induction to ensure that labour and delivery of women with risk factors take place in optimal conditions. When there is some anxiety about the fetus—for example, because of suspected placental insufficiency which may require emergency delivery by caesarean section if fetal distress develops—it is logical to plan that the labour should occur on a weekday during daylight hours, when all obstetric services are readily available to deal with any emergency that might arise. Whether we like it or not obstetric practice has had to accept some limitation of the 24-hour service that obstetrics requires. Statutory regulations ensure that midwives, laboratory technicians, and now junior hospital doctors have adequate off duty. This affects staffing on the labour ward and the ready availability of such vital services as anaesthesia and haematology.

A liberal policy of induction can be advocated only if it is safe for both mother and baby and is acceptable to the mother. In general, as a study from Glasgow showed,[6] induction as practised in most centres fulfils these criteria. Nevertheless, the modern tools of induction, amniotomy and intravenous oxytocin, may be dangerous if misused. If oxytocin is given without careful monitoring of uterine action it will inevitably produce hyperstimulation in some patients, resulting in fetal distress[7] and even uterine rupture. Failure to recognise the importance of a ripe cervix as an index of uterine sensitivity[7-9] may lead to failed induction, intrauterine infection, and a greater risk of oxytocin-induced uterine hyperstimulation. A ripe cervix is possibly the greatest safeguard for mother and fetus when a decision is made to induce labour, and the obstetrician who disregards it when deciding whether to induce labour does so at his peril.

Finally, is an induced labour as satisfying to the pregnant woman as a spontaneous one? Undoubtedly some women feel that they have missed out if they have a labour which is not natural because induction, epidural anaesthesia, or forceps delivery has interfered with an event they wished to be in full control of. Further inquiry[10] suggests that such resentment stems not from the procedure but from the fact that an adequate explanation was not given to them about why it was necessary for the obstetrician to intervene. Once convinced that it was for the benefit of the baby or themselves they are usually satisfied. The weight of available evidence suggests that induction is not generally abused. Undoubtedly a few centres have tended to put too much emphasis on the risk of uncomplicated postmaturity as an indication for induction. This has led to a higher incidence of induction than the 25-30% found in most hospitals. If there is a lesson that comes out of the induction debate, it is that we as doctors must, whenever possible, ensure that our patients are made to feel that they have taken part in decisions that affect their lives.

[1] *Hansard*, House of Commons, 10 December 1975 (Written answers), cols 226-8.
[2] Baird, D, Walker, J, and Thomson, A M, *Journal of Obstetrics and Gynaecology of the British Empire*, 1954, **61**, 433.
[3] Turnbull, E P N, and Baird, D, *British Medical Journal*, 1957, **2**, 1021.
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[5] Department of Health and Social Security. *Report on Induction of Labour*. London, HMSO, 1975.
[6] Cole, R A, Howie, P W, and MacNaughton, M C, *Lancet*, 1975, **1**, 767.
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[9] Steer, P J, *et al*, *British Journal of Obstetrics and Gynaecology*, 1975, **82**, 433.
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Pandemic and interpandemic influenza A

The last major influenza pandemic, in 1968-70, was caused by the A/Hong Kong/68 virus; doctors who were in general practice in Britain in the winter of 1969-70 will clearly recall its impact. The pandemic before that, the Asian 'flu caused by the A/Singapore/57 virus, began in 1957 and had an even greater worldwide effect. This periodicity in the emergence of new pandemic strains of influenza A prompts the fear that a fresh one may be due.

