
Liverpool Handbook of Geriatric Medicine

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This handbook is intended as an aid to medical student studies as part of the overall geriatric medicine course in Liverpool. It is not intended to be used as a substitute for consulting a doctor.

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Fundamental Principles

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Fundamental principles

- Giants of Geriatrics: immobility - patient takes to bed/chair; incontinence - secondary to immobility/non-urological disease; instability - falls \pm trauma; intellectual impairment - acute or acute on chronic
 - Atypical presentation - classical syndromes only in the young
 - Multiple pathology - patients have 6 different pathologies
 - Polypharmacy as a dangerous consequence of (iii) above
 - Multidisciplinary team approach involving most health professionals
 - Rehabilitation from day one to prevent functional decline
 - Discharge planning and objective setting to engender rapid return to community
-

In addition students should have some grasp of the demography, biology and psychology of ageing and the broader aspects of health service provision. Emphasis should be placed on community aspects.

Students should be able to:

- Take a comprehensive and thorough medical history and perform a full and detailed clinical examination combined with a pre-morbid functional assessment
- Appreciate the social context of disease affecting the elderly and how patients may present as a "social crisis".
- They need to develop communication skills so they can cope with confused elderly patients and/or relatives and learn the techniques of communicating with people who are visually and auditory impaired
- Be able to sympathetically make a behavioural and affective evaluation
- Plan appropriate investigations and interpret results
- Formulate a plan of management which would include the acute medical event and appreciate the need for pre-discharge planning
- Institute a full functional rehabilitation program as necessary
- Understand the involvement of other health care professionals and appreciate their contributions
- Contribute to case conferences and planning the management of both in-patients and out-patients (day hospital)
- Appreciate the need to keep not only patients informed but also their relatives and other carers whilst maintaining patient autonomy/confidentiality. This includes communication with community professionals
- Be aware of the basic aids and appliances, how to choose between them and how they can be delivered to a patient
- Appreciate what Social Services support is available in the community, including personnel (district nurses, community psychiatric nurses, general practitioners, home helps, shoppers etc) and the financial support mechanisms including various welfare benefits

Problem solving:

- Plan the discharge of patients with appropriate support services if required
- Manage elderly patients in non-medical/geriatric settings such as the orthopaedic ward, the Accident and Emergency Department, the post operative period etc.
- Debate ethical issues concerned with old age and geriatric medicine
- Appreciate problems of confidentiality and patient's autonomy
- Organise approaches to terminal care in hospital/hospice/community

Attitudes:

- Develop confidence with their approach to elderly patients who are often very ill, frightened and may be terminal
- Deal with prejudicial attitudes towards elderly patients and people
- Appreciate and acknowledge elderly patients religious and other spiritual requirements

Knowledge base:

- Appreciate that care of the elderly requires a sound foundation in good clinical medical practice
- Appreciate that the gains to be achieved from treatment of elderly people do not diminish with increasing age of patient - in fact the opposite may apply

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The ageing process and its clinical implications

"Old age never harmed anybody" Lye, 1978

The difficulty of defining ageing in biological terms and in particular as far as the human is concerned, is due to the interaction between physiological ageing and age-related disease(s). Most of what we see as ageing is in fact the accumulation of minor or major disease(s).

We therefore have to apply a number of criteria which will help us to differentiate true ageing from age-related disease.

Ageing is: universal, deleterious, progressive, intrinsic, irreversible.

At all levels, biological, chemical, even nuclear, we are reacting to "random events" occurring within the environment. By random chance alone one of these reactions is going to be harmful and the longer the organism has been exposed to risk the more likely it is that any interaction will have scored a "detrimental hit". This is reflected in the human condition by the increasing prevalence of pathology in older people who have been exposed to risk longer than younger people. Thus apart from specific congenital childhood diseases and the self-inflicted diseases of road traffic (motorcycle) accidents and drug abuse, most disease states in humans and indeed in all other mammals increase in prevalence with increasing age.

The impact of disease or trauma causes more harm the older the organism is at the time of the crisis. This increased impact is due directly to impaired homeostatic responses and the delay in responding to perturbation of the normal equilibrium state by older people. In the older organism this is due to age effects on primarily the nervous and autonomic systems, the cardiovascular system and on renal function. In the very old there may even be a domino effect across body systems. The moral is clear. If you are going to have a heart attack or develop pneumonia it is best done when you are young - it will have less effect on the whole body, will be less disabling, recovery should be quicker and overall you are more likely to survive.

The knock-on indirect effects of disease/trauma in elderly subjects are often not fully appreciated by the patient, relatives or even by clinicians. The secondary complications of disease and trauma in the elderly organism are much more devastating than in the young individual. If the elderly do have a specific disease unique to them it is the disease called dependency.

"I do not want two diseases - one nature made, one doctor made" Bonaparte, 1820.

Bed is dangerous for all and unfortunately is more dangerous the older we are. Putting fit young people to bed leads to many problems including death. Blood clots in legs may travel to the lung causing collapse, ascending infection of the kidneys may cause renal failure and hypostatic pneumonia is particularly common. Muscles waste and bones thin. Bed-rest is rather like prolonged space flight - not for the faint hearted nor the unfit. Remember the early astronauts trying to stand on the aircraft carrier decks in the 1960s on their return from space flight. They couldn't maintain blood flow to the brain and fell over. The complications and sequelae of bed rest occur more rapidly, are more frequent and more severe by an order of magnitude in elderly patients.

"How to kill your aunt, inherit and live happily ever after" "Bodkin Adams, 1958" In addition to "medical" complications is the major functional complication of dependency. We all like to be waited on and the elderly patient is no exception. If they feel unwell in bed and worse out of it then they will not mobilise. Wonderful caring nurses reinforce this and we are likely to end up with a "cured" patient totally unable or unwilling (usually the former) to do anything for themselves. The patient is on a downward spiral of increasing dependency. At the very least dependency will prolong hospital stay by up to several weeks. If dependency is allowed to develop and is not corrected rapidly it will become permanent and the patient will require long term institutionalisation.

All sick elderly patients require active rehabilitation from day one of their acute illness. Geriatricians and in particular nurses working on active geriatric units, realise the importance of this. It is often more important to maintain an elderly patient's independence than it is to treat

their electrocardiogram. Preferably both need to be addressed. This demands a high degree of skill, particularly amongst nursing staff, and also some courage. Strict discipline and a military background helps! It may be worth taking a leaf out of NASA's book and provide some form of artificial gravity for our elderly patients confined to bed. Perhaps we should put hospital beds on the end of a centrifuge!

"The old man wheezes, not because he is old but because he has bronchitis, asthma etc." Lye, 1980.

There is no such disease as senility. The elderly are afflicted by the same diseases as younger people. In particular, diseases affecting the joints and cardiorespiratory systems are prominent. These diseases may be acute - self limiting and we can cure them or - chronic -prolonged and we are unable to cure them.

Causes of being housebound in patients of more than 65 years of age:

Cause	%*
Arthritis	36.2
Cardiovascular	27.0
Pulmonary	17.2
Cerebrovascular	14.9
Visual	14.4
Trauma	9.8
Psychiatric	4.6
Miscellaneous	29.9

*Some have more than one condition

Apart from diseases being more severe, more prolonged and prone to secondary complications (dependency) in old age they also give rise to other problems from the doctor's point of view:

- Multiple pathology - old patients cheat
- Atypical symptoms - old patients have not read the textbooks
- Polypharmacy - doctors cause confusion with multiple drugs
- Lack of homeostatic reserve - old patients have the effrontery to be frail

The ageing process is important in human medicine but only becomes highly significant when combined with disease/trauma. A single disease in an old person may in itself give rise to little in the way of dependency problems. It is the complication of disease in old age, which leads to problems for our health service. It is the prevention and management of these complications, in addition to medical treatment, which is the core of Geriatric Medicine as a medical specialty.

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Presentation of ill health in old age

Unlike medical students, elderly patients have not read in detail the medical text books and have not learned how to present their acute illnesses in a classical fashion. Equally, many of them have the effrontery to have more than one disease. It is not uncommon for elderly patients to have up to six separate pathological conditions. All these conditions interact and the waters may be muddied still further by the contribution of polypharmacy from well-meaning doctors. Because old age impairs homeostatic mechanisms, even minor perturbations may have a dramatic effect on an elderly person's equilibrium.

Thus elderly patients with acute illnesses present with functional disability;

- Immobility
- Instability
- Incontinence
- Intellectual impairment.

These are the Giants of Geriatrics (Isaacs, 1976).

Not one of these giants is caused by old age. The giants are the result of pathology. These conditions must be very carefully sought and, as far as possible, remedied. If they cannot be remedied their effects can be ameliorated thus restoring functional capacity to the patient and restoring independence.

The older patient often presents as a "social problem" or as a "social crisis". Before attributing a patient's breakdown as being due to social problems, the student and the doctor should deduct 40 years from the patient's age and think again. Thus 40 year old ladies who present to the A&E Department with recurrent falls are admitted to hospital for intensive investigations even though they may live on their own. The investigations would cover the full diagnostic work-up of history taking and physical examination, routine screening investigations and special investigations which might include 24 hour ECG monitoring, exercise testing, postural stress testing, EEGs, CT scans etc. etc. Pity then the poor old lady of 80 years, also living alone, who presents in a similar fashion and is referred to the social worker for a cure!

This is not to say that social crises do not occur in old age, they certainly do, but they are invariably precipitated by pathology, not by social inadequacy. People who are socially inadequate rarely survive into old age and certainly would have been unable to have withstood World Wars, the depression and the social and technological changes which have occurred in the United Kingdom over the century.

In addition, many elderly patients suffer from diseases causing chronic impairment. These particularly are likely to effect the major faculties so that patients may have visual impairment or hearing difficulty. This may not be apparent on first meeting the patient and because patients try to be helpful their responses to detailed questioning may be inappropriate and ascribed by the inexperienced student or doctor to the patient's failing mental ability. Equally, many elderly patients, particularly on admission to hospital, are very frightened. The history taker who can constantly reassure his elderly patient will gain much more information as well as going a long way towards helping the patient. The lesson is clear, make sure that the patient can see you, can hear you and is not suffering from an acute anxiety or panic attack to which you are contributing. Do not be afraid of holding the hand of a distraught patient of any age. You yourself are a very strong placebo and hopefully have very few adverse effects which is more than can be said for many of our powerful modern pharmaceutical remedies.

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What to emphasise in history and examination

Introduction

The history and examination is the same as for the young but there are a few important differences. Multiple pathology is common in the elderly and it is even more important that "no stone is left unturned". It is not only important to diagnose and treat disease in the elderly but it is essential that you are aware of their social circumstances and support structure so that you can be sure that they will attain a good quality of life in the community and therefore the social history is even more important in this age group. The history will point to a differential diagnosis in most instances and the examination will confirm your suspected diagnoses and may even uncover some new ones.

Clinical history

This must include the following:

- **Age**
- **Presenting complaint**
- **History of presenting complaint**
- **Systemic enquiry:**
 - **cardiovascular system**
 - chest pain; exertional dyspnoea, orthopnoea; paroxysmal nocturnal dyspnoea; palpitations; ankle oedema
 - **respiratory system**
 - cough; dyspnoea; wheeze; sputum; haemoptysis
 - **gastrointestinal system**
 - appetite; abdominal pain; weight loss; bowel habit; dysphagia; nausea/vomiting
 - **genito-urinary system**
 - frequency; dysuria; haematuria; nocturia; age at menopause; postmenopausal bleeding
 - **locomotor system**
 - joint swelling; joint pain; morning stiffness
 - **central nervous system**
 - loss of consciousness; weakness; speech abnormalities; fit; headaches
- **Drug history**
- **Allergies**
- **Past medical history:**

Significant illnesses e.g. diabetes, myocardial infarction, hypertension, operations, rheumatic fever, scarlet fever
- **Family history:**

Ask about inherited diseases e.g. diabetes, thyrotoxicosis, ischaemic heart disease

- **Social history:**

- Marital status.
- Who do they live with?
- Where do they live? e.g. house/bungalow/flat.
- Do they have to climb stairs?
- Activities of daily living? e.g. bathing.
- Support services e.g. home helps, meals on wheels
- Finances - allowances e.g. attendance allowance, mobility allowance
- Pets
- smoking
- alcohol

Clinical examination

General:

Does the patient look ill? Nutritional status - look for evidence of significant skin disease e.g. pressure areas, tumours

Hands:

Finger clubbing; splinter haemorrhages; koilonychia; leuconychia; palmar creases for pallor; Dupuytren's contracture; liver palms

Face:

Conjunctivae - anaemia, jaundice. Mouth and lips - anaemia, central cyanosis

Orientation

Evidence of confusion. Formal test with Mini-mental State Examination or AMTS

Cardiovascular system

Pulse: rate, rhythm, volume, character, arterial wall Jugular venous pressure (JVP: patient positioned at 45 degrees = 3 cm vertically from the sternal angle.

Chest:

Observe for scars, apex beat (position and character), thrills in all areas, parasternal heave, auscultate (heart sounds and added sounds) Others: peripheral and sacral oedema, crackles, peripheral pulses, hepatomegaly, blood pressure, fundoscopy

Respiratory system

Inspection:

chest expansion, respiratory rate, abnormal appearances and scars

Palpation:

chest expansion, tactile vocal fremitus

Percussion:

normal, decreased, stony dull, hyper-resonant

Auscultation:

breath sounds (decreased, vesicular sounds, bronchial sounds), added sounds, crackles, wheezes

Gastro-intestinal system

Inspection:

Look for distension, abnormal masses and scars

Palpation

Ask the patient if he/she has any painful areas before palpating the abdomen. Your humerus and forearm must be parallel to the abdominal wall which requires you to crouch down for this examination. Palpate beginning in the right iliac fossa and circling the abdomen clockwise. Start with light palpation and then deep palpation. Then palpate for each of the organs individually. This should also include palpating the hernial orifices and a rectal examination. The latter should not be done routinely unless instructed by medical staff.

Percussion:

Across areas of the abdomen e.g. masses and organs, shifting dullness for ascites

Auscultation:

Listen for bowel sounds (increased, decreased, absent), bruits (over a hepatoma, stenosed renal artery, abdominal aortic atheromatous area)

Central nervous system**Cranial nerves**

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Peripheral Nervous System

Examine all limbs for tone, power, coordination, sensation, vibration and position sense

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Theories of ageing

Introduction

The fundamental biological problem which all theories of ageing are seeking to explain was stated very elegantly in 1957 by Williams when he wrote, "It is indeed remarkable that after a seemingly miraculous feat of morphogenesis, a complex metazoan should be unable to perform the much simpler task of merely maintaining what is already formed".

The difficulty in attempting to establish an understanding of that ageing is that it is not a single physiological process. It is multifaceted and hierarchical in its expression with subtle changes occurring simultaneously at the molecular, cellular, tissue and organ levels. The considerable heterogeneity that characterises many species, particularly humans, and the complexity of environmental interactions and polygenic controls, results in an enormous phenotypic variability being associated with ageing. This variability is frequently confounded by the symptoms of underlying pathology and invariably increases between individuals with ageing.

It is therefore necessary to extract the essential attributes of the ageing process from this phenotypic complexity as a foundation on which to base an understanding of the biochemical processes involved. Strehler has attempted this and has identified four criteria to define the ageing process and to distinguish it from chronic pathology. Are these characteristics sufficient and adequate to distinguish ageing from morbidity?

Attributes of the Ageing Process

- Universal - the change occurs in all older members of the species;
- Intrinsic - ageing is a process which occurs even when all environmental influences are eliminated.
- Progressive - the onset of the process is gradual and the change accumulative.
- Deleterious - the change must shorten life.

Biochemical Theories of Ageing

In the scientific literature there are published over 300 theories seeking to explain the biochemical mechanisms of ageing. What conclusions can you draw from this statement? It is necessary and possible to simplify the issue by categorising the many disparate theories into two basic types; self-destructive genetic programme, or random chemical damage to proteins and DNA reducing the metabolic efficiency of the cell. Other schemes can also be used to provide a rationale approach to the literature. What other schemes can you suggest that could be used to categorise the theories of ageing?

The Program Theory of Ageing

The central idea of the programmed theory is that ageing is the result of a sequence of events encoded in the genome, just as the developmental sequence is controlled by gene expression. Ageing is considered to be programmed into the genome and genes exist whose function, when expressed, is to kill the organism. Clearly individual species have characteristic life-spans, and within a species genetically determined differences in life-span occur. It is assumed in this theory that there is some genetic program that determines the maximum life-span for each species. It has been suggested that the process of ageing and senescence may be under neural and endocrine control in a manner similar to development, growth and maturation.

Can you construct an argument from the available data in the literature to support/contradict this proposal?

Your approach to resolving this question could consider the following:

1. Studies on longevity in monozygotic and dizygotic twins, (McGue et al. 1993). In the classic study of Kallman and Sander, they reported that in a group of 59 pairs, the mean intra-pair differences in life-span for mono-zygotic twins was 36.9 months while in dizygotic twins it was 78.3 months. This finding was confirmed by later studies. This data has been reviewed by Jacquard (1982) who noted that although the difference in the life spans of monozygotic twins (36.9 months) is significantly less than the difference in the life spans of dizygotic twins (78.3 months), these differences decrease with age and finally disappear towards 80 years of age. Thus the contribution of genetic heterogeneity to the observed variability in life span seems to operate only during the early stages of life and strongly decrease with age.
What interpretation can you offer to explain this data?
Have recent studies confirmed or contradicted this interpretation of twin studies? What is the estimated contribution of genetics to eventual survival in the human?
2. Although human life expectancy has increased throughout recorded history, the rate of ageing has remained constant. What justification is there for this statement and if correct, does this statement support or contradict the program theory of ageing?
3. The program theory of ageing has been given some support by the work of Hayflick and others in studying cell mitotic potential under culture conditions. Prior to 1961 it was thought that animal cell populations could be propagated, apparently indefinitely in vitro. It is now known that normal cells derived from almost all vertebrate and most insect tissue can be cultured only for a finite number of generations. This has been interpreted as support for a programmed theory of ageing. Explain why you agree/disagree that this observation is evidence for a program theory of ageing (see Hayflick 1985).
4. Ageing may arise because of the pleiotrophic effect of some genes, i.e. genes may have more than one phenotype or be expressed at different times in the life span. Genes which are detrimental to survival will be eliminated from the genome if they are expressed early in the life span, but genes with detrimental effects late in the life span may not be eliminated so readily from the genome. Why should this be so? It is proposed that the total effect of detrimental, late acting genes may underlie the ageing process. Is there any evidence to support this idea, (see (Williams 1957; Partridge and Barton 1993).

Random Chemical Damage and Information Transfer Theories.

These theories which are based around the idea of 'genetic damage and impaired information transfer', focus attention on the process of information transfer from DNA to its metabolic expression in the cell. Central to these theories is the idea of 'incomplete repair' of metabolic damage resulting in the accumulation of chemical or structural modification of cellular proteins or DNA with time. A number of theories are present in the literature which are based on the observation that biochemical repair processes are not totally efficient and that with time random chemical damage will accumulate. This is damage which has escaped the normal turnover processes and repair capacity of the cell.

The Somatic Mutation Theory

The central concept of this theory is that if somatic cells accrue sufficient mutational damage in their DNA then physiological decrements characteristic of ageing will occur. (See (Partridge and Prowse 1994). Based on the available data would you consider this theory a credible idea?

