ing with two negative small British studies^{8,9} and a positive open, non-blind Norwegian trial, where a low saturated fat and high polyunsaturated fat diet was reported to reduce the incidence of recurrent myocardial infarction and coronary deaths in men under 60 with initially high concentrations of serum cholesterol (7.7 mmol/l; 296 mg/100 ml): a 14% difference between the diet and control groups was achieved over the five years of this study.¹⁰ Without clearer, more convincing evidence of benefit we are not justified in imposing on all patients dietary or drug regimens which many find unpleasant.

Though measurement of serum cholesterol concentrations gives little guidance to prognosis and there is little to be gained from intervention for the average survivor of infarction, detection of hypercholesterolaemia is important. Its recognition should lead physicians to search for families with polygenic, or even perhaps the rare monogenic, hypercholesterolaemia. Families of patients who have had a myocardial infarction are likely to be well motivated to permit screening of all first degree relatives in order to improve their prognosis, and those with severe hypercholesterolaemia are likely to accept vigorous control measures.¹¹ The most satisfactory way of doing this at present is to combine a diet with a high polyunsaturated to saturated fat ratio with 24-30 g cholestyramine daily.¹²

For these reasons it is helpful to be reminded that measurement of cholesterol within the first 24 hours after a myocardial infarction is a reliable way of assessing serum concentrations (p 1651). Contrary to a common belief, three months do not have to pass before an evaluation of plasma lipid values is possible.¹³ Serum concentrations of both cholesterol and low density lipoproteins decrease, however, during the second 24 hours and remain relatively low during the next two to three weeks. This is a message of practical value since blood is likely to be taken during the first 24 hours of myocardial infarction for various other biochemical measurements and less likely to be taken routinely three months after the heart attack. Estimation of serum cholesterol concentrations should be routine during the first 24 hours in all young and middle aged patients with myocardial infarction in order to detect and treat those and their close relatives with concentrations around and above 8.0 mmol/l (309 mg/100 ml).

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- 1 Frank CW, Weinblatt E, Shapiro S. Prognostic implications of serum cholesterol in coronary heart disease. In: Jones RJ, ed. *Atherosclerosis. Proceedings of the second international symposium*. Berlin: Springer Verlag, 1970:390-9.
- 2 Shanoff HM, Little JA, Csima A. Studies of male survivors of myocardial infarction: XII. Relation of serum lipids and lipoproteins to survival over a 10-year period. *Can Med Assoc J* 1970;103:927-31.
- 3 Proudfoot WL, Bruschke AVG, Sones FM. Natural history of obstructive coronary artery disease: ten-year study of 601 nonsurgical cases. *Prog Cardiovasc Dis* 1978;21:53-78.
- 4 Coronary Drug Project Research Group. Natural history of myocardial infarction in the Coronary Drug Project: long-term prognostic importance of serum lipid levels. *Am J Cardiol* 1978;41:489-98.
- 5 Coronary Drug Project Research Group. Clofibrate and niacin in coronary heart disease. *JAMA* 1975;231:360-81.
- 6 Dewar HA, Oliver MF. Secondary prevention trials using clofibrate: a joint commentary on the Newcastle and Scottish trials. *Br Med J* 1971;iv:784-6.
- 7 Rosenhamer G, Carlson LA. Effect of combined clofibrate-nicotinic acid treatment in ischaemic heart disease. *Atherosclerosis* 1980;37:129-38.
- 8 London Hospitals Research Committee. Low fat diet in myocardial infarction. A controlled clinical trial. *Lancet* 1965;ii:501-4.
- 9 Rose GA, Thomson WB, Williams RT. Corn oil in treatment of ischaemic heart disease. *Br Med J* 1965;ii:1531-3.
- 10 Leren P. The effect of plasma cholesterol lowering diet in male survivors of myocardial infarction. *Acta Med Scand* 1966; suppl 466.
- 11 Oliver MF. The case for intervention in those at high risk for coronary heart disease. *Advanced Medicine* 1984;20:114-24.
- 12 Oliver MF. Hypercholesterolaemia and coronary heart disease: an answer. *Br Med J* 1984;288:423-4.
- 13 Fyfe T, Baxter RH, Cochran KM, et al. Plasma lipid changes after myocardial infarction. *Lancet* 1971;ii:997-1001.

