proximal tubular reabsorption of sodium which is thought to cause the hypertension.⁴⁵

Canessa and her colleagues first reported increased maximal rates of Na^+/Li^+ countertransport in erythrocytes from some patients with essential hypertension.⁴⁶ This has been confirmed, although abnormalities are hardly evident in hypertensive women⁴⁷ and blacks,⁴⁸ and body weight seems to have an effect in its own right.⁴⁹⁻⁵⁰ Lithium is not a natural substrate of the exchanger, which may function normally in a Na^+/Na^+ or Na^+/H^+ mode. The Na^+/H^+ antiport is well characterised: it is inhibited by amiloride, helps regulate intracellular pH, and promotes cell growth.⁵¹⁻⁵³ If Na^+/Li^+ exchange in erythrocytes is equivalent to Na^+/H^+ exchange, which is uncertain,⁵⁴⁻⁵⁵ then increased Na^+/H^+ exchange might be linked to the pathogenesis of hypertension in two ways: overactivity of the exchanger in the proximal renal tubule might cause sodium retention⁵³⁻⁵⁶; or overactivity in the erythrocyte might be a manifestation of increased growth factor activity causing hyperplasia or hypertrophy of vascular smooth muscle and hence increased vascular resistance.⁵⁷

Studying sodium transport across the cell membrane has given at best tantalising glimpses of the mechanisms in hypertension. We need to know more about these transporters in the myocytes of resistance vessels and more about their relation to transporters in blood cells.

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Pacemaker syndrome

Permanent cardiac pacing is the treatment for symptomatic bradycardia due to ventricular conduction defects, sinoatrial disease, and the carotid sinus syndrome. About 114 new pacemakers for every million people are implanted each year in Britain, and the rate is increasing. A stable rate of 300-400 per million may be reached nationally over the next five years and has already been approached by a few British centres and reached in several European countries.¹ It is increasingly important, therefore, that doctors appreciate that apparently satisfactory function of an implanted ventricular pacemaker may be associated with a whole range of cardiac symptoms.

such as dizzy spells, syncope, breathlessness, impaired exercise capacity, and postural hypotension. These symptoms may constitute the "pacemaker syndrome," which once recognised is readily treated.

The syndrome results from atrial contraction occurring during ventricular systole and is a consequence of ventricular pacing. The atria contract against closed atrioventricular valves and produce raised mean pressures in the right and left atria with reduced cardiac output and blood pressure.^{2,3} The pacemaker syndrome occurs when retrograde atrioventricular conduction is intact and conduction spreads from the paced ventricles to the atria generally through the atrioventricular node; this may happen even when antegrade conduction is blocked. Ventricular pacing competing with sinus rhythm may be similarly symptomatic.^{4,5} Dual chamber pacing, in which pacing and sensing occur in both the atrium and ventricle, is rarely associated with the pacemaker syndrome because the physiological relation of atrial contraction preceding ventricular contraction is maintained.⁶

Patients with pacemaker syndrome complain of syncope or near syncope, dizziness, chest pain, fatigue, breathlessness, palpitations, and neck pulsations or pressure. Examination may show cannon waves in the jugular venous pulse or palpable liver pulsations. The diagnosis is made by observing or reproducing the adverse haemodynamic effects, signs, and symptoms during ventricular pacing.^{4,5,7}

Though the incidence of this symptomatic pacemaker syndrome is low, many "asymptomatic" patients have felt better with dual chamber pacing when this has been compared with ventricular pacing by within patient randomised control studies.⁸⁻¹² This is mainly because sequential atrioventricular contraction is restored.

Up to a fifth of patients with preserved retrograde atrioventricular conduction develop symptoms suggestive of the pacemaker syndrome.⁵ Retrograde conduction is present in 40% of patients with complete antegrade atrioventricular block and 90% of patients with normal atrioventricular conduction who have the sick sinus or carotid sinus syndromes.¹³ Therefore atrial or dual chamber pacing is best for patients with the sick sinus syndrome¹¹ and dual chamber pacing for those with the carotid sinus syndrome.^{14,15}

Symptoms of the pacemaker syndrome may be reduced by reprogramming a ventricular pacemaker so that competition between paced and sinus rhythm is minimised by adding rate hysteresis such that pacing starts only after a pause substantially longer than the pacing interval.¹⁶ The antiarrhythmic agents flecainide and disopyramide are effective in blocking retrograde conduction in patients with pacemakers^{17,18} and may ameliorate pacemaker syndrome. Nevertheless, flecainide increases the pacing threshold and requires careful control of pacemaker output. Patients who do not respond to these measures must be upgraded to dual chamber pacing. In new patients who require pacing but retain antegrade conduction and have preserved retrograde conduction the syndrome may be prevented by implantation of a dual chamber pacemaker.

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Weight gain in pregnancy: eating for two or just getting fat?

What advice should a doctor give to a woman about her weight during pregnancy? Should he tell her to eat for two, or if she is gaining weight too fast should he advise her to diet? These are everyday questions, but, as so often happens, research has provided some conflicting answers.

The average woman gains 12.5 kg during pregnancy, though normal women may lose weight or gain over 20 kg.¹ Much of the increase is accounted for by the uterine contents, enlargement of maternal organs, and—particularly in the third trimester—increased body water.¹ The remainder, averaging about 3.5 kg, is fat. By a mechanism that is not fully understood² pregnancy alters carbohydrate metabolism,³ and subcutaneous fat is laid down over the abdomen, back, and upper thighs, mainly in the first and second trimesters.^{1,3} The fat is usually lost again afterwards—a process aided by lactation^{4,5}—and for most women long term weight gain is an effect of age rather than parity.^{1,3} Nevertheless, during pregnancy fat women tend to put on more weight than thin ones.³

Women who are overweight at the start of pregnancy face an increased risk of complications.^{3,6} Hypertensive disorders are more frequent,^{7,8} though the incidence in different reports varies widely—from 7%⁹ to 46%.^{3,10} Abnormal glucose tolerance is also more common, affecting 7-17% of obese women.⁷⁻¹⁰ Increased risks of urinary tract infections and possibly thrombophlebitis have been reported,⁸ but the risks of anaemia⁸ and preterm delivery¹¹ may be lower than in underweight women. Older studies found that perinatal mortality was higher among obese women,^{3,12} but more recent reports have not confirmed this^{7,9,10}; perinatal mortality may have been reduced in these patients because of recognition of the risks and consequent careful supervision.⁶ A recent American study concluded, however, that if

¹ Feruglio GA, Steinbach K. Pacing in the world today. World survey on cardiac pacing for the years 1979 to 1981. In: Feruglio GA, ed. *Proceedings of the second European symposium on cardiac pacing*. Padua: Piccin Medical, 1982:953-67.