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Rationale for Prophylactic Surgery in the Diabetic Foot

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The Diabetes Control Activity has established diabetes control programs in 20 states. Each program has investigated the extent and nature of diabetes morbidity within its state by means of a descriptive analysis of selected health status indicators. In a 1983 study by Most and Sinnock, data from six states were pooled to provide a profile of lower extremity amputations (LEA) in diabetic individuals.[1] Results indicated that 45 percent of all LEAs were performed in patients with diabetes. An age-adjusted LEA rate of 59.7 per 10,000 diabetic individuals was computed. Diabetes-related amputation rates increase with age and are higher in males. The overwhelming majority of LEAs were either toe or above the knee (AKA), with few performed on the foot. The relative risk of LEAs for diabetic patients compared with the nondiabetic population was highest in the under-45 age group, although the attributable risk was highest in the older population, 191.5 per 10,000 diabetic individuals.

Overall diabetic persons have a 15-fold higher risk of LEA than nondiabetic individuals.[2] According to Bild et al., the direct cost of an amputation including hospitalization, surgery, and anesthesia is $8,000 to $12,000 per case.[3,4] It is estimated that the annual yearly outpatient costs for diabetes sufferers is $1.7 billion, of which $372 million is expended on 13.4 million visits to physicians.[5] This information, provided by the National Center for Health Statistics, does not show the costs or percentage of the total figure for foot-related conditions. Some have estimated this figure to be as high as 20 percent of hospitalizations for diabetic foot-related conditions.[6] Approximately half the amputations performed in Veterans Affairs hospitals occurred in individuals with d iabetes.[1] The 3-year survival for people with diabetes who have undergone an LEA is only 50 percent.[7] It is further estimated that more than 50 percent of the amputations within the diabetic population could be prevented by reducing risk factors for amputation and improving foot care. Integral to many programs for preventative foot care, prophylactic surgery in the diabetic foot has been advocated for the treatment of forefoot and midfoot deformities that are predisposed to concentrated pressures on localized areas.[8-10] There appear to be no studies to date correlating reduction in the number of LEAs performed on diabetic individuals who have previously undergone prophylactic surgery of the foot specifically to change deforming forces once identified as a risk factor for LEA.

In reviewing literature and statistics related to surgery in the diabetic foot, prophylactic surgery appears to be a small percentage of all diabetic foot surgery.[11-13] In a 5-year review at the University of Chicago Hospital and clinics, prophylactic surgery was only 12.3 percent of all diabetic foot surgery. It was found that most surgery in diabetic patients was ablative in nature and was usually performed to debride infected soft tissue or bone.[14] Their study indicated the rate of complications from prophylactic diabetic foot surgery approached 31.2 percent even with a thorough preoperative workup. Most of these complications occurred when ulcerations had been previously

present for more than 1 year and had been localized to a weight-bearing metatarsal head surface. Deformities that place a diabetic individual at significant risk for ulceration include ingrown toenails, digital contractures, bunion and tailor's bunion, and midfoot protrusion secondary to muscle imbalance or Charcot joint.[6] With nearly all LEAs in diabetic patients being directly or indirectly related to ulceration, it would be prudent from concerns about both health care costs and patient morbidity to identify those diabetic individuals who are at risk for tissue breakdown and consider prophylactic surgical reduction of deformities among the treatment plans. The success rate of prophylactic foot surgery should be able to achieve the success level of surgeries performed on the nondiabetic foot, provided the high-risk patient can be identified and treated appropriately by means of preoperative criteria and analysis before complications occur.[14]

Surgery of the foot in patients with diabetes mellitus should not be considered taboo but it does require specific criteria that must be strictly followed. Careful and comprehensive preoperative assessment and strictly scheduled preoperative management are the hallmarks of successful care of the diabetic foot. A close working relationship with the diabetologist or internist responsible for medical management of the patient is of utmost importance for a successful outcome of any foot pathology correction. This becomes a cardinal rule when discussing the surgical management of the diabetic foot, and one that the competent practitioner will never break.

