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**The Impact of Folic Acid and Vitamin B12 Supplementation on Blood Pressure and Arterial Stiffness in Subjects with Subnormal Micronutrient Intake**K.S. Woo<sup>1,\*</sup>, P. Chook<sup>1</sup>, L.L.T. Chan<sup>1</sup>, D.S. Celermajer<sup>2</sup><sup>1</sup> *The Chinese University of Hong Kong, Hong Kong;* <sup>2</sup> *The University of Sydney, Sydney, Australia***Introduction:** Folic acid (FA) supplementation improves atherogenic processes in coronary and asymptomatic subjects, but its impact on blood pressure and arterial stiffness remains unknown.**Methods:** 207 asymptomatic subjects (aged  $45 \pm 8$  years) in rural northern China were randomised to take FA (5 mg/day), vitamin B12 (500 µg/day), B12 + FA or placebo in double-blinded design for 6 months, followed by open-label B12 + FA for 6 more months. Radial artery augmentation index (AI) and pulse wave velocity (PWV) were measured by SphygmoCor.**Results:** Blood B12 and folate levels were low at baseline, but significantly increased after FA, B12 and B12 + FA, and not after placebo treatment, while fasting homocysteine decreased significantly after FA and B12 + FA treatments ( $p < 0.001$ ). Systolic (SBP) or diastolic pressures (DBP) decreased marginally ( $p < 0.05$ ) during placebo and all three active treatment periods ( $p < 0.001$ ). There was no significant change in AI or PWV.

	Placebo (n=53)		B12 (n=52)		FA (n=51)		B12+FA (n=51)	
	0-month	6-months	0-month	6-months	0-month	6-months	0-month	6-months
FA (nmol/l)	10.4 ± 2.9	12.3 ± 7.5	10.4 ± 2.9	12.3 ± 7.5	10.7 ± 5.0	28.7 ± 19.2***	13.1 ± 8.3	25.3 ± 18.1***
B12 (µg/l)	201 ± 113	173 ± 81	214 ± 109	305 ± 131**	182 ± 88	163 ± 86	188 ± 109	289 ± 170***
SBP (mmHg)	128 ± 14	124 ± 17 <sup>‡</sup>	131 ± 20	130 ± 20	126 ± 20	119 ± 19**	134 ± 20	126 ± 24**
DBP (mmHg)	85 ± 8.5	82 ± 10 <sup>‡</sup>	87 ± 12	82 ± 13**	83 ± 11	79 ± 10**	88 ± 9	81 ± 11***

Compared with baseline: <sup>‡</sup> $p < 0.05$ ; <sup>\*\*</sup> $p < 0.001$ ; <sup>\*\*\*</sup> $p < 0.0001$ .Sustained decrease in SBP ( $p = 0.001$ ) and DBP ( $p < 0.0001$ ) were observed after open-label FA + B12 treatment, associated with a significant decrease in AI ( $135 \pm 22\%$  to  $122 \pm 21\%$ ;  $p < 0.001$ ).**Conclusion:** Long-term FA and B12 supplementation improves blood pressure and arterial stiffness in subjects with subnormal intake.

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**Coronary Heart Disease in New Zealand 2001-2003: Estimates of Incidence and Prevalence Based on Routinely Collected Data**Martin Tobias<sup>1</sup>, Wing-Cheuk Chan<sup>2</sup>, Craig Wright<sup>1</sup>, Rod Jackson<sup>2</sup>, Stewart Mann<sup>3,\*</sup>, Li-Chia Yeh<sup>1</sup><sup>1</sup> *Public Health Intelligence, Ministry of Health, Wellington, New Zealand;* <sup>2</sup> *Department of Public Health, University of Auckland, Auckland, New Zealand;* <sup>3</sup> *Department of Medicine, University of Otago, Wellington, New Zealand***Objective:** We wished to estimate the incidence, prevalence, survival and mortality of coronary heart disease

(CHD) in New Zealand in 2001-2003 using routinely collected data.

**Method:** We estimated incidence from the sum of first CHD hospital admissions and out-of-hospital CHD deaths without a hospital admission in the preceding 5 years. Mortality was calculated from the sum of deaths coded to CHD and to related causes but with prior hospitalisation for CHD. We collated data from the New Zealand Health Information Service and carried out record linkage using the unique national identifier used throughout the New Zealand health care system. From these estimates, we built multi-state lifetables and thereby calculated estimates for prevalence, survival, lifetable risk, and median age at onset.**Results:** The lifetime risk of acquiring CHD was estimated at 35% for males and 28% for females, prevalence rising from around 2% for males and 0.5% for females at age 40-44 and to around 18% and 12%, respectively at age 85-89. Median age at onset of CHD was 67.5 years for males and 77.5 years for females. Median survival duration from the initial event was 9.5 years for males and 6.2 years for females. On average, males with CHD lost 6.9 years of life expectancy and females 5.7 years.**Conclusion:** We have developed an internally consistent picture of the descriptive epidemiology of CHD for the whole New Zealand population in 2001-2003 which has

relevance to prioritisation and planning of relevant health care services.

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**Irukandji Syndrome, Cause for Troponin Leak and Stress Cardiomyopathy**Keith Tiong<sup>1,2,\*</sup>, Sang-Won Jin<sup>1,2</sup>, Jamie Seymour<sup>1,2</sup>, Peter Pereira<sup>1,2</sup><sup>1</sup> *Cairns Base Hospital, Cairns, Queensland, Australia;* <sup>2</sup> *James Cook University, Cairns, Queensland, Australia***Introduction:** Carukia Barseni (also known as Irukandji) is a type of box jelly fish unique to North Queensland. We present a case series of irukandji syndrome which presented to Cairns Base Hospital recently.**Method/Design:** Consents were obtained by all subjects. Regular observations were performed, baseline electrocardiograms, serial serum troponins and differential white cell counts. Serial transthoracic echocardiograms were performed in cases of positive troponins.

