

Training Letter 00-06
Training letter on diabetes mellitus and its complications

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Director (00/21) 211A
All VBA Regional Offices and Centers Training Letter 00-06

SUBJECT: Training letter on diabetes mellitus and its complications

1. Enclosed is training material that includes both medical information and some rating guidelines on diabetes mellitus and its complications. This letter is not intended to make policy but to restate and clarify existing policy.
2. If you have any questions or comments about the content of this letter, or note any errors, please check the appropriate Calendar pages at: <http://vbaw.vba.va.gov/bl/21/calendar/calendar.htm>.

/S/

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Enclosure

Diabetes mellitus and its complications

What is diabetes mellitus (DM)?

Diabetes mellitus is a metabolic disorder in which the body is unable to use glucose (a type of sugar obtained from food) effectively. Hyperglycemia, an abnormally high level of blood sugar, results.

What does the pancreas have to do with diabetes?

Glucose is the main source of fuel for the body for energy and growth. There is a narrow range of blood glucose that is optimal—70 mg/dl to 110 mg/dl. Insulin and glucagon are hormones produced by the pancreas that regulate the level of blood glucose. Most of the pancreatic tissue produces enzymes that aid in

digestion, but about 5% produces hormones instead. Insulin is produced in the pancreas by specialized cells called beta cells in the islets of Langerhans or pancreatic islets (groups of cells scattered throughout the pancreas), and glucagon is produced by alpha cells in the islets of Langerhans.

What is the action of insulin and glucagon?

After eating sugar or starch, the blood glucose level rises. This high blood glucose is the signal for the pancreas to release insulin, which then lowers the blood glucose by moving it out of the blood into cells. The blood glucose then falls to normal. Insulin helps convert glucose to glycogen, which is stored in the liver and muscles and released when glucose is needed, for example, during exercise. As the blood glucose falls, the amount of insulin secreted by the pancreas goes down.

Too much insulin causes hypoglycemia (low blood glucose level), and too little insulin produces hyperglycemia (high blood glucose level), an indication of diabetes mellitus.

Glucagon is produced in the opposite situation from insulin. When the blood glucose rises, no glucagon is secreted by the pancreas, but when blood glucose is low, glucagon is released. Glucagon makes the liver release stored glucose so that the blood glucose rises. Then the amount of glucagon secreted falls.

When does diabetes mellitus develop?

Diabetes mellitus arises in 2 situations that make the blood glucose rise and finally spill into the urine and make the cells of the body become starved for energy.

when the pancreas produces an inadequate amount of insulin

when the body cells do not respond effectively to the insulin that is produced in normal amounts.

What is diabetes insipidus?

Diabetes insipidus is a condition with a name similar to diabetes mellitus, but it is unrelated. It is due to a pituitary gland disorder and is characterized by extreme thirst and the excretion of large amounts of very dilute urine.

The urine is "insipid" or tasteless, in comparison with the sweet, sugar-filled urine of diabetes mellitus (mellitus = sweet as honey).

When the term "diabetes" alone is used, it refers to diabetes mellitus.

How common is diabetes?

In the U.S., 16 million people have diabetes mellitus, and about half of those do not know they have it. About 160,000 Americans die from diabetes each year.

There is variation in the incidence of diabetes among different ethnic groups. About 5-6% of whites have diabetes; 12-15% of African-Americans have it; 20% of Hispanics have it; and 35% (and up to 65% in some tribes) of Native Americans have it. The disease is very rare in undeveloped countries.

What causes diabetes?

The causes of diabetes are not known. Insulin-dependent diabetes may be more than one disease and may have many causes—hereditary factors, viruses, etc. Noninsulin-dependent diabetes is associated with obesity and with the development of resistance of the body to the action of insulin.

Risk factors for diabetes are:

a family history of diabetes.

sedentary lifestyle

central (truncal) obesity

being a member of a high-risk ethnic population

having delivered a baby > 9 lb. or having had gestational diabetes

age >45.

What are the different types of diabetes mellitus (DM)?

There are 3 common types of diabetes:

1. Type 1 (or type I) diabetes mellitus, formerly called juvenile or brittle or insulin-dependent diabetes (IDDM).
2. Type 2 (or Type II) diabetes mellitus, formerly called noninsulin-dependent or adult-onset diabetes (NIDDM).
3. Gestational diabetes mellitus (GDM).

Type I

An autoimmune disorder - the immune system body attacks and destroys beta cells in the pancreas, so the pancreas makes too little or no insulin.

Usually appears suddenly.

Most common under age 30 and more common in whites than in nonwhites.

Treatment is daily insulin, a planned diet, and regular exercise.

Type 2

Makes up 90 to 95% of diabetes.

The pancreas makes some insulin, sometimes too much, but it is not effective because the cells are resistant to insulin.

Usually gradual in onset.

Occurs most often over age 40-45 and is commonly associated with obesity, especially central obesity.

