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Knowledge-Based HomeCare eServices for an Ageing Europe

FP 6 Specific Support Action
Thematic Priority 2: “Information Society Technology”

Deliverable D06
Formal Intervention Plans III

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1 Introduction

Formal Intervention Plans (FIP) are formal structures representing the health care procedures to assist patients suffering from particular ailments or diseases. They contain indications to all the actors involved in the care process (i.e. health care professionals, patients and relatives, etc.) in order to provide the best coordinated action plan.

FIPs in K4CARE provide a response to how to assist a patient with a particular disease or syndrome; therefore, it is a formal representation of the health care know-how that complements the K4CARE know-what knowledge described by the Agent Profile Ontology (APO) and by the Case Profile Ontology (CPO) in [3].

In the K4CARE project, the construction of FIPs is based on one of the following procedures:

a) Adaptation of Clinical Practice Guidelines published by health care institutions of well-known in the field of medical knowledge dissemination.

b) Creation of new FIPs for those diseases and syndromes whose treatment has not been captured in a Clinical Practice Guideline, or the existing Guidelines are not appropriate within the K4CARE health care context. In this case, FIPs are the result of the integration of the knowledge and experiences of the health care experts participating in the project and colleagues.

c) Application of inductive machine learning techniques to elicit behavioral patterns from the data stored in the K4CARE Electronic Health Care Record [4][5]. This is a technological approach that requires a final validation of the FIPs obtained by the health care partners in the project. This sort of creating FIPs is not considered in this document.

1.1 The K4CARE Project in a Nutshell

The K4CARE project (IST-2004-026968) is a European Commission 6FP project aiming at the development, the integration and the use of several Information and Communication Technologies (ICT) and intelligent Computer Science (CS) technologies in the framework of Home Care (HC).

K4CARE is based on:

- A model for HC service which can be shared by the EU countries;
- An Electronic Home Care Record;
- A telematic and knowledge-based CS platform;
- An Actor Profile Ontology for representing the profiles of the subjects involved in the K4CARE model;
- A Case Profile Ontology for representing symptoms, diseases, syndromes;
- Computer-Based Formal Intervention Plans.

The K4CARE project (www.k4care.net) is developed by thirteen EU partners: eight centers with geriatric, medical and healthcare competencies and five ICT and CS centers.
1.2 The Document

This document is the second one of a series of documents completing the set of FIPs managed within the K4CARE Project: Anemia, Arthritis, Chronic Ischemic Heart Disease, Chronic Obstructive Pulmonary Disease, Cognitive Impairment, Delirium, Dementia, Depression, Diabetes, Heart Failure, Hypertension, Immobility, Mobility Impairment, Parkinson’s Disease and Parkinsonism, Post-stroke, and Pressure Ulcer.

The distribution of these diseases and syndromes across the series of three publications (this one included) is:

- Deliverable D06.1 [23]: FIPs on Post-stroke and Diabetes.
- Deliverable D06.2 [24]: Cognitive Impairment (Dementia), and Mobility Impairment (Pressure Ulcers).
- Deliverable D06.3 (this one): Anemia, Arthritis, Coronary Heart Disease, Chronic Obstructive Pulmonary Disease, Delirium, Depression, Heart Failure, Hypertension, Immobility, Parkinson’s Disease and Parkinsonism.

All these documents will share the same structure in three parts: first, the description of the disease or syndrome considered and the knowledge sources (i.e., Clinical Practice Guidelines), then the process of transformation of these extensive guidelines to only highlight the aspects that are relevant to the K4CARE needs, and finally the formal representation of the FIPs in the SDA notation [1].

Intermediate flowcharts are used before the construction of the final FIPs as a bridge between the medical knowledge available in the guidelines, and the way this knowledge is organized in a single structure. The main difference between the flowcharts and the FIPs provided is that flowcharts are produced by physicians pursuing the correctness of the health-care procedures, while FIPs obtained from flowcharts are produced by CS technicians that pursue the logical correctness of the procedures described making them executable.

1.3 Clinical Practice Guidelines and Health Care Knowledge Sources

A Clinical Practice Guideline (GL) is defined [13] as systematically developed statements to assist practitioners and patients in arriving at decisions on appropriate health care for specific clinical circumstances. Guidelines are intended to describe the diagnosis and management of a particular condition, and should provide a clear indication of the best choices for the clinical management of the patient. They can also include recommendations on the organization of services.

Normative treatment guidelines are built to provide the mechanism to link patient outcomes to the care provided and improve quality without increasing costs. However, few GLs have been developed for the homecare setting. Existing GLs for chronic diseases (e.g. for congestive heart failure, diabetes, chronic obstructive pulmonary disease, falls, osteoarthritis, depression, and medication management) should be modified to be applicable in home care. Special issues in generating and modifying GLs in home care patients are represented by co-morbidity (several GLs have to be applied simultaneously) and reliability of GLs related to elderly patients. These are the main con-
cepts leading to the selection and the adaptation of existing GLs that have been realized for K4CARE.

Clinical guidelines used to start defining FIPs in K4CARE were selected based on the review of the scientific literature and guidelines already published by international healthcare organizations such as the National Library of Medicine and the National Guideline Clearinghouse in the USA or National Institute for Health and Clinical Excellence (NICE) in UK. The chosen guidelines are those that – according to the experience of the K4CARE medical partners – summarize and evaluate the best evidence – according to the target of the project. One of the criteria in the choice of GLs was the presence of clear and reliable diagrams (flow-charts) that can be more easily formalized into the SDA notation.

1.4 **Highlighting the parts in GLs and Flowcharts that are relevant to K4CARE**

GLs are targeted at health professionals and patients providing information for decision-making. They are aimed at reducing variations in medical practice in order to guarantee an optimum level of quality and improve health care. GLs are based on systematic reviews of biomedical literature and they recommend different clinical intervention strategies depending on the quality of scientific evidence on which they are based. However, it is generally recognized that GLs are only one of the elements that influence medical practice. This is particularly true when dealing with particular subgroups of patients, home care patients in our case.

Thus, it is not easy to recommend an intervention strategy for a specific circumstance (reality) completely based on what has been studied in some rather ideal conditions (i.e. a randomized controlled trial). At the same time, while discussion is going on about how to implement GLs more reliably in “real” environments, the need remains for “individualized yet systematic clinical decision making” predicated on *individual patient priorities* [12]. The K4CARE choice was that of first selecting, then merging sound GLs in terms of possibility of real use in real chronic home patients.

Clinical guidelines are generally composed of several parts. In K4Care attention has been focused on the parts that relate to the management and treatment options. As a matter of fact, diagnosis is not the basic issue in managing the Home-Care Patient (HCP), while long term treatment of different and concomitant conditions is the main task. Inside each GL some parts have been selected that include advice on clinical management, prescribing aids, and practical procedures. Recommendations are selected among published evidence, and the 'missing link' between this evidence and practical clinical care provided, by taking current expert consensus into account to realize the “merge” among different parts from different GLs.

A major issue is that of deriving reliable schemas from the GLs to be transformed in FIPs, through the translation in the SDA notation. Usually GLs contain flow-charts. These structures have to be modified both to enclose information deriving from different sources and to avoid loops and dead-end paths. The interaction between medical partners and ICT engineers produced the versions of FIPs in SDA which reflect the whole process of selection, merging, integration, and formalization. The work regarding the post-stroke patient is a major example of the entire process, while the proposal of diabetes management is more related to the choice of selecting a reliable and
measurable parameter capable of monitoring the complex management of a multi-apparatus disease as diabetes and to give advice for the treatment.

1.5 FIPs in the SDA Notation

There are several formal languages to represent FIPs: Asbru [9], GLIF3 [7], PROforma [10], PRODIGY [8], EON [11], SAGE [12], SDA [1], etc. Among them, it is the SDA Model the one closer to the requirements of the K4CARE project and also the one meeting some desirable properties that the other ones do not satisfy or they satisfy to an insufficient extent. These properties are representation capability and simplicity [2].

The SDA Representation Model [1] is a formal language to describe actionable procedural knowledge in health care. This model is based on a set of terms that are the vocabulary of the health care domain in which the model is being applied (e.g. Cognitive Impairment). Each single term can be either:

- A state term if it can be used to describe the condition of a patient in that domain,
- A decision term if it is useful to state a patient trait that causes a differential treatment, or
- An action term if it represents some medical, surgical, clinical or management action on the patient.

Action terms have related a set of petitioners and a set of performers in order to permit the description of collaborative medical treatments in which several professionals may interact. Any petitioner in the set of petitioners is allowed to request the action to be executed. Performers in the set of performers are the persons allowed to execute the action. Both petitioners and performers are restriction sets, therefore if no petitioner or performer is provided for an action term, this action can be petitioned or performed by any agent involved in the treatment of the patient.

In the SDA representation model, state terms are grouped to form states, decision terms are grouped to form decision branches, and action terms are grouped to form actions.

1.5.1 State

States represent patient conditions, situations, or statuses that deserve a particular course of action which is totally or partially different from the actions followed when the patient is in other state.

1.5.2 Decision

Decisions gather alternative branches which allow the introduction of variability in a treatment.

1.5.3 Action

Actions constitute the proper health care activity in the treatment the SDA represents.
1.5.4 Combination of States, Decisions and Actions

States, Decisions, and Actions are combined to describe clinical algorithms in such a way that any state, decision branch, or action can be connected to any state, decision, or action in the algorithm.

From an operational point of view, the states are the *entry points* of patients to the clinical algorithm, and the connectors are the paths that the treatment of patients can follow, conditioned to the decision branches these patients meet.

1.5.5 Non-Determinism and Parallelism

In a FIP, the connection of states, decisions, and actions constitute the courses of actions in the treatment of a disease or syndrome. Decisions are used to derive one treatment in one direction or another, with regard to a particular known condition. This defines a *deterministic decision*. In health care, however, not all the possible decisions are deterministic. For example, a patient in a particular condition can follow alternative treatments and there is not an accepted criterion to decide among them. These are called *non-deterministic decisions*.

The SDA language has not been designed to represent the parallel actions on a patient as parallel threads but to consider the treatment of a patient as a unique thing that evolves in time. So, if a patient deserves a parallel treatment conducted by a specialist (or several treatments conducted by several specialists), this is not represented as the launch of several parallel treatments, but as the incorporation of these treatments in the current treatment of the patient. Therefore, *parallelism in SDA* is represented by all the action terms that are in any action and the times related to them.

1.5.6 Time constraints

Time can be part of the SDA FIPs as time constraints. In the SDA model there are two sorts of time constraints: term-related constraints and connector-related constraints.

*Term-related time constraints* are used to indicate the time a term is valid and the frequency within this validity time interval. For example, if the time constraint \([X, Y, Z]\) is given to a state term representing fever, this means that the state describes patients that have had fever between \(X\) and \(Y\) (e.g. \(X=1w\) for one week, and \(Y=1d\) for one day ago), and the frequency of measuring fever has been \(Z\) (e.g. \(Z=6h\) for every six hours). If this time constraint is attached to an action term as for example antidepressant, it means that the antidepressant must be taken between time \(X\) and \(Y\) with a frequency \(Z\).

On the other hand, *connector-related time constraints* are related to SDA connectors and they represent delays in the course of action of a treatment, as for example, wait between one week and one month before continuing the treatment. These constraints have the general form \([X, Y]\), with \(X\) the minimum delay and \(Y\) the maximum delay.
1.5.7 SDA graphical representation

The SDA model has two alternative representations: graphical and textual. The graphical representation is based in the figures depicted in Table 1. The textual representation is based in an XML Schema described in [6].

Table 1. Elements and connections of the SDA Model.

2 Anemia

Anemia is a condition where there is a lower than normal number of red blood cells in the blood, usually measured by a decrease in the amount of hemoglobin. Hemoglobin is the oxygen-carrying part of red blood cells.

The cause of this condition may vary with the type of anemia. Potential causes include blood loss, poor diet, many diseases, medication reactions, and various problems with the bone marrow, where blood cells are made. Iron deficiency anemia is most common in women who have heavy menstrual periods. Risk factors include heavy periods, pregnancy, older age, and diseases that cause anemia.

Anemia is common in the elderly and its prevalence increases with age. Using World Health Organization criteria for anemia (hemoglobin of less than 12 g per dL [120 g per L] in women and less than 13 g per dL [130 g per L] in men), the prevalence of anemia in the elderly has been found to range from 8 to 44 percent, with the highest prevalence in men 85 years and older.

Even though the high prevalence of anemia in the elderly makes it a condition that clinicians might expect to find frequently, several features of anemia make it easy to overlook. The onset of symptoms and signs is usually insidious, and many elderly patients adjust their activities as their bodies make physiologic adaptations for the condition. Typical symptoms of anemia, such as fatigue, weakness and dyspnea, are not specific and in elderly patients tend to be attributed to advancing age. Pallor can be a helpful diagnostic clue, but pallor can be hard to detect in the elderly. Frequently, patients have signs of a disorder that is made worse by the anemia, such as worsening congestive heart failure, cognitive impairment, dizziness and apathy. Unless clinicians consider anemia as a possibility in the elderly, it can be easily overlooked.
Anemia in the elderly is evaluated in a manner similar to that in younger adults, including an assessment for signs of gastrointestinal blood loss, hemolysis, nutritional deficiencies, malignancy, chronic infection (such as subacute endocarditis), renal or hepatic disease, and other chronic disease. In patients without evidence of an underlying disease, the initial laboratory evaluation should include a complete blood count, red blood cell indices, a reticulocyte count and peripheral blood smear.

Anemia algorithms used for evaluation of younger adults are based on the mean corpuscular volume. Such algorithms may be less helpful in the elderly because the classic changes in erythrocyte size do not often accompany anemia in this age group. In most elderly patients with anemia, red cell indices disclose normocytic, normochromic anemia. Clinicians therefore might begin the evaluation of anemia as they would in younger adults, but, if they do not find one of the classic causes of microcytosis or macrocytosis, the search for a cause might need to be enlarged.

The principal causes of anemia in the elderly are: Anemia of chronic disease (30% to 45%), Iron deficiency (15% to 30%), Posthemorrhagic (5% to 10%), Vitamin B$_{12}$ and folate deficiency (5% to 10%), chronic leukemia or lymphoma (5%), Myelodysplastic syndrome (5%), no identifiable cause (15% to 25%).

