Prevention of Coronary Disease

SIR—I would like to comment on your attitude to the prevention of coronary heart disease at a population level as expressed by you in your leading article "The Prevention of Coronary Heart Disease" (21 September, p. 689).

You are discouraging about a preventive programme at a population level and you base a rather nihilistic outlook on the lack of certainty that such a programme might succeed and on the difficulties of its application. But, in my view, the preventive approach is promising in the field of coronary heart disease until we try it, and I submit that the accumulated experience of epidemiologists and clinicians would strongly suggest the logic and efficacy of prevention at a population level. In this regard I would agree with the view of J. B. Hickie quoted by you.

I do not question the reservations you make about the unproved significance of risk factors, about the efficacy of secondary prevention, and about patient co-operation, but it is regrettable that you should take such a discouraging attitude in this important public health field. If the time is not yet ripe to institute preventive measures on a national level, when will the time be ripe? Surely it is not too early to start a public educational campaign based on the considerable knowledge of risk factors which has already accrued. To my mind coronary heart disease is a highly predictable disease, and, because many of the risk factors are man-made and capable of modification, it is preventable at a population as well as a personal level.—I am, etc.,

Dublin 4, Eire.

RISTEARD MULCAHY.

REFERENCE


Asthma from Aspirin

SIR,—Your observations on this subject (4 January, p. 6) may turn out to be the thin end of a very big wedge. From the writer's perspective it appears that an allergic rhinitis and sinusitis are extremely common reactions of aspirin consumers in the early stages of a cold. This aggravates and complicates a perfectly harmless and transient rhinorrhea.

But why stop here? One can infer that the tractitis and bronchitis often noted in aspirin-treated flu and coryza are primarily allergic reactions to the therapy. This statement can easily be confirmed by treating such cases by placebo only and noting the unequivocal resolution without a cough. The acute gastritis with occult bleeds and the acute gastric erosions with haematemesis could be allergic signals in another target area.

Aspirin has been used for 50 years in ever-mounting quantities among vastly spreading populations under many disguises. Allergy to the drug will escalate. For this reason upper respiratory tract infections by virus will become increasingly complicated by drug allergy, will involve longer disability, and will require the deployment of expensive antibiotoics to control a drug-induced disease. I am, etc.,

Romford, Essex.

P. D. MULKEN.

Correspondence

Hong Kong Flu

SIR—I am surprised no one has written to you before concerning the hysterical outburst by the press and the B.B.C. with regard to this subject. Every day there is a headline. We must expect a severe epidemic or not; we should be vaccinated or cannot be a vaccine. It is as common as thousands of cases in America and hundreds of deaths; or, it's not very serious, no worse than previous epidemics: 250,000 doses of vaccine released next week.

Cannot the press and the B.B.C. be controlled or at least persuaded to behave more responsibly in a matter which affects every family doctor, and, by the unnecessary anxiety they have created, increases their work tremendously? Not to mention the disastrous effect it will have on production when the epidemic starts.

Am I right in assuming that the immediate effect of vaccination is to lower resistance for a couple of weeks and that by now it is too late to vaccinate, even if the vaccine becomes available? I managed with great difficulty to obtain 30 doses about six weeks ago and 10 doses last week. I am, etc.,

F. DESMOND MACCARTHY.

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Fibrinolysis in Renal Disease

SIR,—A decreased fibrinolysis in blood and tissues has been reported in patients with chronic renal disease. On the other hand, increased fibrinolysis has occasionally been observed in patients with carcinoma of the kidney. The findings indicate that kidneys of subjects without renal disease make a significant contribution of plasminogen activator to the circulation by supplying quantities greater than those removed in the liver (prompted us to investigate the activator content in the blood of patients with chronic renal failure to find out whether the diseased kidneys behaved similarly.

Twelve patients aged between 13 and 40 with chronic renal failure, all of whom were on regular outpatient haemodialysis, were investigated. Blood samples were collected from the arteriovenous shunt immediately before haemodialysis was begun and heparin administered. The Euglobulin-lysis time (E.L.T.), which was chosen for its value as a measure of plasminogen activator content, was estimated in duplicate by von Kaulla's method modified. The fibrinolytic activity was derived from these lysis times and expressed in units by multiplying their reciprocals by 10,000. Four separate estimations on four different days were made in all the patients. The E.L.T. in the nephrectomized patients with renal disease, no significant difference between the two groups was observed. In spite of this, it cannot be concluded that the diseased kidneys have no deleterious effects on production of plasminogen activators. The clear picture will not emerge until blood from the veins is studied.

The level of activator content in venous and arterial blood is different,

This study of patients undergoing renal dialysis and I. S. Menon have demonstrated that from venous blood the major part of this content is removed during the passage through the lungs. Consequently they suggested that this organ may play a part in the regulation of the plasminogen activator content in the circulation. If this hypothesis is proved to be correct, then even if there is a decrease in the activator content of the venous blood in patients with chronic renal failure this cannot be demonstrated by our study of arterial blood from the shunt.

Our thanks are due to Professor D. N. S. Kerr and Dr. H. A. Dewar for their encouragement, and Mr. Alan Martin for technical assistance.

—We are, etc.,

I. SUDHAKARAN MENON, T. S. RASTOGI.

Royal Victoria Infirmary and University of Newcastle upon Tyne, Newcastle upon Tyne.

