Liuzzo *et al* found that 24% of patients with myocardial infarction did not have raised C reactive protein values on admission.²

We are undertaking a prospective multicentre study of 2000 patients with unstable angina sponsored by the Italian National Research Council to assess the incremental prognostic value of markers of the acute phase response and their correlation with conventional risk factors and with the mechanisms that lead to enhanced production of C reactive protein. This study should improve our understanding of the role of inflammation. In the meantime what conclusions can we draw from available evidence about the usefulness of C reactive protein measurements?

Blood concentrations of C reactive protein (and possibly of serum amyloid A protein) may provide useful information beyond that available from the clinical presentation and other tests in two conditions. In patients presenting with unstable angina C reactive protein concentrations greater than 3 mg/l, and especially greater than 10 mg/l, predict a poor response to intensive medical treatment and indicate high risk of myocardial infarction. Such patients should be given intensive medical treatment but also be considered for urgent revascularisation. In patients with chronic ischaemic heart disease C reactive protein measurements identify a gradient of risk for myocardial infarction or cardiovascular death. The risk of these outcomes over three years rises from 0% in patients in the upper third of cholesterol concentrations (> 263 mg/dl) but the lower third of C reactive protein and fibrinogen concentrations to 10% in patients in the highest third of both cholesterol and C reactive protein and fibrinogen values. In this second, high risk, group reducing cholesterol concentra-

tions may have a much greater effect in reducing the incidence of events than in the first group, who appear to be at a considerably lower risk.

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Managing peanut allergy

Demands aggressive intervention in prevention and treatment

Sup 1074

The average American now ingests about 11 pounds (5 kg) of peanut products each year (United States Peanut Council, personal communication), about 55% as peanut butter and the rest in sweets, baked goods, and table nuts. As the American lifestyle has become more frenetic over the past 20 years, peanut products (such as peanut butter crackers, peanut butter and jelly sandwiches, and peanut butter sweets) have increasingly served as "snacks" or "quick meal substitutes" replacing more standard mealtime fare. Consequently, infants and young children are being exposed to peanut products earlier in life.

A study examining the efficacy of food allergen avoidance in preventing atopic disease in infants defined as being at high risk for atopy found that of 185 control infants, 80% had been exposed to peanut products by their first birthday and 100% by their second birthday.¹ Follow up at 7 years of age revealed that about 7% of high risk children had positive skin tests to peanut and 4% were felt to be reactive on the basis of history or oral food challenges.² The prevalence of peanut allergy seems to have increased over the past two decades. In comparable groups of children referred to us for evaluation of severe atopic dermatitis and possible food allergy, peanut sensitisation (positive skin prick test) increased by 55% while allergic reactions increased by 95% over a 10 year period. Today peanuts are believed to be one of the leading causes of food allergic reactions in the United States^{3,4} and, together with tree nuts, are probably the leading cause of fatal and near fatal anaphylaxis induced by food.⁵⁻⁸

It has long been assumed that peanut allergy was largely an American problem, but press reports in 1993 of six deaths due

to peanut allergy in Britain made it apparent that the problem was no longer confined to the North American continent. An article in this issue of the *BMJ* addresses the scope of the problem in Britain. Ewan (p 1074) reports 62 cases of peanut and/or nut allergy evaluated in a one year period. Peanuts accounted for nearly half of the allergies, with 55% of the allergies presenting by age 2 years and 92% by age 7 years. The author concludes that peanut allergy is occurring in very young children, that people allergic to peanuts are at increased risk of nut allergies, that peanut and nut allergy are rarely "outgrown," and that the prevalence of peanut and nut allergy is increasing.⁹ This is certainly in agreement with our data and those of other investigators in the United States (SA Bock, AW Burks, R Zeiger, personal communication). With this rising number of individuals at risk for potentially lethal reactions, aggressive intervention in both prevention and treatment is essential.

Firstly, some measures should be instituted in an attempt to stem the increasing prevalence of peanut and nut allergy. Infants at increased risk for developing peanut or nut allergy should be identified. These are infants from atopic families or families with other food allergies or atopic disorders. Their parents should be advised to eliminate all peanut products from the child's diet for at least three years, and mothers who are breast feeding should eliminate peanut products from their own diet. Children under 3 years of age who are being evaluated for other allergies should be tested for peanut allergy, and any child with peanut specific IgE antibodies should avoid all peanut and nut products for three to five years. If no reactions to inadvertent ingestions have occurred in the interim, the

child should be re-evaluated for evidence of peanut and nut specific IgE antibodies and clinical reactivity to peanuts.