Error Theory - Protein Synthesis

Lesley Orgel, when working at the MRC Mill Hill Genetics Division, proposed a model to explain biological ageing based on the fidelity (accuracy) of protein synthesis. Ageing is postulated to result from errors introduced in the process of information transfer from DNA

through transcription and translation to the functional protein. Such errors are inevitable and most are likely to be unimportant but others may lead to further errors, i.e. mis-specified RNA polymerises. Errors induced in the proteins controlling transcription and translation could amplify the original error to the point where the cell was no longer viable. Such a mis-specified critical enzyme could initiate an 'error catastrophe'. What evidence is there to support the contention that the accuracy of protein synthesis changes with ageing? You may wish to read Sharma (1988) and (Harley, Pollard et al. 1980).

The Free Radical Theory

Free radicals are highly reactive atoms or molecules in which an electron pair has been separated into two electrons that exhibit independence of motion. They are capable of initiating a chain reaction with stable molecules to generate more free radicals. Free radicals can pose a considerable hazard to biological systems because of their unique chemistry. The highly reactive nature of free radicals and their generation in all cells would lead to widespread damage, particularly in the mitochondrial and microsomal membranes which contain oxidative enzymes, if protective enzyme system were not present.

Further damage has been envisaged to occur by oxidation of collagen, elastin, DNA, mucopolysaccharides, lipoproteins and proteins in general. Some of the products of free radical chemistry are completely foreign to the repair or turnover enzymes of the cell, i.e. the cross-linked product of two proteins may be resistant to the proteolytic enzymes responsible for protein degradation and protein turnover. This would lead to the accumulation of age pigments within cells.

This theory has attracted much attention because of its universal application to all cells to explain random and diverse types of damage. Free radicals are known to be involved in certain pathological states; cancer, cardiovascular disease and some diseases of the central nervous system. How has this theory been evaluated in animal experiments? What conclusions can you draw from these studies? These experiments often suffer from the effect called 'confounding', what does this mean?

Summary

These short notes should be seen as a guide into the complex and confusing literature in this field, and as an aid to developing a critical approach to the interpretation of this data.

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Demography of ageing (U.K.)

Remarkable changes in the age structure of the population have occurred over the last century for two main reasons: (1) reductions in fertility and (2) declines in infant mortality (not from better health care but from socio-economic development). The result of this has meant that the number of old people have risen from about 2 million in 1901 to around 10 million in 1991.

Old age may last as long as 20 or 30 years and thus it is helpful to split the elderly up into smaller age groups: the young old 65-74, the old old 75-84, and the oldest old 85+. The social and biological characteristics of people in each of these age groups is sufficiently different to make these categories meaningful. For example, the young old are recently retired, tend to be wealthier than other elderly people and have a good social network. By contrast the oldest old were born at the turn of the century, many of the women never married or were widowed young during the world wars, they went through the economic slump of the 1930's and tend to live in old unmodernised houses. They are much more likely to be disabled by physical and mental illness and to live alone. Furthermore, they have a very poor social network and are not as well off as the young old.

Most old people want to live in their own home and fortunately the vast majority do. 96% of elderly people live in the community and only 4% in institutions. The Community Care Act allows elderly people to live in their own homes for as long as possible.

The services that are offered to old people fall into 6 main categories:

1. Informal carers - these are made up of relatives and friends who can help in everyday tasks, anything from personal hygiene to financial help.
2. Local authority social services - these services include home helps, meals on wheels, respite care, district occupational therapy and Day Centres. They also provide residential part III homes.
3. Local health services - this includes the family practitioner who helps with treatment of any illness or screening the over 75 year olds. It includes hospital beds and home nursing as well as out-patients etc.
4. Independent services - these are private or voluntary sector services and include private nursing homes and limited home care services. The voluntary services include good neighbourhood schemes, age concern projects, crossroads, luncheon clubs and day centres.
5. Local housing department services.
6. Central government department of social security.

The major change with the community care legislation (1993) was that the local authority social service departments would take the leading responsibility for planning and monitoring of care for the elderly and disabled people. It was intended that the independent sector should take a greater part in providing basic services for these people. A further change was that individual clients would be individually assessed and have a care plan and services provided for them to match their needs, these of course would be monitored regularly.

Sheltered accommodation: this is characterised by purpose built bungalows or flats which are served by a resident Warden or a paid protectorate Warden with a radio pager. They had been mainly built by local authorities or housing associations but private schemes are becoming increasingly common. Tenants rarely move and therefore old schemes can cause much stress and strain on the Warden because of their old and frail occupants. Sheltered accommodation is very attractive to active elderly couples and they also need to be suitable for disabled or single persons. Elderly people are often encouraged to apply for sheltered accommodation by their relatives who feel that their responsibilities and obligations have been fulfilled. Some residents often make heavy demands on supporting services. On the whole the Warden alone is rarely sufficient and officially is only available to summon help when required, however, many Wardens often do much more.

Institutional care:

This is provided in 3 main settings - local authority residential homes (Part III homes), the hospital long stay wards and the independent sector (private residential and nursing homes). In general Part III homes and residential homes will take clients who are independently mobile (with or without mobility aids) who can dress themselves and who are continent of urine and faeces. Some will not take patients with dementia syndromes if they are associated with behavioural problems. Residential homes are not staffed by nurses but by care attendants and do not have sufficient staff or equipment to look after very dependent people. In contrast, nursing homes must have at least one trained nurse on the staff but in practice the bulk of the care is provided by untrained care assistants. A major difference between long stay hospital care and other institutionalised care is that the hospital care is free. Consequently there can be a conflict in deciding who should have a free hospital bed and who should have to use their own resources.

Reasons for institutionalisation:

Severe physical disabilities, immobile without help, severe mental disabilities, constant supervision needed, passive dependent personality, hostile community or non-existent community support, unpredictable and frequent care needs. Institutionalisation however does have some complications: depersonalisation, marked restriction of choices and accelerated dependence.

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 - c. Intestine (relaxation)
 - d. Liver (glycogenolysis)
2. ALPHA-2
 - a. Lipolysis (decreased)
 - b. Renin (reduced)
3. BETA-1
 - a. Heart
 - b. Inotropism
 - c. Chronotropism
 - d. Refractory period
4. BETA-2
 - a. Muscles (vasodilatation)
 - b. Bronchioles (dilatation)
 - c. Uterus (relaxation)
 - d. Intestine (dilatation)
 - e. Liver (glycogenolysis)

NB – subject to “age-change

The autonomic nervous system (ANS) is both the monitor and regulator of the ‘milieu interieur’ and as ageing is associated with the inefficient maintenance of body homeostasis, particularly under stress, it has been assumed that many aspects of normal ageing are mediated via the ANS (Table I). It is, however, quite difficult to pick out specific age lesions in the ANS. As far as the parasympathetic nervous system is concerned the changes are slight. Alternatively, the sympathetic component has been studied in relation to increasing age and though some of the studies are conflicting a consensus is emerging. There is a basic problem however in interpreting many of these studies which have failed to differentiate between actual changes in receptors and physiological age changes in end organs. Alpha 1 receptors are little effected by increasing age but are effected by disease. Studies of age effects on alpha 2 receptors are inconsistent. Beta 1 and beta 2 receptors are less active probably at a post-receptor level involving adenylate cyclase and cyclic AMP dependent protein kinase. Beta receptors have a diminished ability to up regulate with increasing age whereas down regulation is unaltered.

There are two important syndromes where age-related dysfunction of the ANS is thought to be an aetiological factor. Additional pathological factors are equally important. These two syndromes of postural hypotension and accidental hypothermia are confined to the very young and very old.

Postural hypotension

Approximately 1 in 5 elderly people will show an abnormal fall in systolic blood pressure (of more than 20 mmHg) on moving from the supine to the upright position. Whether this fall in blood pressure is accompanied by symptoms or not tends to vary from individual to individual and within individuals at different times. The decrease in cerebral perfusion may lead to dizziness, lightheadedness or frank loss of consciousness. Repeated falls may cause direct trauma but also lead to psychological loss of confidence causing immobility and prolongation of rehabilitation.

Many factors both underlie the syndrome and act synergistically with it (See below). Drugs in particular are often contributing factors.

Aetiology of postural hypotension

1. ENVIRONMENTAL

- Immobility/recumbency
- Hot weather/ bath /bed
- Exercise
- Post-prandial
- Altitude

2. CARDIOVASCULAR

- Myocardial infarct
- Arrhythmia
- Mitral valve prolapse
- Aortic stenosis
- Myxoma

3. BLOOD VOLUME

- Dehydration
- Hypokalaemia
- Haemorrhage
- Hypnoatraemia
- Anaemia (B12)
- Varicose Veins

4. PHYSIOLOGICAL

- Cough
- Micturition
- Defaecation
- Isometric exercise
- Rectal examination
- Pyrexia

5. ANTIHYPERTENSIVES

- Ganglion-blocking agents
- Diuretics
- Alpha methyl dopa
- Calcium channel antagonists
- ACE-inhibitors
- Prazosin
- Beta-blockers
- Reserpine
- Glyceryl nitrate

6. SEDATIVES

- Phenothiazines
- Barbiturates
- Tranquillisers
- Tricyclic antidepressants
- Antihistamine

7. METABOLIC

- Diabetes mellitus
- Hyperthyroidism
- Hypoadrenalism
- Hypopituitarism

8. MISCELLANEOUS

- Insulin
- Anti-Parkinson agents
- Alcohol

Management

It is rare that detailed neurological, neurohumoral and cardiovascular investigations help in the management of the syndrome. The history and physical examination usually outline a commonsense management approach. The avoidance or removal of precipitating factors, particularly drugs, is very important. Modification of lifestyle by encouraging slow postural changes can be helpful. In severe cases, physical measures including waist length elastic support (TED) stockings, are of great benefit.

A final physical measure would entail elevating the head of the bed at night which to some extent down regulates cerebral blood flow. It also prevents the nocturnal diuresis which occurs in these patients.

Finally drugs may be tried. All drugs lead to supine hypertension and/or cardiac failure. Non steroidal anti inflammatory agents such as flurbiprofen, perhaps combined with low dose fluid retaining fludrocortisone would be the first approach. Fludrocortisone precipitates heart failure usually before it relieves the postural symptoms. Xamoterol, a beta 1 partial agonist, may be quite helpful and has minimal side effects. Occasionally some patients respond to dopamine antagonists such as metoclopramide.

Accidental hypothermia

Aetiology of hypothermia

1. IMMOBILITY

- Parkinson's
- Stroke
- Dementia

2. SUBNUTRITION

3. METABOLIC

- Hypoglycaemia
- CO poisoning

4. PATHOLOGICAL

- Chest infection
- Myocardial infarct

5. DRUGS

- Alcohol
- Phenothiazines
- Antidepressants
- Hypnotics
- Barbiturate

In any consideration of thermoregulation it is important to differentiate between the inner core and the more usually recorded peripheral temperatures. Maintenance of a core temperature of 37°C is vital for metabolic processes. Core temperature can be measured with a low reading rectal thermometer. The peripheral temperature is that temperature recorded directly from skin surfaces. In a cold environment, the difference between the two is a measure of thermal insulation. If a core temperature drops below 35°C the individual is hypothermic. 1% of healthy elderly people have impaired thermoregulation leaving them at risk of developing hypothermia in a cold environment. The reason old people develop hypothermia is related to changes in the ANS impairing heat conservation by the skin and failure to increase heat production by metabolic processes. However, as always in the elderly, the aetiology of accidental hypothermia is usually multifactorial and other precipitating or contributing factors always need to be considered (Table III). For diagnosis a high index of suspicion is required as hypothermia may develop in a seemingly quite warm environment. If in doubt the rectal temperature must be recorded. All community nurses should be equipped with such thermometers and should use them.

Mild Core = 35- 32°C the patient may be asymptomatic. They usually have a cold periphery particularly of the anterior abdominal wall. The skin is pale but shivering may not be apparent. The blood pressure and pulse rate are usually elevated. Patients may have been incontinent because of a brisk diuresis due to increased renal blood flow.

Moderate Core = 32-28°C tachycardia is replaced by bradycardia and the blood pressure falls. Arterial thromboses occur due to increased blood viscosity. The skin is still cold but now also shows generalised puffy oedema, particularly of the face and hands. The patient takes on the appearance myxoedema. Respiration is usually slow though there may be respiratory infection. The muscle tone is generally increased though the tendon reflexes are slow relaxing. The patient will be very lethargic and usually mentally confused. Cerebral blood flow will be decreased and the patient may become obtunded.

Severe Core = less than 28°C The chances of survival of an elderly patient with this degree of hypothermia is unlikely. The patient is usually comatose with dilated pupils, are areflexic and grossly hypotensive. The body itself is like a stone and is unable to conserve centrally generated core temperature. The hypothermia may protect against brain damage however so that it is always worthwhile trying resuscitation.

Management

Prevention is better than cure and it is important to maintain a warm environment and mobility of "at risk" elderly individuals. Removal of precipitating agents, particularly drugs is obvious. A high index of suspicion by all primary care team members during cold months is important. Treatment of a hypothermic elderly patient is a medical emergency. The patient should be wrapped in a space blanket to prevent further heat loss. The objective then is to raise the temperature by 0.5 °C per hour. Excessive cutaneous vasodilatation may lead to problems with hypotension and cerebral and cardiac perfusion. Gentle rewarming can be achieved by transfer to a warm environment combined with prevention of further heat loss. Cardiac monitoring is important as is maintenance of the airway and treatment of respiratory infection. If the patient has been immobilised because of hypothermia for some time it is highly likely that they are dehydrated and warm intravenous fluids will be necessary. Increased plasma viscosity may lead to various vascular complications including myocardial, cerebral and pancreatic infarction. Acute pancreatitis and diabetes mellitus are common complications. These should be treated in their own right.

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Cardiac Disease

Introduction

Cardiovascular disease is common in the elderly and affects 50% of those over the age of 65 years. It is the leading cause of death, the mortality increases with age, 65-74 years, death rate = 1,300 per 100,000; over 85 years death rate = 7,300 per 100,000.

Pathology and physiology of ageing

Pathology:

The weight of the heart is 0.5% of the body in the young. This increases to 0.8% in the elderly. It represents thickening of the ventricular myocardium, (L>R). There is increased fat, collagen and elastic tissue. There is also de-position of amyloid protein with increasing age and 50% of hearts over the age of 80 years have evidence of cardiac amyloidosis. Most of this is of a mild degree and located in the atria but 10% show massive involvement.

Deformities and thickening of the valve leaflets occur due to fibrosis and calcific deposits. About 20% of those over 65 years have aortic valvular calcification, calcification of the mitral valve is less common. The mitral valve is most commonly "floppy" due to mucoid degeneration of the valve's fibrous tissue and occurs in 8% of those over 50 years. The conduction system is involved in calcification and fibrosis which result in bradyarrhythmias with increasing age.

Physiology:

The resting heart rate is not affected by age. However, there is a decrease in the inherent rhythmicity of the SA node with ageing. There is a decrease in the mean maximum heart rate during exercise. There is no change in cardiac output (5.5.1/min) with age at rest or during exercise. There is an increase in systolic blood pressure with ageing but no change in diastolic pressure.

Hypertension

The incidence of stroke, coronary artery disease and congestive heart failure are all significantly greater in hypertensive elderly individuals than in normotensive elderly individuals. Hypertension can be defined as a blood pressure persistently higher than 160/90 (at least 6 separate readings). In 80% of cases hypertension is primary (essential) the rest being secondary to other diseases e.g. renal disease.

Old people do not tolerate high blood pressures better than their younger counterparts. The risk of subsequent cardiovascular or cerebrovascular disease goes from about 1 in 10 with a systolic blood pressure of 105 mmHg to approximately 1 in 3 at a systolic pressure of 195 mmHg. Elderly women experience a greater risk for overall mortality, cardiovascular mortality and cardiovascular morbidity than similarly aged hypertensive men.

Clinical studies have shown that treating blood pressure greater than 160/90 in patients up to 80 years of age is beneficial in terms of morbidity (stroke/congestive heart failure, transient ischaemic attacks and angina) as well as mortality from stroke and total mortality.

Cardiac arrhythmias

Supraventricular tachyarrhythmias:

Atrial fibrillation or atrial flutter is the commonest arrhythmia in the elderly. It is most commonly due to ischaemic heart disease but may also occur in association with thyrotoxicosis, pulmonary embolism, pneumonia and cardiomyopathy.

Bradyarrhythmias:

Sinus bradycardia (rate <60 per minutes), or heart block may reflect an underlying sick sinus syndrome, sinus node ischaemia or the effects of drugs, e.g. beta receptor blocking agents. If the bradyarrhythmia occurs in the presence of an acute myocardial infarction it may be transient. If there is no acute myocardial ischaemia the patient may present in a variety of ways e.g. dizzy spells, falls, blackouts or confusion. A permanent pacemaker is usually required.

Ventricular arrhythmias:

Premature beats are common in the elderly and are usually asymptomatic. If they produce symptoms and are frequent e.g. bigeminal rhythm, ventricular tachycardia, they require treatment.

Congestive heart failure

This is defined as cardiac dysfunction leading to symptoms of breathlessness and/or fatigue. It is commoner in the elderly, the prevalence being 10% in those over the age of 80 compared to 1% in the 50-60 age group.

Aetiology:

IHD 53%, VHD 31%, hypertension 25%, cor pulmonale 16%, unknown 16%, cardiomyopathy 6%

Symptoms:

LVF - cough, dyspnoea, orthopnoea, PND, fatigue and poor exercise tolerance

RVF - oedema, ascites, anorexia, nausea

Both - palpitations, weight loss, nocturia, syncope

On examination

The following may be found, dyspnoea, pale, jaundiced, sweating, tachycardiac (pulses alternans) raised JVP, basal crackles, wheezes, cardiac asthma, third heart sound, fourth heart sounds, ascites, hepatomegaly, oedema.

The prognosis

Is poor and is worse in the older patient. The mortality in severe heart failure is about 60% in the first year. The overall mortality of all grades of heart failure is 50% at 5 years.

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Diabetes mellitus

About 1 in 20 of the elderly population have diabetes mellitus and probably an equal number have impaired glucose tolerance. It is responsible for a great deal of morbidity and recent studies have shown that it is associated with an increased mortality. Over 2% of acute medical beds are occupied by elderly diabetic patients, and half of all non-traumatic amputations are because of diabetes (particularly in the elderly).

Aims of treatment

Good glycaemic control is still very important in the elderly diabetic patient.

Other aims should be:

- relieve troublesome symptoms of hyperglycaemia
- prevent hypoglycaemia, ketoacidosis and hyperosmolar coma
- assess the impact of co-existing disease
- screen for complications

Clinical features and treatment

Insulin-dependant diabetes can occur *de novo* in the elderly but it is uncommon. It presents abruptly as in the young and is characterised by signs and symptoms such as weight loss, polyuria, polydipsia, and, in some cases ketoacidosis. These patients need insulin from the beginning to maintain life.

The majority of elderly diabetics have non-insulin dependant diabetes (NIDDM). This may be found incidentally e.g. with glycosuria or following admission into hospital for an unrelated problem or with one of the diabetic complications.

The diagnostic criteria are the same as for younger patients (including the use of oral glucose tolerance test) requiring: a) one abnormal blood test (random or fasting) if symptomatic or b) two such blood tests if asymptomatic.

Dietary treatment can be very effective in lowering blood glucose levels and should be the first treatment in nearly all NIDDM patients. When diet fails to improve glycaemic control, and/or symptoms such as polyuria persist, an oral hypoglycaemic agent is then added, usually a sulphonylurea (provided not very overweight) e.g. tolbutamide or gliclazide. Chlorpropamide and glibenclamide should not be used in the elderly because of their relatively long half-life and the risk of hypoglycaemia associated with their use. Metformin is useful in the obese diabetic (it does not cause hypoglycaemia in therapeutic doses). It should not be used in patients with renal, hepatic or circulatory disease because of the risk of causing lactic acidosis.