The podiatric physician and internist must be well versed in perioperative protocols for diabetic patients because of the surgical stresses placed on either insulin-dependent diabetics (IDDM, type I) or non-insulin-dependent diabetic patients (NIDDM, type II). Impaired renal function and macrovascular disease significantly increase the morbidity and mortality among diabetic patients.[6,9] and become an issue when the podiatric physician becomes involved in extensive reconstructive foot surgery, in neuropathic osteoarthropathy (Charcot foot deformity) or when amputation is considered. Urinary tract infections, pneumonia, and wound infections are common in the postoperative period particularly in cases of persistent hyperglycemia. Blood glucose greater than 240 mg/dl can result in impaired fibroblast function, which may then cause incisional dehiscence.

Patients who are undiagnosed for diabetes may be prone to excessive hyperglycemic osmotic diuresis and dehydration following a prolonged foot procedure.[15,16] It therefore becomes essential that all patients be adequately evaluated preoperatively and that all diabetic patients are carefully controlled via an approved perioperative diabetic surgical protocol. The true meaning of the multidisciplinary team approach to patient management is realized in effective management in the care of the diabetic foot.

One of the conditions most often seen and just as commonly mismanaged is diabetic foot ulceration.[18] Two of the more widely published authorities on diabetic foot care have diverging opinions in the issue of foot soaks. One believes that the foot should be well hydrated to maintain the normal skin integrity, stating that the best way to accomplish this is to get water to the keratin layer via soaking in water for 15 to 20 min/day.[18] The other author believes that excessive soaks lead to excessive dryness, fissuring, and cracking of the skin. He states that "foot soaks lead to more complications in diabetes than any other home remedy."[19] This controversy points out the larger differences and often empirical methods employed in managing the diabetic foot. Limited objective research exists that firmly establishes a causal effect of a particular treatment modality. As a result, management of diabetic foot ulcerations is placed in the hands of practitioners who often employ techniques passed down from generations of clinicians whose treatment protocols lack scientific scrutiny. The time has come to investigate the simplest of methods and to firmly establish proven and successful techniques in the management of the diabetic foot ulcer. Recent introduction of wound healing factors has added another dimension to the treatment of the diabetic foot wounds. Careful scrutiny of these newer modalities must be made by the podiatrist as the claims of the manufacturer may be somewhat optimistic. It appears unclear to many who have taken the responsibility of treating the diabetic foot whether the wound healing factors are responsible for the enhanced rates of healing or if the more intensive comprehensive care being used is the determining factor. At this junction it would appear that research into the efficacy of wound healing factors must be

carefully viewed and the results of double-blind, placebo-controlled protocols be scrutinized before final judgment passed on use of such factors in the management of foot ulcerations.

Guidelines for minor foot surgery were reviewed by Gavin.[22] Table 36-1 indicates an approved protocol for both type I (IDDM) and type II (NIDDM). Minor foot surgery is considered that form of surgery requiring only local anesthetic agents. Major cases, those performed under general anesthetic agents, may require continuous insulin infusion that should be planned to begin the night before surgery. Good metabolic control can be maintained via this method in a procedure that may be somewhat prolonged. Most forefoot cases including amputations can be performed under local anesthesia whether or not there is evidence of peripheral neuropathy, and consequently it would be doubtful that insulin infusion would be utilized. More extensive rearfoot cases would be classified as major surgery as these are often performed under general anesthesia.

Table 36-1. Guidelines for Diabetes Control: Minor Surgery and Invasive Diagnostic Procedures

Insulin-Treated Patient	Oral Hypoglycemic Agent (OHA)-Treated Patient
NPO Day of Surgery	
• IDDM: Cover with insulin-glucose infusion. Check fasting blood glucose (FBG) q2h–q4h with bedside meter. • NIDDM: Hold *morning* NPH/Lente. Check FBG and administer regular insulin q4h–q6h as shown below.	• Hold morning OHA • Check FBG pre- and post-surgery. • Give evening OHA. • Insulin *rarely* needed. If necessary, use regular human insulin as shown below.

