

Clinical nutrition in primary health care

Mark Wahlqvist, Boyd Strauss



Mark Wahlqvist, BMedSci, MD (Adel), MA (Uppsala), FRACP, (pictured) is Professor, Department of Medicine, Monash University, Monash Medical Centre, Melbourne, Victoria.

Boyd Strauss, MB, BS (Monash), PhD (Deakin), FRACP is part-time Senior Lecturer, Department of Community Medicine, Monash University.

Nutrition has always been an important component of medicine. Hippocrates had a strong interest in the subject and Maimonides, in the Middle Ages, considered diet in relation to the perceived health problems of the day.

In the long history of medical practice it is only for perhaps the last 20 or 30 years that nutrition has had a relatively minor role. Nutritional problems, especially malnutrition and infectious disease, continue to constitute the world's major health problems.

The discovery of micronutrients provoked the developed countries to extend skills in nutrition assessment and diagnosis into medical practice. The importance of nutrition in general practice has now been redefined, and alongside dermatology and psychological disorders, nutritional disorders are among the most common problems encountered by doc-

tors. The Social Issues Committee of the Royal Australasian College of Physicians also recognises the growing importance of nutrition, and has released a report on nutrition diagnosis.

We all know that many patients come to us with their own nutritional diagnosis, with their own views and interpretations. However, the general practitioner should be able to provide competent nutritional assessment, diagnosis and management.

Table 1 lists clinical situations in which a nutritional diagnosis is required.

Why nutrition diagnosis?

The three dimensions of medical practice that concern nutrition are shown in *Figure 1*.

We often speak about the need to develop nutritional assessment skills in clinical practice. But what distinguishes those of us in medicine from other areas of health care practice? While others may make an assessment, the business of *diagnosis* is the business of medicine. That is the core of this discussion. Nutritional management flows on naturally from our ability to establish a nutritional diagnosis.

Medicine is distinguished from its alternatives by its ability to bring together different lines of information and different ways of gathering evidence before determining priorities for management.

An assessment that ultimately leads to nutritional management may have very little to do initially with nutritional assessment. A nutritional diagnosis of obe-

■ Nutritional diagnosis and management are important aspects of general practice. This information, which is presented in two parts, offers the general practitioner a practical framework and an approach to nutritional advice.

Part 1 outlines the clinical conditions and principles involved in nutritional diagnosis with a management approach to macrovascular disease and obesity.

Part 2 covers protein malnutrition, eating disorders, osteoporosis, nutrient toxicity, cancer, inherited metabolic disorders, nutrient deficiency and diabetes mellitus.

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sity, for example, may have more to do with physical inactivity than with nutrition. Nevertheless, it has nutritional implications.

Table 2 lists the principles of making a nutritional diagnosis.

Nutrition and genetics

The interplay between genetics and food needs to be underscored at the outset. Recently, for example, the genetic basis of obesity was established by Stunkard in the Danish Adoption Study.¹ If obesity can be clearly ascribed to genetic factors, then what can be done about it? An analogy is phenyl-ketonuria, which is principally genetically determined, but its expression and its management depend very much on the way in which the food intake is controlled: this strongly genetic condition is amenable to management.

The assessment and management of lipid disorders is another example. On the one hand, familial hypercholesterolaemia is genetically determined: a condition in which the metabolic or molecular biological basis in the LDL receptor defect is now fairly well understood. That particular defect is very poorly responsive to diet and it would be foolish to suggest that that particular problem can be managed by nutritional means alone.

Nevertheless, the risk from this condition can be greatly lowered by nutritional management, irrespective of the plasma cholesterol level. At the other end of the spectrum of lipid disorders, however, are forms of hypercholesterolaemia that are very responsive to nutritional management.