Aseptic rituals unmasked

Debunking venerable medical customs and procedures is a beguiling pastime which may have practical as well as recreational value. Among the most attractive targets are the surgical rituals introduced by Joseph Lister and his direct disciples. The recent advances in our knowledge of the microbiology of the skin, mucous membranes, and gut have stimulated reappraisal of the measures designed to protect the patient from infection. Traditionally these have included the surgical scrub, as well as shaving, disinfecting, draping, and subsequently dressing the operation site, and also preparing the bowel before surgery on the gut.¹

Probably the most visually impressive feature of aseptic ritual is the cocooning of staff in bizarre gowns, hats, and masks. At one extreme a complete physical barrier may be donned by the surgical team in the form of the body exhaust ventilated "space suit," which is an integral part of the Charnley-Howarth system of ultraclean air in operating rooms. This degree of protection of the patient from the staff's microflora may be justified only in high risk procedures such as replacement of the hip joint.² The more conventional types of operating costume form a negligible barrier to micro-organisms unless the normal woven fabrics are replaced by impermeable—and generally uncomfortable—materials.³

At first glance facemasks may seem a small and unimportant part of the protective uniform. They can, however, prevent the direct contamination of the operation field with respiratory tract droplets containing pathogenic bacteria. In obstetric practice this appears to be a less frequent source of infection than the patient's own microflora. In one recent study, for example, staff stopped wearing masks during vaginal delivery and the incidence of puerperal infection in 1750 consecutive cases (3.6%) was the same as in the previous 1750 cases when masks had been worn.⁴ The wearing of masks was continued for all caesarean sections, yet a quarter of the 192 patients developed puerperal infections, which were apparently endogenous in most cases.

In this study, fortunately, no infections occurred with Lancefield group A haemolytic streptococci, but nevertheless four of the five infections with "streptococcal species" (other than group A or B) were in patients attended by unmasked staff. For almost 200 years the principal danger facing women during labour has been known to be infection acquired from their attendants.⁵ The arch enemy, the haemolytic streptococcus, is harboured in the upper respiratory tract of about 1% of the general population⁶ and is transmitted to the birth canal infrequently but with potentially disastrous consequences.^{7,8}

This risk is far less than 50 years ago in the heyday of puerperal fever, when around one fifth of the population were carriers. The high incidence at that time helped to establish clearly the importance of droplet borne infection from staff, notably in a classic *BMJ* paper of 1935.⁹ This included impressive epidemiological data to show "that genital infection is not homo-exogenous but hetero-exogenous—that is, from the respiratory tract of obstetric attendants." The paper also described pioneer experiments on the protective value of masks in obstetrics and surgery. An efficient mask must be capable of arresting "low momentum droplets," which contaminate "the front of the operator's gown and his sterile gloves, transference to the patient occurring at the next manual interference," as well as the

evidently less important "high momentum droplets," which infect "by direct implantation on to the vaginal introitus."

A mask consisting of four layers of fine dental gauze or eight double layers of surgical gauze arrested both types of droplets, but half that thickness increased the amount of dangerous low momentum spray. The inclusion of a "snout" in the design and secure fastening below the chin reduced the risks of down flow and the escape of droplets at the sides of the mask. Paradoxically, saturation of the mask increased its efficiency but also increased the danger of indirect transfer from the sodden material.

During the past two decades the reusable natural fabric mask has been replaced in operating theatres by disposable synthetic types, usually incorporating an efficient fibreglass or plastic filter.^{10 11} Cheaper disposable paper masks are very satisfactory when used for short periods, however—for example, in the protective isolation of highly susceptible patients¹² and during procedures such as lumbar puncture, inserting intravenous lines, and dressing large wounds.¹¹ They should also prove adequate during routine vaginal delivery.

Nevertheless, if the obstetricians' luck holds, their face-masks will tend to go the way of the traditional preparatory enema¹³ and shaving the pubis.¹⁴ The clinical microbiologist must, however, view with serious misgivings this latest example of iconoclasm by his colleagues.

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- 1 Selwyn S. Skin preparation, the surgical 'scrub' and related rituals. In: Karran S, ed. *Controversies in surgical sepsis*. Eastbourne and New York: Praeger, 1980:23-32.
- 2 Lidwell OM, Lowbury EJJ, Whyte W, Blowers R, Stanley SJ, Lowe D. Effect of ultraclean air in operating rooms on deep sepsis in the joint after total hip or knee replacement; a randomised study. *Br Med J* 1982;285:10-4.
- 3 Mackintosh CA. A testing time for gowns? *J Hosp Infect* 1982;3:5-8.
- 4 Turner MJ, Crowley P, MacDonald D. The unmasking of delivery room routine. *Journal of Obstetrics and Gynaecology* 1984;4:188-90.
- 5 Gordon A. *A treatise on the epidemic puerperal fever of Aberdeen*. London: Robinson, 1795.
- 6 Hope-Simpson RE. Streptococcus pyogenes in the throat: a study in a small population, 1962-1975. *J Hyg (Camb)* 1981;87:109-29.
- 7 Anonymous. Puerperal streptococcal infection [Epidemiology]. *Br Med J* 1969;iii:788.
- 8 Eastmon CSF. What is the risk of beta-haemolytic streptococcal infection in obstetrics?: discussion paper. *J R Soc Med* 1984;77:302-8.
- 9 Paine CG. The aetiology of puerperal infection with special reference to droplet infection. *Br Med J* 1935;i:243-6.
- 10 Quesnel LB. The efficiency of surgical masks of varying design and composition. *Br J Surg* 1975;62:936-40.
- 11 Rogers KB. Face masks: which, when, where and why? *J Hosp Infect* 1981;2:1-4.
- 12 Selwyn S. Protective isolation: what are our priorities? *J Hosp Infect* 1980;1:5-9.
- 13 Romney ML, Gordon H. Is your enema really necessary? *Br Med J* 1981;282:1269-71.
- 14 Romney ML. Pre-delivery shaving: an unjustified assault. *Journal of Obstetrics and Gynaecology* 1980;1:33-5.

