
As-Salamu Alaykum

APPROVED

T2DM MED 341 FEB 2021

AlMaatouq MA, MD


Photo by
3M

OBJECTIVES:

APPROVED

1. SCOPE OF DIABETES
2. MAKING THE DIAGNOSIS
3. PATHOPYSIOLOGY
4. DISEASE CONSEQUENCES
5. MANAGEMENT
6. CONCLUSION

1

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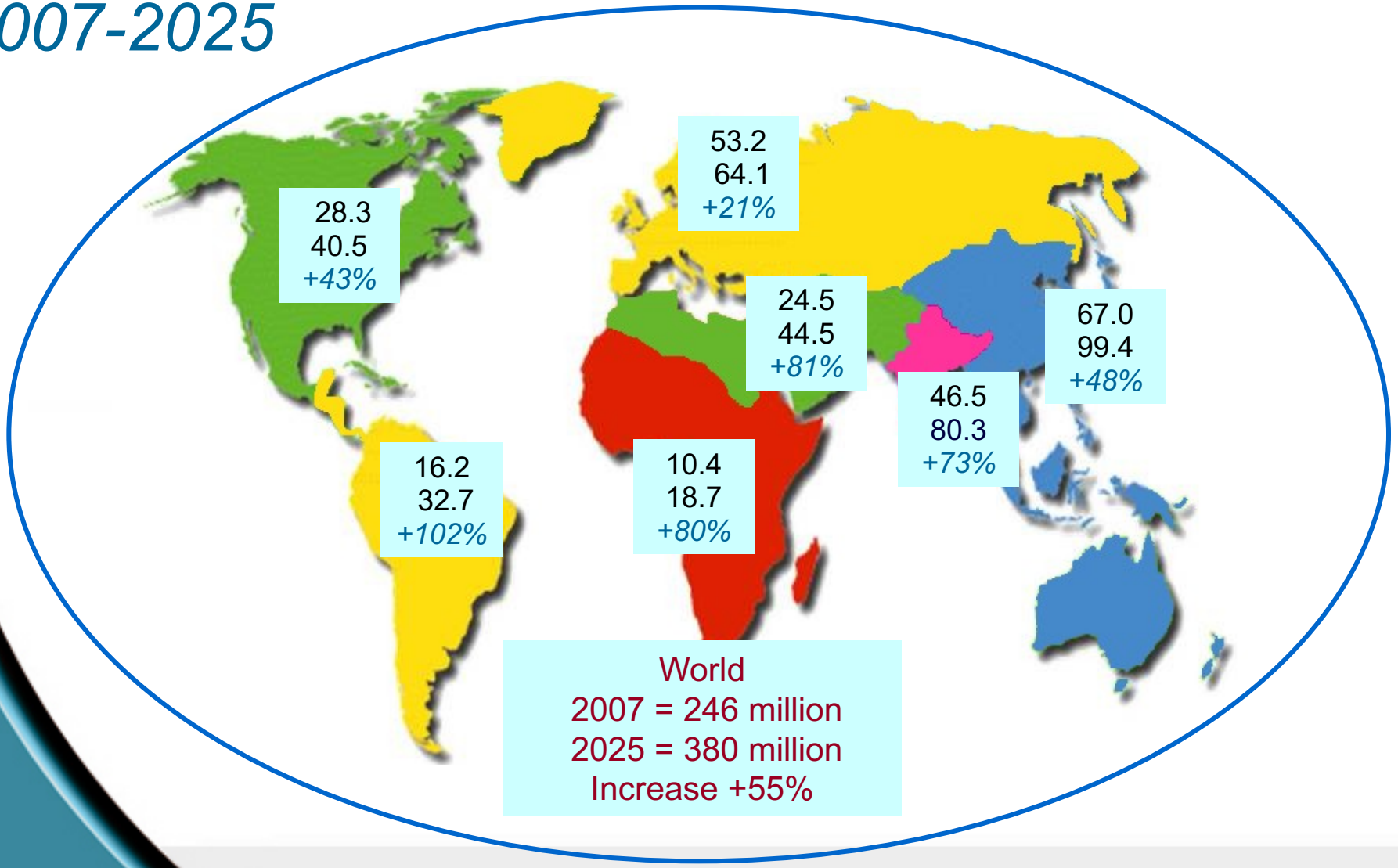
Global projections for diabetes (millions)
2007-2025



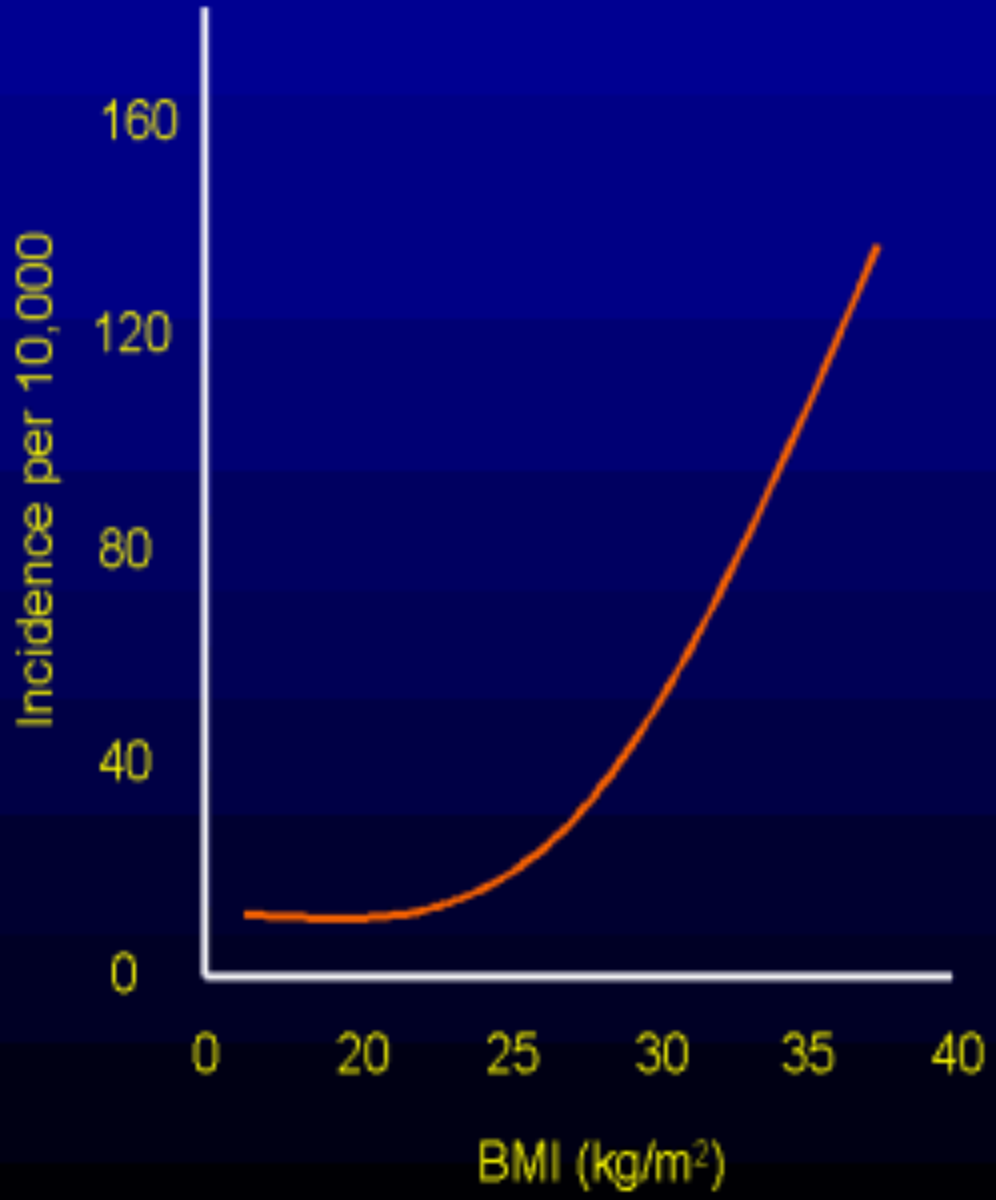
Diabetes Atlas, 3rd edition, IDF 2006

Obesity and type 2 diabetes

- Elevated BMI (> 25) is associated with an increased risk for the development of Type 2 diabetes.

- Prevalence of Type 2 diabetes closely matches the prevalence of obesity.

- Elevated TNF-α levels may be partly responsible.



