2009 IHS Combined Councils Meeting

Treatment of Diabetes in the Indian Health System

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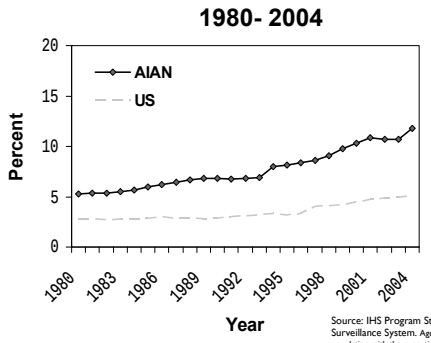
Prevalence of Diagnosed Diabetes

AI/ANs compared to U.S. population



Source: IHS Program Statistics and National Diabetes Surveillance System. Age-adjusted to the 2000 US standard population with the exception of 1981–1993 data for AIAN, which was age-adjusted to the 1980 US standard population.

Treatment of Diabetes in the Indian Health System

- **Issues & controversies in diabetes - 2008**
- **Diabetes treatment patterns and costs in IHS**
- **Treat the defect, not the number**
- **Cherokee treatment algorithms**

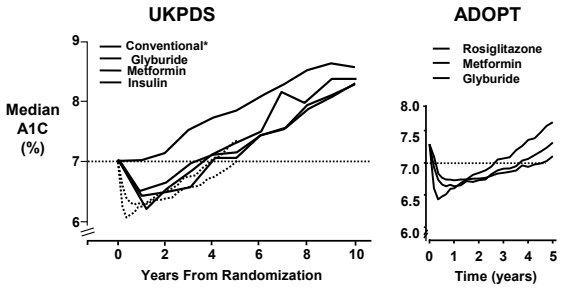
Issues & Controversies in Diabetes - 2008

- **Legacy effect in diabetes**
- **Glycemic control & CVD: ACCORD, ADVANCE and the VA Diabetes Trial**
- **HbA1c for screening & diagnosis of diabetes?**
- **Lessons from bariatric surgery in pts with T2DM**
- **Post-prandial glucose in managing diabetes**
- **Bone fractures and TZDs**
- **GDM: New guidelines coming; Rx with oral agents?**
- **Estimated average glucose (eAG); Pre-diabetes**

Diabetes Research Findings from Last 20 yrs

- Type 2 diabetes can be prevented or delayed
  - Diabetes Prevention Program (DPP) Study
  - Finnish Diabetes Prevention Study
  - Da Qing Prevention Trial
- Tight control can delay the complications of diabetes (DCCT, UKPDS)
- Specific treatments can reduce the risk of complications of diabetes

Over Time . . . Glycemic Control Deteriorates



*Diet initially, then sulfonylureas, insulin, and/or metformin if FPG >15 mmol/L.

UKPDS Group. *Lancet*. 1998:352:854-865; Kahn SE, et al. *New Engl J Med*. 2006;355:2427-2443.

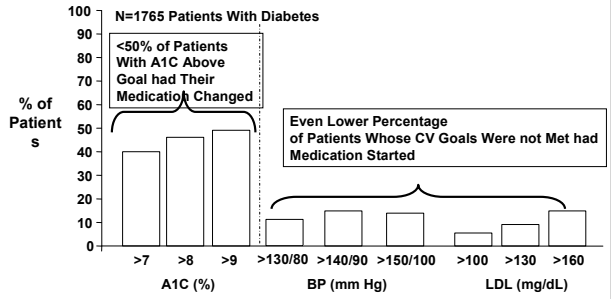
Prevention/Treatment of Diabetes and its Complications: Evidence – based Medicine

Retinopathy	Nephropathy	Neuropathy	CVD	Amputations
⇓	⇓	⇓	⇓	⇓
BP	BP	HbA1c	HbA1c	Foot Exams
⇓	⇓	⇓	⇓	⇓
HbA1c	HbA1c	⇓	BP	Revasc
⇓	⇓	⇓	⇓	⇓
Laser	Dialysis	⇓	Lipids	⇓
⇓	⇓	⇓	⇓	⇓
Vitrectomy	Transplantation	⇓	Revascularization	
⇓	⇓	⇓	⇓	⇓
29 -76%	**39-84%**	**25-60%**	**10-58%**	**10-70%**

Reductions in Risk

Room for Improvement in Therapy Implementation and Change

Rates of Medical Regimen Change in 30 US Academic Medical Centers



*P<.05 for A1C and LDL (P values are derived from Mantel-Haenszel test for trend).

Grant RW, et al. *Diabetes Care*. 2005;28:337-442.

Glycemic Goals of Therapy

Goal	ADA	ACE
Premeal plasma glucose (mg/dl)	70-130	<110
2-h postprandial plasma glucose	(<180)*	<140
HbA1c	<7%†	<6.5%

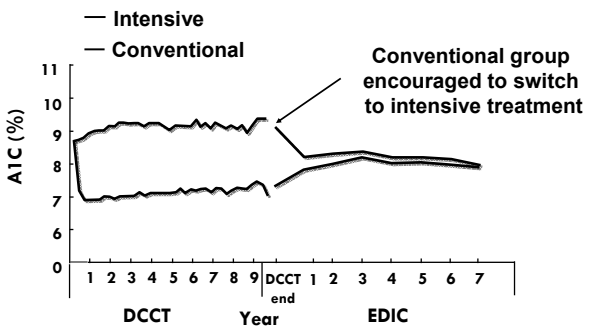
*If A1C remains above the desired target, postprandial levels, usually measured 90–120 min after a meal, may be checked. They should be <180 mg/dl to achieve A1C levels in the target range.

†An A1C of ≥7% should serve as a call to action to initiate or change therapy with the goal of achieving an A1C level as close to the nondiabetic range as possible or, at a minimum, decreasing the A1C to 7%.

Diabetes Care 30: S4-41, 2007 (http://care.diabetesjournals.org/cgi/reprint/30/suppl_1/S4)
http://www.aace.com/pub/press/releases/diabetesconsensuswhitepaper.php

Legacy Effect

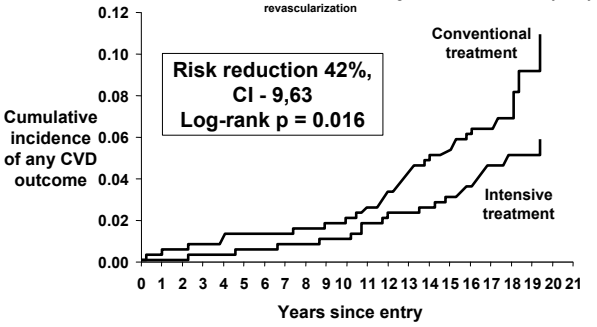
A1C During DCCT and Follow-Up



Adapted from: *N Engl J Med* 329:977–86,1993; EDIC: *JAMA* 287: 2563–9;2002

Cumulative Incidence of the First of Any Predefined CVD Outcomes

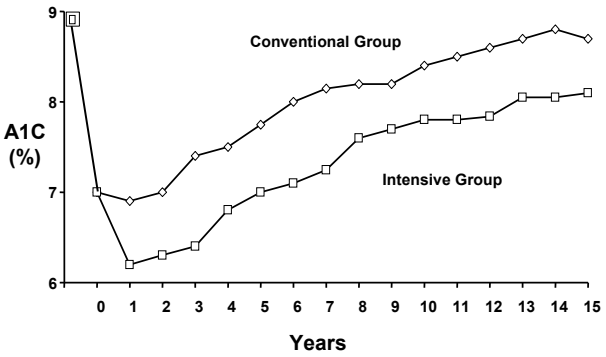
nonfatal MI, stoke, death from cardiovascular disease, confirmed angina, or the need for coronary-artery revascularization



DCCT/EDIC Study Research Group, *N Engl J Med* 2005; 353:2643-53.