The inter-pandemic influenza A outbreaks that have occurred since 1971-2 have been caused by a series of viruses related to A/Hong Kong, a relationship dependent on antigenic cross-reactivity between the haemagglutinin and neuraminidase antigens on the surface of the virus. In Britain the strains mainly responsible were A/England/42/72 in the 1972-3 winter; A/Port Chalmers/1/73 in 1973-4; A/Port Chalmers and A/Scotland/840/74 in 1974-5; and this winter A/Victoria/3/75. The nomenclature of these viruses[1] may be confusing. The geographical name refers to the place of origin of the first-identified isolate of the new strain, the number that follows is the sequential laboratory number allocated to the virus, and the last number is the year of its isolation. Furthermore, all these strains are designated H3 N2—their haemagglutinins are related antigenically to that of the A/Hong Kong (H3 N2) virus, and their neuraminidase antigens to those of A/Hong Kong and the A/Asian (H2 N2) virus of 1957. This means that the different strains are characterised by definite but incomplete changes ("drifts") in their surface antigens. Immunity to influenza is directed against these antigens, particularly the haemagglutinin; and it is antigenic drift that helps a virus cause outbreaks in populations that have become immune to its precursors.[2 3] Such outbreaks are unlikely to be severe, because population immunity to the preceding related viruses is good enough to damp down the effects of each new strain. Nevertheless, these variants of the Hong Kong virus have caused influenza throughout most of the world. A fresh pandemic is believed to require the appearance of a virulent strain that has a new haemagglutinin, one unrelated to the H3 antigen or to the previous H2 or H1 antigens; it is this event, due to a so-called "shift" in the haemagglutinin antigen, that is mostly to be feared. If the neuraminidase antigen should also undergo a complete antigenic shift, with the emergence of a virulent H4 N3 virus, the pandemic could be specially severe. The measures that should be taken to minimise the impact of such a virus were considered recently by an international study group which met in January 1976 in Rougemont, Switzerland. Their deliberations have been given added point by recent events in Fort Dix, USA.[4]

In an influenza outbreak among armed forces recruits at Fort Dix, New Jersey, in late January 1976 the virus isolated from four young men proved to have surface antigens resembling those of the swine influenza virus (see also report in our Epidemiology columns). One of the recruits died, and the post-mortem appearances were compatible with death from influenzal pneumonia. Serological studies indicated that other recruits had also been infected. The swine influenza virus is believed to be derived from—possibly identical with—that responsible for the most serious influenza pandemic on record, the Spanish influenza of 1918-19. The virus stopped circulating in man in the early 1930s but has remained endemic in the pig population in the USA ever since. It has been identified in a few cases of human influenza in the intervening years, usually

where there has been close contact with pigs, but this is the first report of its case-to-case transmission. If this virus should spread, and if it retains or acquires the virulence for man of the 1918 virus, then the world could be faced with a similar pandemic. The 1918-19 influenza pandemic not only caused many deaths among the elderly but also an unusually high mortality in young adults.[5] At the present time many people over about 50 years of age possess serum antibodies against the swine virus; a swine influenza pandemic now might therefore spare older people, but younger adults and children would be expected to possess no basal immunity.

The epidemiological features of the swine influenza virus are under close scrutiny by the Centre for Disease Control in Atlanta, USA, and by the World Health Organisation. Plans are in hand to start large-scale production of killed vaccine if evidence of significant spread appears. Careful observation is being kept in Britain, where the Department of Health, the Medical Research Council, and the Public Health Laboratory Service, together with vaccine manufacturers and field workers, are also putting preliminary plans into operation. Small amounts of killed vaccine are being made for clinical trials—in order, for example, to determine the dose that will ensure an adequate serological response, particularly in young adults and children who might respond poorly to a completely new influenza vaccine; they may require two injections. Live, attenuated vaccines will also be prepared, though they may well not be available soon enough to combat the first effects of a pandemic because their development and the essential potency and safety testing takes time. Nevertheless, their study is important; many workers believe that the eventual control of pandemic influenza is most likely to be achieved by means of live vaccines which can be given easily and economically by the nasal route.

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[2] Pereira, M S, *et al*, *Journal of Hygiene*, 1969, **67**, 551.
[3] Kilbourne, E D, *Journal of Infectious Diseases*, 1973, **127**, 478.
[4] US Department of Health, Education and Welfare, *Morbidity and Mortality Weekly Report*, 1976, **25**, 47.
[5] Ministry of Health, *Reports on Public Health and Medical Subjects No 4*. London, HMSO, 1920.

