ANEXOS

ÍNDICE DE LOS ANEXOS

ANEXO I: ÁRBOL DE GÉNEROS MÉDICOS DEL GRUPO DE INVESTIGACIÓN GENTT (GÉNEROS TEXTUALES PARA LA TRADUCCIÓN[321,322])

Clínicos

Características de productos para profesionales

Carta de resultados

Citación

Dieta

Ficha de seguridad de medicamento

Hoja de vigilancia de enfermedades

Guía clínica

Historia clínica

Consentimiento informado

Electrocardiograma

Estudio post mortem

Evolución de enfermería

Hoja clinicoadministrativa de hospitalización

Hoja clinicoadministrativa de consulta externa

Hoja de administración de medicamentos

Hoja de anamnesis y exploración

Hoja de anestesia

Hoja de autorización de ensayo clínico

[321] Hemos decidido emplear las siguientes convenciones de formato a la hora de ilustrar el árbol de géneros médicos del grupo GENTT: los macrogéneros se muestran en negrita, los géneros en redonda y los subgéneros en cursiva.

[322] En palabras de la directora del grupo de investigación GENTT, Isabel García Izquierdo, esta taxonomía debe entenderse como una propuesta "con finalidad explicativa, cuya pertinencia ha sido consensuada después de varios años de investigación, pero no por ello pretende erigirse en definitiva. Más bien al contrario, esperamos que el devenir de la propia investigación nos permita ir modificando y mejorando esa primera aproximación taxonómica. La finalidad de la propuesta es, pues, dibujar las líneas de conocimiento general y conocimiento experto y las relaciones sociales existentes en cada una de las áreas estudiadas" (2009: 21).

Hoja de autorización de autopsia

Hoja de evolución

Hoja de examen radiológico

Hoja de infección hospitalaria

Hoja de informe citológico

Hoja de informe de alta

Hoja de informe quirúrgico

Hoja de interconsulta

Hoja de informe anatomopatológico

Hoja de observaciones y curas

Hoja de órdenes médicas

Hoja de urgencias

Hoja de circulante

Hoja de codificación

Hoja de constantes

Hoja de control de exámenes complementarios

Hoja de demanda quirúrgica programada

Hoja de determinaciones analíticas

Hoja de donación de órganos

Hoja de lista de problemas

Hoja de petición de alta voluntaria

Hoja de postanestesia

Hoja de preanestesia

Hoja de seguimiento intensivo

Hoja de solicitud de estudio anatomopatológico

Hoja de solicitud de estudio citológico

Hoja de solicitud de ingreso

Hoja de tratamiento convencional

Hoja de transfusión

Hoja de trabajo social

Hoja de valoración de enfermería

Fórmula lucocitaria

Gráfica anestésica

Hemograma

Informe clínico

Informe de exploraciones especiales

Informe preoperatorio

Parte de asistencia por lesiones

Informe forense

Manual de instrucciones

Manual médico

Parte

Informe de consulta y hospitalización

Parte de alta

Parte de baja

Parte de confirmación

Programa informático de diagnóstico

Prospecto de medicamento

Protocolo

Cuestionario sanitario

Reconocimiento médico periódico

Reconocimiento neonatal

Vademécum

Divulgativos

Artículo de opinión

Artículo temático

Atlas visual

Características de producto para pacientes

Carta

Comunicado de prensa

Conferencia

Cuento médico infantil

Enciclopedia divulgativa

Folleto informativo

Guía divulgativa

Informe anual

Libro divulgativo

Noticia

Parte epidemiológico

Preguntas más frecuentes (FAQ)

Resumen para pacientes

Información para pacientes

Metagéneros

Base de datos médica

Diccionario médico

Manual de estilo

Normas para autores

Tesauro

Pedagógicos

Atlas anatómico

Libro de texto

Tratado

Tutorial

Publicitarios

Anuncio para pacientes

Anuncio para profesionales

Artículo publicitario

Catálogo de productos sanitarios

Folleto publicitario

Publirreportaje

Investigación

Artículo de revisión

Artículo en actas de congreso

Artículo especial

Artículo original

Carta al director

Carta científica

Caso clínico

Conferencia clínica

Documento de consenso

Editorial científico

Expediente de registro farmacéutico

Informe

Informe farmacológico

Nota clínica

Noticia

Original breve

Patente de temática médica

Cuaderno de recogida de datos

Reseña bibliográfica

Tesis doctoral

Trabajo de investigación

ANEXO II: COMPETENCIAS Y SUBCOMPETENCIAS ESTABLECIDAS POR LA RED DE MÁSTERES EUROPEOS EN TRADUCCIÓN (EMT)