The treatment is diet, exercise, oral medication, and sometimes insulin.

Gestational diabetes (GDM)

Occurs during pregnancy and usually disappears when the pregnancy ends. A woman who has had gestational diabetes is at increased risk for later developing Type 2 diabetes.

Other types may occur:

Diabetes may result from many other causes, such as other endocrine diseases, drugs, infections, genetic syndromes, etc.

How is diabetes diagnosed?

In 1997, the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus named 3 ways to diagnose diabetes:

1. Symptoms of diabetes (such as polyuria, polydipsia, and unexplained weight loss) plus casual plasma glucose greater than 200 mg/dL (11.1 mmol/L). Casual means any time of day, without regard to meals.
2. Fasting plasma glucose (FPG) greater than 126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 hours.
3. Glucose greater than 200 mg/dL (11.1 mmol/L) after a 75-g glucose load. This is not recommended for routine clinical use.

✓ Any of these is sufficient for diagnosis but should be confirmed by repeat testing on a separate day.

What is impaired glucose tolerance?

Impaired glucose tolerance (IGT) is a category that refers to those with fasting plasma glucose greater than 110 but less than 126. This group is at increased risk for diabetes.

What are the signs and symptoms of early diabetes?

- Type I DM usually presents with symptomatic hyperglycemia or diabetic ketoacidosis (DKA).
- Type II DM frequently is diagnosed on a routine medical examination, but may present with symptomatic hyperglycemia or hyperglycemic hyperosmolar nonketotic coma (NKHHC). Some patients are not diagnosed until they are found to have a late complication of diabetes.

What laboratory tests are often used to monitor DM?

Fasting or random (casual) plasma glucose.

Lipid testing - total cholesterol, HDL and LDL cholesterol, triglycerides.

Serum creatinine.

Urinalysis for glucose, ketones, protein, sediment, urine culture if indicated.

Test for microalbuminuria.

Electrocardiogram.
Hemoglobin A_{1c} (HbA_{1c})

What is hemoglobin A_{1c}?

Hemoglobin A_{1c} (HbA_{1c}) is the part of red blood cells that carries oxygen to the cells and that also binds to glucose. It is also known as glycosylated hemoglobin (GHb). The higher the level of blood sugar, the more sugar attaches to red blood cells and therefore the higher the level of HbA_{1c}.

The hemoglobin A_{1c} test measures the percentage of HbA_{1c} that is linked to glucose. The glucose stays joined to the cell for the duration of the cell's life (about 4 months), so the test can help track the average blood glucose levels over the past 60-90 days and monitor the effectiveness of treatment.

6% is considered normal. The goal of treatment is a level of less than 7%, the level that has been shown to have fewer complications than with a level of 8% or higher. Levels of 9-12% may be seen in diabetics. Levels greater than 8% may indicate a change in treatment is needed.

What is symptomatic hyperglycemia?

Symptomatic hyperglycemia generally occurs when excessive blood sugar occurs and is excreted in the urine (glucosuria). It results in dehydration as the kidneys try to flush out the extra glucose. Findings include:

polyuria (excessive urination, especially at night)

polydipsia (excessive thirst)

polyphagia (excessive hunger)

weight loss

May also be blurred vision, fatigue, nausea, dry mouth, dry or itchy skin, poor wound healing, impotence, or recurrent infections, such as vaginal candidiasis (yeast) and swimmer's ear.

What are the acute complications of diabetes?

Acute complications of diabetes include both hyperglycemia and hypoglycemia. Hyperglycemic diabetic emergencies include **diabetic ketoacidosis (DKA)** and **hyperosmotic non-ketotic coma (NKHHC)**.

What is nonketotic hyperosmolar hyperglycemic coma (NKHHC)?

NKHHC is a serious complication of Type 2 DM resulting from very high blood glucose (600-2400 mg/dL) with dehydration. It often follows an infection, stroke, myocardial infarction, or surgery, often when there is some problem that interferes with adequate oral hydration (fluid intake by mouth). Sometimes it is due to medications.

In one-third of patients, it is the first indication of diabetes. It is often seen in elderly nursing home residents and may be associated with renal insufficiency, congestive heart failure, or recent discontinuation of insulin or oral hypoglycemic agents.

The mortality rate may be up to 50%. Symptoms include polyuria, polydipsia, evidence of severe dehydration, nausea, weakness, lethargy, tachycardia, hypotension, vision problems, confusion, delirium, convulsions, seizures, and ultimately coma (in about 10%). These symptoms may develop over a period of days or weeks.

What is diabetic ketoacidosis (DKA)?

DKA develops gradually when the blood glucose level rises and there is insufficient insulin to deal with it. This may happen because of stress, acute illness, eating too much, not taking enough insulin, etc. It is much more common in Type 1 DM.

The lack of insulin results in starvation of the body's cells, so they lack energy and start to break down fat for fuel. This results in the production of ketones in the blood, a condition called ketoacidosis. This may result in coma if not treated rapidly.

