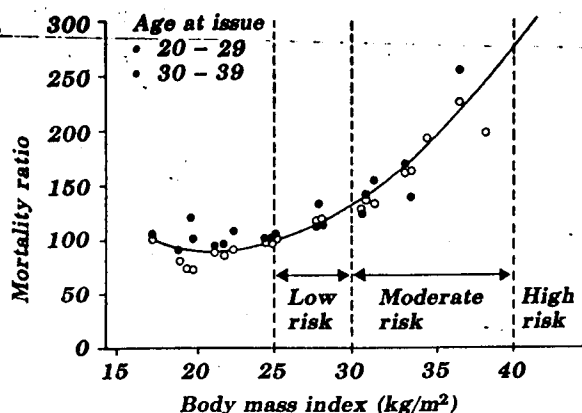


# Energy Undernutrition and Overnutrition

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**W**hat is remarkable is how well most people maintain their bodyweights throughout adulthood given the potential consequences of small, consistent errors in daily energy balance. Consider, for example, the error involved in either the additional consumption of one 100g banana (about 340 kilojoules or 800 kilocalories) each day or the energy cost of light physical activity such as walking slowly, which amounts to about 12 kilojoules (2.9 kilocalories) per minute for a 58kg woman compared with a basal energy expenditure of 4 kilojoules per minute. The energy value of body fat is about 25 to 27.2 kilojoules or 6.0 to 6.5 kilocalories per gram. On this basis, the banana could yield, each day, 12 to 13 grams of fat!

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**FIG. 1.** Relation of body mass index to excess mortality [after Bray (1)].

## Body Fatness and Survival

Body mass index is weight in kilograms divided by height in metres<sup>2</sup>. This expression minimises the effect of height-related body mass and accentuates the effect of body fatness. The overall relationship between mortality rates and body mass index is a J-shaped curve, with the nadir between body mass indices of 20 and 25 (fig. 1). There is significant increase in mortality below and above these levels. Above a body mass index of 30, the increase in mortality is of particular consequence and, since this is an index where the increase in adiposity, rather than lean body mass, must be substantial, this is now the level taken to divide obesity from what otherwise might be regarded as overweight. However, the interaction between obesity and other factors can be seen by considering cigarette smoking, from the American Cancer Society Study (fig. 2).

Great interest has recently been stimulated in the predictive power of distribution of body fatness and this can be seen from figure 3.

Here, once distribution of body fatness is taken into account, the body mass index is not predictive of mortality, stroke rate or ischaemic heart disease in the same way. Similar findings have now been found in several studies insofar as the risk of diabetes is concerned.

## Energy Value of Food

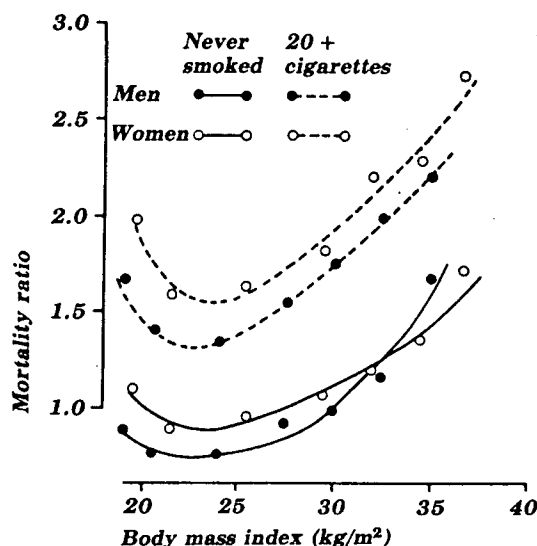
The energy value of food is usually expressed in kilocalories or kilojoules per 100g ( $4.18 \times \text{kilocalories} = \text{kilojoules}$ ). Such values are usually provided in tables of food composition. The Australian tables are presently undergoing extension and revision.

Particular attention must be paid to the effect of food preparation techniques on energy value as can be seen from figure 4. The energy value in kilojoules of macronutrients are fat 37 kJ/g, alcohol 29 kJ/g, protein 17 kJ/g and carbohydrate 16 kJ/g.