TYPE OF COMPETENCE	*DEFINITIONS / COMPONENTS*
TRANSLATION SERVICE PROVISION COMPETENCE	**INTERPERSONAL dimension** - Being aware of the social role of the translator - Knowing how to follow market requirements and job profiles (knowing how to remain aware of developments in demand) - Knowing how to organise approaches to clients/potential clients (marketing) - Knowing how to negotiate with the client (to define deadlines, tariffs/invoicing, working conditions, access to information, contract, rights, responsibilities, translation specifications, tender specifications, etc.) - Knowing how to clarify the requirements, objectives and purposes of the client, recipients of the translation and other stakeholders - Knowing how to plan and manage one's time, stress, work, budget and ongoing training (upgrading various competences) - Knowing how to specify and calculate the services offered and their added value - Knowing how to comply with instructions, deadlines, commitments, interpersonal competences, team organisation - Knowing the standards applicable to the provision of a translation service - Knowing how to comply with professional ethics - Knowing how to work under pressure and with other experts, with a project head (capabilities for making contacts, for cooperation and collaboration), including in a multilingual situation - Knowing how to work in a team, including a virtual team - Knowing how to self-evaluate (questioning one's habits; being open to innovations; being concerned with quality; being ready to adapt to new situations/conditions) and take responsibility

	PRODUCTION dimension - Knowing how to create and offer a translation appropriate to the client's request, i.e. to the aim/skopos and to the translation situation - Knowing how to define stages and strategies for the translation of a document - Knowing how to define and evaluate translation problems and find appropriate solutions - Knowing how to justify one's translation choices and decisions - Mastering the appropriate metalanguage (to talk about one's work, strategies and decisions) - Knowing how to proofread and revise a translation (mastering techniques and strategies for proofreading and revision) - Knowing how to establish and monitor quality standards
LANGUAGE COMPETENCE	**-** Knowing how to understand grammatical, lexical and idiomatic structures as well as the graphic and typographic conventions of language A and one's other working languages (B, C) - Knowing how to use these same structures and conventions in A and B - Developing sensitivity to changes in language and developments in languages (useful for exercising creativity)
INTERCULTURAL COMPETENCE (the dual perspective – sociolinguistic and textual – is in the comparison of and contrast between discursive practices in A, B and C)	**SOCIOLINGUISTIC dimension** - Knowing how to recognise function and meaning in language variations (social, geographical, historical, stylistic) - Knowing how to identify the rules for interaction relating to a specific community, including non-verbal elements (useful knowledge for negotiation) - Knowing how to produce a register appropriate to a given situation, for a particular document (written) or speech (oral) **TEXTUAL dimension** - Knowing how to understand and analyse the macrostructure of a document and its overall coherence (including where it consists of visual and sound elements) - Knowing how to grasp the presuppositions, the implicit,allusions, stereotypes and

	intertextual nature of a document - Knowing how to describe and evaluate one's problems with comprehension and define strategies for resolving those problems - Knowing how to extract and summarise the essential information in a document (ability to summarise) - Knowing how to recognise and identify elements, values and references proper to the cultures represented - Knowing how to bring together and compare cultural elements and methods of composition. - Knowing how to compose a document in accordance with the conventions of the genre and rhetorical standards - Knowing how to draft, rephrase, restructure, condense, and post-edit rapidly and well (in languages A and B)
INFORMATION MINING COMPETENCE	- Knowing how to identify one's information and documentation requirements - Developing strategies for documentary and terminological research (including approaching experts) - Knowing how to extract and process relevant information for a given task (documentary, terminological, phraseological information) - Developing criteria for evaluation vis-à-vis documents accessible on the internet or any other medium, i.e. knowing how to evaluate the reliability of documentary sources (critical mind) - Knowing how to use tools and search engines effectively (e.g. terminology software, electronic corpora, electronic dictionaries) - Mastering the archiving of one's own documents
THEMATIC COMPETENCE	- Knowing how to search for appropriate information to gain a better grasp of the thematic aspects of a document (cf. Information mining competence) - Learning to develop one's knowledge in specialist fields and applications (mastering systems of concepts, methods of reasoning, presentation, controlled language, terminology, etc.) (learning to learn) **-** Developing a spirit of curiosity, analysis and

