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Australian Government
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As we share our own knowledge, teaching, learning and research practices within this University we also pay respect to the knowledge embedded forever within the Aboriginal Custodianship of Country.

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Methamphetamines and the Heart: .....skating on thin ICE

Dr Elizabeth Paratz

29/03/21

Australian Government
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Overview

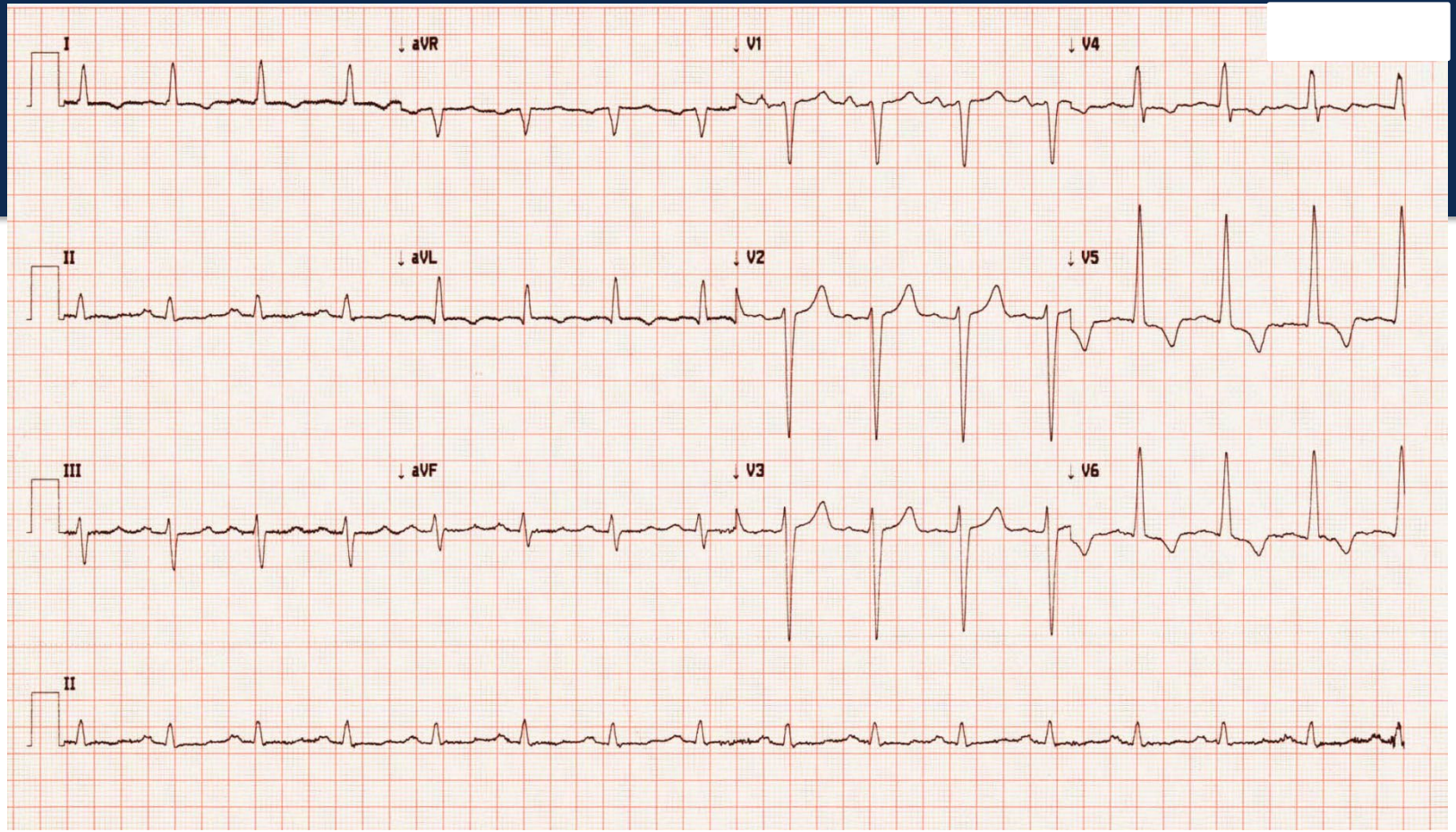
1. Case Study
2. Spectrum of methamphetamine-related complications
3. Focus on methamphetamine cardiomyopathy
4. Q&A

Case study

42yo gentleman,
7 year history of methamphetamine abuse
- $100/day of ice
- Occasional EtOH, ex-marijuana smoker

Presented to Emergency Department with severe peripheral oedema, admitted under Cardiology for further assessment

