Vascular malformations are ideal targets for attack by embolisation, and treatment of these abnormalities is one of the most important applications of the technique. Aneurysms,[12] arteriovenous fistulas, and angiomas[1,10] have all been successfully treated, sometimes when surgical management would have been impossible. The lesion may be anatomically awkward for the surgeon (e.g., in the brain or spinal cord),[13] or it may be of a type (e.g., an angiomatous malformation) which tends to recur after attempted excision or ligation owing to the existence of multiple anastomotic communications which may enlarge and act as new arterial "feeders". Angiomatous lesions of the face are particularly troublesome because the surgeon must avoid postoperative scarring. In such angiomas embolisation has given extremely promising results, especially when the lesion is of a fast-flow type with big feeding arteries and arterialised veins. More difficult to eradicate are capillary and venous hæmangiomas (slow-flow lesions), but the outstanding work of DJINDJIAN and MERLAND,[10] shows that even these lesions succumb to suitable techniques and materials.

The great majority of cases in which therapeutic embolisation is useful fall into one of the three above categories—hæmorrhage, tumour, and vascular malformation. There are other disorders, however, in which embolisation is a therapeutic possibility: in hyperparathyroidism, parathyroid adenomas can be embolised as an alternative to surgery;[14] bilateral adrenal infarction can be produced by balloon-catheter occlusion of the adrenal veins (a technique used as an alternative to bilateral surgical adrenalectomy in advanced breast cancer and in Cushing's disease);[15] and, in hæmatological disorders caused or exacerbated by hypersplenism, embolisation of the spleen is a possibility in the patient who is a poor risk for splenectomy.[1,7,11]

As enthusiasm mounts, it is well to keep in mind the dangers and complications of transcatheter embolisation. Apart from the hazards of any selective arteriographic procedure, specific problems that may arise from the embolisation itself include pain in the embolised organ, inadvertent embolisation of normal structures, passage of emboli into the lungs through large arteriovenous shunts, œdema of the infarcted organ (which may give rise to local complications), adherence of the catheter tip to the vessel wall (when instant-setting polymers are used), and sensitivity reactions to the injected embolic materials. Delayed complications include persistent pain in the infarcted tissues, fever, infection (either systemic, or local leading to abscess formation), necrosis of inadvertently embolised normal tissues and organs, retrograde propagation of thrombus from an embolised artery, and (a theoretical complication) massive release of active humoral substances from infarcted endocrine tissues. In practice the incidence of serious complications is low. The commonest side-effects are discomfort at the site of embolisation and low-grade fever, both of which usually disappear within two or three days. The advances in catheter technology in the past decade have made most regions of the body accessible to the embolising catheter. While the more difficult and dangerous types of embolisation will properly remain the preserve of the specialist, the next decade will probably see embolisation procedures being done in almost every vascular radiology unit. The key to successful embolisation is the selection of suitable cases, and the best way of ensuring good selection is for radiologists, physicians, and surgeons to get to know the potential applications and limitations of this exciting branch of therapy.

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Hypertension—Salt Poisoning?

Two years ago we were sceptical about the value of modest salt restriction in the treatment of essential hypertension without renal failure.[1] Our statement of conventional wisdom did not go unchallenged. A study from Newcastle, New South Wales, revealed a small, but real fall in blood-pressure in a group of hypertensive subjects advised to reduce their salt intake.[2] The Australian workers also demonstrated something well-known about low-salt diets and human nature—that many symptomless individuals allegedly ingesting a salt-restricted diet make no serious attempt to adhere to it. Fortunately, defections from the diet are not difficult to detect. Body losses of sodium are almost entirely achieved by renal excretion, so that a 24-hour urinary sodium provides a good measure of salt intake in subjects who are in a steady state. Before salt restriction, mean urinary sodium in the Australian study was 195 mmol/day: this fell to a mean value of 157 mmol/day when patients were advised to adhere to a diet containing 70–100 mmol/day. The mean value of course includes some patients who clearly adhered closely to the salt-restricted diet and others who equally clearly did not.