	summary
TECHNOLOGICAL COMPETENCE (mastery of tools)	- Knowing how to use effectively and rapidly and to integrate a range of software to assist in correction, translation, terminology, layout, documentary research (for example text processing, spell and grammar check, the internet, translation memory, terminology database, voice recognition software) - Knowing how to create and manage a database and files - Knowing how to adapt to and familiarise oneself with new tools, particularly for the translation of multimedia and audiovisual material - Knowing how to prepare and produce a translation in different formats and for different technical media - Knowing the possibilities and limits of MT

ANEXO III: CUESTIONARIO INICIAL REALIZADO A LOS ALUMNOS

TRADUCCIÓN CIENTÍFICA-TÉCNICA: INTRODUCCIÓN A LA TRADUCCIÓN BIOMÉDICA

NOMBRE Y APELLIDOS:

……

LUGAR DE PROCEDENCIA:

……..

EDAD: ☐ 18-25 ☐ + de 25

PLAN DE ESTUDIOS: ☐ Grado ☐ Licenciatura

CURSO: ☐ 3º ☐ 4º

1- ¿Cuál es tu lengua materna?

2- ¿Cuáles son tus lenguas de trabajo?

3- ¿Has realizado o estás realizando otros estudios superiores aparte de los de Traducción e Interpretación? En caso afirmativo, especifica cuáles y en qué nivel estás.

4- ¿Has cursado o estás cursando alguna otra asignatura relacionada con la traducción o la interpretación biomédica?

5- ¿Has traducido en alguna ocasión textos médico-sanitarios? En caso afirmativo, especifica qué tipos de textos, su temática y a quién iban dirigidos.

6- ¿Has realizado algún encargo profesional relacionado con la traducción/interpretación médicas? En caso afirmativo, especifica qué tipo y en qué contexto.

7- ¿Consideras que la traducción de textos biomédicos es un área relevante dentro de los estudios de Traducción? Explica tu respuesta.

8- ¿Piensas que el área de la traducción científico-técnica y más concretamente de la traducción biomédica es una especialidad demandada en el mercado laboral? ¿Por qué crees que sí/no?

9- ¿Cuál o cuáles consideras que son las principales lenguas de partida de los textos médicos? ¿Por qué?

10- ¿Quiénes son en tu opinión los principales empleadores/demandantes de este tipo de traducción?

11- ¿Cuáles son las dificultades principales que crees que tiene un texto de contenido médico?

12- ¿Consideras que es necesario tener conocimientos de medicina para traducir un texto de dicho ámbito? Explica tu respuesta.

13- ¿Piensas que se dedica suficiente tiempo a la traducción médica en la carrera para poder traducir este tipo de textos profesionalmente o crees que es necesario seguir especializándose después de graduarse? Explica tu respuesta.

14- ¿La traducción médica despierta tu interés como traductor?

15- ¿Qué esperas aprender en estas clases?

ANEXO IV: TEXTOS ORIGINALES TRADUCIDOS POR LOS ALUMNOS

Texto 1: Hepatitis C

The liver is the largest internal organ in our body and weights about 3.5 pounds. It fits into the top of the abdominal cavity and partially covers the stomach.

The two lobes of the liver are divided by a thick ligament, and the entire liver is enclosed by a thin layer of connective tissue. The liver's functional units are microscopic columns of cells, called lobules. Each lobule contains vertical sheets of hepatocytes, which are separated by cavities or sinusoids.