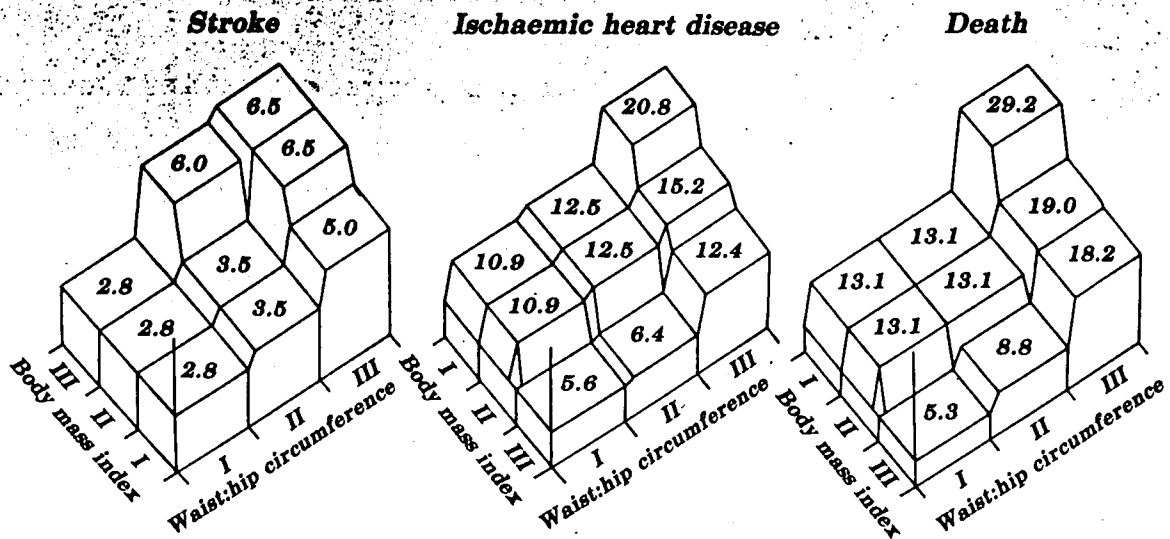
## Maintenance of Energy Balance

### Hunger and Appetite

Castonguay et al. [4] have made the distinction that hunger asks the question "Is there anything to eat?" and appetite "What do I want to eat?". The variety of factors which might influence either hunger or appetite are shown in figure 5.

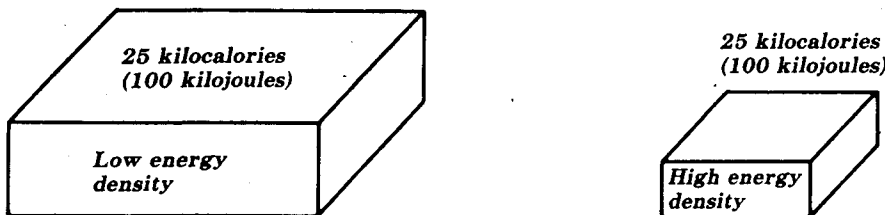


**FIG. 2.** Relation of smoking to relative risk and body mass index in the American Cancer Society Study [after Bray (1)].

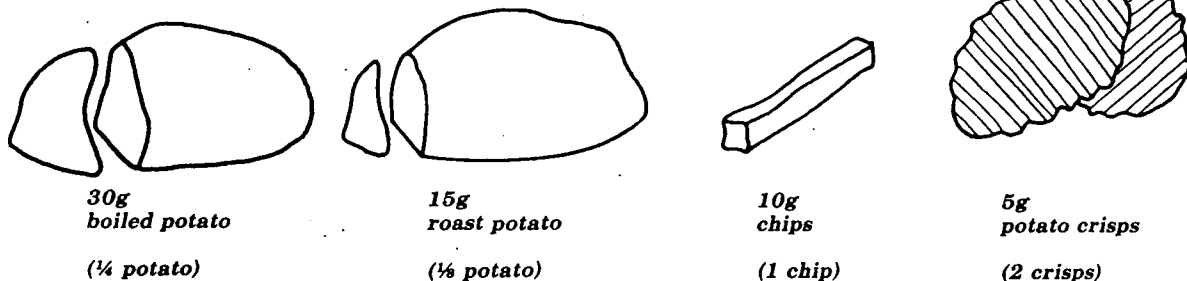


**FIG. 3.** Percentage probabilities of stroke, ischaemic heart disease and death from all causes in relation to tertiles of body mass index and waist to hip circumference ratio. The body mass index axes are reversed for death and ischaemic heart disease [after Larsson (2)].

