for defects, and with a greater degree of certainty, than after intravenous diprydamole. Reversibility of defects at 3 h was similar for the two techniques. Six patients had no defects on either test. Of these, four had normal coronary arteriograms. One patient developed angina and ST segment depression after oral but not with intravenous diprydamole and these were abolished by 50 mg of aminophylline intravenously.

We conclude that oral diprydamole is at least as effective as intravenous diprydamole in ¹⁸¹stallium myocardial imaging. The technique is safe, easy to perform and suitable for out patient use.

17. DOES HOME BLOOD PRESSURE RECORDING CORRELATE WITH INTRA-ARTERIAL AND CLINIC BLOOD PRESSURES?
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The variability of blood pressure is well documented and multiple readings are necessary to improve the precision and to obtain a stable baseline. Many automated devices are available but have not withstood validation. Intra-arterial ambulatory blood pressure monitoring provides interesting, accurate and valid information, but is not suitable for routine use. We have therefore correlated the blood pressure of 34 hypertensive patients, measured by themselves at home with anaeroid sphygmomanometers, with clinic pressures, recorded with a random-zero sphygmomanometer and with intra-arterial ambulatory blood pressure. The latter was monitored with the Oxford transducer perfusion unit and Medilog tape recorder. Mean daytime intra-arterial blood pressure was computed on a purpose-built hybrid computer. Discrepancy values were calculated for inter-group comparisons. Scatter plots and between-method frequency distribution graphs have been plotted. In general the comparison of intra-arterial blood pressure with the mean of home recordings and the mean clinic pressures showed minor discrepancies between the different groups, but modest scatter on the graphs. The discrepancy value was much larger for the diastolic pressures, which showed a clear trend to be lower on intra-arterial recording. Comparison of home blood pressure with clinic pressures showed generally good agreement whereas a comparison of a random clinic pressure with mean clinic pressure showed considerable variability.

We think that home blood pressure measurement is a simple, cheap and effective method of obtaining relatively accurate serial blood pressure recordings.

18. THYROIDROPHIN-INDUCED STIMULATION OF LIPOGENESIS IN ADIPOCYTES
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Whereas hypertriglyceridaemia is known to be associated with primary hypothyroidism, in hypothyroidism secondary to pituitary disease it does not usually occur. Adipocytes are known to have thyrotrophin receptors and those obtained from thyroidectomized animals are also known to synthesize lipids and triglycerides significantly more rapidly than those from controls. We have hence investigated the possibility that thyrotrophin (TSH) and thyroid hormones may have an influence on the rate of triglyceride synthesis by adipocytes. We used the incorporation of ¹⁴C-glucose into the lipid fraction in adipocyte suspensions, obtained from rat epididymal fat pads, as an index of triglyceride synthesis. Tri-iodothyronine and TSH in graded concentrations were incubated with the adipocyte suspensions mentioned above.

Thyrotrophin was shown to induce a dose-related stimulation of triglyceride synthesis in adipocyte suspensions. The minimal stimulatory concentration of TSH in our experiments was 1 munit/ml, which is comparable with the circulating TSH concentrations in some patients with severe myxoedema. In contrast, T3 induced a significant reduction in the rate of lipogenesis but only at pharmacological concentrations (1000 times those observed in thyrotoxic patients). Since TSH is known to stimulate lipolysis, it would appear that the overall effect of TSH on the adipose tissue would be that of enhanced conversion of glucose into fatty acid residues.

We conclude from these experiments that the enhanced TSH concentration may be an important mediator of the enhanced lipid synthesis observed in thyroidectomized experimental animals. This effect of TSH may be relevant to the pathogenesis of hyperlipidaemia in primary myxoedema.

19. SHORT-TERM REGULATION OF LIPOPROTEIN LIPASE: STUDIES WITH VARIANT LIPOPROTEINS
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We have previously reported two variant triglyceride-rich lipoproteins which show impaired catabolism by lipoprotein lipase (EC 3.1.1.3). One lipoprotein had an acquired deficiency of apolipoprotein C-II and failed to activate lipoprotein lipase (Reckess et al., 1979. Clinical Science, 57, 139). A second, which contained excess of apo-C-III-2 peptide, provided an inefficient substrate for lipoprotein lipase although activation was normal (Stocks et al., 1979, Lancet, ii, 667-671). We now report a third variant in which the triglyceride-rich lipoprotein contains excess of apo-C-II peptide, activates lipoprotein lipase normally, but is an inefficient substrate.

The variant lipoprotein was obtained from a hypertriglyceridaemic man (aged 22 years, plasma triglyceride 12-5 mmol/l, cholesterol 4-2 mmol/l) with thalassaemia major, who had repeated blood transfusions since the age of 21 months. Triglyceride-rich lipoproteins (d < 1-006 g/ml) were isolated from patient and controls (with type IV or V hyperlipidaemia) by ultracentrifugation. The lipoproteins were delipidated by incubation with tetramethylurea or ethanol/ether. The C-peptides were electrophoresed on polyacrylamide gel (8 mmol of urea/l) or by isoelectric focusing (at pH 4-0-6-0). Gels were scanned in a Vitatron densitometer.

The peptide composition (%) of the variant lipoprotein compared with that of controls was: C-II, 45-2 vs 13-9 (3) vs 21-5 ± 4-9 (30) (P < 0-001); C-III-1, 28-1 ± 5-9 (9) vs 49-1 ± 10-5 (30); C-III-2, 26-4 ± 0-78 (9) vs 28-0 ± 5-2 (30); by isoelectric focusing, apo-C-II, 45-4 ± 2-7 (3) vs 20-7 ± 5-2 (7). When incubated with purified bovine milk lipoprotein lipase, the release of free fatty acids, measured by complexing with ⁶⁷Ni, was impaired from the variant lipoprotein compared with hypertriglyceridaemic controls 0-356 ± 0-38 (4) vs 0-769 ± 0-11 (4); P < 0-001 μmol of fatty acid h⁻¹ ml⁻¹, whereas activation of the enzyme was normal.

Electron microscopy of the variant and control lipoprotein was performed after negative staining at room temperature with 1% solution of phosphotungstic acid. Micrographs were taken at a magnification of x 10 000 and revealed non-spherical and flattened particles from the variant lipoprotein.

We conclude that abnormal 'packaging' of the variant lipoprotein with excess apo-C-II peptide may alter its reaction with lipoprotein lipase and lead to a defect in clearance of plasma triglyceride.

20. EFFECT OF A MIXED MEAL ON THE APO-PROTEINS OF VERY-LOW DENSITY LIPOPROTEINS
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