

'my doctor' in privacy and with adequate space and time; the patient will have more help from other professionals and self-help and patient groups, as will the doctor from in-practice discussion and group learning and support systems of different kinds - we do not all have to be Balint-boys, even if some of us have found that kind of group enhancing and invigorating. For the foreseeable future, we shall go on needing our patients, for income if nothing else, and they will need us, even if not exclusively.

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trachea or bronchi. Distraction during eating by talking, laughing or playing increases the risk of inhalation¹. Sixty-five percent of childhood deaths from choking on food occur in boys^{1,4}. Food associated with food choking is usually either round and smooth, or pliable and non-friable^{1,2}. The former tend to slip easily into an airway and obstruct it completely, and the latter to resist expulsion.

Avoidance of certain foods might reduce choking deaths. Peanuts need to be chewed with a grinding motion, which is not well-developed until at least 4 years of age. Those foods specifically marketed for children should be appropriately shaped. For instance, lollipops and sweets should be flat rather than spherical. Death from choking may also be prevented by efficient first aid. There is, however, disagreement about whether the Heimlich manoeuvre or a combination of back blows followed by chest thrusts are more effective^{5,6}. Available experimental evidence does not relate to children⁷.

In England and Wales there has been a recent sharp decline in the number of childhood deaths reported to be due to choking on food. The number of deaths from this cause has fallen from 144 in 1974 to 46 in 1984⁸ (Table 1). The numbers were fairly static until 1979, since when there has been a marked decline. Further analysis shows this fall to be restricted to infants under one year old. The fall in deaths has been most marked in those under 3 months of age, with a smaller decline in those aged 3-5 months. This is not merely a reflection of the decline in birth rate, as the infant mortality rate attributable to choking has fallen in this time from 0.23 deaths per 1000 live births to 0.05 deaths per 1000 live births in boys, and from 0.16 deaths per 1000 live births to 0.05 deaths per 1000 live births in girls. We think this is unlikely to be due to changing patterns of reporting, for example an increase in the diagnosis of the sudden infant death syndrome (SIDS). The pattern of change of SIDS deaths has been different, reaching a peak in 1982 and declining slightly in 1983 and 1984. Furthermore, the causes of death that tend to be mentioned with 'cot death' are particularly respiratory tract infections^{9,10}.

Why should there have been such a marked decline in the number of young babies choking to death on food? A change in infant feeding practice may be the

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Decline in deaths from choking on food in infancy: an association with change in feeding practice?

Most children who choke to death do so on food rather than other objects. More than 60% of childhood deaths due to choking occur in the first year of life¹, and choking is the leading cause of accidental death in infants in the USA². Some risk factors have been identified. Infants are particularly at risk because they have ineffective chewing and swallowing mechanisms and are edentulous. Both are factors which are known to increase the risk of choking at all ages^{1,3}. Furthermore, in infancy the larynx is not only the narrowest part of the upper airway, but it is also relatively smaller than in older children and adults⁴, and this increases the risk of occlusion by a foreign body. Thus, in children less than one year of age, most inhaled foreign bodies are found in the larynx⁴, whereas in older children more lodge in the

Table 1. Total deaths from 'inhalation and ingestion of food causing obstruction of respiratory tract or suffocation' ▲ in children under 15 years of age, England and Wales, 1974-1984

	0-2 months	3-5 months	6-11 months	12-23 months	24-59 months	5-9 years	10-14 years	Total
1974	61	43	22	13	5	●	●	144■
1975	42	34	17	16	8	5	3	125
1976	38	41	18	13	7	2	7	126
1977	41	35	11	14	6	2	8	117
1978	38	35	17	15	4	1	4	114
1979	59	32	19	6	10	5	7	138
1980	37	28	9	9	10	1	1	95
1981	27	23	12	6	7	2	9	86
1982	13	16	12	10	5	3	7	66
1983	11	14	5	6	5	3	9	53
1984	10	14	6	9	2	2	3	46

● Figures not available.

■ Those less than 5 years only

▲ Code E911 in International Classification of Disease

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explanation. In the early 1970s there was concern that babies were receiving solid food far too early, and it was recommended that the early introduction of solid food should be avoided. In 1975, 85% of infants received solids before 3 months of age and 45% before 2 months¹¹. By 1980, although there had been some improvement, 55% of infants still received solids before 3 months¹². Concern was reiterated¹³, with the recommendation that parents should be advised that in general infants should not receive solids before the age of 3 months. This is consistent with the observation that the major reduction in choking deaths has occurred in the first few months of life. The avoidance of early introduction of solid food, by delaying exposure to gluten, may have been responsible for the sharp decline in the incidence of coeliac disease in infancy¹⁴. It seems possible that the advice to delay the introduction of solids may also have had the fortuitous effect of reducing the number of small children who choke to death on food. We suggest that avoidance of food choking is a further argument in favour of delaying the introduction of solid food to babies.

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Growth hormone releasing factors

The existence of a human growth hormone-releasing hormone (GHRH) was demonstrated in 1964 by the ability of hypothalamic extracts to release growth hormone (GH) from pituitary tissue¹. Attempts at extracting and sequencing GHRH were unsuccessful until 1984 when Ling and co-workers² isolated both a 44 and a 40 amino acid peptide from 1032 human pituitary stalk-hypothalamic median eminences which had been preserved by freezing. The 44 amino acid peptide (GRF 1-44) was present in greater quantities than the 40 amino acid form (GRF 1-40) in this preparation (ratio 1.4 to 1), although another preparation from 2050 acetone preserved pituitary-hypothalamic fragments had greater quantities of the 40 amino acid peptide (ratio 2.2 to 1). A third preparation of mixed acetone and frozen preserved fragments produced near equal proportions of GRF 1-44 and GRF 1-40. It is therefore possible that acetone causes degradation of the 44 amino acid peptide. Whilst both peptides have similar potencies *in vivo*, the larger peptide has the greatest *in vitro* potency and it would appear logical to denote the 44 amino acid peptide as human GHRH.

That these peptides are highly specific for GH release was already known, as they are identical to peptides which had been extracted from a human pancreatic tumour in 1982. This tumour had caused somatotroph hyperplasia and acromegaly³, and had intrinsic GH-releasing properties attributable to its content of three peptides designated hp (human pancreatic) GRF 1-44, hpGRF 1-40 and hpGRF 1-37. A second pancreatic tumour studied at the same time contained hpGRF 1-40 alone⁴. There is no international agreement on the nomenclature of these peptides, but if GHRH is defined as the 44 amino acid form, then the shorter peptides which act as GH-releasing factors can be abbreviated as GRFs, followed by numbers representing the number of amino acids present. The chemical structure of the shorter peptides is identical except for the number of amino acids that have been deleted. Further deletion of the carboxy-terminal amino acid residues produces peptides with progressively reduced *in vitro* bioactivity, though maximal GH release can be obtained down to GRF 1-21 as the dose-response curves remain parallel⁵. De-amidation of the carboxy-terminal also reduces potency, and deletion or substitution of peptides at the amino terminal causes a profound, reduction in bioactivity⁶. No analogue of GHRH with