The decision to start insulin treatment for poorly controlled elderly NIDDM should not be delayed since many feel much better on insulin and can cope with the injections. There are many who cannot administer the insulin themselves and may need to teach spouse, friend or other relative to inject or use district nurse service.

Complications

Large vessel disease is a major problem in NIDDM. Particular diabetic complications found more often in the elderly include diabetic maculopathy, hyperosmolar coma and diabetic amyotrophy. All elderly patients require annual review of eyes (dilated pupils), feet (nail care, neuropathy, excess callus etc.), renal function (and liver function if taking metformin) metabolic control and blood pressure.

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Dermatological disorders

Dermatological problems are prevalent in the elderly population and a full spectrum of disorders is seen.

Aging of the skin

Skin ages due to both intrinsic aging and photoaging, i.e. the damage caused by chronic sun exposure. Intrinsic aging causes fine wrinkles, while photoaging produces coarse furrows. Aging skin also exhibits fragility, purpura and slower wound healing.

Pruritus

"Senile" pruritus is common. It is often accompanied by dry skin, and the skin may become widely excoriated. Systemic causes of pruritus should be excluded, including iron deficiency anaemia, hypothyroidism, hepatic or renal dysfunction, and haematological malignancy. Treatment is with emollients and antihistamines.

Eczema (dermatitis)

All types of eczema occur in the elderly, including atopic, seborrhoeic, discoid, contact and gravitational eczema. Particularly common is asteatotic eczema, which occurs when the skin is dried out by frequent use of soap, hot baths and the low humidity of central heating. A cracked, crazy-paving appearance results. Eczema is investigated by patch testing if contact allergy is suspected. Treatment is by avoidance of precipitating factors, and the use of emollients, topical steroids, antihistamines, and antibiotics when infected.

Benign tumours

Seborrhoeic keratoses, cherry angiomas, skin tags and horns are common benign skin tumours, increasing in prevalence with age. Seborrhoeic keratoses range from skin colour to black, have a rough "wart" surface, and can reach a large size. Cutaneous horns may have a malignant lesion at their base.

Malignant and pre-malignant tumours

Solar keratoses, Bowen's disease, squamous cell carcinoma (SCC), basal cell carcinoma (BCC) and lentigo maligna melanoma (LMM) are related to chronic sun damage. Hence they increase in prevalence with age and usually occur on sun-exposed sites.

Solar keratoses are small scaly plaques which rarely (less than 0.1%) transform to malignancy, while **Bowen's disease** is SCC in situ. Both respond to cryotherapy or topical 5-fluorouracil. BCC and SCC are confirmed histologically and treated by surgery or radiotherapy. LMM are slowly growing pigmented macules on the face of elderly people, which may eventually become invasive. Unlike the aggressive malignant melanoma, urgent surgery is rarely required. However, an early Dermatological opinion should be sought where malignancy is suspected.

Leg ulcers

The elderly are the population in which most leg ulcers are seen. The majority (90%) are venous in nature; most of the remainder are arterial ulcers. Venous ulcers are painless, frequently occur just above the medial malleolus, and may be accompanied by changes of venous stasis, e.g. oedema, hyperpigmentation, eczema. Compression bandaging aids their healing. Leg ulcers of arterial origin are painful and have a "punched-out" appearance. The opinion of a vascular surgeon is needed. Less frequently leg ulceration is due to an inflammatory disorder, e.g. vasculitis, pyoderma gangrenosum, or a malignancy, e.g. SCC. The ulcer edge should therefore be biopsied when there are atypical features.

Infections

Bacterial infections: Cellulitis of the lower leg is common in the elderly, with leg oedema a predisposing factor. The skin is red, hot and swollen, and pyrexia may occur. The usual pathogen is *Streptococcus pyogenes*. Treatment is with penicillin, given iv in the event of extensive infection or systemic features.

Viral infections: Herpes zoster (shingles) occurs most frequently in the elderly. Unilateral pain precedes blister formation in a dermatomal distribution. Patients with ophthalmic zoster should be seen by an ophthalmologist. Acyclovir is helpful if started in the first 48 hours. Post-herpetic neuralgia is a more frequent and severe complication in the elderly.

Fungal infections: Tinea (ringworm) infects skin and nails, and is diagnosed from cultured skin scrapings and nail clippings. *Candida* infects skin folds (intertrigo) and mucous membranes, and swabs are taken. Anti-fungal creams are mostly used, but systemic agents are usually required for nail and widespread involvement.

Infestations: Scabies should be suspected in patients with pruritus and skin excoriation of recent onset. The mite causes characteristic skin burrows. Patient and close contacts require thorough treatment with anti-scabetic agents.

Blistering disorders

Bullous pemphigoid is an autoimmune disorder, with 80% patients being >60 years of age. Large, tense blisters develop. Possible association with internal malignancy is controversial. Biopsy shows sub-epidermal blistering, with IgG deposition along the dermo-epidermal junction. Treatment is with systemic steroids and azathioprine, but the condition usually resolves in 3-6 years.

Other skin disorders in the elderly

Nodular prurigo is a poorly understood disorder, commonest in the elderly, where troublesome pruritus accompanies widespread excoriated nodules.

Psoriasis is a common disorder occurring in all age groups, which is treated with standard topical and systemic agents. Drug reactions including photosensitivity are seen, since older patients are more likely to be receiving multiple drugs. Dystrophic toenails occur, aggravated by ill-fitting footwear, and may lead to the extreme condition of onychogryphosis. Fungal infection should be excluded and chiropody requested.

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Musculoskeletal disorders

The incidence of musculoskeletal disorders in elderly people increases with age. The most common disorders being osteoporosis, osteoarthritis, rheumatoid disease, osteomalacia and Paget's disease.

Osteoporosis

As we age, bone mass is lost. This loss is more severe in women who lose 60% of their trabecular bone and 35% of their cortical bone. Osteoporosis results in fractures of the vertebrae, hip and distal forearm. One third of women over the age of 65 having vertebral fractures. Osteoporosis is caused by immobility, endocrine disorders (thyrotoxicosis), alcohol, drugs (heparin, steroids) and excessive ethanol intake. The patient is usually symptom free until a fracture occurs. Signs include deformity and loss of height in vertebral fractures.

Diagnosis and treatment

This is initially on radiography although DEXA scans are able to quantify bone loss. There are no characteristic biochemical abnormalities of osteoporosis. Treatment includes pain management but bed rest should be avoided if possible. Occasionally orthopaedic appliances may be required and measures to prevent falls should be taken. All patients with osteoporosis should have a diet adequate in calcium, protein and vitamins, and should be advised to give up smoking and moderate alcohol consumption. Diphosphonates and calcium increase bone density and serve to reduce pain in patients with established fractures. Prevention of osteoporosis in women is aided by perimenopausal oestrogen replacement.

Osteomalacia

Osteomalacia is a condition characterised by impaired mineralisation of newly formed bone matrix. Although less common than osteoporosis, in contrast, treatment is highly effective. The main causes of osteomalacia in the elderly are vitamin D deficiency (dietary, lack of sunlight, malabsorption), renal disease (impairment of 1 α hydroxylation) and drugs (phenytoin, carbamazepine and phenobarbitone).

Diagnosis and treatment

Clinical features include bone pain, proximal myopathy, tenderness and skeletal deformities with an increased incidence of fracture. Diagnosis on X-ray is of pseudo fractures (loosers zones) with biochemical changes and may show a low serum calcium and phosphate and a raised alkaline phosphatase. Treatment of osteomalacia due to vitamin D deficiency is by supplementation and those with gluten sensitive enteropathy respond to a gluten free diet.

Paget's Disease

Paget's disease is characterised by excessive bone turnover. The aetiology of this is uncertain although slow viruses, possession of certain autosomal dominant characteristics or trauma may play a role. Osteoclasts cause reactive bone resorption, additionally osteoblasts result in excessive bone formation. The newly formed bone is architecturally abnormal and mechanically weak, therefore resulting in deformity and fractures. Most patients are asymptomatic at presentation although deformity and pain either due to the Paget's or secondary osteoarthritis are common. Complications include fractures, neoplasia, nerve compression and high output cardiac failure.

Diagnosis and treatment

Clinical diagnosis is made at sites of bony deformity and characteristic X-ray appearances include areas of excessive bone resorption with the cortex appearing thickened and irregular. Biochemically, urinary hydroxyproline excretion and serum alkaline phosphatase are raised. Both technetium99 and gallium67 scanning show increased uptake although this is not pathognomonic of Paget's disease. Treatment is usually symptomatic for pain but calcitonin and bisphosphonates may inhibit osteoclast activity.

Osteoarthritis

This is a common degenerative disorder showing increasing incidence with age. It commonly affects the hips, knees, feet and hands and may be primary or secondary. Women have a higher prevalence and severity of osteoarthritis of the hands and knees whereas in men, prevalence and severity is greater in the hips and spine. Secondary osteoarthritis arises in joints where pre-existing diseases such as gout, rheumatoid arthritis or Paget's are present or trauma has occurred.

Diagnosis and treatment

A history of pain and stiffness may be associated with no physical findings, however, X-ray changes of osteophytes, sclerosis, erosions and decreased joint space are classical. Treatment is generally with analgesics and non-steroidal anti-inflammatory agents. Surgery may be required at later stages of the disease.

Rheumatoid arthritis

This is a symmetrical inflammatory polyarthropathy which usually presents in middle age. The onset of the condition is usually insidious with stiffness and swelling of the small joints of hands, metatarsophalangeal joints, wrists and knees.

Diagnosis and treatment

Rheumatoid factor may be present in low titre although this is not pathognomonic. During the acute phase rest is required, although this is not ideal in the elderly. Heat treatment and exercise in order to preserve the range of movements, avoidance of contractures and maintenance of muscle strength is essential. Drugs such as analgesic and anti-inflammatory agents may be required but few patients require orthopaedic intervention except for complications such as tendon rupture or secondary osteoarthritis.

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Parkinson's Disease

Parkinson's Disease and Parkinsonism

Parkinsonism refers to akinesia or bradykinesia which is associated with rigidity and/or tremor. *Idiopathic Parkinson's Disease* has a distinct pathology with cell loss of the neuromelanin containing cells in the substantia nigra. Affected cells contain an inclusion body called a Lewy Body.

Akinesia is an essential symptom complex consisting of slowness of movement, poverty of movement, fatigue, decrement of alternating movements and difficulty in initiating movement. Easily recognisable features of akinesia are the loss of facial expression, infrequent blinking, monotonous speech, failure to swing the arms, small handwriting, difficulty in fine movements and a shuffling gait.

Rigidity is an increase in muscle tone which results from smooth plastic rigidity, so-called lead pipe rigidity, superimposed upon a tremor giving rise to "cogwheel rigidity". The rigidity affects all muscle groups but there is slightly more tone in flexion than extension and this gives rise to postural changes. It is important that rigidity is differentiated from spasticity where the increase in muscle tone is more marked in flexion in the arms and extension in the legs and is due to upper motor neurone lesions.

Tremor is the presenting feature of 70% of patients with Parkinson's Disease but may be entirely absent. In the early stages the tremor is localised to the fingers giving a pill rolling pattern. It is usually present at rest and reduced on intentional movement. The classical frequency is 3-5 cycles per second.

The neuropathology

Parkinson's Disease results from the loss of neuromelanin containing cells in the basal ganglia. The cells which produce Dopamine in the substantia nigra project the caudate and putamen in the neo striatum. Dopamine is released there and acts as a modulating neuro transmitter affecting a series of motor loops. The classical changes in Parkinson's Disease is the loss of pigment in the substantia nigra, the presence of Lewy Bodies, eosinophilic intracytoplasmic inclusions looking somewhat like targets and a loss of over 70% of nerve cells in the substantia nigra. It is recognised that this cell loss is associated with a deficiency of Dopamine in the striatum. Striatal dopamine must fall below 70 - 80% for clinical Parkinson's Disease to develop. There is involvement in other neurotransmitter systems particularly noradrenergic systems. In addition acetylcholine, serotonin and some peptide neurotransmitters are deficient.

Epidemiology

There is a cumulative life time risk of developing Parkinson's Disease of 1 in 40. The prevalence and incidence rates increase with increasing age and the disease rarely starts below the age of 55. Prior to the introduction of Levo Dopa patients with Parkinson's disease had a standardised mortality ratio of 2.9 times the general population and subsequently the rate is about 1.3 quite close to the normal expectation of life.

Aetiology

The aetiology of Parkinson's disease is unknown, however, a number of factors have been implicated:

Environmental insult

The toxin MPTP causes a clinical syndrome identical to Parkinson's Disease. MPTP is converted into its active form MPP⁺ by monamine oxidase type B. This is taken up by the Dopamine transport systems of dopaminergic cells to cause disruption in the mitochondrial respiratory chain affecting mitochondrial complex 1 activity. Similarly impaired mitochondrial function is seen in normal Parkinson's disease suggesting that there may be a free radical insult that causes the disease.

Genetic factors

Genetic factors are thought to be of little importance in Parkinson's Disease as a result of twin studies, however, mitochondrial inheritance has to be considered and different expression of the disease in twins with later onset of the disease excluded in the first studies has meant that some authorities still believe that genetic influences are important.

Ageing

Dopaminergic neurones are affected by the ageing process declining at a rate of about 5% per decade. This rate of ageing would not account for Parkinson's disease alone but when combined with an environmental insult may explain its late age distribution.

Problems that occur with Parkinson's Disease

Parkinson's Disease very often disturbs gait causing freezing of movement, shuffling gait. The foot is held in a plantar flexed position which makes the lever action of the foot less efficient. The patient has postural change with characteristic flexion of the limbs, neck and trunk. Postural instability is a late feature of the disease, patients presenting with falls. The autonomic nervous system is affected giving rise to constipation, urinary symptoms, excess sweating and low blood pressure with postural fall in blood pressure. Psychiatric illness is more common in Parkinson patients particularly dementia and depression.

Natural History of the Disease

Parkinson's disease is a progressive disorder which starts with mild unilateral involvement and progresses to complete dependency. It is usually staged in five stages;

- Stage I Unilateral involvement only
- Stage II Bilateral involvement without impairment of balance
- Stage III Impairment of balance and functional restriction
- Stage IV Fully-developed disease retaining ability to walk and stand unassisted
- but otherwise markedly incapacitated
- Stage V Bed-bound or wheelchair bound unless aided

The Parkinson's Disease has to be differentiated from other causes of Parkinsonism the most important of which is drug-induced. This can be caused by any drugs which block dopamine receptors the most important are neuroleptic drugs given in major psychiatric illness such as Largactil and drugs given to treat nausea and dizziness notably Metoclopramide and Prochlorperazine. Parkinsonism may follow viral encephalitis although this is now extremely rare. Arteriosclerotic pseudo Parkinsonism is a disorder which affects predominantly the lower half of the body giving gait apraxias which may mimic those seen in Parkinson's disease is due to small infarcts in the basal ganglia and sub-cortical matter and is usually associated with dementia in hypertensive patients who do not usually respond to anti-Parkinsonian medication.

Multi-system disorders

Parkinson's may arise from disorders which affect more widespread areas in the brain than simply the basal ganglia. Multi-system atrophy is quite rare, one variant called Supra nuclear palsy particularly affects eye movements.

Treatment of Parkinson's Disease

The theory of treating a neuro-degenerative disease such as Parkinson's Disease involves the replacing of the neuro transmitters which are reduced, the main effort is made to increase dopaminergic activity and decrease cholinergic activity.

The drug *Levo Dopa* is a pre-cursor of Dopamine (Dopamine does not cross the blood/brain barrier). Other drugs may stimulate the release of Dopamine (Amantadine). Drugs may mimic the action of Dopamine acting directly on the post synaptic receptor. These are Dopamine agonists such as Bromocriptine, Lysuride and Pergolide. Drugs may act against the cholinergic system ie Benztropine. Cholinergic activity is increased in Parkinson's disease because Dopamine normally inhibits this pathway. Drugs may augment the use of Levo Dopa in particular monoamine-oxidase-B inhibitors such as Selegiline or a COMT inhibitor.

Levo-dopa without a decarboxylase inhibitor cause nausea and vomiting and postural hypertension so it is always combined with a decarboxylase inhibitor. In the case of Madopar this is Benserazide and Sinemet carbidopa. Side effects of Levo-Dopa include involuntary movements, psychiatric disturbance, nightmares, hallucinations, frank psychosis, fluctuations in response including on/off syndrome.

Parkinson's is also helped by non-drug treatments, occupational therapy giving analysis of self-care, providing aids and appliances to improve function, physiotherapy can greatly improve gait, speech therapy can improve both speech and feeding.

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Pressure sores

Definition

The term pressure sore is used to describe a range of destructive lesions of the skin and sub-cutaneous tissues and are of varied pathogenesis. They are referred to as "bed sores", "decubitus ulcers", "pressure ulcers" and "brush burns". They range in severity from erythematous soft tissue lesions to open wounds which extend deep into the tissues.

Incidence

Unfortunately, pressure sores are often ascribed to poor care which inevitably adversely affects comprehensive and accurate documentation of their presence and therefore estimates of incidence and prevalence are vague. Though estimates have been made of incidence (1-5%) and prevalence (3-14%) in hospitalised patients (Barbarell, 1977), there are many problems with the data collected. Studies are frequently performed on a single site, rendering the results unable to be generalised and data collected is not standardised. Though pressure sores frequently contribute to increased morbidity and mortality, they are grossly under-reported as the cause of death on death certificates.

Pathogenesis

Pressure sores are essentially the result of soft tissue compression between bony prominences and external surfaces over a period of time causing localised tissue necrosis. However, there are many local and systemic risk factors.

Local risk factors include:

Direct pressure - the greater the pressure the less time is required for tissue necrosis due to impaired capillary perfusion.

Shear force - skin and superficial tissue adhere to the bed clothes (during movement of the patient) traumatising the underlying blood vessels. Movement may be instituted by nursing staff, the patient themselves or simply gravity.

Moisture and increased skin temperature - mattresses retain heat, increase metabolic rate in the tissues and potentiate the effects of ischaemia causing breaks in the skin.

Systemic risk factors include:

- Ageing skin - reduced elasticity, thinning and loss of water and fat content.
- Immobility and inactivity - osteoarthritis, hip fractures and other problems may render some people particularly the elderly prone to pressure sores.
- Malnutrition - reduction in serum albumin, cholesterol and haematocrit contribute to the development of pressure sores and adversely affect the healing process.
- Arterial disease and hypotension - adequate oxygenation of tissues depends on blood flow.
- Sensory loss - some patients (eg. stroke) may be less aware of pain and damage caused by continued pressure.

Many risk factor assessment scales have been developed to quantify how "at risk" some people are. They are based on ratings of mobility, incontinence, nutrition etc. (Norton Score, Waterlow score, Braden and Douglas). Though they focus staff attention on particular patients, they do not take into account other factors which influence treatment (eg. unwillingness to practice standing). There are also some problems with these scores as they tend to over predict, ie. those with high scores do not necessarily develop sores.

Clinical features and complications

Pressure sores represent a continuum from superficial to severe and several classification systems have been developed to give an indication of severity and act as a guide to treatment.

