In our own day the large lead industries are much better controlled, and (in the developed countries at least) the emphasis is on the prevention of subclinical rather than clinical effects. The risk of excessive lead absorption is still high, however, in some occupations. Anyone who takes a high temperature flame to old painted metal may inhale a sufficiently great amount of lead fumes to poison himself, particularly if the work is carried out within a confined space. Such a man would not normally describe himself as a lead worker, so the physician needs to be especially careful when taking the occupational history in order to obtain precise details of the type of work undertaken.

Inhalation risks, however, are not the only ones to be considered in occupational lead poisoning. The risk from ingesting lead at work has frequently been played down, but in some circumstances it can be very important indeed. For example, Baxter and his colleagues found raised blood lead concentrations among a group of workers in a stained glass factory whose methods of work had scarcely changed since the middle ages (p 383). As the glass is fitted into the lead bars the hands inevitably become contaminated, and, unless great care is taken, some of this lead is transferred to the mouth. Where the risk to workers is from ingestion control strategies must be based on blood lead analyses; the concentrations of lead in the air are most unlikely to be raised and environmental measurements may thus induce an unwarranted sense of security.

So far as the general public is concerned the risk of contracting lead poisoning is almost entirely confined to ingestion. The ways in which our daily lead may be increased are many and various but new risks—or old risks in new disguises—continue to appear to perplex the physician.

A 19 year old girl who lived in an Arab village on the West Bank was admitted to hospital in Jerusalem with lead poisoning.[1] The most striking clinical feature was her severe muscular weakness, particularly of the shoulder girdle, which improved only slowly after chelation treatment. The patient's mother was also found to have severe lead poisoning, and an investigation of some other members of the village found nine other persons, including a 2 week old baby who needed treatment in hospital and many others with raised blood lead concentrations who were treated at home. Subsequently, the search for patients with raised blood lead concentrations was widened and other affected villages were found.[2] The cases of clinical poisoning clustered in household units but were unrelated to age or sex, and a common dietary item appeared to be the source of the lead. The water, olive oil, salt, sugar, and a wide variety of spices and condiments were analysed and found to be blameless. When locally ground flour from houses of affected families was analysed, however, it was found to contain lead in concentrations from 54 to 532 parts per million; the lead concentrations in locally ground flour from unaffected families was less than 10 parts per million.

The investigators next focused their attention on the flour mills. Freshly ground flour was found to be heavily contaminated although the grain was lead free. When caked flour was removed from grooves in the lower mill stone from one of the mills its lead content was found to be 940 parts per million; that in flour taken from grooves in the upper stone was 93 parts per million. When the stones were cleaned the lead content of the flour was considerably reduced.

The ultimate source of the lead was shown as soon as the driveshaft was examined. Both it and its housing were made of iron. As it had worn the housing had become loose; so to secure it again to the stone lead had been poured into the gap between the mill stone and the housing. The researchers then examined other West Bank mills and found that nearly a quarter produced flour with lead concentrations above 0·5 part per million, thus posing a risk to health on a large scale.

These important studies remind us that lead in the environment may be a serious threat to health if it finds its way into the diet—and that chasing the source may be slow and painstaking and require a great deal of detective work. Shades of the Devonshire colic!

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Gastric microflora

The advent of endoscopy and the use of antiulcer drugs have increased interest in normal gastric physiology and the microflora of the stomach.[1,2] In health, fluid aspirated from the stomach has a pH of less than 2 and is usually sterile, except during meals, which introduce organisms from the mouth and food. The make up of this transient flora depends on the number of bacteria ingested, the food in which they are ingested, the gastric acidity, and the rate of gastric emptying—and the stomach's defence against bacterial invasion, consisting of organic acids, mucus, lysozyme, and immunoglobulins (mainly IgA).[3,4]

The bactericidal nature of gastric secretions is affected by alterations in pH. In hypochlorhydria the number of bacteria found in the stomach increases and may include faecal type bacteria as well as oral flora.[1,5] Such conditions occur in pernicious anaemia and after partial gastrectomy and vagotomy and pyloroplasty. Patients with hypochlorhydria are more susceptible to enteric infections such as cholera and salmonellosis since a lower infective dose will breach the weakened gastric bactericidal barrier.[6]

