Murad Alam
Marisa Pongprutthipan
Editors

Body Rejuvenation

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Chapter 21
Ambulatory Phlebectomy

Marisa Pongprutthipan, Girish Munavalli, and Simon Yoo

Introduction

Ambulatory phlebectomy (AP) is a common, minor, office-based procedure using a specially designed hook inserted through a minute stab incision to avulse and completely remove superficial varicose veins. AP is generally well tolerated and results in a high degree of patient satisfaction. Advantages of AP include short surgical time, ability to be performed under local tumescent anesthesia, low recurrence rate compared to sclerotherapy, minimally obtrusive scars and dyschromia compared to other methods, and immediate postoperative ambulation, which helps prevent vascular complications.[1–4]

Indications

- Superficial varicose venous tributaries of the great saphenous vein (GSV) or small saphenous vein (SSV), perforator veins, and reticular varicose veins when distended, visible, and palpable on the surface of the skin (Fig. 21.1 and 21.2(a))
- Preferred for varicosities greater than 4 mm in diameter and flesh-colored (which are thicker wall and more resistant to treatment with sclerotherapy)
- Large tortuous distal veins (which are difficult to treat with endovascular procedures)

Contraindications

- Infection in the treatment area
- Severe arterial occlusive disease
- Bleeding tendency or coagulopathy
- Allergy to local anesthetics
- Severe peripheral edema or severe lymphedema
- Seriously ill patients (i.e., cardiovascularly compromised, etc.)
- Very elderly patients

Relative Contraindications

- Recent deep vein thrombosis
- Hypercoagulable states
- Pregnancy
- Untreated or poorly managed diabetes mellitus

Clinical Examination and Patient History

Before stripping any vessels or performing extensive phlebectomies, a preoperative evaluation must be completed. The deep venous system should be interrogated, and any source of venous hypertension needs to be identified by using duplex ultrasound.[1]

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Chapter 22
Endovenous Laser and Radiofrequency Treatment of Leg Veins

Marisa Pongprutthipan and Jeffrey T.S. Hsu

Introduction

Superficial varicosity is a common medical condition that is symptomatic in 20–30% of the US population. Classic symptoms of venous insufficiency are ankle edema, leg fatigue, aching, discomfort, and muscle cramps. Some patients develop associated complications, including stasis dermatitis, lipodermatosclerosis, skin atrophy, superficial thrombophlebitis, and venous ulcers. The treatment of varicose veins reduces symptoms and complications of chronic venous insufficiency and improves quality of life. Superficial varicose veins are often due to failure of the valves in the saphenous vein and at the saphenofemoral junction (SFJ), causing venous reflux. Until recently, traditional ligation and stripping has been the standard of care in the treatment of truncal varicosities. But there are some disadvantages, including a 20% recurrence rate in 5 years, requirement of 2–6 weeks of downtime, associated risks of general anesthesia, scars, and possible neurovascular and lymphatic vessel damage. As an alternative, there are currently available endovenous treatment options for superficial varicose veins: ultrasound guided foam sclerotherapy, endovenous radiofrequency, and endovenous laser treatment. These are minimally invasive in-office procedures with less pain, early ambulation, and less recovery time. In this chapter, we review techniques for endovenous laser and endovenous radiofrequency treatments.

Endovenous Laser Therapy[1–7]

