*Leading article*

# Age related changes in gut physiology and nutritional status

With an aging population, the gastroenterologist is seeing increasing numbers of elderly patients with unexplained gastrointestinal disorders or malnutrition. If the presentation can be ascribed to normal aging or if no treatment is judged to be available, the patient may not be investigated. To make such an evaluation, however, the doctor must possess a thorough understanding of how gastrointestinal function and nutritional parameters change in the elderly. This review shows that in the light of current knowledge, active treatment will probably be seen as the most appropriate course to pursue in most cases.

## Malnutrition in the elderly

*The scale of the problem*

Protein-energy malnutrition is common in the elderly. Approximately 10% of acute geriatric patients are malnourished on admission.[1] The prevalence is 50% in elderly people living in institutional care and even in free living people aged over 65 years, it is approximately 3% although most of these people have coexisting disease.[2 3] Unless the possibility is considered, however, malnutrition is rarely diagnosed, as even in grossly undernourished patients, haematological and biochemical parameters can be normal.[4] Even serum transferrin, transthyretin (prealbumin), and retinol binding protein, which are considered the best biochemical markers of acute malnutrition, can be normal in the chronic state.[5]

*Does nutrient intake fall in old age?*

In healthy people, earlier cross sectional studies suggested a fall in energy intake with aging, but this no longer seems to be the case. There was no difference in food intake in old compared with young socially privileged women in the USA, although old men did eat less,[6] and in Finland and the USA, longitudinal data suggest that nutrient and energy intake does not change in healthy people in the eighth or ninth decade.[7 8] Food intake is severely diminished in people in institutions, most of whom are physically disabled,[9] and older studies had already suggested that the age related fall in energy intake is associated with decreasing physical ability.[10] Recently, physically active elderly women were shown to eat more than sedentary women, but a moderate exercise training programme did not increase energy intake.[11] Psychological stresses such as bereavement are important causes of decreased appetite as is the inability to chew food because of poor dentition or a dry mouth, factors often neglected by physicians.[12 13]

Vitamin and trace element deficiencies are well recognised in hospitalised patients. Of more concern are the widespread deficiencies in the 'healthy' elderly. In an American survey, large numbers of healthy people aged over 65 years had very low intakes of vitamins A, E, calcium, and zinc. Not surprisingly, subclinical deficiencies of all vitamins have been widely shown. Indeed, the recommended daily intake for many vitamins, which are based on studies in younger people are probably too low for the elderly.[14]

*Changes in anthropometric parameters in old age*

Advancing years are accompanied by loss of height. The proportion of body fat increases and lean body mass decreases as shown by measurements of total body potassium and total body water, or by densitometry or anthropometry; there may also be a shift of fat to non-subcutaneous sites with ageing. Anthropometric data for healthy people over 65 are available to allow useful comparisons to be made in this age group. Measurements of triceps skinfold thickness and mid-arm circumference with the derived arm muscle circumference in healthy normal subjects in Europe and North America show no pronounced change in these indicators of nutrition until at least 75 years of age. There is also no age related fall in plasma haemoglobin or plasma protein concentration in healthy people.

## Changes in gut function with age

*Mouth*

Some 40% of healthy elderly people complain of dry mouth, but whereas unstimulated salivary flow probably decreases, in response to stimulation, salivation is unchanged in both healthy and edentulous people.[15–17] Medication, diabetes, arthritis, and physical dependence are all associated with mouth dryness and those affected are more likely to report difficulty eating and communicating. However, taste and smell sensitivity do decrease with age.[18 19] It is presumed that this decreased sensitivity results in foods tasting similar, with elderly people consequently eating more bland diets. The manufacturing industry is tackling this problem by developing foodstuffs with sharper tastes, which might be preferred by older people.