The assessment of food intake

The assessment of food intake is perhaps the area in which most of us in the medical profession feel most uncomfortable. In some cases, this may be because the medical undergraduate curriculum was

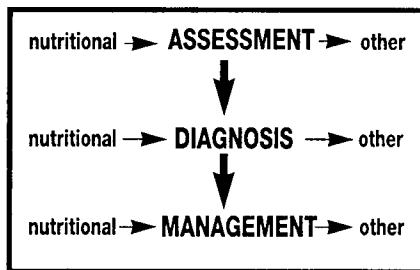


Figure 1. The three dimensions of medical practice that concern nutrition.

good on nutritional biochemistry, but did not have a lot to do with food. However, doctors are well placed to rapidly increase their skills in this area. A supplement to the *Medical Journal of Australia* (July 1989)² gave answers to some common questions from patients about food. It considers food technology, food composition, and disorders of taste and smell and their impact on nutritional status.

Many doctors find it difficult to get access to food composition data. In *Food Facts* published by Penguin (*Further reading*) we have arranged nutrient composition in 100 gram units so that at a glance you can see the foods that are good, poor or moderate sources of virtually any nutrient.

It is important to at least know the foods that are good sources of key nutrients. For example, there are four good sources of folic acid: green leafy vegetables, liver, citrus fruits (in order, oranges, grapefruit, lemons) and cereals (other sources of folate include nuts).

In clinical practice, there are basically two ways to assess food intake:

1. As part of general history taking, when we ask patients to run through the day from morning to evening and describe what they usually eat — it takes between 5 and 7 minutes once one gets used to the routine.
2. Ask the patient to record everything eaten for a week. We suggest that weekend days and week days both be recorded, as food intake can vary markedly as other activities vary.

TABLE 1

Clinical situations requiring a nutritional diagnosis

A nutrition diagnosis is required as part of the management of the following conditions:

- macrovascular disease
- disorders of body composition
 - obesity
 - energy undernutrition
 - protein malnutrition
- eating disorders
- alcohol excess
- osteoporosis
- diabetes mellitus
- micronutrient deficiency
- micronutrient toxicity
- concerns about cancer
- inherited metabolic disorders.

Both techniques can be repeated from time to time to determine whether food intake has changed.

We have prepared simple forms that run through breakfast, morning snack, lunch, afternoon snack, dinner, supper and other times of eating. If, for example, a patient has recently developed colonic polyps, and already made some changes to diet that might not be well founded, it is easy to explore these differences and set the patient on the right track. With newly diagnosed diabetes, food intake may change by the time of further consultation: for example, the patient may eliminate all sugar, which is not particularly useful unless there happens to have been a high intake.

Alternatively, one can give patients a record sheet, or they can provide a diary of their eating activities from Monday through to Sunday. When they bring this back we recommend that it be photocopied, and a highlighting pen be used to

mark the foods that we want to see changed, making a note at the bottom of practical suggestions to effect the change.

We find that this is one of the most important things one can do in clinical practice in the process of nutrition management.

A written record, then, gives a view of the food intake and documents changes that have occurred.

Making a nutritional assessment: case study

The steps in making a nutritional assessment are:

1. understand the patient's problem or concern;
2. find out what components of the food intake are determinants of the problem;
3. make some assessment of body composition.

Other clinical tests, including functional outcomes and laboratory measurements where appropriate, may then be required.

Case study

A 42 year old business executive, married with two children, presents to the surgery. Our patient's concerns include a family history of premature coronary artery disease, a common reason for seeking a consultation these days. His father died at 43, just a year older than the patient, which stimulated him to see what he might do to avoid the same problem. This provides a powerful tool for motivating a change of diet.

On assessing his usual food intake during the consultation and then to further record it over a full week, we found he had the hallmarks of all the food intake problems that would predispose him to macrovascular disease.