Other PMH :
- Bilateral varicose veins
- Mild acquired brain injury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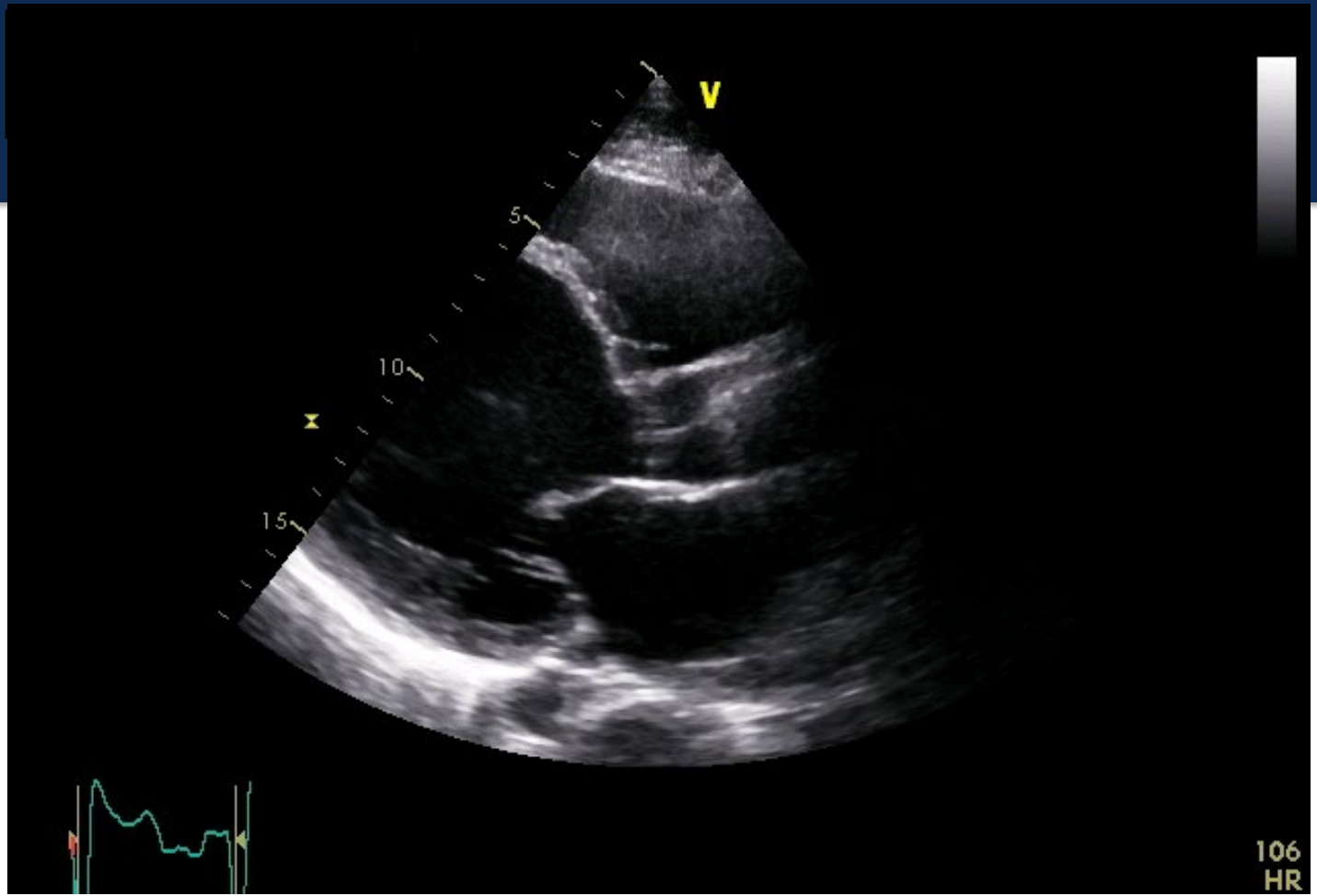
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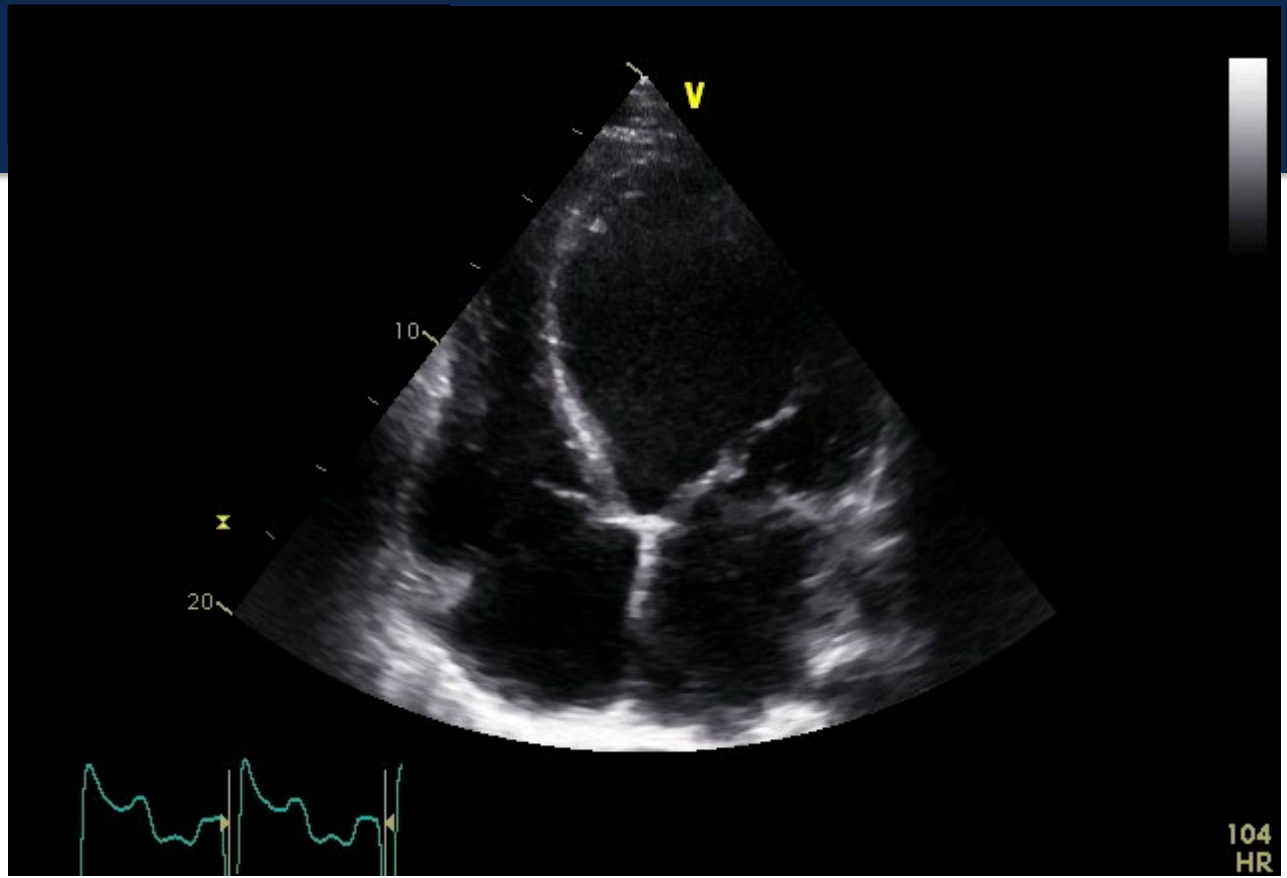
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Commenced on therapy…

- ACE inhibitor : perindopril 2.5mg
- beta-blocker : bisoprolol 2.5mg
- spironolactone : 25mg daily
- frusemide : 40mg mane

Issues with adherence

- initially, NYHA IV -> II on therapy.
- however, issues with non-adherence re-emerged :
    - Spironolactone -> very dramatic and painful gynaecomastia, to extent that mastectomy under consideration
    - Generally non-adherent to other medications
    - Re-adherence to methamphetamine abuse

- worsening of symptoms again
- progression of cardiomyopathy severity on serial TTEs

Progress

- out-of-hospital ventricular tachycardia requiring direct cardioversion
- multiple episodes of conscious ventricular tachycardia with acute pulmonary oedema requiring non-invasive ventilation and GTN infusion

Progress and prognosis:

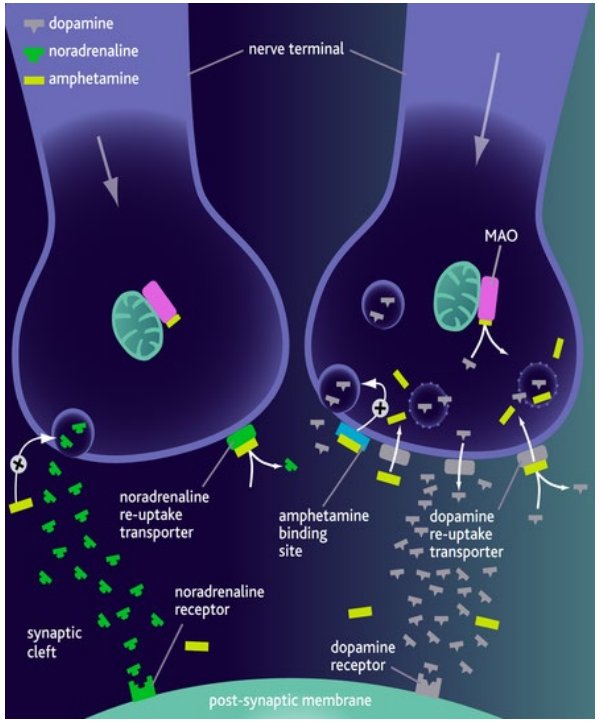
- multiple episodes VT
    - Amiodarone infusion, direct cardioversion >5 times
- extensive discussions
    - Not for device therapy
    - Not for transplant given overall patterns of behaviour
- conservative trajectory of care pursued