Obesity in Saudi Arabia

Mansour M. Al-Nozha, Yaqoub Y. Al-Mazrou, Mohammed A. Al-Maatouq, et al

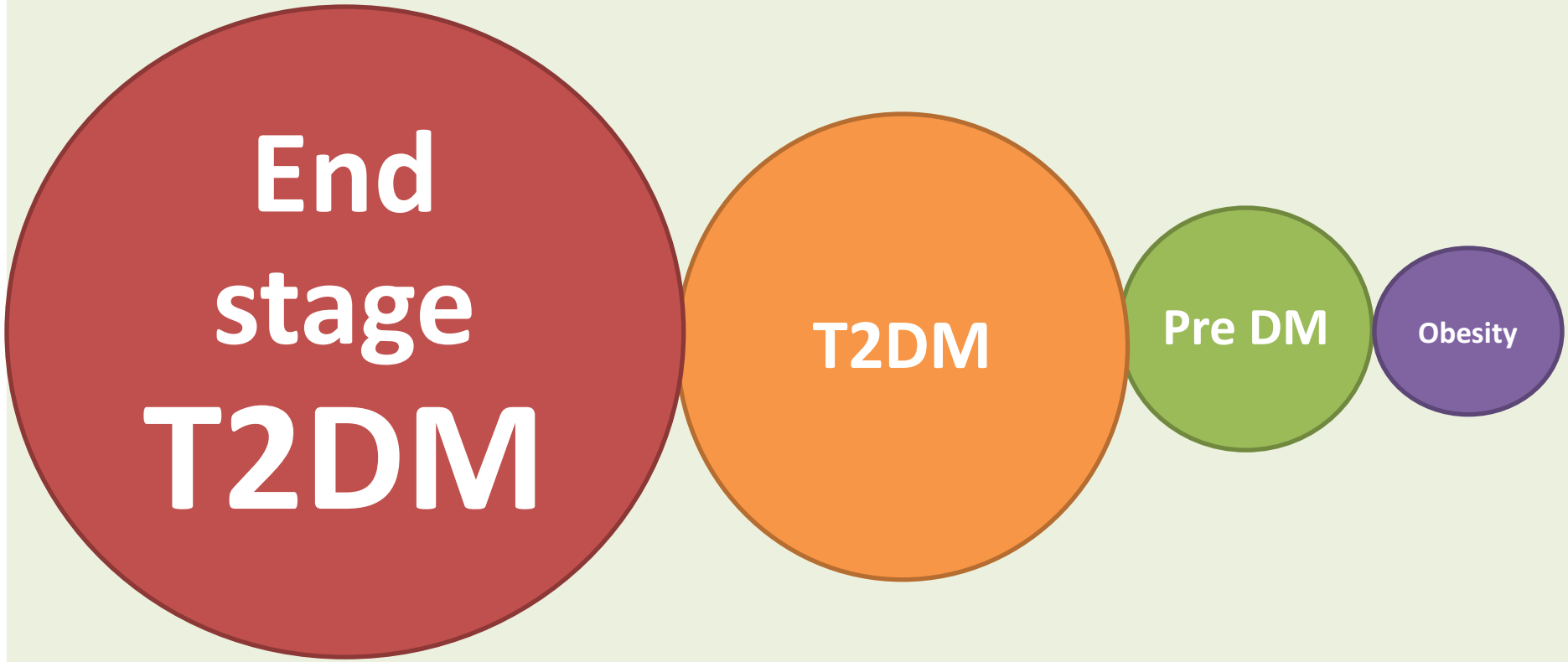
Methods: This study is a community-based national epidemiological health survey, conducted by examining Saudi subjects in the age group of 30-70 years of selected households over a 5-year period between 1995 and 2000 in KSA.

Results: Data were obtained by examining 17,232 Saudi subjects from selected households who participated in the study. The prevalence of **overweight was 36.9%.** Overweight is significantly more prevalent in males (42.4%) compared to 31.8% of females ($p<0.0001$). The age-adjusted **prevalence of obesity was 35.5%** in KSA with an overall prevalence of 35.6% [95% CI: 34.9-36.3], while severe (gross) obesity was 3.2%. Females are significantly more obese with a prevalence **of 44%** than males **26.4%** ($p<0.0001$).

Conclusion: Obesity and overweight are increasing in KSA with an overall obesity prevalence of **35.5%**.

Saudi Med J. 2007 Apr ;28 (4):559-68

Costs of T2DM 2030



Diabetes mellitus in Saudi Arabia

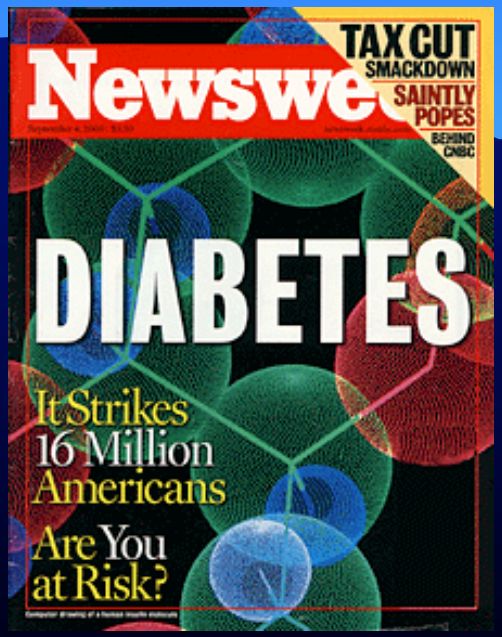
Al-Nozha MM, Al-Maatouq MA, Al-Mazrou YY, et al

- OBJECTIVE: Diabetes mellitus (DM) is a major public health problem worldwide, and it is a known risk factor for coronary artery disease (CAD). New recommendations for the diagnosis of diabetes have changed the epidemiology of DM. Therefore, we designed this study with the objective to determine the prevalence of DM among Saudis of both sexes, between the ages of 30-70-years in rural as well as urban communities. This work is part of a major national project: Coronary Artery Disease in Saudis study (CADISS) that is designed to look at CAD and its risk factors in Saudi population.
- METHODS: This study is a community-based national epidemiological health survey, conducted by examining Saudi subjects in the age group of 30-70-years of selected households over a 5-year period between 1995 and 2000. Data were obtained from history, fasting plasma glucose levels, and body mass index. The data were analyzed to classify individuals as diabetic, impaired fasting glucose and normal, using 1997 American Diabetes Association (ADA) criteria, which was adopted by the World Health Organization (WHO) in 1998, to provide prevalence of DM in the Kingdom of Saudi Arabia (KSA).
- RESULTS: A total of 17232 Saudi subjects were selected in the study, and 16917 participated (98.2% response rate). Four thousand and four subjects (23.7%), out of 16917 were diagnosed to have DM. **Thus, the overall prevalence of DM obtained from this study is 23.7% in KSA**. The prevalence in **males and females were 26.2% and 21.5% (p<0.00001)**. The calculated age-adjusted prevalence for Saudi population for the year 2000 is 21.9%. Diabetes mellitus was more prevalent among Saudis living in **urban areas of 25.5% compared to rural Saudis of 19.5%** (p<0.00001). Despite the readily available access to healthcare facilities in KSA, a large number of diabetics 1116 **(27.9%) were unaware of having DM.**
- CONCLUSION: The overall prevalence of DM in adults in KSA is 23.7%. A national prevention program at community level targeting high risk groups should be implemented sooner to prevent DM. We further recommend a longitudinal study to demonstrate the importance of modifying risk factors for the development of DM and reducing its prevalence in KSA.

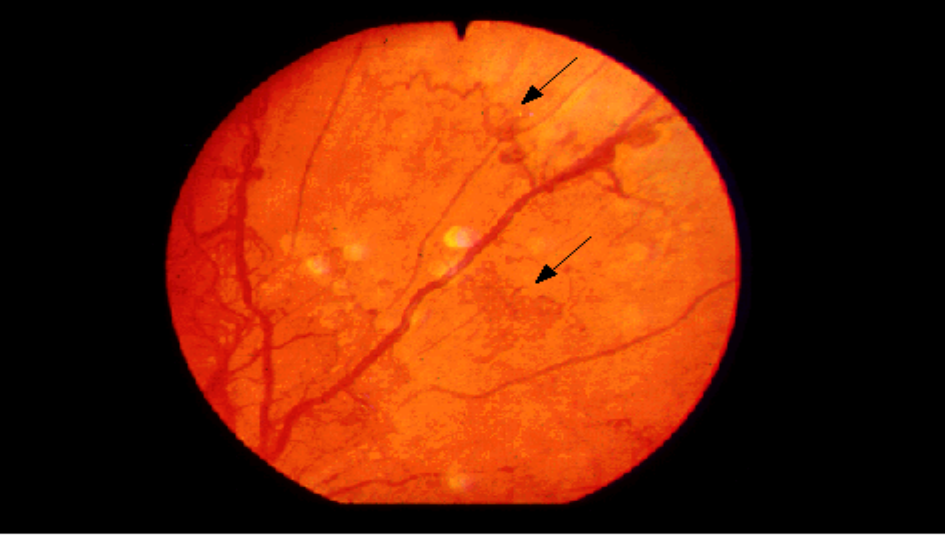
Saudi Med J. 2004 Nov;25(11):1603-10.

KSA Diabetes Prevalence

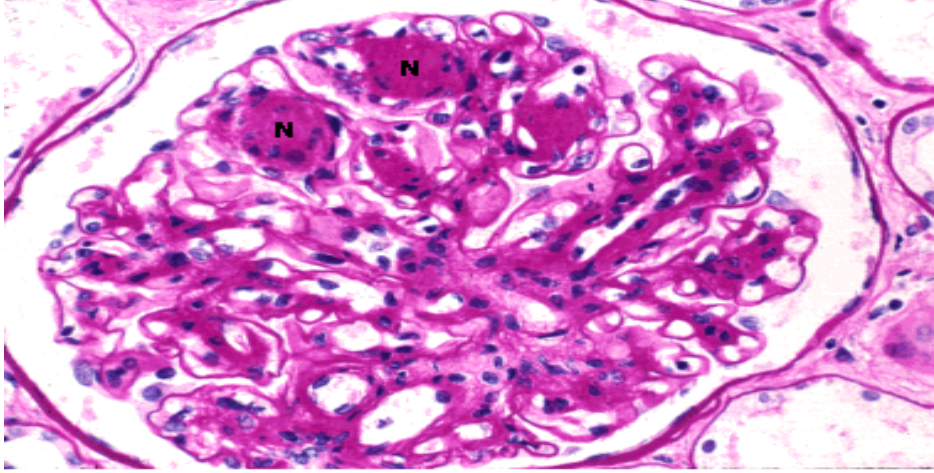
2 Million



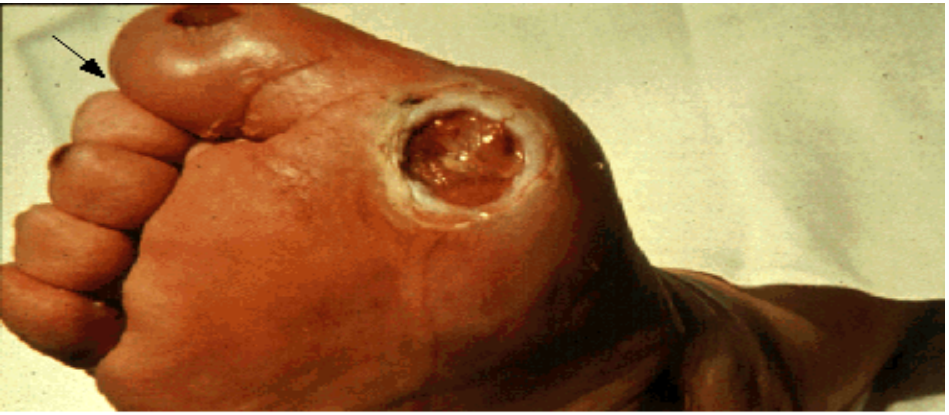
- Diabetes kills 1 Saudi every 30 minutes
- New case diagnosed every 2 MINUTES
- More deaths than TB and breast cancer combined
- Average life expectancy: 15 years less than non-diabetes population