Symptoms may include: fatigue, nausea and vomiting, weight loss, blurred vision, polydipsia, polyuria, abdominal pain, and altered mental status.

Treatment often requires hospitalization and includes fluid and electrolyte replacement and insulin. The mortality rate of DKA is less than 5%.

What is hypoglycemia?

Hypoglycemia is too low blood glucose (below 70). It is the most common complication of the treatment of diabetes. It may occur because of eating too little food after taking diabetes medications, excessive exercise, alcohol intake, or medication. It is more common in those on insulin.

When the blood sugar is 60 or below, it affects the brain, leading to weakness, nervousness, shaking, hunger, drowsiness, sweating, tachycardia, headache, confusion, irritability etc., and finally to seizures or loss of consciousness (insulin shock).

The treatment of hypoglycemia is immediate intake of a source of glucose such as fruit juice, sugar, non-diet soda, or glucose tablets.

What are the chronic complications of diabetes?

The chronic complications of diabetes can affect many different parts of the body—eyes, heart, feet, nervous system, kidneys. On average, complications become evident about 15-20 years after the diagnosis of DM. However, some people never develop complications, and others develop them much earlier, and even have them at the time of diagnosis. About 40% develop complications at some time.

Complications are often divided into several categories.

Microvascular complications (small blood vessel damage)

- retinopathy - eye
- neuropathy - nerves
- nephropathy - kidney

Macrovascular Complications (large blood vessel damage)

- heart problems
- hypertension
- peripheral vascular disease
- stroke

Other Complications

- infections
- impotence
- pregnancy complications
- foot problems
- skin problems

What is diabetic nephropathy?

Diabetic nephropathy is deterioration of the kidneys due to diabetes. It occurs in 30-50% of insulin-dependent diabetics and 10-15% of non insulin-dependent diabetics. There is often a clinical syndrome of albuminuria, hypertension, background retinopathy, and a history of diabetes for more than 10 years.

What is the typical course of diabetic nephropathy?

Diabetic nephropathy is often divided into 5 stages:

I. Silent stage. Diabetic nephropathy may be silent for 10-15 years, although it is damaging the nephrons of the kidney during that time.

II. Microalbuminuria. Persistent proteinuria (protein in the urine), specifically albuminuria (albumin, a type of protein, in the urine), in the range of 30-300 mg/24 h, which is known as microalbuminuria, is the earliest stage of diabetic nephropathy. These trace amounts of albumin leak through the damaged filtering structures of the kidneys. These patients will likely progress to clinical albuminuria.

Normal albumin excretion is 15 to 30 mg/day. Detecting microalbuminuria requires a special test. It is not found on a routine urinalysis because more than 550 mg/day must be excreted for it to show up on routine testing, which is the level called macroalbuminuria or macroproteinuria. Untreated hypertension accelerates renal disease, and increased hypertension occurs in patients with microalbuminuria.

III. Clinical albuminuria or proteinuria. Clinical diabetic nephropathy is said to be present when a patient who has had diabetes for more than five years and has evidence of diabetic retinopathy develops clinically apparent albuminuria (>300 mg per 24 hours) and has no evidence of any other cause of kidney disease. The level can be as much as 2000-4000 mg/day. When albuminuria develops, there is a high likelihood of developing end-stage renal disease within 3 to 20 years.

IV. Renal insufficiency (decreased renal function) - indicated by a rising blood creatinine. About 4 years after the onset of clinical diabetic nephropathy, the serum creatinine level rises to 2 mg/dL or greater. Within an additional 3 years, about half of patients will have developed ESRD.

V. End stage renal disease (ESRD) - usually when creatinine level reaches between 3 and 8. Likely to need kidney transplantation or hemodialysis.

What are some tests for renal disease?

- **creatinine**
- **creatinine clearance test**
- **blood urea nitrogen (BUN)**
- **proteinuria**
- **renal imaging**
- **renal biopsy.**
- **renal angiogram**

What things accelerate diabetic nephropathy?

hypertension

neurogenic bladder

urinary tract infection and obstruction

drugs toxic to the kidney, such as NSAIDS.

What is the treatment of diabetic nephropathy?:

Albuminuria and renal disease may be prevented or delayed by the use of ACE inhibitors, providing tight glucose control, treating hypertension, low protein diet, and control of blood lipids, but treatment will not reverse renal disease.

Hemodialysis or kidney transplant may be needed in late stages. Patients with diabetes tend to start dialysis earlier (at a lower creatinine level) than others because they develop symptoms sooner than non-diabetics.

What are the cardiovascular complications?

Atherosclerosis occurs earlier and is more severe than in the general population, but the reason is unknown. Problems may develop in the legs, with peripheral arterial disease with intermittent claudication, ulcers that don't heal, sometimes progressing to gangrene; heart, with coronary artery disease, cardiomyopathy, or congestive heart failure; and brain, with cerebrovascular accident (stroke).