Methamphetamine-related cardiac complications

pharmacology

- a sympathomimetic psychostimulant that **causes the release** and **blocks the reuptake** of monoamine neurotransmitters, including dopamine, noradrenaline, and serotonin.
  - Causes the release : by displacing noradrenaline, adrenaline, dopamine and serotonin out of cytoplasmic vesicles into the synapse
  - Blocks the reuptake : by inactivating metabolism

- Net effect : +++ monoamines in synapse
- Stimulant, anorexiant, euphoric, hallucinogenic



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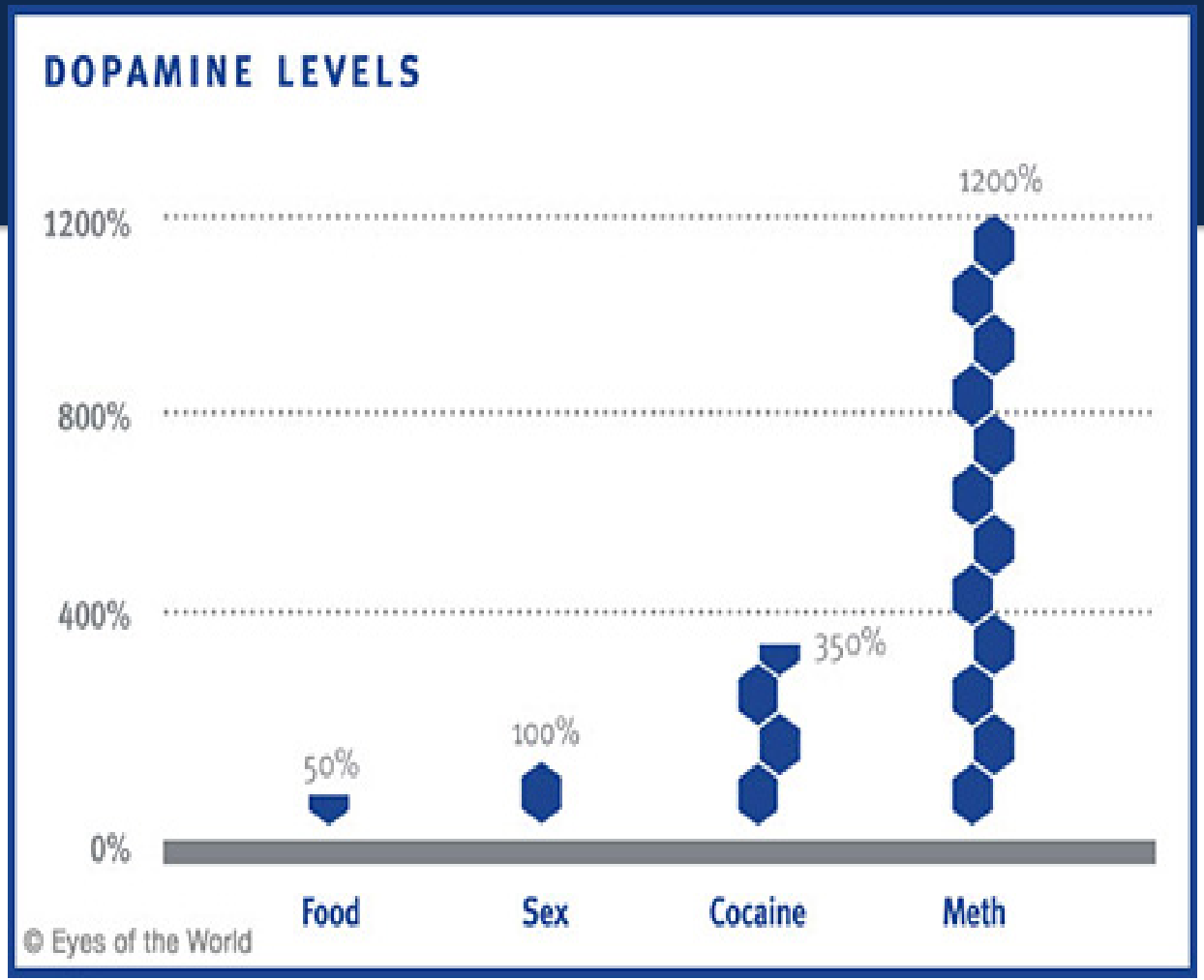

4 Acute intoxication and associated clinical effects of amphetamines[7,8]

System/nature of problem	Symptoms
Central nervous system	Anorexia, headache, bruxism, agitation, aggression, tremor, ataxia, seizures, coma, intracerebral haemorrhage, subarachnoid haemorrhage, hypertensive encephalopathy
Cardiovascular	Hypertension, tachycardia, arrhythmias, acute coronary syndrome, aortic dissection
Pulmonary	Cardiogenic and non-cardiogenic pulmonary oedema, pulmonary hypertension
Gastrointestinal	Nausea, vomiting, mesenteric ischaemia
Musculoskeletal	Hyperthermia, rhabdomyolysis
Adrenergic crisis	Sympathomimetic syndrome, hepatorenal failure, disseminated intravascular coagulation
Electrolyte disturbances	Hyponatraemia, hypernatraemia, hypoglycaemia
Psychiatric	Euphoria, "rush", increased energy, paranoia, anxiety, hallucinations, acute psychosis

Gray et al MJA 2007

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Table 1 Cardiac complications of methamphetamines

Methamphetamine effect	Cardiac outcome
Tachycardia, hypertension	Malignant hypertension Coronary vasospasm Acute myocardial infarction Aortic dissection
Myocardial toxicity	Malignant arrhythmias Methamphetamine associated cardiomyopathy
Pulmonary arterial hypertension	Right heart failure
Neurotransmitter depletion	Dysrhythmias Sudden cardiac arrest
Intravenous drug injection	Infectious endocarditis

Paratz et al 2015

Escalating the risks….

Co-ingestion :

- Co-ingestion with alcohol or other drugs
- This can significantly increase toxicity by cross-inhibition of metabolism or interaction

'Dirty drugs' :

- Also the risks of 'street preparation'
- Case reports for example of direct manganese toxicity causing cardiomyopathy in a methamphetamine abuser whose drugs were contaminated with heavy metals

Addiction

RESEARCH REPORT

doi:10.1111/j.1360-0443.2008.02231.x

Methamphetamine-related fatalities in Australia: demographics, circumstances, toxicology and major organ pathology

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ABSTRACT

Aim To examine the demographic characteristics, circumstances of death, toxicological results and major organ pathology of methamphetamine-related deaths in Australia. **Design** Retrospective review of coronial files. **Setting** Australia. **Methods** Cases in which methamphetamine was listed as a cause of death were identified from the National Coronial Information System (NCIS). **Findings** A total of 371 cases were identified. The mean age of decedents was 32.7 years; 77% were male and 35% were employed. Route of administration was predominantly by injection (89%). Drugs other than methamphetamine were detected in 89% of cases, most commonly benzodiazepines (41%) and morphine (36%). The median blood methamphetamine concentration was 0.2 mg/l (range 0.02–15.0 mg/l). Deaths were overwhelmingly accidental, with 14% determined to be suicides, and occurred in a private home (71%). Cardiovascular pathology, typically coronary artery atherosclerosis, was detected in 54% of decedents. Cerebrovascular pathology, most commonly cerebral haemorrhage and hypoxia, was present in 20% of cases. **Conclusions** Methamphetamine has contributed to a substantial number of deaths in Australia. Users need to be informed of the potential harms of methamphetamine use, particularly those associated with the cardiotoxicity of methamphetamine and the use of methamphetamine in conjunction with other drugs.