Between the sheets of hepatocytes are tiny canals –called "bile canaliculi". These carry bile secreted by hepatocytes into the bile ducts and from the common hepatic duct into the gall bladder. Here the bile is stored and finally released into the duodenum to support fat digestion.

Although the liver has a remarkable ability to regenerate after injuries, it is still affected by a variety of harmful diseases such as hepatitis, the medical term for an inflammation of the liver.

The hepatitis C virus is a blood-borne virus. It is known to have survived in dried blood for longer periods than many other viruses, HIV for example, possibly as long as three months. The Hepatitis C virus is transmitted primarily through the blood contamination.

When the hepatitis C virus gets into the blood stream, it attaches to liver cells, enters them, and starts to reproduce. The new viruses, made within the infected liver cell, exit into the bloodstream where they attach to and infect other liver cells. This process allows the infection to spread through the liver.

The infected cell raises alarm by releasing the so-called interferons. Additionally, surface marks appear on the surface of the cell which are recognized by the killer cells

of the immune system. The killer cells destroy the infected liver cells. This acute phase of inflammation is mild in terms of symptoms. It can be triggered by the abusive use of alcohol, medications, drugs, toxins or viruses. Despite the mild nature of inflammation and liver injury, the disease commonly progresses to fibrosis – the formation of scar tissue in the liver. In this advanced phase of the disease connective tissue cells substitute the killed hepatocytes.

When fibrosis increases, the architecture in the liver is distorted, and the severe disease is called cirrhosis.

TEXTO 2: HIV and AIDS

The human immunodeficiency virus (HIV) is a retrovirus - a virus built of RNA instead of more typical DNA. It attacks the very cells of the immune system that should be protecting the body against it - T lymphocytes and other white blood cells with CD4 receptors on their surfaces. The virus uses the CD4 receptor to bind with and thereby enter the lymphocyte. HIV then integrates itself into the cell's own DNA, turning the cell into a virus-generating factory. The new viruses break free, destroying the cell, then move on to attack other lymphocytes.

HIV kills by slowly destroying the immune system. Several weeks after initial infection, flu-like symptoms are experienced. Then the immune system kicks-in, and the virus mostly retreats into hiding within lymph tissues. The untreated, infected individual usually remains healthy for 5 to 15 years, but the virus continues to replicate in the background, slowly obliterating the immune system.

Eventually the body is unable to defend itself and succumbs to overwhelming opportunistic infections that rarely affect healthy people. Acquired Immune Deficiency Syndrome (AIDS) is the name given to this final stage of HIV infection, and is characterised by multiple, life-threatening illnesses such as weight loss, chronic diarrhoea, rare cancers, pneumonia, fungal conditions and infections of the brain and eye. Tuberculosis has become especially prevalent in AIDS victims.

HIV is found in body fluids such as: blood, semen, vaginal fluids and breast milk. It can be passed on through penetrative sex, oral sex and sharing contaminated needles when injecting street drugs or in hospitals. It can also be transmitted from a mother to her baby during pregnancy, childbirth or breastfeeding - though many children escape infection. HIV cannot be passed on through kissing, coughing, mosquito bites or touching.

Most anti-HIV drugs aim at stalling viral replication. Nucleoside analogues such as AZT (zidovudine) and also non-nucleoside reverse transcriptase inhibitors (NNRTIs), attack

the action of the viral enzyme reverse transcriptase. This prevents it from creating functional DNA which would otherwise integrate into the DNA of infected cells.

A third class block protease, an enzyme essential for generating functional virus particles. Protease inhibitors are the most effective of the three types of drugs, and AIDS mortality fell dramatically in the US when they were first licensed during the late 1990s. Fusion inhibitors are a newer type of drug that work by stopping HIV from binding with CD4 receptors that it uses to enter cells. Drugs that block another enzyme, integrase, are also under development.

TEXTO 3: Heart failure

Heart failure is the end stage of cardiac disease after the myocardium has used all its reserve and compensatory mechanisms. Once overt signs appear, half of patients die within 5 years despite medical management. Heart failure is most often a consequence of hypertension, CHD, valve deformity, diabetes, or cardiomyopathy. The various etiologies tend to coexist. CHD, frequently accompanied by hypertension, is responsible in more than 50 percent of cases and has been increasing in prevalence among new cases of heart failure. Left ventricular hypertrophy, hypertension, and valvular diseases are diminishing determinants. The risk of cardiac failure is increased two-to sixfold with CHD, angina conferring half the risk compared with MI. The dominant cause continues to be hypertension, which precedes failure in 75 percent of patients.