In January 2002, an 810-nm diode laser (Diomed Inc., Andover, MA) received Federal Drug Administration (FDA) clearance for use in endovenous laser therapy. This procedure can be done in-office under local anesthesia. After vein treatment, patients are able to walk immediately and most individuals are able to return to work the next day. The mechanism of action is the ablation of the target vein through delivery of laser energy. Since then, several wavelengths have been used in endovenous laser therapy (810, 940, 980, 1,319, 1320, and 1470 nm). Shorter wavelengths (810, 940, and 980 nm) are absorbed by deoxygenated hemoglobin and transmit heat to the vein wall. The extent of thermal injury when using shorter wavelengths depends on the energy settings, pulse duration, presence of blood in the lumen, pullback rate, and amount of tumescent anesthesia. Since the hemoglobin is the target, endothelial destruction and thrombotic vein occlusions can occur. Thrombus may progress into the deep venous system creating a deep vein thrombosis (DVT), which is usually asymptomatic. The likelihood of DVT is less than 1%.[6] With longer wavelengths (1319, 1320, and 1470 nm), heat is generated when the laser energy is absorbed by the intracellular water of the vein wall and the water content of the blood. Heat is produced endoluminally and eccentrically distributed. This leads to vascular contraction, venous wall (especially tunica intima) destruction, inflammation, and ultimately, fibrosis. Some reports have suggested that this direct heating of the vein wall tends to be more effective, less painful, and results in less incidence of vessel perforation. The reported rates of great saphenous vein (GSV) occlusion from endovenous laser treatments range from 84 to 100%. Recanalization is rare, but may occur as early as 1 week after treatment.[5] The mechanisms of recanalization remain unclear; however, some postulated factors include improper performance, laser fluence, anticoagulant or antiplatelet medication use during the perioperative period, and patients with body mass index greater than 35 kg/m^2. Some commercially available endovenous lasers are provided in Table 22.1.

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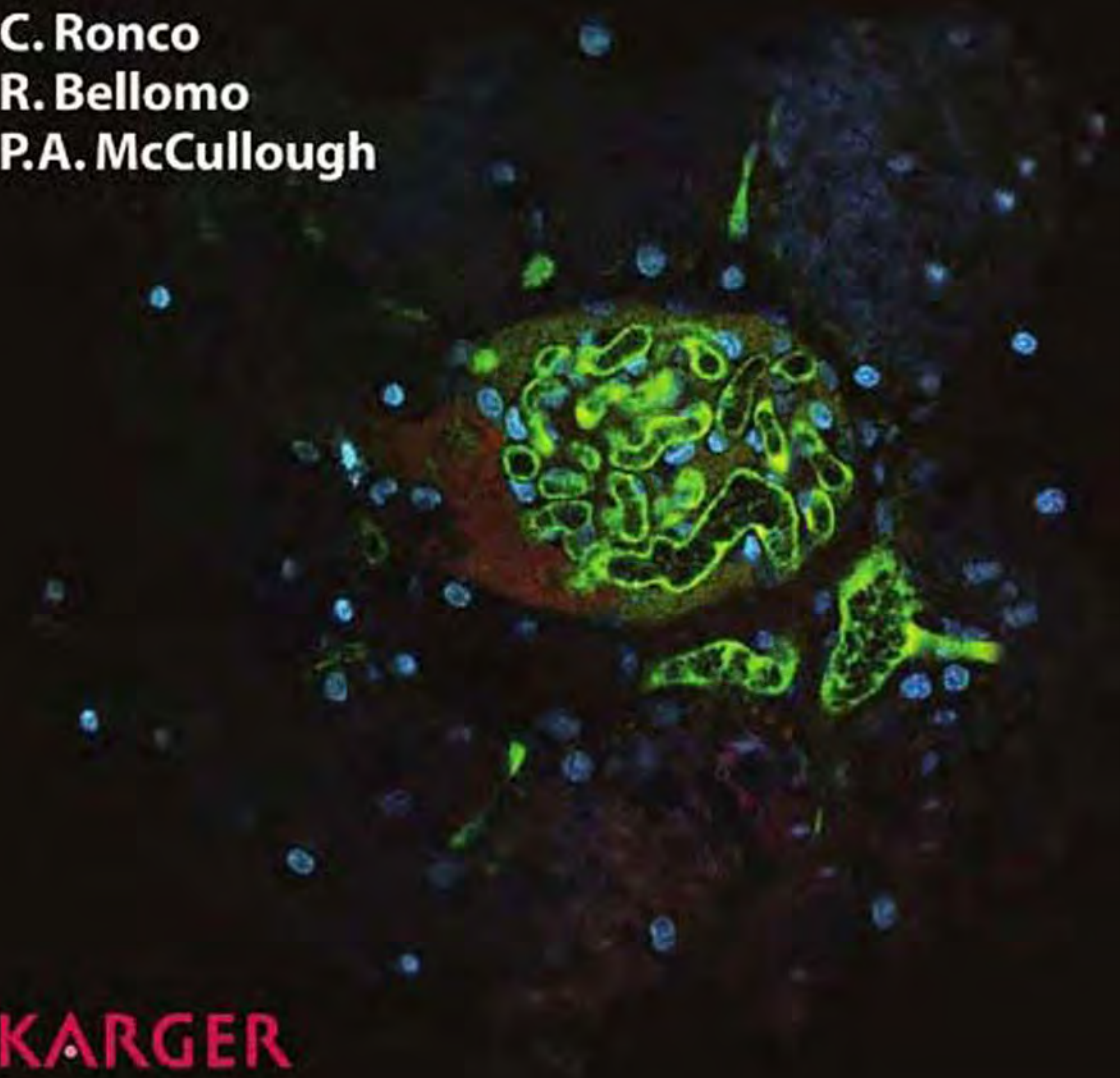
C. Ronco
R. Bellomo
P.A. McCullough