One classification system is as follows:

- Stage I - Non-blanchable erythema of intact skin - the heralding lesion of skin ulceration.
- Stage II - Partial thickness skin loss involving the epidermis and/or dermis. The ulcer is superficial, presenting clinically as an abrasion, blister or shallow crater.
- Stage III - Full thickness skin loss involving damage or necrosis of sub-cutaneous tissue which may extend to (but not through) underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of the adjacent tissue.
- Stage IV - Full thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone and supporting structures.

Other features also aid description of the sores including; site, depth, colour, base, edge, surrounds and pain.

Pressure sores may further complicate patient management and prognosis by contributing to or causing infection (generalised or local sepsis), dehydration, anaemia, electrolyte imbalance and malnutrition.

Prevention

It is important as far as is possible to identify those at risk at an early stage. This involves education of all staff, by the use of risk scales and increased awareness. The main prevention measures can be summarised as follows;

Treatment of medical conditions and care of body fluids, regular turns, frequent weight shifts and proper positioning (lying prone at night), the elimination of shear with or without friction, frequent skin inspections and the use of specialised beds, mattresses or cushions.

Treatment - The principles of treatment can be summarised as follows: The improvement of general health and nutrition, restoration of tissue perfusion (relief of pressure), maintenance of a clean wound, prevent or treat wound infection, stimulation of granulation tissue and arterial reconstruction where necessary.

Topical agents (cleansers, dressings, cavity fillers and desloughing agents) used should only be those detailed in the Regional Wound Care Formulary and Criteria for Management. That is; all epithelial breaks should be occluded, only systemic (no topical) antibiotics to be used, no toxic cleansers to be used and no agents should be used that are likely to debride contaminate or poison granulation tissue.

In terms of prognosis, pressure sores are painful, unsightly, difficult to treat. The human costs are immeasurable.

Conclusions

Pressure sores are extremely costly in both human and financial terms. Costs have been estimated at £150 million annually (1982 - probably £300 million today), with a per patient cost of £26,000. Costs of treatment and prevention must be balanced.

Pressure sores are frequently under reported and the current state of knowledge, and future treatment and prevention strategies must be investigated. Accurate data on prevalence and incidence must be determined and the contribution of pressure sores to mortality and morbidity must be documented.

Pressure sores develop in places where doctors seldom look, but the responsibility for the state of patients skin should be shared by all staff.

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Respiratory disease

Age-related changes:

1. Anatomical

These include kyphosis, rib decalcification (reduced transverse thoracic diameter) and calcification of costal cartilages with resulting increasing chest wall rigidity. There is additionally a fall in the alveolar surface area by 4% per decade after age 30. The alveoli flatten and shallow out reducing gas exchange area. The alveolar ducts enlarge and the alveolar walls thin and contain fewer capillaries. The pulmonary artery branches have increased wall thickness and radius.

2. Physiological

These include a reduction in the FEV1 of approximately 32 ml/yr in males and 25 ml/yr in females. This reduction in the FEV1 is greater than the FVC reduction and therefore the ratio FEV1/FVC% decreases. The residual volume increases, total lung capacity is unchanged but both the anatomical and physiological dead space increase. Lung elastic recoil falls and airway collapse occur more frequently in lower zones, lung perfusion remains predominantly basal resulting in ventilation-perfusion mismatch.

Respiratory symptoms are common in the elderly although the presentation of respiratory disease may be very different from that in younger subjects. Cough is often considered to be "normal". Cough may be classified either as "acute" (less than 2 weeks duration) and usually due to infection, asthma, pulmonary oedema or embolism or foreign body aspiration. If cough persists longer than two weeks it is considered to be chronic with causes such as chronic bronchitis, asthma, neoplasia, tuberculosis, interstitial disease and bronchiectasis, being likely.

Chest pain is a common presenting symptom despite an increased pain threshold in the elderly. The likely diagnosis will be respiratory, cardiac or chest wall pain. Breathlessness is highly subjective and often difficult to quantify. The severity of dyspnoea may not relate to spirometry or blood gases. Cardiac causes of breathlessness include pulmonary oedema secondary to ischaemic heart disease, valvular disease, hypertension or cardiomyopathy. Respiratory causes include asthma, chronic bronchitis, pneumonia or tuberculosis. Additionally, breathlessness may result from neuromuscular disease, renal failure, diabetic precoma or anaemia.

Respiratory diseases in the elderly

1. Pneumonia

This is divided into "community acquired" or "hospital acquired". The former are usually bacterial including strep pneumoniae, haemophilus influenzae, staph aureus or viral including mycoplasma. Hospital acquired infections occur 48 hours or longer after admission and are usually due to more varied organisms for example gram negative bacilli. Clinically, pneumonia does not have the abrupt onset as in younger subjects and may be associated with confusion or non-specific deterioration in health. It is more commonly seen in patients with chronic bronchitis, left ventricular failure, diabetes or in patients in whom drugs such as codeine or sedatives have been given. Unlike younger subjects, fever, rise in white cell count and ESR may be absent.

2. COAD

The MRC definition (1985) "Cough on most days for 3 consecutive months for greater than two consecutive years".

Emphysema The WHO definition (1961) "Abnormal increase in size of airspaces distal to terminal bronchiole with destruction of their walls".

Both usually smoking related.

3. Asthma

This is defined as reversible airway narrowing which occurs either spontaneously or with drug treatment. It is found in 5% of men and 2% of women aged over 70.

4. Pulmonary embolus

This is an increasingly frequent cause of death in the elderly which is often not diagnosed antemortem.

5. Lung cancer

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Renal disease

Renal Function

Kidneys are involved in the excretion of waste and unwanted end-metabolites. Creatinine is synthesized in muscle at a relatively constant rate and excreted largely by glomerular filtration with a smaller contribution from tubular secretion. Its clearance provides a useful measure of glomerular filtration rate (GFR). In adults with a stable muscle mass, plasma creatinine levels are inversely related to the GFR. Metabolic and endocrine functions of the kidneys include synthesis of erythropoietin, which will stimulate erythropoiesis, and hydroxylation of 25-hydroxycholecalciferol to provide active vitamin D, which has a central role on calcium and bone metabolism. The kidneys play a central role in the renin-angiotensin-aldosterone regulation of blood pressure, sodium and potassium balance. Insulin, parathyroid hormone and calcitonin are primarily cleared by glomerular filtration.

Ageing in the kidney

- Reduction in weight of kidney - up to 20% between 5th and 8th decades.
- Renal size diminishes by as much as 2 cm within the same period.
- Microscopically there is a reduction in the number and size of the nephron.
- Increase in number of mesangial cells.
- Increased basement membrane thickening.
- Increased number of diverticulae in the distal convoluted tubule.
- Variable sclerotic changes in the walls of the larger renal vessels.
- Reduction in renal plasma flow from 600 ml/min in young adults to 300 ml/min at age 80.
- GFR decreases linearly after the middle of the fourth decade (8 ml/min/1.73m²/decade).
- Decreased excretory and re-absorptive capacities of the renal tubules.

Clinical aspects of renal disease in the elderly

Haematuria

May result from urinary tract infections, trauma, parenchymal disease and malignancies. Red cell casts may indicate parenchymal disease. The nephrotic syndrome may be caused by membranous glomerulonephritis and may be in association with carcinoma. Investigation of the elderly is often complicated because of ageing changes and co-existing diseases, for example, intravenous urography may precipitate acute renal failure in patients with diabetes or multiple myeloma.

Urinary tract infection

The prevalence of urinary tract infection increases with age and may be as high as 30% in women aged over 65 years. Predisposing factors in women include the shortness of the urethra, the post menopausal vaginal changes and changes in normal flora. In men, lack of prostatic secretion, faecal incontinence, urinary retention and obstruction for any cause particularly prostatic enlargement may result in urinary tract infections. Significant bacteriuria (100,000 organisms per ml of urine) is the basis of the diagnosis of urinary tract infection. Classical symptoms seen in the young may be lacking and UTI may present with confusion, fatigue, dizziness or immobility. Gram-negative organisms such as E-coli are responsible for 90% of UTIs in general practice and up to 50% in hospital. Other organisms such as Proteus, Klebsiella, Pseudomonas and Strep. faecalis are commonly found on culture. Recurrent urinary tract infections may be due to co-existent medical diseases such as diabetes or to structural abnormalities in the urinary tract including renal stones and prostatic enlargement. Definitive treatment of these conditions may be required to prevent reinfection.

Patients presenting with symptomatic infection and renal dysfunction should have antibiotic therapy guided by bacteriological investigations. Unfortunately failure of treatment is high.

Renal failure

The most important aspect of acute renal failure in elderly patients is prevention. Any situation leading to hypovolemia and hypotension should be promptly controlled to avoid pre-renal failure and obstructive conditions such as stones, prostatic hypertrophy, malignancies and neurogenic problems should be dealt with appropriately, to prevent obstructive ARF. Besides these conditions, many systemic diseases may affect the kidneys and lead to acute or chronic renal failure. Diabetic nephropathy is a particularly important problem in elderly patients. Many drugs are nephrotoxic and may also lead to renal insufficiency. Aminoglycosides, tetracyclines, cephalosporins, sulphonamides and iodinated contrasted media can be cited.

The treatment of renal failure involves conservative management in the early stage. This is based on dietary protein and potassium intake restriction. Haemodialysis and continuous ambulatory peritoneal dialysis may be considered for the end stage renal failure.

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Stroke

Definition

This is a clinical diagnosis and is a sudden neurological deficit of presumed vascular origin lasting > 24 hrs (or leading to death within 24 hours). Transient ischaemic attack @ a stroke that clinically resolves within 24 hrs (maybe CT evidence of infarction)

The majority of strokes result from two processes of vascular origin.

1. Blockage of a cerebral artery causing brain damage (cerebral infarction) from lack of oxygen and nutrients.
2. Leakage of blood within or around the brain (intra-cerebral or subarachnoid haemorrhage).

Sudden neurological deficits can also arise from non-vascular lesions such as tumours, metabolic disturbances, epilepsy or from circulatory problems of a different kind, such as migraine or subdural haematoma. These can mimic stroke and must be excluded by appropriate tests.

Incidence

Overall 1.5 to 2 per thousand per year. This is equivalent to 100,000 strokes per year in Britain. On average a GP will see about 5 new cases per year and have 10-20 cases on his list. The incidence of stroke is higher in the elderly and is 15-20 per thousand per year in the over 75 age group. As the population ages and the number of people surviving to 75 and over increases, the total number of strokes may also increase. Thus stroke should be considered one of the Giants of Geriatric Medicine.

Risk Factors for Stroke

Major risk factors:

1. age (incidence doubles every 10-12 years over 30)
2. hypertension (both systolic and diastolic)
3. heart disease (ischaemic heart disease, valve disease, atrial fibrillation and cardiac failure)
4. diabetes
5. previous stroke
6. transient ischaemic attack

Minor Risk Factors:

1. polycythemia (high haemoglobin)
2. smoking
3. alcohol
4. obesity

Diagnosis and Investigation

Stroke is a clinical diagnosis based on an accurate history and examination. In patients with impaired consciousness, confusion, dysphasia or dysarthria it is vital for the admitting doctor to obtain an accurate history from family or friends and to thoroughly examine the patient. Specifically conditions which mimic stroke and must be excluded include hypoglycaemia, subdural haematomas, brain tumours or abscesses and post-ictal paralyses called Todd's paralysis. Investigations are mainly used to clarify diagnosis and other associated illnesses such as myocardial infarction or temporal arteritis and thus they should include a random blood sugar, urea and electrolytes, full blood count and ECG in the first instance. Differentiation between cerebral haemorrhage, cerebral thrombosis, subdural haematomas, brain tumours or abscesses can be distinguished by CT scanning. Equally, the use of CT scanning has implications in further

management. Other investigations that may be appropriate include doppler ultrasound of the neck, echocardiography and an EEG. If the patient is on anticoagulant therapy then an INR is essential.

Complications and survival

In the early stages of stroke there may be associated cerebral oedema and this can cause brain stem compression leading to early death within the first week. Between one and two weeks the patient may develop bronchopneumonia because of immobility, impaired consciousness and aspiration. They are also at risk from pulmonary emboli due to deep venous thromboses. Later complications include the problems of immobility such as infections, pulmonary emboli, pressure sores and heart disease. Only 20% of deaths are due to further strokes and the risk of a recurrent stroke within the first year is 10%. It is then 5% per year thereafter.

Of the stroke patients admitted to hospital 30% are dead at 1 month and 60% dead at 1 year. Between 40% and 70% of survivors are independent in basic self care at a year following a stroke. Of the patients that are unconscious within the first 24 hours due to a stroke, 85% are dead at 1 month and 90% at 1 year. This poor prognosis is reversed in those patients who are continent. 5% are dead at 1 month and 10% are dead at 1 year. 85% or more of these patients are independent in basic self care after a year. Stroke assessment should determine which brain functions have been effected by the stroke and to what extent this interferes with useful function.

At the earliest possibility the following should be assessed:

1. Rule out things which need immediate treatment, i.e. hypoglycaemia, hypoxia, hypotension, left ventricular failure, atrial fibrillation.
2. Exclude other causes of this clinical picture including meningitis, cerebral abscesses, subdural haematomas.
3. Assess conscious level (80-90% of comatose stroke patients die).
4. Check swallowing and ensure adequate intake, IV fluids if necessary
5. Document cognitive function including memory if patient can cooperate, orientation and concentration.

Speech/language

Dysarthria is common after hemisphere strokes as well as brain stem strokes. Dysphasia is best divided into receptive and expressive. Elements of both are often present and this is termed global dysphasia. It indicates more serious brain damage.

Visual deficits

The presence of an homonymous hemianopia should always be ascertained at the bedside by use of confrontation. However, this requires cooperation and concentration on behalf of the patient and may be difficult to obtain in the early stages of stroke. It should however be assessed when the patients condition has become more stable within the first few days of admission. It should not be mistaken for visual inattention, or visual neglect.

Sensory deficit

Left-sided weaknesses or hemipareses are often associated with an alteration in sensation in the effected side and usually reflects right parietal lobe damage. This is much rarer in right-side weaknesses but can occur. However in right-sided weaknesses the speech is more often effected. In the extreme form of sensory deficit, the person may fail to recognise his affected limbs or deny that they even belong to him. However, more usually it manifests itself as a tactile sensory inattention or loss of proprioception in the affected limbs.

Motor Deficit

This should be assessed during the peripheral nervous system examination with tone being assessed before power. Power should be assessed with the muscle groups under assessment, in the position of maximum power. Sometimes there is obvious muscle power present, but complex tasks cannot be performed. This is termed apraxia. It can give rise to specific problems with dressing and gait.

Treatment

There is no single effective treatment for established stroke. However, patient management should include looking for reversible causes that may mimic stroke such as hypoglycaemia or over anticoagulation and ensuring patient safety with regard to the swallow reflex. Nursing in the unconscious position, assessing skin condition and ensuring an adequate fluid intake. The family should be interviewed early on and informed of the prognosis. Blood pressure should be recorded but patients with high blood pressure should not have this lowered in acute stroke as it may result in an extension of the condition. Complications such as pressure sores, aspiration, pneumonia, contractures, deep venous thrombosis should be prevented. Depression which is common in stroke patients and may occur in up to 40% of these patients should be screened for and treated as appropriate. Mobilisation should occur as early as possible and this is best achieved with a team approach directed by the physiotherapist with instructions to nurses to carry this on in the ward. The occupational therapist is essential in the assessment and therapy of simple procedures such as dressing, feeding and cooking.

Aspirin is indicated for any patient who suffers a transient ischaemic attack and as secondary prophylaxis after cerebral infarction unless there is a contra-indication. Anticoagulation is useful in patients under 80 who are in atrial fibrillation or any patient who has a clear source of cardiac emboli. Where treatment with warfarin is considered then cerebral haemorrhage must be excluded by a CT scan. Some patients will die as a result of their stroke and it is the responsibility of all members of staff to ensure a comfortable and dignified death.

Prevention

Aspirin has been shown to be effective in secondary prevention of stroke as has adequate control of blood pressure. Equally in patients with lone atrial fibrillation, warfarin has now been shown to reduce the incidence of stroke in those aged between 60-80. In patients 80 years and older the use of warfarin is debatable as the side effects of formal anticoagulation such as GI haemorrhage and cerebral haemorrhage are much greater in this elderly age group.

In patients with TIAs or minor strokes who have significant carotid stenosis ³ 70% then surgery (carotid endarterectomy) is recommended.

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Mental Health

- **Cognitive impairment**
Shaun O'Keeffe
- **Depression**
Maggie Hammond

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Cognitive impairment

Implies a global disturbance of brain function: memory - especially registration and recall of recent events; orientation - time > place > person; attention - increased distractibility; perception - increased misinterpretations, illusions, hallucinations; logical thought - muddled thinking and speech.

Includes:

- delirium (acute confusional state)
- dementia (chronic confusional state)

Avoid the words 'confusion' or 'confused': no standard definition or understanding of these words; often imply chronic and irreversible impairment.

Cognitive impairment is a syndrome, not a diagnosis. Full assessment to determine the diagnosis and cause is essential.

Assessing a patient with suspected cognitive impairment

The assessment comprises:

1. Interview with carers/relatives
2. Interview with patient
3. Examination of patient
4. Formal mental status test
5. Diagnostic tests

Is cognitive impairment present? Suspicion will be aroused by:

1. Memory/orientation problems: e.g. repeating the same question again and again, repeatedly losing things like pension book or keys, forgetting familiar names, getting lost in familiar surroundings.
2. Social problems: e.g. neglect of appearance, nutrition or hygiene, loss of interest in hobbies, withdrawal from social surroundings.
3. Behavioural problems: e.g. getting lost, wandering, leaving gas on unlit, irresponsible use of money.

How long has it been present? 'When did you first suspect something might be wrong?' 'When was X last his or her normal self?'

What problems is it causing? Social and behavioural problems (see above) Financial problems
Poor compliance with medications Incontinence Stress to carers (potential for abuse)

Interviewing the patient

Observe grooming, mood, behaviour Is patient distractible or drowsy? Does behaviour fluctuate from minute to minute?

Normal social conversation provides most information

- avoid question with yes/no answers
- leave formal mental testing to the end.

Note language use (exclude aphasia)

Note evasiveness in answering questions 'Do you think your memory is all right for your age?'

FORMAL MENTAL STATUS TESTING

Abbreviated Mental Test

Widely used in U.K.

Score 1 point for each correct response

1. Age
2. Time (to nearest hour)
3. Address for recall at end of test: 42 West Street (Ask patient to repeat the address to ensure it has been heard correctly)
4. Year
5. Name of hospital
6. Recognition of two persons (e.g. doctor, nurse)
7. Date of birth
8. Year of start of first World War
9. Name of Monarch
10. Count backwards from 20 to 1

Benefits:

1. Brief, objective screening test for cognitive impairment
2. Acceptable to patients
3. Serial tests useful in monitoring progress

Disadvantages:

1. Only a screening test
2. Tests few areas of cognition (chiefly memory and orientation)
3. Cannot distinguish delirium from dementia
4. If the test is not repeated after resolution of delirium, a patient may be wrongly labelled as 'demented'

Best cut-off for abnormality is 7/8 points However, this cut-off is only 80% sensitive and 80% specific; that is, 1/5 people with a score of 7 or less are normal, and 1/5 people with a score of 8 or more are cognitively impaired.

DELIRIUM

Definition:

Delirium is an acute organic mental syndrome characterized by global disorder of cognition. It may be difficult to distinguish from dementia as these conditions often coexist, a good history from carer is essential, and serial mental tests may be helpful.

