relative bradycardia. Classically the patient has a dry, unproductive cough, though rarely he may produce mucopurulent, rust coloured sputum. Crepitations are often heard but (as in mycoplasmal pneumonia) the radiological changes usually far exceed the physical signs. Patchy consolidation may persist on the chest *x* ray film for several weeks after the resolution of symptoms.[3] Extrapulmonary manifestations include vasculitic skin lesions resembling rose spots and epistaxis. Myocarditis[4] and pericarditis[5] may occur; endocarditis[6,7] is rare but may be fatal.[7] Some patients are confused or semicomatose, and meningoencephalitis has been described.[8] Treatment is with tetracycline, for 21 days in severe cases; children should be given erythromycin. The endocarditis has been successfully treated with rifampicin.[6]

The diagnosis of psittacosis is almost exclusively serological. *C psittaci* grows readily on cell monolayers, but it is a grade B pathogen, and deaths have occurred in laboratory workers.[9] The widely used complement fixation test does not distinguish between *C psittaci* and *C trachomatis*, but the clinical syndromes are dissimilar enough for this to be a minor problem. Patients may show a simultaneous rise in antibody titre to a variety of organisms, which suggests that raised antibody titres to *C psittaci* may sometimes be an anamnestic response.[10] Newer methods which distinguish *C psittaci* and *C trachomatis* antibodies such as enzyme linked immunosorbent assay and monoclonal antibody techniques are being evaluated.[11]

The earliest descriptions of epidemics of psittacosis in late nineteenth century Europe incriminated imported Argentinian parrots as the vectors.[9] Large numbers of caged birds, domestic poultry, and wild birds are susceptible to the infection, which they may occasionally transmit to man.[9] The name ornithosis is therefore sometimes used synonymously with psittacosis. Poultry workers[12] and veterinarians[13] may be infected, and handling feathers is a risk factor.[13] Budgerigars have been responsible for about half the sporadic cases in Britain in which a vector can be identified.[14]

The incidence of psittacosis in Britain is increasing. Over 300 cases have been reported yearly since 1980, and this is probably only partly owing to increased surveillance.[2,10] In one rural Cambridgeshire practice with under 5000 patients 18 symptomatic cases were recognised in 8·5 years, an annual incidence of about one in 2000.[10] In only 10-29% of cases reported in Britain and 17% of Cambridgeshire cases[10] was there a history of contact with exotic, domestic, or wild birds. The lack of seasonal variation suggests that the condition is endemic.[2,10]

Is it possible, then, that psittacosis has a human reservoir? Man to man transmission, presumably by droplet spread, is considered rare but may be dramatic. In 1939 one patient infected 25 contacts, with 13 deaths,[15] and recently a patient in hospital who died from psittacosis infected 11 people including a relative, medical and nursing staff, a cleaner, and a patient in the same room.[16] Carriage of *C psittaci* may continue in the sputum for as long as eight years.[17]

As diagnostic tests improve the epidemiology of psittacosis may become clearer; making psittacosis a notifiable disease, as suggested previously, might expedite matters.[18]

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Disaster at the dining table

To die from a cardiac arrest resulting from a myocardial infarction is an unfortunate tragedy, but to die from a piece of food having "gone down the wrong way" seems a calamity of catastrophic proportion.

Not surprisingly, then, whenever I talk about cardiopulmonary resuscitation, whether the audience is medical or lay, I am invariably persuaded to spend a lot of time on the resuscitative manoeuvres described by Heimlich.[1] We need to remember, however, that many cases of sudden choking occurring during meals are due to cardiac ischaemia simulating food impaction.[2] The victim often clasps his neck or upper chest because of the feeling of pain, panic, and constriction in that area; onlookers may suspect food impaction because of their own experiences of mild choking on food. There are other causes of "collapse" while eating, but when choking seems to be the main presenting symptom the most useful differential diagnosis is between myocardial ischaemia and food impaction.

Food impaction is likely when the victim has been eating with dentures (which disguise the tactile perception of food quality), is edentulous and eating without dentures, is a talkative eater, a food gobbler, or an excessive drinker. True chokers do not lose consciousness for some time but are unable to speak—respiratory silence is more sinister than noisy respiration. Vigorous inspiratory attempts by the victim seem only to increase the impaction.

Typically, the victim follows one of two courses of action. Most commonly he seeks early refuge in the nearest lavatory or kitchen and leaves the dining table without a word—often remarkably unnoticed by most other diners. By this decision he either survives or dies with relative dignity, unless he is pursued into his sanctuary by a wary fellow diner. Perhaps the more extravert will choose to stay put and seek help. Some may have learnt the sign of food impaction, which is placing the skin web between the extended forefinger and thumb of the hand against the larynx. The subject's appraisal of his own catastrophe may, of course, not be accurate—but certainly the rescuer is more justified in concentrating on food impaction in the first instance.

If the presumptive diagnosis is food impaction, the rescuer must be assertive and encourage coughing or vomiting. While the victim remains conscious he should be urged to explore his throat with his own fingers while leaning forward in a sitting position with the rescuer thumping his back. Back thumping cannot be expected to disimpact food or create an expiration, but it seems to stimulate coughing and is a time honoured and expected

therapy. The rescuer should not explore the victim's pharynx with fingers or instruments because this can be fraught with anatomical deception. A small child may be held upside down and gently back slapped.