Kaye et al Addiction 2008

Table 2 Direct cause of death.

Cause of death (%)	*Male (n = 285)*	*Female (n = 86)*	*All (n = 371)*
Methamphetamine toxicity	17	16	17
Combined drug toxicity	51	53	51
Cardiovascular	15	11	14
Cerebrovascular	4	13*	6
Injury	9	8	9
Pulmonary	5	4	5
Hanging	5	4	5
Other	7	6	7

In 14% of cases, the forensic pathologist concluded that cardiovascular complications or disease arising from or complicated by methamphetamine use was the direct cause of death. Cardiovascular events and pathology causing death included coronary artery atherosclerosis/disease (22 cases), cardiac arrhythmia (five cases), cardiomegaly (five cases), ischaemic heart disease (six cases), myocardial infarction (four cases) myocardial ischaemia (two cases), cardiomyopathy (two cases), left ventricular hypertrophy (one case), aortic rupture (one case) and endocarditis (one case).

Kaye et al Addiction 2008

Table 5 Major organ pathology.

Type of pathology (%)	*Males (n = 172)*	*Females (n = 48)*	*All (n = 220)*
Cardiovascular pathology	55	48	54
Atherosclerosis	43	26*	39
Mild	20	15	19
Moderate	3	2	3
Severe	17	7	15
Unspecified	1	2	1
Sites of atherosclerosis	40	17*	35
Coronary arteries	14	17	14
Aorta			
Cardiomegaly	18	6	16
Hypertrophy	13	11	12
Ischaemic heart disease	6	2	5
Cerebrovascular pathology†	19	26	20
Pulmonary pathology‡	42	42	42
Hepatic pathology§	61	60	61
Renal pathology¶	12	13	13

- Cardiovascular pathology was present in 54% of methamphetamine-related fatalities (mean age 32 years old)
- Very high rates of coronary artery disease in this group – likely accelerated

Kaye et al Addiction 2008

methamphetamine-associated malignant hypertension

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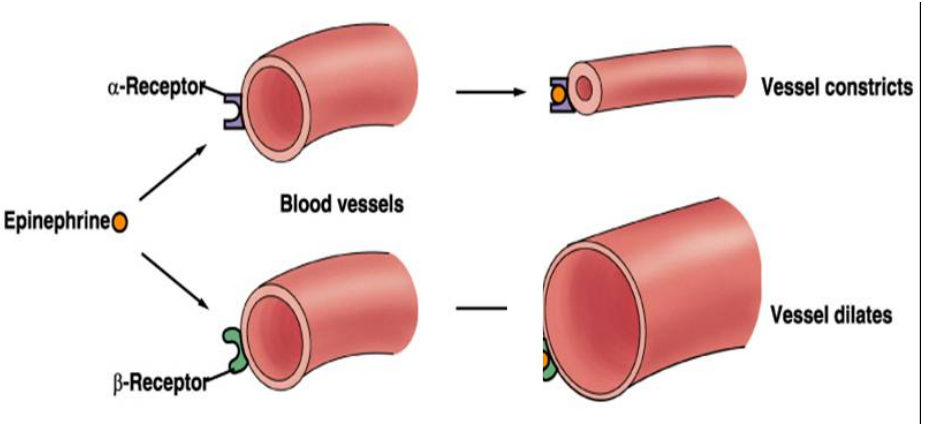
beta-blockers and methamphetamines

- Sympathomimetic toxicity (methamphetamine, cocaine) can cause generalised increase of both alpha and beta-adrenergic tone

- giving B-bs may cause unopposed excess of alpha-adrenergic vasoconstriction (as will lose the beta-2 mediated vasodilation opposing the alpha-adrenergic effects)



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beta-blocker avoidance

- Most of the literature regarding B-bs in sympathomimetic toxicity is to do with cocaine and phaeochromocytomas
    - For example, in one double-blinded RCT, patients in Texas underwent coronary angiogram and were given cocaine then subsequently beta-blockers. Administration of beta-blockers in patients given cocaine worsened coronary artery vasospasm and peripheral hypertension.
- Extrapolating to methamphetamines, current suggestion is to avoid B-bs if at all able until after 'adequate' alpha-blockade has been provided (?10-14 days based on phaeochromocytoma literature).
- Benzodiazepines may be more efficacious in inhibiting CNS over-stimulation.

Lange Annals of Internal Med 1990

Table 10. Specific interventions for cardiovascular related amphetamine toxicity

- Hypertension (avoid beta-blockers)
  - Benzodiazepines
    - Diazepam or clonazepam boluses
  - Vasodilators
    - GTN – 5 µg/min and titrate
    - Nitroprusside – 0.25–10 µg/kg/min
  - Alpha-adrenergic antagonists
    - Prazosin – 2–5 mg orally
    - Phentolamine – 5 mg slow i.v.
- Dysrhythmias (avoid moderate to long acting beta-blockers)
  - Sinus tachycardia
    - Observation only
    - Benzodiazepines if associated with CNS overstimulation
  - Supraventricular tachycardia
    - Adenosine – 6 mg initial bolus
    - Verapamil – 5 mg i.v. (beware of hypotension)
    - Flecainide – 2 mg/kg i.v. over 30 min
    - Electrical cardioversion
  - Ventricular dysrhythmias
    - Standard protocols