Arteriosclerotic heart disease: Coronary artery disease is the major cause of death in diabetics. Angina and myocardial infarction may be silent until they result in unexpected left heart failure. (88% of asymptomatic diabetics undergoing coronary angiography as part of screening before kidney transplant had significant coronary disease.) Another cardiac complication is cardiomyopathy without coronary artery disease.

Diabetics have the same risk factors for arteriosclerotic heart disease as the general population—smoking, hypertension, elevated blood lipids, obesity—but diabetes greatly increases the risk. The risk of death following a myocardial infarction is 40-50% in diabetics, and 25 to 30% in non-diabetics. Unlike other complications, good blood glucose control will not prevent the development of heart disease.

Half of all diabetics have hypertension, but it is not ordinarily due to the diabetes—except when it results from diabetic nephropathy.

What is diabetic neuropathy?

Diabetic neuropathy is a group of disturbances that occur frequently in diabetics that can affect many parts of the nervous system. The peripheral nerves go out from the brain and spinal cord to muscles, skin, and internal organs. Peripheral neuropathy may be asymptomatic until a serious complication, such as foot ulcer or cardiac arrhythmia, develops.

The cause of diabetic neuropathy is not known, but may be due to a disturbance of nerve metabolism or ischemia (inadequate blood supply) of the nerves. Risk factors that contribute are increasing age, male sex, increasing height, long

duration of diabetes, poor glucose control, hypertension, alcohol consumption, and smoking.

Examinations for diabetic neuropathy assess muscle strength, deep tendon reflexes, and sense of touch (temperature, pinprick or pressure sensation, vibratory sensation, position sense). Different functions are affected in different individuals, and symptoms may be out of proportion to the findings on examination. Diagnostic criteria are based on some combination of symptoms, focused neurologic examination, nerve conduction studies, and special quantitative sensory tests, but some tests are difficult and time consuming, and not all are ordinarily done.

Treatment consists of strictly controlling blood glucose and treating symptoms.

There are several different classifications of diabetic neuropathy. One groups it into 3 categories:

- Distal symmetrical polyneuropathy.
- Focal neuropathy.
- Autonomic neuropathy.

Distal symmetric polyneuropathy

This is the most common type of diabetic neuropathy. It is primarily sensory. It can affect the feet, legs, hands, and arms. It is characterized by peripheral neuropathy that is usually bilateral and symmetrical. It typically begins insidiously in the toes and progresses up the legs. It then affects the fingertips and later the chest and abdomen. It always starts distally and moves proximally.

About 12% of diabetics have it at the time of diagnosis of diabetes, and almost 60% have it after 25 years of diabetes.

Symptoms vary, but may include

paresthesias - numbness and tingling

hyperesthesias - increased sensitivity—to touch, etc.

hypesthesia (or hypoesthesia) - decreased sensitivity—to touch, etc.

loss of sensation

pain - often burning, may be lancinating or lightning, may be severe and debilitating, often worse at night

dysesthesia - unusual and unpleasant sensation, sometimes extremely painful, after normal stimulation

muscle weakness.

The findings are typically in a stocking-glove distribution. Ankle jerks are usually decreased or absent. The same patient may show both pain and insensitivity to pain.

Complications include:

abnormalities of gait (sensory ataxia - loss of balance and poor muscle coordination due to loss of position sense)

Charcot joints (neuropathic osteo-arthritis), which includes degenerative changes, instability, and possibly fragmentation of bones, particularly in the joints of the feet and ankle.

neuropathic ulcers of the feet. May lead to gangrene and amputation.
injuries and burns - may be unnoticed and become infected.

Mononeuropathy (focal)

Mononeuropathy is less common than polyneuropathy. It involves isolated neuropathy of a single nerve, often with paralysis of the 3rd, 4th, or 6th cranial nerve (eye muscle nerves), with the 3rd being most common, or the 7th cranial nerve, on one side. It can also affect peripheral nerves, causing a sudden wrist or foot drop. Compression neuropathies, such as carpal tunnel syndrome, may also occur. Mononeuropathy may improve spontaneously after weeks to months and is believed to be due to nerve infarction.

Radiculopathy (focal)

Neuropathy of a spinal nerve root may occur, producing pain over the distribution of one or more spinal nerves, usually on the chest wall or abdomen. It also causes sensory loss. Like mononeuropathy, the lesion is usually self-limited.

Autonomic neuropathy

The autonomic nervous system includes sympathetic and parasympathetic nerves that supply heart muscle, smooth muscle (such as the muscle lining walls of arteries and the digestive tract), and glands, all organs that work without our conscious control. Neuropathy of the autonomic nervous system can have broad effects on the cardiovascular, digestive, and genitourinary systems, and on the sweat glands.

Cardiovascular autonomic neuropathy: Among the effects are decreased cardiac sensation so that angina or the pain of myocardial infarction goes unnoticed until congestive heart failure develops.