An estimated 4.8 million Americans have CHF. The prevalence increases with age to exceed 10 percent after age 60 (see Fig. 1-9). Each year there are an estimated 400,000 new cases. Based on the Framingham Study, heart failure is equally frequent in men and women, and the annual occurrence approaches 10 per 1000 population after 65 years of age (see Table 1-1). Survival following the diagnosis of heart failure is worse in men than in women, but even in women fewer than 15 percent survive much longer than 8 to 12 years (see Table 1-6). The prognosis is not much better than for most forms of cancer. The 1-year fatality rate for heart failure is high, with one in five patients dying. Sudden death is a common mode of exitus, occurring at six to nine times the general population rate. With an increasing geriatric population, heart failure is a formidable problem.

There is little indication that the declines in death rates from heart disease in general and from CHD in particular in the United States have been accompanied by an improvement in the incidence of heart failure. This cannot be readily explained. Some postulate that improved survival of patients with angina, MI, and hypertensive heart disease may result in an increased prevalence of chronic heart disease and ultimately

heart failure. Data from the Framingham Study indicate very little improvement to date in the ominous outlook following the onset of CHF. The median survival of 652 incident cases of CHF was only 1.7 years in men and 3.2 years in women, and the overall survival rates at 5 years were only 25 percent for men and 38 percent for women. The mortality increased with age in both sexes. If one adjusts for age, no significant changes in the prognosis of CHF are evident over the past four decades despite improvements in treatment. Advances in treatment of hypertension, myocardial ischemia, and valvular heart disease have not resulted in dramatic improvements in survival once heart failure ensues.

Despite the availability of potent glycosides, diuretics, and hypertensive agents, heart failure continues at a high incidence. Because of the high attributable risk of hypertension and CHD, their prevention and effective treatment would appear to be required to make a significant impact on the incidence of congestive heart failure.

TEXTO 4: Low-density lipoprotein cholesterol

Evidence of several types supports the concept that LDL is the primary atherogenic factor, and controlled clinical trials show that lowering LDL reduces the risk for coronary heart disease (CHD). Accordingly, the National Cholesterol Education Program (NCEP) has identified LDL cholesterol as the primary target of lipid-lowering therapy. Five decades of research on the role of LDL in the pathogenesis of CHD represents one of the major advances in modern medicine and public health.

Many studies in laboratory animals indicate that raising serum levels of LDL and related lipoproteins will initiate and sustain atherogenesis. Moreover, humans with genetic forms of severely elevated LDL exhibit premature atherosclerotic disease. For many years, it was believed that the major action of LDL was merely to deposit its cholesterol within the arterial wall. More recently, LDL has been found to be a proinflammatory response that is the hallmark of the atherosclerotic lesion. Elevated LDL appears to be involved with all stages of atherogenesis: endothelial dysfunction, plaque formation and growth, plaque instability and disruption, and thrombosis. Elevated LDL-cholestererol levels in the plasma lead to increased retention of LDL particles in the arterial wall, their oxidation, and the secretion of various inflammatory mediators and chemoattractants. One sequela of this is the disruption of endothelial cell function by oxidized LDL, with subsequent loss of production of nitric oxide. Treatment of elevated LDL-cholesterol levels has been shown to reestablish normal coronary vasodilatory response to acetylcholine. LDL is also a potent mitogen for smooth muscle cells.

The primacy of LDL as a pathogenic agent is supported by epidemiologic data of several types. In different populations, the risk for CHD is positively correlated with the serum total cholesterol level; the total cholesterol level in turn is highly correlated with LDL-cholesterol levels. The association between serum cholesterol levels and CHD risk is curvilinear (or long-linear). Risk rises exponentially at higher cholesterol levels. In populations that have very low total (and LDL) cholesterol, risk for CHD likewise is low, even when other CHD risk factors (cigarette smoking, hypertension, and diabetes) are

common. This latter observation strongly suggests that an elevated LDL cholesterol is the primary risk factor.

