tissue interventions may represent alternatives for some patients and avoid the theoretical vascular stresses associated with high-velocity cervical manipulation.

Treatment selection should remain individualised and should take account of the patient's complete cardiovascular, neurological and vascular risk profile rather than stimulant exposure in isolation. Interprofessional collaboration may also be appropriate. Patients with suspected stimulant-associated vascular disease should be considered for medical assessment where clinically indicated, particularly when presenting with neurological symptoms, uncontrolled hypertension or other features suggestive of underlying cardiovascular or cerebrovascular pathology. (45) Appropriate monitoring for emerging cardiovascular or neurological symptoms remains important during the management of individuals with recognised vascular risk factors. (46)

Areas where further study is needed

Several clinically important questions remain unanswered. Prospective research is required to determine whether chronic cocaine use independently increases the risk of CeAD following cervical spinal manipulation or whether cocaine exposure should instead be understood as one component of a broader vascular risk profile. Large multi-centre observational studies may be particularly valuable in addressing this question because individual vascular events are rare and appropriately powered prospective interventional studies may be difficult to conduct.

Chronic hypertension

Numerous studies have identified an association between chronic cocaine exposure and the development of hypertension. Repeated stimulation of the sympathetic nervous system may produce recurrent elevations in blood pressure through vasoconstriction and increased cardiac output. (47) Hypertension is an important consideration during vascular risk assessment before cervical adjustment because chronically elevated blood pressure may coexist with other conditions that affect vascular integrity. However, the available literature has not established a direct relationship between long-term cocaine use, cervical spinal adjustment and subsequent cervical artery dissection. Further research is needed to clarify the long-term effects of cocaine and other sympathomimetic substances on blood-pressure regulation, cervical arterial integrity and the risk of vascular events in patients undergoing cervical manual interventions.

Venous thrombosis

Although the arterial complications of cocaine use, including myocardial infarction and stroke, have been extensively investigated, its effects on the venous system are less well characterised.

Emerging evidence suggests that cocaine may contribute to an increased risk of deep-vein thrombosis and pulmonary embolism through mechanisms including endothelial injury, platelet activation and hypercoagulability. (48) These processes may promote venous thrombus formation and create potentially life-threatening complications when a thrombus embolises to the pulmonary circulation. Data regarding the prevalence and specific risk factors for deep-vein thrombosis and pulmonary embolism among cocaine users remain limited. Further investigation is also required into the effects of other sympathomimetic drugs on cervical vascular integrity. Although such substances share some pharmacological characteristics with cocaine, their specific relationship with cervical arterial pathology has not been adequately investigated.

Future studies should also examine long-term vascular outcomes among chronic stimulant users and determine whether clinically useful risk-stratification tools can be developed to identify patients who may require modification of manual therapy techniques or referral for medical assessment before treatment. (24, 49, 50)

Cocaine- and sympathomimetic drug-related vascular disease, may possess distinctive clinical features, including earlier onset, severe presentations and occurrence in younger individuals without conventional cardiovascular risk factors. However, evidence concerning the prevalence and magnitude of any additional CeSAM-related risk in cocaine users remains limited. Further research in this area could inform more targeted screening, prevention, referral and management strategies for patients with significant stimulant exposure. (45)

Conclusion

Drug misuse represents a significant social and public-health concern. The occurrence of coronary, cerebrovascular and other vascular disease among individuals who use cocaine highlights the importance of recognising potentially modifiable vascular risk factors and

improving strategies for early identification and prevention of cardiovascular and cerebrovascular morbidity.

Adverse vascular events reported in temporal association with cervical spinal adjustment or manipulation have frequently involved younger patients who may not have previously recognised arterial pathology. Epidemiological evidence and case reports demonstrate that cocaine use is associated with both haemorrhagic and ischaemic stroke, while heavy or prolonged exposure has been linked to substantial vascular dysfunction. Amphetamines and methylphenidate may also affect vascular function through mechanisms including vasoconstriction, hypertension and alterations in platelet activity.

Although the current literature does not establish a direct causal relationship between sympathomimetic drug use, cervical spinal adjustment or manipulation and cervical artery dissection, the known vascular effects of these substances provide a biologically plausible basis for increased caution. Patients who are heavy users of cocaine, amphetamines or methylphenidate may have underlying vascular pathology that could increase their susceptibility to cervical arterial complications.

Consequently, clinicians should specifically enquire about sympathomimetic drug use as part of a comprehensive clinical history and incorporate this information into individualised vascular risk assessment before undertaking CeSAM. Heavy active use should be recognised as a potentially modifiable risk factor and may constitute a relative contraindication to cervical spinal adjustment or manipulation pending appropriate clinical assessment. If significant vascular risk are identified, consider conservative management strategies and, when indicated, medical referral.

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Cite: Battiston ALM, Nunn N, Breeze S, Harrison J. Chiropractic spinal adjustments in patients with heavy sympathomimetic drug use. Asia-Pac Chiropr J. 2026;7.3 https://www.apcj.site/BattistonDrugUse.pdf

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Funding

This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Ethics approval and consent to participate

Ethics approval was not required because this study is a narrative review of published literature and did not involve human participants or identifiable patient data.

Competing interests

The authors declare no potential conflicts of interest concerning the research, authorship and/or publication of this article.

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