Healthy elderly subjects open their mouths less widely and chew with less power than young people, a finding that parallels the loss of muscle bulk with age, and which is greater in edentulous people.[20 21] It has been suggested that some of the changes noted are not just a normal part of aging but represent preclinical manifestations of neurodegenerative disorders such as Parkinson's disease.[17]

*Oesophagus*

Classic radiographical studies showed pharyngeal hypotonicity with incomplete opening of cricopharyngeus muscle in 22% of asymptomatic subjects over age 65.[22 23] Manometric studies have confirmed that the upper oesophageal sphincter resting pressure is lower in old age with delayed relaxation on swallowing accompanied by increased pharyngeal contraction pressures.[24 25] In a radiological study of 56 asymptomatic patients in their 80s, two thirds had difficulties in the oral phase of swallowing, one

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quarter had dysfunction of the pharyngeal phase, and oesophageal abnormalities were described in almost 40%.[23] The prevalence of silent acid reflux does not change with age.[26]

*Stomach*

Most studies suggest that a large proportion of 'normal' asymptomatic subjects above 60 years of age have atrophic gastritis. Bird *et al* found only 50 histologically normal gastric biopsy specimens in 201 asymptomatic subjects aged 65 to 90 years, but there was no relation between age and the degree of gastric atrophy in this series.[27] Furthermore, in a study to evaluate the development of gastritis, no subject aged over 60 with initially normal mucosa or superficial gastritis went on to develop atrophic gastritis during a 10 year follow up.[28] Taken together, these findings suggest that gastric atrophy is not a normal part of aging, rather that it is due to specific abnormal mechanisms (see below).

Baron's classic study suggested that both basal and peak gastric acid output decrease with increasing age.[29] This change is probably due to the increased prevalence of atrophic gastritis, however, with most healthy old people actually maintaining gastric acid secretion.[30] Recently, serum gastrin concentration has been shown to be increased only in *Helicobacter pylori* infected subjects but not in elderly uninfected subjects.[30 31] Pepsin secretion does not change with age either.[32]

The importance of *H pylori* infection in the pathogenesis of gastric atrophy and hypochlorhydria is now well recognised and previous or current *H pylori* infection is found in most patients with atrophic gastritis. More surprisingly, *H pylori* might also have a role in the development of pernicious anaemia as in a recent study, 83% of patients with clinical pernicious anaemia also demonstrated antibodies to *H pylori*.[33 34] Whether these findings are of pathogenetic significance is not yet clear. The histological and functional changes in the stomach, however, previously attributed to aging can probably all be explained by the presence of this pathogenic agent, the prevalence of which also rises with age.

Isotope studies have shown a considerable prolongation of gastric emptying for liquids in healthy elderly subjects compared with younger controls. However, gastric emptying for solids is unchanged in old age and the gastric electrical rhythm is maintained in the elderly.[35–37]

*Small intestine*

There is no age related change in small intestinal anatomy and enterocyte height and intraepithelial lymphocyte counts are unchanged.[38 39] Increased cell proliferation in response to injury has been shown in a rodent model which, if also true in humans, would present a possible mechanism for the increased susceptibility to gastrointestinal cancers in the elderly.[40]

The healthy small intestine also maintains its absorptive function for carbohydrates with no change in duodenal brush border membrane enzyme activity of glucose transport; and for fats, although adaptive reserve is more limited.[41–45] Vitamin B12 absorption is also maintained in the healthy elderly.[44 45] 'Malabsorption in the elderly' does not seem to be either a non-specific occurrence or to be due to a specific age related process, but should be explicable in terms of recognised abnormal mechanisms. Bacterial overgrowth may be a comparatively common cause reflecting the high incidence of gastric hypochlorhydria and jejunal diverticulosis,[41 46 47] and although tests of absorptive capacity are normal in most cases, empirical antibiotic treatment has been shown to have a profound impact on nutritional state.[48 49]