Anemia of chronic disease, also called anemia of chronic disorders, is the most common form of anemia in the elderly. Numerous diseases are associated with anemia of chronic disease, but in many cases an underlying disease is not identified.

Iron deficiency anemia, the second most common cause of anemia in the elderly, usually results from chronic gastrointestinal blood loss caused by nonsteroidal anti-inflammatory drug-induced gastritis, ulcer, colon cancer, diverticula or angiodysplasia. Chronic blood loss from genitourinary tract cancer, chronic hemoptysis and bleeding disorders may result in iron deficiency but are much less common causes. Older persons may become iron deficient because of inadequate intake or inadequate absorption of iron. Without blood loss, anemia takes several years to develop.

While studies suggest that vitamin B$_{12}$ (cobalamin) deficiency is the cause of anemia in 5 to 10 percent of elderly patients, the actual prevalence of vitamin B$_{12}$ deficiency is likely to be much higher in the elderly. Vitamin B$_{12}$ deficiency is difficult to detect in the elderly. First, the symptoms and signs of vitamin B$_{12}$ deficiency are not reliably present in the elderly. Only about 60 percent of patients with vitamin B$_{12}$ deficiency are anemic. In addition, neurologic symptoms of B$_{12}$ deficiency can develop before the patient becomes anemic. Second, although anemia due to vitamin B$_{12}$ deficiency is usually macrocytic and megaloblastic, it can be normocytic or even microcytic. Third, serum B$_{12}$ levels do not reliably reflect tissue B$_{12}$ deficiency. Up to 30 percent of patients with low-normal serum vitamin B$_{12}$ levels have anemia and neurologic disease. This observation has prompted a search for more reliable ways of detecting vitamin B$_{12}$ deficiency.

Unlike vitamin B$_{12}$ deficiency, folate deficiency usually develops as a result of inadequate dietary intake. The body stores very little folate, only enough to last four to six months. Like vitamin B$_{12}$ deficiency, folate deficiency classically causes macrocytic anemia, although a significant proportion (25 percent) of elderly patients with folate deficiency have normocytic anemia. The symptoms of folate deficiency are nearly indistinguishable from those of vitamin B$_{12}$ deficiency.
Myelodysplastic syndrome is a relatively uncommon cause of anemia, but is a more common cause in the elderly than in younger patients. The syndrome, thought in the past to represent pre-leukemia, is characterized by a defect in the development of one of the marrow cell lines, limiting the release of functioning cells. Anemia results when the red cell lines are affected. Myelodysplastic syndrome should be a diagnostic consideration when white cell or platelet abnormalities accompany the anemia. The diagnosis of this syndrome is usually made by bone marrow biopsy.

2.1 General Treatment of Anemia

There is no specific therapy for anemia of chronic disease except to manage or treat the underlying disorder. Iron therapy is of no benefit. Erythropoietin may be helpful in some patients with anemia of chronic disease. The dosage is 50 to 100 U per kg three times a week. The dosage can be increased to 150 U per kg per dose if the response to a lower dose is inadequate.

In addition to treatment of the cause of bleeding, iron supplementation should be initiated for the treatment of iron deficiency anemia. The usual recommended dose of elemental iron is 50 to 100 mg three times a day; however, a smaller amount of elemental iron, such as a single 325-mg tablet of iron sulfate, may minimize side effects and improve compliance. This dose, equivalent to approximately 97.5 mg of elemental iron, is usually sufficient to replace iron stores, albeit at a slower rate.

Vitamin B<sub>12</sub> deficiency is treated by vitamin B<sub>12</sub> supplementation, parenterally or orally. The intramuscular dose is 1,000 µg, often given daily for one week to build up stores, then weekly for one month and then monthly thereafter. Oral therapy with 1,000 to 2,000 µg of vitamin B<sub>12</sub> daily has been shown to be as effective as intramuscular injections and in some ways may be superior. A response to therapy, characterized by an increase in reticulocytosis, often occurs within a week of the initiation of vitamin B<sub>12</sub> therapy.

Folate deficiency is treated with oral folic acid, 1 mg daily.

Myelodysplasia is treated supportively with transfusions.

2.2 K4CARE Knowledge on how to deal with Anemia

The American Medical Directors Association proposed in 2007 the flowchart in Figure 1 [31]. In [29] and [30], this knowledge is extended and provides a particularization of the treatment of anemia in the elder. This knowledge is reflected in Figure 2.

2.3 Anemia Formal Intervention Plans

The formal intervention plans employed in the K4CARE project are the ones depicted in Figure 3 and Figure 4. Figure 3 describes the steps that have to be followed in the general treatment of anemia. These steps are Recognition, Assessment, Treatment, and Monitoring. The diagram also reflects the details of each one of these steps.

Figure 4 is a concrete description of the process of managing anemia in the elderly.
Figure 1. Recognition, Assessment, Treatment, and Monitoring of Anemia.
Figure 2. Management of anemia in the elderly
Figure 3. SDA on the recognition, assessment, treatment, and monitoring of Anemia.
Figure 4. SDA on the management of anemia in the elderly
3 Arthritis

Arthritis is the inflammation of one or more joints, which results in pain, swelling, stiffness, and limited movement. There are over 100 different types of arthritis.

Arthritis involves the breakdown of cartilage. Cartilage normally protects the joint, allowing for smooth movement. Cartilage also absorbs shock when pressure is placed on the joint, like when you walk. Without the usual amount of cartilage, the bones rub together, causing pain, swelling (inflammation), and stiffness.

3.1 General Treatment of Arthritis

Osteoarthritis is the most prevalent form of arthritis, with an associated risk of mobility impairment and related disability. It is the clinical and pathological outcome of a range of disorders that results in structural and functional failure of synovial joints. Traditionally, it has been considered a disease of articular cartilage. The current concept holds that osteoarthritis involves the entire joint organ, including the subchondral bone, menisci, ligaments, periarticular muscle, capsule, and synovium. Osteoarthritis is caused by aberrant local mechanical factors acting within the context of systemic susceptibility. Systemic factors that increase the vulnerability of the joint to osteoarthritis include increasing age, female, sex, and possibly nutritional deficiencies. The diagnosis of osteoarthritis can usually be made clinically and then confirmed by radiography. The main features that suggest the diagnosis include pain, stiffness, reduced movement, swelling, crepitus, and increased age (unusual before age 40) in the absence of systemic features (such as fever).

Typically osteoarthritis presents as joint pain. The joint pain of osteoarthritis is typically described as exacerbated by activity and relieved by rest. In more advanced stages, the disease is more painful at rest and at night. The source of pain is not particularly well understood and is best framed in a biopsychosocial framework. Of the local events in the joint, loss of cartilage probably does not contribute directly to pain as it is aneural. In contrast, the subchondral bone, periosteum, synovium, and joint capsule are all richly innervated and could be the source of nociceptive stimuli in osteoarthritis.

There is no cure, and current therapeutic strategies are primarily aimed at reducing pain and improving joint function. Osteoarthritis should be managed on an individual basis and treatment will probably consist of a combination of treatment options. Treatment should be modified according to the response obtained. The recommended hierarchy of management should consist of non pharmacological treatments as first choice, followed by drugs, and then, if necessary, surgery. The nonpharmacological approach includes: exercise and weight loss associated, if necessary, with additional treatment. Exercise increases aerobic capacity, muscle strength, and endurance and also facilitates weight loss. All people capable of exercise should be encouraged to take part in a low
impact aerobic exercise programme (walking, cycling, or swimming or other aquatic exercise). It is also important to encourage overweight patients with osteoarthritis to lose weight through a combination of diet and exercise. Additional treatments consist of local heat and cold, electrotherapy (TENS), acupuncture, glucosamine sulphate, appropriate footwear (shock absorbing shoes, insoles), assistive devices (walking sticks and tap turner). If non pharmacological approach fails, and patient still complains about his pain, it is possible to try with pharmacological treatments. As first choice, it is suggested to use Paracetamol which is the oral analgesic of choice for mild to moderate pain in osteoarthritis. It would be useful to associate topical treatment with NSAID and/or capsaicin. If there is no improvement it can be used oral NSAID or COX-2 inhibitor. Oral NSAID/COX-2 inhibitors should be used at the lowest effective dose for the shortest possible period of time. All oral NSAIDs/COX-2 inhibitors have analgesic effects of a similar magnitude but vary in their potential gastro-intestinal and cardio-renal toxicity and therefore the choice of agent and dose should take into account individual patient risk factors. Opioid analgesics are useful alternatives in patients in whom NSAIDs are contraindicated, ineffective, or poorly tolerated. If there is no improvement despite pharmacological treatments and the patient still presents with pain and signs of local inflammation, there is an indication for intra-articular corticosteroid injections. Intra-articular injections depend on the joint affected and if it is susceptible of this treatment, for example knee or shoulder. If the symptoms cannot be managed by all these treatments modalities the last choice is surgery. The typical indications for surgery are debilitating pain and major limitation of functions such as walking and daily activities that impact substantially on their quality of life.

3.2 K4CARE Knowledge on how to deal with Arthritis

Recommendations about osteoarthritis are mostly derived from guidelines of NICE [49]. The National Institute for Clinical Excellence (NICE) is a part of the NHS in UK. It produces guidance for both the NHS and patients on medicines, medical equipment and clinical procedures and where they should be used. We have reviewed these guidelines and other studies; recommendations are the same used in the clinical practice of K4CARE medical partners.

First, rehabilitative procedures are described - as recommended - next to weight loss and additional treatments. Then, pharmacological treatment is proposed; it consists in paracetamol or local NSAID (non-steroidal anti-inflammatory drug), followed if insufficient by NSAID or COX-2 inhibitors or analgesic opioid if NSAID are contraindicated. The final step proposes intra-articular corticosteroid injections or surgery.

The K4CARE knowledge employed in the treatment of arthritis is summarized in Figure 5. In the next section this knowledge is transformed into a K4CARE Formal Intervention Plan (Figure 6).
Patient with osteoarthritis
Pain, reduced function, stiffness

Exercise
Local muscle strengthening, general aerobic fitness

There is improvement?
Yes
End treatment

No
Consider weight loss

Intra-articular corticosteroid injections
There is improvement?
Yes
End treatment

No
+ one or more Additional treatments

Consider joint replacement surgery

If the treatment is insufficient -> substitute COX-2 inhibitor or NSAID + proton pump inhibitor

If NSAID are controindicated add or substitute OPIOID analgesic

Re-assess the patient after short time (1 - 2 week) in order to finish PT

There is improvement?
Yes
End treatment

No

Pharmaceutical treatment:
• Oral analgesy: Paracetamol
• Topic treatment: NSAID and/or capsaicin

Re-assess the patient after short time (1 - 2 week) in order to finish PT

There is improvement?
Yes
End treatment

No
+ one or more Additional treatments until 3 months

End treatment

Additional treatments:
- Local heat and cold
- Electrotherapy (TENS)
- Acupuncture
- Glucosamine sulphate (1500 mg daily)
- Advice on appropriate footwear (shock absorbing shoes, insoles)
- Assistive devices (walking sticks and tap turner)

Figure 5. Treatment of Osteoarthritis.
3.3 Arthritis Formal Intervention Plans

Figure 6. SDA on the treatment of Osteoarthritis.
4 Cognitive Impairment Syndrome

Cognitive Impairment Syndrome is a broad term to describe a wide variety of impaired brain function (relating to ability to think, concentrate, react to emotions, formulate ideas, problem solve, reason and remember, etc.), sufficient to impair social or occupational function. There is also a wide range of severity in impairment from mild through to severe.

4.1 General Treatment and prevention of Cognitive Impairment Syndrome

In the K4CARE project a cognitive impairment syndrome is defined by contemporary presence of cognitive impairment (MMSE score < 24 points) and functional impairment. Functional impairment is defined as an alteration in Barthel Index (BI) and/or in Instrumental Activity Daily Living (IADL). BI and IADL are useful in evaluating a patient’s state of independence before treatment, his progress as he undergoes treatment and his status when he reaches maximum benefit, but the total score is not as significant or meaningful as the breakdown into individual items, since these indicate where the deficiencies are.

It can result from Alzheimer’s disease, other form of dementia or depression or other diseases.

In scientific literature there are no guidelines for the cognitive impairment syndrome, but only for individual diseases that may be associated with cognitive impairment. From medical experience and various existing guidelines the following indications are born.

4.2 K4CARE Knowledge on how to deal with Cognitive Impairment Syndrome

Because the likelihood of chronic general medical illnesses and the likelihood of dementia both increase with age, the two commonly coexist. Memory impairment and aphasia, both of which interfere with the patient’s ability to provide a reliable description of symptoms, complicate the assessment and treatment of general medical conditions. Resistance to physical examination can also complicate assessment, so laboratory testing and radiological procedures may become particularly important. The involvement of family members and other caregivers in providing history is essential. Many medical conditions are known to have a significant impact on cognitive functioning. The identification and treatment of medical and psychiatric disorders that can adversely affect cognition are especially important. For example, appropriate management of diabetes mellitus may have beneficial effects on cognition.

Dementia predisposes to the development of delirium, especially in the presence of general medical and other neurological illnesses. Delirium in persons with dementia negatively affects cognitive and functional ability, quality of life, and life span, as well as increases the need for institutionalization and rehospitalization and increases mortality. A comprehensive approach to delirium includes prevention by avoidance of unnecessary medications and use of the lowest effective dosage, early recognition of delirium through vigilant monitoring at regular intervals, and—when delirium does develop—a thorough search for other causes and prompt treatment to decrease the associated morbidity.
Major depression is an important element of the differential diagnosis of memory difficulties. Particularly in elderly persons, major depressive disorder may be associated with reports of memory impairment, difficulty concentrating, and a reduction in intellectual abilities described by history or observed on mental status examination. Depression and progressive dementia may sometimes be distinguished on the basis of an assessment of the course and onset of depressive and cognitive symptoms and by response of cognitive symptoms to treatment of the depression. However, even when the onset of depressive symptoms precedes or coincides with the onset of cognitive symptoms and both resolve with antidepressant treatment, more than 50% of patients go on to develop dementia or mild cognitive impairment within several years of the depressive episode. In addition, among patients with mild cognitive impairment, evidence suggests that those who are also depressed have a greater likelihood of developing Alzheimer’s disease. Dementia must be distinguished from malingering and factitious disorder, which generally manifests patterns of cognitive deficits that are inconsistent over time and are uncharacteristic of those typically seen in dementia.