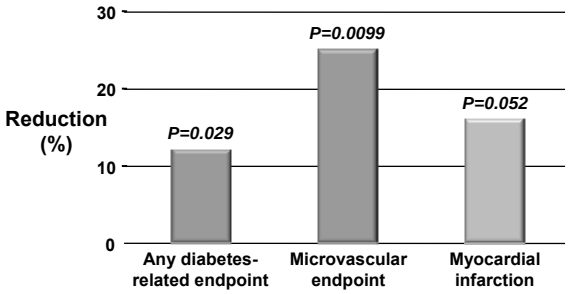
Fatal MI, CVA, or CVD death ↓57%

UKPDS: A1C



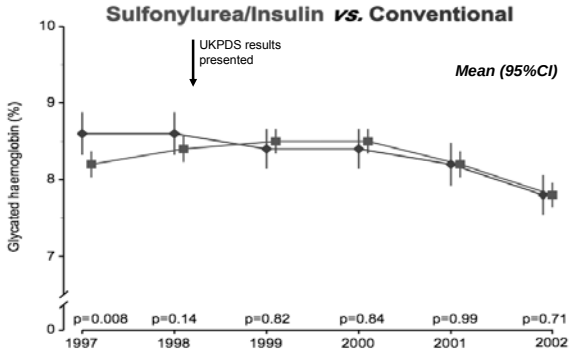
UKPDS Group *Lancet* 352:837–53;1998

UKPDS: Reduction in Complications in Intensive Group



UKPDS Group *Lancet* 352:837–53;1998

UKPDS: Post-Trial Changes in A1C



Holman RR, et al. *New England Journal of Medicine* 2008; 359:1577-1589

UKPDS: "Legacy Effect" of Glucose Therapy

After median 8.8 years post-trial follow-up

Aggregate Endpoint		1997	2007
Any diabetes related endpoint	*RRR:*	12%	9%
	P:	0.029	0.040
Microvascular disease	*RRR:*	25%	24%
	P:	0.009	0.001
Myocardial infarction	*RRR:*	16%	15%
	P:	0.052	0.014
All-cause mortality	*RRR:*	6%	13%
	P:	0.44	0.007

RRR = Relative Risk Reduction P = Log Rank

Holman RR, et al. *New England Journal of Medicine* 2008; 359:1577-1589

UKPDS: "Legacy Effect" of Metformin Therapy

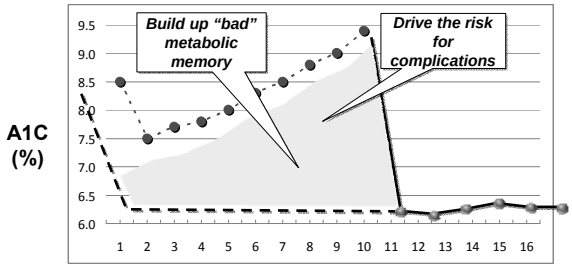
After median 8.8 years post-trial follow-up

Aggregate Endpoint		1997	2007
Any diabetes related endpoint	*RRR:*	32%	21%
	P:	0.0023	0.013
Microvascular disease	*RRR:*	29%	16%
	P:	0.19	0.31
Myocardial infarction	*RRR:*	39%	33%
	P:	0.010	0.005
All-cause mortality	*RRR:*	36%	27%
	P:	0.011	0.002

RRR = Relative Risk Reduction P = Log Rank

Holman RR, et al. *New England Journal of Medicine* 2008; 359:1577-1589

The "Natural History" of Type 2 Diabetes



TIME (yrs since diagnosis)

Glycemia and CVD Risk

How low should we go?

Recent Glycemia & CVD Studies

Characteristic	ACCORD	ADVANCE	VADT
N	10,251	11,140	1,791
Mean age	62	66	60
Hx CVD	35%	32%	40%
BMI	32.2	28	31.3
BL mean A1C	8.3%	7.5%	9.4%
BL meds	34% Ins/1.4 OAD	1% Ins/1.5 OAD	51% Ins/??
Statin use	62% → 88%	28% → 47%	58% → 84%
Antiplatelet use	54.5% → 75.5%	48% → 62%	76% → 93%
Study duration	3.4	5.0	6.0
Target A1C	<6% vs 7-8%	<6.5% vs "Usual"	<6% vs 8-9%
Achieved mean A1C	6.5% and 7.5%	6.3% and 7.0%	6.9% and 8.4%
LDL at end	91	102	75
BP at end	127 / 67	136 / 74	127 / 69
Smokers at end	9.9%	8%	17%

Skyler J, et al. *Diabetes Care*. 2009; 32:187-92.

Effect of Intensive Glucose Lowering on Macrovascular Complications in Type 2 Diabetes

	VADT	ACCORD	ADVANCE
Primary outcome	Non-fatal MI Non-fatal stroke CVD death Hospitalization for CHF Revascularization	Non-fatal MI Non-fatal stroke CVD death	Non-fatal MI Non-fatal stroke CVD death
Hazard Ratio for primary outcome (95% CI)	0.87 (0.73 – 1.04)	0.90 (0.78 – 1.04)	0.94 (0.84 – 1.06)
Hazard Ratio for mortality (95% CI)	1.07 (0.80 – 1.42)	1.22 (1.01 – 1.46)*	0.93 (0.83 – 1.06)

*P=0.04

Skyler J, et al. *Diabetes Care*. 2009; 32:187-92.

ADA – AHA – ACC Revised Glycemic Control Recommendations

- Lowering A1C to below or around 7% has been shown to reduce microvascular and neuropathic complications of type 1 and type 2 diabetes. Therefore, for microvascular disease prevention, the A1C goal for non-pregnant adults in general is <7%. (A)

ADA/AHA/ACC. *Diabetes Care*. 2009; 32:187-92.

ADA – AHA – ACC Revised Glycemic Control Recommendations

- In type 1 and type 2 diabetes, randomized controlled trials of intensive vs. standard glycemic control have not shown a significant reduction in CVD outcomes during the randomized portion of the trials. Long-term follow-up of the DCCT and UKPDS cohorts suggests that treatment to A1C targets below or around 7% in the years soon after the diagnosis of diabetes is associated with long-term reduction in risk of macrovascular disease. Until more evidence becomes available, the general goal of <7% appears reasonable for many adults for macrovascular risk reduction. (B)

ADA/AHA/ACC. *Diabetes Care*. 2009; 32:187-92.

ADA – AHA – ACC Revised Glycemic Control Recommendations

Subgroup analyses of clinical trials such as the DCCT and UKPDS, and the microvascular evidence from the ADVANCE trial, suggest a small but incremental benefit in microvascular outcomes with A1C values closer to normal. Therefore, for selected individual patients, providers might reasonably suggest even lower A1C goals than the general goal of <7%, if this can be achieved without significant hypoglycemia or other adverse effects of treatment. Such patients might include those with short duration of diabetes, long life expectancy, and no significant cardiovascular disease. (B)

ADA/AHA/ACC. *Diabetes Care*. 2009; 32:187-92.

ADA – AHA – ACC
Revised Glycemic Control Recommendations

- Conversely, less stringent A1C goals than the general goal of <7% may be appropriate for patients with a history of severe hypoglycemia, limited life expectancy, advanced microvascular or macrovascular complications, extensive co-morbid conditions, and those with longstanding diabetes in whom the general goal is difficult to attain despite diabetes self-management education, appropriate glucose monitoring, and effective doses of multiple glucose lowering agents including insulin. (C)

ADA/AHA/ACC. *Diabetes Care*. 2009; 32:187-92.