Features of Delirium and Dementia

	Dementia	Delirium
Onset	Gradual (months to years)	Acute (usually hours to days)
Progression	Progressive	Non- progressive
Duration	Usually irreversible	Days to weeks
Impaired attention	Mild except in very	Prominent feature
Level of consciousness	Normal except in very severe dementia	Fluctuating (often impaired)
Organic cause	Usually NOT found	Usually found

Epidemiology

- 10-30% of acute medical admissions in elderly people
- 10-15% after general surgery
- 30-60% after hip fracture repair or cardiac surgery

Course and prognosis

- 15-25% are dead within a month
- Majority of episodes clear within a few days to two weeks.
- Some have a more prolonged course.
- Rarely progress to dementia (more often, dementia has not previously been recognised).

Delirium is associated with:

- Increased duration of hospital stay
- Increased hospital-acquired complications (falls, pressure sores, infections, urinary incontinence)
- Increased risk of admission to long-term care on discharge from acute hospital.

Clinical subtypes.

1. Agitated: hyperactive, hyper-alert prominent delusions/hallucinations risk of self injury
2. Quiet: hypo-active, hypo-alert often appear depressed easily missed unless high index of suspicion.

Predisposing factors:

- Increased age (reduced capacity for homeostasis; reduced acetylcholine synthesis)
- Decreased vision and hearing
- Dementia

Precipitating factors:

- Medications (anticholinergics; digoxin; psychoactive; steroids + almost any other)
- Infection
- Metabolic disturbance (hyponatraemia, hypokalaemia, hypoglycaemia; dehydration; organ failure)
- Intracerebral disease (stroke, trauma, infection)
- Hypoxia (cardiac, respiratory)
- Alcohol abuse or withdrawal;
- Benzodiazepine withdrawal
- Myocardial infarct

Investigations:

History:

- Confirm diagnosis - talk to carers/G.P., read old notes
- ? underlying dementia
- Note recent medication changes
- Ask about alcohol/benzodiazepines

Examination:

- ? infection (delirium may precede X-ray/lab abnormalities)
- ? hydration adequate
- Careful neurological exam

Tests:

- Serum electrolytes, full blood count, glucose, chest X-ray, ECG
- Other tests as appropriate
- In difficult cases, consider brain CT, EEG, lumbar puncture

Management:

- Treat the underlying cause
- Relieve distress and prevent self-harm reassurance, esp. during procedures

- calm approach, quiet room
- frequent family visits
- sedation only if essential to prevent harm or relieve severe distress. Haloperidol or Thioridazine for most circumstances, benzodiazepines if alcohol or benzodiazepine withdrawal
- Maintain hydration and nutrition consider vitamin supplements if delirium prolonged

DEMENTIA

Definition:

An acquired chronic, and frequently irreversible brain disorder characterized by global cognitive impairment which is of such severity as to interfere with social or occupational functioning.

Areas of cognitive affected include:

- memory (recent > long-term)
- abstract thinking
- judgement
- personality
- language
- visuo-spatial skills

Causes:

Alzheimer's disease - AD (50-60%)

- degenerative brain disorder
- 10-15% familial inheritance

Pathological hallmarks:

- neurofibrillary tangles and neuritic plaques
- impaired acetylcholine neurotransmission

Multi-infarct disease - MID (15-20%)

- abrupt onset
- step-wise progression
- focal neurological signs
- often a history of hypertension
- frequent gait disturbance (Binswanger's disease)

(Alzheimer's disease and multi-infarct disease coexist in a further 10-20%)

Lewy-body dementia (frequency disputed ?1%, ?20%)

- prominent early parkinsonism
- fluctuating clinical features
- do badly if prescribed neuroleptics

Potentially treatable dementias (10-20%) (Most only partially reverse with treatment)

- Depression
- Normal pressure hydrocephalus
- Infections such as:
 - Meningitis
 - brain abscess
 - syphilis
 - AIDS (+ associated opportunistic infections)
- Vascular disease such as:
 - subdural haematoma
 - vasculitis
 - cardiac disease
- Systemic or metabolic disorder:
 - nutritional deficiency (esp. vitamin B12 and thiamine)

- endocrine disease (esp.hypothyroidism, Cushing's disease, hyperparathyroidism)
- systemic lupus
- erythematosus
- Toxic disorders such as:
 - drugs (esp. psychoactive)
 - alcohol
- Neoplasms (direct and indirect effects of primary and metastatic tumours)
- Others:
 - Parkinson's disease
 - Pick's disease
 - Huntington's chorea
 - Jacob-Creutzfeldt disease
 - Anoxic brain damage

Differential diagnosis

- Delirium
- Depression (both a cause and a consequence of cognitive impairment)
- Benign forgetfulness of old age

Prevalence (European data)

- 65-70 years: 1.5%
- 70-75 3%
- 75-80 6%
- 80-85 12%
- 85-90 20%
- 90+ 30%

Course and prognosis

AD progresses gradually while MID has a step-wise deterioration associated with repeated (often clinically inobvious) strokes.

Terminal dementia: neurological signs and symptoms (parkinsonism, primitive reflexes, seizures) become common; patients may lose mobility, continence, speech and swallowing; they are almost totally dependent on others.

Average survival in AD or MID is 7 years (the same as many malignancies).

Investigations

Exclude potentially treatable causes:

- serum electrolytes
- calcium, glucose
- full blood count
- ESR
- vitamin B12
- syphilis serology
- thyroid function tests
- urinalysis
- chest X-ray and ECG.

Other tests will be determined by history and physical findings

Brain imaging is not necessary if there is a classical history of dementia present for 6 months or more; however, if there are any atypical features, CT or MRI should be performed.

Management

Multidisciplinary approach to assessing and planning interventions (esp. geriatrician and/or psychogeriatrician, social worker, occupational therapist, community nursing staff).

Treat reversible factors, especially depression.

Treat complications: eg, delirious episodes and incontinence.

Analyze environment for safety and security.

Regular, familiar daily routine.

Support carers, e.g. day centre, respite care.

Plan for future, e.g. finances; sheltered accommodation or long-term care will become necessary for some patients.

Drugs:

- small doses of phenothiazines may help with otherwise uncontrollable behavioural or sleep disturbances
- avoid anticholinergic medications
- tetrahydroaminoacridine (THA), a cholinergic agent, may be of slight benefit to a proportion of AD patients
- low-dose aspirin and blood pressure control may help MID patients

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Depression

Depression is the most common mental illness in the elderly. It increases morbidity, mortality, and seriously affects quality of life. Approximately one in ten community-dwelling over-65s (and one in five in nursing homes) are affected to an extent which requires treatment. Dementia is more common than depression only in the very elderly.

Depression goes largely untreated because:

- a) It is underdiagnosed. Depression may be unrecognised by patients, families and physicians. Persistent sadness, loss of pleasure in life, and the somatic symptoms of depression may be accepted as normal signs of ageing or circumstances. Emphasis on physical illness by patients and doctors means that emotional well-being may be overlooked; patients may not volunteer details of their mental state and doctors may not ask.

- b) It is undertreated. Some drug treatments are contraindicated in the elderly person with physical illness. Many people assume that psychological therapies are inappropriate for the elderly or that elderly people are unwilling to accept a diagnosis of mental illness and/or reluctant to take antidepressants.

Presentation

Depressive illness is characterised by:

1. dysphoria

- a low mood
- sadness,
- irritability,
- worthlessness,
- helplessness,
- hopelessness

2. anhedonia (loss of interest or pleasure) which is present most of the time for at least 2 weeks.

3. Other symptoms may include:

- poor appetite;
- sleep disturbances;
- crying;
- complaints of poor concentration;
- loss of energy;
- agitation; retardation;
- complaints of pain;
- suicidal ideas;
- feelings of guilt.

In severe depression there may be delusions (for example of illness such as cancer or sexually transmitted disease, or of persecution) or hallucinations.

Pseudodementia

Depression may be accompanied by a reversible, non-progressive cognitive deficit of relatively sudden onset. It is not certain whether this is due to an alteration in the neurochemistry of the brain, or to loss of motivation associated with depression. One in ten severely depressed people may be misdiagnosed as demented, and not receive the treatment they need. Depressed people whose presenting complaint is insomnia may receive sedatives which compromise cognitive function. Depression is also seen in people with dementing illness, particularly in the early stages when the individual may be aware of loss of function.

Aetiology

Depression may arise for the first time in old age. There is no evidence that ageing itself causes depression, but the social stressors and losses associated with ageing may be implicated: bereavement; loss of employment, social status and social network; loss of opportunities for positive reinforcement; loss of functioning due to chronic illness and disability; chronic pain; financial stress; social isolation; reduced physical activity; visual and hearing impairment.

Chronic illness has been found to strongly predict depression. In a vulnerable person, prolonged stress of any kind may lead to depressive illness. Primary biochemical changes resulting from certain conditions (stroke, Parkinson's Disease, liver failure, anaemia, hypothyroidism) and many medications may result in depression.

Treatment and outcome

Psychological therapies (cognitive, behavioural, psychodynamic, counselling) are as effective in elderly as in younger people. Drug treatments are equally effective as in younger people, but require careful management, and response may take several weeks. The side effects of tricyclic drugs may be anticholinergic (dry mouth, urinary retention, constipation, worsening of glaucoma); adrenergic (hypotension), and histamine (sedation). Studies of newer antidepressants (serotonin re-uptake inhibitors) are showing promising results for efficacy and more acceptable side effect profiles. In the severely depressed, suicidal patient, electro-convulsive therapy is a safe and affective treatment.

Untreated depression may remit in around one half of cases; the natural course of depression is one and a half to four years. Loss of motivation and appetite may severely compromise physical well-being, with mortality at least twice as high in depressed elderly. Suicide is more common in elderly men than in any other group. Around 75% will recover from an individual episode with treatment, but relapses occur in approximately 50% of those who recover (most often within eighteen months).

Poor prognostic factors include physical illness, and long duration of depression before treatment. Best prognosis is associated with early intervention, adequacy of treatment, optimising physical health, and planned aftercare. Maintenance or prophylactic therapy (social, psychological and/or drug) may be indicated.

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Immobility

Definition

Immobility can be defined as an inability to occupy space - the life-space from anywhere in the wide world to the confines of an upstairs bedroom. Its causes may be as diverse as inability to afford a car or an aeroplane ticket to agoraphobia, deafness, fear of falling, or the severe restrictions imposed by arthritis or a stroke. The effects of restricted mobility on an individual are equally diverse. Immobility is usually considered as a restriction in everyday activity.

How commonly does restricted mobility occur?

Outside the home - An investigation by Age Concern in 1978 of people past retirement age revealed that about 40% had no difficulties getting about. In the remaining 60% difficulties with hills and ramps were the most commonly reported (31%), while others were traffic and road crossing (22%), uneven pavements (16%), and steps and kerbs (4%).

More severe restriction - The prevalence of more severe immobility rises with increasing age as shown in the table below:

Age Group	%Bedfast	%Housebound	%Needing Assistance
65-69	0	1.1	3.2
70-74	0	2.5	6.6
75-79	0.4	4.9	9.6
80-84	1.0	9.7	12.5
85+	1.9	17.7	26.8

Causes of immobility in old age

Physical barriers -

Unfortunately old age brings with it an increased prevalence of a number of diseases; often more than one at a time.

- Joint problems especially osteoarthritis in women
- Neurological deficit: impaired balance, stroke, Parkinson's Disease
- !Previous falls
- Sensory deprivation: deafness, impaired vision
- Cardiovascular and respiratory diseases
- Mental barriers
- Reduced expectations of an 'active' life
- Loss of adaptability and creativity
- Introversion with reduced social contact
- Anxiety and fear of going out (or of allowing others in)

Social barriers

- Retirement brings with it dangers of reduced social contract and a drop in income. The retired person may regard a motor car as an unnecessary expense. Many regret having got rid of their car.
- Living alone: an epidemic problem in ageing women
- Nowhere to go. Insufficient outside interests or activities
- Urgency incontinence. Need to get to toilet quickly
- What are the consequences of immobility?
- Loss of choice

Any of the following choices may be lost to us:

- Being able to get to where we want to be and thus be able to do what we want to do.
- Being alone or with others.
- Having the TV/radio on or off (look round any Hospital ward).

Loss of capability

- Getting to the toilet in time/answering the door/getting upstairs.
- Social responsiveness.
- Worsening physical dependency

An old person's world may thus contract and after becoming housebound he (or more often she) then becomes restricted to the lower half of the house and eventually to what Geriatricians sometimes call a 'triangular' existence - from bed to chair to commode.

A vicious circle **IMMOBILITY (any cause) WORSENING CONFINEMENT IMMOBILITY SENSORY DEPRIVATION DULLING OF SENSORY AND SOCIAL RESPONSIVENESS**

Prevention of Immobility

The seeds of immobility in later life are sown in middle age and prevention involves:

- Maintenance of physical fitness
- Adequate diagnosis and treatment of minor ailments before they become major ones.
- Financial planning - money in our society often gives us the means to maintain mobility.

Patient with impaired mobility

Your report should include, but need not be confined to, answers to the following questions:-

- Is the patient a reliable witness?
- When did he last go out of the house unaccompanied: to use public transport; to cross a traffic-bearing street; to go to the Post Office/Shops/Pub/Church etc.
- Why did he stop going out: because of pain; breathlessness; fear - of what?
- Does he go out accompanied?
- What difficulties are experienced in: ascending stairs; descending stairs; walking; rising from a chair; getting on and off the chair or toilet; getting in and out of bed.
- What aids are used?
- What footwear is worn?
- Examine..... general pattern of movement, gait speed, step length, symmetry; transfer efficiency, style; range of movements of joints; muscle strength, tone, co-ordination.
- What is the causal condition?
- What are the contributory conditions?
- What is the treatment?
- What is the prognosis?

Rehabilitation

Definitions

- **Rehabilitation**
 - the restoration of the individual to the optimal level of ability within the needs and desires of the individual and his or her family.
- **Impairment**
 - damage to an organ or part of the body e.g. hemiplegia.
- **Disability**
 - the way impairment affects the function of an individual e.g. immobility (due to the hemiplegia).

- **Handicap**

- the way impairment and disability affect a persons resettlement into the community e.g. loss of job due to immobility due to hemiplegia. Some disability may produce very different handicaps.

Categories of Rehabilitation

e.g. SPReAd

- **Specific control** of underlying disease or impairment by medical surgical and psychological measures e.g. curative surgery.
- **Prevention** of secondary disability e.g. pressure sores, contractures, etc.
- **Restorative** measures e.g. physiotherapy, continence promotion etc.
- **Adaptation** Including the provision of aids and appliances and structural alterations to accommodation etc.

How it should be done:

Team working - no individual has the requisite knowledge, skills, or expertise to be considered the most important although at any time one may take a lead role and be the prime mover. The family should be considered as part of the team and early involvement is beneficial. Team members need a common philosophy, to share common goals and a knowledge and respect for the expertise and contribution of other team members.

Alternative approach is to have a single multipurpose rehabilitational agent e.g. conductor at la Peto Institute.

Special problems of rehabilitating elderly people

- **Multiple pathology** - complex combinations of disabilities and impairments increases complexity of goal setting, prognosis, etc.
- **Mental impairment** - learning difficulties, inability to grasp new concepts or ways of doing things, behavioural problems.
- **Personality** - a key factor in determining outcome. Complex factors but a few oversimplified stereotypes are:
 - i) **Constructors** - Flexible, tolerant, plan for the future, well adjusted and a pleasure to rehabilitate
 - ii) **Dependents** - pleasant but happy to let others do things. Passive partners and frequently "molycoddled at home".
 - iii) **Hostiles** - Aggressive, constantly complaining, inflexible. Resists all help.
 - iv) **Defensives** - "I'll be alright when I get home". Fear of dependency and resists attempts to help. Rigid and afraid inadequacies may be revealed.
 - v) **Self Haters** - see no future and don't help themselves. Passive but resist encouragement. On discharge sit and do nothing for themselves.
 - vi) **Low expectations** - and low status - "What can you expect at your (my) age".
 - vii) **Carer stress** - strength of pre-morbid relationships, other family commitments, financial and housing problems etc.

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Impaired balance and falls

Balance

Maintenance of man in an erect posture requires balancing a large mass over a very small base. Mechanisms involved include ocular, vestibular and proprioceptive receptors. The whole process is initially learned in childhood. Under normal circumstances the body sways from a fixed point with women having a greater sway than men. Sway also increases with normal ageing.

Ocular mechanisms

We all require visual clues to help prevent falls. In the elderly, both visual acuity and the threshold for light stimulation is reduced. This coupled with poor environmental lighting increases the likelihood of falls.

Vestibular mechanisms

The vestibule is involved with rotatory movements of the head. These mechanisms become relatively inefficient as patients age.

Proprioceptive mechanisms

The main receptors are situated in the cervical interfacetal joints in the neck. Impulses generated in the neck are coordinated in the cerebellum with the efferent pathway being via the medial longitudinal fasciculus. Cervical spondylosis or the wearing of a cervical collar renders these receptors inefficient. Additionally vascular problems in the carotid may lead to balance disturbances.

Falls

There is an increasing incidence of falls with age. Approximately 25% of 70 year olds experiencing at least one fall per year rising to 35% in the over 75s. In the over 65s falls are the sixth commonest cause of, and account for 5% of all deaths. Although only 5% of falls result in fractures and 5% soft tissue injuries, the remaining 90% may result in loss of confidence.

Causes of falls can be summarised as:

- impaired balance due to ageing
- environmental factors
- medical factors
 1. non-specific illness
 2. specific diseases
 3. Neurological and vision
 - a) Confusion (acute and chronic)
 - b) Cerebrovascular disease
 - c) Parkinsonism
 - d) Epilepsy
 - e) Visual impairment
 - f) Vertigo
 - g) Drugs - see below

4. Cardiovascular
 - a. Postural hypotension
 - b. Arrhythmias
 - c. Aortic stenosis
 - d. Other causes of syncope - micturition, cough
 - e. Vasovagal
 - f. Carotid sinus syndrome
5. Locomotor
 - a. Arthritis
 - b. Muscle weakness/myopathy
 - c. Foot problems
 - d. Footwear
6. Drugs
 - a. Sedatives (benzodiazepines, antidepressants, phenothiazines)
 - b. Alcohol
 - c. Drug induced parkinsonism
 - d. Drug induced hypotension/postural hypotension (eg GTN, phenothiazines, prazosin)
 - e. Drug induced electrolyte disturbance (eg hyponatraemia, hypokalaemia)

Management

It is essential to take a good history, particularly with regard to loss of consciousness, position or activity at the time of the fall and specific symptoms such as chest pain. It is important to know how long the patient was on the floor and, if possible, to obtain an eye-witness account. Full examination with particular reference to lying and standing blood pressure, any neurological disorders, vision and gait should be performed. Investigation is guided by history and examination. All identifiable causes such as polypharmacy or untreated Parkinson's disease should be treated. Rehabilitation and the multidisciplinary approach will not only restore confidence but also increase stability and ensure safety if falls occur. A home visit is mandatory when patients present with recurrent falls and will identify environmental risks and help provide a safe environment for the patient.

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Impaired vision

Definition

Blindness - Visual acuity worse than or equal to 3/60

Partially sighted - no strict definition although visual acuity of < 6/18 with field loss is an arbitrary cut-off point

Impaired vision - 6/18 or less

Many elderly people have deteriorating eye-sight and do not recognise impaired vision as a specific problem but relate it all to the changes associated with ageing. However, the vast majority of patients with impaired vision can be improved with simple measures. Patients with a vision of 6/9 are legally entitled to drive. Those with vision of 6/18 or less have difficulty watching television across the room. Those with poor vision are more likely to fall, the vast majority of which are correctable. Thus visual impairment is grossly under-reported as many elderly people accept this as a normal part of ageing.