Some foods allow you to eat more per 25 kilocalories (100 kilojoules)



All these foods are 25 kilocalories (100 kilojoules)



**FIG. 4.** Fat markedly alters energy value, largely dependent on the surface area available for absorption of fat [after Briggs & Wahlqvist (3)].

## Metabolic Rate

The basal metabolic rate (BMR) is the main component of energy expenditure in the human adult with light or moderate levels of physical activity.

Heat loss is proportional to body surface, which is, in turn, proportional to  $W^{2/3}$  (where  $W$  = weight or mass).

Heat production must relate to heat loss, but temperature regulation is not the main determinant of the basal metabolic rate, since cold-blooded vertebrates, unicellular organisms and even certain trees obey a similar formula. Lean body mass or fat-free weight is a better predictor of basal metabolic rate than total bodyweight. The decline in basal metabolic rate with age may represent nothing more than a decrease in active cell mass. The various factors affecting the rate, therefore, will include body composition, neuroendocrine factors and cellular metabolism. The prediction of basal energy expenditure (BEE) is usually made by the Harris-Benedict equations.

For women,  
$$BEE = 655 + (9.6 \times W) + (1.8 \times H) - (4.7 \times A)$$

For men,  
$$BEE = 66 + (13.7 \times W) + (5 \times H) - (6.8 \times A)$$
  
where  $W$  = ideal body weight in kilograms;  
 $H$  = height in centimetres;  $A$  = age in years

This matter has been under review recently and, as a consequence, alternative predictive equations may be preferred.

To the basal energy expenditure, calculated from the Harris-Benedict equation, must be added percentages to adjust for activity, i.e. sedentary - 30%, moderate activity - 50%, strenuous activity - 100%. Non-stressed hospitalised patients usually require 120% of basal energy expenditure. Catabolic patients usually require 150 to 200% to prevent tissue breakdown or to allow anabolism. Other factors which need to be taken into account in assessing energy expenditure are fever - an increase by about 13% over basal per degree Celsius, burns - 40 to 100%, trauma - 40 to 100%, hyperthyroidism - 10 to 100%, and malabsorption - oral intake may not prevent starvation.

## Energy Intake

The various food factors influencing energy intake are energy density, macronutrient composition, and palatability. We can consider these issues in the following way:

*Macronutrient Composition:* Avoidance of what have been regarded as carbohydrate-rich foods has become part of folklore in regard to reduced risk of obesity. Carbohydrate craving has been suggested as a basis for obesity in a sub-group of obese individuals [6]. However, so-called carbohydrate-rich foods are often also fatty foods, examples being sweet biscuits, pastries and ice cream. Rats have been shown to prefer, overeat and become obese on sweet-fat foods [7]. These foods may be preferred by obese people [8] and hedonistic responses to food in humans can be shown dependent on sugar-fat interactions [9].