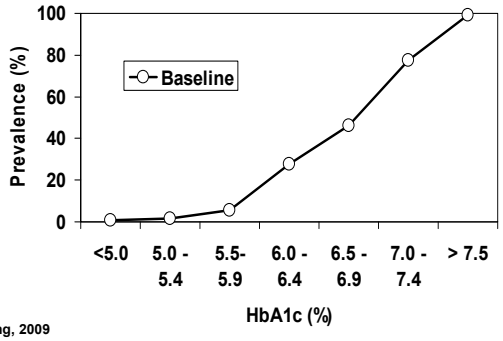
Should A1c be used to screen for and diagnose diabetes?

Practical Issues

- OGTT not routinely performed in primary care
- 2-hr glucose poorly reproducible
- Fasting glucose requires fasting
- Sample processing - glycolysis

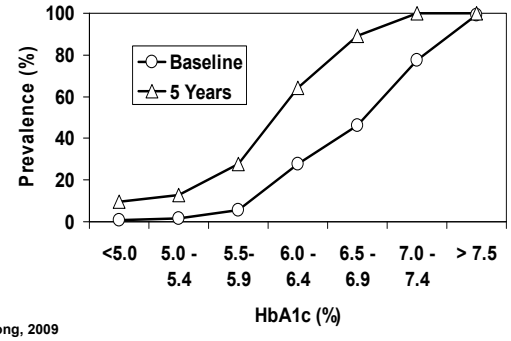
► Is there a simpler, more reliable way to screen or diagnose diabetes?

Prevalence of Diabetes (OGTT criteria) in 2435 Screened Pima Indian Adults



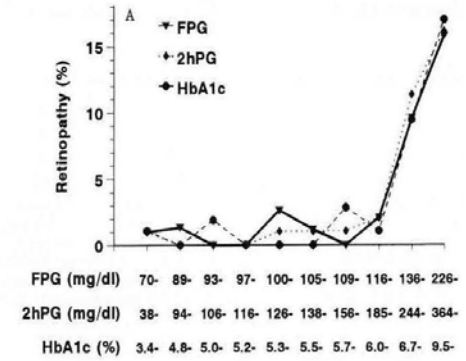
Q. Gong, 2009

Prevalence & 5-yr Predicted Prevalence of Diabetes (OGTT criteria) in 2435 Screened Pima Indian Adults



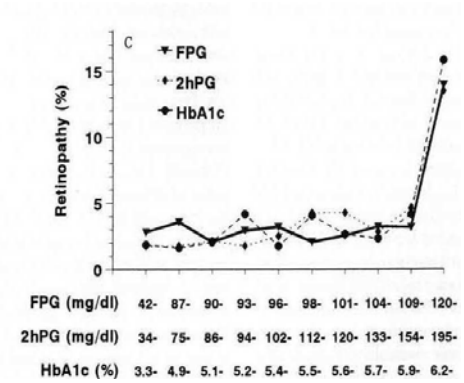
Q. Gong, 2009

ADA Expert Committee on Diagnosis, 1997 Relation of FPG, 2hPG, HbA1c to Retinopathy: Pima Indians



ADA Expert Committee, 1997

ADA Expert Committee on Diagnosis, 1997 Relation of FPG, 2hPG, HbA1c to Retinopathy: NHANES III



ADA Expert Committee, 1997

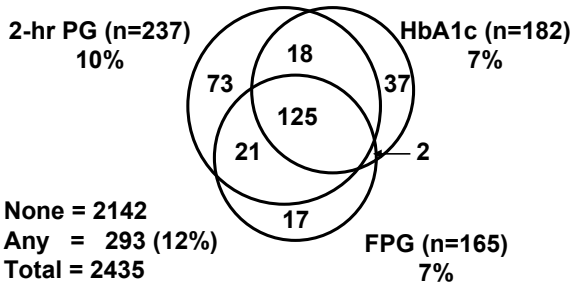
ADA 1997 Criteria Based on These and Other Similar Data

- FPG, 2-hr PG, HbA1c have similar relationships to complications
- New criterion: FPG ≥ 126 mg/dl (previously ≥ 140 mg/dl)
  - Simpler and cheaper than OGTT
  - Widespread use should lead to more cases diagnosed
- HbA1c not sufficiently standardized

HbA1c Advantages Compared with FPG, OGTT

- The HbA1c assay is standardized and aligned to DCCT/UKPDS
- Better index of overall glycemic exposure
- Equivalent in predicting risk for long-term complications
- Predicts CVD
- Substantially less laboratory variability
- Substantially less pre-analytic instability
- No need for fasting or timed samples
- Unaffected by acute (e.g. stress or illness-related) perturbations in glucose levels
- Used to guide management and adjust therapy

Numbers of people with HbA1c ≥ 6.5%, FPG ≥ 126 mg/dl, or 2hPG ≥ 200 mg/dl. 2435 adult Pima Indians without previous dx



If we use HbA1c in place of FPG or OGTT, do we lose the ability to identify people at high risk for diabetes or CVD?

Lessons from Bariatric Surgery in pts with Type 2 DM

Role of the Gut in Glucose Regulation Lessons from Bariatric Surgery

- Weight loss after gastric bypass not simply mechanical
- Decreased appetite and changes in food preferences
- Effects on energy expenditure
  - Increased oxygen consumption and body temperature in rodents
  - Pair feeding suggests half of weight loss due to increased diet induced thermogenesis and half to reduced appetite (not malabsorption)
  - In man EE unchanged despite large weight loss
- Improved glucose homeostasis prior to weight loss

Potential Mechanisms of Bypass

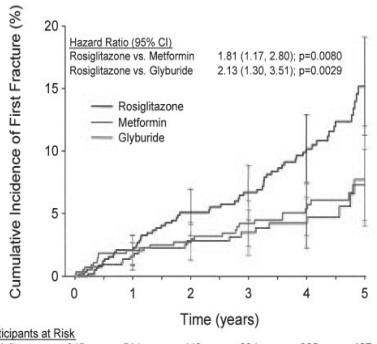
- Altered gut peptides
  - Foregut (Ghrelin)
  - Hindgut (PYY, GLP1, GIP, etc)
- Multiple components to surgery
  - Gastric exclusion and/or resection
  - Duodenal exclusion
  - Accelerated nutrient delivery to jejunum
  - Partial vagotomy
  - Altered GI flora

Is it important to specifically target post-prandial glucose?

Recommendations re Post-prandial Glucose

- **Focus on the pre-prandial glucose (target: 70-130 mg/dl)**
- **If pre-prandial target reached and A1c still >7.0%, <u>then</u> focus on post-prandial glucose (target: < 180 mg/dl)**
- **If pre-prandial goals are reached and A1c ≤ 7.0%, no added benefit to lowering post-prandial glucose.**

Bone Fractures in Women Treated With Glitazones



Participants at Risk						
Rosiglitazone	645	514	448	394	325	127
Metformin	590	464	414	366	293	117
Glibenclamide	605	435	361	279	209	67

Kahn SE, et al. *Diabetes Care.* 2008;31:845-851.

Recommendations re TZDs & Bone Fx

- **Avoid TZDs in those at high risk for bone fracture or that have osteoporosis**
- **If using TZDs, recommend Ca^{++} , Vit D and regular exercise**
- **Anti-resorptives have been effective in glucocorticoid osteoporosis, so…**

Gestational diabetes

New Diagnostic Cutpoints?
New treatment Strategies?