Blood Glucose (BG) (mg/dl)	Subcutaneous Regular Insulin (U)
<180	0
181–220	5
221–260	6
261–300	8
300	10

• After the procedure, give usual evening dose of insulin if dinner allowed. If patient remains NPO, continue the selected regimen.	
Breakfast on Day of Surgery	
• Give normal morning insulin dose: Check BG q2h-q4h. • Supplement with regular insulin: BG >220, 5 U, etc. • Avoid hypoglycemia.	• Give normal morning OHA. • Check BG pre-and post-surgery. • Given evening OHA.

An understanding of the disease process in diabetes is mandatory to being able to analyze treatment choices and identify preoperative criteria when prophylactic surgery is being considered for the diabetic patient. Vascular disease combined with neuropathy leads to a significant portion of the morbidity we see in the lower extremities of diabetic individuals.[6, 23-25] Progressive symmetrical distal polyneuropathy of motor and sensory nerves causes sensory deficits, which can lead to significant anesthesia so that the skin cannot handle repetitive stress.[15] Consequently skin and underlying tissues may break down and become an attractive environment for infection.

It is believed that during repetitive stress the normal sensate individual is aware of subsequent repeated trauma to the skin and underlying tissues.[26] As a result, gait and stride may be altered as a means of protection. The patient who has lost this protective sensation threshold and continues to traumatize local regions of the foot is predisposed to callus formation, foot ulceration, and possibly amputation.[27] Polyneuropathy begins distally and spreads proximally with initial sensory deficits in a stocking distribution. These patients show varying degrees of diminished touch, pain, vibration, and joint position sense, with depressed reflexes. It is interesting to note that the achilles reflex is often absent in the early stages of diabetes and neuropathy. Motor nerve changes also lead to rapid increases in the formation of digital, forefoot, and midfoot deformities, and in certain circumstances neuropathic osteoarthropathy.[28] Mononeuropathy is believed to be the result of vascular occlusion of an arterial supply to a peripheral nerve.[10] Peroneal palsy is the most common disorder. Clinically the patient may present with a motor, sensory, and reflex impair-

Table 36-2. Signs and Symptoms Associated With the Insensitive Foot

Paresthesias
Hypesthesia
Anesthesia
Nocturnal cramping
Diminished or absent deep tendon reflexes
Diminished or absent vibratory sensation
Diminished or absent temperature or pain sensation
Anhidrosis
Callus formation
Ulceration
Intrinsic muscle atrophy
Digital deformity
Cavus foot deformity or pes valgus deformity
Increased skin temperature
Edema
Change in function (foot-drop)

Table 36-3. Perioperative Risks of Diabetic Patients

Autonomic neuropathy
Difficulty in maintaining stable blood pressure
Postural hypotension
Painless myocardial infarction
Increased sensitivity to drugs
Coronary artery disease (three to four times as frequent in age-matched population)
Nephropathy
Difficulty metabolizing and excreting drugs
Fluid and electrolyte imbalance
Nutritional imbalance

ment in the distribution of a specific peripheral nerve (Table 36-2).

Vascular changes in the diabetic extremity can be the result of three mechanisms: occlusive peripheral vascular disease, autonomic neuropathy, and microvascular insufficiency, most likely caused by basement membrane hypertrophy.[15,29] The development of arterial occlusive diseases is approximately five times more common in diabetics than nondiabetics. Pathologic changes occur within the walls of the small, medium, and large blood vessels of diabetic persons, causing lesions in lower extremity vessels.[30] The diabetic patient often develops arterial occlusion in both large and small vessels, as evidenced by calcification of larger arteries in the tunica media as well as hemorrhagic plaques and calcification in the intima. The process occurs more frequently, at an earlier age, and with more complications for the diabetic patient.[15] Arteriosclerosis in the diabetic person appears to occur more distally and progresses in distal to proximal fashion, resulting in the development of a less effective collateral circulation[14] (Table 36-3).