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Recovery from Acute Kidney Injury: Determinants and Predictors

Nattachai Srisawat[a] · Raghavan Murugan[a] · Xiaoyan Wen[a] · Kai Singbartl[a] · Gilles Clermont[a] · Somchai Eiam-Ong[b] · John A. Kellum[a]

[a]The CRISMA (Clinical Research, Investigation, and Systems Modeling of Acute Illness) Laboratory, Department of Critical Care Medicine, University of Pittsburgh School of Medicine, Pittsburgh, Pa., USA, and [b]Division of Nephrology, Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

Abstract

Predicting recovery of renal function following acute kidney injury (AKI) is one of the top ten questions in the field of AKI research. Accurate prediction would help physicians distinguish patients with poor renal prognosis in whom further therapy is likely to be futile from those who are likely to have good renal prognosis. Proper stratification of patients with AKI is also critical to design clinical trials to target patients with poor prognosis. Unfortunately, current general clinical severity scores (APACHE, SOFA, etc.) and AKI-specific severity scores (Mehta's score, Liano's score, Chertow's score, etc.) are not the good predictors of renal recovery. Recent progress on the pathophysiology of renal injury and recovery is encouraging. Repopulation of surviving renal tubular epithelial cell with the assistance of certain renal epithelial cell and specific growth factors such as neutrophil gelatinase-associated lipocalin (NGAL), hepatocyte growth factor (HGF), epidermal growth factor, and insulin-like growth factor-1, etc., play a major role in the recovery process. Such findings provide a great opportunity to test and validate these potential biomarkers as candidate markers of renal recovery. This review will describe the current understanding of the renal recovery process, and the role of clinical severity scores and novel biomarkers such as NGAL, HGF, and cystatin C in predicting renal recovery.

The prevalence of renal recovery varies by the definition and the study population (ICU vs. non-ICU). Results from the Beginning and Ending Supportive Therapy (BEST) Kidney Study group, a large multicenter, multinational,

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Review: Molecular Biology Approach to Viper Venoms

Ponlapat Rojnuckarin[1]*, Chuanchom Muanpasitporn[1],
Pon Singhamatr[1] and Jaradpong Arpijuntarangkoon[1]