Another potential cardiovascular problem is orthostatic (postural) hypotension (low blood pressure) with syncope (fainting). Cardiorespiratory arrest and sudden death have been reported.

Urinary tract autonomic neuropathy: Can cause incomplete bladder emptying, with stasis of the urine predisposing to infection of the bladder and kidneys. Incontinence is possible because of decreased sensation or difficulty controlling urination. Chronic catheterization may be necessary.

Digestive tract autonomic neuropathy: May cause bloating, nausea, vomiting, diarrhea (especially at night), weight loss, constipation, difficulty swallowing, or loss of bowel control.

Delayed gastric emptying (sometimes with a dumping syndrome) may occur due to vagus nerve involvement. When severe, it is called gastroparesis (literally, paralysis of the stomach). It may cause nausea, vomiting, early satiety (feeling of fullness in the stomach), bloating, abdominal pain, weight loss. If longstanding, bezoars (masses of hardened food) may develop and obstruct the stomach. It is treated by diabetes treatment, drugs, and at times a feeding tube or parenteral nutrition.

Sweat gland autonomic neuropathy: May cause problems with regulation of body temperature or excess sweating (often at night or during meals).

Sexual dysfunction includes impotence and retrograde ejaculation, usually irreversible. Impotence occurs in 50 to 60% of men with diabetes. It is believed to be due to nerve damage and decreased circulation, but may stem from medications used to treat DM.

What are the foot complications?

Diabetics are at risk for foot problems if they have distal symmetrical polyneuropathy and/or peripheral vascular disease with poor circulation. There are 42 muscles, 26 bones and 29 joints in the foot that may be damaged due to diabetes.

Ulcers are a common foot problem. They may be due to abnormal pressure resulting from neuropathy or poor fitting shoes, combined with a lack of sensitivity to pain. They may be preceded by a callus. Injuries of which the patient is unaware are common. Osteomyelitis and gangrene may follow.

Amputations are 15 times more common in people with diabetes than in those without it. Peripheral neuropathy, peripheral vascular disease, and infection are all contributory causes. Amputation of one lower extremity predisposes to amputation of the other because of increased stress on the opposite leg, resulting in ulcers, infection, etc.

What are the skin complications?

In addition to infections (for example, Candida, dermatophytes, and bacterial infections) and ulcers, there are several specific conditions that may affect diabetics.

- Necrobiosis lipoidica diabetorum - plaque-like yellow to brown lesions over the anterior tibial surfaces of the legs that may ulcerate. It develops over months and may last years. The cause is unknown.
- Diabetic dermopathy ("shin spots") - small plaques with a raised border, also usually over the anterior tibial surfaces that may also ulcerate. Cause is unknown.
- Bullosis diabetorum - blisters spontaneously appearing on the hands or feet that heal in 2-5 weeks, sometimes with scarring and atrophy.
- At the site of insulin injections, fatty tissue may atrophy, or the skin may thicken with an accumulation of fat resembling a lipoma.

What are the eye complications?

The most common eye problems in diabetics are:

Diabetic retinopathy - impairment or loss of vision due to damage to the blood vessels of the retina.

Cataract--clouding or opaqueness of the lens of the eye.

Glaucoma--increased fluid pressure in the eye. Causes loss of visual fields due to optic nerve damage.

How common are diabetic eye complications?

Diabetes is the leading cause of adult blindness. About 2% of people with Type 2 DM suffer total loss of vision.

Who is likely to get diabetic eye complications?

Anyone with diabetes may get eye complications, but they are more likely the longer someone has had diabetes. Almost half of diabetics will develop diabetic retinopathy during their lifetime.

Two early warning signs of retinopathy are microalbuminuria and decreased dark adaptation. Diabetic eye disease is associated with poor control of blood glucose and blood pressure.

What causes eye damage?

This is largely due to blood vessel damage from high blood sugars:

Leakage (hemorrhage): from damage to capillaries.

Blood vessel blockage: partial or total, decreases blood supply.

What is the retina?

The retina is a very thin light-sensitive tissue at the back of the eye. When light enters the eye, the retina changes the light into nerve signals and sends them along the optic nerve to the brain to make vision possible.

What are the types of retinopathy?

The 2 main types of retinopathy are:

background, or simple retinopathy (BDR)

proliferative retinopathy

preproliferative retinopathy is sometimes listed as a 3rd type

What is the course of retinopathy?

Retinopathy affects the small blood vessels of the eye. It begins as background retinopathy (BDR), which is an early stage of damage that can be diagnosed before vision is impaired by an examination of the eyegrounds by ophthalmoscopic examination. The characteristics of background retinopathy are:

- **Microaneurysms**
- **Hemorrhages**
- **Hard Exudates** -

About 80% of people who have had diabetes for over 20 years have some background diabetic retinopathy, but 75-80% of those never develop serious vision problems. However, BDR can progress to macular edema or proliferative retinopathy.