Dementia must also be distinguished from milder symptoms. Subjective memory complaints are common as people get older. Many individuals with these complaints have subtle, nonprogressive declines in memory, but some have more significant impairment that is more likely to represent the prodromal phase of Alzheimer’s disease or another dementia. The category of mild cognitive impairment was developed to describe individuals in this prodromal phase.

The goal of the treatment of cognitive impairment syndrome is to reduce the severity of the symptom and any associated disability. This is summarized in Figure 7.

The K4CARE FIP obtained from Figure 7 is depicted in Figure 8.
Clinical assessment: MMSE <24 AND functional impairment (alteration in Barthel index and/or in IADL)

Yes

Cognitive Impairment syndrome

Are medical conditions present? yes

Treat medical condition and reassess after stabilization

no

Is delirium present? yes

Treat delirium (see DELIRIUM FIP) and reassess after three days

no

Is depression present? yes

Treat depression (SEE DEPRESSION FIP) and reassess after 3 months

no

Is dementia present? yes

Treat dementia (SEE figure 1 in DEMENTIA FIP) and reassess after 1 month

no

Is cognitive impairment confirmed? yes

Are behavioural disturbances present?

no

Reassess after three months

Treat behavioural disturbances (SEE figure 6 in Dementia FIP) and reassess

yes

Are alterations in social assessment present?

no

Reassess after one month

yes

Treat modifiable social problems and reassess after three months

Figure 7. Cognitive Impairment Syndrome.
Figure 8. SDA for Cognitive Impairment Syndrome.
5 Coronary Heart Disease

Coronary heart disease is the end result of the accumulation of atheromatous plaques within the walls of the arteries that supply the myocardium (the muscle of the heart) with oxygen and nutrients. Most individuals with coronary heart disease show no evidence of disease for decades as the disease progresses before the first onset of symptoms, often a "sudden" heart attack, finally arise. After decades of progression, some of these atheromatous plaques may rupture and (along with the activation of the blood clotting system) start limiting blood flow to the heart muscle. The disease is the most common cause of sudden death, and is also the most common reason for death of men and women over 20 years of age. Coronary heart disease is a chronic illness that requires continuing medical care and patient self-management education to prevent acute complications.

5.1 General Treatment and prevention of Coronary Heart Disease

Therapeutic options for chronic coronary heart disease today are based on four principles:

1. Life style changes;
2. Medical treatment - drugs;
3. Coronary interventions as angioplasty and stent-implantation;

Acute deteriorations in patients with coronary heart disease should be managed in a hospital setting unless the patient has already been commenced on a palliative care pathway.

Lifestyle modifications remain the single most effective way to combat coronary heart disease. Lifestyle changes specifically shown to help in reducing the risk or improving the symptoms of heart disease include:

• Quit smoking
• Maintain a normal blood pressure
• Keep cholesterol levels within normal range
• Engage in regular physical activity
• Maintain as near normal body weight as possible
• When diabetes is present, keep blood glucose levels within normal range
• Reduce the amount of stress in life
• Prevention of an infection

Pharmacological treatment: drug management of patients with coronary heart disease involves individualised regimens addressing the particular needs of the patient. Medical treatment is often necessary to maintain a normal blood pressure and normal cholesterol levels.

Guidelines have been produced with the aim to provide a series of evidence-based recommendations related to coronary heart disease and its secondary prevention (for patients already after myocardial infarction). The guidelines which fit better with K4CARE aim are:

• AACVPR/ACC/AHA 2007 performance measures on cardiac rehabilitation for referral to and delivery of cardiac rehabilitation/secondary prevention services. Circulation. 2007

These guidelines were taken as the basis for determination of the FIP for secondary prevention of coronary heart disease in the K4CARE project.

5.2 K4CARE Knowledge on how to deal with Coronary Heart Disease

People with coronary artery disease should similar to diabetic patients receive medical care from a physician-coordinated team. It is necessary in this team approach that patients with coronary heart disease play an active role in the management of their care. Many strategies and techniques should be used to provide adequate education of the patient. In developing the plan, consideration should be given to the patient’s age, school or work schedule and conditions, physical activity, eating patterns, social situation and personality, cultural factors, and presence of complications of diabetes or other medical conditions.

For individuals with coronary heart disease life-style changes are of the biggest importance and may have the best outcomes. However lifestyle interventions are very limited because of the nature of states requiring home care. That is for example cognitive impairment and immobility, which make lifestyle intervention such as physical activity quite difficult. At the same time the usually poor nutritional status of the home care patients limits the dietetic changes. Despite these problems there are still many ways, how the patient’s behavior may be influenced.

• Quit smoking. Cigarette smoking is considered the biggest risk factor for sudden cardiac death. Smokers' risk of heart attack is more than twice that of a nonsmoker. Smokers' who do suffer a heart attack are more likely to die than are nonsmokers who have a heart attack. By quitting smoking, an individual can immediately reduce their risk for heart disease. Patients should be forced to stop smoking regularly.

• Control blood pressure. High blood pressure (hypertension) increases the workload of the heart. Over time, the heart muscle begins to enlarge. This increases the risk of heart attack, congestive heart failure, stroke and kidney failure. Individuals should have their blood pressure checked regularly every day in the morning and if necessary also more often during the day. The goal for blood pressure is 140/90 or less (e.g. 130/80 for diabetic patients). Medication is usually needed when lifestyle changes are ineffective in keeping the blood pressure within normal range.
  • It is recommended to begin drug therapy with ACE inhibitors (especially beneficial for diabetic patients) and/or beta blockers. If ineffective add diuretics, angiotensin-receptor blocker, calcium channel blocker. The choice of medications in combination depends on individual circumstances and must be managed by specialist.
- **Control blood cholesterol.** As levels of cholesterol rise in the blood, the risk for coronary heart disease increases. By adopting a diet that is lower in saturated fat and cholesterol, it is often possible to reduce cholesterol levels. When diet alone proves ineffective in reducing cholesterol level, medications may be needed. Blood cholesterol (low density lipoprotein) LDL levels should be no higher than 160 mg/dl if no more than one risk factor is present or less than 130 mg/dl if two or more risk factors are present. LDL cholesterol should be less than 100 mg/dl in people with a history of heart attack, stroke or diabetes. HDL (high-density lipids) greater than 40 mg/dl and triglycerides less than 150mg/dl are recommended. Cholesterol level should be checked annually. If the patient takes hypolipidemic medication, lipid levels should be checked every 3 months.

- **Increase physical activity.** Lifestyle intervention such as physical activity is in HCP quite difficult. However, minimal increase in physical activity is beneficial and can help to reduce blood cholesterol levels and blood pressure, as well as decrease risk for developing diabetes and obesity.

- **Maintain and achieve a desirable body weight.** Persons with excess body fat are more likely to develop coronary heart disease. Obesity not only puts undue strain on the heart muscle but it can adversely influence blood pressure, blood cholesterol levels and increase risk for developing diabetes. Even modest weight loss (5 to 10 %) can reduce risk of heart disease and diabetes. A body mass index (BMI) of 21 to 25 is recommended. The body weight should be checked regularly (every day) especially in patients with concomitant heart failure.

- **Maintain normal glucose levels.** Diabetes increases risk of developing heart disease, even when blood sugars are kept under control. It is very important to monitor and control as many risk factors for coronary heart disease as possible. Blood glucose should be checked in respect to patient’s type of diabetes and actual treatment. Refer to diabetes mellitus FIP.

- **Attempt to control the amount of stress in life.** Is has been noted some evidence between coronary heart disease and stress. Stress might increase risk for heart disease and it is generally considered wise to try to avoid stressful events, especially if they seem to contribute to other unhealthy behaviors like over-eating, smoking or increased alcohol consumption.

- **Influenza vaccination.** Patients with cardiovascular disease should have an influenza vaccination. Patients should be vaccinated annually.

In the HC setting, visits and clinical reviews on a regular basis as well as regular medication intake by HCP are necessary for successful therapy and prevention of symptoms worsening. Visits and actions should be scheduled to meet the needs of each patient. Clinical reviews should assess functional capacity, fluid status, cardiac rhythm and also monitor blood biochemistry.

Both, [38] and [39], have been considered to elaborate the knowledge that is being used in the K4CARE project to assist coronary heart disease. This knowledge is summarized by Figure 9 (split over two pages).
Acute deteriorations in patients with coronary heart disease have to be managed in a hospital setting unless the patient has already been commenced on a palliative care pathway.

Prevention of coronary heart disease

START

Lifestyle factors optimised?

NO

YES

Weight loss program if overweight or obesity present
Smoking cessation program – refer to smoking cessation clinic
Exercise Program and increase physical activity
Cardiac Rehabilitation program
Alcohol intake advice
Influenza vaccination (annually)

Patients with arterial hypertension: BP must be checked daily
Goal: BP less than 140/90 mm Hg or less than 130/80 mm Hg if patient has diabetes or chronic kidney disease?

NO

YES

Start with ACE inhibitors (especially in diabetic patients and chronic kidney disease) and/or beta blockers and titrate dose upwards.

Add diuretics, angiotensin-receptor blocker, calcium channel blocker and titrate dose upwards

Optimal blood pressure control?

NO

YES

Regular appointments by specialist (cardiologist) every 3-6 months.

Optimal blood pressure control?

NO

YES

Seek specialist input

Patients with diabetes mellitus: Goal: HBA1C <7%?

NO

YES

See FIP for diabetes mellitus

Lipid management:
Goal: LDL-C <100 mg/DL, if triglycerides are >200 mg/DL, non-HDL-C should be <130 mg/DL;
(if not known dislipidemia, lipid levels should be checked annually)
Goal: HBA1C <7%

NO

YES

Start dietary therapy and increase physical activity

Normal lipid levels after 3 months?

NO

YES

Normal lipid levels?

NO

Seek specialist input

Start therapy with hypolipidemic drugs and titrate dose upwards. Use statins, fibrates or niacin depending on type of dislipidemia. Should be started by specialist.

Normal lipid levels?

NO

Seek specialist input

Figure 9. Coronary Artery Disease (part I).
Does the patient take antiplatelets or anticoagulants?

- Add aspirin 100 mg per day for all patients and continue indefinitely in all patients unless contraindicated.

- Start and continue clopidogrel 75 mg/d in combination with aspirin for up to 12 months in patients after acute coronary syndrome or percutaneous coronary intervention with stent placement.

Does the patient already take renin-angiotensin-aldosteron blockers?

- ACE inhibitors: Start and continue indefinitely in all patients with left ventricular ejection fraction \( \leq 40\% \) and in those with hypertension, diabetes, or chronic kidney disease, unless contraindicated. Consider for all other patients.
  If patient intolerant to ACE inhibitors use angiotensin receptor blockers.

- Aldosteron blockade: Use in postmyocardial infarction patients, without significant renal dysfunction or hyperkalemia, who are already receiving therapeutic doses of an ACE inhibitor and-blocker, have a left ventricular ejection fraction \( \leq 40\% \), and have either diabetes or heart failure.

Does the patient already take beta blockers?

- Beta blockers: Start and continue indefinitely in all patients who have had myocardial infarction, acute coronary syndrome, or left ventricular dysfunction with or without heart failure symptoms, unless contraindicated.

If the patient is clinically stabilized, regular appointments by specialist (cardiologist) every 3-6 months with other examinations (ECG, Echocardiography, carotid ultrasound, lab tests) are necessary to improve the therapy.

Figure 9. Coronary Artery Disease (part II).
5.3 Coronary Heart Disease Formal Intervention Plans

The treatment of coronary artery disease described in Figure 9 (parts I & II) is converted to the formal intervention plan in SDA notation of Figure 10.

Figure 10. SDA on the treatment of Coronary Heart Disease.
6 Chronic Obstructive Pulmonary Disease

Chronic Obstructive Pulmonary Disease (COPD) is a common clinical problem. It is also known by various other names, such as Chronic Obstructive Lung Disease (COLD), Chronic Obstructive Airway Disease (COAD), Chronic Airflow Obstruction (CAO), Chronic Airway (or Airflow) Limitation (CAL), or simply as Chronic Bronchitis and Emphysema. COPD, which includes chronic bronchitis and emphysema, is a progressive disease characterized by airflow limitation/obstruction that is either not reversible at all or only partially reversible. It is generally difficult to separate out the two conditions (chronic bronchitis and emphysema), hence these are grouped together as COPD. COPD does not include asthma in which the airflow obstruction is largely reversible. The airflow obstruction in COPD is associated with abnormal inflammatory response of the lungs to chronic inhalational exposure from smokes, dusts and other air pollutants.

COPD manifests as chronic cough with or without sputum production. To define COPD, the presence of these symptoms for more than three months of a year for at least two consecutive years is considered essential. It may or may not be accompanied with progressive breathlessness. The disease progresses with time ultimately leading to respiratory disability and death.

Acute exacerbations of COPD occur whenever there is an episode of infection or some other complication. There is worsening of symptoms, deterioration of clinical condition and impairment of lung function during the period of exacerbation.

After an acute exacerbation, most patients experience a transitory or permanent decrease in quality of life, and nearly 50% of patients discharged from hospitals after acute exacerbations are readmitted more than once in the following 6 months. Therefore, one of the main treatment goals for patients with COPD is reducing the number and severity of annual exacerbations.

6.1 General Treatment of COPD

The treatments explained in this section are based on [43], [44], [45], [46], [47], [48], and [49].

The important components of managing patients with stable COPD include (a) minimization of risk factors, (b) pharmacotherapy appropriate to the disease severity and (c) supportive nonpharmacological measures (such as patient education and rehabilitation).

6.1.1 Therapeutic interventions

None of the existing medication for COPD has been shown to modify the long-term decline in lung function. Therefore, pharmacotherapy for COPD (see Figure 11) is used only to decrease symptoms and complications. Patient education is necessary to improve skills, ability to cope with illness and the health status. Health education is particularly effective for sustained smoking cessation. In addition, appropriate information about the nature of the disease, instructions on how to use different medications and inhalers, and clues to recognize symptoms of exacerbation are mandatory.