## ***Non-stressed hospital patients usually require 120% of basal energy expenditure***

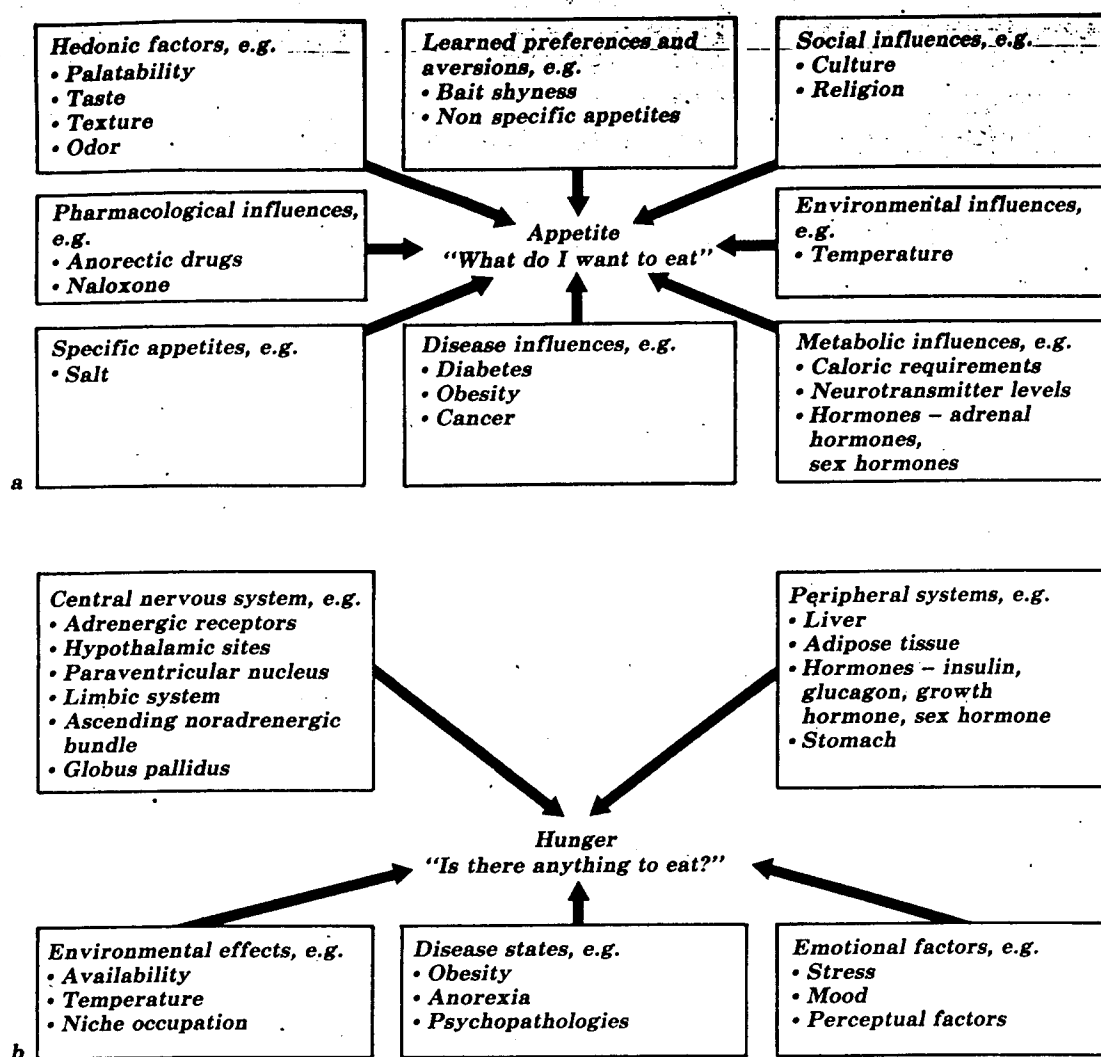
*Dietary Fibre:* To increase dietary fibre intake in its own right has been popularised as a way of increasing satiety [10]. Few well controlled studies have been carried out on the use of dietary fibre in weight control, but 2 recent studies lend some support to this approach [11,12].

## Energy Expenditure

Several of the factors which alter energy expenditure have already been referred to under 'Metabolic Rate' and further discussion on the value of exercise in control of body fatness is provided below under 'Obesity'.

## ***Wasting States (Energy Undernutrition)***

Energy undernutrition, in combination with protein undernutrition and usually many other essen-



**FIG. 5.** a) A partial list of the ever growing number of factors that are known to influence the onset of hunger. b) Some of the factors that determine appetite [after Grande (5)].

tial nutrient deficiencies, constitutes the world's most prevalent nutritional problem. It principally affects children under the age of 5 years in developing countries. However, in hospitalised patients in developed countries, the problem of protein energy malnutrition is commonly seen.

Even in the community it is generally thought that in developed countries the dominant energy nutritional problem is one of overnutrition. However, the Geelong growth study by Rutishauser

and Hunter [13] showed that, where both parents are Australian-born, underweight was more common than obesity.

It must be recognised, nevertheless, that the health outcomes of underweight in developed countries, at least in children, are not defined in the same way as they are in developing countries. For example, whereas the nexus between infection and undernutrition in developing countries is clear, this may or may not be the case in devel-

oped countries, where hygiene and the management of infectious diseases is of a different order.

There are various ways in which energy undernutrition may arise.

**Starvation (Reduced Intake):** For most of the world's population who suffer from starvation, the problem is one of inadequate availability of food.

In developing countries, the problem of malabsorption can be common, on account of intestinal parasitic infestation, tropical sprue, or other problems. Malabsorption is also sometimes a cause of reduced energy availability in developed countries.