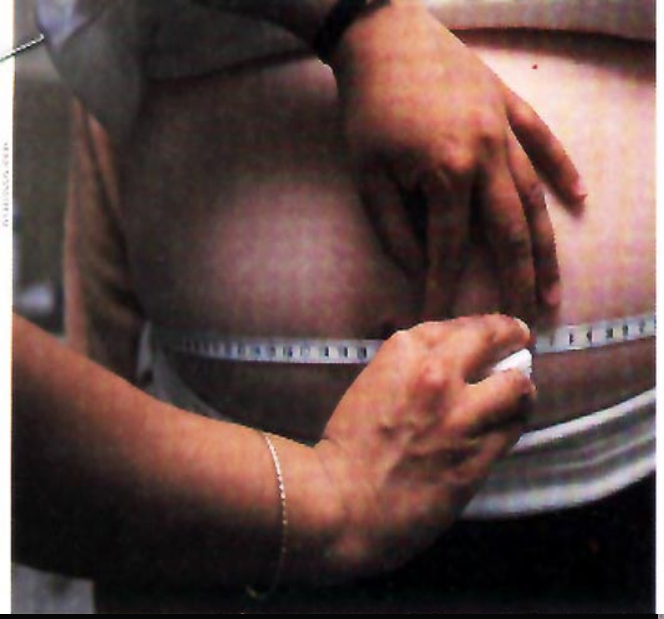
Early diabetic retinal neovascularization Diabetic retinopathy, showing irregular changes in venous caliber, tortuosity of blood vessels, and proliferation of networks of fragile new vessels, arising from both arteries and veins (arrows). Courtesy of David McCulloch, MD.



Diabetic nephropathy Light micrograph showing diffuse and nodular (N) glomerulosclerosis in diabetic nephropathy. Note the dense appearance of the deposits and the rim of cells around the nodules, which distinguish this disorder on light microscopy from fibrillary glomerulonephritis or amyloidosis. Courtesy of Helmut Rennke, MD.

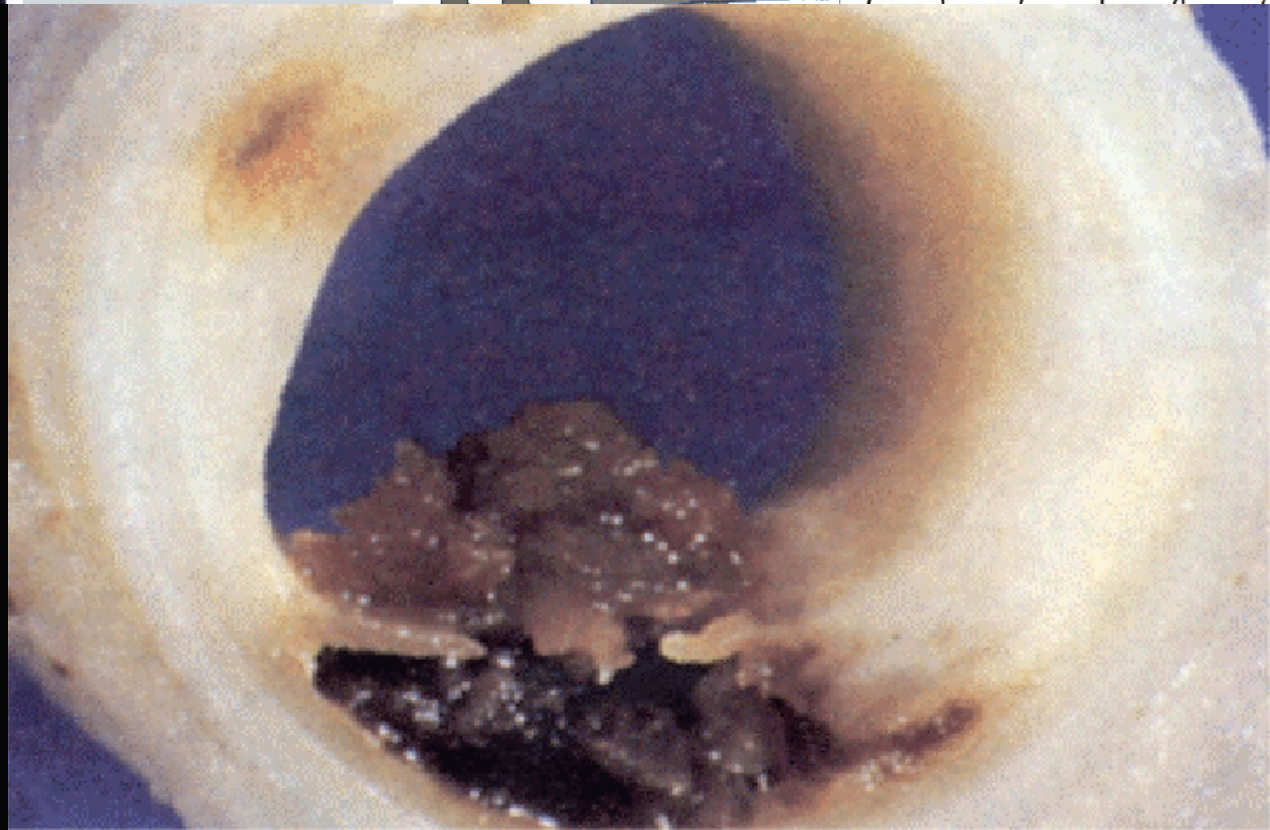


Wagner grade 2 ulcer and claw toe Foot from a diabetic patient with a penetrating neuropathic ulcer that is not associated with abscess formation or bone involvement (Wagner grade 2). The toes have been pulled anteriorly because the anterior tibial muscles are unopposed due to motor neuropathy–induced weakness of the intrinsic foot muscles. This promotes subluxation of the proximal interphalangeal–metatarsal joints, resulting in a claw toe appearance (arrow) and in increased pressure on the metatarsal heads, predisposing to ulcer formation at this site. Courtesy of David McCulloch, MD.




Hypertension
oh oh!


Cholesterol



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What is the

GOLD standard

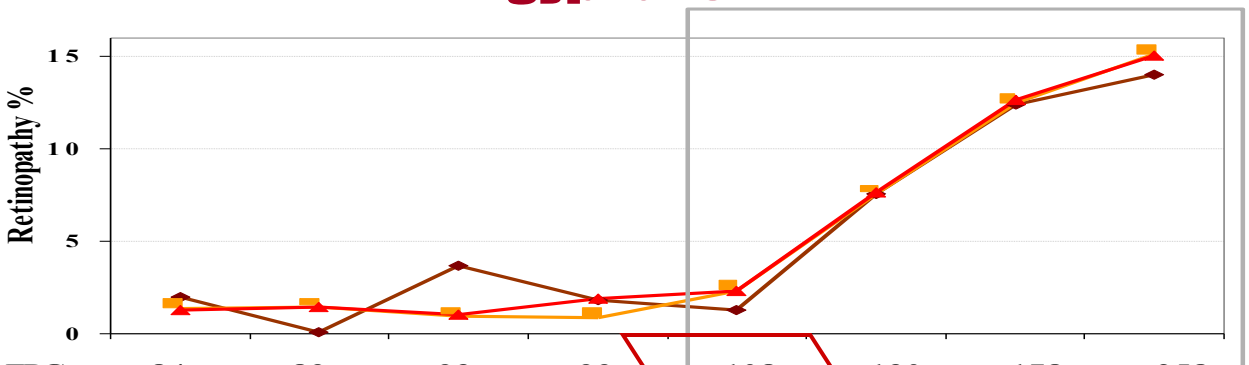
for the Diagnosis of Diabetes?