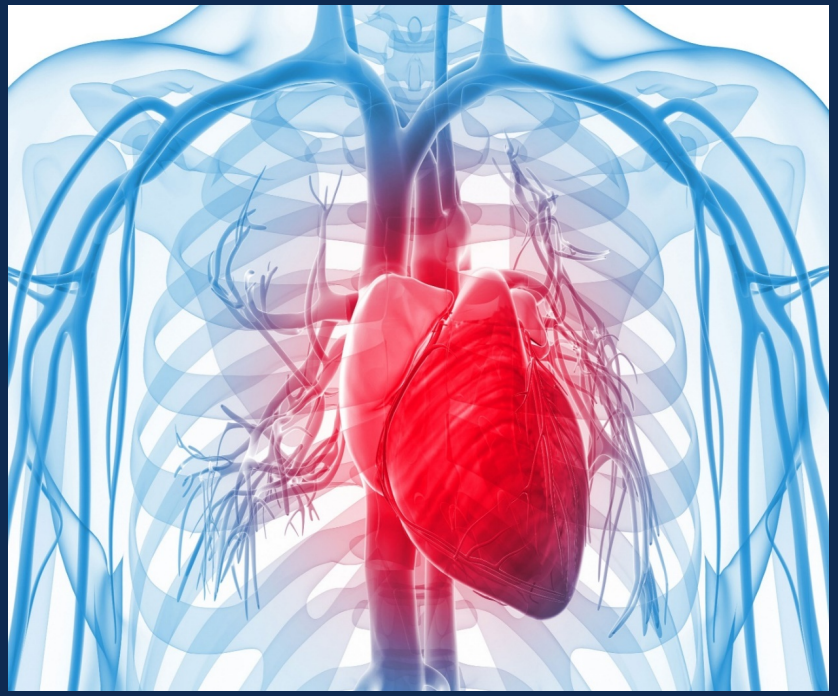
Greene et al Emerg Med Australasia 2008

Table 3 Management approaches to methamphetamine-related cardiac complications

Complication	What is still recommended?	What is unique in methamphetamines?
Hypertension	Nitrate therapy α-antagonist ACE inhibitor Calcium channel blocker	Consider benzodiazepines in acute setting to reduce catecholamine surge Use α-antagonists prior to initiating β-antagonists Consider carvedilol or labetalol in longer-term (dual α- and β-antagonism)
Aortic dissection	Blood pressure control Surgical intervention	Avoid β-antagonists: use alternative agents to control heart rate and blood pressure Drug history and consider urinalysis in young patients with aortic dissection
Acute coronary syndrome	Coronary angiography Anticoagulation, analgesia	May have significant burden of disease for age Use α -antagonists prior to initiating β-antagonists Consider carvedilol as β-blocker of choice in longer-term (dual α- and β-antagonism)
Pulmonary arterial hypertension	Right heart catheterisation Vasculitic screen 6 minute walk test Exclude chronic thromboembolic pulmonary hypertension Echocardiogram	Drug history and consider urinalysis in a diagnostic workup of idiopathic pulmonary arterial hypertension Counselling regarding abstinence from methamphetamines
Cardiomyopathy	ACE inhibitor B-blocker (after α-blocker) Coronary angiogram	Use α -antagonists prior to initiating β-antagonists Consider carvedilol or labetalol as β-blocker of choice in longer-term Drug history and consider urinalysis in young patients with cardiomyopathy

Paratz et al 2015

Methamphetamine-associated cardiomyopathy



CASE REPORT Open Access

Report of methamphetamine use and cardiomyopathy in three patients

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Abstract

Background: Methamphetamine (meth) is a stimulant used illegally around the world, including in Iran. Cardiomyopathy and cardiac failure may occur following chronic meth use and may cause the patients referred to the emergency department.

Case reports: A 28-year old man and two women, ages 29 and 31-year-old, with a history of meth use, were admitted to the emergency department with severe dyspnea at rest. Each had sinus tachycardia with tachypnea and an echocardiogram that showed severe systolic dysfunction consistent with heart failure. Additional evaluation in the hospital revealed cardiomyopathy with no other etiology other than the meth use.

Conclusion: There are several reports that show an increase in frequency of meth use, suggesting that cardiomyopathy and acute heart failure may be a new medical concern.

Keywords: Cardiomyopathy, Congestive heart failure, Methamphetamine

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CLINICAL RESEARCH STUDY

The Association of Methamphetamine Use and Cardiomyopathy in Young Patients

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ABSTRACT

PURPOSE: Methamphetamine is the most widespread illegally used stimulant in the United States. Previously published case reports and series suggest a potential association between methamphetamine exposure and cardiomyopathy. The objective of this study is to demonstrate an association between methamphetamine use and cardiomyopathy.

SUBJECT AND METHODS: Case-control study based on chart review of discharges from a tertiary care medical center from January 2001 to June 2004. Patients were ≤45 years old. Cases included patients with a discharge diagnosis of either cardiomyopathy or heart failure. Controls included hospitalized patients who had an echocardiographic assessment of left ventricular function with ejection fraction ≥55% and no wall motion abnormalities.

RESULTS: One hundred and seven cases and 114 controls were identified. Both groups had similar gender distribution, length of hospital stay, rates of health insurance, prevalence of coronary artery disease, diabetes mellitus, hypertension, cigarette smoking, alcohol abuse, and marijuana and cocaine use. Cases were older than controls (mean age: 38 vs 35 years; $P=.008$), had higher body mass index (BMI) (mean BMI: 37 vs 30 kg/m^2; $P<.001$), and higher prevalence of renal failure (13% vs 4.4%; $P=.03$). Methamphetamine users had a 3.7-fold increased odds ratio [95% confidence interval, 1.8-7.8] for cardiomyopathy, adjusting for age, body mass index, and renal failure.

CONCLUSIONS: Methamphetamine use was associated with cardiomyopathy in young patients.

Yeo KK, et al. Am J Med 2007.

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Young patients with CM

- In one cohort of young patients with a diagnosis of 'idiopathic' cardiomyopathy in Hawaii, prevalence of methamphetamine abuse was found to be above-average on screening (questioning and urinalysis)
  - 107 young patients with cardiomyopathy (<45yo) vs age-matched controls
  - 40% of the young patients with cardiomyopathy were abusing methamphetamines vs 20% of the controls (OR 3.0, 95% CI 1.6-5.7)
- Examining within the cohort of young patients with cardiomyopathy, mean LVEF was significantly lower amongst heart failure patients abusing methamphetamines (26%) vs those who were not (35%) (p=0.009)

Yeo KK, et al. Am J Med 2007.

Table 2 Differing patterns of methamphetamine-associated cardiomyopathy

Pattern	Presumed mechanism	Reported in literature
Dilated	Direct toxicity of methamphetamine to cardiac myocytes	Rajs 1979, Jacobs 1989, Nestor 1989, Hong 1991, Wijetunga 2003, Ito 2009 [11–16]
Hypertrophic	Profound hypertension (increased peripheral vascular resistance) from activation of peripheral α- and β-adrenoreceptors	Nishida 2003 [10]
Stress cardiomyopathy (Takotsubo or reverse-Takotsubo pattern)	Acute effect of catecholamines on adrenoreceptors in myocardium	Movahed 2008, Srikanth 2008 [17,18]

Paratz et al, 2015

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Heart, Lung and Circulation (2016) **25**, 325–332
1443-9506/04/$36.00
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REVIEW

The Cardiac Complications of Methamphetamines

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Methamphetamines are increasingly popular drugs of abuse in Australia, and are rising in purity. The rising popularity and purity of methamphetamines has notably increased demands upon Australian medical services.

Methamphetamines are sympathomimetic amines with a range of adverse effects upon multiple organ systems. Cardiovascular complications are the second leading cause of death in methamphetamine abusers, and there appears to be a high prevalence of cardiac pathology. Cardiovascular pathology frequently seen in methamphetamine abusers includes hypertension, aortic dissection, acute coronary syndromes, pulmonary arterial hypertension and methamphetamine-associated cardiomyopathy.

The rising prevalence of methamphetamine abuse is likely to increase the burden of cardiovascular pathology in Australians. A National Parliamentary Enquiry was opened in March 2015 to address concerns regarding the medical and social impacts of methamphetamine abuse. From April 2015, a National 'Ice

Paratz et al, 2015

What provokes methamphetamine associated cardiomyopathies?