How A1c relates to estimated Average Glucose

Estimated average glucose (eAG):

A1C of 6%	= eAG of 126 mg/dl
A1C of 6.5%	= eAG of 140
A1C of 7%	= eAG of 154
A1C of 7.5%	= eAG of 169
A1C of 8%	= eAG of 183
A1C of 8.5%	= eAG of 197
A1C of 9%	= eAG of 212
A1C of 9.5%	= eAG of 226
A1C of 10%	= eAG of 240

** ADA online calculator at www.diabetes.org/AG

Translating the hemoglobin A1c assay into estimated average glucose values. Nathan D, Kuenen J, Borg R, Zheng H, Schoenfeld D, and Heine RJ, for the A1c-Derived Average Glucose (ADAG) Study Group. Diabetes Care 2008

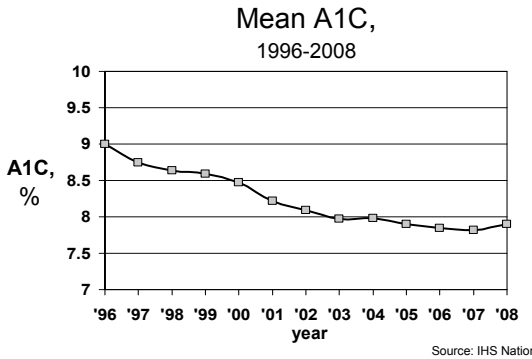
Treatment of Diabetes in the Indian Health System

- **Issues & controversies in diabetes - 2008**
- **Diabetes treatment patterns and costs in IHS**
- **Treat the defect, not the number**
- **Cherokee treatment algorithms**

IHS Annual Diabetes Care & Outcomes Audit

- Since 1986
- Measures based on IHS Standards of Care for Diabetes
- In 2008:
  - 63,840 charts representing care to 127,204 people
  - 320 facilities (32 Urban)
- Benchmark comparisons within & outside system
- Track trends over time
- Evaluate performance on the local, regional and national levels

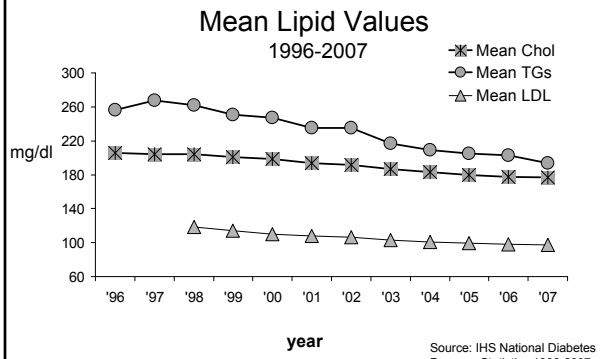
IHS Diabetes Care & Outcomes Audit



Source: IHS National Diabetes Program Statistics 1996-2008

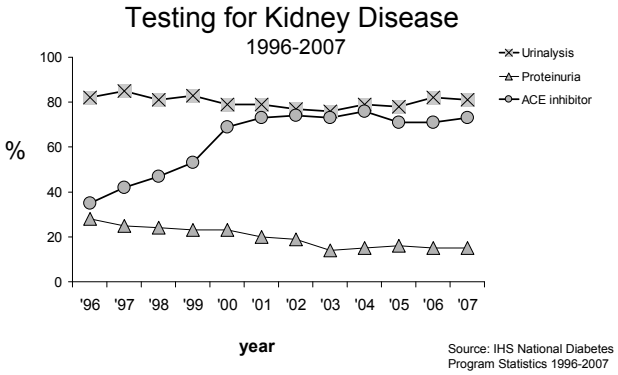
*p<0.0001 comparing mean A1C levels in FY96 and FY08

IHS Diabetes Care & Outcomes Audit



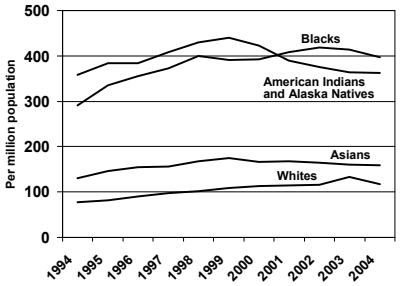
Source: IHS National Diabetes Program Statistics 1996-2007

IHS Diabetes Care & Outcomes Audit



Source: IHS National Diabetes Program Statistics 1996-2007

Age-adjusted* rate of persons initiating therapy for end-stage renal disease with diabetes as the primary diagnosis, by race, United States, 1994–2004



*Based on the 2000 US population

Source: CDC. Racial Differences in Trends of End-Stage Renal Disease, by Primary Diagnosis --- US, 1994--2004. MMWR March 23, 2007 / 56(11):253-256

Costs of Diabetes

- **Diabetes cost U.S. $174 billion in 2007 (direct & indirect costs)**
  - ✓ **$116 B medical expenditures**
  - ✓ **$58 B reduced productivity**
- **Care for people with diagnosed diabetes is ~ 2.3 X more expensive than for those without diabetes ($11,744 vs $5,095)**
- **~ $1 in $10 health care dollars spent in the U.S. is attributed to diabetes**

ADA Position Statement: Economic Costs of diabetes in the US; , Diabetes Care 31, 2008

Expenditures for Persons with and without Diabetes Comparing Spending within IHS to a US Estimate

Ratio of expenditures for persons with diabetes as compared to persons without diabetes

Within IHS: $7,003 / $2,205 = 3.2

US Estimate*: $9,789 / $4,096 = 2.4

Medical service expenditures include those for hospital, office, and home-based services and pharmaceuticals. They exclude those for nursing home and hospice services. Estimates are adjusted for age differences and expressed in 2005 dollars. *US Estimate: ADA 2008.

Wilson C & O'Connell J. Treatment Costs of Diabetes within the Indian Health Service. SDPI 2007

Total Costs

Adults with diabetes accounted for one third of costs for all adults in this population in a one year period.

	Percent of all adults	Total Costs	
		Dollars	Percent
Persons with diabetes	10.6%	$23,713,428	33.5%
Persons without diabetes	89.4%	$47,087,931	66.5%
Total	100.0%	$70,801,359	100.0%

Wilson C & O'Connell J. Treatment Costs of Diabetes within the Indian Health Service. SDPI 2007

Total Costs among Persons with Diabetes by Cardiovascular Disease Status

Total health expenditures per person per year for persons with diabetes and CVD were 2 times higher than expenditures for persons with diabetes but not CVD.

Type of Utilization Rate	Diabetes with CVD Costs per person	Diabetes without CVD Costs per person	Ratio with CVD / without CVD
Unadjusted utilization rates	$12,693	$5,917	2.1
Adjusted utilization rates*	$12,693	$6,049	2.1

* Utilization rates were adjusted for differences in the age distribution of the two populations.

Wilson C & O'Connell J. Treatment Costs of Diabetes within the Indian Health Service. SDPI 2007

Costs of Diabetes

- Every percentage point drop in A1C (e.g. from 8% to 7%) reduces the risk of microvascular complications (eye, kidney, & nerve diseases) by 40%
- Expenses are significantly higher for each percentage point increase above an A1C of 7%.
- Costs are even higher if the person also has heart disease and high blood pressure.

CDC National Diabetes Fact Sheet, 2005

Diabetes Treatment Cost Control Flow Chart

Levels of Hemoglobin A1c (%) Being Compared **	Greater Per-Person Treatment Cost Associated with a One Percentage Point Higher Hemoglobin A1c Value
10% to 9%	$1,200 - $4,100
9% to 8%	$900 - $3,100
8% to 7%	$600 - $2,200
7% to 6%	$400 - $1,500
** Less than 7% is the recommended hemoglobin A1c value.	

Gilmer TP, O'Connor P, Manning WG, Rush WA. The cost to health plans of poor glycemic control. Diabetes Care 1997;20(12):1847–53.

Costs of Diabetes: Treatment vs Prevention

- **The DPP study showed that a lifestyle-change program could *delay the onset* of diabetes by an average of 11 years and *reduce the risk* of developing diabetes by 58%, when compared with no intervention.**
  - ✓ **Cost of *preventing diabetes* in one person with lifestyle intervention: $2,780 over 3 years**
  - ✓ **Cost of *treating one person with diabetes*: $11,744 per year**

"The Cost-Effectiveness of Lifestyle Modification or Metformin in Preventing type 2 Diabetes in Adults with Impaired Glucose Tolerance," Annals of Internal Medicine, Vol. 142, No. 5, March 2005, pp. 323-332.