**Results:** Four cases with positive troponins were identified. The age group range from 14 to 40 years of age, median age 24.3 are males and 1 female. These cases presents within 24 h of contact. Two cases were documented with persistent hypertension, one with persistent tachycardia, three with heart rate variabilities. Only two electrocardiograms were abnormal. Intravenous magnesium and/or nitrates infusion were given in three of the cases. The eosinophils counts were normal in all cases. These were associated with a rise in the level of serum troponins. Serial transthoracic echocardiograms were performed to identify the cause of troponin rise which included regional wall motion abnormalities, left ventricular function by Simpson's method and tissue doppler imaging. There were only one case of impaired left ventricular function, identified as a case of stress cardiomyopathy. **Conclusion:** Irukandji syndrome is associated with sudden catecholamines release. The sympathetic overdrive is a cause of transient troponin rise, which precipitates stress cardiomyopathy.

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### Angiotensin-Converting Enzyme Inhibition Reduces Left Ventricular Outflow Tract Diameter in Marfan Syndrome; Potential Role of Reduced Transforming Growth Factor- $\beta$ Signalling

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**Background:** We have previously shown that the angiotensin-converting enzyme (ACE) inhibitor perindopril reduced aortic diameter by 3.7 mm in Marfan syndrome (MFS) patients ( $p < 0.0001$ ). Excessive signalling by the transforming growth factor- $\beta$  (TGF- $\beta$ ), which also contributes to matrix metalloproteinase (MMP) activation and extracellular matrix degeneration, is believed to play a crucial role in the development of aortic dilatation. We hypothesised that reduction in aortic diameter would correlate with reduction in plasma TGF- $\beta$  and MMP levels. **Methods:** 17 MFS patients (aged  $33 \pm 5$  (mean  $\pm$  S.D.)) on standard  $\beta$ -blocker therapy were randomised to also receive either perindopril (8 mg od,  $n = 10$ ) or placebo ( $n = 7$ ) for 24 weeks in a double blind study. Venous blood samples were analysed for latent and active TGF- $\beta$ , and MMP 2 and MMP 3 levels.

**Results:** Compared to placebo perindopril significantly reduced left ventricular outflow tract (LVOT) diameter by  $2.8 \pm 0.4$  mm (placebo  $+1.1 \pm 0.3$  mm,  $p < 0.0001$ ), latent TGF- $\beta$  levels by  $14.0 \pm 4.5$  ng/ml (placebo  $+2.0 \pm 2.3$  ng/ml,  $p = 0.01$ ), active TGF- $\beta$  levels by  $4 \pm 1$  ng/ml (placebo,

$+3 \pm 1$  ng/ml,  $p = 0.02$ ), MMP 2 levels by  $22 \pm 6$  ng/ml (placebo  $+8 \pm 3$  ng/ml,  $p < 0.001$ ), and MMP 3 levels by  $5 \pm 1$  ng/ml (placebo  $+2 \pm 1$  ng/ml,  $p < 0.001$ ). There were moderately strong correlations between the pre/post intervention change in LVOT diameter and the change in both latent ( $r = 0.49$ ,  $p = 0.04$ ) and active TGF- $\beta$  ( $r = 0.74$ ,  $p = 0.002$ ), MMP 2 ( $r = 0.75$ ,  $p = 0.001$ ), and MMP 3 plasma levels ( $r = 0.81$ ,  $p < 0.0001$ ).

**Conclusion:** ACE inhibition reduced LVOT diameter in MFS patients possibly through attenuation of TGF- $\beta$  signalling. Plasma TGF- $\beta$  could be a useful prognostic indicator of progression of aortic dilatation and response to therapy in MFS.

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### Outcome & Prognostic Factors on 57 Cases Of Infective Endocarditis in a Single Center

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A single centre retrospective review of all cases of IE for a 5-year period from June 2002. There were 57 episodes of IE in 47 patients. Seventy percent were definite IE using modified Duke Criteria (2000). Forty five episodes were native valve endocarditis, the remaining were prosthetic valve endocarditis and one case of pacemaker lead endocarditis. The mitral valve was most commonly involved. Mean age was 66 with bimodal peak at age group 21-30 and 81-90. The most commonly isolated organisms were Streptococci (37%) and Staphylococcus Aureus (35%). Forty nine percent of patients remained event free (survive without recurrence or operation) at the end of follow up period. Eight patients had recurrent endocarditis within the study period. Five cases (8.5%) had early recurrence of endocarditis within 60 days. Twelve patients (26%) died during follow up (mean 14 months). There was no significant increase in mortality of patients with history of recurrent endocarditis (38% vs. 28%;  $p = 0.39$ ). Staphylococcus Aureus was associated with increased mortality or need for valve surgery (OR 4.5; 95% CI 1.38-14.8), risk of neurological events (OR 8.9; 1.5-52), renal failure (OR 7.2; 1.7-30) and thrombocytopenia (OR 5.6; 1.4-22). Haematological parameters, renal function or inflammatory markers were not shown to be predictive of increased mortality or need for valve surgery.

**Conclusion:**

1. The mortality of IE remains high. Less than half of this cohort remained event free.
2. The micro organism involved is more predictive of mortality or need for surgery than recurrent endocarditis.