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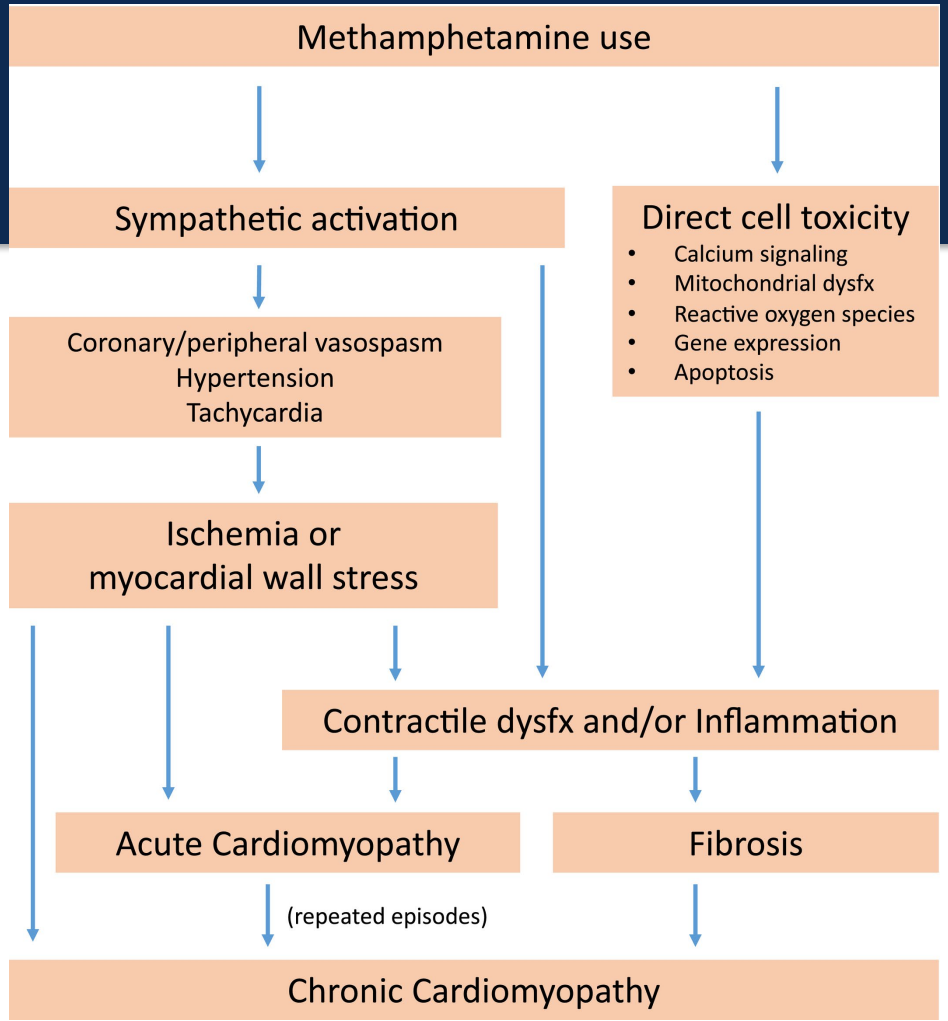

Reddy et al 2020

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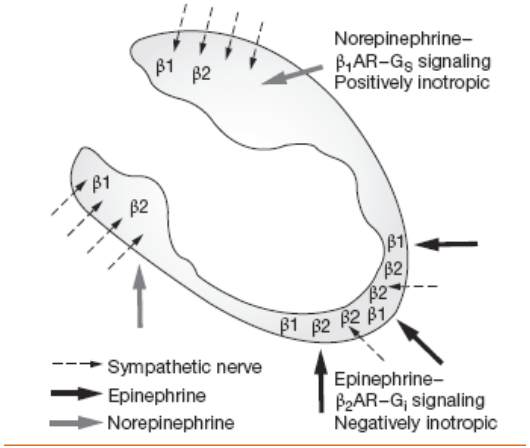
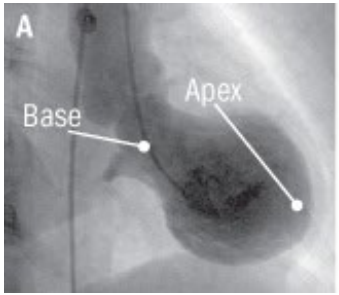
catecholamine excess theory

- adrenergic-driven with sustained periods over time, similar to mechanism of a tachycardia-induced cardiomyopathy [*catecholamine excess theory*]
- may have a Takotsubo-like behaviour





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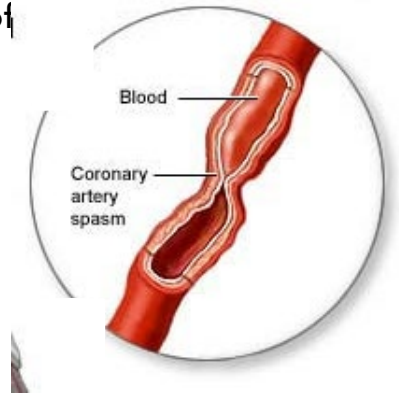
coronary vasospasm theory

- cumulative effect of multiple recurrent hypertensive crises leading to progressive LV failure [*coronary vasospasm theory*]
  - impact of HTN in causing LV remodelling
  - impact of recurrent episodes of



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direct myocardial toxicity

- rats given methamphetamines demonstrate severe LV systolic dysfunction with ventricular myoglobin loss and mitochondrial injury with increases in reactive oxygen species.
- proposed to activate Ca2+/calmodulin protein kinase II which leads to myocardial hypertrophy and fibrosis
- human autopsy studies show interstitial fibrosis and contraction band necrosis in the myocardium
    - **These create a substrate for re-entrant arrhythmias including VT**

Wijetunga et al J Clin Toxicol 2003

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more severe cardiomyopathy

- Appear to progress more rapidly than either idiopathic dilated cardiomyopathy or ischaemic cardiomyopathy patients
    - Case-control study of 59 patients (<45yo) with idiopathic dilated cardiomyopathy vs methamphetamine-induced cardiomyopathy
    - Methamphetamine abusers had TTE findings of more severe DCM compared to non-abusers for same progression of time from diagnosis
    - LA volume (120mL vs 86mL), LV end-diastolic volume (202mL vs 157mL), LV end-systolic volume (136mL vs 92mL), LVEF (33% vs 45%)

- Compared to ischaemic cardiomyopathy patients, also have more severe disease with worsened myocardial energy usage for same degree of LVEF
    - mitochrondrial swelling, disruption of oxidative phosphorylation processes

Ito H et al. Clin Cardiol 2009
Won SK et al Circulation 2013

How is methamphetamine-associated cardiomyopathy identified?

Can it be treated / reversed?