Bronchodilators: Bronchodilator medication is central to the symptomatic management of COPD. Inhaled drugs are preferred to oral preparations. However, the choice of drugs depends on
the availability of medications and patient’s affordability. Short acting bronchodilators can be used ‘as-needed’ to relieve intermittent or worsening symptoms, and on regular basis to prevent or reduce persistent symptoms. Stepwise treatment should be recommended. In general, nebulized therapy for stable patients is not appropriate unless it has been shown to be better than conventional dose therapy. Regular treatment with short-acting bronchodilators is cheaper but less convenient than treatment with long-acting bronchodilators. The long acting inhaled beta agonist salmeterol has been shown to improve health status significantly in doses of 50µg twice daily. Similar data for short acting beta agonists are not available. Use of inhaled tiotropium (an anticholinergic) once daily also improves symptoms and health status. Combining drugs with different mechanisms and durations of action may increase the degree of bronchodilatation for equivalent or lesser side effects. A combination of a short-acting beta agonist and the anticholinergic drug ipratropium in stable COPD produces greater and more sustained improvements in FEV1 than either alone and does not produce evidence of tachyphylaxis. The addition of oral theophylline should normally be considered only if inhaled treatments have failed to provide adequate relief. Sustained release preparations are better. All studies that have shown efficacy of theophylline in COPD were done with slow-release preparations. Addition of theophylline to β2-agonists or anticholinergics may produce additional improvements in lung function and health status. However, combination of salbutamol with theophylline in a single tablet is not recommended.

**Corticosteroids:** Inhaled corticosteroids do not change the rate of decline in lung function, but can increase postbronchodilator FEV1, reduce the number of exacerbations, and slow the rate of decline in health status. Regular treatment with inhaled glucocorticosteroids should be prescribed for symptomatic patients with COPD with a documented spirometric response to glucocorticosteroids or for those with FEV1<50% predicted and repeated exacerbations requiring treatment with antibiotics or oral glucocorticoids. Long-term treatment is required in such patients; in fact, withdrawal of inhaled corticosteroids can lead to increase in symptoms and exacerbation rate. Chronic treatment with systemic glucocorticosteroids should be avoided because of unfavorable benefit-to-risk ratio.

**Antibiotics:** Antibiotic treatment is beneficial in selected patients with acute exacerbation of COPD. In particular, the patients with more severe exacerbations (type 1) are more likely to experience benefit than those whose exacerbations are less severe. Typical administration periods ranged from 3 to 14 days, and tetracycline, amoxicillin, and trimethoprim–sulfamethoxazole were the most common antibiotics. Although most of randomized, placebo-controlled trials were done before the emergence of multidrugresistant organisms, they show only a minimal benefit with antibiotic treatment in the more severe exacerbations. On the basis of these data and the emergence over time of more resistant organisms, particularly Streptococcus pneumoniae, it has become common practice to use more broad-spectrum antibiotics in acute exacerbations of COPD.

**Oxygen Therapy:** Ample evidence shows that oxygen therapy provides important benefits to inpatients with acute exacerbations of COPD and hypoxemia. The major concern with the administration of this therapy is the risk for resultant hypercarbia and respiratory failure.

**Mucolytic Agents and Mucus Clearance Strategies:** Mucolytic agents shorten disease course or improve patient outcomes. Mechanical percussion of the chest as applied by a physical
or respiratory therapist is ineffective (that is, does not increase FEV1) in patients with acute exacer-
berations of COPD.

**Noninvasive Positive-Pressure Ventilation**: Noninvasive positive-pressure ventilation is fre-
quently used in the inpatient management of patients with acute exacerbations of COPD. It not only improves ventilation and decreases PCO2 levels but, in many instances, is also a means of avoiding intubation.

### 6.1.2 Management of acute exacerbations

Exacerbation of COPD is defined as “a sustained worsening of the patient's condition, from the stable state and beyond normal day-to-day variations, that is acute in onset and necessitates a change in regular medication”.

The symptoms of an exacerbation are increased breathlessness often accompanied by wheezing, increased cough and sputum, change of the color or tenacity of sputum, and fever.

The common causes of an exacerbation are infection of the tracheobronchial tree and air pol-
lution. The cause of approximately one-third of severe exacerbations cannot be identified. Condi-
tions that may mimic an acute exacerbation include pneumonia, congestive heart failure, pneuo-
mothorax, pleural effusion, pulmonary embolism, and arrhythmias. These conditions should be ruled out by clinical examination and investigations.

The assessment of severity of acute worsening is based on the patient's medical history before
the exacerbation, symptoms, physical examination, lung function tests, arterial blood gas meas-
urements, and other laboratory tests. The medical history should cover the period of worsening since the new symptoms have been present, the frequency and severity of breathlessness and coughing attacks, sputum volume and color, limitation of daily activities, any previous epi-
sodes/exacerbations, hospitalization, and the present treatment regimen.

There is no widely accepted definition of acute exacerbation of COPD, but most published definitions encompass some combination of three clinical findings: worsening dyspnea, increase in sputum purulence, and increase in sputum volume. A severity scale for acute exacerbations devel-
oped by Anthonisen and colleagues is based on these findings as well as others. Type 1 exacerbations (severe) have all three clinical findings, and type 2 exacerbations (moderate) exhibit two. Type 3 exacerbations (mild) have one of these clinical findings plus at least one of the following: an upper respiratory tract infection in the past 5 days, fever without other apparent cause, increased wheezing, increased cough, or a 20% increase in respiratory rate or heart rate above baseline.

Acute exacerbations can be triggered by tracheobronchial infections or environmental expo-
sures, and patients often have associated clinical conditions, such as heart failure, extrapulmonary infections, and pulmonary embolism. Therefore, acute exacerbation is mainly a clinical diagnosis.

Current practices for the diagnosis and management of acute exacerbations of COPD are varied. Some commonly used tests and therapies are not supported by evidence, while others are.
6.1.3 Treatment of acute exacerbations

Bronchodilators are the cornerstone of managing exacerbations of COPD. Patients need to increase the dose and/or frequency of existing bronchodilator therapy. New drugs, which patient is not taking at the time of worsening, may be added. Short-acting bronchodilators should ideally be administered using inhalers (preferably with spacers). In a severe case, nebulizers may be used for drug administration. In situations where these drugs are not available, parenteral aminophylline can be used with due attention to its toxicity. Aminophylline dose should be appropriately modified in elderly patients, those in congestive cardiac failure or having liver cirrhosis, and those already taking oral methylxanthines, cimetidine, ciprofloxacin or erythromycin.

Antibiotics should be used when symptoms of breathlessness and cough are increased and sputum is purulent and increased in volume. The choice of antibiotic depends on the affordability of the patient, the severity of exacerbation and the bacterial spectrum. Amoxycillin, doxycycline, co-trimoxazole, fluoroquinolones or a second generation macrolide/cephalosporin are used as the first choice. For severe exacerbations higher-grade antibiotics, such as coamoxiclav or a fourth generation cephalosporin can be used.

Systemic glucocorticoids should be used in acute exacerbations. They shorten recovery time and help to restore lung function more quickly. A dose of 40 mg oral prednisolone per day (or equivalent) for 5-10 days is recommended. Carefully look for tuberculosis by sputum examination and chest radiograph before starting corticosteroids.

Controlled oxygen therapy can be administered at low flow rates (preferably with a Venturi mask) with monitoring for features of CO2 retention. Chest physiotherapy, inhaled corticosteroids and mucolytic agents are generally not useful in the management of acute exacerbations.

Patients with the following features should be hospitalized for further management:
- Marked increase in intensity of symptoms, such as sudden development of resting dyspnea
- Onset of new physical signs (e.g. cyanosis, drowsiness, confusion, flaps, peripheral edema)
- Failure of exacerbation to respond to initial medical management
- Significant co morbidities such as diabetes or associated cardiac disease
- Newly occurring arrhythmias
- Diagnostic uncertainty

6.2 K4CARE Knowledge on how to deal with COPD

All the above indications on the treatment of COPD pharmacologically, and the treatment of acute exacerbations is reflected in the diagrams of Figure 11 and Figure 12.

Figure 12 contains the concepts PEFR (Peak expiratory flow chart), URI (upper respiratory infection), CXR (Chest X-ray), O2 (Oxygen therapy), NPPV (Noninvasive positive pressure ventilation), and PRN (as needed). Moreover it makes references to the footnotes:

(1) Use anticholinergic bronchodilators first, once at maximum dose, then add B2 agonist bronchodilators
(2) Dosing regimen used in the SCOPE trial: 3 days intravenous methylprednisolone, 125 mg every 6 hours followed by oral prednisone, taper to complete the 2 week course (60 mg/day on days 4-7, 40 mg/day on days 8-11 and 20 mg/day on days 12-15)

(3) NPPV should be administered under the supervision of a trained physician

(4) Use narrow-spectrum antibiotics; the agent favored in the trials were amoxicillin and trimethoprim-sulfamethoxazole and tetracycline

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**Figure 11. COPD Pharmacotherapy.**
COPD
(Increase in symptom from baseline)

Physician examines patient for three diagnostic criteria for acute exacerbation of COPD:
1. Increase in dyspnea
2. Increase in sputum volume
3. Increase in sputum purulence

Criteria present?

None of 3 diagnostic criteria present
Consider other diagnosis

One or more criteria present?

One diagnostic criterion with at least one of the following?
1. URI in the past 5 days
2. Fever without apparent cause
3. Increased wheezing
4. Increased cough
5. 20% increase in heart rate or respiratory rate over baseline

Three criteria

1. Chest X-Ray
2. Inhaled bronchodilators(1)
3. Systemic corticosteroids(2)
4. Antibiotics (4)
5. O₂ PRN
6. NPPV PRN(3)

Two only

1. Chest X-Ray
2. Inhaled bronchodilators (1)

1. Chest X-Ray
2. Inhaled bronchodilators (1)

Figure 12. Treatment of COPD acute exacerbations.

6.3 COPD Formal Intervention Plans

The treatments described in the previous section are formalized in the FIPs of Figure 13 and Figure 14.
Figure 13. SDA on COPD Pharmacological Treatment.
Delirium is a complex neuropsychiatric syndrome with an acute onset and fluctuating course; it is common in all medical settings. It occurs with higher frequency in elderly people and in those with pre-existing cognitive impairment. The definition of delirium is “An acute reversible alteration in cognitive function and/or behaviour that occurs as a direct toxic effect of a substance or secondary to some underlying medical condition”.

The symptoms of delirium are wide ranging, and although they are non-specific, their fluctuating nature is highly characteristic and is a valuable diagnostic indicator. The core disturbance involves an acute generalised impairment of cognitive function that affects orientation, attention, memory, and planning and organisational skills. Other disturbances, such as those of the sleep-wake cycle, thought processes, affect, perception, and activity levels, are underemphasised in diagnostic systems but contribute substantially to problems in identifying and managing delirium.
Depending on which symptoms are apparent, delirium may be mistaken for a variety of disorders including dementia, mood disorders, and functional psychoses.

The causes of delirium are many. In a typical case, predisposing and precipitating factors interact with multiple aggravating or perpetuating factors, which influence the course. The multifactorial nature is often underemphasised, but studies that have accounted for the possibility of multiple causes have found that between two and six factors may be present in any single case. It is therefore vital to be aware of risk factors and, having identified an explanation for delirium, remain vigilant as to the possibility of additional factors. Attempting to identify and treat a single cause is overly simplistic: each case needs detailed, repeated assessment for multiple potential factors.

Age, pre-existing cognitive impairment, severe comorbidity, and exposure to medication are robust predictors of the risk of delirium.

Although many risks for delirium reflect the enduring characteristics of the patient, some factors can be modified to prevent onset. Medications are implicated in 20-40% of cases: most prescribed drugs can cause delirium but benzodiazepines, narcotics, and drugs with anticholinergic activity have a particular propensity. Many drugs and their metabolites may unexpectedly contribute to causing delirium because their anticholinergic effects are unrecognised. It is therefore prudent to minimise exposure to drugs and to reduce doses or stop administration of high risk compounds especially during high risk periods, such as the perioperative period. Many risk factors may simply be markers of general morbidity, and studies showing the preventive impact of modification of these risk factors are lacking but important. None the less, preliminary evidence indicates that interventions that reduce sensory deficits, immobility, sleep disturbance, dehydration, and cognitive impairment can reduce the number of episodes of delirium and their duration.

7.1 General Management and Treatment of Delirium

The most important step in delirium management is early recognition. If delirium is not diagnosed, it is doubtful that any efforts will be made to reverse it. Once delirium is detected, efforts should focus on identifying the etiology. Often this can be done by assessing for the presence of known risk factors. Both prevention and treatment should focus on the minimization and/or elimination of predisposing and precipitating factors. The theoretical goals of management are to improve the patient's cognitive status and reduce the risk of adverse outcomes such as aspiration, prolonged immobility, increased length of acute care, institutionalization, and death. The major role of biomedical investigations is to exclude or diagnose non-toxic causes of delirium. Investigations should be selected according to clinical judgment and may include:

- Arterial blood gases
- Blood sugar level
- Chest X-ray
- CT of the head
- ECG
- Electroencephalogram
- Pulse oximetry
• Renal and hepatic function tests
• Serum electrolytes
• Thyroid function tests

Treatment is essentially supportive and should be continued until cognitive function and behavior returns to normal. General supportive measures are aimed at alleviating distressing symptoms, preventing self-harm and maintaining adequate fluid and electrolyte fluid balance. These measures may include:

• Intravenous fluids (if the patient is not taking oral fluids)
• Provision of a calm environment (no bright lights, no noise, away from windows)
• Reassurance
• Protection of the patient from self-inflicted injury
• Relief of urinary retention (anticholinergic agents)
• Pharmacological sedation

The first step in pharmacologic treatment of delirium is to assess the patient’s current medications for any offending agents that may be causing or exacerbating the delirium. Inappropriate use of sedatives or analgesics may exacerbate delirium symptoms. Delirious patients may become more confused when treated with sedatives, causing a paradoxical increase in agitation as the sedative effects wear off. In fact, benzodiazepines and narcotics that are often used to treat “confusion” (delirium) actually worsen cognition and exacerbate the problem. A thorough review of a patient’s medications will help identify any sedatives, analgesics and/or anticholinergic drugs that may be removed or decreased in dose. There are currently no drugs with FDA-approval for the treatment of delirium. The American Psychiatric Association [37] and the Society of Critical Care Medicine clinical practice guidelines [35] recommend haloperidol for the treatment of delirium, though it is acknowledged that this is based on sparse outcomes data from nonrandomized case series and anecdotal reports (i.e., level C data). Haloperidol is a dopamine receptor antagonist that works by inhibiting dopamine neurotransmission, with resultant improvement in the positive symptomatology (hallucinations, agitated and combative behavior, etc) and often results in a sedative effect. Haloperidol and similar agents (e.g., droperidol) have not been extensively studied, though they are widely used anecdotally. In addition to haloperidol, other candidate antipsychotics/neuroleptic agents (e.g., risperidol, ziprasidone, quetiapine, and olanzapine) may prove to be helpful for both hyperactive and hypoactive delirium, especially with their broader receptor affinities. Patients receiving all of these antipsychotics should be monitored for adverse side effects such as QT prolongation, arrhythmias and extrapyramidal side effects. Prospective randomized controlled trials are needed to evaluate the effectiveness and safety of these agents relative to one another.