Median A1c of AI/AN Patients with Diabetes Duration < 5yrs on Specific Treatment Regimens

Treatment		N (%)	Median A1c %	95% CI
Diet and Exercise Alone		3412 (25%)	6.3	6.2-6.3
Oral Monotherapy		4976 (36%)	6.5	6.4-6.5
	Metformin	3394	6.4	6.4-6.5
	Sulfonylurea	1010	6.8	6.8-6.9
	TZD	531	6.2	6.2 -6.3
Dual Combination Therapy		2936 (21%)	7.2	7.1-7.2
	Sulfonylurea + Met	1631	7.4	7.3- 7.5
	TZD + Metformin	885	6.7	6.6- 6.9
	Sulfonylurea + TZD	332	7.3	7.1-7.5
Triple Oral Therapy		691 (5%)	7.7	7.5-7.9

Treatment of Diabetes in the Indian Health System

- **Issues & controversies in diabetes - 2008**
- **Diabetes treatment patterns and costs in IHS**
- **Treat the defect, not the number**
- **Cherokee treatment algorithms**

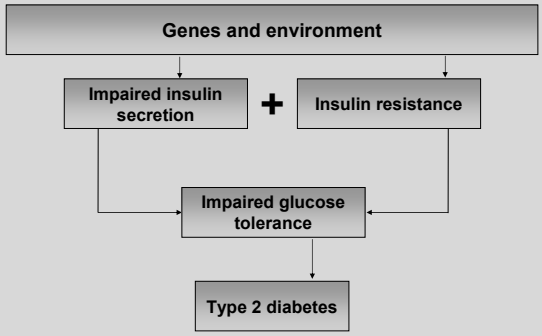
Impaired Glucose Tolerance, Pre-diabetes

- **Individuals with IGT:**
  - **are maximally / near maximally insulin resistant**
  - **have lost ~80% of beta cell function**
  - **have an incidence of retinopathy ~10%**
- **"Pre-diabetes:" is this actually Type 2 DM?**
- **Intervene at causes of beta cell failure**

Etiology of Beta Cell Failure

- ↑ age
- genetics
- insulin resistance
- ↑ FFA & lipotoxicity
- glucotoxicity
- abnormalities in incretin effect
- ? amyloid deposition in pancreas

Etiology of Type 2 Diabetes
Impaired Insulin Secretion and Insulin Resistance



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Pathophysiology of Diabetes

- Impaired insulin secretion
- Decreased glucose uptake (insulin resistance)
- Increased basal hepatic glucose production
- Increased lipolysis (↑ FFA & lipotoxicity)
- Incretin effects (GLP-1 & GIP)
- Islet alpha cell glucagon secretion (hyperglucagonemia)
- Kidney resorbs glucose in hyperglycemia
- Brain neurotransmitter dysfunction (impaired appetite regulation & altered hypothalamic function)

Recommendations for Treatment of Type 2 Diabetes

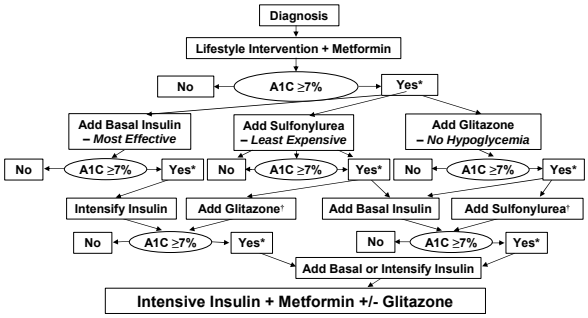
- T2DM will require multiple drugs in combination to treat multiple pathogenic disturbances
- Treatment should be based on known pathogenic abnormalities, not simply the A1c
- Treatment must be started early in the natural history of T2DM if progressive beta cell failure is to be prevented

Antihyperglycemic Agents in Type 2 Diabetes

Class	A1C Reduction	Hypo-glycemia	Weight Change	CVD Risk Factors	Dosing (times/day)	Diabetes Comorbidity Contraindications
Metformin	1.5	No	Neutral	Minimal	2	Kidney, liver
Insulin, Long-acting	1.5 - 2.5	Yes	Gain	TG	1, Injected	None
Insulin, Rapid-acting	1.5 - 2.5	Yes	Gain	TG	1-4, Injected	None
Sulfonylureas	1.5	Yes	Gain	None	1	Essentially none
Thiazolidinediones	0.5 - 1.4	No	Gain	Variable	1	CHF, liver
Repaglinide	1 - 1.5	Yes	Gain	None	3	Essentially none
Nateglinide	0.5 - 0.8	Rare	Gain	None	3	Essentially none
Alpha-glucosidase Inhibitors	0.5 - 0.8	No	Neutral	Minimal	3	Essentially none
Amylin-mimetics (pramlintide)	0.5 - 1.0	No	Loss	With weight loss	3, Injected	None
Incretin Agonists (exenatide)	0.5 - 1.0	No	Loss	With weight loss	2, Injected	Kidney
DPP-IV Inhibitor (sitagliptin)	0.6 - 0.8	No	Neutral	None	1	None
Bile acid sequestrant (colesevelam)	0.5	No	Neutral	LDL	1-2	Severe hypertriglyceridemia

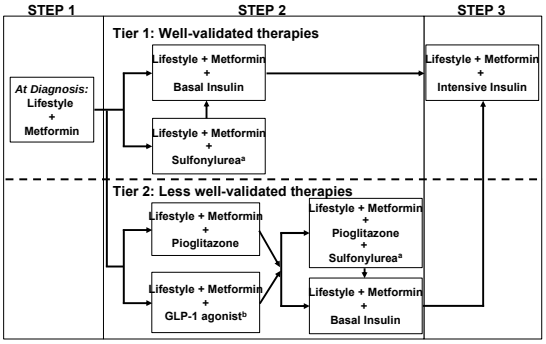
Adapted from: Nathan DM, et al. *Diabetes Care*. 2007;30:753-759; Nathan DM, et al. *Diabetes Care*. 2006; 29:1963-1972; Nathan DM, et al. *Diabetes Care*. 2008;31:173-175. ADA. *Diabetes Care*. 2008;31:S12-S54. WelChol PI. 1/2008

ADA/EASD Consensus: *Treatment Algorithm*



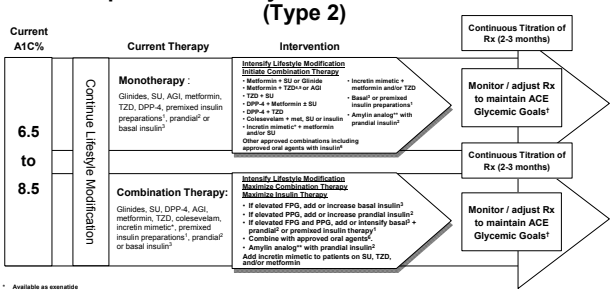
***Check A1C every 3 months until <7% and then at least every 6 months. †Although 3 oral agents can be used, initiation and intensification of insulin therapy is preferred based on effectiveness and expense.**
ADA/EASD. *Diabetes Care*. 2006;29:1963-1972.

Updated ADA/EASD Consensus Algorithm



Reinforce lifestyle interventions at every visit and check A1C every three months until A1C < 7.0 %, then at least every 6 months thereafter. Change interventions whenever A1C ≥ 7.0 %.
[a]Sulfonylureas other than glybenclamide (glyburide) or chlorpropamide.
[b]Insufficient clinical use to be confident regarding safety.
Nathan DM, et al., *Diabetes Care* 2008;31:1-11.