The gastric pH becomes less acid with age, increasing the quantity of bacteria,[7] and a syndrome associated with bacterial overgrowth has been recognised as a cause of malabsorption.[8] A similar picture has been described in association with the chronic diarrhoea of malnutrition in Third World countries.[9]

The type and numbers of organisms in the stomach are of importance in gastric surgery. Postoperative infections are much more frequent in patients with hypochlorhydria. Possibly a gastric aspirate could be Gram stained or cultured preoperatively to help in the choice of prophylaxis,[10] but this is rarely practicable. A gastric aspirate with a pH of greater than 4 would suggest bacterial overgrowth and the need for prophylactic antibiotics. If the patient is taking an acid reducing drug this should be stopped 24 hours before surgery.[11] Patients in intensive care units develop Gram negative pneumonias more frequently, possibly owing to organisms from the stomach entering the respiratory tract,[12] and the use of H_2 blockers may have increased this problem.

Endoscopy has allowed more detailed investigation of the gastric microflora for the presence of bacteria, viruses, fungi, and parasites. The gastric parasites *Anisakis*[13] and hookworm[14]

have been removed at gastroscopy. Cytomegalovirus and herpes simplex virus inclusions have been found in gastric biopsy specimens—usually in immunosuppressed patients as part of a local or generalised infection causing gastric erosions and ulceration.[15 16] Gastric candidal infection has been found in 15% of patients undergoing routine endoscopy of the upper gastrointestinal tract.[17] It produces no characteristic lesions and is not certainly pathogenic, although it occurs secondary to mucosal damage.

Since the turn of the century spiral bacteria have been seen in the stomach by many workers.[18 19] They lie on the gastric mucosa and in the gastric crypts but are protected from gastric acid by the mucus layer.[20-22] These fastidious organisms (now called *Campylobacter pyloridis*) have defied culture until recently when prolonged microaerobic incubation and the use of selective media have proved successful,[23] and many centres have repeated this success.[24-27] These organisms are found in most patients with gastritis, and several workers have shown a strong correlation with peptic ulceration. They elicit a polymorph response in the mucosa[28] as well as provoking a serological response[25 29]—and a complement fixation test may be used to predict the presence of gastritis. The campylobacters are not found in normal stomachs,[20 24] suggesting that they are not merely part of the normal flora. Whether they play a part in the aetiology of gastritis and peptic ulceration or occur secondary to breakdown in the mucosal barrier remains uncertain. No animal model has been found, though several workers are undertaking longitudinal studies to investigate the serological response and mucosal changes with appropriate antimicrobial treatment. Should these organisms prove important in the aetiology of gastritis and duodenal ulceration we may have been approaching their treatment incorrectly. Antacids and histamine H_2 receptor blockers decrease gastric acid secretion, permitting colonisation of the stomach by other bacteria,[30] and may allow *C pyloridis* to multiply. Antiulcer agents such as De-Nol (tri-potassium di-citrato bismuthate) act as mucosal protection agents; but we have recently shown that these also inhibit *C pyloridis*, which may explain their success in the treatment of peptic ulceration.

Prolonged use of drugs which increase gastric pH has been thought to increase the long term risk of gastric cancer.[31] Among the increased numbers of gastric bacteria found in these patients are some capable of reducing salivary and dietary nitrate to nitrite.[30] Subsequently the nitrite may react with constituents of normal gastric juices to form carcinogenic nitroso compounds. Nevertheless, this nitrosation requires the presence of hydrogen ions—that is, gastric acid—so achlorhydria may make their formation less likely.[31] Some studies have shown increased concentrations of nitroso compounds in patients taking cimetidine[30 32]; this is probably because the secretion of diluting gastric juice is inhibited, causing an increased concentration of all gastric juice constituents (including nitroso compounds). Several other studies have failed to confirm this proposed carcinogenic mechanism.[33 34] The postmarketing surveillance programme with cimetidine has failed to substantiate any increased carcinogenic risk of long term treatment.[35]

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Correction

Blood transfusion and surgery

In the fourth paragraph of the leading article "Blood transfusion and surgery" (27 July, p 234) there was an error in the final sentence. This should have read: "Concentrations below 10 g/dl are accompanied by a progressive increase in the bleeding time,[7] and below 9 g/dl tissue oxygenation can be maintained only by a compensatory increase in cardiac output." We apologise for this error.