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Prevention of Migraine

SIR.—Frequent attacks of migraine are notoriously difficult to treat. As the advertisement says, you can count on one finger the drugs that may prevent them. But methysyergide maleate has a large variety of adverse reactions and many physicians are loath to use it. For the past 15 months I have been trying amitriptyline hydrochloride (Tryptizol) in small doses, with most encouraging results. So far 12 patients with two or more attacks of typical migraine headache per week have been treated. All except one are highly satisfied with the results. The dosage used was three or four mg. tablets a day. On stopping the drug the attacks tended to return to their former frequency. No untoward side-effects were observed.
Fulminating Hyperthermia in General Anaesthesia

Sir,—The complications of hyperpyrexia during anaesthesia (21 December, p. 750) are very similar to those of extensive body temperature rise occurring elsewhere, irrespective of the body temperature level from which this rise occurs, and it would seem likely have a similar set of acutely life-threatening complications occurring during rewarming following prolonged induced hyperthermia were practically identical to those reported in heatstroke, and to a varying degree a similar pattern is found in other situations of large body temperature rise.

Increase in body temperature is accompanied by increase in blood volume. This change has been observed whether from hypothermic, normothermic, or hyperthermic levels, whether due to artificially imposed or to climatic conditions, to infection, or even in the presence of dehydration, in man and in animals. Increase in blood volume occurs as part of a generalized transfer of body water from the intracellular to the extracellular position, and within the latter from the interstitial to the intravascular position during body temperature rise, the reverse occurring with decrease in body temperature. These observations have been reviewed elsewhere.4,5 During rewarming following prolonged induced hyperthermia of 30–198 hours at 29–33°C,6 body volume increases by as much as 37% above the value at precooling and is then much higher than the value during hyperthermia. This was associated variably with increase in lumbar cerebrospinal fluid pressure and with progressive cerebral compression, leading to acute neurogenic pulmonary oedema and gastrointestinal complications (gastric dilatation and retention, intestinal mucosal haemorrhage), and with central circulatory failure. These complications were largely preventable or could be controlled by the proper rewarming measures, which, if the above hypothesis is correct, should apply equally to the management of complications occurring in other situations in which a large rise in body temperature occurs.

(1) Lumbar C.S.F. pressure should be reduced immediately if raised above normal, and this should be repeated as frequently as is necessary to control pressure rise. Pressures may rise to 500–600 mm Hg and can be reduced by 200 mm Hg or more without risk, with dramatic relief of cerebral compression and of acute neurogenic pulmonary oedema. Following accidental hypothermia lumbar C.S.F. pressure should be checked regularly during rewarming, and should be reduced as necessary to prevent the onset of cerebral compression.

(2) A rapidly acting diuretic (frusenide) should be given by the intravenous route. In the case of hyperpyrexia during anaesthesia these first two measures should be accomplished within a few minutes in an operating-theatre geared to meet this emergency, and should go a long way to prevent the two complications of body temperature rise most likely to lead to clinical deterioration. They should precede cooling. In the absence of sweating an effective diuretic is of value.

(3) Cooling should be induced as soon as possible following the above. In the unanesthetized subject this is likely to result in shivering and increase in heat production. This can be controlled effectively using a nitrous-oxide/oxygen mixture. There seems little point in reducing body temperature level to below normal.

(4) Gastric contents should be withdrawn at regular intervals and should be returned to the stomach if normal in colour, volume, and consistency.

(5) Since blood volume increases during body temperature rise, intravenous transfusion should be limited to the minimum volume necessary for the administration of essential drugs, etc.

(6) Metabolic abnormalities should be corrected.

(7) Patients should be observed for at least 24 hours in a special-care unit, and renewed evidence of cerebral compression, pulmonary oedema, and circulatory failure should be looked for.

(8) Rewarming, where applicable (accidental hyperthermia), should be slow. Patients should be covered by a sheet or one or two blankets, and rewarming should be spontaneous. Occasionally even spontaneous rewarming may be unduly rapid and lead to severe clinical deterioration. If this cannot be controlled by the above measures the patient should be cooled to 2.4°C C using pethidine, chlorpromazine, and surface cooling, and body temperature stabilized for four to six hours before permitting further rise in temperature.

—I am, etc.,
St. Mary's Hospital,
MAURICE BLOCH,
London.

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Through-knee Amputations

Sir,—In the article by Mr. P. F. Early (16 November, p. 418) and in the letter by Mr. J. F. Newcombe (30 November, p. 580) advocating through-knee amputations for ischaemic limb both writers comment on the tendency for necrosis of the long anterior flap necessary in this type of amputation with the consequent delay in healing. In most centres this also causes delay in fitting an artificial limb.

Almost all the patients one sees requiring amputation at this level are cases of vascular disease rather than trauma, and there seems, therefore, to be a strong case for avoiding the through-knee amputation owing to its poor record of healing. There is no doubt that a mid-thigh amputation should be avoided if at all possible because the average elderly patient does not become proficient in using a mid-thigh prosthesis, and all too often he prefers a wheel-chair.

It seems there is a strong case for an amputation slightly proximal to the through-knee level which will avoid the necessity for long skin flaps to cover the bulky knee joint. Mr. Robin Burkitt (30 November, p. 580) advocates the Gritti–Stokes supracondylar amputation, and certainly I agree that this heals much more satisfactorily. However, there is no doubt about getting the patella to balance on the divided end of the femoral shaft is not always easy, as Mr. K.