IF YOU HIT THIS SIGN,
YOU WILL HIT THAT BRIDGE

Etiologic classification of diabetes mellitus

Impaired glucose tolerance **IGT**
Impaired fasting glucose **IFG**

Prevalence of Retinopathy by Deciles
Egyptians



FPG	84	89	93	99	108	130	178	258
2h PG	90	99	110	125	155	218	304	386
HbA1c	4.9	5.1	5.4	5.6	6.0	6.9	8.5	10.3

Diabetes Care 1997;20, 1183-1197

FPG	93	97	100	105	109	116	136	226
2h PG	106	116	126	138	156	185	244	364
HbA1c	5.0	5.2	5.3	5.5	5.7	6.0	6.7	9.5

Diabetes Care 1997;20, 1183-1197

FPG	90	93	96	98	101	104	109	120
2h PG	86	94	102	112	120	133	154	195
HbA1c	5.1	5.2	5.4	5.5	5.6	5.7	5.9	6.2

Diabetes Care 1997;20, 1183-1197

Diagnostic criteria for T2DM

1.	8 hours fasting plasma glucose value ≥ **126** mg/dL (7 mmol/L)

OR

2.	Polyuria, polydipsia, unexplained weight loss and random plasma glucose value ≥ **200** mg/dL (11.1 mmol/L)

OR

3.	2 hour plasma glucose during a (75 g anhydrous glucose) OGTT ≥ **200** mg/dL (11.1 mmol/L)

OR

4.	HbA1c ≥ 6.5 %

1, 3 & 4 to be confirmed by repeat testing on a different day if no unequivocal hyperglycemia

APPROVED

Etiologic classification of diabetes mellitus

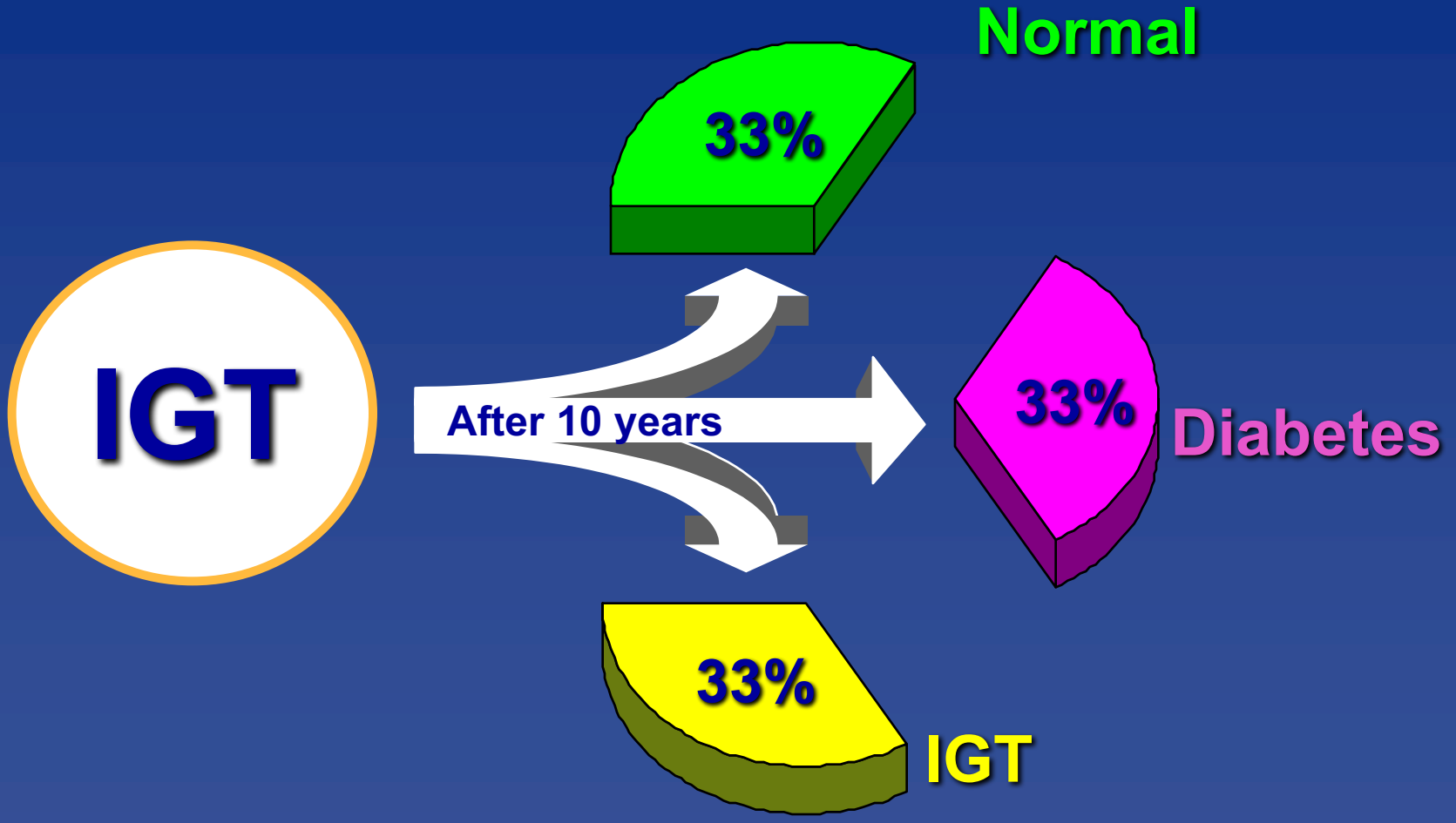
Impaired glucose tolerance IGT
Impaired fasting glucose IFG

- **They are known risk factors for future diabetes and cardiovascular disease.**
- **Intermediate stage for all types of diabetes.**
- **Associated with insulin resistance syndrome or :**

Syndrome X :

Insulin resistance
Hyperinsulinemia
Obesity
Dyslipidemia (high triglyceride and/or low HDL)
Hypertension

Natural History of IGT



Etiologic classification of diabetes mellitus

I- Type 1 diabetes:

II- Type 2 diabetes.

III- Other specific types.

IV- Gestational diabetes mellitus.