Local manifestations of autonomic neuropathy in the diabetic foot can be manifest as medial vascular calcification and neuropathic edema.[6,14,15,31] It has been associated with an increased incidence of ulceration and the development of a painful neuropathy. Long-term sympathetic denervation has been shown to cause structural damage to the peripheral arteries.[28] The effects of long-term sympathectomy include smooth muscle atrophy in the vessels, leading to ultimate structural changes in the arterial tree. This increase in blood flow has been implicated as an important factor in the development of Charcot joint and pedal ulceration. Ward et al.[16] postulated that, flow in the small distal vessels is inadequate as a result of faster flow from ateriovenous shunting. Abnormally high blood flow, vasodilation, and arteriovenous shunting that result from sympathetic denervation lead to abnormal venous pooling.[16] The neuropathic edema that develops interferes with the normal mechanism of skin function and predisposes the patient to the development of pedal ulcerations.

Biomechanical processes are significantly altered with diabetes.[15] It has been shown by sonographic examination that diabetic patients who suffer from neuropathy have atrophic plantar fat pads.[17] Brand conducted an experiment that suggested that tissue will most likely break down at those areas of high pressure with repetitive stress.[18] Stokes and coworkers found, in diabetic patients, that peak loads were shifted laterally on the foot and that increasing abnormalities in loading occurred with a corresponding evidence of peripheral neuropathy. Their most striking factor was a reduction of load of the toes. Stokes theorized that lateral shifting and weight-bearing could be caused by weakness of the muscles or loss of coordination from loss of physiologic impulses from the tendon receptors and denervation of the intrinsic muscles. This would result in overpowering of the extrinsic extremity muscles, contributing to significant digital contracture and leading to hammer toes and submetatarsal head lesions.[20,21]

Several authors have demonstrated the importance in the accumulation of dynamic plantar pressure data in the diabetic foot.[32,33] Recent technologic advances

by instruments such as the EMED (Novel), Pedobarograph (Biokenitics), and Tekscan (Physical Support Systems, Inc.) now can provide reproducible vertical force and pressure data invaluable in the comprehensive care of the diabetic patient. Although no 'normal" values or normal feet have been firmly established in the literature, several studies have demonstrated the force and pressure data of diabetic feet and how these differ from nondiabetic subjects.[32,33] A recent chapter by Cavanagh and Ulbrecht[32] concluded, "Circumstantial evidence has shown that elevated plantar pressure is a risk factor for ulceration in the diabetic foot. Elevated plantar pressure, however, even in the presence of sensory neuropathy, has not been proved to cause a plantar ulcer." Despite this we can unequivocally state that peak plantar pressure is one factor in plantar ulcer development and that this pressure must be determined if we hope to be in the position of prospective determination of the likelihood of a neuropathic diabetic patient to develop a foot ulcer. At this point prophylactic intervention via custom orthotic devices, custom footwear, and routine lesion debridement will be irrefutable as to their importance in ulcer prevention. It requires more than technologic sophistication to achieve a well-coordinated plan for perioperative care. Careful integrated planning as well as collaboration by all care-givers will achieve optimal results. Without this effort by the podiatric surgeon, internist, endocrinologist, anesthesiologist, and nursing and other staff members, the result would be only an increase in morbidity and unsatisfactory surgical outcomes.

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Appendix 36-1. University of California, San Francisco, Diabetes Service

PERIOPERATIVE MANAGEMENT OF DIABETES GENERAL GUIDELINES

- □ Admit patient the evening before surgery and schedule early surgery.
- □ Physician, anesthesiologist and surgeon liaison to plan regimen.
- □ Aim to keep blood glucose (BG) in a safe range (120–240 mg/dl).
- □ Bedside monitoring of BG with METER must be available.

DEFINITIONS:
IDDM: Insulin-dependent diabetes mellitus. These patients (pts.) need frequent (q4–6h) insulin to prevent DKA.

NIDDM: Non-insulin-dependent diabetes mellitus. Generally treated with diet ± oral hypoglycemic agent (OHA). However, many (30%) take insulin to achieve optimum control.

MAJOR SURGERY: General anesthesia–Invasion of body cavity—Duration > 2 h – NPO > 24 h.

MINOR SURGERY: Mild Sedation—Short duration (<2 h)—Food OK post-op.