Road Map to Achieve Glycemic Goals: Treated Patients (Type 2)



* Available as exenatide
** Available as pramlintide
1 Analog preparations preferred
2 Prandial insulin (rapid-acting insulin analogs available as lispro, aspart, glulisine, or regular insulin) can be added to any therapeutic intervention at any time to address persistent postprandial hyperglycemia
3 Available as glargine and detemir
4 *A recent meta-analysis suggests a possible link of rosiglitazone to cardiovascular events; other studies do not confirm or exclude this risk. The FDA has stated "In their entirety, the available data on the risk of myocardial infarction are inconclusive."*
5 Cannot be used in NYHA CHF Class 3 or 4
6 According to the FDA, rosiglitazone not recommended with insulin

Endocr Pract. 2007;13:260-268

†ACE Glycemic Goals
≤ 6.5% A1C
< 110 mg/dL FPG
< 110 mg/dL Preprandial
< 140 mg/dL 2-hr PPG

Access Roadmap at:
www.aace.com/pub

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Algorithm-based Diabetes Care

Ann Bullock, MD
Chris Lamer, PharmD, BCPS, CDE

Why Cherokee algorithms were developed

- Noticed many pts with diabetes (DM) coming in for walk-in care but DM not addressed
  - -even when BG or BP elevated on chart
- Walk-in providers often temporary or "green"
  - -little knowledge of DM SOC and/or local formulary options
- Not enough room in Diabetes Clinic to care for all DM pts
  - -SOC not being met for non-DM clinic pts
  - ***So how to help those providers do this??***

Algorithm Cards Created:1997

- Simple to use by providers who don't "do" diabetes
  - -Found other algorithms cumbersome
  - -Goal: "every visit is a diabetes visit"
- Easy and inexpensive to produce
  - -written on computer, printed locally
- Updateable (this is 4th update)
  - -changes in standards of care
  - -formulary changes

Algorithms (cont'd)

- *Only* Cherokee formulary medications
  --minimizes frustration
- Dosing information
  --providers don't need to look up dose
- ADA, IHS, JNC VII, ATP III, KDOQI, and other international standards of care
- Fit in white coat pocket
- Collaborative: written by MD-PharmD team with input from rest of clinical staff
- Cards on Glucose Control, HTN, Lipids, Insulin and CKD

Why a card on CKD? Isn't that too specialized for our clinics?

- Chronic Kidney Disease—no longer can a few providers "do" all the CKD
  - -Cherokee has ***355 pts*** with a GFR of <60 ml/min (Stage 3+)
  - -CKD SOC can help pts feel better and prolong time to starting dialysis

National Diabetes Treatment Algorithms

- IHS Core Formulary meds
- Both meds and algorithms are customizable and updatable in electronic format
  - -to local formulary
  - -to local preference for treatment paths

National Review

Type 2 DM – Glucose Control

DM DX – *at least two (same or combination)*

1. FPG ≥ 126
2. 2 * (OGTT) ≥ 200
3. Non-fasting tab glucose ≥ 200 with symptoms

Impaired Fasting Glucose = IFPG 100-125
Impaired Glucose Tolerance= 2* OGTT 140-199

DM BG Targets
Premeal: < 70-130
2*PP: <160-180
A:C: 6.5-7%

Individualize if elderly or co-morbidity

Metformin with Lifestyle Interventions

Add Sulfonylurea

Basal Insulin
Bedtime NPH 10 units
Long Acting Insulin 10 units

TZD

If FPG > 250
OR Non-fasting glucose >300
OR A1c > 10%
OR Active liver disease/ ETOH abuse
OR ketonuria
OR weight loss

(Hospitalize if acidotic)

Intensify Insulin Therapy
(See Insulin Card)

Immunizations
Pneumovax—At Dx & again at age 65 (If ≥5 yrs. since 1st shot)
Flu shots yearly
Td (Tdap (routine)
PPD once after Dx of DM (Pos is ≥10mm)

Monitoring of DM
A1c every 3 months
Creatinine and GFR yearly
UA yearly
Microalbumin yearly
Lipid Panel yearly
EKG every 2-5 years
Complete Foot Exam yearly
- Foot inspection each visit
Retinopathy exam yearly
Pap, Mammograms, Contraception
Evaluate sexual function
Depression, Tobacco, ETOH, DV screening yearly

Don't Forget
Glucose toxicity— Insulin production↓'s if prolonged hyperglycemia; insulin shots short-term reverse this.
Pancreatic Exhaustion— Almost all Type 2 diabetics will eventually require insulin.

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Type 2 DM – Glucose Control

Biguanides: Metformin & Metformin XR (Glucophage®)
Start 500 mg daily with meals and increase no faster than 500 mg each week. If GI sx occur may increase more slowly.
Max. dose: 2000mg daily or divided with XR tablets. Do not split XR tablets.
2500 mg divided B/D-T/D with regular release tablets.
Can decrease weight. Pt. must have normal creatinine (males <1.5, females <1.4), no heart failure or liver disease (check ALT) and no significant ETOH use. Discontinue before surgery or IV contrast dye administration.

Sulfonylureas: Glyburide (Micronase®) and Glipizide (Glucotrol®)
Start 2.5-5mg daily – Max 10 mg B/D
Can increase weight and cause hypoglycemia

Thiazolidinediones: Pioglitazone (Actos®)
Start 15mg daily; may increase to 30mg daily (little benefit doing over 30mg)
Max A1c changes may take up to 12 weeks to occur
Check ALT at baseline & periodically. No underlying liver dz or significant ETOH use.
Warning: may cause Heart Failure. May use in renal insufficiency. Can cause weight gain.

Sitagliptan (Januvia®) - DPP-4 inhibitor
May reduce weight, mild to mod A1c lowering Dose: 100mg PO daily
Reduce dose if ≥ Stage 3 CKD

Vildagliptin (Galvus®) - DPP-4 inhibitor
May reduce weight Dose: 50-100mg PO daily

Exenatide (Byetta®) - GLP1 mimetic
May reduce weight, mild to mod A1c lowering
Start 5 mcg/dose BID Administer 60 minutes before meals
Weekly dose (Byetta LAR) is under investigation
May increase to 10 mcg/dose B/D after 1 month of treatment
Do not use if ≥ Stage 4 CKD Do not mix in same syringe as insulin
May be associated with pancreatitis – seek medical care if persistent severe abdominal pain with or without vomiting

Pramlintide (Symlin®) - Amylin mimetic
Use in Type 2 Diabetes unclear; May consider in Type 1 Diabetes
Start 60mcrograms daily subcutaneously immediately before a major meal
(Reduce prepramlat (short acting) insulin by 50% as appropriate)
May increase to 120mcrograms after significant nausea is gone x 3-7 days
Do not mix in same syringe as insulin

Drugs names in italics are not on the IHS National Core Formulary

Ref: ADA Clinical Practice Recommendations 2007, 2008
http://care.diabetesjournals.org/content/vol30/suppl_1/

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Type 2 DM – Hypertension

First Line
Therapeutic Lifestyle Changes
↓
ACE Inhibitor: Lisinopril / Captopril
ARB (if cough/angioedema on ACEI)