The ageing eye

Eye-lids

The decrease in elastic tissue of the skin and loss of muscle tone will produce typical appearances of age, namely loose skin fold or baggy eyelids (blepharochalasis) and skin wrinkling. The eyelid can either turn in (entropion) or out (ectropion) due to toxicity.

Cornea

The cornea becomes more opaque with age however this is not often clinically important. Arcus senilis of the cornea - this appears as a whitish ring in the periphery of the cornea with a clear zone separating it from the limbus. The appearance is due to deposition of phospho-lipids in the corneal periphery and can be seen in most people after late middle age. It is harmless and does not effect vision.

Lens

The most important contributor to ageing. It increases in diameter due to the addition to new lens fibres throughout life. This may have the effect of moving the iris anteriorly predisposing to acute angle closure glaucoma.

Vitreous

With age the vitreous becomes more liquid and the fine vitreous fibrils close together giving rise to "floaters". Myopic patients may experience these floaters at an earlier age.

Retina

With increasing age arteriosclerosis of the retinal vessels occurs and this may be observed in the fundus as a narrowing of the retinal arterials and occasional variations in calibre of the vessel.

Macula

Central part of retina shows accumulation of waste products known as drusen. These indirectly interfere with the function of the retinal pigment epithelium and consequent loss of central vision.

Cataract

Definition

A cataract is an opacity in the crystalline lens some of which are so small they do not effect vision. There are many types of cataract but the commonest seen in elderly are senile cataracts. Almost all people over the age of 65 years have some degree of cataract. They may occur earlier in life, especially in people where nutrition has been poor or those who have diabetes mellitus. There are three types of senile cataract, however, the symptoms of all types of senile cataract are similar. They are a slowly progressive, painless decrease in visual acuity. This may take place over several months or years and usually effects both eyes. This is associated with glare in bright lights or sunlight. There are fixed dark spots in their field of vision and there is a deterioration in colour vision. There may be double or multiple images seen with one eye (polyopia).

The types of senile cataract are:

- nuclear sclerosis
- cuneiform cataract
- posterior subcapsular cataract

Treatment:

Spectacles may help those with an early cataract. When vision becomes so poor that an individual cannot do normal activity then a cataract operation is indicated. At present the operation of choice is extracapsular cataract extraction. The opaque lens is removed leaving the posterior capsule intact and a new artificial lens inserted.

Glaucoma

Definition :

This is a condition usually effecting both eyes in which there is visual field loss, raised intraocular pressure and pathological cupping of the optic disc. The raised intraocular pressure causes ischaemia to the optic nerve head with consequent damage to the retinal fibres resulting in loss of visual field. Glaucoma is a common condition in the population and effects 1.5% of people over 40. It is one of the worlds most common causes of blindness. In normal circumstances aqueous fluid drains into the blood stream through the pores in the trabecular system which lie between the base of the iris and the cornea. There is a constant production of aqueous fluid associated with a continuous exit through the eye and thus the intra ocular pressure is kept within the range of 11-22 mm/hg.

In the case of an acute angle closure glaucoma, the obstruction occurs at the periphery of the iris and the pressure rises within a few hours. This is associated with rapid loss of vision, a painful red eye and nausea. The patient may also notice halos. On examination the eye is red, the cornea is cloudy and the pupil is half dilated, oval and does not react to light. There is usually a very high intraocular pressure over 50 mm/hg. In chronic glaucoma the site of obstruction is in the trabecular mesh work and the pressure rises gradually over months and years so that damage to the optic nerve head is often gradual. Because of the gradual rise in intraocular pressure the condition frequently goes unnoticed in early stages. The patient usually presents with loss of vision. Eventually the peripheral loss of vision becomes so great that the patient is reduced to tunnel vision.

Senile macular degeneration

Gradual deterioration of vision over several years and distortion of vision are the symptoms of this condition. As the name implies it is largely confined to the elderly and is one of the commonest causes of registered blindness in western countries. The signs on fundoscopy are usually symmetrical and consist of granular, pigmented and speckled areas confined to the macular region. Although no specific treatment is available, there are several ongoing studies using antioxidants such as vitamin E as there is speculation that senile macular degeneration may be related to oxidative stress.

Diabetic retinopathy

Diabetic patients are prone to eyelid infections, early development of senile cataracts and specific retinal damage called diabetic retinopathy. The development of diabetic retinopathy is related to two factors: i) The duration of the diabetes; ii) The control of the diabetes.

Gradual or rapid visual deterioration may occur. Vitreous haemorrhage or macular haemorrhage will cause sudden loss of vision.

The severity of retinal disease can be assessed by fundoscopy and they are:

- dilatation of veins
- microaneurysms
- blot haemorrhages
- hard exudates
- cotton wool spots
- neovascularisation

When new vessels are present this is called proliferative diabetic retinopathy and effective laser therapy to destroy the new vessels and microaneurysms in the retina can prevent serious haemorrhage at the back of the eye with loss of sight.

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Impaired hearing

Deafness is a common problem in the elderly, causes of which are multifactorial.

Pathology -

Presbycusis is a loss of pure tone hearing in the higher frequency which is an age-related phenomena caused by degeneration and atrophy of the sensory cells and neuronal connections within the cochlea. Additional acquired causes of deafness are either conductive or sensorineural in aetiology.

Symptoms -

Many patients with presbycusis also experience hypersensitivity to very loud speech. Tinnitus is present in approximately 10% of patients in their sixth decade although hearing may not necessarily be lost. Patients with impaired hearing may have problems with sound localisation, particularly in a noisy environment.

Social consequences -

Any inability to communicate experienced by an elderly person leads to loss of independence and social isolation. Additionally, the sufferer is robbed of the ability to listen to radio and television programmes and of telephone conversation. Ignorance may result in the deaf patient being labelled as confused or demented and increasing social isolation may precipitate transfer from the community to residential care.

Treatment -

Treatable causes of deafness such as wax in the auditory canal must be identified and treated. The second step is to provide an adequate hearing aid together with careful instruction, follow-up and encouragement. Additionally, special adaptors for use with the telephone are available. Hearing aids unfortunately magnify extraneous sounds and therefore in certain situations, lip-reading may be helpful.

How to speak to deaf persons

For the hearing impaired the visual clues of speech become very important. Simple actions may determine the ease with which these visual clues can be followed:

1. Keep your face visible and make sure it is well lit;
2. Do not hide your lip movements behind your hands, cigarette, pipe;
3. Keep your head still and do not distract the observers attention with unnecessary hand movements;
4. Attract the observers attention before you start talking;
5. Do not shout, speak clearly, not too fast;
6. Be prepared to write down important facts
- 7. Be patient.**

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Impairment of speech and language

Speech and Language therapists assess, diagnose and treat a range of speech and language disorders of acquired neurological origin: Cerebrovascular accident - head injury - degenerative disease - tumours

Speech & Language

Verbal communication is divided into Speech and Language.

SPEECH

Is the motor process involved in articulation and pronunciation. Disorders of speech are identified as weaknesses of the bulbar muscles and affect intelligibility of communication. Disordered articulation is called **dysarthria**. The total inability to move the articulators for speech is called **anarthria**.

LANGUAGE

Is a neuropsychological process where thoughts and ideas become verbalised. It involves the selection of words to be spoken (semantics) and the formulation of appropriate sentences or phrases (syntax).

Language is made up of comprehension (our understanding of what is said) and expression (what we choose to say). Reading, writing and gesture are all included in language processing.

An impairment of language is called **dysphasia**. This may be a receptive dysphasia affecting comprehension, or an expressive dysphasia affecting expression. Total inability to use language is called **aphasia**.

The distinction between dysarthria and dysphasia is not absolute. Occasionally dysarthria occurs with dysphasia, however, a dysarthric person who has no dysphasia will be able to read, write and gesture normally.

Causes and types of Dysarthria

Dysarthria is caused by lesions in the motor areas of the cerebral hemispheres or by disruption to the integrated action of the upper motor neurones, basal ganglia and cerebellum. Lesions in the brain stem affecting the lower motor neurones can also result in dysarthria.

Clinically, dysarthria may present in a variety of ways depending on the site and nature of the lesion.

- Slurred, weak articulation; weak voice caused by pseudobulbar palsy following cerebrovascular accident. Usually symptoms are unilateral corresponding to a left sided hemiplegia. Brain stem cerebrovascular accident may lead to bilateral signs of dysarthria or anarthria.
- Slurred, scanning and staccato speech caused by cerebellar lesions in multiple sclerosis.
- Dysrhythmic, dysphonic and monotonous voice caused by disorder of the extra pyramidal system in Parkinson's Disease.
- Indistinct articulation, hypernasality, bilateral weakness caused by lower motor neurone disorders, eg., motor neurone disease.

Management of Dysarthria

Speech Therapy involves thorough assessment of bulbar and facial muscles. On the basis of this a programme of exercises aimed at increasing muscle tone, rate of movement, etc., is drawn up for the individual's needs. It is essential to be patient with a dysarthric person and try to make out what is being said. If this is impossible, encourage them to write the message down or use an electronic communicator if this has been provided.

DYSPHASIA

Causes:

Dysphasia is the loss of ability to formulate, express or understand the meaning of spoken words, due to a lesion in the language area of the dominant cerebral hemisphere.

There may also be difficulty in reading and writing, understanding and using gesture.

The cause is usually stroke but dysphasia also results from tumour and other space occupying lesions, head injury and dementia.

Handedness

The left cerebral hemisphere is almost always dominant for speech and language in right-handers. Therefore dysphasia is usually associated with lesions of the left side of the brain.

The left cerebral hemisphere is also dominant for language in about 70% of left handers.

Dysphasia will only rarely result from right hemisphere lesions, but when it does, the patient is likely to be left handed.

Clinical types of dysphasia

Fluent (receptive) dysphasia

Fluent dysphasics speak rapidly with normal rhythm and articulation but their words fail to convey meaning. Their understanding of words is impaired.

Non-fluent (expressive) dysphasia

Non-fluent dysphasics have difficulty forming words and sentences. There may be grammatical errors and word finding difficulties. In severe cases the patient may be unable to talk spontaneously. Conversation is usually understood though not always perfectly.

Diagnostic Approach

Try to determine the patient's abilities in speaking, understanding speech, reading, writing, gesturing and understanding gesture. The language defect, in association with other clinical findings, will give you a clue as to where the lesion is located (predominantly fluent, probably temporal lobe; predominantly non-fluent, probably frontal lobe).

When testing comprehension beware of "social cover up" - many dysphasic patients are still able to follow basic greetings and social-type conversation, eg., "How are you?", when comprehension of other language is very limited. Patients often give the impression that they have understood what is being said when they are actually responding to non-verbal cues, eg., facial expression, tone of voice. Ensure you ask questions that require "Yes" as a response and questions that require "No" as a response, eg., (holding a pen) "Is this a pen?" and later (holding a spoon) "Is this a pen?"

Look out for jargon (meaningless speech), neologisms (structures which sound like words but are not, eg., "bonter", "tupley" and recurrent utterances, eg., Yes..... Yes..... Yes....

When testing expressive language ask questions that are open ended. Ask the patient to name everyday and less common items.

Check the patient's abilities in the use of:-

Conversational speech - Can the patient formulate complete and original sentences?

Social speech/fillers - Does the patient use normal or excessive amounts of standard phrases, such as "How are you?", "You know what I mean"?

Automatic and lists serial speech - Does the patient use "yes" and "no", can the patient recite of numbers, days of the week, etc?

Look out for word-finding difficulties (the patient knows what s/he wants to say but can't find the word) and perseveration (inappropriate repetition of the same word/s in reply to different questions).

Ref. Espir M.L.E. & Rose F.C. (1983) The Basic Neurology of Speech & Language Blackwell Scientific Publications

DYSPHAGIA

Difficulties swallowing can occur following CVA or in association with a neurological degenerative disease. One or more of the following could mean a patient experiencing dysphagia and being at risk of aspiration:

1. reduced conscious level
2. cognitive problems including, a). poor attention or motivation, b). inability to identify food, c). no chewing, d). poor sequencing skills
3. inadequate lip seal or poor or disorganised tongue movement
4. tongue scarring
5. primitive reflexes
6. lack of sensation

All of the above can affect the oral preparation and/or oral stages.

The following can affect the pharyngeal stage:

1. no swallowing reflex
2. delayed swallow reflex
3. lack of nasopharyngeal seal by soft palate
4. poor laryngeal protection
5. no cough reflex/phonation
6. weak or non-existent pharyngeal peristalsis
7. cricopharyngeal sphincter failing to relax

The third stage is the oesophageal stage when reflux may result in aspiration.

Swallowing problems need identifying early to minimize the risk of a) aspiration-related chest infections developing and b) malnutrition or dehydration. Although many patients cough when they aspirate, this is not always the case and the patient may 'silently' aspirate.

If you have any doubt about the patient's ability to swallow safely (i.e. not just to swallow) refer the patient to a Speech and Language Therapist.

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Urinary Incontinence

CAUSES

- Urinary tract infection - This should always be considered in patients presenting with urinary incontinence.
- Constipation - This may cause urinary incontinence by causing bladder outlet obstruction or by reducing bladder capacity.
- Drug induced Diuresis
- Poorly controlled Diabetes Mellitus
- Renal failure
- Excessive fluid Intake
- Nocturnal diuresis (this may be due to autonomic dysfunction)
- Loss of awareness of bladder filling
- Dementia
- Unconsciousness

Urinary incontinence may also accompany less severe degrees of impaired consciousness e.g. secondary to sedative drugs.

Unstable bladder

Patients with bladder instability experience strong detrusor contractions during bladder filling which may be followed by involuntary expulsion of urine. This may be preceded by the sensation or urgency but often no bladder sensation is experienced.

Unstable bladder contractions occur due to loss of the normal cerebral inhibitory effect on the bladder. They are common in patients with global cerebral disease e.g. dementia, cerebrovascular disease. Patients with autonomic dysfunction may also exhibit bladder instability.

Urge incontinence is present when the interval between the desire to urinate and involuntary expulsion of urine is very short. Patients with urge incontinence usually have an unstable bladder.

Immobility/Environmental

Environmental and physical factors may contribute towards urge incontinence. Immobile patients take longer to reach the toilet, especially when there are stairs to negotiate. (When they reach the toilet they may be unable to unfasten their clothes quickly enough.

Urinary retention

- Bladder outlet obstruction e.g. prostatic enlargement.
- Atonic bladder.

Urinary retention may also occur in patients with, spinal cord disease e.g. multiple sclerosis, paraplegia in whom bladder sensation is impaired or absent in association with an atonic bladder.

Stress incontinence -

The underlying problem in female patients with stress incontinence is either pelvic floor muscle or urethral sphincter weakness. Stress leakage occurs with increases in intra-abdominal pressure as a result of standing or coughing especially when the bladder is full.

Pelvic floor muscle weakness may occur due to damage of their innervation during childbirth or as result of chronic straining at stool. Vaginal prolapse may also be present in these patients.

Urethral sphincter weakness may be due to atrophic urethritis which is often associated with pruritus vulvae and frequency of micturition. In the elderly this is usually due to oestrogen deficiency.

Behavioural

Patients with severe dementia and/or psychiatric illness may exhibit severe behavioural disturbances which include inappropriate urination, (and defaecation).

Assessment and Investigation

- History - to include drug history.
- Rectal examination - assess all patients for the presence of faecal impaction and assess the size of prostate in males.
- Vaginal examination - examine for stress leakage, atrophic vaginitis which is associated with atrophic urethritis and can be confirmed by vaginal cytology), and vaginal prolapse.
- MSSU/Urine Cytology - cystoscopy is indicated if haematuria is present without urinary infection.
- Incontinence chart - keep record chart initially for 48 hours. Record:
- Fluid intake (aim for 1500-2000 ml/day)
- Urine output at each voiding (gives indication of bladder capacity)
- Episodes of urinary and faecal incontinence.
- Urodynamic assessment - in most elderly patients it is possible to diagnose the cause of incontinence from the history and examination findings. Urodynamic assessment is indicated when there is difficulty making precise diagnosis, when treatment has not been successful or when surgical treatment is being considered.
- Pelvic floor muscle assessment.

Treatment of Urinary Incontinence

Review medication

- Stop offending drug(s) if possible or use an alternative drug. When diuretic therapy is required a short acting diuretic with a predictable effect should be used e.g. loop diuretic.
- Urinary tract infection - antibiotic medication as indicated from urine culture sensitivity results.
- Constipation - use laxatives or enemas.

Urge incontinence/unstable bladder

- Toileting - bladder drill or timed voiding to empty the bladder before the onset of urgency and/or bladder contraction. The usual interval between micturition is 2 hours. Restrict fluid intake after 6.00 pm. if nocturnal incontinence persists.
- Environmental adjustment - Useful aids/adaptations that could be considered include:
 1. Commode -a commode placed beside the patient's bed or close to where they sit during the day is very useful for patients with nocturia and with limited mobility.
 2. Chemical toilet- for patients living alone this is an alternative to a commode which does not need to be emptied as often.
 3. Bottle urinal- useful for males. Non return valves are available for these.
 4. Toilet seat raise
 5. Frame around toilet
 6. Modifications to clothing e.g. velcro fastenings.
- Anticholinergic drugs - Oxybutynin (3 mg b.d. - 5 mg t.d.s.), for example, may be used to increase bladder capacity and reduce the distending volume at which urgency and/or unstable detrusor contractions occur.

Stress incontinence

A pelvic floor assessment is performed by assessing pelvic floor muscle contractions with a finger in the rectum (male) or vagina (female). The abbreviation PERFECT summarises the assessment:

Power

Modified MRC scale

- 0 - MI
- 1 - flicker
- 2 - weak
- 3 - moderate
- 4 - good
- 5 - strong

Endurance

- hold time for contraction

Repetitions

? how many

Fast

- 30% fast fibres
- 70% slow fibres
- record number of fast twitches

Every

Contraction

Timed

Examination result may be 2/4/5 = P E R

Exercise programme is individual, guided by assessment and should be designed to push the muscle to the point of fatigue with a combination of slow contractions and fast twitches. 2/4/5 assessment suggests treatment should be 5 contractions ("squeeze and lift") held for 4 seconds followed by 5 fast twitches on the hour every hour.

It takes 3-6 months to strengthen the muscles though continence may be restored before then. The exercises should be continued indefinitely thereafter.

Vaginal cones - Use of weighted plastic vaginal cones which are available in sets of 5 (10-100g) are also helpful in pelvic floor rehabilitation. The aim is to start with a light cone which is left in the vagina during normal activity for 10-15 min, 3-4 times per day and kept in place by contracting the pelvic floor muscles and build up to the heaviest cone.

Electrical stimulation of the pelvic floor - techniques for the electrical stimulation of pelvic floor muscles are being developed to augment pelvic floor exercises. The main benefits, however, appear to be due to pelvic floor muscle exercises and until further evidence is available, electrical stimulation should only be used in a research setting.

Oestrogen for atrophic vaginitis - traditionally this has been given in the form of pessaries or vaginal cream. Elderly patients are generally unable to apply these correctly. It is therefore advisable when treating atrophic urethritis to prescribe either transdermal oestrogen 25 mcg/week for 6 weeks or oral oestrogen, ethinyloestradiol 10 micrograms daily for 21 days as a single course of treatment. Oestrogen alone does not usually cure incontinence but is effective

for alleviation of the atrophic symptoms. Oestrogens have an effect on urethra, reduce detrusor instability and alter vaginal flora.