LDL lowering can be accomplished with nondrug and drug therapies. The importance of nondrug therapies must not be minimized. Chief among them are reducing intake of cholesterol-raising fatty acids (saturated and *trans* fatty acids) and dietary cholesterol. Statins head the list of cholesterol lowering drugs. Most patients tolerate statins with few side effects. Occasional patients will have a mild rise in liver transaminases, but this change is currently not believed to be an indication of hepatotoxicity. Rare patients will exhibit signs and symptoms of myopathy. This side effect is more likely to occur in patients who have chronic renal failure or liver disease or who are on drugs that utilize or inhibit the cytochrome P450 3A4 pathway. For every doubling of the dose of a statin, the LDL-cholesterol level will fall by about 6 percent; a more efficacious way to enhance LDL lowering is to combine statins with bile acid sequestrants. For patients with borderline elevated triglycerides (200 to 400 mg/dL) and high LDL, niacin or a statin is an acceptable first-line drug. When triglycerides exceed 400 mg/dL, a fibrate or niacin is usually the most appropriate first-line agent.

TEXTO 5: Drug Treatment of Lipid Disorders

ARTERIOSCLEROSIS of the coronary and peripheral vasculature is the leading cause of death among men and women in the United States and worldwide. In 1992, for example, cardiovascular disease accounted for 38 percent of deaths from all causes among men and 42 percent of all deaths among women in Washington State; nationwide, the mortality rate for cardiovascular disease is approximately 50 percent.

MECHANISMS OF ATHEROGENESIS

Central to the pathogenesis of arteriosclerosis is the deposition of cholesterol in the arterial wall.

Nearly all lipoproteins are involved in this process, including cholesterol carried by very-low-density lipoprotein (VLDL), remnant lipoprotein, and low-density lipoprotein (LDL), particularly the small, dense form. Conversely, cholesterol is carried away from the arterial wall by high-density lipoprotein (HDL). In healthy persons, these lipoproteins function to distribute and recycle cholesterol (Fig. 1).

Hepatic overproduction of VLDL can lead to increases in the serum concentrations of VLDL, remnant lipoprotein, and LDL, depending on the ability of the body to metabolize each of these types of lipoprotein. The most common and important lipid disorder involving this mechanism is familial combined hyperlipidemia (also referred to as mixed hyperlipemia). The primary disorders of lipoprotein metabolism are described in Table 1 and have been reviewed elsewhere.

The chief risk factors for cardiovascular disease are listed in Table 2. When these risk factors occur in combination with hyperlipidemia and low serum HDL concentrations, early cardiovascular disease is commonplace. Keys to prevention and treatment are the elimination or modification of risk factors, if possible, in conjunction with treatment of the specific lipid disorder.

A SECONDARY CAUSES OF HYPERLIPIDEMIA

Closely related to the numerous risk factors for cardiovascular disease are conditions that cause hyperlipidemia, including obesity, diabetes mellitus, hypothyroidism, and the nephrotic syndrome; alcohol ingestion; and therapy with oral estrogen, isotretinoin, sertraline hydrochloride, human immunodeficiency virus (HIV)–protease inhibitors, *b*-adrenergic

antagonists, glucocorticoids, cyclosporine, and thiazide diuretics. In general, each condition should be treated and any offending medications discontinued before a program to lower serum lipid concentrations is initiated. Patients with severe hyperlipidemia usually have two disorders — for example, diabetes mellitus and familial combined hyperlipidemia, familial hypertriglyceridemia, or remnant removal disease.

TARGET SERUM LIPOPROTEIN CONCENTRATIONS

The threshold serum total cholesterol and LDL cholesterol concentrations above which diet and drug therapy should be initiated, as well as the goals of therapy, have been defined by the National Cholesterol Education Program (Table 3).

The target serum LDL cholesterol concentration is less than 160 mg per deciliter (4.3 mmol per liter) for patients with no risk factors for heart disease or only one risk factor, less than 130 mg per deciliter (3.4 mmol per liter) for patients with two or more risk factors, and less than 100 mg per deciliter (2.6 mmol per liter) for those with cardiovascular disease (Table 3). Persons with diabetes also fall in this third category, even those with no apparent cardiovascular disease.