Second
Diuretic
HCTZ

Third/Fourth
β-Blocker
Metoprolol / Atenolol
↔
Calcium Channel Blocker
Diltiazem

May Consider adding
Clonidine
Alpha Blocker
Doxazosin/Terazosin

BP TARGET
<130/80
Treat to Achieve This Goal

Ref: JNC VII;
www.nhlbi.nih.gov/guidelines/hypertension/index.htm

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Type 2 DM – Hypertension

ACE Inhibitors (ACEI)/ARBs	
Renal protective in diabetics—consider using if Microal (+), even if BP < 130/80. Can cause ↑ K+, ↑ creatinine, cough (not with ARB), rarely angioedema.	
Lisinopril (Prinivil®/Zestril®)	Start 2.5-5mg daily; usually 20-40mg daily
Captopril (Capoten®)	Start 12.5 B/D-T/D; max 150mg T/D
Losartan (Cozaar®)	Start 25-50mg daily; usually 100mg daily Consider if unable to tolerate ACEI
Telmisartan (Micardis®)	Start 40mg daily; usually 20-80mg daily Consider if unable to tolerate ACEI
Diuretics	
HCTZ	*Start 12.5-25 mg daily; usually 25mg daily* *Can ↓ K+ (Problems ? with higher doses > 25mg)*
Maxzide®	*Dose: ½ tab daily (to keep HCTZ dose at 25mg); 1 tab = 50mg HCTZ/75mg triamterene; K+ sparing – Caution esp. in CKD*
β-blockers (BABA)	
Don't use if bradycardia or 2nd/3rd degree block. Caution in Severe CHF, Asthma, or Renal dysfunction	
Atenolol (Tenormin®)	Start 25-50mg daily-B/D; usually 50-100mg daily Eliminated renally (caution Renal Failure)
Metoprolol (Lopressor®)	Start 50-100mg B/D; usually 100-400mg daily in 1-2 divided doses. (XR formulation dosed once daily) Eliminated hepatically (caution in Liver Failure) Preferred β-Blocker for renal dysfunction or heart failure
Carvedilol (Coreg ®)	Start 3.125-6.25mg. Usual dose 25mg B/D Consider in patients with heart failure
Calcium Channel Blockers (CCBs)	
Diltiazem CD (Cardizem®)	Start 120mg daily; usually 120-420mg daily
Amlodipine (Norvasc ®)	Start 5mg daily; 5-10mg daily consider in patients with angina or CHF
Nifedipine XL (Adalat Procardia®)	Consider use if patient cannot tolerate diltiazem; Start 30mg daily; usually 30-120mg daily; Caution edema, CHF, and MI
Nisoldipine (Sular®)	Consider use if patient cannot tolerate diltiazem; Start 20mg daily; usually 10-40mg daily; NVT 60mg daily; Caution edema, CHF, and MI
Alpha Blockers	
Doxazosin (Cardura®)	Start 1mg immediate release HS; Max dose 16mg daily; Can cause dizziness, drowsiness, and weakness. Titrate up slowly
Terazosin (Hytrin®)	Start 1mg HS; Max dose 20mg daily; Can cause dizziness, drowsiness, and weakness. Titrate up slowly
Central Acting	
Clonidine (Captopres®)	Start 0.1mg B/D; usually 0.1-0.3mg B/D; Can cause ↑ sedation/dizziness/weakness; Titrate ↑ slowly. Do not withdraw abruptly

Drugs names in italics are not on the IHS National Core Formulary

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Type 2 DM – Lipids

Step 1: Ensure triglycerides (TG) are not dangerously high

TG > 500 → Restrict fats, Intensify blood glucose control, R/O hypothyroidism or other causes. Consider: Gemfibrozil, Fish Oil, Niaspan

Step 2: Check LDL LDL Goal is < 100
Consider LDL < 70 for known CVD

LDL < 100 → Therapeutic Lifestyle Changes

LDL > 100 → Start Simvastatin 40mg at bedtime If goal LDL not achieved with Simvastatin 80mg, consider switching to Atorvastatin 80mg daily

Step 3: Check Triglycerides Triglyceride Goal is < 150

TG > 150 → Start/Add Fish Oil, Gemfibrozil 600mg BID, or Niaspan 500mg HS

All Adults with Diabetes > Age 30
ASA 81mg daily if no history of CVD

Ref: ADA Clinical Practice Recommendations 2007 & ATP III
http://care.diabetesjournals.org/content/vol30/suppl_1/
http://www.nhlbi.nih.gov/guidelines/cholesterol/index.htm

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Type 2 DM – Lipids

Medication	LDL	HDL	TG	Start Dose	Max Dose
Statins					
Simvastatin (Zocor ®)	-20-50%	+5-10%	-15-30%	40mg HS	80mg HS
Atorvastatin (Lipitor ®)	-20-55%	+5-10%	-15-30%	10-20mg daily	80mg daily
Fibrates					
Gemfibrozil (Lopid ®)	-0-20%	+10-25%	-20-35%	600mg BID	
Fenofibrate (Tricor ®)	-20%	+9-15%	-20-35%	145mg daily	
Others					
Niacin (Niaspan ®)	-5-20%	+10-30%	-10-35%	500mg HS	2-3g HS
Ezetimibe (Zetia®)	-13%	+1-4%	-8-11%	10mg daily	
Cholesevelam (Welchol ®)	-15-20%	+3%	+0-5%	3 tab BID or 6 tab daily	
Fish Oil (Omacor ®)	+45%	+9%	-45%	4g / day	
Fish Oil (OTC)	+5-10%	+1-3%	-28-40%	2-4g EPA + DHA	

Drugs names in italics are not on the National Core Formulary
Note: Atorvastatin 80mg is on the formulary while lower doses are not

Contraindications
Statins: active liver disease, ETOH abuse, pregnancy & lactation
Niacin: active gout or PUD, active liver disease, pregnancy & lactation
Gemfibrozil: active liver disease, gallbladder disease, pregnancy & lactation

Lab Monitoring

ALT
Simvastatin Baseline, 6 mos then yearly
Simvastatin 80mg Baseline, 3 mos, 6 mos then yearly
Atorvastatin 80mg Baseline, 12 weeks, then every 6 mos
Niacin Baseline, Q 3 mos for 1 year then Q 6 mos
Gemfibrozil Baseline, Q 3 mos for 1 year then Q 6 mos
Discontinue or restart at lower dosage if ALT > 3 times ULN
Omacor Periodic ALT

Other Lab Tests – Check CK if muscle pain
Niacin Follow glucose closely; Check uric acid levels at baseline and when dose is stable; may check in patients suspected of gout.

Combination Therapy
Fibrates (Gemfibrozil) & Statin: begin with low doses of Simvastatin (5-20mg); obtain baseline CK and recheck if complaints of muscle symptoms.
Other Combinations: Obtain baseline ALT and CK. Follow strictest monitoring guidelines.

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Type 2 DM – Insulin

STEP 1: Target Fasting Plasma Glucose with **Basal Insulin**
Fasting Plasma Glucose (FPG) Target = 70-130mg/dl

HS NPH or Long Acting insulin – start 10 units or 0.2 units/kg
↓
Increase dose 2 units every 3 days until FPG is 70 - 130mg/dl
May increase by 4 units every 3 days if FPG is > 180mg/dl

STEP 2: Target Pre-Meal Glucose (target one at a time)
Premeal Glucose Target = 70-130mg/dl

If Pre-lunch glucose > 130mg/dl
Start 4 units Bolus Insulin before breakfast

If Pre-supper glucose > 130mg/dl
Start 4 units Bolus Insulin before lunch
OR Add/Increase morning NPH

If Bedtime glucose > 140mg/dl
Start 4 units Bolus Insulin before supper OR Increase evening NPH

→ Increase Bolus Insulin by 2 units every 3 days
As insulin doses get larger, (over 10 units), begin to change insulin dose by 10-20%

STEP 3: If A1c not at goal: Target Post-Prandial Glucose
with **Bolus** premeal insulin
2 Hour Post-Prandial Glucose Target <160-180mg/dl

Ref: Nathan, Buse, Davidson, et al. Management of Hyperglycemia in Type 2 Diabetes : a Consensus Algorithm for the Initiation and Adjustment of Therapy. (2006). Diabetes Care 29, 1963-1972

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Type 2 DM – Insulin

Basal Insulin – longer acting insulin

Insulin	Onset	Peak	Duration
Basal Insulin - Intermediate Acting Insulin			
NPH (Humulin N ®, Novolin N ®)	1-3 hours	6-10 hours	12-20 hours
Basal Insulin – Long Acting Insulin			
Glargine (Lantus ®)	1 hour	None	24 hours
Levemir (Detemir ®)	1 hour	None	12-24 hours