The duration of delirium varies with the intoxication but rarely lasts longer than 72 hours. The patient should be carefully and continuously observed until mental status normalizes. Mental status and respiratory function must be carefully monitored after sedation.
7.2 K4CARE Knowledge on how to deal with Delirium

K4CARE takes [32], [33], [34], [35], [36], and [37] as sources of knowledge on delirium. This knowledge is synthesized in Figure 15 where the presence of diseases deserve specific treatment, and the treatment of delirium alone is started with pharmacological consideration to continue with a non-pharmacological treatment if the previous one does not achieve the expectancies. The treatment of pain is also considered in the flowchart. Any sort of treatment proposed is followed with an observation phase of 72h.

Figure 15. Delirium.
Numbers in some of the blocks in Figure 15 have the following meaning:

1. Consider stopping or substituting for deliriogenic medications such as benzodiazepines, anticholinergic medications (metoclopramide, H2 blockers, promethazine, diphenhydramine), steroids, etc.
2. Non-pharmacological protocol:
   - **Orientation**
     - Provide visual and hearing aids
     - Encourage communication and reorient patient repetitively
     - Have familiar objects from patient’s home in the room
     - Attempt consistency in nursing staff
     - Allow television during day with daily news
     - Non-verbal music
   - **Environment**
     - Sleep hygiene: Lights off at night, on during day. Sleep aids (zolpidem, mirtazapine)?
     - Control excess noise (staff, equipment, visitors) at night
     - Ambulate or mobilize patient early and often
   - **Clinical parameters**
     - Maintain systolic blood pressure > 90 mm Hg
     - Maintain oxygen saturations >90%
     - Treat underlying metabolic derangements and infections
3. Analgesia – Adequate pain control may decrease delirium. Consider intermittent narcotics if feasible. Asses with objective tool.
4. Typical or atypical antipsychotics - While tapering or discontinuing sedatives, consider haloperidol 2 to 5 mg IV initially (0.5-2 mg in elderly) and then q 6 hours. Guideline for max haloperidol dose is 20 mg/day due to ~60% D2-receptor saturation. May also consider using any of the atypicals (e.g. olanzapine, quetiapine, risperidone, ziprasidone, or abilifide). Discontinue if high fever, QTc prolongation, or drug-induced rigidity.

### 7.3 Delirium Formal Intervention Plans

The flowchart in Figure 15 is transformed into the K4CARE SDA formal intervention plan depicted in Figure 16.

### 8 Depression

Depression is a common mental disorder that presents with depressed mood, loss of interest or pleasure, feelings of guilt or low self-worth, disturbed sleep or appetite, low energy, and poor concentration. These problems can become chronic or recurrent and lead to substantial impairments in an individual's ability to take care of his or her everyday responsibilities. At its worst, depression can lead to suicide, a tragic fatality associated with the loss of about 850,000 lives every year. Depression is the leading cause of disability and the 4th leading contributor to the global burden of disease in 2000. Depression occurs in persons of all genders, ages, and backgrounds.

The older person may describe the usual signs and symptoms of depressive disorders, being: low mood, reduced energy, anhedonia, loss of interest, sleep and appetite disturbance, guilty ideas, psychomotor reduction or increase, impaired concentration and reduced clarity of thought.

The Differential Diagnosis in an elderly person will include:
• Dementia
• Delirium
• The signs and symptoms of a medical illness itself
• Substance dependence and abuse
(Although these conditions may also be co-existing with a depression.)
However, particularly relevant to the elderly are:
• Cognitive impairment generally of recent onset, which may be severe
• Somatic symptoms over a range of systems

Figure 16. SDA on the treatment of Delirium.
The assessment of depressive disorder in the person who already has a dementia is difficult, especially in the person who has impaired expressive language function. Relevant issues to consider include the occurrence of recent changes such as:

- A recent change in behaviour
- A recent change in psychomotor function
- A recent deterioration of biological function – sleep, appetite
- Development of depressed affect.

It may be difficult to distinguish the natural progression of a dementia but sudden downturns in the above, unexplained by the development of new intercurrent physical discomfort or illness, should signal that depression be considered.

The newly depressed older person warrants investigation for presence of common underlying physical conditions and investigations relevant to the expression of somatic symptoms at the time of presentation. However, there should not be a delay in commencing treatment for a depressive illness, which is severe – investigations can continue in parallel. Depression is not a diagnosis of exclusion. Its recognition as a cause of morbidity in the older person will lead to more effective management. Too often physical symptoms are pursued to exhaustive ends while clear psychological symptoms are ignored. In all cases of major depression of new onset or altered presentation compared to previous episodes, notwithstanding investigations being conducted for a particular physical illness, the following investigations may be considered after physical examination:

- Full blood count.
- Electrolytes, creatinine, urea.
- Random blood sugar.
- Thyroid function.
- Urine microscopy.
- Cerebral scan.

Other tests may include B12/folate, chest x-ray, liver function, calcium.

Medication is likely to be needed where there is any sustained depressive disorder and when nonpharmacological strategies are not achieving their goals. Useful signs to indicate commencing medication are:

- Presence of biological signs, disturbed sleep,
- Appetite and energy changes.
- Diurnal variation in mood worse in the morning.
- Agitation or retardation.
- Depression with any psychotic features.
8.1 General Treatment of Depression

This section describes the general treatment of depression according to [40], [41], and [42]. Beneficial effects may take 2 to 3 weeks to begin and continue from then. All antidepressant medications have been shown to be effective. Personal familiarity with a few, across different groups, is of assistance in making a choice for patients with different clusters of symptoms and sensitivity to side effects.

An adequate trial of any one medication is a minimum 4-6 weeks at the maximum tolerated dose. Once remission has occurred, the same dose should be maintained.

Medication may not be forever, but 12 months is a reasonable period of treatment after full recovery from a first episode. Continuation beyond this should be discussed with a psychiatrist, and will depend on:

- The severity of the index episode
- The frequency of past episodes
- Adequacy of remission.

8.1.1 Anti-depressant medicines

First line: A patient who has been successfully treated with an anti-depressant in the past may have a trial of that same medication in a recurrence, provided there are not any new contraindications for that medication. On the basis of a more acceptable profile of side effects in the elderly, the usual first choices for antidepressants would come from the following:

- SSRI – citalopram, fluoxetine, fluvoxamine, paroxetine, sertraline
- SNRI – venlafaxine, duloxetine
- MAO-I – moclobemide
- Nefazadone
- Mianserin
- Mirtazepine

Second line: The medications used as a second line are all effective antidepressants, which have the potential to give more side-effects to the elderly, such as:

- Anticholinergic effects, dry mouth, confusion, constipation.
- Postural hypotension
- Toxicity in overdoseage
- affect cardiac rhythm in those predisposed

The second line medications are:

- Tricyclic antidepressants – agents without significant active metabolites are preferred for the elderly, being nortryptiline, dothiepin
- MAO-inhibitors – phenelzine.
Other drugs

Antipsychotic medications are often used in the early stages of a depression where psychotic features are troublesome – a few weeks might be the maximum time. They are usually a temporary addition to the antidepressant and have a strong potential to cause extra-pyramidal movement disorders if continued longer term.

The use of other herbal preparations has some community use but at this stage the effectiveness of these preparations remains contentious given the small number of published studies and cohort selection. It is necessary to ask about their use by every patient because of the known interactions with conventional antidepressant medications.

8.2 K4CARE Knowledge on how to deal with Depression

Figure 17, Figure 18, Figure 19, and Figure 20 summarize the management of depression in K4CARE. As far as Figure 17 is concerned, two footnotes are identified:

1. First line antidepressant SSRI – citalopram, fluoxetine, fluvoxamine, paroxetine, sertraline; SNRI – venlafaxine, duloxetine; MAO-I – moclobemide; nefazadone; mianserin; mirtazepine

2. The second line medications are the Tricyclic antidepressants – agents without significant active metabolites are preferred for the elderly, being nortryptiline, dothiepin and MAO-inhibitors – phenelzine.

The knowledge in Figure 17, Figure 18, Figure 19, and Figure 20 is converted to the K4CARE formal intervention plans that are shown in Figure 21, Figure 22, Figure 23, and Figure 24, respectively.
Adults with acute major depression or dysthmia

Choose an first line antidepressant (1) based on:
- Side effect profile
- Past response
- Severity of depression

Adequate Antidepressant Trial is 6 weeks at maximum or near-maximum recommended dose

Response

Inadequate response

Review diagnosis
Choose another antidepressant from a different drug class

Not tolerated

Maintain effective dose for 12 months after first episode

Response

Not tolerated

Further adequate Antidepressant Trial

Inadequate response/ not response

- Are there underlying medical conditions?
- Are there untreated psychosocial stressors?
- Is the Carer depressed?

Treat medical condition, psychosocial stressors or carer depressed

Yes

Re-evaluate depression diagnosis:
Is depression present?

No

Follow-up

Yes

Consider a drug of second line (2) or an antipsychotic

No

Figure 17. Pharmacological treatment of depression.
Figure 18. Management of depression with cognitive impairment at initial presentation.
Figure 19. Management of depression in a patient with dementia.
Figure 20. Suicide: risk assessment and management.
8.3 Depression Formal Intervention Plans

The SDA diagrams obtained from the flowcharts in the previous section are depicted in Figure 21, Figure 22, Figure 23, and Figure 24.

Figure 21. SDA on Depression Pharmacological Treatment.
Figure 22. SDA on Depression with CI Treatment.
Figure 23. SDA on Depression with dementia treatment.
Heart failure, also known as congestive heart failure (CHF), means the heart cannot pump enough blood to meet the body's needs. Over time, conditions such as coronary artery disease or high blood pressure gradually leave the heart too weak or stiff to fill and pump efficiently.

Heart failure represents a growing health problem. The past decade has seen significant advances in the treatment of heart failure, despite this it is still a major cause of hospital admissions – particularly in the elderly. Estimated prevalence of symptomatic heart failure ranges from 0.4% to 2% in the general European population and increases rapidly with age, suggesting that 10-20% of the elderly (over 70 years old) have heart failure. Mean age of the heart failure population is about 74 years. The increasing prevalence of heart failure might be partly explained by growing proportion of elderly population in Europe. Studies have confirmed the poor long-term prognosis of the disease. Annual mortality for those with heart failure ranges from 10% to over
50% depending on the severity. The accuracy of diagnosis is necessary to optimize the treatment of heart failure.

Heart failure is a complex syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the heart to function as a pump to support a physiological circulation. The syndrome of heart failure is characterised by symptoms such as breathlessness and fatigue, and signs such as fluid retention as well as signs associated with the underlying disorder. The most common cause of heart failure is left ventricular systolic dysfunction, in which the contraction is reduced. Coronary heart disease is the commonest cause of this but non-ischaemic cardiomyopathy secondary to hypertension, alcohol excess, thyroid disease, valvular disease or myocarditis can also be a cause.

9.1 General Treatment of HF

Much of the evidence base for the management of heart failure relates to heart failure due to left ventricular systolic dysfunction. Although this is the most common underlying cardiac abnormality in patients with heart failure, it should not be forgotten that other cardiac abnormalities may be the cause of the heart failure – for example valve disease, or ‘diastolic’ dysfunction of the left ventricle. In some patients several abnormalities may co-exist. Treatment must be multi-disciplinary and involves a holistic approach covering various aspects of the patients care:

1. Lifestyle modifications: weight reduction, exercise programs, stopping smoking, avoidance of alcohol, fluid and salt intake monitoring form a vital starting point for heart failure treatment.

2. Pharmacological treatment: drug management of patients with heart failure involves individualized regimens addressing the particular needs of the patient.

- Diuretics should be routinely used for the relief of congestive symptoms and fluid retention in patients with heart failure, and titrated according to need.
- ACE-inhibitors confer a prognostic benefit in heart failure and should be commenced prior to beta-blockade. Many large clinical trials have shown that several ACE-inhibitors increase life expectancy in patients with heart failure due to LV systolic dysfunction, compared with placebo. This effect has been seen in patients with all functional classes of heart failure (NYHA classes I – IV).
- Beta-blockers licensed for use in heart failure should be initiated in patients with heart failure due to LV systolic dysfunction after diuretic and ACE inhibitor therapy regardless of whether or not symptoms persist.
- Aldosterone antagonists such as spironolactone have been shown to improve mortality and should be introduced in those with more severe heart failure.
- There is limited evidence for the routine use of other agents including digoxin, amiodarone. Aspirin and anti-coagulation should be considered in the context of coronary vascular disease and atrial fibrillation.
Full guidelines assessing interventions and outcome benefits in Heart Failure management that fit within the K4CACE include:


2. **SIGN (Scottish Intercollegiate Guidelines Network)**: Management of Heart Failure: A National Clinical Guideline; 2007 Scottish Intercollegiate Network

3. **ESC (European Society of Cardiology) Guidelines**: Guidelines for the diagnosis and treatment of Chronic Heart Failure: update 2005; The Task Force for the diagnosis and treatment of CHF by the European Society of Cardiology

### 9.2 K4CARE Knowledge on how to deal with HF

The management of chronic heart failure is a complex process involving a multidisciplinary team. Acute deteriorations in these patients should be managed in a hospital setting unless the patient has already been commenced on a palliative care pathway. It is important to develop strong links between hospital and HC settings to ensure a smooth transition from a hospital to a HC facility. The management of established heart failure should be aimed at improving the quality of life for patients by improving symptoms or slowing their deterioration, reducing frequent re-admissions to hospital and improving the end of life experience for patients.