A growing problem in developing countries is that of eating disorders. Anorexia nervosa is perhaps the most dramatic of these states.

**Hypercatabolism:** There are a number of states in which increased catabolism occurs. These include febrile states, post-trauma, especially head injury, neoplastic disease and hyperthyroidism.

**Energy Loss:** Malabsorptive states may be thought of as states of energy loss. It is also possible for energy loss to occur in the urine, by way of glycosuria as in diabetes. Large energy losses can occur before diabetes comes under control.

## Obesity (Energy Overnutrition)

In Australia, the National Heart Foundation Risk Factor Prevalence Surveys of 1980 and 1983 have provided important data on the prevalence of

obesity. These prevalences, at least for 1980, have been compared with those in Britain and the USA in table I.

It should be noted that, here, obesity is defined as a body mass index above 30 kg/m<sup>2</sup> and, on this basis, the prevalence of obesity in Australia is less than in the USA. In the range of 25 to 30 kg/m<sup>2</sup>, the extent to which the extra weight is attributable to adiposity, will depend very much on the level of physical activity. The trained athlete may have an increase in lean body mass contributory to this figure.

***Causes of obesity such as hypothyroidism and Cushing's diseases are rare when compared with obesity due to an unsatisfactory lifestyle***

Obesity can sometimes be secondary to states like hypothyroidism, Cushing's Disease (hyperadrenocorticism), insulinoma, hypothalamic disorders and a result of treatment with certain medications. However, these are rare by comparison with what might be regarded as primary obesity, namely that associated with an unsatisfactory lifestyle.

Factors which need to be taken into account with primary obesity are genetics, energy expenditure, energy intake and efficiency of energy utilisation.

**TABLE I. Percentage of overweight (body mass index 25-30 kg/m<sup>2</sup>) and obese (body mass index over 30 kg/m<sup>2</sup>) people in English-speaking countries [after Bray (1)]**

	Age	Overweight (%)		Obese (%)	
		Male	Female	Male	Female
Australia	25-64	34	24	7	7
Britain	16-65	34	24	6	8
United States	20-74	31	24	12	12

**TABLE II. Intake of energy and macronutrients in men who died from all causes and survivors (after Krahnouk [16])**

	Intake	
	All deaths (n = 107)	Survivors (n = 764)
Energy (Kcal)	2833	3077
Energy/kg bodyweight (Kcal/kg)	39.5	43.2
Vegetable protein (g)	33.6	38.2
Monounsaturated fat (g)	58.4	62.1
Polyunsaturated fat (g)	18.5	20.2
Oligosaccharides (g)	129.6	140.2
Polysaccharides (g)	179.5	206.3
Dietary fibre (g)	26.9	30.9

## Energy Expenditure

It is a common observation in various studies that obese persons may be less physically active than their non-obese counterparts.

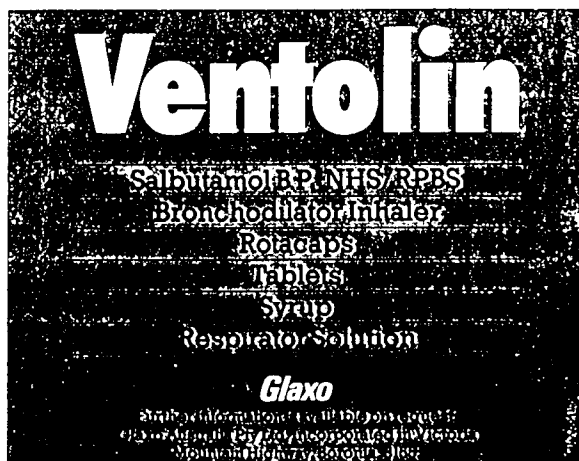
There are several reasons why increased levels of physical activity can be expected to be of value in the prevention and management of obesity. These are a sense of well-being, appetite more correct, energy cost of any event is increased with mass increase, and lean body mass is increased.

## Energy Intake

It is a common error to assume that the energy intake of obese people when seen must have been that when their weight increased. They may well now be being seen in a new steady state. It is most important to enquire what the times were when weight increased. These may have occurred at different times of life, in childhood, adolescence, or early adulthood, for example. They may have occurred with pregnancy, weights not returning to those preconception. It may well be that no increase in intake has been apparent, but there may have been a clear decline in physical activity at some point in life.