3

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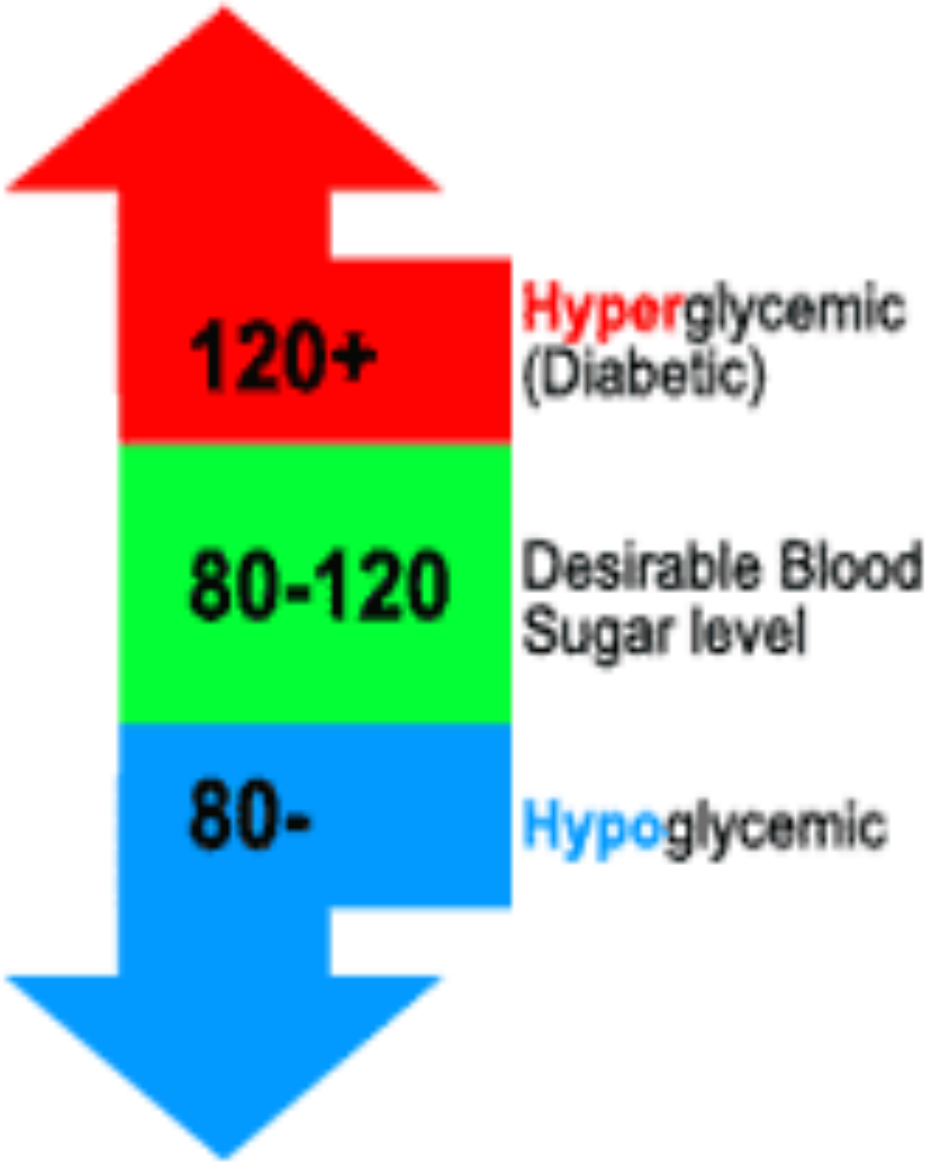
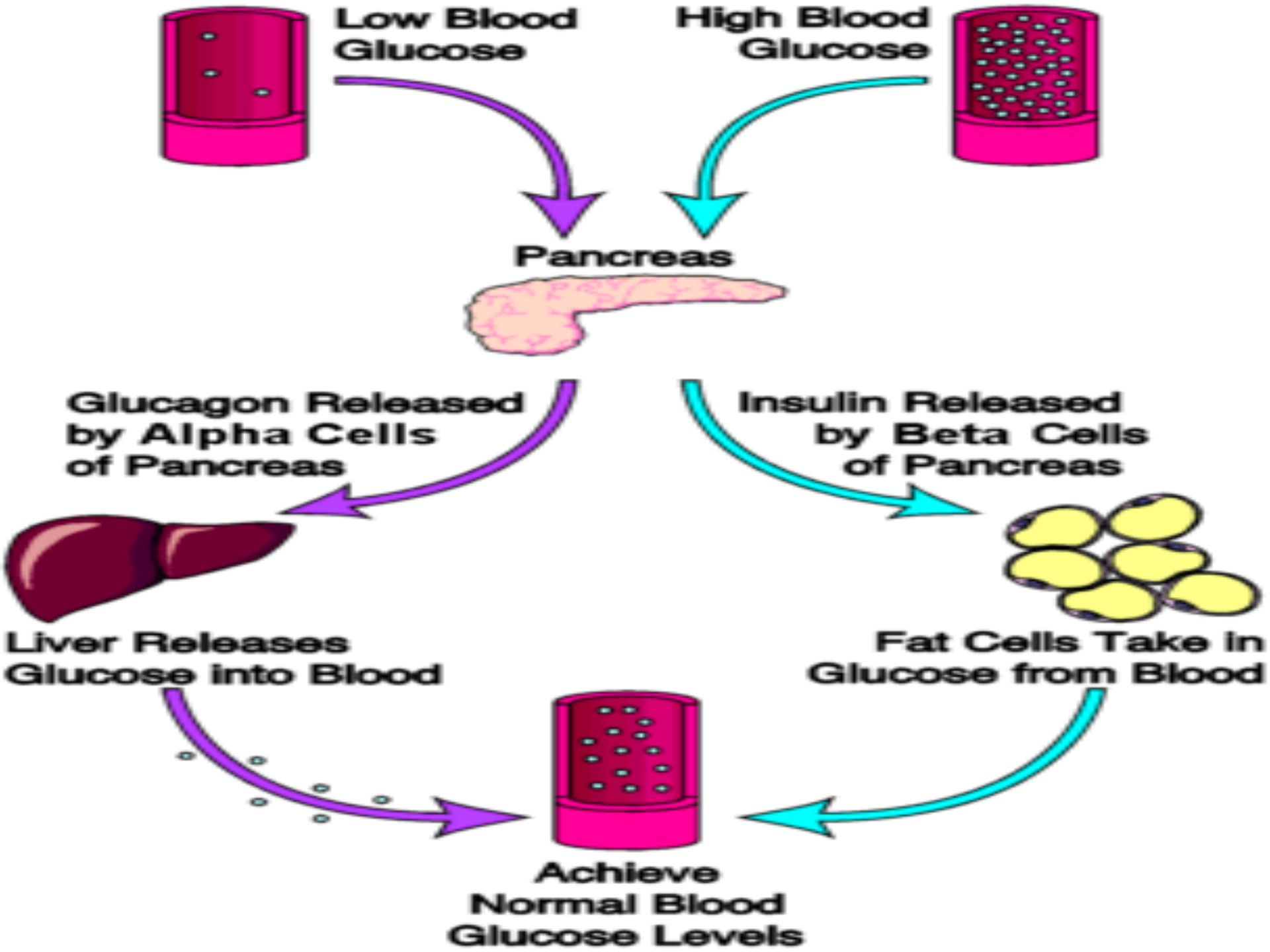
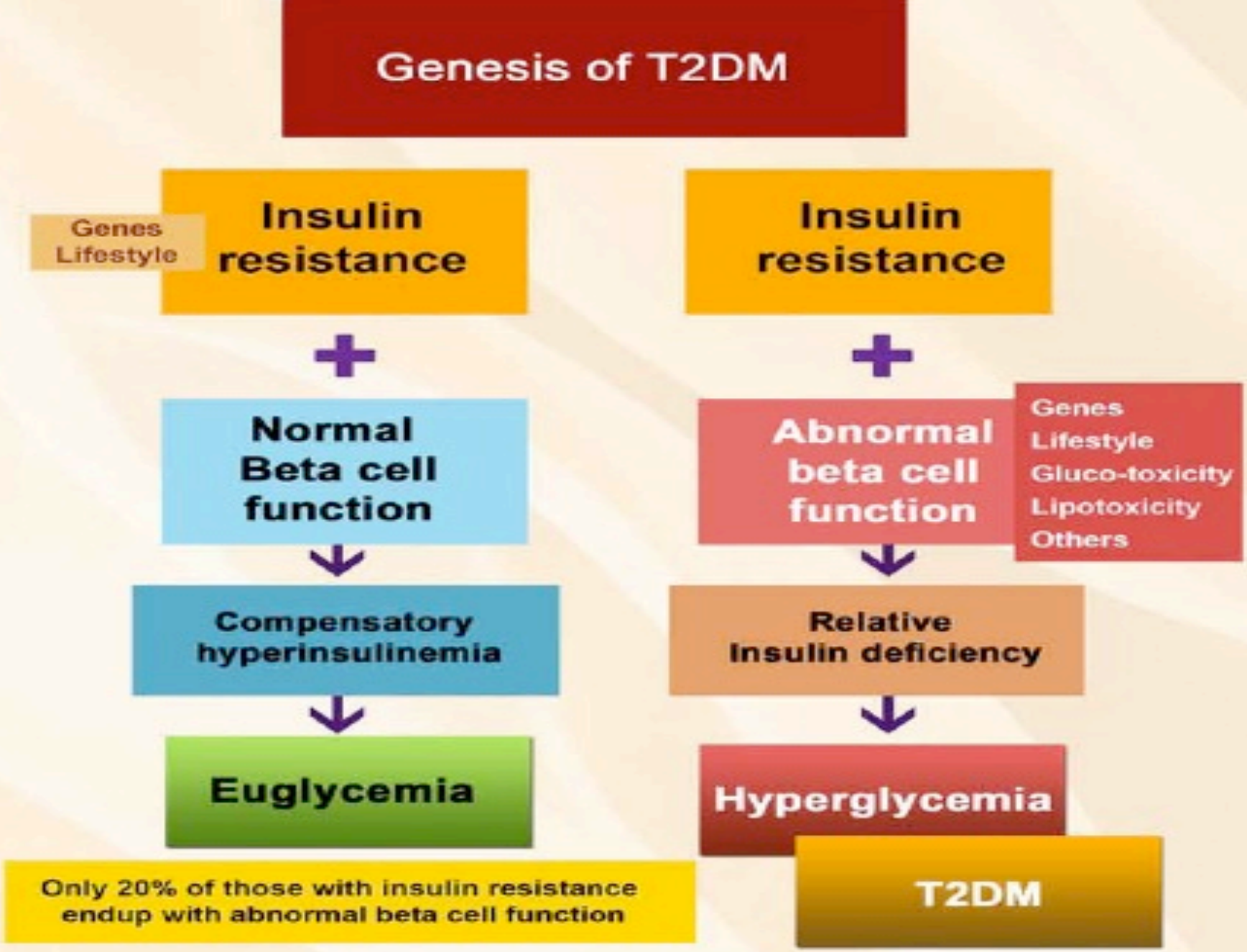
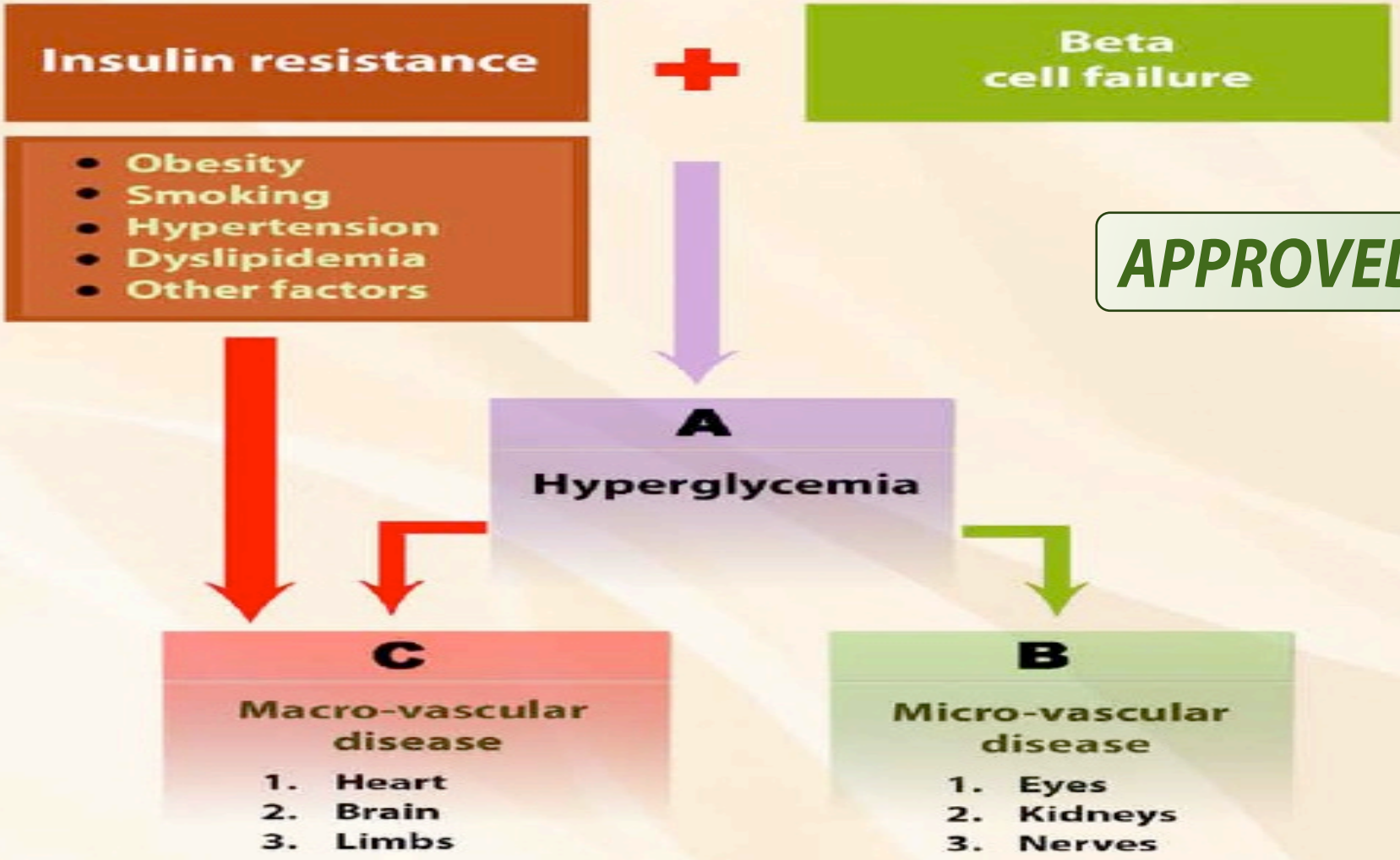