If these measures bring no relief the victim will be in panic if still conscious and the rescuer will be in a state of high alert. Nevertheless, thought and observation are the priorities. Is this food impaction or something else? If the victim is becoming cyanosed this means that his circulation is brisk but hypoxic, indicating acute respiratory obstruction. If he is pallid this indicates circulatory failure. Frank cyanosis warrants further attempts at food disimpaction, while pallor suggests that either the cause of the catastrophe is a myocardial ischaemia or that the original choke is passing into hypoxic ventricular fibrillation. A pale victim should be placed on the floor.

The Heimlich manoeuvre is usually demonstrated in the standing position, the rescuer standing behind the victim. The victim's knees may, however, buckle beneath him and he may fall through the arms of the rescuer. For this reason the sitting manoeuvre is more practical, when the rescuer leans over the back of the chair. In either case the rescuer clenches one wrist with the other hand in the victim's epigastrium and thrusts sharply inwards and upwards several times if necessary in the hope of blowing out the impaction. The epigastric thrust may also invoke a regurgitation from the stomach up the oesophagus which may assist in disimpaction. On the other hand, a regurgitation may compound the food problem—and could create an aspiration hazard in someone with a myocardial infarction.

Whatever the original cause of the disaster, unless he has revived, the victim will now be passing into unconsciousness and a cardiopulmonary arrest is imminent or in progress. In either case the victim should be placed supine on the floor. The head should be firmly hyperextended and vigorous mouth to mouth ventilation attempted while lifting up the jaw. If ventilation is not achieved this is further evidence of food impaction and an enthusiastic probing by the rescuer's fingers is now justified in an attempt to identify and remove the object. If ventilation is achieved, either a food impaction has resolved or it was never present. In either case, standard cardiopulmonary resuscitation should be instituted if ventilation alone does not resolve the problem.

Failure to ventilate supposes either inadequate manipulation of the head and chin or continued obstruction of the airway from food impaction. As the victim is now lying on a hard floor surface in the supine position I recommend closed cardiac compressions, say 15 compressions, followed by a further digital exploration of mouth and pharynx. As well as creating blood circulation the chest compressions act like a Heimlich manoeuvre. If a further attempt at mouth to mouth ventilation proves unsuccessful the victim's airway must now be entered as a matter of life or death: circulatory arrest is likely so that there is nothing to lose.

The operation required is cricothyrotomy (laryngotomy), not tracheotomy. Maintain the hyperextended head position and place a cushion under the shoulders. The blade of the sharpest available knife is scraped horizontally down the midline of the thyroid cartilage from the laryngeal eminence (Adam's apple) down into the groove between it and the cricoid cartilage. (Practise this with the blunt edge on yourself.) This manoeuvre is, to my mind, the easiest way of finding the cricothyroid groove. An alternative is to use a fingernail instead of a knife.[3] Keeping the head extended and the skin taut, saw briskly back and forth over the midline so as to limit the incision to below 2 cm if possible. (If you doubt the sense of vigour that I suggest, try incising the skin of an uncooked chicken with a table knife.) If the cricothyroid membrane has not been cut with the skin incision, it may now be entered by a vertical thrust (vertical to the floor but in the horizontal plane of the neck) with a sharp pointed knife. Either way, an entry into the cricothyroid area should be made with the knife which is then rotated through a right angle in order to open up the space.

Little or no bleeding indicates circulatory arrest and facilitates mouth to neck (laryngostomy) ventilation after removal of the knife. Closed cardiac compression will be needed to complement the mouth to neck ventilation. With the return of spontaneous circulation and respiration some means will be needed to maintain the patency of the laryngostomy—a knife, drinking straws, or the barrel of a retractable ballpoint pen. To minimise bleeding into the trachea, the victim should be placed in the lateral recovery position on a bed or settee tilted to a 30° head down inclination. Close supervision will be needed until the patient reaches hospital. An ambulance will provide oxygen, which may be offered by a conventional facemask to the laryngostomy, but its continued patency is of paramount importance.

At hospital the obstruction can be removed by direct laryngoscopy and, in most cases, endotracheal intubation can be performed and the laryngeal opening closed.

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Prophylaxis against unipolar depression

Lithium has an established place in the prophylactic treatment of bipolar affective disorders but not in the treatment of unipolar disorders. Its efficacy has recently been examined in a Medical Research Council multicentre trial.[1]

Unipolar depression is a non-specific term with no agreement about the types of affective disorder that it includes. Sometimes it is used to mean any depression—including minor or neurotic depressions—occurring in patients who do not have a bipolar disorder. On other occasions it is used to mean only serious or psychotic depressions, and on still others to mean only recurrent depression. Recently Boyd and Weissman have proposed that the term non-bipolar depression would overcome this confusion.[2] In America that would correspond to the diagnosis of major depressive disorder as categorised by the American Psychiatric Association's diagnostic system DSM III.

Such depressions are common. Weissman and Myers have estimated that the point prevalence for non-bipolar depression is 4·3% (3·38% in men, 5·2% in women) with a life time expectation of illness of around 20%.[3] Figures for prevalence vary considerably from study to study, however, and it is extremely difficult to determine how much this is due to differences in study design or definitions and how much to true differences in prevalence.

One consistent finding is that recent studies have tended to show higher prevalence figures—indicating that affective