Optimizing medical treatment should be a priority for patients with chronic heart failure and this can be achieved in the HC setting by careful up-titration of treatment according to each patient’s needs (see Figure 25). Pharmacological therapy needs to be balanced by non-pharmacological interventions such as dietary advice and monitoring of salt intake, smoking cessation and avoidance of alcohol. Most patients will require some support and counseling to assist with understanding heart failure and coming to terms with the restrictions on their lifestyle.

In the HC setting, visits and clinical reviews on a regular basis as well as regular medication intake by HCP are necessary for successful therapy and prevention of symptoms worsening. Visits and actions should be scheduled to meet the needs of each patient (see Figure 26). Clinical reviews should assess functional capacity, fluid status, cardiac rhythm, and also monitor blood biochemistry. The guidelines in [25][26][27] have been taken as baseline of the procedures represented in Figure 25 and Figure 26.
New Diagnosis of Heart Failure

START

Lifestyle factors optimised?

Weight loss program
Smoking Cessation program
Exercise Program
Cardiac Rehabilitation program
Alcohol intake advice
Dietary Advice

Drug therapy optimised?

Add ACE-inhibitor and titrate dose upwards.

Intolerance to ACE-I or impaired renal function

Consider angiotensin-II receptor antagonist

Add Beta-blocker and titrate dose upwards

Patient symptomatic despite optimal drug therapy

Add Spironolactone

Patient remains symptomatic?

Seek Specialist input

END

Figure 25. Chronic Heart Failure. Initial FIP: Treatment optimization.
Chronic Heart Failure. Secondary FIP: Monitoring.

Patient’s pharmacological treatment optimised. **START**

HC team (physician and nurse) contact patient

**Help needed with Smoking or Alcohol?**

**YES**

Referral to Cessation clinics

**NO**

Counselling or Heart Failure advice needed?

**YES**

Referral to Counselling & Support services

**NO**

Patient stable and enters monitoring.

Weight gain of 1kg on two consecutive days

**YES**

Symptoms of breathlessness and/or ankle oedema

**YES**

Check blood chemistry
Check drug compliance
Modify fluid and salt intake
Increase Furosemide by 40mg daily
Discuss with Heart Failure Consultant

**NO**

Modify fluid and salt intake
Re-check within 48 hours

Oedema/weight returns to baseline value

**YES**

Consult with Cardiologist or hospital physician

**NO**

**Cut back diuretic to original dose**

**Oedema/weight returns to baseline value**

**YES**

Consider elective admission to hospital

**NO**

**Oedema/weight returns to baseline value**

**YES**

**Oedema/weight returns to baseline value**

**YES**

Consult with Cardiologist or hospital physician

**NO**

**Continue Observation of weight in HC facility**

Figure 26. Chronic Heart Failure. Secondary FIP: Monitoring.
9.3 HF Formal Intervention Plans

The clinical algorithms in Figure 25 and Figure 26 are translated into the FIPs depicted in Figure 27 and Figure 28, respectively.

Figure 27. SDA on the treatment of Chronic Heart Failure.
Figure 28. SDA on monitoring Chronic Heart Failure.
10 Hypertension

Hypertension is one of the most common worldwide diseases. Because of the associated morbidity and mortality and the cost to society, hypertension is an important public health challenge. Hypertension is the most important modifiable risk factor for coronary heart disease, stroke, congestive heart failure, end-stage renal disease, and peripheral vascular disease. Therefore, health care professionals must not only identify and treat patients with hypertension but also promote a healthy lifestyle and preventive strategies to decrease the prevalence of hypertension.

Defining abnormally high blood pressure is extremely difficult and arbitrary. Furthermore, the relationship between systemic arterial pressure and morbidity appears to be quantitative rather than qualitative. A level for high blood pressure must be agreed upon in clinical practice for screening patients with hypertension and for instituting diagnostic evaluation and initiating therapy. Because the risk to an individual patient may correlate with the severity of hypertension, a classification system is essential for making decisions about aggressiveness of treatment or therapeutic interventions.

Based on recommendations of the Seventh Report of the Joint National Committee of Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VII) [28], the classification of blood pressure (expressed in mm Hg) for adults aged 18 years or older is as follows*:

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic BP (mmHg)</th>
<th>Diastolic BP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>and</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>or</td>
</tr>
<tr>
<td>Hypertension stage I.</td>
<td>140-159</td>
<td>or</td>
</tr>
<tr>
<td>Hypertension stage II.</td>
<td>&gt;160</td>
<td>or</td>
</tr>
</tbody>
</table>

*Based on the average of 2 or more readings taken at each of 2 or more visits after initial screening

†Normal blood pressure with respect to cardiovascular risk is less than 120/80 mm Hg. However, unusually low readings should be evaluated for clinical significance.

Prehypertension, a new category designated in the JNC VII report, emphasizes that patients with prehypertension are at risk for progression to hypertension and that lifestyle modifications are important preventive strategies.

Evaluation

Diagnostic workup of hypertension
- Assess risk factors and comorbidities.
- Reveal identifiable causes of hypertension.
- Assess presence of target organ damage.
- Conduct history and physical examination.
- Obtain laboratory tests: urinalysis, blood glucose, hematocrit and lipid panel, serum potassium, creatinine, and calcium. Optional: urinary albumin/creatinine ratio.
- Obtain electrocardiogram.

Asses for major cardiovascular diseases risk factors
• Hypertension
• Obesity (body mass index >30 kg/m²)
• Dyslipidemia
• Diabetes mellitus
• Cigarette smoking
• Physical inactivity
• Microalbuminuria, estimated glomerular filtration rate <60 mL/min
• Age (>55 for men, >65 for women)
• Family history of premature CVD (men age <55, women age <65)

Assess for identifiable causes of hypertension

• Sleep apnea
• Drug induced/related
• Chronic kidney disease
• Primary aldosteronism
• Renovascular disease
• Cushing’s syndrome or steroidtherapy
• Pheochromocytoma
• Coarctation of aorta
• Thyroid/parathyroid disease

**Blood pressure measurement technique – Blood pressure diary**

In home care services the patients blood pressure has to be measured by the family doctor, by the physician in charge and by the nurse too in every visit.

In case of treated hypertension these controls should be regular and indicated in the patients blood pressure diary. Regular measurements should be repeated at least every third day. In case of moderately elevated blood pressure (140-180/<100mmHg) the controls should follow twice a day until it’s normalization. In case of highly elevated blood pressure (>180/>140mmHg) a doctor has to see the patient as soon as it is possible. In case of highly elevated blood pressure causing complaints such as thoracic or head pain immediate medical help is needed, it should be considered as medical emergency.

The blood pressure diary contains all the results of blood pressure measurements, its date, hour and minute. Patient self-check and the blood pressure diary provide information on response to therapy. They may help improve adherence to therapy and are useful for evaluating “white coat hypertension.” If the patient is not capable to measure his/her blood pressure for any reason (e.g.: dementia, paresis etc.), then family caregiver, social worker should do it instead of him/her and the results should be recorded in the diary.

The family doctor, the physician in charge and the nurse should control this diary at every visit. Family doctor and physician in charge can modify antihypertensive therapy according to the results indicated in the diary. If the nurse see reason then the family doctor or the physician in charge should be noticed that the patient’s blood pressure changed.

In home care the blood pressure diary is the base of antihypertensive therapy.

**10.1 General Treatment of Hypertension**

**Principles of Hypertension Treatment**

• Treat to BP <140/90 mmHg or BP <130/80 mmHg in patients with diabetes or chronic kidney disease.
• Majority of patients will require two medications to reach goal.
**Principles of lifestyle modifications**

- Encourage healthy lifestyles for all individuals.
- Prescribe lifestyle modifications for all patients with prehypertension and hypertension.
- Components of lifestyle modifications include weight reduction, DASH eating plan, dietary sodium reduction, aerobic physical activity, and moderation of alcohol consumption.

<table>
<thead>
<tr>
<th>Class of Medication</th>
<th>When to Use</th>
<th>When Not to Use</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diuretics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loop diuretics</td>
<td>Renal insufficiency (additional therapy)</td>
<td>Gout</td>
</tr>
<tr>
<td>Potassium-sparing</td>
<td>Primary hyperaldosteronism (additional therapy in combination with thiazide diuretics)</td>
<td>Renal insufficiency</td>
</tr>
<tr>
<td>Thiazides</td>
<td>Uncomplicated hypertension (preferred therapy), systolic hypertension in elderly people (preferred therapy), for older diabetic patients without nephropathy</td>
<td>Gout, dyslipidemia (high-dose)</td>
</tr>
<tr>
<td><strong>Beta-adrenergic antagonists</strong></td>
<td>Post–myocardial infarction, uncomplicated hypertension (preferred therapy), diabetes (without nephropathy)</td>
<td>Asthma, peripheral vascular disease (severe)</td>
</tr>
<tr>
<td><strong>ACE inhibitors</strong></td>
<td>Diabetes, post–myocardial infarction, heart failure, renal disease, uncomplicated hypertension (preferred therapy)</td>
<td>Bilateral renovascular disease, pregnancy</td>
</tr>
<tr>
<td><strong>Angiotensin II antagonists</strong></td>
<td>Diabetes (alternative therapy), heart failure (alternative therapy), uncomplicated hypertension (preferred therapy)</td>
<td>Bilateral renovascular disease, pregnancy</td>
</tr>
<tr>
<td><strong>Calcium channel blockers</strong></td>
<td>Nondihydropyridines</td>
<td>Uncomplicated hypertension (alternative therapy)</td>
</tr>
<tr>
<td></td>
<td>Dihydropyridines</td>
<td>Systolic hypertension (preferred therapy), uncomplicated therapy (alternative therapy)</td>
</tr>
<tr>
<td><strong>Alpha-adrenergic antagonists/central acting agents</strong></td>
<td>Uncomplicated hypertension (alternative therapy)</td>
<td>Autonomic dysfunction</td>
</tr>
</tbody>
</table>

*Table 2. Synopsis of Considerations in the Use of Antihypertensive Drug Classes*.

**Recommendations for management of hypertension**

The JNC recommends certain situations for which a specific class of drug may be administered. An ACE inhibitor should be the initial treatment in situations in which hypertension is associated with congestive heart failure, diabetes mellitus with proteinuria, and postmyocardial infarction with systolic left ventricular dysfunction. In patients who develop persistent cough while on ACE inhibitor therapy, an angiotensin II receptor antagonist may be substituted, but these agents’ efficacy in lowering cardiovascular mortality rates...
has not yet been proven. A beta-blocker should be prescribed following an acute myocardial infarction. A diuretic or a long-acting calcium channel blocker may be more effective in elderly patients with isolated systolic hypertension.

The 2004 Canadian Hypertension Society recommendations (similar to JNC VII guidelines) for the management of hypertension in specific patient groups are listed in Table 2 and Table 3.

Several situations demand the addition of a second drug because 2 drugs may be used at lower doses to avoid adverse effects, which may occur with higher doses of an individual agent. Diuretics generally potentiate the effects of other antihypertensive drugs by minimizing volume expansion. Specifically, the use of the diuretic thiazide in conjunction with a beta-blocker or an ACE inhibitor has an additive effect, controlling blood pressure in up to 85% of patients.

Most drug combinations using agents that act by different mechanisms have an additive effect. The combination of a calcium channel blocker with either an ACE inhibitor or a dihydropyridine calcium channel blocker and a beta-blocker has additive effects. An ACE inhibitor may be combined with an angiotensin II receptor antagonist because the blocking of angiotensin I receptors may lead to increased plasma angiotensin II concentration, which may compete with a drug for the receptor. Some combinations may not be additive, including a beta-blocker and ACE inhibitor, a beta-blocker and an alpha1-blocker and an alpha2 stimulant, and, more controversially, a diuretic and a calcium channel blocker. Some combinations may have additive adverse effects; these include a beta-blocker combined with verapamil or diltiazem, which leads to cardiac depression, bradycardia, or heart block.

Clinical trials have shown that the effective control of blood pressure reduces the risk of cardiovascular events in high-risk patients. In the patients who achieved optimal blood pressure control compared with those with uncontrolled hypertension, significant reductions in the incidence of cardiac events, stroke, and all-cause mortality occurred. Recent studies have consistently shown that newer antihypertensive agents, such as ACE inhibitors and calcium channel blockers, reduce cardiovascular events to a similar, or possibly greater, extent as older therapies, such as diuretics and beta-blockers. ACE inhibitors specifically offer additional benefits beyond blood pressure reduction, which include reduction of cardiovascular events and renal protection. Similarly, ARBs have demonstrated beneficial effects in heart failure, stroke, and renal protection.