NEED FOR INSULIN DURING MAJOR SURGERY:
All insulin-treated diabetic pts. (IDDM and NIDDMs)
NIDDMs (not on insulin) with average FBG > 180 during elective major and emergency surgery.
Variable: NIDDMs with average FBG < 180 on diet ± OHA. These may not need insulin for short-duration major surgery not invading a body cavity and when allowed food post-op.

INSULIN REGIMENS: INSULIN **DRIP** (Regular [R] Human[Novolin] 25 u in 250 ml N-Saline): This provides excellent flexibility for elective major and emergency surgery in all insulin-treated diabetic pts. Required for CABG, organ transplant (Tx).

INSULIN-GLUCOSE **INFUSION** (Regular [R] Human [Novolin] 7–15 u in 1000 ml of 5% D.W.): Suitable for NIDDM pts. during elective major surgery, except CABG and Organ Tx.

Pre-Op **INTERMEDIATE** INSULIN (NPH 30–50% AM dose): Reasonable for NIDDM but *not* IDDM during elective major surgery. These pts. should be taken to OR early and receive 5% D.W. (100 ml/h) in order to avoid potential hypoglycemia during a protracted NPO period.

INSULIN DOSE: Individualize re type of DM, FBG control, renal status and stress of surgery. Average normal dose: NPO, 0.3 u/Kg/day (dose needs may increase 2–3 fold during severe stress). Food OK—0.6 u/kg/day.

REGIMEN DURING MINOR SURGERY AND INVASIVE DIAGNOSTIC PROCEDURES (**Bedside Meter for BG Test**):

NPO: IDDM: Cover with "Insulin-Glucose Infusion." Check BG q4–6h.
NIDDM: Hold (AM) DM med, Rx Regular (sc) q4–6h for BG > 180.

FOOD OK: Give usual (AM) DM med: OHA or insulin.

Post-surgery, if food OK give (PM) DM med., if NPO continue regimen.

DKA—EMERGENCY SURGERY:

1. Diagnosis: BG > 300 mg/dl, pH < 7.3, HCO_3 < 15, and plasma (urine) positive for ketones.
2. Stabilize patient prior to surgery with rehydration (fluid and electrolytes) and BG correction (BG < 240). This generally requires 3–4 h.
3. Insulin is best delivered by DRIP. Insulin needs are high during the initial phase (0.1 u/kg/h). Once stabilized, the insulin drip can be used for perioperative control as per detailed guidelines.
4. Glucose should be added to maintenance fluids once BG < 240 mg/dl.
5. Check serum potassium q2–4h to determine ongoing needs.

TPN should be considered in pts. **NPO** > 72h:

1. Most diabetic pts. will need insulin cover during TPN.
2. Nondiabetic pts. develop hyperglycemia (10–15%) and need insulin.
3. The safest insulin regimen is to add Regular Human (Novolin) directly to the standard TPN solution (Dextrose 25%, Amino Acids 4.25%).
4. Guidelines for initial insulin dose for diabetes based on pre-TPN BG:

Blood Glucose (mg/dl):	< 120	121–180	181–240	> 240
Regular/Liter TPN:	10 u	15 u	25 u	35 u

5. Supplement with Regular (sc) q4h for BG > 240 mg/dl. Make adjustments to Regular dose in subsequent bottles.
6. If insulin needs exceed 50 u/TPN liter, control BG with an Insulin Drip.

DETAILED GUIDELINES

WRITE INSULIN ORDERS the evening before surgery, and send to IV Additives.

1. The evening before surgery administer usual diabetes medication.

2. Hold morning diabetes medication.
3. Obtain 6 AM STAT FBG. (Lab and Bedside) (split sample).
4. Check BG hourly (bedside meter) perioperatively until patient is stable.
5. Assess potassium needs daily.

INSULIN DRIP: Regular (R) 25 u in 250 ml N-saline (1 u per 10 ml).

A. Begin INSULIN DRIP as per sliding scale (*see* B) for BG > 80.

Use a Microdrip (60 drops/ml), infusion pump. Flush 50 ml through line before connecting "piggyback" to the maintenance IV fluids.

Maintenance fluids must contain glucose (5%) infused at a constant rate.