However, it should be noted that prospective studies in London and Zutphen [16] indicate the beneficial effect of higher energy intakes in de-

creasing coronary mortality and total mortality. This presumably means that, for a particular weight/height relationship, those with the higher levels of physical activity live longer. Thus, although in the short term there may be advantage in decreasing energy intake to prevent or control obesity, in the long term it would be better to be more physically active – the same studies indicate that the extra food should be plant food.



**TABLE III** Percentage of expected deaths from various causes of overweight men and women aged 25 to 74 years compared with the general population [after Mayer (18)]

Cause of death	Men	Women
Principal cardiovascular-renal diseases	149	177
Organic heart disease, diseases of the coronary arteries and angina pectoris	142	175
Cerebral haemorrhage	159	162
Chronic nephritis	191	212
Cancer		
Liver and gallbladder	168	211
Breast		69
Diabetes	383	372
Tuberculosis, all forms	21	35
Cirrhosis of the liver	249	147
Appendicitis	223	195
Hernia and intestinal obstruction	154	141
Biliary calculi and other gallbladder diseases	152	188
Biliary calculi	206	284
Puerperal condition		162
Suicide	78	73
Accidents, total	111	135
Automobile	131	120

## Efficiency of Energy Utilisation

As already indicated, the work of Garrow [15] would suggest that the range of efficiencies of energy utilisation are similar between obese and non-obese persons. However, it is possible that subgroups could behave differently and that in the small numbers of subjects possible to study with whole body calorimetry, these subtleties have not yet emerged.

The health outcomes of obesity are reflected in increased total mortality rates as already indicated. The components of this increased total mortality, and of increased morbidity short of increased mortality, have been in evidence for some time.

The American Cancer Society Study, by Lew and Garfinkel [17], shows that increase in degrees of overweight in men are associated with a statistically increased risk of cancer of the colon, rectum and prostate. For women, there was a progressive increase in risk of cancer of the breast, uterus and cervix. Although this does not mean a causal relationship, it is noteworthy that there are

enhanced rates of oestrogen production in the overweight which may well predispose to the increased risk of certain cancers. There are also increases in risk of cancer of the gall bladder and biliary system with increased weight.

Problems of increased morbidity, not reflected in the mortality data, with obesity include: arthritis, respiratory impairment, psycho-social problems, and menstrual disturbances.

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## CARTIA™ PRESCRIBING INFORMATION

### COMPOSITION

Each 'Cartia'™ 'Duentic'® enteric coated tablet contains 100mg acetylsalicylic acid (aspirin).

### PHARMACOLOGY

In platelets, endothelial and smooth muscle cells, arachidonic acid produced from cell membrane phospholipids is metabolised to various prostaglandins including thromboxane (TXA<sub>2</sub>) and prostacyclin (PGI<sub>2</sub>) by the enzyme cyclo-oxygenase. TXA<sub>2</sub>, which acts as a platelet aggregator and a vasoconstrictor, is produced in platelets while PGI<sub>2</sub>, which is an inhibitor of platelet aggregation and a vasodilator, is produced in endothelial and smooth muscle cells.

Aspirin, by irreversibly acetylating cyclo-oxygenase, reduces the production of TXA<sub>2</sub> in platelets. Aspirin can also reduce PGI<sub>2</sub> production in endothelial cells. As endothelial cells are nucleated, cyclo-oxygenase can be regenerated, though how quickly after aspirin is uncertain. Whether platelet cyclo-oxygenase is more sensitive to aspirin than endothelial cell cyclo-oxygenase has not been fully established.

Studies in human volunteers using 'Cartia'™ tablets have shown that thromboxane B<sub>2</sub> production is suppressed by 99% while prostacyclin production as measured by urinary 6-Keto-PGF<sub>1α</sub> (a PGI<sub>2</sub> metabolite) remains at a mean of 94% of control values. Platelet aggregation induced by arachidonic acid and collagen and malondialdehyde production is suppressed by 96%, 60% and 97%, respectively. The slow release of aspirin from enteric coated 'Cartia'™ tablets allows platelets to be irreversibly acetylated in the portal circulation by continual low doses of aspirin. As hepatic metabolism of these low doses of aspirin to salicylate approaches totality, little or no aspirin is released into the systemic circulation to affect vascular wall PGI<sub>2</sub> production, salicylate having no effect on cyclo-oxygenase.