120+
Hyperglycemic
(Diabetic)
80-120
Desirable Blood
Sugar level
80-
Hypoglycemic


Low Blood Glucose
High Blood Glucose
Pancreas
Glucagon Released by Alpha Cells of Pancreas
Insulin Released by Beta Cells of Pancreas
Liver Releases Glucose into Blood
Fat Cells Take in Glucose from Blood
Achieve Normal Blood Glucose Levels

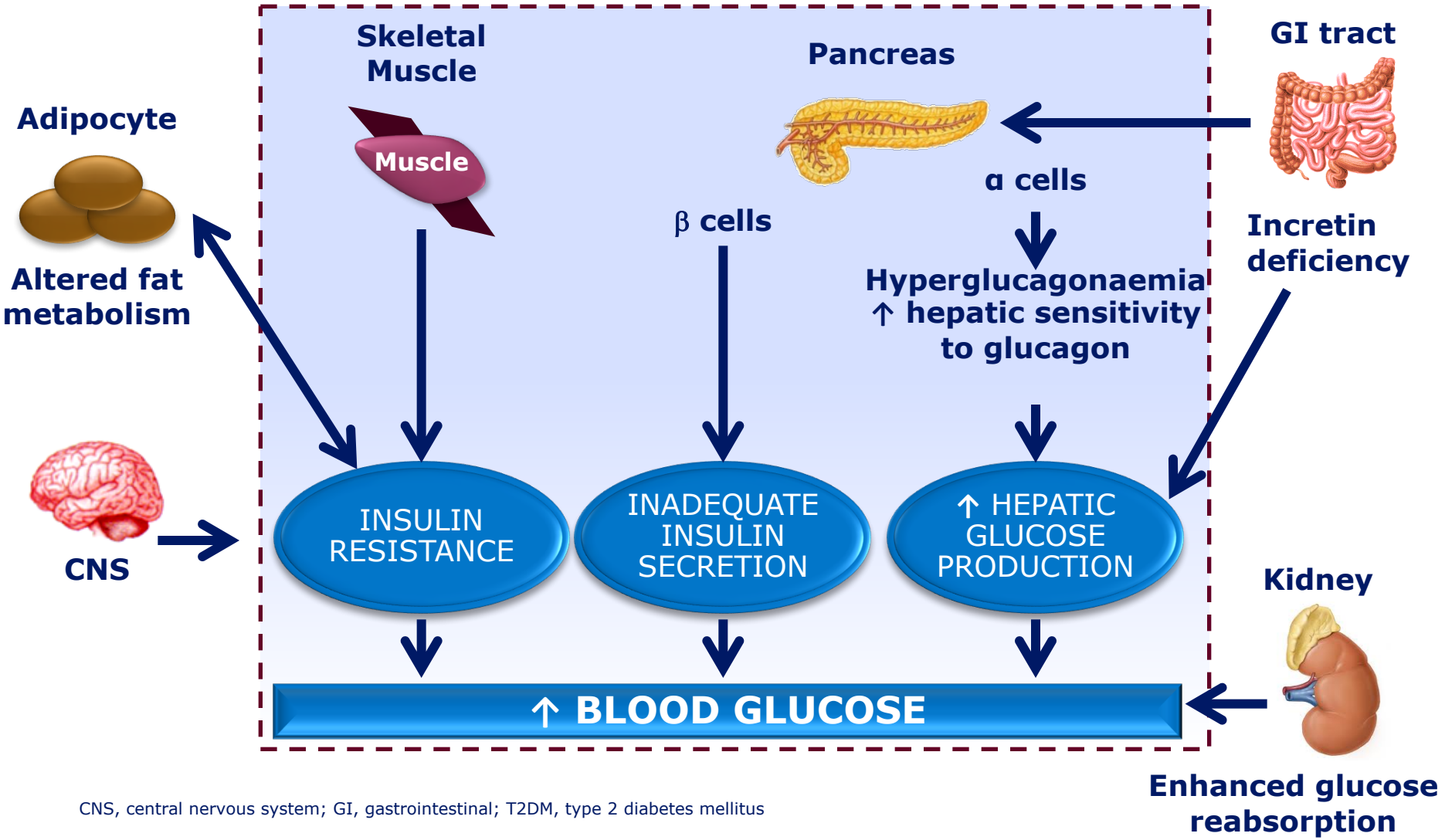


T2DM at a glance



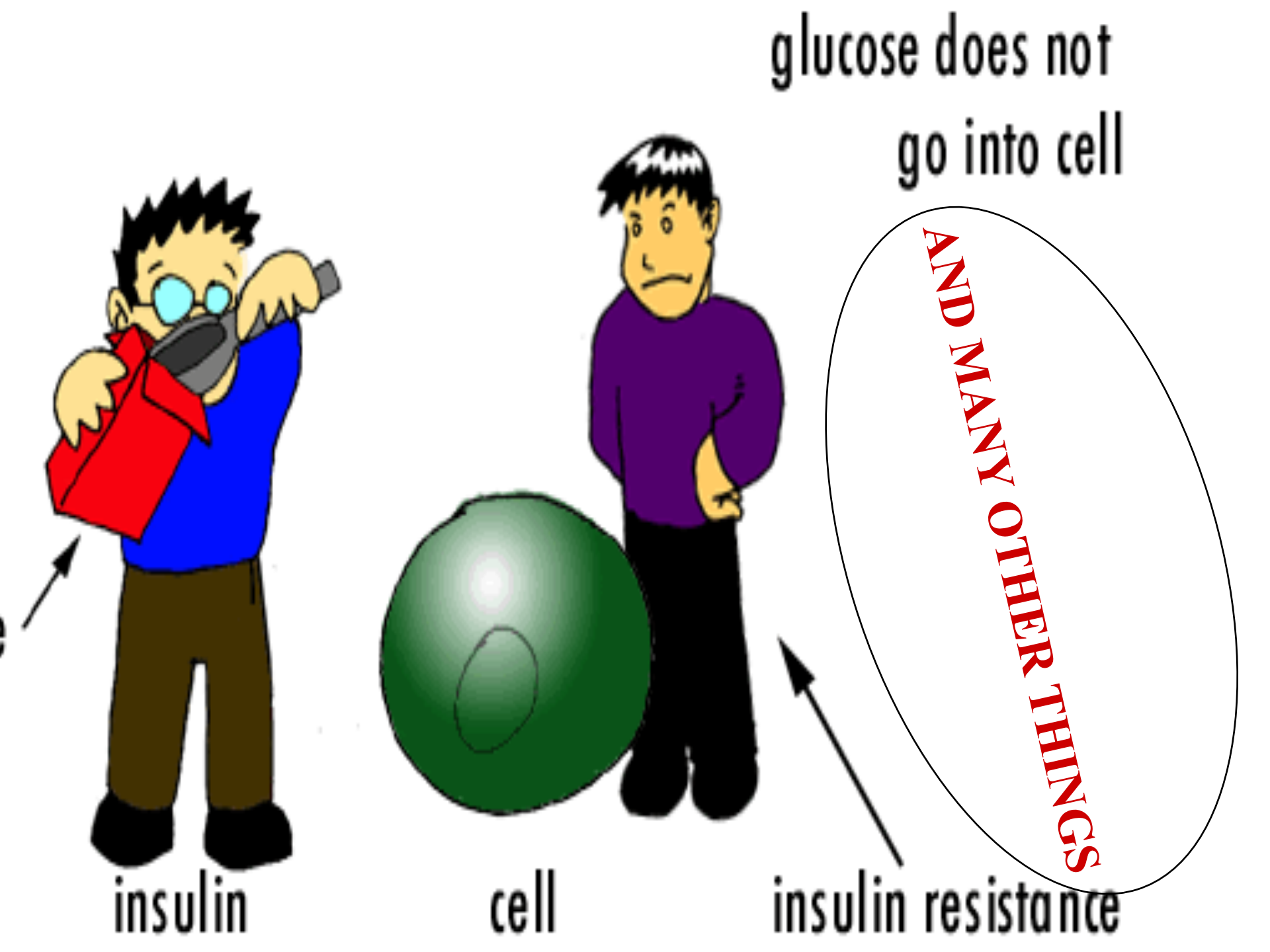
See ABC of T2DM care next

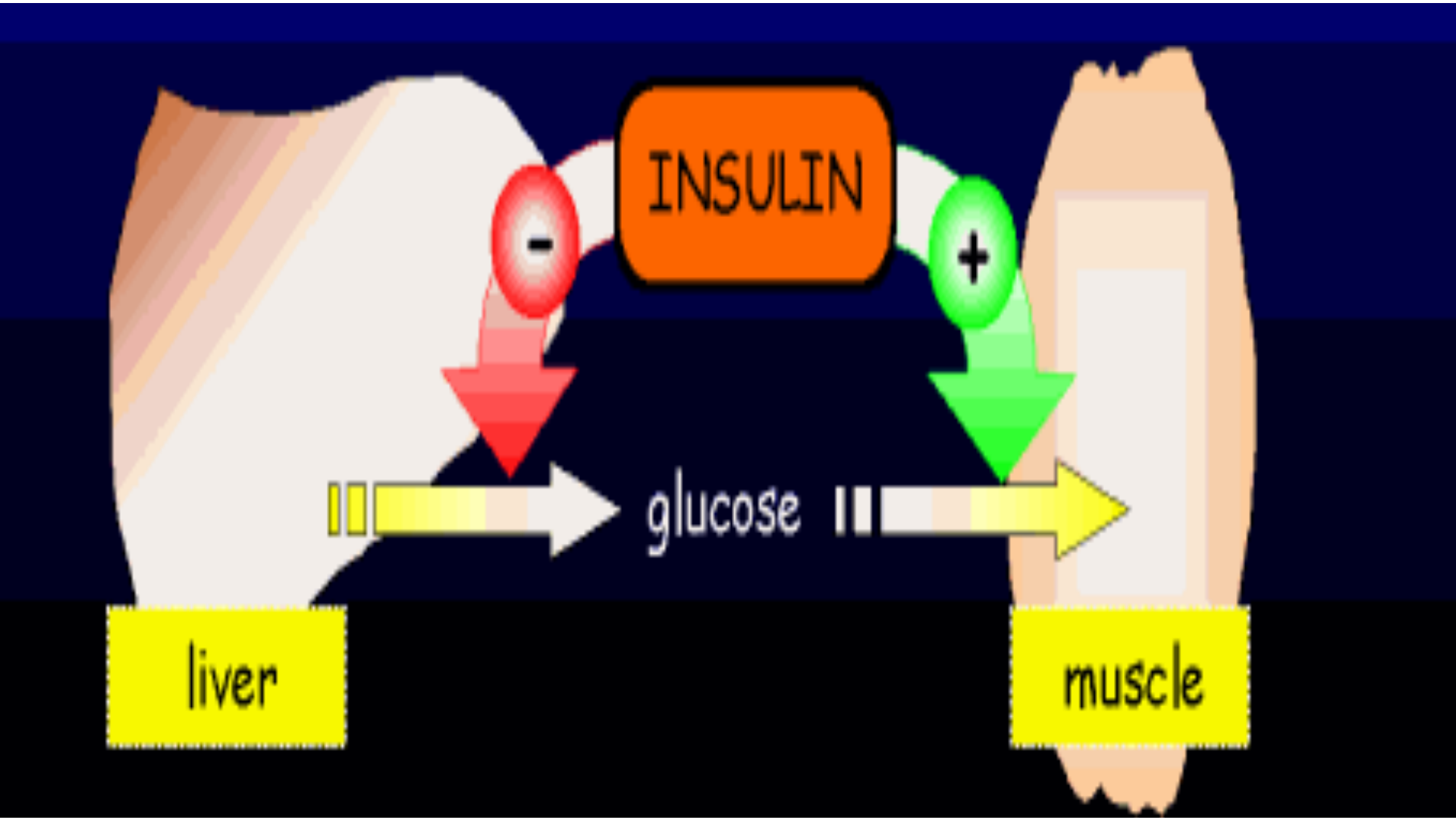
Pathophysiology of type 2 diabetes



CNS, central nervous system; GI, gastrointestinal; T2DM, type 2 diabetes mellitus

Cernea S & Raz I. *Diabetes Care* 2011;34(suppl 2):S264–S271

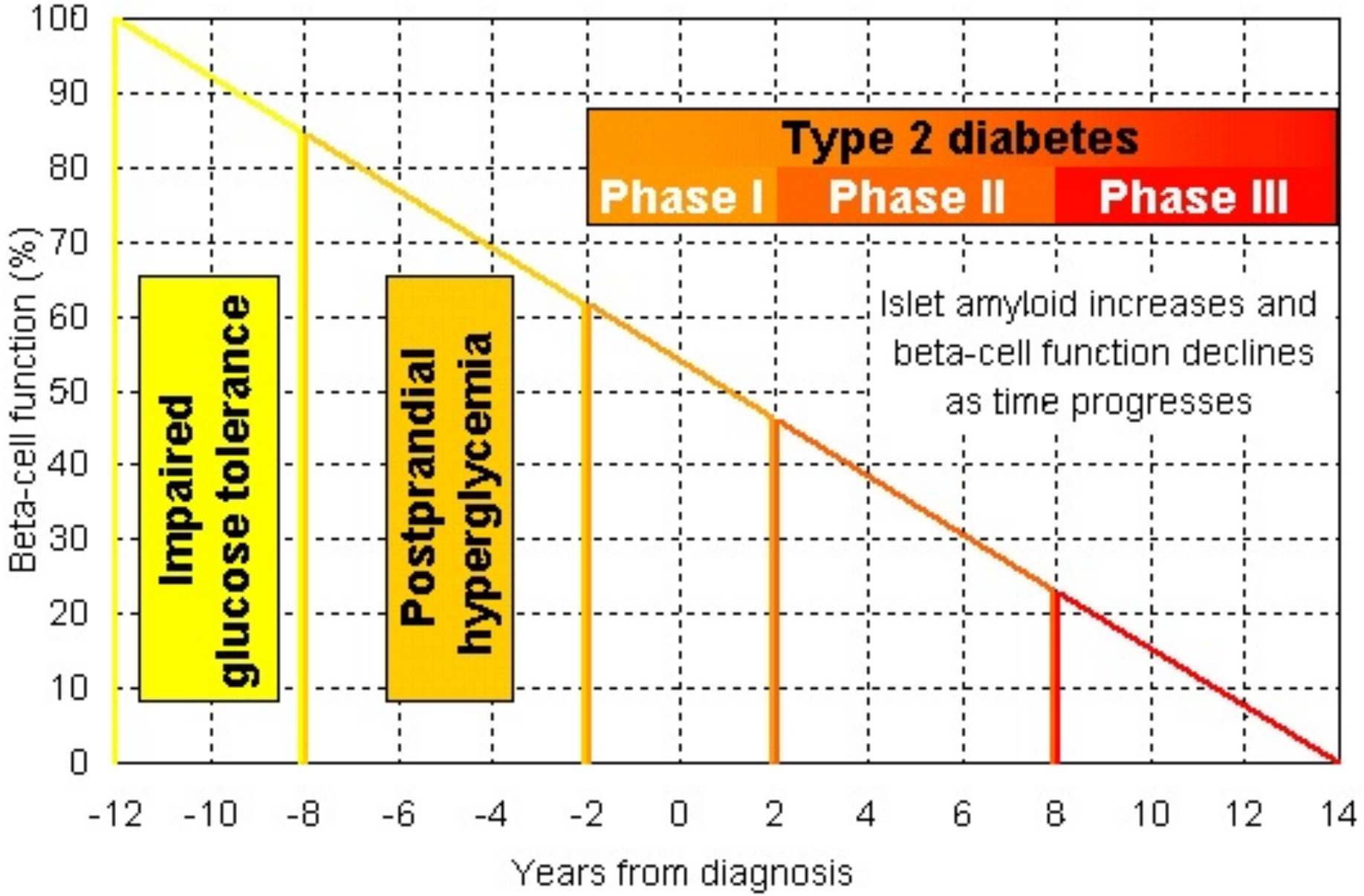

glucose does not