B. SLIDING SCALE FOR DRIP RATE ADJUSTMENT

Blood Glucose (mg/dl)	Insulin Drip (*IV*) U/H
<80	0.0 ____
81–100	0.5 ____
101–140	1.0 ____
141–180	1.5 ____
181–220	2.0 ____
221–260	2.5 ____
261–300	3.0 ____
301–340	4.0 ____
>340	5.0 ____

BG + 100 gives a reasonable estimate of insulin dose (U/H).
Individualized dose re type of DM, renal status and stress.

C. HYPOGLYCEMIA: BG < 80 but > 60 mg/dl, STOP Insulin Drip. Once BG > 80, restart Insulin Drip. If BG < 60 mg/dl but pt. conscious, bolus with 25 cc of 50% D.W. If comatose, give 50 cc of 50% D.W., call M.D. and recheck BG in 15 minutes.
D. HYPERGLYCEMIA: Persistent BG > 340 mg/dl, despite 5 u/h insulin, call M.D. for reevaluation.
E. If HIGH-DOSE INSULIN: (>5 u/h) is anticipated (e.g., CABG, Organ Tx), use a more concentrated Insulin Drip. A special **DRIP** may also be needed to avoid excess fluids and for pediatrics.

INSULIN-GLUCOSE INFUSION: Base Regular (R) insulin (Novolin) dose on average FBG during the previous week. Begin infusion at 100 ml/h.
A. FBG 120–180 mg/dl: Add 7 u R to 1000 ml of 5% D.W. (0.7 u/h).
FBG 181–240 mg/dl: Add 10 u R to 1000 ml (1.0 u/h)
FBG > 240 mg/dl: Add 15 u R to 1000 ml (1.5 u/h).
FBG < 120 mg/dl: Do not add insulin to infusion for NIDDM.
However, for IDDM add 5 u R to 1000 ml (0.5 u/h).
To avoid hypokalemia add KCl (20 mEq/L) except in renal failure.
B. During post-op period check BG q4h once patient stable. BG < 80, STOP infusion and treat as above in "Insulin Drip," C. Should BG > 240 mg/dl supplement with R (Novolin) sc.
Adjust insulin dose in subsequent infusions as per supplements.

POST-OP TRANSITION FROM NPO TO FOOD INTAKE (Need Lab FBG and 4 PM BG qd):

A. Pre-op **INTERMEDIATE** INSULIN (NPH) Regimen: If post-op food OK—Rx usual pm Meds. If NPO-Rx R (sc) q6h for BG > 180 as per BG sliding scale (bedside meter).

B. Peri-op DRIP/INFUSION Regimen: Continue infusion (IV) until normal feeding established, then plan new regimen. Do not routinely give insulin by IV push. Its biological action is too short-lived (15 min.).

1. NIDDM previously treated with diet ± OHA: Rx pre-op medication if BG < 180. Higher BG may required transient R (sc) q6h.

2. Insulin-treated Diabetic patients: Use pt's usual insulin regimen or develop a new **BASIC DOSE** regimen:

The dose selected should be 60–80% of the previous day's total insulin dose. Needs may be higher during persistent stress (infection, pain, steroids, etc.) or high food intake (TPN, etc.).

The selected BASIC DOSE may be given as pre-meal [Breakfast (25%), Lunch (25%), Dinner (25%)] Regular (sc) and NPH at Bedtime (25%).

Use BG sliding scale to ADJUST the **PREMEAL BASIC DOSE.**

BG PREMEAL (*Bedside Meter*)		**BASIC DOSE** *Regular Insulin*
<80 mg/dl		4 units less
81–120 mg/dl		3 units less
121–180 mg/dl	.....GIVE.....	BASIC DOSE
181–240 mg/dl		2 units more
241–300 mg/dl		3 units more
>300		4 units more

Aim to keep BG in safe range (120–240).

The **BASIC DOSE** should be regularly modified (1–2 days) according to the sliding scale needs.

Additional doses of R may be needed at other times, e.g., 10 PM, 2 AM.

C. Prior to discharge establish the most suitable insulin regimen.

SUGGESTED READINGS

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