Food intake was high in saturated fats, which constitutes a risk, but additionally his dietary fibre intake as an index of

TABLE 2

Principles of making a nutritional diagnosis

1. Think of food or nutrient intake as a possible explanatory model for the patient's problem.
2. Consider the importance of food intake relative to other components of diagnosis, including:
 - genetic predisposition or cause
 - other aspects of lifestyle such as physical activity, alcohol consumption, cigarette smoking and stress
 - the presence of other disease processes
 - the effect of medication
 - interaction between these factors.
3. Include the nutritional assessment as part of the overall clinical assessment.
4. Keep functional outcome, diagnosis and management in mind as you assess.
5. Involve the patient while coming to a diagnosis, explaining, clearing up misconceptions and giving practical hints about changes in food habits while this is being done.
6. Formulate the problem or sets of problems and indicate the consequences in terms of action and the time frame.
7. Develop 'clinical nutrition repertoires' for common situations such as:
 - premature macrovascular disease
 - back pain and osteoporosis
 - obesity
 - the use of nutrient and herbal remedies.
8. Try to make a formal statement, at least mentally, about the nutritional diagnoses involved.

TABLE 3

Managing macrovascular disease

- Achieve energy balance, preferably through increased physical activity — there is now much evidence to suggest that it is energy *throughput* that is most related to a reduction in cardiovascular mortality, providing that weight remains constant, and this can only be achieved by an increase in physical activity.
- Decrease the total saturated fat intake, while conserving some polyunsaturated fat intake (omega-3 and omega-6, from vegetables and fish) and monounsaturated fat intake (such as those found in olive oil and avocado).
- Decrease dietary cholesterol intake, especially in those with a serum cholesterol >5.5 mmol/L and inadequate HDL cholesterol. The effect of dietary cholesterol on serum levels is greater when it is consumed with saturated fats — the effect of an egg fried in butter is much greater than that of a boiled egg.
- Increase the intake of plant food. These foods have a high nutrient density and contain certain non nutrients like allium and saponins that may have cholesterol-lowering effects of their own.
- Increase fish intake. It is important to note that the benefits on coronary mortality may be related to total fish consumption, rather than the consumption of fish oils alone.
- Decrease the ratio of dietary sodium to dietary potassium.
- Have no more than one or two standard alcoholic drinks a day, preferably with food. Alcohol affects energy intake and increases blood pressure.

TABLE 4**Body mass index and risk implications**

BMI	Grade	Implications
20-24.9	0	
25-29.9	I	Increased risk
30-39.9	II	Obesity
>40	III	Morbid obesity

plant food intake was low. Saturated fat intake is often accompanied by increased sodium and low fibre intake.

Because people often follow their family tradition, it is important to work through their food practices, their recipes, the things mother did, the things that the spouse now does. In this case, there was a need to progressively reduce saturated fat. His intake of fish was low: an increase in fish intake has emerged as a protective factor against premature coronary disease and death.

It is remarkable how little fish most Australians actually eat, and it may even have been lower had it not been for the Catholic tradition of Friday fish. For people who have no fish, the risk of cardiovascular disease is significantly increased, and even one meal of fish a week may considerably reduce the risk of cardiovascular disease.

The patient's body composition was assessed: the body mass index (described below) was 33 and his waist-hip ratio 1.2, both being significant risk factors for cardiovascular disease. A waist-hip ratio above about 0.9 for men and 0.85 for women is now recognised as a key risk factor for cardiovascular disease (both coronary and cerebrovascular), together with the expression of non-insulin dependent diabetes mellitus.

The appropriate investigations in this case are blood lipids. Measuring HDL

cholesterol is necessary, otherwise it is difficult to make sense of the lipid values. One also needs to assess, and exclude where appropriate, secondary hyperlipidaemia.

The final picture is one of premature vascular disease with a strong family history, hypercholesterolaemia, abdominal obesity, and a food intake that is high in saturated fat from dairy foods and meat, and little physical activity.

Nutritional management

The nutritional approach to the management of macrovascular disease in this example is shown in *Table 3*.

In managing diet in relation to macrovascular disease, the general practitioner should be able to answer questions such as:

- What are the major sources of fat in the Australian diet?
- How safe is it to reduce the intake of dairy products?
- How valuable are plant and fish fats in the diet?
- What are the sources of sodium in the diet?
- How can food affect macrovascular disease and its outcomes?
- When are blood tests necessary for nutritional intervention?