Bolus Insulin – shorter acting insulin

Insulin	Onset	Peak	Duration
Bolus Insulin – Rapid and Short Acting Insulin			
Lispro (Humalog ®) *Aspart (Novolog ®)* *Glulisine (Apidra ®)*	15-30 min	30-90 min	3-5 hours
Regular (Humulin R ®, Novolin R ®)	30-60 min	1-2 hours	5-8 hours

Premixed Insulin – longer and shorter acting
Use **ONLY** if the patient cannot mix insulin

Insulin	Onset	Peak	Duration
Mixed Insulins			
Humulin, Novolin, Novolog 70/30	30 min	2-4 hours	14-24 hours
Humulin 50/50	30 min	2-5 hours	18-24 hours
Humalog 75/25	15 min	½-2½ hrs	16-20 hours

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Type 2 DM – Chronic Kidney Disease

Stages of Chronic Kidney Disease (CKD)

	1	2	3	4	5
eGFR	> 60	> 60	30-59	15-29	< 15 ml/min
Proteinuria	micro	macro			

Referrals

Nephrologist: GFR < 60ml/min or sooner if unsure of etiology of renal disease
Nutrition: Refer to RD for consult (protein, Na+, K+, PO_4, fluids, saturated fat)

Managing Complications of CKD – Stages 3-5

Acidosis

If CO_2 < 22mmol/L	Start sodium bicarbonate 325-650mg (1-2 tabs) TID-QID	Goal: CO_2 ≥ 22mmol/L

Anemia

Check Hb at least yearly: Anemia = Hb <13.5 g/dL adult men, <12 g/dL adult women
Consider r/o B12/folate deficiency, GI blood loss, other causes
Baseline Labs: Ferritin, transferrin % sat, iron studies (Fe, % Sat, TIBC), and CBC with diff
Start oral iron therapy if ferritin/iron studies low
Ferrous Sulfate ($FeSO_4$) 325mg daily to TID
Consider discussing 100mg BID to reduce constipation
Monitor ferritin to avoid iron overload
Consider IV iron if needed (see iron protocol)
Consider erythropoiesis stimulating agents (ESA) for Hb < 10 (see epo protocol)
Dose ESA to maintain Hb > 10.5 and < 12

Diabetes & Cardiovascular Disease (CVD)

Blood sugar control—as renal fxn declines pts' BGs often improve—titrate meds down as needed
D/C metformin when Creatinine >1.5 men or >1.4 women
Insulin usually the only/main medication used for patients with CKD
Peripheral Neuropathy: Foot ulcers common, check feet each visit, refer to shoe clinic
Retinopathy: Ophthalmologist visits regularly
Autonomic Neuropathy: Frequent BP fluctuations, including orthostatic sx.
HTN: BP goal ≤130/80; continue ACE/ARB (watch K+)
CVD: CKD increases CVD risk – patients on aspirin (if no contraindications)
Achieve lipid targets, encourage tobacco cessation

Edema/Fluid Overload

Establish patient's dry weight
Titrate furosemide as needed 20-240mg BID (diuresis lasts 6 hours-give AM & mid-day)

Ref: KDOQI/NKF and UK Renal Assoc 4th Ed. Clinical Practice Guidelines for Complications of Chronic Kidney Disease

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Type 2 DM – Chronic Kidney Disease

Metabolic Bone Disease

Phosphorus (PO_4): if >4.6 mg/dL start binder (calcium); Refer to RD for dietary PO_4 restriction
Calcium (Ca): target: 8.4-9.5 mg/dL
If <8.4, start/increase calcium supplementation
If >10.2, correct causes (often 2° meds, need to hold Ca and/or Vit D/calcitriol
iPTH: targets - Stage 3: 35-70pg/mL, Stage 4: 80-150pg/mL
If PTH elevated, measure 25(OH)D (Vitamin D)
If 25(OH)D <=30ng/mL, start calcitriol
If 25(OH)D <30ng/mL, start ergocalciferol (Vit D2)
Follow Ca, PO_4, iPTH, and 25(OH)D (Vitamin D). If Ca or PO_4 above target or if PTH below target, hold calcitriol and/or calcium

CKD Stage	eGFR	iPTH goal	PO_4 goal	Ca goal	Ca X PO_4
3	30-59	35-70	2.7-4.6	8.4-9.5	< 55
4	15-29	80-150	2.7-4.6	8.4-9.5	< 55
5	< 15	150-300	3.5-5.5	8.4-9.5	< 55

Medication*	iPTH effect	PO_4 effect	Ca effect	Comments
Phosphate Binders				
$CaCO_3$ (Cyal-Cal or TUMS) 500-2000mg with meals (no more than 7g/d)	-	↓	↑	Use if Ca < 8.4
Ca Acetate 1334-2668mg with meals	-	↓↓	↑	Use if Ca < 8.4 & PO_4 > 5
Sevelamer (Renagel) 800-1600mg TID	-	↓↓	-	Decrease PO_4, no effect on Ca
Lanthanum 1500-3750mg/day w/ meals	-	↓↓	↓	Decrease PO_4 and Ca++
Aluminum 600-1200mg TID between meals & HS	-	↓↓	-	ONLY if PO_4 > 7 and Ca x PO_4 > 55, not more than 30 days
Vitamin D and Analogs				
Vit D (Ergocalciferol) 1.25-5mg daily	↓	-	↑↑	Use if Vit D < 30ng/ml
Calcitriol 0.25-1mcg daily or 0.5-3mcg TIW	↓	-	↑↑	Use only if Ca & PO_4 in hi range
Doxercalciferol 1-3mcg daily or 10-20mcg TIW	↓	-	↑	Hold if Ca x PO_4 > 55
Other				
Cinacalcet 30-180mg daily	↓	↓	↓↓	Do not use if Ca < 8.4

*Always include dietary phosphorus restriction
Drugs in **italics** are not on the IHS National Core Formulary

Lab Monitoring

Parameter	GFR > 60	GFR 30-59	GFR 15-29	GFR < 15 not on dialysis
Creatinine	Annual	Each visit	Each visit	Each visit
GFR	Annual	Each visit	Each visit	Each visit
Hb	Annual	Q3-4 mos*	Q3-4 mos*	Q3-4 mos*
Serum Fe		Q3-4 mos	Q3-4 mos	Q3-4 mos*
Transferrin Sat		Q3 mos	Q3 mos	Q3 mos*
Ferritin		Q3 mos	Q3 mos	Q3 mos
iPTH		At least annually*	Q3 mos*	Q3 mos*
Ca & PO_4		At least annually*	Q3 mos*	Q month*

* Monitor more often if on medications that affect these labs

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Where to Get the Algorithms

- IHS DDTP website: www.ihs.gov/MedicalPrograms/Diabetes/
  - -508 compliant text version
  - -Word file in Algorithm format

Customize them

- Adjust meds to your local formulary
- Send out electronically to all clinical and diabetes staff for feedback
  - Be sure your Nephrology group is on board with CKD card
- Once consensus reached, print cards (laminate?), post electronically on clinic website
- Emphasize algorithms are not "the law"

Update them

- We will review cards at least annually
  - -check for updates on DDTP website
- You should also update cards when:
  - you make a significant formulary change
  - when there's a major change in national standards—you don't have to wait til we adjust the cards on the DDTP website!
  - But don't print new cards for every small change—you'll drive staff a little crazy….

Conclusions

- It's hard for non-DM providers to keep up with changes in SOC and meds
- DM and its complications are increasingly devastating our communities
  - "Every visit MUST be a diabetes visit"!
- Algorithm cards can be a useful tool to help staff remember to do DM care—and feeling more confident to do so
- Goal: Better DM care leading to healthier lives for our patients and communities

Advances in Indian Health Conference

- 9th year of IHS' primary care conference
- April 21-24 at Radisson Albuquerque
- 28 hours of CME
  - Diabetes track sponsored by DDTP
- UNM Office of CME website: hsc.unm.edu/cme/

"Not everything that can be counted counts, and not everything that counts can be counted."

Albert Einstein

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