### PHARMACOKINETICS

**Absorption:** After oral administration aspirin is released from 'Cartia'™ tablets when the pH is > 6 i.e. in the duodenum and small intestine. Thus the stomach is not exposed to the local effects of aspirin. **Metabolism:** Aspirin is converted to salicylic acid mainly in the gastrointestinal mucosa and the liver. Systemic plasma levels of acetylsalicylic acid resulting from the administration of aspirin as 'Cartia'™ (one tablet daily for seven days) are below the level of sensitivity of standard assays for acetylsalicylic acid. However, ng or pg/mL levels have not been ruled out.

**Protein binding:** Salicylate is 80-90% bound to plasma protein especially albumin, at clinical concentrations of salicylate.

**Excretion:** The mean recovery of aspirin as total urinary salicylate accounted for 87 ± 9% of the 'Cartia'™ 100mg dose administered.

### INDICATIONS

'Cartia'™ is indicated for vascular conditions where platelet aggregation inhibition and sparing of vascular wall prostacyclin are indicated.

### CONTRAINDICATIONS

- Aspirin hypersensitivity manifested for example as anaphylaxis, asthma or angioneurotic oedema.
- Bleeding disorders such as haemophilia and Von Willebrand's disease.
- Gastric haemorrhage, erosive gastritis or active peptic ulcer disease.
- Not recommended for use in children.

### PRECAUTIONS

Care should be taken when 'Cartia'™ is administered with uricosuric agents, non-steroidal anti-inflammatory agents or anticoagulants or to

patients with a history of peptic ulcer disease or bronchial asthma. There is no experience with 'Cartia'™ use in patients with renal or hepatic disease and care should be taken when aspirin is administered to these patients. It may be advisable for 'Cartia'™ to be withdrawn one week before surgery. Blood donors should cease all forms of aspirin at least a week before giving blood.

### USE IN PREGNANCY

'Cartia'™ should not be used in pregnant women or those likely to become pregnant unless the benefits outweigh the risks. Aspirin has been used in high-risk pregnant women, after the first trimester, for the prevention of pre-eclampsia in doses of aspirin 60mg/day and aspirin 150mg/day combined with dipyridamole 300mg/day resulting in a significantly lower incidence of pre-eclampsia.

**Use in Lactation:** Aspirin is excreted in breast milk.

**Use in Children:** Not recommended for use in children.

### ADVERSE REACTIONS

Side effects of aspirin are mainly gastrointestinal including heartburn, nausea, bleeding and activation of peptic ulcer. 'Duentic'® enteric coating has been shown to decrease the risk of gastric damage. Other possible adverse events include tinnitus, vertigo and deafness but in the doses of aspirin administered in 'Cartia'™ these risks appear to be minimal. Asthma, vasomotor rhinitis, urticaria and angioneurotic oedema are common manifestations of aspirin sensitivity but other skin reactions, e.g. erythema and pustular eruptions have also been described.

### DRUG INTERACTIONS

Aspirin, because of its effect on platelets should be avoided in patients using oral anticoagulants or heparin, unless indicated. Care should be observed when coadministering aspirin and methotrexate, chlorpropamide, corticosteroids, sulphapyrazone, probenecid and spironolactone.

### Interference with clinical and laboratory tests

- Heparin activity:** Decreased platelet activity caused by aspirin may exaggerate heparin activity tests such as Lee-White clotting time and activated partial prothrombin time.
- Urinary glucose oxidase:** Aspirin may cause a false negative test in the presence of glycosuria.

### DOSAGE AND ADMINISTRATION

**Adults:** One 'Cartia'™ tablet daily.

**Children:** Only under strict medical supervision.

### OVERDOSAGE

Symptoms are those of salicylate intoxication. In mild overdosage these may include rapid and deep breathing, nausea, vomiting, vertigo, tinnitus, flushing, sweating, thirst and tachycardia. Severe cases may show fever, haemorrhage, excitement, confusion, convulsions or coma, respiratory failure. Suggested treatment is: Gastric lavage or emesis. Force fluids, but if patient is unable to retain fluids orally, infuse saline or sodium bicarbonate depending on electrolytes and pH. Sodium bicarbonate will increase renal excretion of salicylate. In severe cases dialysis should be considered.

### PRESENTATION

Calendar pack of 28 tablets.

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