go into cell
AND MANY OTHER THINGS
insulin
cell
insulin resistance


INSULIN
-
+
glucose
liver
muscle

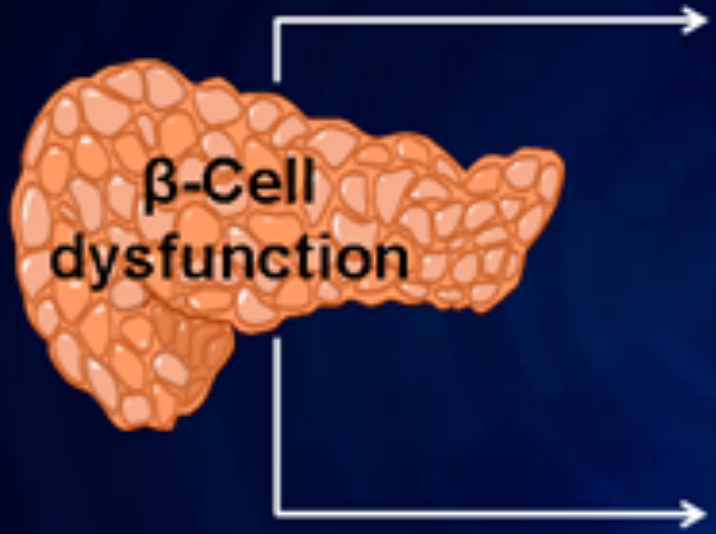
TNF-alpha may induce insulin resistance in obesity

The cytokine tumour necrosis factor-α (TNF-α) is produced from adipose tissue, and TNF-α levels are often elevated in obesity.

- Administration of TNF-α leads to insulin resistance.
- Over-expression of TNF-α in adipose and muscle of obese, insulin resistant diabetic subjects is positively correlated with insulin resistance.
- Polymorphisms at the TNF-α locus correlate with insulin resistance.
- TNF-α inhibits insulin receptor signalling in adipocytes.
- TNF-α deficiency (knockout mice) prevents diet-induced insulin resistance.