Reducing serum LDL cholesterol concentrations below the target levels does not necessarily result in a proportional reduction in the risk of cardiovascular disease, because of the attenuation of the cholesterol-heart disease relation at lower serum cholesterol concentrations.

Drug therapy is not recommended for premenopausal women and men under 35 years of age unless they have serum LDL cholesterol concentrations of more than 220 mg per deciliter (5.7 mmol per liter), because their immediate risk of heart disease is low.

TEXTO 6: Warfarin and Aspirin in Patients with Heart Failure and Synus Rhythm

Chronic heart failure is a major cause of illness and death. Heart failure is associated with a hypercoagulable state, formation of left ventricular thrombus, and cerebral embolism. It is also associated with both sudden death and death resulting from progressive heart failure that may be caused by unrecognized atherothrombotic events. As a result, there is a rationale for using oral anticoagulants to treat patients with chronic heart failure who are in sinus rhythm. However, the role of oral anticoagulants as compared with aspirin has not been clarified in patients with chronic heart failure. Early studies showed that anticoagulation reduced the rates of embolic events and death, but many patients in these trials had atrial fibrillation and clinically significant valvular heart disease, making interpretation of the results difficult. In retrospective analyses of data from large trials involving patients with a reduced left ventricular ejection fraction (LVEF), conflicting results have been reported. Unfortunately, these findings are of limited value, since the use of anticoagulants was not randomized or controlled, data were collected retrospectively, end points were not predefined or standardized, and patients with atrial fibrillation were included.

Several prospective studies comparing oral anticoagulants with aspirin were too small to provide conclusive evidence for the superiority of either agent. In the Heart Failure Long-Term Antithrombotic Study (HELAS), 197 patients were randomly assigned to warfarin, aspirin, or placebo; there was no significant difference among the groups in the incidence of embolic events. In the Warfarin/Aspirin Study in Heart Failure (WASH), 279 patients were randomly assigned to warfarin, aspirin, or placebo; there was no significant difference among the groups in the composite end point of death, stroke, or myocardial infarction, but the rate of hospitalization was highest among those receiving aspirin. The Warfarin and Antiplatelet Therapy in Chronic Heart Failure trial (WATCH), which was the most recent and the largest study, enrolled 1587 patients who were randomly assigned to warfarin, aspirin, or clopidogrel, with a mean follow-up period of 1.9 years. The results of this trial, which was terminated

prematurely owing to difficulties with recruitment, suggested that there was a reduction in the rate of ischemic stroke with warfarin as compared with aspirin but showed an increase in hospitalization for heart failure in the aspirin group as compared with the warfarin group. The Warfarin versus Aspirin in Reduced Cardiac Ejection Fraction (WARCEF) trial was designed to compare the efficacy and safety of warfarin with those of aspirin among a substantially larger number of patients, with the use of a double-blind, randomized design.

METHODS

Study Patients

Eligible patients were 18 years of age or older and had normal sinus rhythm, no contraindication to warfarin therapy, and an LVEF of 35% or less as assessed by quantitative echocardiography (or a wall-motion index of ≤1.2) or as assessed by radionuclide or contrast ventriculography within 3 months before randomization. Patients who had a clear indication for warfarin or aspirin were not eligible. Patients in any New York Heart Association (NYHA) functional class were eligible, but patients in NYHA class I could account for no more than 20% of the total number of patients undergoing randomization. Additional eligibility criteria were a modified Rankin score of 4 or less (on a scale of 0 to 6, with higher scores indicating more severe disability), and planned treatment with a beta-blocker, an angiotensin-converting-enzyme (ACE) inhibitor (or, if the side-effect profile with ACE inhibitors was unacceptable, with an angiotensin-receptor blocker), or hydralazine and nitrates. Patients were ineligible if they had a condition that conferred a high risk of cardiac embolism, such as atrial fibrillation, a mechanical cardiac valve, endocarditis, or an intracardiac mobile or pedunculated thrombus.