Bladder neck repair - this is indicated for patients with stress incontinence who do not respond to the above treatment and are fit for an anaesthetic.

Vaginal ring pessaries to control vaginal prolapse are usually ineffective in the treatment of stress incontinence.

Treatment of other causes

Prostatic enlargement

Prostatic enlargement may cause bladder outflow obstruction leading to urinary retention and overflow incontinence. The treatment of choice is transurethral prostatectomy. Post-operatively many patients experience urinary incontinence. This is usually a short term problem and can be cured by bladder retraining. More persistent urinary incontinence may be experienced by a few patients due to the presence of another cause of urinary incontinence e.g. unstable bladder.

Atonic bladder

- these patients may be managed by intermittent self-catheterisation.

Nocturnal diuresis

-(autonomic dysfunction) - additional treatment possibilities include 10 degrees head up tilt at night and possibly fludrocortisone or antidiuretic hormone (DDAVP) at night for resistant cases.

Loss of awareness

Usually secondary to severe dementia - these patients should be managed by toileting at planned regular intervals.

Behavioural

Behaviour modification may be successful for these patients.

Control of Urinary Incontinence

Control aids are used for patients with intractable incontinence and for selected patients undergoing treatment for their incontinence.

- *Pads and Pants* - A full range of pads and pants are available for light to heavy incontinence. These enable social continence to be achieved.
- *Underpads* - Disposable under pads should not be used in the management of incontinent patients. Reusable underpads (absorbent bed sheets) are useful in the management of patients with intractable heavy nocturnal urinary incontinence. They are easily laundered and dry quickly. However they should not be used if the patient is also faecally incontinent.
- *Penile sheaths* - These are appropriate for male patients with moderate or heavy incontinence especially if this is only nocturnal. They are contra-indicated in men with latex sensitivity. The penile shaft needs to be at least 4cm long for successful fixation. Each sheath system is accompanied with fixation instructions which should be followed carefully.
- *Body-worn urinals* - A disposable pouch system is available which is effective for some female patients.

Catheters

Catheter material - Many catheters currently on the market are available as uncoated latex. Some latex however can contain chemical additives which may constitute up to 6% of its content. These can be leaked from the latex and in some cases have led to urethral irritation and stricture

formation. The lumen of uncoated latex catheters soon becomes encrusted with struvite which will eventually block the catheter. This invariably happens subsequent to the appearance of infecting bacteria in the urine. Only products which are non-toxic should be used for long-term drainage e.g. hydrogel coated (Bard Biocath), some All-Silicone or some Silicone Elastomer coated catheters. Teflon coated catheters are now available as an alternative to latex for short to medium term catheterisation. Only those Teflon coated catheters that are non-toxic should be used.

Short-term catheterisation - indications Incontinence in an unconscious patient Heavy incontinence in a patient with sacral pressure sore(s) Urinary retention (may require suprapubic catheterisation)

Long term catheterisation - This is sometimes associated with reduced life expectancy and should be avoided unless there is intractable urinary incontinence which cannot be contained by other means.

Catheter size - It is important that a small balloon (10 ml) and a small diameter (12-16Ch) catheter is used. Larger balloons increase the risk of catheter expulsion or bypassing with unstable detrusor contractions. Large diameter catheters cause discomfort and increase site risk of leakage.

Female patients where appropriate should only be catheterised with a female length catheter (females 23+cm), males with a standard length (40-46 cm).

Collection System - The catheter should be connected to the inlet tube of a leg bag. The leg bag should be positioned such that it is concealed from view by attaching it to the thigh or to the lower leg when trousers are to be worn. The bag should be emptied at regular intervals during the day by opening the tap. Ensure that the tap can be easily opened and closed by the patient or their carer. At night time, a night drainage bag should be connected to the leg bag and the leg bag tap opened. The night bag should be positioned on a stand beside the patient's bed.

Catheterisation and urinary infection - Urinary infection is inevitable with long term catheterisation. Asymptomatic bacteriuria however is not an indication for antibiotic treatment. Systemic illness in association with urinary infection is an indication for treatment. Bladder washouts used prophylactically do not prevent urinary infection and there is now evidence which suggests that the use of regular bladder washouts may encourage colonisation with the resistant bacterial strains.

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Faecal Incontinence

Many mechanisms that contribute towards the maintenance of faecal continence are determined by the anatomy in the anus and rectum.

The internal anal sphincter prevents faecal leakage by maintaining resting pressure above rectal pressure. The external anal sphincter makes little contribution to resting pressure. Its main contribution to continence lies in its reflex activity in response to rectal distension or increased intra-rectal pressure, such as coughing. The anorectal angle is normally maintained by the puborectalis muscle. Continence may be lost when it exceeds 110 degrees. Other factors include anal sensation, the slit shape of the anal canal and anal cushions (vascular projections that aid closure of the anal canal).

Causes of faecal incontinence

The causes of faecal incontinence are variable. They include:

- anorectal incontinence (idiopathic faecal incontinence)
- colorectal disease
- diarrhoea
- faecal impaction
- neurological (including dementia and unconsciousness)
- immobility.

Young faecally incontinent patients who are mostly female, tend to suffer from anorectal incontinence, while in the elderly the aetiology is usually multifactorial.

Anorectal incontinence - The main abnormality in anorectal incontinence is weakness of the external anal sphincter and pelvic floor muscles. Although this may be due to surgical or traumatic division of the anal sphincters, most cases were, until recently, termed idiopathic. Atrophy of the external sphincter and pelvic floor muscles owing to pudendal neuropathy has now been traced to chronic straining at stool and difficult childbirth (especially with forceps) for many of these patients.

External sphincter weakness in old age is also caused by denervation, but age-related changes in the spinal cord appear to be the cause, rather than pudendal neuropathy. The presence of faecal incontinence in elderly people, however, does not appear to be related to the degree of external sphincter weakness. Age related external sphincter weakness may, however, be a predisposing factor for the development of faecal incontinence in the elderly because external sphincter pressures even in continent elderly subjects are similar to those recorded in younger faecally incontinent patients. Reflexion traction of the external sphincter in responses to rectal distension is also absent in many elderly individuals.

Low anal resting pressure (internal sphincter pressure) is one of the main factors leading to faecal incontinence in elderly patients, diabetic patients and in those with perineal descent or rectal prolapse. The suggestion that faecally impacted patients are incontinent because of stretch or reflex inhibition of the anal sphincter muscles has recently been disproved.

Anal sensation is impaired in incontinent patients. It is impaired in most elderly people, but more so in incontinent elderly patients. Incontinent patients do however, have normal rectal sensation but many leak faeces before they are aware of rectal distension, probably because anal sphincter relaxation (which is necessary for normal defaecation) occurs before they felt the call to stool. Reflex external sphincter contraction in response to rectal distention is also absent in many of these patients.

Colorectal disease

The following causes should be considered:

- infective diarrhoea
- carcinoma of the rectum or colon
- proctitis or colitis (secondary to inflammatory bowel disease or ischaemia)
- diverticular disease
- drugs (laxatives)

Faecal impaction

This is the commonest cause of faecal incontinence in the elderly. Traditionally, this term has been used to describe faecal loading of the rectum with hard faeces. Many elderly patients, however, have massive faecal loading with soft or even liquid stool. Faecal impaction is better defined therefore as faecal loading of the rectum with stool of any consistency.

A history of chronic constipation, although common in these patients, is not always present. Deficient colonic propulsion, defaecatory difficulty and impaired rectal sensation may all contribute towards the development of constipation and/or faecal impaction in these patients. Colonic propulsion normally occurs via a series of colonic mass movements.

The main stimuli for these mass movements are physical activity and the ingestion of food. It is not surprising therefore that constipation is relatively common in immobile patients. Whole gut transit time is often used as a measure colonic propulsion.

It is markedly prolonged in constipated elderly patients and in many young constipated patients. Patients with slow transit times appear to be a heterogeneous population. In some there is evidence of a myenteric plexus abnormality which could be either the primary cause of their delayed transit or secondary to long term laxative abuse.

The delay in gut transit in constipated elderly patients appears to occur mainly in the pelvic colon and rectum, possibly as a result of mechanical obstruction by faecal masses impacted in the rectum. Intrarectal pressure normally increases and anal resting pressure is inhibited during defaecation, to produce a pressure gradient.

The increase in intrarectal pressure may be produced by rectal smooth muscle contraction, contraction of the abdominal muscles and diaphragm or a combination of both.

A number of defaecatory abnormalities have recently been demonstrated in young women with severe slow transit constipation.

Obstructed defaecation may be caused by a paradoxical contraction of the external sphincter during attempted defaecation, rectal intussusception or failure of the anorectal angle to increase during defaecatory straining.

The ability to defaecate is also dependent upon stool consistency and it is more difficult to defaecate hard stool than soft stool. Some elderly constipated patients develop a megarectum, probably owing to a myenteric plexus abnormality.

Faecal soiling in impacted patients is more common when a stool is soft. The increased anorectal angle in these patients may also contribute towards their incontinence, as may the treatment of the faecal impaction. Immobile patients tend to be slow to respond to the "call to stool". They are therefore liable to incontinence when they are given a laxative, especially with potent preparations. This should always be remembered when elderly patients are being prepared for a barium enema, colonoscopy or surgery.

Neurological

Neurological causes of faecal incontinence may be divided into two types.

- These are loss of awareness
- Behavioural problems.

Loss of awareness may be owing to impaired consciousness in severely ill patients, as in those with stroke. Dementia may also produce loss of awareness of the "call to stool".

Rectal motility is normally under inhibitory cerebral control. This control is absent in many demented patients, who have rectal contractions similar to the uninhibited detrusor contractions that may cause urinary incontinence.

Rectal distension studies using simulated stools suggest that in elderly incontinent patients involuntary faecal expulsion is often preceded by a strong rectal contraction. It appears, however, that although uninhibited rectal contractions may play a role in the expulsion of soft stool, similar to the release of urine that follows an unstable detrusor contraction, they are unlikely to contribute to the expulsion of hard stool.

Many demented patients with behavioural disturbances defaecate in inappropriate places. These patients pose a major care problem in local authority homes and psychogeriatric units, as treatment for them may be difficult since the behavioural problem is the result of an untreatable primary condition.

Treatment of faecal incontinence

This varies according to the cause.

The aim in the treatment of patients with faecal impaction and constipation is to empty the rectum and colon. Patients impacted with soft stool should not receive lactulose as this will exacerbate the situation.

The preferred treatment is to empty the rectum from below by administering an enema each day until the faecal mass is cleared.

Phosphate enemas are the most commonly used. They increase the water content of the stool which then induces defaecation by stimulating rectal contractions. Occasionally they are ineffective usually because the enema is not retained and in these patients the best result may be achieved with a microenema or suppository but some patients will require manual evacuation, especially if they are impacted with very hard faeces.

More drastic treatments to produce bowel clearance are sometimes required. For instance, sodium picosulphate (a stimulant laxative) with magnesium citrate (an osmotic laxative) may be appropriate. Sodium picosulphate is a very potent preparation, but its effect is unfortunately difficult to control. The starting dose should therefore be reduced to one quarter of the manufacturers recommended dose.

Many elderly impacted patients have a continuing tendency to constipation even after their impaction has been cleared. The next step in the management is preventing the recurrence of constipation. Increased dietary fibre, however, is only partially effective in restoring constipated patients' stool weight and gastrointestinal transit time to normal.

High fibre intake has also been shown to be associated with colonic faecal loading in immobile elderly patients. It is therefore not advisable to recommend increased dietary fibre routinely for constipated elderly patients, as this may add to their existing constipation. It may also increase their risk of faecal incontinence.

An alternative approach is to use a laxative, the choice of which must be guided by the character of the stool and the patient's ability to defaecate.

An osmotic laxative, such as lactulose, is indicated for patients with hard stool.

A stimulant laxative is required for patients with soft or formed stool.

The most commonly used are senna, sodium picosulphate, and bisacodyl. They exert their effect approximately 10 hours after ingestion and should therefore be given at night to avoid faecal incontinence.

Although laxatives are often administered to elderly patients to prevent recurrent constipation they are not always successful. Many patients require regular emptying of the bowel from below, using either enemas (preferably miniature ones) or suppositories (glycerine or bisacodyl). Diarrhoea is often associated with faecal incontinence.

While the cause is sought, antidiarrhoeal agents should be administered to alter stool consistency. This alone should restore faecal continence. In the initial stages, however, application of a faecal collection device can help.

Codeine phosphate has been the traditional anti diarrhoeal drug, but the action of the newer antidiarrhoeal agent Loperamide is better understood. It may prevent faecal incontinence by mechanisms other than just that of changing stool consistency.

Loperamide is a synthetic opiate, devoid of central nervous system side-effects. It has a direct effect on the internal sphincter and has been found to increase anal resting pressure and to improve the ability of patients with chronic diarrhoea and faecal incontinence to retain liquid stool in the rectum.

Treatment of the faecally incontinent demented patient must be carefully planned. Such patients are usually unaware of the presence of faeces in the rectum and the need to defaecate; their incontinence is usually secondary to faecal impaction. This should be treated as described above. If faecal incontinence and/or constipation continue then a regimen of planned defaecation should be implemented.

Defaecation should be stimulated by the use of an enema 1-3 times weekly. (Most patients need twice-weekly enemas). To prevent leakage between enemas, a constipating drug (loperamide or codeine phosphate) may be needed.

The principles for treating patients with anorectal incontinence are similar to those described above. Maintaining firm stool will cure most, but resistant cases may need further treatment. Biofeedback, electrical stimulation and post-anal repair have all been tried.

The operation of post-anal repair appears to be a moderately successful method of restoring continence to patients with anorectal incontinence, particularly in those aged 50-69 years. Results in older patients are not as good, probably because their problems are multifactorial.

Faecal incontinence should be seen as a curable and preventable problem. It must not be accepted as inevitable.

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Nutrition

Energy and Protein Requirements

Energy requirements of elderly people are slightly lower than those for younger adults. However, requirements for all other nutrients are as for the young adult. Therefore, the diet needs to become more nutrient dense, i.e. a diet which provides a large number of nutrients relative to the overall energy intake.

Energy requirements are determined by many factors including the basal metabolic rate, levels of activity, the presence of infections or fever and the need for weight loss or gain. It is often assumed that with old age, organ function decreases, physical activity decreases, lean body mass decreases and hence, metabolic rate decreases. This may not necessarily be the case if physical activity is maintained.

Protein requirements remain similar to those for younger adults. If energy intakes are inadequate, the body uses protein stores (muscle) to obtain energy. Therefore, an adequate energy intake is essential to minimise protein loss. This is particularly important during illness when energy and protein requirements are usually higher.

Nutritional requirements in illness

Good nutrition is essential for maintaining health but is also very important during periods of ill health and during recovery from illness. If periods of ill health occur repeatedly, nutritional status may gradually decline over several months or years and remain unnoticed because of its slow, progressive nature.

Illness may affect:

1. Food intake
2. Nutritional requirements - e.g.
 - a rise in body temperature of 1°C increases energy requirements by 10%
 - an elderly person with pressure sores is likely to have higher requirements for energy, protein and nutrients such as vitamin C, iron and zinc to help heal the wound.
3. Nutrient absorption - e.g.
 - will be reduced in any condition where the nutrients are lost, such as vomiting or diarrhoea
 - many commonly prescribed drugs can affect absorption and therefore nutritional status (see below examples).

Surgery is likely to affect nutritional status and good nutrition before and after surgery is very important to:

1. speed up wound healing;
2. reduce the risk of postoperative complications;
3. reduce the length of hospital stay;
4. prevent weight loss and deterioration of nutritional status.

Malnutrition in the elderly

Factors contributing to malnutrition in elderly people may be divided into primary and secondary:

<i>Primary Social & Environmental</i>	<i>Secondary Physical & Mental</i>
Ignorance	Impaired appetite (due to disease or depression)
Poverty	Poor dentition
Social Isolation and loneliness	Reduced absorption
Mental Disturbance	Increased requirements (Eg disease trauma, surgery, burns , pressure sores)
Physical Disability	Alcohol Intake
Chronic disease states	Drug therapy
Iatrogenic (Eg. low fat diet for gallstones, Problem if patient is underweight)	

* Examples of drug-nutrient interactions include:

- antibiotics (neomycin) which results in malabsorption of fat soluble vitamins and impairs iron absorption; tetracycline
- steroids which impair vitamin C status
- metformin may result in vitamin B12 deficiency

Malnutrition is high among the elderly with studies showing that institutionalised elderly are at the greatest risk. Elderly in the community fare better, although living alone may result in impaired nutrition for a variety of reasons.

Detection of malnutrition

Malnutrition is difficult to diagnose but risk factors such as pressure sores; burns; fractures and other trauma increasing catabolism, e.g. surgery, anorexia, successive periods of starvation for test i.e. the NBM syndrome, renal/liver failure, and should be considered in all elderly patients. Indicators of malnutrition include clinical features such as weight loss, bruising/bleeding gums, angular stomatitis, poor wound healing and osteomalacia, together with laboratory evidence of anaemia or hypoalbuminaemia

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Carers

Although most elderly adults are able to live independently, taking care of an older family member is a concern of many people. Research has shown that families provide most of the care that the elderly need and that the majority of care-givers are women, with 29% of care-givers being daughters, 23% wives and 19% husbands. Care-giving spouses tend to be in their late 60s or 70s, so the carers themselves are often elderly and have some degree of disability. Male care-givers are older on average than females.

Government policy and the realisation that the elderly often cope and manage better in their own environment, has led to the emphasis on 'community care'. It has often been suggested that the extended family and its ensuing support networks have disintegrated over the years, with increasing emphasis being put on the provision and use of formal support networks. However, there are, however, extensive informal support networks.

The problems of caring

Physical exhaustion may result from lack of sleep, heavy physical work, particularly if the person requires help with activities of daily living such as washing, going to the toilet, eating and walking. Physical exhaustion is often compounded by essential activities which require to be performed such as laundry and the housework. Behavioural problems also add to the physical load.

Physical disease may be present in elderly carers or may result from prolonged caring.

Psychological distress is seen in 50% of carers and may lead to anxiety and depression. Having an elderly relative living with a couple may lead to marital disharmony.

Isolation due to alteration in the carers lifestyle, particularly when caring for somebody who requires 24 hour supervision such as an elderly person with dementia.

Financial problems caused by increased heating, laundry and food bills may be incurred by carers. Special aides are often bought by carers to bypass waiting lists. Returning to work may be impossible and transport may become increasingly costly.

Providing support

Health care professionals should be aware that carers are at risk and should endeavour to identify those requiring help at an early stage which minimises stresses and strains of caring. Additional support can be provided by respite care, either in day care centres, or through respite admission to hospitals. Shorter duration respite may enable carers to have a break from an otherwise 24 hour situation. Additional help with personal care and household tasks provided by home care service or district nurses again alleviates the carer. Counselling is essential particularly when a carer first takes on this role. Additional financial help in the form of attendance allowance, mobility allowance and invalid care allowance are often available. Psychological support may be provided, particularly through support groups such as the Stroke Association.

Elder abuse and neglect

Violence in the home and abuse of family members who are less capable of defending themselves have always existed, but the problems have been hidden until recently. In 1978, the issue of ELDER ABUSE and neglect started to receive attention as a result of an increasing awareness of the burgeoning of a number of older adults in our society and their unique problems; as a result of the ground work laid by practitioners who treat children and women suffering from domestic abuse; and finally due to the evolution of a society that is more compassionate than it once was.