ABSTRACT

Viper venoms comprise a large array of proteins affecting endothelium, platelets, blood coagulation and anti-coagulation, fibrinolysis, blood circulation, extracellular matrix, as well as cancer biology. Surprisingly, this plethora of functions is generated from limited molecular structures, e.g. snake venom serine protease, snake venom metalloprotease/disintegrin, C-type lectin-like protein and phospholipase A_2. Venom gland accelerated evolution greatly diversifies their activities to fit a wide variety of preys. Several derivatives of these components have been, or potentially are, useful diagnostic or therapeutic agents. Molecular cloning and recombinant expression of these peptides give us complete and accurate amino acid sequences to correlate with their functions and to compare with related proteins. This will provide the deeper insights in pathogenesis of snakebites and the molecular mechanisms of protein functions. The cDNA allows us to make a large amount of pure recombinant proteins and perform mutagenesis for detailed structure-function studies. Therefore, current molecular technology may be able to mimic the 'nature experiments' by engineering proteins with desirable activities from these molecular platforms for clinical and/or research uses.

Key words : Viper venom, molecular cloning, serine protease, snake venom metalloproteinase, disintegrin, C-type lectin-like protein, phospholipase A_2

* *Snake Venom and Snakebite Research unit Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand.*
* *Corresponding author* : E-mail : rojnuckarinp@gmail.com

ANDRES M. LOZANO
PHILIP L. GILDENBERG
RONALD R. TASKER
EDITORS

Textbook of Stereotactic and Functional Neurosurgery

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110 Diagnosis and Medical Management of Cervical Dystonia

R. Bhidayasiri · D. Tarsy

Torticollis is not a diagnosis but a physical sign which is characterized by twisting contractions of the neck muscles resulting in forced turning of the head. Therefore, there are many causes of torticollis, both dystonic and non-dystonic in origin. Although primary cervical dystonia (CD) represents the most common cause of torticollis, other non-dystonic (pseudodystonia) causes of torticollis, such as congenital disorders or local musculoskeletal changes in the cervical region, may resemble CD. This chapter will discuss the differential diagnosis of torticollis, particularly how to make a diagnosis of CD and how CD can be distinguished from non-dystonic causes of torticollis and other abnormal head postures. In addition, this chapter will cover a wide variety of disorders affecting the central and peripheral nervous systems, which are associated with secondary forms of CD. The last section of the chapter will focus on the medical management of CD. Although unfortunately relatively limited in efficacy, a variety of pharmacologic agents have been tried, including drugs affecting cholinergic, dopaminergic, serotonergic, and γ-aminobutyric acid (GABA) systems.

Many Causes of Torticollis: When to Diagnose Cervical Dystonia

The diagnostic evaluation of patients with torticollis requires a determination of the following: First, physicians have to observe whether the head deviation represents dystonia, non-dystonic torticollis, or some other type of hyperkinetic movement disorder. Second, it must be determined whether the dystonia is localized to the neck region or is a combination of a segmental or generalized pattern of distribution. Third, it must be determined whether the CD is idiopathic or symptomatic (secondary) to an underlying, identifiable cause. Lastly, other associated movement disorders (e.g., tremor) or secondary complications (e.g., cervical spondylosis) should be identified.

By definition, torticollis refers to abnormal cervical postures, which are characterized by tonic or intermittent spasms of neck muscles that cause involuntary deviation of the head from the normal position [1,2]. Therefore, there are many causes of torticollis which are either dystonic or non-dystonic in nature [3–5]. The terms "non-dystonic disorders" or "pseudodystonia" refer to disorders that are associated with sustained muscle contractions possibly occurring as a reflex mechanism or reaction to some other disturbance such as, for example, a trochlear nerve palsy or hemianopia which result in cocking of the head to improve vision, or Sandifer syndrome in which gastric reflux causes spasmodic retrocollis [6]. On the other hand, CD is a primary focal dystonia which manifests as sustained involuntary contractions of the neck muscles that result in abnormal movements and postures of the head [7]. With these two operational definitions, several non-dystonic causes of torticollis can be broadly