Obesity Assessment

Obesity is present when the fat mass of an individual is increased to a level where there is a likelihood of impairment of health. The easiest clinical assessment of obesity is by use of the body mass index:

$$\text{Body mass index (BMI)} = \frac{\text{weight (kg)}}{\text{height (m)}^2}$$

Garrow³ has produced a simple classification of the body mass index associated with the relative degree of risk increase (*Table 4*).

Of course, the BMI needs to be assessed in the context of the patient's phys-

TAGAMET® (cimetidine). Abridged Product Information.

INDICATIONS: Treatment of duodenal ulcer, gastric ulcer; short-term treatment of persistent gastro-oesophageal reflux disease unresponsive to other measures; maintenance treatment for recurrent duodenal ulcer; maintenance treatment for up to 1 year of chronic benign gastric ulcer; prevention of stress ulcer; gastrinoma (Zollinger-Ellison Syndrome); scleroderma oesophagus.

CONTRAINDICATIONS: Hypersensitivity to cimetidine.
PRECAUTIONS: Exclude malignant gastric neoplasm before therapy with TAGAMET is instituted. * It is then important to re-endoscope the patient after 8 - 12 weeks of Tagamet therapy to check that the ulcer has healed. Dosage adjustment may be necessary in patients with impaired renal function or undergoing haemodialysis (see full Product Information for dosage); avoid rapid i.v. injection and monitor BP and pulse during i.v. use; 800mg effervescent tablets contain 432mg sodium therefore caution is advised in patients on salt restricted diets. Clinical experience in children is limited; cimetidine is distributed into breast milk.

* Agents that elevate gastric pH may increase the already present risk of nosocomial pneumonia in intubated ICU patients receiving mechanical ventilation.

* The Tagamet 800mg effervescent tablet should be used with caution in persons with phenylketonuria. The Tagamet 800mg effervescent contains 25mg aspartame per tablet.

Use in Pregnancy category: B1

INTERACTIONS: *Cimetidine may modify the pharmacokinetics and pharmacodynamic response of some drugs by modulating their absorption, hepatic metabolism or renal tubular excretion. Effects on the cytochrome P-450 mixed function oxidase system in the liver or on renal tubular excretion may increase the plasma concentration of some co-administered drugs by delaying their elimination. Toxicity may occur rarely for drugs with a narrow therapeutic index e.g. warfarin-type anticoagulants, phenytoin, theophylline, lignocaine, quinidine, procainamide, flecainide; and also with nifedipine.

In the case of warfarin anticoagulants, closer monitoring of prothrombin time is recommended, and adjustment of the anticoagulant dose may be necessary when cimetidine is administered concomitantly.

In the case of the other drugs listed, plasma levels should be monitored more closely when starting or stopping concomitantly administered cimetidine, and dosage adjustments made as necessary.

Other drugs which may be affected by cimetidine include β-blockers, calcium channel blockers, tricyclic anti-depressants, benzodiazepines, chlormethiazole and metformin. Although concomitant cimetidine administration may result in increased blood levels of these drugs, clinically significant effects occur in relatively few cases. Special caution should be exercised in the elderly and those with liver or renal disease.

ADVERSE REACTIONS: More frequent: headache, diarrhoea, tiredness, muscular pain, dizziness, rash, constipation. Less frequent: increases in plasma creatinine and serum transaminases, severe skin rash, reversible alopecia and gynaecomastia.

Rarely: reversible confusional states, interstitial nephritis, hepatitis, fever, pancreatitis, leukopenia, (including agranulocytosis), thrombocytopenia, pancytopenia, aplastic anaemia, sinus bradycardia, tachycardia, heart block, anaphylaxis, reversible impotence.

DOSAGE: ORAL: NOTE: effervescent tablets should be dissolved in a glass of water before swallowing.

Acute Duodenal Ulceration and Acute Gastric Ulceration: 800mg at bed-time for 4 - 8 weeks; severe cases 400mg 3 times daily and at bed-time. Maintenance: 400mg at bed-time. Zollinger Ellison Syndrome (Gastrinoma): 200mg 3 times daily and 400mg at bed-time. Dosage may be increased up to 2g/day.