Research into elder abuse and neglect came in three waves. During the late 1970s and early 80s it was accepted that elder abuse existed and that the usual victim was over 75 with physical

and/or mental impairment. The abuser was generally a relative in the same household but could be other society members such as the GP, the lawyer or bankers. During the late 1980s the existence of elder abuse and neglect was confirmed and specific cases found that the elderly were not necessarily more frail but were those with least social support. Additionally it showed that the abusers were frequently dependent on the elder, usually financially or for housing. The research of the 1990s is based on randomised population surveys looking more at the nature of the circumstances and the incidence of elder abuse.

Types of abuse

Abuse may be physical, psychological, financial or violation of basic rights. Physical abuse includes assault, trauma, bruises, burns, fractures, dehydration, malnutrition, pressure sores, hypothermia and over-sedation. Psychological abuse includes threats of abandonment or placing in a nursing home or verbal abuse. Violation of basic rights includes not being able to vote, practice ones religion or open ones own mail. Financial abuse is more common as elders have more assets, particularly their own home and their savings.

Reasons for abuse

Culmination of intolerable stress carer lashes out after years of isolation, sleep loss, emotional stress, and physical labour. Dependency of the victim, the transgenerational nature of family violence, and the personality of the care-giver. Cramped or intolerable living conditions. Mental impairment of the care-giver psychiatric disorders, dementia, substance abuse, or sociopathic personalities. Low value placed on the elderly in this society, especially older women. Ignorance of the problems of increasing age deafness may be seen as awkwardness, immobility as laziness etc. Greed alone may be a factor.

Treatment of the problem

The primary aim in treatment is the recognition and reporting of elder abuse. Stopping of abuse may require legal services particularly to block bank accounts and to remove guardianship. The reversal of physical effects may be simple as the provision of meals and home helps. Counselling of both victims and abusers is vital as is provision of respite for the carer and reduction of social isolation. Education of all carers into the nature of ageing and conditions associated with it minimise these as reasons for abuse.

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Law and the elderly (U.K.)

Although the law applies to everyone, there are certain aspects which become particularly associated with the needs of elderly persons.

Testamentary capacity

The law requires that a person making a will has a 'sound disposing mind'. Doctors are often asked to assess a patient prior to making a Will. It is important that accurate records are taken and kept of the assessment in order that your decisions can be justified by contemporaneous note keeping.

In addition to establishing and documenting the absence of cognitive impairment it is necessary to specifically assess the following:

1. Does the patient understand the nature of the act of making a Will and its effects?
2. Does the patient know the nature and extent of his/her property?
3. Does the patient know which persons have a claim upon his/her property?
4. Can he/she form a judgement on the strength of the claims made by these people on his/her property?
5. Has the patient expressed him/herself clearly and without ambiguity (this need not necessarily be in writing as nods and gestures can be allowed)?

Section 47 (U.K. ONLY)

Section 47 of the 1948 National Assistance Act gave the Medical Officer of Health the power to apply to a Magistrate for the compulsory removal of persons who:-

1. are suffering from grave chronic disease or being aged, infirm or physically incapacitated, are living in insanitary conditions **AND**
2. are unable to devote to themselves and are not receiving from other persons proper care and attention.

The original Act required 7 days notice in order to authorise a persons detention in a suitable hospital or other place (e.g. Nursing Home) for a period not exceeding 3 months.

Because of the tragic death of a patient during the 7 days notice the original Section 47 Act was amended in 1951 as: 1951 - National Assistance (Amendment) Act of 1951

This allowed the compulsory removal of the patient to be effected immediately providing that the Medical Officer of Health's opinion was supported by that of another registered medical Practitioner. The period of detention is limited to 3 weeks.

The Section 47 Act is not commonly used with only around 250 cases admitted per annum in the whole of the UK. It is good practice that Geriatricians be involved and it is probably better that patients compulsorily detained are initially admitted to hospital where they can be given the full benefit of a medical assessment.

It is important to realise that both parts of the Act have to be satisfied and the Act is inapplicable to patients who are allowing care to be given in their own home.

Usually we are able to persuade patients to come into hospital for a full appraisal and Section 47 Act is not well known.

Power of Attorney (UK)

This is a legal document which gives total control of a persons financial affairs to someone else. It must be made by a mentally competent person but ceases to be valid once the patient loses the mental capacity to withdraw it. This is only really of any use for physically incapacitated patients who wish their attorney to deal with their affairs.

Enduring Power of Attorney (UK)

This is a document taken out by a person when they have mental capacity, which will continue if it is subsequently lost. This is the preferred method for elderly persons to safeguard their financial affairs.

Once it is decided that mental capacity is lost the Enduring Power of Attorney must be registered with the Court of Protection. This usually requires that the document is accompanied by a doctors opinion that the patient has now lost the mental capacity to make decisions. The patient concerned is notified and if they disagree and feel that they have not yet lost their mental capacity they may challenge the decision so providing some safeguard. It is wise that two attorneys are established, one person being a family member (usually a grown-up child) and the other to be the representative from a legal firm.

Court of Protection (UK)

This is an Office of the Supreme Court of Judicature under the direction of a master, deputy master and assistant masters. The Court's primary function is to safeguard the financial interests of a patient by providing for his/her maintenance and that of his/her family dependants and management of his/her property. It can take months to grant receivership which is usually granted to either a firm of solicitors or to a relative, but the Court itself has to oversee all the expenditure which it must also approve. This is really the only mechanism of controlling the financial affairs of a mentally incompetent patient if no Enduring Power of Attorney has been produced. This is extremely expensive and is not to be recommended if it can be avoided.

The family solicitor will usually arrange for the patient to be examined by a doctor who will determine whether or not the patient is capable or incapable of managing and administering his/her property and affairs by virtue of mental disorder. The patient is usually served with notice of proposed proceedings so they may protest if they feel they have not yet lost their powers. The doctor may choose to recommend that the service of notice is dispensed with if either the patient is incapable of understanding it or that service would be injurious to health or for any other reason.

The Court of Protection is usually receptive to providing things which will improve the quality of life of a patient. For example, they may well fund a holiday for both themselves and their carer and may be in a position to approve purchase of a vehicle, if funds permit, in order to allow for trips out etc.

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Loss and bereavement

Population statistics from the 1991 Census show that there are 3 million widows in Britain and ¾ million widowers. One man in 35 is a widower, 1 woman in 9, but over 65s 1 man in 6 and 2 women in 5. On Merseyside the figures for the over 65s are 1 man in 5 and 1 woman in 2.

The grief process

Grief is a normal, natural reaction to loss with most people experiencing loss or bereavement going through the following stages usually in this order but not necessarily so. In the normal course of events they will eventually emerge from most of them, there are however some who will not move forward through the various stages and this needs to be identified particularly when there are complicating factors such as this person providing the care for another or a underlying mental illness.

1. **SHOCK** for a few days, including unreality and denial.
2. **EMOTIONAL RELEASE**, often around time of funeral. Weeping to be welcomed.
3. **DEPERSONALISATION**, erratic behaviour, unreality. Futile searching. Throwing away of important documents. Hearing the sounds, even seeing the person. Feeling the deceased is near -this is often reassuring rather than distressing.
4. **PHYSICAL SYMPTOMS OF FEAR**, distress. Tight breathing etc.
5. **DEPRESSION** - understandable!
6. **GUILT FEELINGS** about life lived, treatment of deceased, could I have done something to prevent death?
7. **DEEP HOSTILITY** to something or someone, irrational anger towards medics, ambulance service, undertakers, clergy, God, even deceased.
8. Degree of **WITHDRAWAL** from normal activity.
9. **GRADUAL REORIENTATION**
10. **ACTUAL READJUSTMENT**, even freedom to do new things.

Bereavement in the elderly may be different from the younger person in that there are special factors which need to be considered.

1. Often a greater degree of readiness.
2. Different expectations of others in community.
3. Different standards of mourning - stiff upper lip etc.
4. More physical, less psychological symptoms recognised.
5. Happens amidst many other losses which multiply effects, eg retirement, physical disabilities, poorer mobility, etc.
6. Less willingness, expectation, willingness to return to 'normal' living.
7. Increased possibility of suicide.
8. Trying to find solace in rapid succession of partners.
9. Often feelings that children should be doing more.

Nevertheless most elderly people do recover and go on to have a good quality of life. Help is available for all persons who have been bereaved through Cuse Bereavement Care which provides counselling, social groups and activities, advice and teaching. Their Liverpool branch is found at 25 Hope Street, Liverpool L1 9BQ. National Headquarters is found at Cruse House, 126 Sheen Road, Richmond, Surrey TW9 1UR.

Reference Bereavement: Studies of Grief in Adult Life by Colin Murray-Parkes (Penguin)

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Palliative Care

DEFINITIONS

Palliative care is `The active total care of patients whose disease is *not* responsive to *curative* treatment. Control of pain, of other symptoms, and of psychological, social and spiritual problems is paramount. The goal of Palliative care is achievement of the best quality of life for patients and their families. Many aspects of Palliative Care are also applicable earlier in the course of the illness in conjunction with anti-cancer treatment`.

Ref: Pain Relief and Palliative Care (Technical Report Series 804 - Geneva) W.H.O. 1990.

Palliative Medicine is `The appropriate medical care of patients with active, progressive and advanced disease for whom the prognosis is limited and the *focus* of care is the *quality of life*. Palliative medicine includes consideration of the family's needs before and after the patients death`.

Ref: Association for Palliative Medicine of Great Britain and Ireland. Palliative Medicine Curriculum. Southampton: Association for Palliative Medicine; 1992.

Specialist Palliative Care services are `Those services with palliative Care as their core speciality. Specialist Palliative Care services are needed by a significant minority of people whose deaths are anticipated, and care may be provided:

- Directly through the specialist service
- Indirectly through advice to a patients present professional advisers/carers

Ref: National Council for Hospice and Specialist Palliative Care Services. Specialist palliative Care: A statement of definitions. Occasional Paper 8 London October 1995.

The following are areas to consider in this Speciality;

Communication

1. Basic communication skills e.g. active listening, acknowledging, summarising, reflecting back open and closed questions, appropriate responses
2. Breaking bad news
3. Preventing and dealing with collusion
4. Handling difficult questions and uncertainty

Pain Control

Relates to the principles of pain control in this situation

1. The concept of total pain
2. The W.H.O. analgesic ladder
3. Titration of morphine
4. Appropriate use of analgesia (morphine is not a panacea for everything)
5. Matching appropriate analgesia to the type of pain
6. Conversion from oral morphine to syringe driver

Control of other Symptoms

1. Nausea and vomiting
2. Types of drugs to use
3. Routes of administration
4. Constipation

Psychological Support

1. Life threatening illness
2. Peoples reactions to life threatening illness and how to deal with such reactions
3. Spirituality - meaningfulness of life. Guilt, anger

Ethical Consideration

1. Principle of autonomy
2. Truth telling
3. Confidentiality
4. Whose life is it

The Critical Care Pathway

This particular pathway deals with the last days of a patients life and through specified criteria indicates when a patient is considered to be dying. It is a multidisciplinary tool designed to indicate what appropriate action is to be taken to provide pain and comfort measures in the journey of the dying patient with relevant support for the family or those near to the patient.

Useful reading list:

- A guide to symptom relief in advanced cancer. Claude F.B. Regnard, Sue Tempest.
- A-Z Pocket book of symptom control. Peter Kaye - Pub. EPL Publications
- Pain relief in advanced cancer. Robert Twycross
- The Management of terminal malignant disease. Saunders and Sykes. Pub. Edward Arnold.
- How to break bad news - A guide for Health Care Professionals. Dr. Robert Buckman - Pub. Papermac.
- Oxford Textbook of Palliative Medicine. Derek Doyle, Geoffrey W.C. Hans, Neil Macdonald. Pub. Oxford Medical Publications.

MAIN INDEX

Sub Index

Drugs and the elderly

Drug prescribing in the elderly is important to consider because:

1. over 65s constitute » 18% of the population but receive 39% of prescribed drugs (i.e. twice as many as younger people)
2. chronic illness and multiple pathology increase with age, leading to polypharmacy
3. pharmacokinetics and pharmacodynamics may be altered by age and disease
4. 1,2 and 3 increase susceptibility to adverse drug reactions (ADRs)

In Cartwright's survey *Elderly People: Their Medicines and Their Doctors* (1988): it was found that 70% of sample had been prescribed medication (average 2.8/person) with the most widely prescribed drugs being

- Diuretics 25%
- Analgesics 20%
- Hypnotics, sedatives and anxiolytics 15%
- Antirheumatic drugs 15-20%
- β -Blockers 11%
- Digoxin 6%

Duration and review of treatment -

60% of prescriptions had been given for more than 2 years, 30% for >5 years and 16% for >10 years. 88% were by repeat prescriptions and 40% had not been discussed with a doctor for ³6 months.

Prescription errors -

Inappropriate dosage, pharmacologically "unsound" prescriptions, duplication (5%), potentially harmful interactions (20%) were noted.

Comprehension and compliance

Drug compliance (70% as prescribed) and drug knowledge (10% with no or erroneous knowledge) comparable to younger age groups.

Concordance

30% of prescribed medicines identified by GP unrecorded by patients; doctor unaware of 36% medicines reported by patient. Inaccurate GP and hospital records and independent prescriptions from OP clinics, GDH contribute.

THE PATIENT

Pharmacokinetics (What the patient does to the drug)

1. **ABSORPTION** -age changes of limited clinical relevance, but formulation, rate of gastric emptying, effect of food, effect of disease on small bowel remain important factors.
2. **DISTRIBUTION** - body fat, $\bar{\bar{}}$ lean body mass results in increased volume of distribution (Vd) of lipophilic drugs (e.g. benzodiazepines) and reduced Vd for water-soluble drugs.
3. **PROTEIN BINDING** less affected by age per se than by disease. Changes in plasma albumin or "reactive" proteins may be clinically relevant for those drugs which are extensively protein-bound (e.g. warfarin, sulphonylureas).

4. METABOLISM

- Renal: For water soluble drugs, drug clearance depends on GFR (and secretion). GFR usually declines with age (chiefly because of disease). Serum creatinine is not a good indicator of renal function in the elderly, as endogenous production diminishes with age. If renal function is impaired, water-soluble drugs, especially those with narrow therapeutic index (e.g. digoxin, aminoglycoside) should be given in reduced dose (the loading dose is unchanged).
- Hepatic: reduction in liver blood flow with age contributes to reduced clearance of drugs which undergo extensive first-pass metabolism (e.g. propranolol, verapamil, warfarin and many psychotropics).

Oxidation and other Phase I (mono-oxygenase) reactions may show some reduction but conjugation, Phase II reactions are usually unaffected by age.

As in younger people, factors such as cigarette smoking, alcohol consumption, diet and nutritional status, disease and concomitant medication affect drug metabolism. Enzyme induction (but not inhibition) is blunted in old age. Genetic factors (slow/fast acetylators, oxidative polymorphism) are unchanged with age.

Pharmacodynamics (What the drug does to the patient)

Changes in receptor responsiveness, target organ and sensitivity homeostasis are as important as kinetic factors in explaining the variability of drug responses in the elderly.

Receptors and target organs

Most studies of β -adrenoreceptor-mediated responses (to both agonists and antagonists) have shown some reduction with age. Other drug receptor sites have been less well studied. Increased brain sensitivity is the likely mechanism to explain psychomotor impairment with psychosedative drugs (e.g. benzodiazepines, anticholinergics, opiates). Coagulation factor synthesis is more sensitive to warfarin, leading to reduced dose requirements in elderly.

Homeostasis

Response to drugs may be accentuated or modified by changes in homeostatic mechanisms such as postural control (sedative drugs), orthostatic circulatory responses (antihypertensives, diuretics, ACE inhibitors, nitrates, phenothiazines, tricyclic antidepressants, levodopa), thermoregulation (phenothiazines, many other centrally sedating drugs, alcohol), visceral muscle function (bowel, bladder, eye; drugs with anticholinergic activity, diuretics), higher cognitive function (drugs affecting cholinergic transmission, many other (almost every) drugs (digoxin, cimetidine, NSAIDS.....)).

Adverse drug reactions

Often present with nonspecific symptoms such as confusion, incontinence, falls.

10% acute geriatric medicine admissions solely or in part due to drug side-effects.

Drugs commonly associated with ADRs - diuretics, digoxin, antihypertensives, analgesics, (non-steroidal anti-inflammatory drugs), anti-Parkinsonian drugs, psychotropics.

Most ADRs are dose-dependent, predictable exaggerations of the normal drug response.

Considerable morbidity or even mortality may ensue, and the underlying drug reaction may be overlooked.

Conversely a superimposed insult (e.g. diarrhoea, acute chest infection) may induce unexpected toxicity from previously tolerated medication.

Compliance

More than 90% of medicines consumed in form of tablets or capsules. Pills "sticking" as swallowed is a common complaint and pill-oesophageal transit time may be slowed even in those with normal oesophageal motility. There is then a risk of pill disintegration causing ulceration (esp iron tablets, slow-release potassium). Medication should be swallowed while upright, sitting or standing with 50-100 ml liquid and at least 15 minutes before lying down at bed time.

Compliance - poor in about 30%

- Poor motivation - especially if asymptomatic.
- Lack of understanding - complex regime, forgetful, running out of tablets.
- Practical problems - taste, size, 'sticking', vision, dexterity (bottle tops, inhalers).
- "Intelligent" non-compliance.

THE PHARMACIST

Over-the-counter (OTC) medicines taken by about 20% of elderly people. More potentially toxic compounds are now available over-the-counter, especially non-steroidal anti-inflammatory agents and H₂ blockers. Patients may not mention these compounds unless specifically asked. Child-proof containers are often also elder-proof (poor manual dexterity, arthritis of hands, poor vision).

Typed labels are now mandatory; "as before/as directed/as required".

The pharmacist (hospital and community) has a vital role in contributing to patient education and in maintaining a 'check' for incorrect dosages, interactions, etc.

The role of the doctor:

Safe and effective prescribing

- Maximum benefit with minimum hazard.
- Make a full diagnosis and aim at key problems in order of priority - not everything needs a pill, remember lifestyle changes (diet, alcohol, exercise, aids and appliances, social support)
- Consider the effects of organ failure on kinetics, dynamics and any likely drug interactions.
- Start with low doses and adjust cautiously, especially if drug has narrow therapeutic index (digoxin, phenytoin, theophylline) and/or reduced clearance.
- Prescribe only drugs with which you are familiar (know how metabolised and excreted), and which are of proven efficacy.
- Monitor and record response (or lack of it).
- Monitor compliance - short, simple list (twice/day better than 4/day... and perhaps once/day)
- Review drugs regularly, stop those no longer indicated, care with repeat prescriptions.
- Careful drug history from patients, relatives, GP, home help; look for hoarding of tablets; don't forget patients may share drugs; remember OTC preparations.

Drug	Complications
Diuretics	Postural hypotension, hypokalaemia, urinary incontinence
NSAIDS	Peptic ulceration renal failure
Digoxin	Cardiac arrhythmias vomiting delirium
Gentamicin	renal failure deafness
Warfarin	Bleeding
Benzodiazepines	Postural hypotension delirium, reversible dementia, addiction
Neuroleptics	Postural hypotension, delirium, parkinsonism, hypothermia,
ACE Inhibitors	Postural hypotension renal failure
Tricyclic antidepressants	Delirium postural hypotension, constipation

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