Slow release theophyllines and chronic bronchitis

Liquid concoctions containing methylxanthine have been used for asthma and bronchitis for over 100 years, and strong coffee has been shown to be an effective bronchodilator.¹ The systematic investigation of methylxanthines as bronchodilators began in the 1930s,² but they have become widely popular in Britain only in the past few years with the development of sustained release theophylline preparations.^{3 4} The benefits that come from theophylline have to be balanced against possible difficulties,^{5 6} especially in patients with asthma, and these hazards may be even greater in patients with chronic bronchitis with airflow obstruction. Theophylline has several drawbacks that result from its pharmacological features—not only its narrow therapeutic margin^{7 8} but its variable pharmacokinetics among in-

dividuals^{9 10} and the fact that saturable pathways may be affected by dietary methylxanthines.^{11 12} Furthermore, the clearance of theophylline may be affected by age, smoking, cor pulmonale, respiratory viral infections, impaired liver function, and erythromycin.¹³⁻¹⁸

These six factors are frequently present in patients with chronic obstructive bronchitis, for whom the oral methylxanthines appear to be widely prescribed. Their efficacy in such patients is controversial. Possible short term improvements have been reported as measured by spirometry,¹⁹ but these have not always been found in long term treatment.²⁰ In one study a small but statistically highly significant improvement in the 12 minute walking test²¹ was produced with a single dose of slow release aminophylline (450 mg Phyllocontin), but 4 mg salbutamol produced an identical response.²² The simultaneous administration of the two drugs produced a further spirometric improvement but did not affect the exercise tolerance. Surprisingly, no such improvement in exercise performance or indeed breathlessness was seen in patients with chronic obstructive bronchitis treated for a week with placebo or low dose or high dose oral theophylline, despite improvements on spirometric assessment.²³ In patients with severe airflow obstruction diaphragmatic fatigue may well be a factor in the development of respiratory failure, and theophylline may improve diaphragmatic contractility,²⁴ but the clinical importance of the effect of methylxanthines on diaphragmatic function is disputed.²⁵

The paper by D W V Evans (p 1649) may, therefore, be an important contribution. Evans investigated the response to dosages of slow release theophylline given as a single daily dose at 2200 hours of 20 patients with chronic obstructive bronchitis (14 were considered to have accompanying emphysema based on abnormally low diffusion coefficients; KCO <68% predicted). Dosages from 200 to 800 mg a day were given, though in four patients the highest dose was omitted because lower doses (given acutely) were intolerable. Twelve hours after the dose of oral theophylline (Uniphyllin) or placebo was given measurements were made of the peak expiratory flow rate, the forced expiratory volume in one second, the forced vital capacity, the heart rate, and the plasma concentration of theophylline. Patients also carried out a six minute corridor walk.²⁶ Their breathlessness was assessed by a visual analogue scale and spirometry, and the walking test was then repeated one hour later. Theophylline produced no improvement in the formal respiratory function tests—but this was not necessarily to be expected in Evans's patients, for patients who responded to an unspecified dose of an inhaled β_2 selective agent had been excluded from the study. Patients with severe airflow obstruction frequently need high doses of inhaled β_2 stimulants to achieve any substantial bronchodilatation,²⁷ and the forced or residual vital capacities may be more appropriate measurements for assessing response to bronchodilators. Furthermore, unless the patient shows a bronchodilator response to salbutamol alone theophylline will rarely produce a response, but if he does respond to salbutamol there may be an additional bronchodilator effect of theophylline.²⁸ Evans showed a correlation between the dose of theophylline and its plasma concentrations. He found no correlation between dosage and improvement in walking distance, and no subjective reduction in the degree of breathlessness assessed by visual analogue score. The higher drug dosages achieved plasma concentrations in the higher therapeutic range. Treatment with placebo achieved an improvement (7%; 21 metres) in walking distance, and the study design suggested that this was not a