Gastro-oesophageal reflux: 800mg at night or in divided doses for up to 12 weeks.

Scleroderma oesophagus: 1200mg/day in divided doses.

* See full Product Information for dosage with impaired renal function.

PARENTERAL: Prophylaxis of Stress Ulceration: 200mg every 3 - 6 hours or 300mg every 4 - 8 hours i.v.i.

See full Product Information for administration instructions.

OVERDOSAGE: Acute overdosage of up to 20g without significant ill effect has been reported. Management see full Product Information.

PRESENTATION: 800mg cimetidine - pale green tablets, 30s - effervescent white tablets, 2 tubes of 15 per pack; 400mg cimetidine - pale green tablets, 60s; 200mg cimetidine - pale green tablets, 120s; 200mg/2mL cimetidine hydrochloride injection solution, 10s.

MANUFACTURED BY: SmithKline Beecham (Australia) Pty Ltd ACN 004 170 932, 300 Frankston Road Dandenong, Victoria 3175.

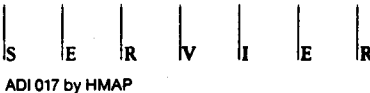
* Please note change(s) in Product Information

For further information consult the Product Information in MIMS Annual or PP Guide, or contact SmithKline Beecham (Australia) Pty Ltd. 21 February 1992



ABRIDGED PRESCRIBING INFORMATION

NAME: Adifax (dexfenfluramine hydrochloride). INDICATIONS: Simple, complicated or refractory obesity unresponsive to non-pharmacological treatments. MODE OF ACTION: Adifax has its effects on feeding behaviour via alterations in brain serotonin levels. WHILST THE EXACT PROCESSES RESPONSIBLE REMAIN UNCLEAR, ADIFAX STIMULATES THE RELEASE AND INHIBITS THE RE-UP TAKE OF SEROTONIN IN THE CENTRAL NERVOUS SYSTEM. ADIFAX MAY INFLUENCE THE DIETARY COMPOSITION OF FOOD INTAKE. UNLIKE AMPHETAMINE AND ITS DERIVATIVES, ADIFAX DOES NOT CAUSE STIMULATION OF THE CENTRAL NERVOUS SYSTEM AND DOES NOT HAVE ADDICTIVE POTENTIAL. CONTRAINDICATIONS: Glaucoma, history of psychosis, severe affective disorder, known alcohol or drug abuse. CONCOMITANT USE WITH CENTRALLY ACTING APPETITE SUPPRESSANTS, L-TRYPTOPHAN AND MAOI'S (A 2 WEEK INTERVAL AFTER STOPPING MAOI'S AND APPETITE SUPPRESSANTS IS NECESSARY). WARNING: There are conflicting reports that fenfluramine can cause toxic effects on serotonergic nerve terminals in the brains of rats and guinea pigs. THESE EFFECTS HAVE NOT BEEN CONSISTENTLY REPORTED. REFER TO FULL PRESCRIBING INFORMATION FOR FURTHER DETAILS. PRECAUTIONS: Treatment duration should be limited to three consecutive months. Adifax may potentiate the action of anti-hypertensive, anti-diabetic, sedative and anti-depressant drugs including L-tryptophan. Adifax is not recommended during pregnancy (category B3) or lactation. ADVERSE REACTIONS: The most common side-effects include: Drowsiness, dizziness, somnolence, asthenia, depressed mood, dry mouth, polyuria and diarrhoea. THESE ARE USUALLY MILD, TRANSIENT AND RARELY REQUIRE CESSATION OF TREATMENT. ABRUPT CESSATION OF TREATMENT WITH ADIFAX MAY BE ACCOMPANIED BY SYMPTOMS OF TRANSIENT DEPRESSION. DOSAGE AND ADMINISTRATION: The dose of Adifax is one 15mg capsule in the morning and evening (15 mg b.d.). Weight loss becomes apparent after 2 weeks and continues during treatment. The dose should not be exceeded. Adifax is not recommended for children. PRESENTATION: Packs of 60 capsules. NAME OF MANUFACTURER: Servier Laboratories (Australia) Pty. Ltd., 13 Cato Street, Hawthorn, Victoria 3122. Refer to full product information before prescribing.