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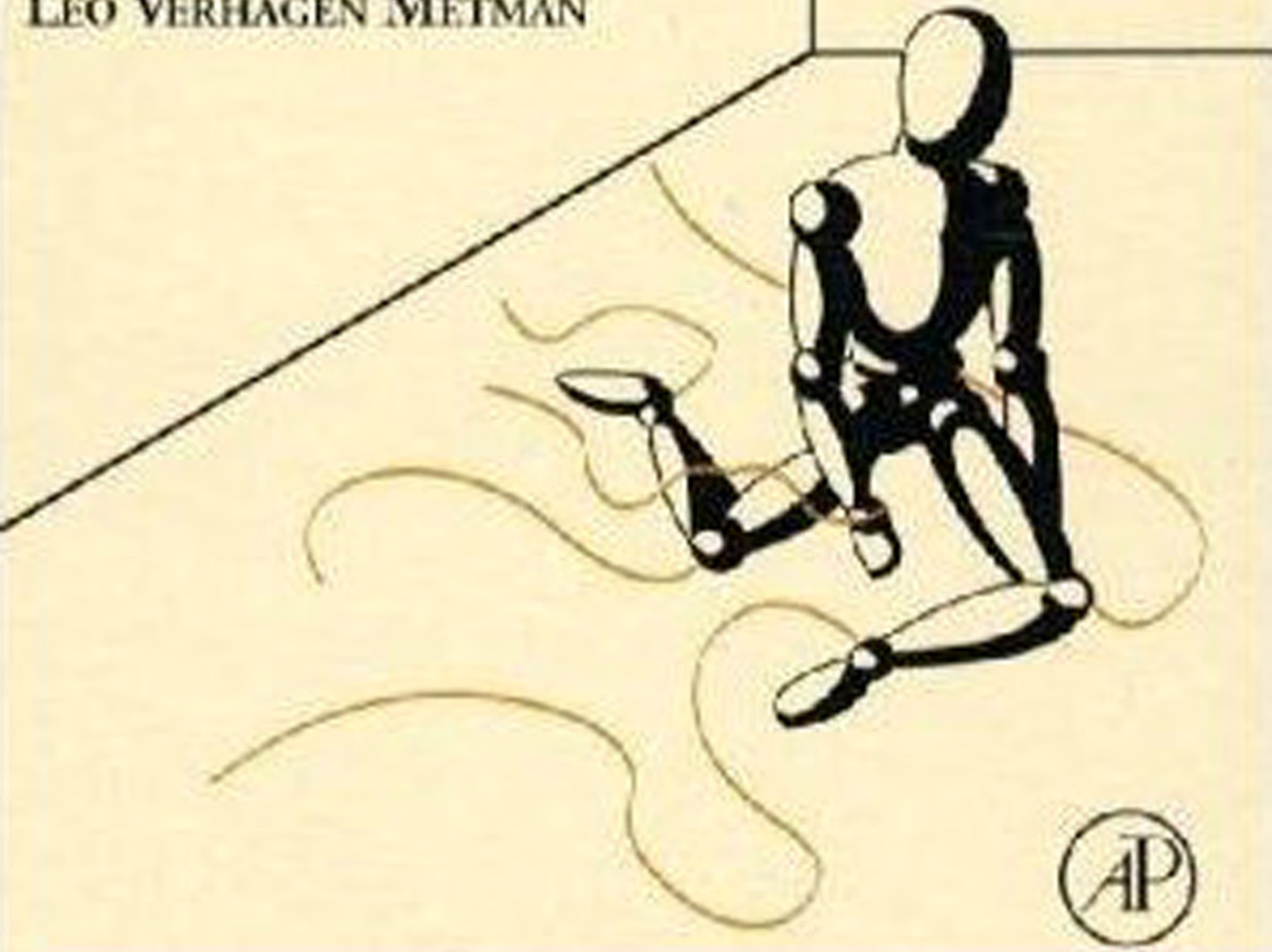
THE ENCYCLOPEDIA OF MOVEMENT DISORDERS