Zinc absorption is also decreased from a mixed meal in the elderly, but excretion is similarly diminished so that zinc balance remains.[50] Calcium absorption probably also declines over 60 years of age,[51] but is also lessened in the presence of gastric atrophy. Recent work suggests a fall in vitamin D receptor concentration in the small bowel may decrease intestinal responsiveness to vitamin D activity.[52] Parathyroid hormone secretion and 1,25 dihydroxyvitamin D production may then need to rise to maintain serum calcium homeostasis with the consequence of increased bone loss. The implications of these changes are unclear and further study into their effect on health maintenance and chronic disease in the elderly are required.[53]

*Pancreas*

There is a steady increase in the calibre of the main pancreatic duct with age, and the other branches show areas of focal dilatation or stenosis not associated with any other abnormality.[54] *x* Rays obtained during endoscopic retrograde pancreatography should be interpreted with caution, but this finding is otherwise of no obvious physiological relevance.

Aging changes pancreatic secretion through a decrease in flow rate, bicarbonate and enzyme secretion while calcium secretion is increased. Pancreatic secretion has also been shown to fall significantly upon repeated stimulation.[55] Thus, rare cases of pancreatic exocrine insufficiency could be explained on the basis of aging alone without malnutrition.

*Splanchnic blood flow*

Splanchnic blood flow declines with increasing age both absolutely and as a fraction of cardiac output.[56 57] The splanchnic circulation is also susceptible to hypoxia associated with cardiac or respiratory insufficiency and to hypovolaemia or systemic hypotension.[58] Very few cases of painless occult malabsorption resulting from vascular insufficiency have been described in geriatric patients. The classical syndromes associated with vascular insufficiency of both small and large intestine, which are almost always painful are usually seen in very elderly people.

*Gastrointestinal motility*

Constipation is a common symptom in the elderly. Orocaecal transit time is not generally changed in elderly volunteers[59] although it does seem to be exquisitely sensitive to thyroid hormone status and can be considerably prolonged even in subclinical hypothyroidism.[60] Colonic transit may slow with aging, but is highly variable[61] and interestingly, strength training shortens bowel transit, with the effect being limited entirely to the colon.[62]

*Rectum*

Mechanical changes in the rectum probably cause most of the problems old people suffer in evacuating as well as the increasing prevalence of faecal incontinence with increasing age. A reduction in rectal wall elasticity means that tonic activity of the external sphincter is lost at a smaller volume.[63] An age dependent increase in rectal pressure threshold to produce initial sensation of rectal filling has been found[64] and structural changes also occur in the internal anal sphincter, which has been shown by endosonography to be thicker and hyperechoic in older subjects.[65] Maximum resting anal pressure and maximum

squeeze pressure have been shown to decline with age, particularly in postmenopausal women. The decrease in squeeze anal pressure has been shown in one study to be accompanied by increased mean pudendal nerve terminal motor latency in women in the fifth decade, indicating damage to this nerve. Compensatory reinnervation of this muscle does, however, seem to develop after the menopause.[66] The difference between maximum resting anal pressure and rectal pressure also falls in old age.[67] Taken together, these findings may explain the increased prevalence of faecal incontinence in the elderly and suggest that at least in women, oestrogen treatment may be valuable. This hypothesis remains to be tested.

## Conclusion

Few gastrointestinal functions decline to an important extent as a result of old age alone and there is little clinical evidence that significant malnutrition occurs in any normal elderly person as a result of the aging process itself. Nevertheless, decreased gastrointestinal reserve makes older people highly sensitive to minor insults and decompensation can rapidly occur. Drugs appreciably affect taste sensation, which is already blunted and psychological as well as physical disability can have a major impact on appetite. Malabsorption can be caused by gastric hypochlorhydria with small bowel bacterial overgrowth and while gastrointestinal dysmotility can be caused by subclinical hypothyroidism, it can improve in response to physical exercise. Evidence is now mounting that thorough investigation of gastrointestinal disturbances in elderly patients coupled with intensive nutritional support can make a very real impact on their outcome. Gastroenterologists should therefore seek out and actively treat gastrointestinal disorders in the elderly and not just ascribe them to old age.

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