Macular edema can occur if the microaneurysms, hemorrhage, and exudates of BDR occur within the macula, which is the central 5% of the retina most critical to vision. Macular edema may lead to blurred vision, and there may be progressive visual loss and inability to focus clearly. This may occur at any stage of retinopathy.

Some call the following changes preproliferative diabetic retinopathy:

- Intraretinal Microvascular Abnormalities (IRMA):
- Cotton Wool Spots: - white areas in the retina where blood vessels are blocked, and localized areas of nerves have been damaged.

Proliferative retinopathy: occurs when small, fragile, abnormal new blood vessels develop and grow out of control across the eye. This is called neovascularization. It may be associated with retinal detachment or hemorrhage, which can cause blindness.

The new vessels grow out into the vitreous gel (the clear jelly-like substance that fills the middle of the eyeball) and are very prone to bleeding, especially with a

sudden motion or rise in blood pressure. Until they bleed, someone is unlikely to know they have eye problems. Hemorrhage can cause blurred vision or temporary blindness.

If extensive or repeated bleeding occurs, fibrous tissue or scarring can form near the retina. Since the retina is so thin, being made up of only a few layers of cells, scarring can pull or detach the retina away from the back of the eye. Retinal detachment may be noted as wavy lines or a curtain-like effect that appears in one area of vision. It may result in permanent visual impairment.

How is proliferative diabetic retinopathy treated?

- Laser surgery can reduce the risk of visual loss from proliferative diabetic retinopathy by 60%. It is used to seal or shrink the abnormal blood vessels. Can also reduce visual loss from macular edema by 50%.
- Vitrectomy surgery (removal of the clear vitreous gel in the eye and replacement with a salt solution) may restore useful vision when retinopathy is too advanced for laser surgery—e.g., those who have vitreous hemorrhage or scarring with retinal detachment.

How common are cataracts in diabetics?

Diabetics are twice as likely to get a cataract as a person who does not have the disease, and they develop at an earlier age in people with diabetes.

What are the types of cataracts that diabetics get?

Senile cataract - the most common type. Almost exclusively in those > age 60. The underlying damage begins decades earlier. Diabetes raises the risk about 40%.

Vitrectomy - In one study, 63% of eyes that had had a vitrectomy developed cataracts compared to only 4% in the non-vitrectomised eye.

Sugar cataract - mostly young adults with poor control of Type I DM. Can grow rapidly with complete loss of vision in as little as 3 days.

How common is open-angle glaucoma in diabetics?

Open-angle glaucoma is 1.4 to 2 times more common in the diabetic population. The older a person is and the longer a person has had diabetes, the greater the risk of glaucoma. It results from high fluid pressure within the eye. As the pressure increases, it can compress the optic nerve and the blood vessels that nourish the retina and cause a slow loss of peripheral vision and eventual blindness.

Treatment may include medications, laser, or other forms of surgery.

What are some miscellaneous complications?

Vaginal and oral thrush or moniliasis - may be troublesome during periods of high blood glucose and urine spillage of glucose.

Other infections

Hyperglycemia causes the white blood cells of the immune system to function poorly. In addition, all of the body's fluids have higher levels of sugar and nutrients, which make them more inviting for bacteria to grow and multiply. Therefore, with poorly controlled diabetes, there is a higher risk of infection, and often the infections are more serious and harder to cure. Urinary tract and vaginal infections are particularly common.

Four unusual infections appear to have a specific relationship with diabetes.

- **Malignant external otitis**
- **Nasopharyngeal mucormycosis**
- **Emphysematous cholecystitis**
- **Emphysematous pyelonephritis**

Pregnancy Complications of DM

- Gestational diabetes occurs in 4% of women. They have an increased risk of developing diabetes later in life.
- Twice as likely to have a large baby than women without diabetes.
- Cesarean sections are three to four times more likely.

Can complications be prevented?

A large 10-year clinical study (The Diabetes Control and Complications Trial (DCCT)) assessed the effects of intensive therapy on the long-term complications of diabetes. It showed that strict control of blood sugar by intensive management slowed the onset and progression of eye, kidney, and nerve diseases caused by diabetes. Specifically, it showed:

- 76% reduced risk of retinopathy
- 50% reduced risk of clinical nephropathy
- 60% reduced risk of neuropathy

The chief adverse event associated with intensive therapy was a two-to-threefold increase in severe hypoglycemia.

These results do not necessarily apply to type 2 diabetics. In fact, concern has been raised that insulin treatment in type 2 patients may accelerate macrovascular disease.

What role does insulin have in diabetes management?

The pancreas of type 1 DM patients makes no insulin or insufficient insulin, so daily injections of insulin, usually multiple, are necessary. The exact types, amounts, and timing of the insulin injections are individually determined, and food intake must be matched to this.