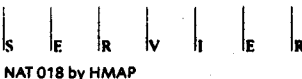


ADI 017 by HMAP

Natrilix

ABRIDGED PRODUCT INFORMATION:

NAME: Natrilix (indapamide hemihydrate). INDICATIONS: Essential hypertension. CONTRAINDICATIONS: Anuria, progressive and severe oliguria, hepatic coma. Known hypersensitivity to indapamide or sulfonamide derivatives. WARNINGS: A larger dose than 1 tablet (2.5mg) of Natrilix results in electrolyte disturbances becoming significant consequent to diuresis without appreciable additional antihypertensive effect. The maximum recommended dose is therefore 1 tablet daily. Hypokalaemia or hyperuricaemia may occur with Natrilix at all doses though gout has rarely been reported. PRECAUTIONS: Electrolytes, blood urea nitrogen and uric acid should be assessed during therapy. Special caution should be exercised in patients with severe hepatic disease. When Natrilix is given with other non-diuretic antihypertensive agents, the effects on blood pressure are additive. Orthostatic hypotension may occur and be potentiated by alcohol, barbiturates or other antihypertensives. The use of Natrilix is not recommended during pregnancy or lactation, and safety and effectiveness has not been established in children. Natrilix should not be used with a diuretic since the combination may produce hypokalaemia and hyperuricaemia. ADVERSE REACTIONS: The most common adverse effect is electrolyte imbalance and some 6% of patients may need potassium supplements. CNS side effects, including asthenia, headache, dizziness and vertigo have occasionally been reported. For uncommon side effects consult full Product Information. DOSAGE AND ADMINISTRATION: One Natrilix tablet (2.5mg indapamide) daily taken in the morning. Since higher doses do not appreciably increase antihypertensive efficacy but do provoke a diuresis, the dose should not be exceeded. Natrilix is compatible with other antihypertensive agents, but should not be used with a diuretic. PRESENTATION: Packs of 2.5mg tablets in a single blister strip of 30. (N.H.S. General Benefit of 60 tablets plus one repeat.) Manufacturer: Servier Laboratories (Aust.) Pty. Ltd., 13 Cato Street, Hawthorn, Vic, 3122, Australia. ACN 004 838 500



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ical activity. It is possible for a sedentary individual to have a BMI within grade 0, but nevertheless to have a raised fat mass and a lowered lean mass. Conversely, an active sportsman may have a raised BMI because of a raised lean body mass.

Recently it has become obvious that the distribution of body fat is as important a risk factor as its total amount.

Abdominal fat increases risk for total mortality, coronary artery disease, hypertension and non-insulin dependent diabetes mellitus. It is most easily assessed clinically by measuring and comparing body circumference around the waist and the hip. A ratio greater than 0.9 indicates the presence of abdominal obesity.

Nutritional assessment in a patient with obesity, involves the following processes:

- define the problem by measurement of body mass index and body circumferences;
• assess the evolution of the problem in this particular patient;
• determine the motivation of the patient for seeking help in the management of his or her problem;
• identify changeable behaviours in relation to food and physical activity.

Diagnosis

Once assessment is complete, it is possible to state a formal diagnosis, for example: "Obesity grade II with a major contribution from physical inactivity, and with a component of the weight increase due to binge eating and winter depression".

Management

Management of obesity involves the following steps:

- 1. Establish short and long term goals, which will involve 'contracts' by both the patient and the physician.

2. Consider endpoints other than weight change as management outcomes. These can include:

- a change in body composition, as evidenced by a change in fat distribution that may occur even without a large change in total weight;
• adoption of lifestyle changes, such as cessation of cigarette smoking or by increasing physical activities;
• change food intake in directions known to be associated with less risk;
• understand and develop changes in food purchasing and preparation habits.

References

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Further reading

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Reprint requests

Professor M Wahlqvist
Department of Medicine
Monash University
Monash Medical Centre
Melbourne