The initial step in managing any patient with suspected peanut or nut allergy is to positively identify the food that is provoking the allergic reaction. When the patient's history and laboratory studies are not definitive, this may require a food challenge supervised by a physician in a specialised centre. Food allergic individuals at increased risk for severe anaphylactic reactions—that is, patients with histories of previous severe anaphylactic reactions or asthma, or both—should be provided with self injectable adrenaline (such as Ana-Kit or Epi-Pen) and antihistamine (liquid diphenhydramine or hydroxyzine). In a survey of patients with peanut allergy, Bock found that 25% had experienced an accidental ingestion and reaction in the past year (SA Bock, personal communication). The patient, parent, and any care providers should be given detailed information about the allergy, how to recognise anaphylaxis, how to administer adrenaline and antihistamine, and a detailed "emergency plan" for transporting the patient to a medical facility. About a third of children with fatal or near fatal reactions experienced a recurrence of anaphylactic symptoms, so all patients with systemic symptoms should be observed for at least four hours.⁶

An educational process is vital to ensure that the patient, family, and any care providers understand how to avoid all forms of peanut and nut allergens and the potential consequences of an inadvertent ingestion. Groups such as the Food Allergy Network in the United States and the British Allergy Foundation and the Anaphylaxis Campaign in Britain can provide patients with information about food allergen avoidance and educational materials for schools and parents of children with food allergies and anaphylaxis. When eating outside the home, people allergic to peanuts and nuts must always be extremely cautious and should not hesitate to ask specific and detailed questions about the preparation and ingredients of foods they are planning to eat. Peanut and nut products may be encountered at any meal—in breakfast cereals, trail mixes, chili and spaghetti sauces, gravies, oriental cooking (including

egg rolls), pastries, sweets, ice creams, desserts, and as garnishes for almost any food. Simply removing peanuts and nuts from a dish does not remove the contaminating protein. In addition, foods may be contaminated with peanut protein from "pressed or extruded" peanut oils, or oils or utensils used to cook foods containing peanuts. Unfortunately, it is not uncommon for patients dining in restaurants to react to a food that they were assured by a waiter did not contain peanuts or nuts. If there is any doubt about a food's ingredients, peanut and nut allergic individuals must not eat it.

Finally, the medical community should put pressure on governmental agencies responsible for product labelling and purity. All foods known to contain even minute amounts of peanut or nuts should be clearly labelled. Cross contamination in packaging and production plants is not infrequent, and when it occurs, there needs to be a system which provides this information quickly to the public. In addition, funding for research on the basic mechanisms of food allergy and potential therapeutic interventions needs to be increased.

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Smoking in public places

Self regulation of businesses is not working

Britain's environment minister, James Clappison, has released results of a National Opinion Poll study of provision of smoke free indoor environments by businesses that deal with the public.¹ Attempting to put a flattering spin on the findings, his press release claims "a substantial increase in the number of establishments covered by a smoking policy (between 1992 and 1995). In the travel sector, the percentage has doubled." But reading the report itself, only those content with bleak expectations bound to the incrementalist gains of self regulation could share the minister's enthusiasm. Only 38% of businesses in the travel sector were described as having effective policies. Just 36% of restaurants qualified and, most damning, even health establishments (at 71%) failed to reach the government white paper's modest target of 80%.²

Workplaces and restaurants are the public venues that expose the greatest number of people to environmental tobacco smoke. Because of their prolonged exposure,^{3,4} staff in bars and dining rooms are most at risk; the 50% increase in lung cancer risk among food service workers may be in part attributable to workplace exposure to tobacco smoke.⁵ Levels of environmental tobacco smoke in restaurants have been found to be double those in offices and 1.5 times higher than in homes with at least one smoker.⁵ Levels in bars can be more

than six times those in offices that allow smoking.⁵ In Australia, concern about litigation from staff is now driving workplaces to become smoke free faster than is nominal concern for workers' health and amenity (National Heart Foundation of Australia, personal communication).

While public preference for smoke free dining is growing, restaurateurs often grossly underestimate the potential demand. An Australian study surveyed restaurateurs and their patrons to determine whether restaurateurs' perceptions and policies matched customers' preferences.⁶ While less than 2% of restaurants were totally smoke free and 22% provided some smoke free areas, 47% of patrons dining in those restaurants stated that they believed restaurants should be totally smoke free, with a further 42% supporting smoke free areas. Only a third of restaurateurs who thought they should provide smoke free areas actually provided them.

In the report of the National Opinion Poll, 76% of restaurateurs believed that a policy to ban or restrict smoking would harm their business.¹ Californian laws require smoking areas to be separate and ventilated, and New York City bans smoking in restaurant dining areas with more than 35 seats. About 11 000 of some 15 000 restaurants are affected. Analysis of American restaurant sales tax data shows no evidence that