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http://dx.doi.org/10.1016/j.hlc.2016.11.005

ORIGINAL ARTICLE

Is an Abnormal ECG Just the Tip of the ICE-berg? Examining the Utility of Electrocardiography in Detecting Methamphetamine-Induced Cardiac Pathology

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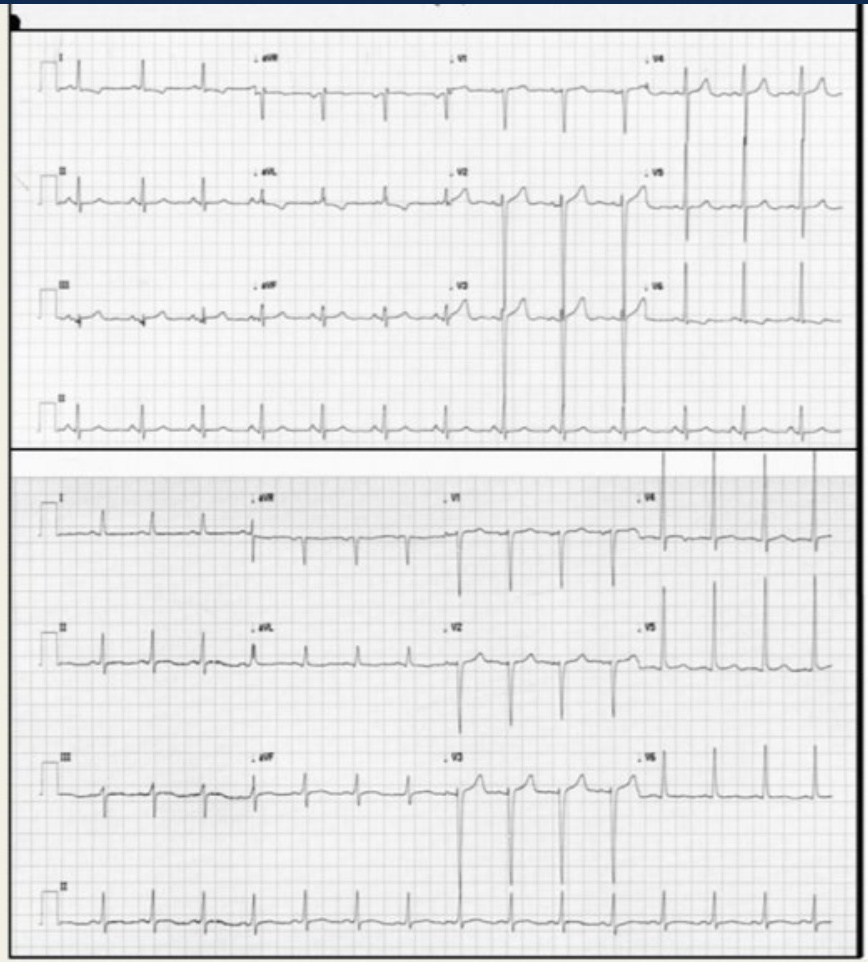
Received 7 March 2016; received in revised form 2 November 2016; accepted 9 November 2016; online published-ahead-of-print xxx

Background Methamphetamine use is escalating in Australia and New Zealand, with increasing emergency department attendance and mortality. Cardiac complications play a large role in methamphetamine-related mortality, and it would be informative to assess the frequency of abnormal electrocardiograms (ECGs) amongst

Paratz et al, 2015



3. A 37-year-old female with a 5 year history of smoking methamphetamines.
- ECG shows left ventricular hypertrophy and strain pattern, with a normal QTc interval.

4. A 27-year-old male with a 7 year history of smoking methamphetamines
- ECG shows left ventricular hypertrophy and anterior Q waves, with a normal QTc interval.

Paratz et al, 2015

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ECG abnormalities

ECG abnormalities seen in 72% of methamphetamine users, mean age 33 years
Compared to age and gender-matched controls:

- More tachyarrhythmias
- More right axis deviation
- More features of left ventricular hypertropy
- More signs of ischaemia: T wave inversion, Q waves
- Longer QTc interval

Of those that had echocardiograms performed, 38% had LV dysfunction.

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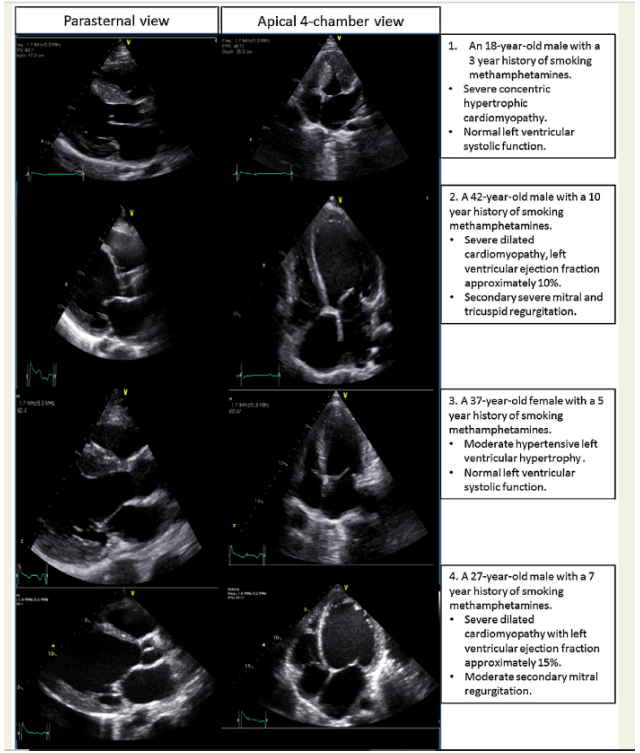
Echocardiography

Reduced systolic (pumping) function?

Remodelled heart – altered chamber sizes?

Any important other disease that needs intervention?



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reversibility

- methamphetamine-associated cardiomyopathy appears to be reversible, at least initially
- some patients have a Takotsubo-like pattern or even reverse-Takotsubo pattern on imaging that may reverse completely with abstinence
- multiple case studies of patients with significant dilated cardiomyopathy who abstained from methamphetamines and commenced medical therapy with excellent improvements
- the 'window period' of reversibility remains unclear

Won et al. Clin Cardiol 2013

Journal of Cardiovascular Magnetic Resonance

BioMed Central

Case report

Open Access

Recovery of methamphetamine associated cardiomyopathy predicted by late gadolinium enhanced cardiovascular magnetic resonance

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Abstract

Methamphetamine is known to cause a cardiomyopathy which may be reversible with appropriate medical therapy and cessation of use. Late gadolinium enhancement cardiovascular magnetic resonance (CMR) has been shown to identify fibrosis in ischemic and non-ischemic cardiomyopathies. We present a case of severe methamphetamine-associated cardiomyopathy in which cardiac function recovered after 6 months. Evaluation by CMR using late gadolinium enhancement was notable for an absence of enhancement, suggesting an absence of irreversible myocyte injury and a good prognosis. CMR may be useful to predict recovery in toxin-associated non-ischemic cardiomyopathies.

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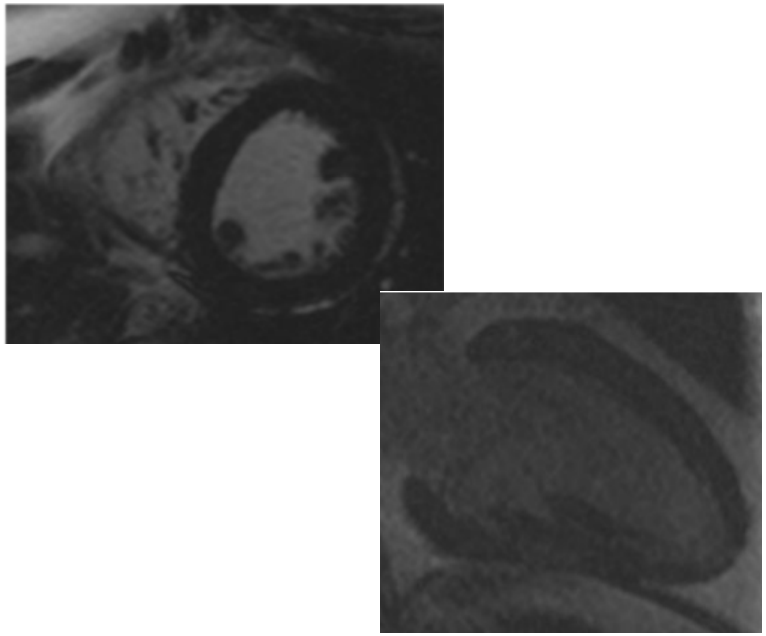
predicting reversibility

- cardiac MRI may be useful in identifying patients with recoverable function :
  - one case report describes a patient with severe methamphetamine-associated cardiomyopathy who underwent cardiac MR : this demonstrated no delayed gadolinium enhancement to suggest any significant fibrosis
  - subsequently abstained from methamphetamines and commenced appropriate medical therapy
  - LVEF improved from 37% to 64%, LV mass from 234g to 185g



Lopez et al. J Cardiovasc Mag Res 2009

INTERNAL MEDICINE JOURNAL

Methamphetamine-associated cardiomyopathy: patterns and predictors of recovery

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Key words
methamphetamine, cardiomyopathy, reverse Takotsubo, heart failure.