Key messages of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VII) are as follows:

- In those older than 50 years, systolic blood pressure (BP) of greater than 140 mm Hg is a more important cardiovascular disease risk factor than diastolic BP.
- Beginning at 115/75 mm Hg, the cardiovascular disease risk doubles for each increment of 20/10 mm Hg.
- Individuals who are normotensive at 55 years will have a 90% lifetime risk of developing hypertension.
- Prehypertension (systolic 120-139, diastolic 80-89) requires health-promoting lifestyle modifications to prevent the progressive rise in blood pressure and cardiovascular disease.
- In uncomplicated hypertension, a thiazide diuretic, either alone or combined with drugs from other classes, should be used for the drug treatment of most.
<table>
<thead>
<tr>
<th>Risk Factor/Disease</th>
<th>Preferred Therapy</th>
<th>Alternative Therapy</th>
<th>Avoid Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncomplicated hypertension (&lt;60 y)</td>
<td>Low-dose thiazidelike diuretics, beta-blockers, ACE inhibitors, or long-acting dihydropyridine calcium channel blockers</td>
<td>Combinations of first-line drugs</td>
<td>...</td>
</tr>
<tr>
<td>Uncomplicated hypertension (&gt;60 y)</td>
<td>Low-dose thiazidelike diuretics, ACE inhibitors, or long-acting dihydropyridine calcium channel blockers</td>
<td>Combinations of first-line drugs</td>
<td>...</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>As for uncomplicated hypertension</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Diabetes mellitus with nephropathy</td>
<td>ACE inhibitors</td>
<td>Angiotensin II receptor blockers</td>
<td>High-dose diuretics and centrally acting agents (in the setting of autonomic neuropathy)</td>
</tr>
<tr>
<td>Diabetes mellitus without nephropathy</td>
<td>ACE inhibitors or beta-blockers</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Diabetes mellitus without nephropathy, with systolic hypertension</td>
<td>Low-dose thiazidelike diuretics or long-acting dihydropyridine calcium channel blockers</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Angina</td>
<td>Beta-blockers (ACE inhibitors as add-on therapy)</td>
<td>Long-acting calcium channel blockers</td>
<td>...</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>Beta-blockers, ACE inhibitors</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Systolic dysfunction</td>
<td>ACE inhibitors (thiazide or loop diuretics, beta-blockers, spironolactone is additive therapy)</td>
<td>Angiotensin II receptor blockers, hydralazine/isosorbide dinitrate, amlodipine</td>
<td>Nondihydropyridine calcium channel blockers (diltiazem, verapamil)</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>Most antihypertensives reduce LVH</td>
<td>...</td>
<td>Hydralazine, minoxidil</td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
<td>As for uncomplicated hypertension</td>
<td>As for uncomplicated hypertension</td>
<td>Beta-blockers (with severe disease)</td>
</tr>
<tr>
<td>Renal disease</td>
<td>ACE inhibitors (diuretics as additive therapy)</td>
<td>Dihydropyridine calcium channel blockers</td>
<td>ACE inhibitors in cases of bilateral renal artery stenosis</td>
</tr>
</tbody>
</table>

*Short-acting calcium channel blockers are not recommended in the treatment of hypertension

Table 3. Considerations in the Individualization of Antihypertensive Therapy*
• In specific high-risk conditions, there are compelling indications for the use of other antihypertensive drug classes (eg, ACE inhibitors, angiotensin-receptor blockers, beta-blockers, calcium channel blockers).

• Two or more antihypertensive medications will be required to achieve goal BP (<140/90 mm Hg or <130/80 mm Hg) for patients with diabetes and chronic kidney disease.

• For patients whose BP is more than 20 mm Hg above the systolic BP goal or more than 10 mm Hg above the diastolic BP goal, initiation of therapy using 2 agents, one of which usually will be a thiazide diuretic, should be considered.

• Regardless of therapy or care, hypertension will be controlled only if patients are motivated to stay on their treatment plan.

### Compelling indications for individual drug classes

- Heart failure: THIAZ, BB, ACEI, ARB, ALDO ANT
- Post myocardial infarction: BB, ACEI, ALDO ANT
- High CVD risk: THIAZ, BB, ACEI, CCB
- Diabetes: THIAZ, BB, ACEI, ARB, CCB
- Chronic kidney disease: ACEI, ARB
- Recurrent stroke prevention: THIAZ, ACEI

**Abbreviations:**

- THIAZ: thiazide diuretics
- BB: beta blockers
- ACEI: ACE inhibitors
- ARB: angiotensin receptor blockers
- ALDO ANT: aldosterone antagonists
- CCB: calcium channel blockers

### 10.2 K4CARE Knowledge on how to deal with Hypertension

The procedure followed for lifestyle modification is represented in Figure 29. The treatment of Stage I hypertension is represented with the flowchart of Figure 30, which is split up in seven consecutive pages. Finally, Stage II hypertension is treated with the procedure depicted in Figure 31, which is split up in seven consecutive pages.
Elevated blood pressure >140/90Hgmm or >130/80Hgmm and diabetes mellitus, but blood pressure does not exceed 160/100

Lifestyle optimised?

Lifestyle modification:
- DASH eating plan
- dietary sodium reduction
- aerobic physical activity,
- moderation of alcohol consumption
- weight loss program if overweight or obesity present
- smoking cessation program
- exercise program and increase aerobic physical activity

Goal blood pressure? <140/90 or 130/80DM
Blood pressure measurement twice a day for 3 days

YES

Optimised anti-hypertensive treatment: blood pressure in normal range

YES

Permanently elevated blood pressure, but under 160/100Hgmm Hypertension stage I.

NO

Permanently elevated blood pressure, over 160/100Hgmm Hypertension stage II.

Figure 29. Treatment of Hypertension: lifestyle modification.
Figure 30. Treatment of Hypertension: after lifestyle optimization. Stage I (140/90HGMM-160/100HGMM) (Part I).
Figure 30. Treatment of Hypertension: after lifestyle optimization. Stage I (140/90HGMM-160/100HGMM) (Part II).

1. Chronic kidney disease?
   - Yes: THIAZ, ACEI. Give only one of them.
   - No: Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB. Give only one of them.

2. No matter how many compelling indication there are, chose only one antihypertensive drug, and start only that in half of maximal dose. The strongest compelling indication should define the drug. Preferably thiazide-type diuretics are proposed.

3. Goal blood pressure?
   - <140/90 or 130/80DM
   - Blood pressure measurement twice a day for 3 days

4. Permanent elevated blood pressure, but under 160/100Hgmm Hypertension stage I. In spite of low dose monotherapy.

5. Permanent elevated blood pressure, over 160/100Hgmm Hypertension stage II.

Optimised anti-hypertensive treatment: blood pressure in normal range HT FIP STOP
Figure 30. Treatment of Hypertension: after lifestyle optimization. Stage I (140/90HGMM-160/100HGMM) (Part III).

Double the dose of the previously started antihypertensive monotherapy. (This will be the maximum dose of the previously started drug.)

Goal blood pressure? <140/90 or 130/80DM
Blood pressure measurement twice a day for 3 days

YES

Optimised anti-hypertensive treatment: blood pressure in normal range HT FIP STOP

NO

Permanently elevated blood pressure, but under 160/100Hgmm Hypertension stage I. In spite of maximum dose monotherapy.

ADD

Add a different type of antihypertensive drug than the previously started. Chose half of the maximal dose. Consider compelling indications!!!

NO

Permanently elevated blood pressure, over 160/100Hgmm Hypertension stage II.
Figure 30. Treatment of Hypertension: after lifestyle optimization. Stage I (140/90HGMM-160/100HGMM) (Part IV).
Figure 30. Treatment of Hypertension: after lifestyle optimization. Stage I (140/90HGMM-160/100HGMM) (Part V).
Fig. 30. Treatment of Hypertension: after lifestyle optimization. Stage I (140/90HGMM-160/100HGMM) (Part VI).

Goal blood pressure? <140/90 or 130/80DM
Blood pressure measurement twice a day for 3 days

YES: Optimised anti-hypertensive treatment: blood pressure in normal range HT FIP STOP

NO: Permanently elevated blood pressure, but under 160/100Hgmm
Hypertension stage I. In spite of low dose triple therapy.

Permanently elevated blood pressure, over 160/100Hgmm
Hypertension stage II.

Double the dose of the previously started third antihypertensive drug.
(This will be the maximum dose of the three antihypertensive drug.)
Goal blood pressure?  
<140/90 or 130/80DM  
Blood pressure measurement twice a day for 3 days

YES

Optimised anti-hypertensive treatment: blood pressure in normal range  
HT FIP STOP

NO

Permanently elevated blood pressure, but under 160/100Hgmm  
Hypertension stage I.  
In spite of maximum dose triple therapy.

SEE SPECIALIST !!!  
CONSIDER SECONDARY HYPERTENSION !!!  
STOP HT FIP

NO

Permanently elevated blood pressure, over 160/100Hgmm  
Hypertension stage II.

Figure 30. Treatment of Hypertension: after lifestyle optimization. Stage I (140/90HGMM-160/100HGMM) (Part VII).
Figure 31. Treatment of Hypertension: after lifestyle optimization. Stage II (OVER 160/100Hgmm) (Part I).
Figure 31. Treatment of Hypertension: after lifestyle optimization. Stage II (OVER 160/100HGMM) (Part II).

Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB.

No matter how many compelling indication there are, chose two antihypertensive drug, and start them in maximal dose. The two strongest compelling indication should define the drugs.

Goal blood pressure? <140/90 or 130/80DM Blood pressure measurement twice a day for 3 days

Optimised anti-hypertensive treatment: blood pressure in normal range HT FIP STOP

Permanently elevated blood pressure, over 160/100Hgmm Hypertension stage II. In spite of maximum dose double therapy.

Permanently elevated blood pressure, but under 160/100Hgmm Hypertension stage I. In spite of maximum dose double therapy. See FIP HT stage I.
Give a third type of antihypertensive drug in half of the maximum dose.

Goal blood pressure? <140/90 or 130/80DM
Blood pressure measurement twice a day for 3 days

NO

YES

Optimised anti-hypertensive treatment: blood pressure in normal range HT FIP STOP

NO

Permanently elevated blood pressure, over 160/100Hgmm Hypertension stage II. In spite of low dose triple therapy.

Double the dose of the previously started third antihypertensive drug. (This will be the maximum dose of the three antihypertensive drug.)

Permanently elevated blood pressure, but under 160/100Hgmm Hypertension stage I. In spite of low dose triple therapy. See FIP HT st.I.

Figure 31. Treatment of Hypertension: after lifestyle optimization. Stage II (OVER 160/100HGMM) (Part III).
Goal blood pressure? 
<140/90 or 130/80DM
Blood pressure measurement twice a day for 3 days

YES

Optimised anti-hypertensive treatment: blood pressure in normal range
HT FIP STOP

NO

Permanently elevated blood pressure, over 160/100Hgmm
Hypertension stage II.
In spite of maximum dose triple therapy.

SEE SPECIALIST !!!
CONSIDER SECONDARY HYPERTENSION !!!
STOP HT FIP!!!

Permanently elevated blood pressure, but under 160/100Hgmm
Hypertension stage I.
In spite of maximum dose triple therapy.

SEE SPECIALIST !!!
CONSIDER SECONDARY HYPERTENSION !!!
STOP HT FIP!!!

Figure 31. Treatment of Hypertension: after lifestyle optimization. Stage II (OVER 160/100HGMM) (Part IV).
10.3 Hypertension Formal Intervention Plans

The K4CARE knowledge in the previous section is converted to the SDA structures of Figure 32, Figure 33, and Figure 34. These SDAs represent K4CARE formal intervention plans for Hypertension.

![SDA for Hypertension lifestyle modification](image)

**Figure 32. SDA for Hypertension lifestyle modification.**
Figure 33. SDA on Hypertension (Stage I).
Figure 34. SDA on Hypertension (Stage II).
11 Immobility

Immobility refers to the results of short-term as well as long-term immobilization. Prolonged inactivity and bed rest causes in fact pathological changes in most organs and systems of the body, generally known as immobilization syndrome or Immobility. Immobility is a highly prevalent problem in older people and is a frequent pathway through which many diseases produce further disability in elderly people. This results in pathological changes and decreased functional capacity of multiple body systems with clinical manifestation of Immobility (1). Most of the complications result from prolonged periods of rest. In fact, while the effects of immobilization are mostly reversible in young subjects, older persons have considerably more difficulty in recovering and prolonged Immobility can even lead to the loss of independence. The etiology of Immobility is often multifactorial; many physical, psychological and environmental factors can cause Immobility in older people (2). Among physical factors, acute and chronic diseases are the causes of Immobility in older people. Several common and treatable diseases and their associated impairments cause loss of mobility in older people. Musculoskeletal diseases are the most important causes of Immobility, followed by cardiovascular and neurological diseases. Many older people with Immobility have more than one disease or impairment, and these may interact to limit mobility. Psychological variables including personality trait, depression and fear of falling are also important risk factor to develop Immobility. An important component of mobility problems in older people is the home environment. Environmental conditions can be conceptualized as those which decrease performance, such as barriers, and those which maintain activity performance, as supports. Thus, the physical environment influences disability by moderating performance parameters of an activity or task. Even more, insufficient social support appears to be an important risk factor. Pathological changes derived from Immobility are produced in almost every body systems such as cardiovascular, musculoskeletal, respiratory, gastrointestinal, urinary systems; in addition pressure ulcers can develop also after a short period of immobilization.

The effects of the immobilization on some systems are summarized below:

- Skin: pressure ulcers.
- Musculoskeletal: loss of muscular strength, loss of muscular endurance, loss of bone mass (osteoporosis), loss of bone strength, decreased range of motion of joints, contractures.
- Cardiovascular: orthostatic hypotension (hypotension that occurs when a person assumes a standing position), venous thromboembolism.
- Pulmonary: decreased ventilation, atelectasis (a state in which the lung is collapsed), aspiration pneumonia.
• Genitourinary: urinary infection, urinary retention, bladder calculi, incontinence.

• Gastrointestinal: loss of appetite, constipation, faecal impaction (a condition when constipation is so important that there is a large mass of dry, hard stool in the rectum).

• Psychological: depression, sensory deprivation.

• Social: isolation, caregiver strain.

Immobility adversely affects the quality of life of older people, threatens independent living and personal autonomy, and increases both formal and informal care needs. Persons with difficulty in mobility use a disproportionate share of health care, and costs of caring for such persons will rise dramatically over the next two decades, since inactivity increases the risks of osteoporosis, diabetes, and cardiovascular disease.