Some people with type 1 DM require an insulin pump for adequate glucose control. It delivers small amounts of insulin throughout the day, through a needle, usually inserted in the abdominal wall. The amount is tailored to the individual's activities and mealtimes and requires close monitoring of blood glucose. Brittle diabetics are type 1 DM patients who exhibit frequent, rapid swings in glucose levels without apparent cause.

Patients with type 2 DM may also need insulin if they do not respond well to diet, exercise, and oral medications.

Types of insulin include mostly genetically engineered human insulin, porcine (from pigs) insulin, and bovine (from cows) insulin. Insulin is injected subcutaneously (under the skin) with disposable insulin syringes or a multiple-dose insulin injection device or insulin pen.

The duration of insulin is another way it is classified. There are short-acting (rapid-acting), intermediate-acting, and long-acting types, based on the rate of insulin absorption from the injection site.

Rapid-acting insulins

Regular insulin, the only insulin preparation that can be given IV, works for two to four hours.

Lispro insulin works for about 2 hours.

Semilente insulin is a slightly slower rapid-acting insulin.

Intermediate-acting insulin

NPH (neutral protamine Hagedorn).

Lente.

Long-acting

PZI (protamine zinc insulin).

Ultralente.

Mixtures of insulin preparations with different onsets and durations of action are frequently given in a single injection.

In the DCCT study, type I DM patients received an average total dose of about 40 U insulin a day. Type II DM patients are insulin resistant and may require much more insulin. The dose is adjusted to maintain preprandial (before a meal) plasma glucose between 80 and 150 mg/dL (4.44 and 8.33 mmol/L).

What are the most frequent complications of insulin treatment?

- **Hypoglycemia**
- **Weight gain.**
- **Generalized insulin allergy**
- **Insulin resistance**
- **Local fat atrophy or hypertrophy**

What role does diet play in treatment?

All insulin-treated patients require detailed diet management, including a prescription for their total daily caloric intake; guidelines for proportions of carbohydrate, fat, and protein in their diets; and instruction on distributing calories among individual meals and snacks.

In obese type 2 DM patients, the aims of diet management are losing weight and controlling hyperglycemia.

What role do oral medications have in type 2 DM?

If improvement in hyperglycemia is not achieved by a diet to achieve weight reduction in type II DM patients, a drug (an oral hypoglycemic agent) will be used.

Oral hypoglycemic agents are not useful in type I DM patients. They are unable to prevent symptomatic hyperglycemia or DKA in such patients.

The following categories of drugs are now available:

- **Sulfonylureas** - action is to stimulate the pancreatic beta cells to produce more insulin.
- **Biguanides** - action is to slow the ability of the liver to produce and release too much glucose.
- **Glucoside inhibitors** - action is to slow the digestion of some carbohydrates (starches).
- **Thiazolidinediones** - action is to make the muscle cells more sensitive to insulin.
- **Meglitinides** - action is to stimulate the pancreas to produce more insulin.

Sulfonylureas

First-generation sulfonylureas:

Tolbutamide (Orinase)

Acetohexamide (Dymelor)

Tolazamide (Tolinase)

Chloropropamide (Diabinese)

Second-generation sulfonylureas (more recently developed and about 100 times more potent than the first generation drugs):

Glipizide (Glucotrol)

Glyburide (Diabeta, Micronase)

Sulfonylureas have been available since the 1950's but don't work in 40% of type 2 diabetics. Because of progressive resistance after 1-2 years of use, they may have to be used with insulin.

Side effects/complications of sulfonylureas:

hypoglycemia - can be severe and may last or recur for days after treatment is stopped.

weight gain.

upset stomach.

skin rash or itching.

Biguanides

Metformin (Glucophage) - approved 1995.

Promotes weight loss and can improve blood fat and cholesterol levels. It does not result in hypoglycemia.

Side effects/complications of biguanides:

- digestive side effects - may be transient.
- contraindicated with kidney and liver diseases or alcoholism.
- may produce metallic taste in mouth.
- occasionally, weakness, fatigue, dizziness, shortness of breath.

Glucoside inhibitors (or alpha glucoside inhibitors)

Acarbose (Precose) - approved 1995.

Mitagliol (Glyset) - approved 1996.

Do not cause hypoglycemia.

Side effects/complications of glucoside inhibitors:

Gas, bloating, and diarrhea - often transient

Meglitinides

Repaglinide (prandin) - approved 1997.

Side effects/complications of meglitinides
hypoglycemia
weight gain

Thiazolidinediones

Pioglitazone (Actos) - approved 1999.

Rosaglitazone (Avandia) - approved 1999.

Troglitazone (Rezulin) - approved 1997, withdrawn 2000.

In March 2000, the U.S. Food and Drug Administration ordered an immediate recall of troglitazone (Rezulin) after it was linked to 28 deaths and cases of liver failure. It has been withdrawn from the market.

Side effects/complications of thiazolidinediones
symptoms of upper respiratory tract infection
headache
sinusitis
muscle pain
tooth disorder
sore throat.
weight gain
edema
anemia.