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Abstract

Background: Methamphetamine abuse is a growing public health problem, and increasing numbers of patients are admitted with methamphetamine-associated cardiomyopathy (MAC).

Aim: We sought to characterise the patterns of this disease and identify predictors of recovery.

Methods: We retrospectively studied consecutive patients diagnosed with MAC between January 2006 and July 2015.

Results: We identified 20 patients (14 males, 6 females) with mean age 35 ± 9 years. Most had very severe systolic dysfunction (mean left ventricular ejection fraction (LVEF) 19.7 ± 11.4%) at presentation with 14 requiring inotropes and 5 requiring mechanical support. The pattern of systolic dysfunction was global in 14 patients, while 6 patients had a 'reverse Takotsubo' (RT) pattern with severely hypokinetic basal-mid segments and apical preservation. RT patients were predominantly female, had a short history of methamphetamine abuse and had higher cardiac enzyme levels. Patients with global dysfunction tended to have mid-wall fibrosis on cardiac magnetic resonance imaging. On follow-up transthoracic echocardiography, 6 out of 19 (32%) had normalisation of LVEF (LVEF ≥ 50%) within 6 weeks. Smaller left ventricular and left atrial

Voskoboinik et al 2016

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Key points

Very unwell population – mean age 35yo but 20% needed mechanical support and 70% needed inotropic support

Factors predicting recovery:

Smaller LV and LA size (less adverse remodelling)

Shorter duration of methamphetamine use

Adverse features:

Fibrosis on cardiac MRI

Bigger LV and LA size

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medical therapy

- As with our patient, the current standard of care consists of :
    - **Abstinence** from methamphetamines
    - **Appropriate anti-heart failure therapy**
        - ACE inhibitor
        - Beta-blocker
        - Spironolactone
        - Diuretics
    - **Device therapy** if qualifies and is felt to be clinically appropriate

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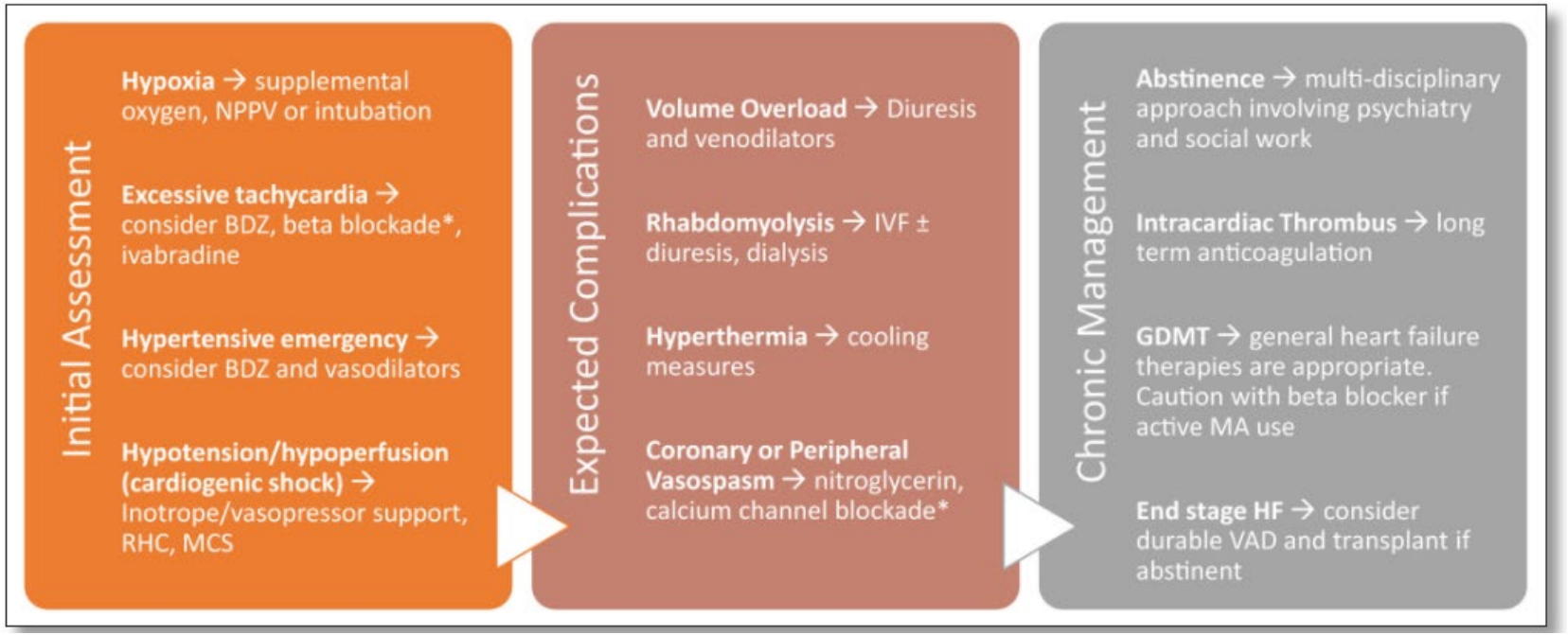
Clinical management



Reddy et al 2020

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Summary

Methamphetamines cause a spectrum of cardiac damage. Key things to think about are:

- Malignant hypertension (incl risk aortic dissection)
- Accelerated coronary artery disease
- Severe cardiomyopathy

What can clinicians do?

- 'screening approach' is reasonable: ECG for all, low threshold to consider echo
- Be aware of management differences in treating hypertensive crises
    - Sedation and vasodilators may be more important than beta-blockade
- Multidisciplinary involvement and support with cardiology teams to discuss prognosis, feasibility of advanced therapies

Q & A

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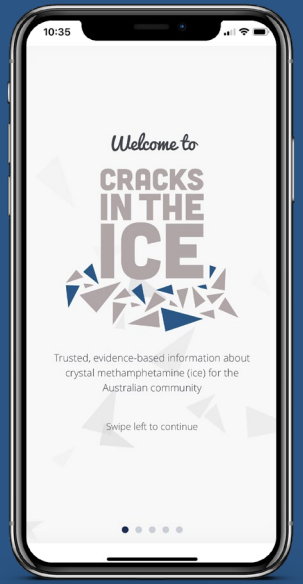

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