The explanation of such a severe failure in human compliance is the unpleasantness of salt re-

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striction. The Kempner rice-fruit diet[3] (which reduced sodium intake to 10 mmol/day or less) undoubtedly lowered blood-pressure, but most individuals found it intolerable. More modest salt restriction[4] had little effect upon blood-pressure. The advent of potent antihypertensive drugs seemed to render salt restriction obsolete in the treatment of hypertension, but in the past few years control of salt intake has attracted new interest. There are two reasons for this. Firstly, there is the feeling that even the most apparently innocuous medication (such as thiazide diuretics[5]) may prove toxic when administered to large populations for long periods. Secondly, there is the intriguing theory that essential hypertension is a disease of excessive salt intake. The evidence for this comes largely from anthropological studies. Various primitive cultures subsist upon a vegetarian diet with a low salt content. Studies of such groups, ranging from the Yanomamo indians of Brazil[6] to Polynesian islanders[7] and New Guinea natives,[8] have revealed a low prevalence of hypertension: in addition there is no rise in blood-pressure with age. By contrast, inhabitants of northern Japan ingest large quantities of dietary salt[9] and this is associated with a high prevalence of hypertension and cardiovascular disease. There are many other differences between "primitive" and "civilised" societies which may account for the observed differences in blood-pressure; nevertheless, these observations are suggestive, and have led to the hypothesis that the "civilised" man's diet contains quantities of sodium far in excess of his physiological needs and that this excess produces hypertension in genetically-predisposed individuals.[10-13] It should be emphasised that the cultural differences in sodium intake associated with blood-pressure changes are extreme, varying from less than 10 mmol/day to more than 350 mmol/day. Compared with such values, the range of dietary sodium intake encountered by Western man (100–250 mmol/day) is small. It is therefore not surprising that there are no differences in 24-hour urinary sodium between hypertensive and normotensive individuals[14,15] within a single culture, although even in this context a recent Australian study begs to differ.[16] Most of the evidence indicates that, in man, large changes in sodium intake are required to produce substantial changes in blood-pressure. Moderate reduction in sodium intake may have a minor effect upon blood-pressure, perhaps roughly equal in magnitude to the effect of diuretics. It is therefore all the more surprising that a preliminary report from Massachusetts[17] links blood-pressure levels in schoolchildren to the sodium content of drinking-water. Water contributes only about 4% of total daily sodium ingestion. Despite this, significantly higher blood-pressure levels were observed in children from communities where drinking-water sodium was high (4·65 mmol/l) than where it was low (0·34 mmol/l). Clinicians who have failed to detect any effect of dietary sodium upon blood pressure will tend to regard the effect of such minuscule differences with incredulity: certainly, the possibilities for errors due to unrecognised cultural differences are great. Nevertheless, differences in drinking-water sodium operate for many years in stable communities, and a sustained minor deviation in sodium intake might have a different action from an acute change of dietary habit. This requires further examination. The alternative proposal,[12] that a massive public-health programme is required in order to return Western man to his ancestral low salt intake, would demand an unprecedented change in human nature.

A study from Bethesda attempts to elucidate some of the epidemiological confusion by postulating that sodium handling (like most other variables in essential hypertension) is heterogeneous.[18] The late Lewis K. Dahl, whose name is closely associated with the salt hypothesis, bred a strain of rats which became hypertensive only when exposed to a high salt intake.[19] Hypertension then persisted even when salt intake was reduced to normal. Kawasaki et al.[18] have proposed that patients with essential hypertension may be divided into those whose blood-pressure responds to changes in salt intake, and those who show no change. The former retain more sodium on a high-salt diet as their blood-pressure rises and lose less sodium when receiving a low-salt diet diet: they also share some characteristics with "low-renin" hypertension in that their plasma-renin responds poorly to low-salt diet. The distinction between the two groups seems arbitrary and the differences in renal physiology may be secondary to longstanding hypertension; nevertheless, if the proportion of salt-sensitive hypertensive patients does prove to vary between populations, a puzzling discrepancy in published reports on salt intake and hypertension will be resolved.

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