What are the benefits of treatment?

- Decreased risk of DKA or NHHHC.
- Less blurred vision and less risk of polyuria, polydipsia, fatigue, weight loss, vaginitis, etc.
- Greatly decreased risk of development or progression of diabetic retinopathy, nephropathy, and neuropathy.
- Improvement in blood lipids.

How is diabetes rated?

10% requires 1 criterion
diet.
20% requires 2 criteria
oral drugs and diet or
insulin and diet.
40% requires 3 criteria
insulin
diet

regulation of activity.

60% requires 5 criteria, one of which provides alternatives

insulin

diet

regulation of activity

(a) episodes of ketoacidosis or hypoglycemic reactions requiring one or two hospitalizations per year or (b) twice a month visits to a diabetic care provider complications that would not be compensable if separately evaluated.

100% requires 5 criteria, 2 of which provide alternatives

more than one daily injection of insulin

restricted diet

regulation of activities

(a) episodes of ketoacidosis or hypoglycemic reactions requiring at least three hospitalizations per year or (b) weekly visits to a diabetic care provider

(a) progressive loss of weight and strength or (b) complications that would be compensable if separately evaluated.

Why are frequency of hospitalizations and visits to a diabetic care provider part of the evaluation criteria?

Frequency of health care visits and contacts and hospitalizations are used as criteria because they are an indicator of diabetic control and the severity of complications.

- * Some patients require hospitalization for initiation or change of therapy.

- * When starting insulin treatment or having a major change in insulin program, a patient may need to be in contact with their health care providers as often as daily.

- * Patients beginning treatment with oral drugs may need to be in contact as often as weekly.

- * Frequent contact may be required if undergoing intensive insulin therapy, are not meeting blood glucose or blood pressure goals, or have evidence of progressive microvascular or macrovascular complications.

What is different about examinations for DM?

Examinations for diabetes are generally more complex and time consuming than the average examination. Sometimes a complication is overlooked or fails to be reported by an examiner in the flood of medical findings.

In some cases, several medical specialty exams may be called for, although a general medical exam should be sufficient in most cases, except for vision and hearing problems, or unusually complex situations.

Failure of a rater, for whatever reason, to address all of the existing complications of diabetes is one of the common rating errors.

How are complications rated?

Complications of compensable degree are rated separately unless used to support a 100% evaluation. Therefore, many diabetic ratings will have to be evaluated under the eye, cardiovascular, genitourinary, musculoskeletal, neurologic, and other systems.

Cardiovascular complications are rated as discussed in the cardiovascular training - coronary artery disease, cardiomyopathy, peripheral atherosclerosis, amputation, etc. Strokes require rating in the neurologic system and sometimes genitourinary, digestive, and respiratory systems.

Diabetic eye diseases are rated as glaucoma, cataract, or retinopathy based on visual acuity, visual field loss, or diplopia, depending on the specific findings.

There is a wide range of possible ratings for **diabetic nephropathy**. It will be rated as renal dysfunction if renal function is affected, but as voiding dysfunction if there is incontinence from autonomic nephropathy. If chronic pyelonephritis is the problem, rating as urinary tract infection may be appropriate. Many may need to be rated as kidney transplant, hemodialysis, or nephrectomy.

Diabetic neuropathy has an even broader range of possible ratings. The effects may not be limited to paresthesias of the extremities that could be rated under 8521, for example, but may involve virtually any body system, since the entire nervous system except the brain may be involved. Therefore, there may be findings of the digestive system, cardiovascular system, genitourinary system, musculoskeletal system, etc., that need rating. Neuropathic joints, for example, may be rated as fracture or arthritis or instability.

Residuals of infections are another source of potentially great variation in rating. They range from osteomyelitis to stroke and cranial nerve paralysis, hearing loss, destruction of sinuses, foot ulcers, or gangrenous gall bladder.

On top of all this, there is the bilateral factor, the amputation rule, pyramiding, unemployability, and SMC to consider, since seldom is there a single issue to consider in diabetics once complications begin.

It's safe to say that if you can rate diabetes well, you can rate almost anything well!

What are some Internet sources of information on diabetes?

<http://www.niddk.nih.gov/health/diabetes/diabetes.htm>

Excellent comprehensive website from NIH.

<http://www.medscape.com/>

Extensive information, fairly technical, has search capability.

<http://www.retinopathy.org/info02.htm>

Clear information on diabetic eye disease.

<http://www.intmed.mcw.edu/gimcme/nephropathy/sld001.htm>

Slide program with notes on nephropathy.

<http://telemedicine.org/dm/dmupdate.htm>

Atlas of skin diseases with color photographs of diabetic skin lesions.

<http://www.nei.nih.gov/publications/retinopathy.htm>

Diabetic eye disease in user-friendly language.

http://pharminfo.com/disease/diabetes/diab_info.html#drugs

Detailed technical information about drug treatment of diabetes.