Oxygen in myocardial infarction

Patients with acute myocardial infarction commonly have hypoxaemia, and this tends to be more pronounced when left ventricular failure or shock complicates the infarct.[1-3] Severe hypoxaemia is associated with a higher incidence of cardiac arrhythmias and a higher mortality. Several mechanisms have been proposed to explain the hypoxaemia; imbalance of ventilation-perfusion, collapse of small airways with physiological right-to-left shunting, and pulmonary oedema. Oxygen treatment restores the arterial oxygen tension to normal more readily in uncomplicated cases than in those in which there is left ventricular failure or shock, and it usually results in increased total peripheral resistance, a rise in the arterial pressure, and a reduction in cardiac output.[3 4]

Oxygen is given routinely to many patients with myocardial infarction, but there is still no evidence based on controlled clinical trials about its effect on either morbidity or mortality. It seems reasonable to correct hypoxaemia and even to achieve higher-than-normal arterial oxygen tensions in the belief that more oxygen would then be available to the myocardium. This might be expected to reduce the incidence of arrhythmias and the size of the infarct, improve myocardial performance, and reverse metabolic acidosis by improving aerobic respiration in other organs such as skeletal muscle.[4 5] Whether or not these results do occur, however, remains controversial.

Based on work in dogs, Maroko and co-workers[6] have recently suggested that 40% inspired oxygen might benefit patients with uncomplicated myocardial infarction. They found a significant reduction in the extent of acute ischaemic injury and in the size of myocardial infarcts produced by occlusion of the left anterior descending artery in dogs breathing 40% oxygen (resulting in a Pao_2 of 25 kPa (188 mm Hg)) when compared with dogs breathing 20% oxygen (Pao_2 13 kPa (98 mm Hg)); the inhalation of 100% oxygen offered no added advantage. The same group has also shown that hypoxaemia (Pao_2 <6kPa (<45 mm Hg)) substantially increased myocardial damage,[7] though Madias *et al*[8] found that hypoxaemia did not further depress ventricular function after myocardial infarction. The results of these studies[6 7] are encouraging, but relatively few animals were used and several questions are left unanswered. The oxygen inhalation was given for only 24 hours, and its protective effect over a longer period would be of interest. Since most patients with acute myocardial infarction do not come under medical care immediately after the infarct it is also important to know whether the protective effect of 40% oxygen inhalation is influenced by a delay of several hours.

Controlled clinical trials in man seem warranted before such treatment could be recommended with confidence. The information we have at present about the value of oxygen inhalation is conflicting. Some investigators state that inhalation of 100% oxygen does not augment the availability of oxygen to the myocardium in patients with coronary artery disease (nor in patients with rheumatic or congenital heart disease) and may in patients with triple coronary artery disease[9] even reduce coronary blood flow sufficiently to increase myocardial ischaemia. On the other hand, Horvat *et al*[10] showed an increase in the threshold of pacing-induced angina by oxygen inhalation, and Ganz *et al*[11] found a beneficial effect of high oxygen concentration (Pao_2 >53 kPa (>398 mm Hg)) in coronary disease.

Hyperbaric oxygen is probably of marginal, if any, value in patients with acute myocardial infarction.[12 13] Thurston and his co-workers[13] did appear to show some reduction in mortality in patients treated with hyperbaric oxygen compared with those given oxygen via an MC mask, but the evidence available suggests that, even if it were feasible, this form of treatment cannot be recommended for wide use.

Mild hypoxaemia can be corrected easily by using nasal catheters or cannulae at a flow rate of 1-2 l/min or a Ventimask delivering 24-28% oxygen at 4 l/min.[14 15] These devices are particularly helpful for patients with chronic respiratory disease, in whom hyperoxaemia should be avoided.[16] For more profound hypoxaemia the MC mask will deliver about 60% oxygen and achieve a Pao_2 of $25 \pm 5{\cdot}5$ kPa (188 ± 41 mm Hg).[17] Delivery of 40% oxygen can be achieved by a 40% Venturi mask, an MC or Edinburgh mask at 4-6 l/min, or nasal cannulae at 4 l/min.[1 15] Whichever device is used arterial oxygen tension should be measured in the more severely ill patients, and administration should be continued for three days or longer if the patient's condition warrants it.[1 4]

The uncertainty about the value of oxygen therapy is shown clearly enough by the range of practices in different coronary care units, most of which report similar mortality rates. Except when carbon dioxide retention is present, the most