EDITORS
KATIE KOMPOLITI AND
LEO VERHAGEN METMAN



Friedreich's Ataxia and Variants

R Bhidayasiri, Chulalongkorn University Hospital, Bangkok, Thailand; David Geffen School of Medicine at UCLA, Los Angeles, CA, USA

Glossary

Ataxia – Impaired coordination, dexterity, or gait in the absence of significant muscular weakness.
Frataxin – A highly conserved nuclear-encoded protein localized in mitochondria.
***FRDA* gene** – The *FRDA* gene is localized on the long arm of chromosome 9, just below the pericentromeric heterochromatic region.
Friedreich's ataxia – The most common autosomal recessive and hereditary ataxia due to the hyperexpansion of a GAA triplet repeat in the first intron of the *FRDA* gene.

Definition and History

In 1863, Nicholaus Friedreich described a specific form of spinal degeneration with distinctive clinical and pathological findings in nine members of three siblings attending the university clinic. Originally termed 'degenerative atrophy of the posterior columns of the spinal cord,' Friedreich was able to pinpoint all essential clinical and pathological features of the disease, including progressive ataxia, sensory loss, muscle wasting and weakness, often with scoliosis, foot deformity, and heart disease, but just to miss deep tendon reflexes. He also noted the tendency of the disease to afflict several individuals in a sibship, but not affect parents. Initially suspected to be a form of neurosyphilis, it was not until 1882 when the new disease was given the name Friedreich's ataxia [MIM229300] (FRDA) or Friedreich's disease by some authors since ataxia is not always a principal feature. During the past 40 years, a number of landmark studies were carried out to establish the autosomal recessive (AR) pattern of inheritance as well as define vigorous diagnostic criteria, which are the key for the collection of clinically homogeneous families to be used for biochemical and molecular genetic studies.

Pathogenesis

Neuropathology

Despite Friedreich's emphasis on the degeneration of the posterior columns of the spinal cord as the hallmark of the disease, the main pathological features of FRDA are not limited to the central nervous system, but start with an early loss of large sensory neurons in dorsal root ganglia, followed by degeneration of posterior columns, spinocerebellar and pyramidal tracts. Indeed, the pattern of atrophy of the long tract fibers suggests a 'dying back' process (severely atrophic at the lumbar level, much less so in the cervical cord and in the brainstem, and of normal appearance in the cerebral peduncle), suggesting that the degenerative process more likely affects first the axon before cell bodies.

Difference from the severity of ataxia is observed in patients, and cerebellar atrophy is not a characteristic feature of FRDA. Only a mild loss of Purkinje cells and axonal torpedoes are observed in the cerebellar cortex. This is in contrast to the early loss of large primary sensory neurons in the dorsal root ganglion, while the motor component is well preserved, resulting in the presence of sensory axonal polyneuropathy on clinical examination.

In summary, the main neuropathological findings in FRDA are characterized by atrophy of the central sensory and cerebellar efferent pathways as well as the distal portion of the corticospinal tracts, which carry crucial information to the brain and cerebellum for correct execution of movement and for equilibrium. As a result, the degeneration of each of these systems contributes to the characteristic clinical picture of FRDA.

Systemic Consequences

Since many patients with FRDA die as a consequence of heart disease, we may assume that degeneration probably affects independent sites outside the nervous system. Indeed, the heart is clinically or subclinically affected in the vast majority of patients. While hypertrophic cardiomyopathy is the typical finding in most patients, dilated cardiomyopathy is frequently observed after a long disease course. Microscopically, hypertrophic cardiomyocytes are intermingled with fibers undergoing atrophy or granular degeneration. In addition, 10% of FRDA patients have diabetes mellitus at later stages due to a loss of β-pancreatic islet cells.

Genetics and Frataxin

By the application of molecular genetic methods, we now understand that degeneration in FRDA develops as a result of a loss of frataxin, a highly conserved