11.1 General Treatment of Immobility

Immobility and its resultant complications can often be prevented. Bed rest and immobilization are amongst the most frequently prescribed therapies. However, inactivity, elimination of gravity and immobilization lead to multiple physiological changes. Early mobilization and measures to support mobility in chronic cases are of major importance in avoiding structural damage. When Immobility has already developed, the assessment starts with a careful history-taking and a thorough physical examination. The aim of the assessment is to identify physical, psychological and environmental factors that lead to Immobility and the time frame of the onset of Immobility. Immobility may be sudden and of recent onset; it may be caused by a sudden event such as a fall leading to a hip fracture; or a steady deterioration in mobility may be caused by a progressive illness such as Parkinson’s disease. Other useful information includes medical conditions that influence mobility and a review of the patient’s medications (e.g. antipsychotic drugs commonly induce Parkinsonism in elderly patients). Psychological factors such as depression and fear should receive special attention. The physical examination should search for causes and consequences of Immobility in an elderly patient. The skin should be assessed repeatedly to identify pressure ulcers. The pulse and blood pressure should be checked both lying and standing. A detailed musculoskeletal assessment should include evaluation of muscle tone and strength, joint range of motion and potentially reversible podiatric problems. The neurological examination should identify focal weakness as well as sensory and perceptual problems. Formal assessment of the mental state may reveal cognitive impairment that can frustrate rehabilitative efforts. Most importantly, the patient’s mobility should be assessed on a regular basis. There are several levels of mobility and the person’s level of functioning should be carefully documented.

Therapy is multifactorial and management of immobile patients involves a thorough assessment leading to a list of problems. Treatment focuses on restoration of function and improvement
of autonomy and quality of life by rehabilitation, and on preventing and treating all the clinical consequences of the immobilization. The mobilization of the patient should begin as soon as possible. The rehabilitation objectives may involve controlling or reversing disease activity, decreasing pain, restoring function or modifying the environment so that the patient’s needs are better met.

The references [56], [57], [58], and [59] have been taken into consideration.

11.2 K4CARE Knowledge on how to deal with Immobility

To our knowledge, clinical guidelines on Immobility or immobilization syndrome do not exist in the realm of evidence-based medicine. In the medical literature, there are some studies dealing with how to prevent the development of Immobility and about how to treat the main consequences of bed rest. But none of them deals with Immobility as a whole complex entity. We derived some recommendations for treatment from published papers and textbooks (2-3), while some result from expert clinical practice; in other cases, flowcharts derive from existing guidelines for the complication of immobility, such as bladder dysfunction, pain, etc. In the first phase (Figure 35) the patient affected by immobility undergoes a Multidimensional Evaluation; details about mobility level and self-dependency are obtained from IADL, Barthel Index and Tinetti scale. In order to provide practical suggestions, we distinguish a rehabilitative treatment for completely bedridden patients and one for patients with inability to perform mobility tasks (if the score of the Tinetti scale is under a definite value). All patients will be evaluated through a clinical history and a physical examination; if it is observed bowel or bladder dysfunction, and pain, recommendations expand in problem-specific flowcharts, one for each problem. If some patient scores lower than 14 at the Norton scale, the diagram refers to the FIP for pressure ulcers. The flowchart regarding the bedridden patient (Figure 36) describes actions derived from general recommendations and interventions normally used in clinical practice, mainly in rehabilitative settings. If the patient achieves the sitting position, treatment continues with training for standing up and ambulation. At the end of the rehabilitative treatment, a particular attention is paid to the prescription of assistive devices (wheelchair, canes, and walker) to support the patient in his activities of daily living. Management of both bladder and bowel problems should be seen as an essential part of the patient’s rehabilitation (Figure 37 and Figure 38) as they can seriously hamper progress in other areas. If either constipation or diarrhea is detected, actions to carry out are proposed; goals of management are to ensure adequate intake of fluids and fiber, and to help the patient establish a regular toileting schedule. If bladder dysfunction is detected, it is proposed to distinguish between two forms of incontinence and to adopt strategies to relieve symptoms; evaluation and treatment of bladder dysfunction are complex, simplified here with the proposal of two decision point and some therapeutic options. Pain occurring in Immobility may include joint pain from spasticity, stillness, muscle weakness, headache, centrally mediated pain etc. Prevention, assessment, and treatment of pain should continue throughout re-
habilitation care. In Figure 39 we distinguish two types of pain, for the need of simplification and without any intent to be exhaustive. Among the actions, some options for these types of pain are described.

\[ \text{Multidimensional Assessment} \]

- IADL = 3/8 and/or
- Barthel Index = 5 and/or
- Tinetti between 0 and 1
  (at least two)

\[ \text{Immobility Syndrome} \]

- Medical history
- Physical examination

\[ \text{Patient bedridden} \]

- Bowel dysfunction
- Bladder dysfunction
- Pain

\[ \text{See the section} \]

- Norton scale < 14

\[ \text{Pressure ulcers} \]

\[ \text{See the FIP} \]

- Mini Nutritional Assessment < 23.4

\[ \text{Malnutrition (and/or dehydration)} \]

\[ \text{See the section (in a different FIP)} \]

- MMSE < 23

\[ \text{Cognitive impairment} \]

\[ \text{See the FIP} \]

Figure 35. Immobility Syndrome Assessment.

11.3 Immobility Formal Intervention Plans

Flowcharts in Figure 35, Figure 36, Figure 37, Figure 38, and Figure 39 are represented as FIPs in the SDA notation in Figure 40, Figure 41, Figure 42, Figure 43, and Figure 44, respectively.
Figure 36. Patient bedridden Immobility treatment
Constipation or diarrhoea

Bowel dysfunction

Rectal examination

If it is negative for fecal impaction

- Adequate fluid intake
- Stimulatory laxative
- Dietary manipulation
- Bowel training

If it is positive for fecal impaction

Remove fecal impaction

Constipation

There is improvement?

End of treatment

Yes

No

Urinary retention

Bladder dysfunction

Urinary retention

Intermittent catheterization

There is improvement?

Yes

End of treatment

No

Urge incontinence

- Bladder training
- Anticholinergic drugs

There is improvement?

Yes

End of treatment

No

Bladder catheterization

Figure 37. Treatment of bowel dysfunction for Immobility

Figure 38. Treatment of bladder dysfunction for Immobility
Pain?

Musculoskeletal pain

Non pharmacological treatment:
- Exercises
- Passive movements

There is improvement?

Yes

End of treatment

No

Pharmacological treatment

Central post-stroke pain

Figure 39. Treatment of pain for Immobility

Figure 40. SDA on Immobility Syndrome Assessment.
Figure 41. FIP about Patient bedridden in Immobility treatment
Figure 42. Bowel dysfunction in Immobility treatment
Figure 43. Bladder dysfunction in Immobility treatment

Figure 44. Pain in Immobility treatment
12 Parkinson’s Disease and Parkinsonism

Parkinson's disease is a disorder of the brain that leads to shaking (tremors) and difficulty with walking, movement, and coordination.

Parkinson's disease was first described in England in 1817 by Dr. James Parkinson. The disease affects approximately 2 of every 1,000 people and most often develops after age 50. It is one of the most common neurological disorders of the elderly. Sometimes Parkinson's disease occurs in younger adults, but is rarely seen in children. It affects both men and women.

In some cases, Parkinson's disease occurs within families, especially when it affects young people. Most of the cases that occur at an older age have no known cause.

Parkinson's disease occurs when the nerve cells in the part of the brain that controls muscle movement are gradually destroyed. The damage gets worse with time. The exact reason that the cells of the brain waste away is unknown. The disorder may affect one or both sides of the body, with varying degrees of loss of function.

Nerve cells use a brain chemical called dopamine to help send signals back and forth. Damage in the area of the brain that controls muscle movement causes a decrease in dopamine production. Too little dopamine disturbs the balance between nerve-signaling substances (transmitters). Without dopamine, the nerve cells cannot properly send messages. This results in the loss of muscle function.

Some people with Parkinson's disease become severely depressed. This may be due to loss of dopamine in certain brain areas involved with pleasure and mood. Lack of dopamine can also affect motivation and the ability to make voluntary movements.

Early loss of mental capacities is uncommon. However, persons with severe Parkinson's may have overall mental deterioration (including dementia and hallucinations). Dementia can also be a side effect of some of the medications used to treat the disorder.

Parkinson's in children appears to occur when nerves are not as sensitive to dopamine, rather than damage to the area of brain that produces dopamine. Parkinson's in children is rare.

The term "parkinsonism" refers to any condition that involves a combination of the types of changes in movement seen in Parkinson's disease. Parkinsonism may be caused by other disorders (such as secondary Parkinsonism) or certain medications used to treat schizophrenia.

12.1 General Treatment of Parkinson's Disease and Parkinsonism

Parkinson's Disease (PD) is a progressive neurodegenerative condition resulting from the death of the dopamine containing cells of the brain. People with PD classically present with the symptoms and signs associated with parkinsonism, namely hypokinesia (i.e. poverty of movement), bradykinesia (i.e. slowness of movement), rigidity and rest tremor. These symptoms usually develop after age 60, although some people affected by Parkinson's disease are younger than age 50. The diagnosis is primarily a clinical one based on the history and examination [53]. Early symptoms of
PD are subtle and occur gradually; nevertheless in some people the disease progresses more quickly than in others. Parkinson's disease is progressive, meaning the signs and symptoms become worse over time. But although Parkinson's disease may eventually be disabling, the disease often progresses gradually, and most people have many years of productive living after a diagnosis. Then, as these symptoms become more pronounced, patients may have difficulty walking, talking, or completing other simple tasks. Although PD is predominantly a movement disorder, other impairments frequently develop, including psychiatric problems such as depression and dementia. Difficulty in swallowing and speaking, urinary problems or constipation, skin problems, sleep disruptions and pain may later ensue, and the condition progresses to cause significant disability and handicap with impaired quality of life for the affected person and their families. No one can predict which symptoms will affect an individual patient, and the intensity of the symptoms also varies from person to person. Several other disorders have certain features that are similar to those of PD, and are sometimes mistaken for PD. Evidence suggests that the diagnosis of PD should be reviewed regularly and reconsidered if atypical clinical features develop [53].

**General Treatment of Parkinson's disease**

Symptomatic therapies for PD treat the symptoms of the disease but do not necessarily slow the rate of progression of the condition. For many people with Parkinson's, the initial response to treatment can be dramatic. Over time, however, the benefits of drugs frequently diminish or become less consistent, although symptoms can usually still be fairly well controlled. While no treatments have yet been shown conclusively to slow the disease, a large number of drugs are available to treat symptoms, as well as several forms of surgery and numerous non-pharmacological approaches. It was evident from reviewing the evidence-base that there is no single drug of choice in the initial pharmacotherapy of early PD. The standard symptomatic therapy for PD for more than 30 years has been levodopa; however, levodopa preparations contribute to the development of motor complications in PD (abnormal involuntary movements, dyskinesias, dystonia, response fluctuations in which people experience ‘on-off’ of state). To avoid motor complications, the strategy of delaying the introduction of levodopa has developed. Dopamina agonists were introduced as adjuvant therapy to levodopa in later disease, but, more recently, trials have examined their effects as initial monotherapy in the hope that they may delay the onset of motor complications. The choice of drug first prescribed should take into account:

- Clinical and lifestyle characteristics
- Patient preference, after the patient has been informed of the short- and long-term benefits and drawbacks of the drug classes [53].

Treatment of PD includes other drugs with different actions such as β-blockers, amantadine, anticholinergics, MAO-B inhibitors, etc. Guidelines usually divided patients in two groups: a
first group with early disease to guide the choice of the first pharmacological treatment and a second one with later disease, already in treatment with levodopa, that have developed motor complications. Besides, evidence suggests different approach to detect and treat non-motor complications. When symptoms are poorly controlled by pharmacological treatment, surgery options can be considered. Next to pharmacological therapy other interventions have to be carried out, such as physiotherapy, occupational therapy and speech therapy. Evidence demonstrated that these therapies should be available for patients with PD [53]. Physical therapy, especially, can be extremely helpful for people with Parkinson's disease — both in the early stages and later, as the disease progresses. It can help improve mobility, range of motion and muscle tone. Although specific exercises can't stop the progress of the disease, improving muscle strength can help patient feel more confident and capable.

12.2 K4CARE Knowledge on how to deal with Parkinson’s Disease and Parkinsonism

On the basis of the recommendations of the guidelines and from clinical experience, some flowcharts have been derived regarding the steps to follow in the patients with PD. Because of the difficulty to treat as a whole symptoms and complications, both of the disease and of pharmacological therapy, it has been decided to make schemas only for major indications. In detail, in the first part of Figure 45 we focused on the detection of non-motor complications such as dysarthria and swallowing difficulties and on the education of patients and families about how to prevent some complications, such as environmental issues. If these kinds of problems are detected, patients have to be referred to a specialist or to speech therapy. Afterwards, we focused on physiotherapy. In order to distinguish patient with minimal or no functional impairment from patients with more pronounced disability or confined to bed or wheelchair unless aided, we subdivided patients according to a simplified version of the Hoehn and Yahr classification [54]. In Figure 46 it is reported a flowchart about the choice of the pharmacological therapy in early disease; this diagram is derived from Italian guideline formulated by LIMPE and suggests some recommendations on best practice [55]. Moreover, given the error rate in making a diagnosis of PD, even when made by experts, it is apparent that the diagnosis should be kept under regular review and revised if patients do not respond to conventional therapy.
Figure 45. Treatment of Parkinson’s disease.
Figure 46. Pharmacological Treatment in early Parkinson’s disease.

12.3 Parkinson’s disease and Parkinsonism Formal Intervention Plans

Treatments described in Figure 45 and Figure 46 are represented as K4ACRE FIPs in Figure 47 and Figure 48, respectively.
Figure 47. Treatment of Parkinson's disease
Figure 48. Initial pharmacotherapy in early Parkinson’s disease
13 Conclusions and Acknowledgements

This document complements and closes a series of three documents on the description and formal transformation of procedural knowledge about the treatment of the diseases the K4CARE project deals with. The procedure followed for the construction of all the Formal Intervention Plans has been the same: select relevant bibliography (primarily guidelines) that provides the perspective of the diseases that K4CARE requires, expert analysis and integration of the contents of the bibliography for each disease, elaboration of a direct and concise description of all the relevant aspects of the treatment that are useful to K4CARE, transformation or development of health-care diagrams to summarize all the procedural knowledge as a continuous care, and transformation of these diagrams in FIPs as a result of a cyclic knowledge engineering process.

Once the final FIPs are validated by the medical experts of the K4CARE project, this document was structured to describe all these elements: disease description, general remarks on the treatment of the disease, adaptation of the treatment to the K4CARE needs with a final representation as flowcharts, and final FIPs.
14 Bibliography


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