Type 2 diabetes
Phase I
Phase II
Phase III
Impaired glucose tolerance
Postprandial hyperglycemia
Islet amyloid increases and beta-cell function declines as time progresses
Beta-cell function (%)
100
90
80
70
60
50
40
30
20
10
0
-12
-10
-8
-6
-4
-2
0
2
4
6
8
10
12
14
Years from diagnosis

Abnormalities of β-Cell Function in Type 2 Diabetes



- Disrupted pulsatile insulin response[1]
- ↓ First phase
- ↑ Proinsulin/insulin ratio[1]
- ↓ β-cell responsiveness to glucose[2,3]
- ↓ Insulin production[4]
  - ↓ insulin
  - ↓ insulin granules

1. Buchanan TA. *Clin Ther.* 2003;25(suppl B):B32-B46. 2. Buse JB et al. In: Larsen PR et al. *Williams Textbook of Endocrinology*. 10th ed. Saunders; 2003;1427-1483. 3. Ward WK et al. *J Clin Invest.* 1984;74:1318-1328.
4. Marchetti P et al. *J Clin Endocrinol Metab.* 2004;89:5535-5541.

4

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Patients with type 2 diabetes are at heightened risk of disabling complications versus non-diabetics

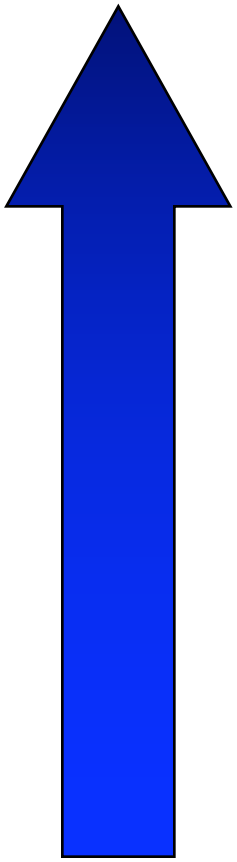
Complication	Relative Risk*
Blindness	↑ 20
End-stage renal disease	↑ 25
Amputation	↑ 40
Myocardial Infarction	↑ 2-5
Stroke	↑ 2-3
Lifespan	↓ 6 years

* Diabetics versus non-diabetics

Ryden L. Eur Heart J. 2007; 28: 88-136

Burden of Type 2 Diabetes

15 Years Follow-up



100 Patients - Age 55

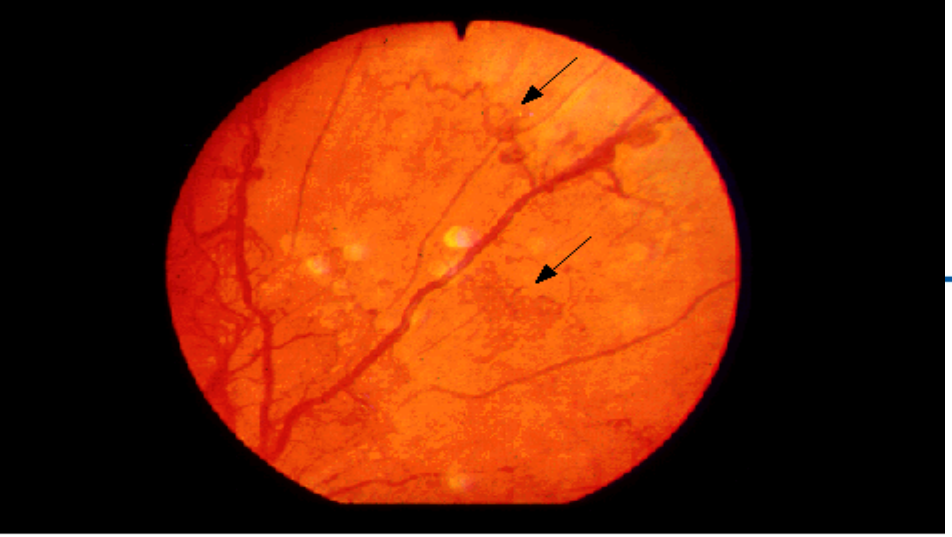
Complications

Heart attacks	27 patients
Stroke	10 patients
Retinopathy	23 patients

Premature Mortality

Diabetes deaths	28 patients
Life expectancy	5-7 years ↓

UKPDS 33. Lancet 1998; 352: 837-53



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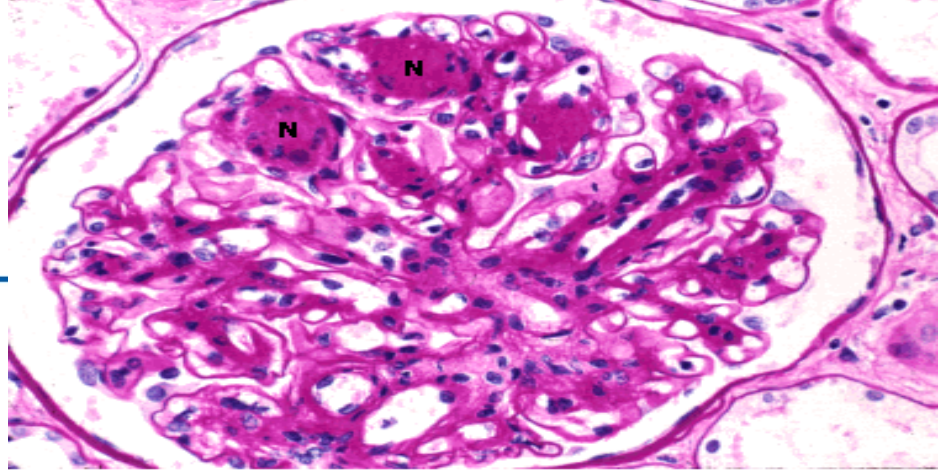
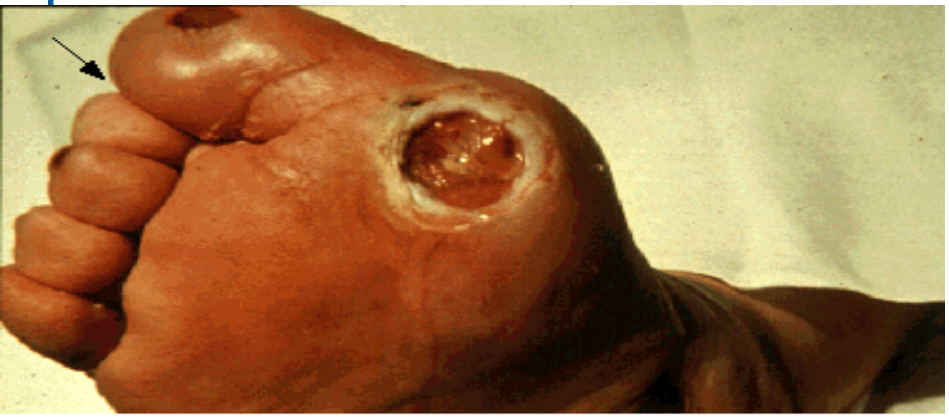


Diabetic nephropathy Light micrograph showing diffuse and nodular (N) glomerulosclerosis in diabetic nephropathy. Note the dense appearance of the deposits and the rim of cells around the nodules, which distinguish this disorder on light microscopy from fibrillary glomerulonephritis or amyloidosis. Courtesy of Helmut Rennke, MD.



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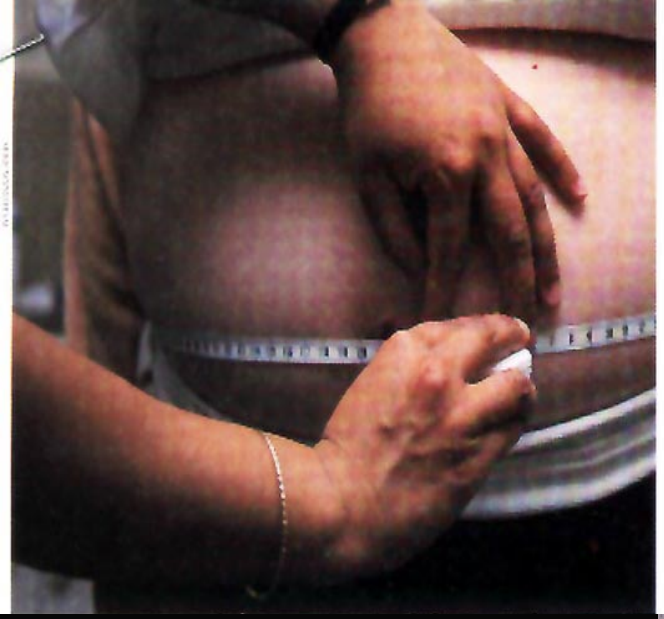
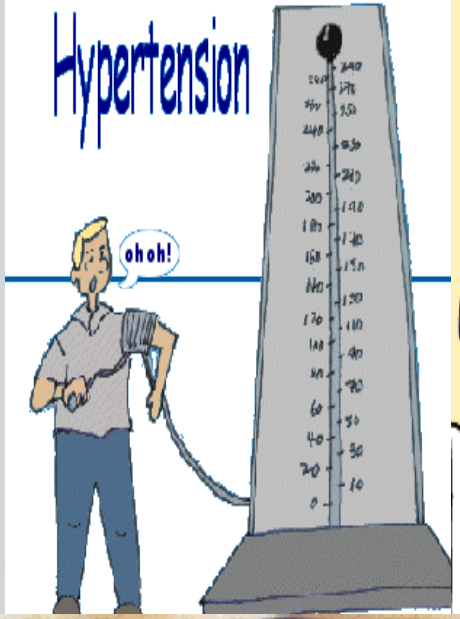
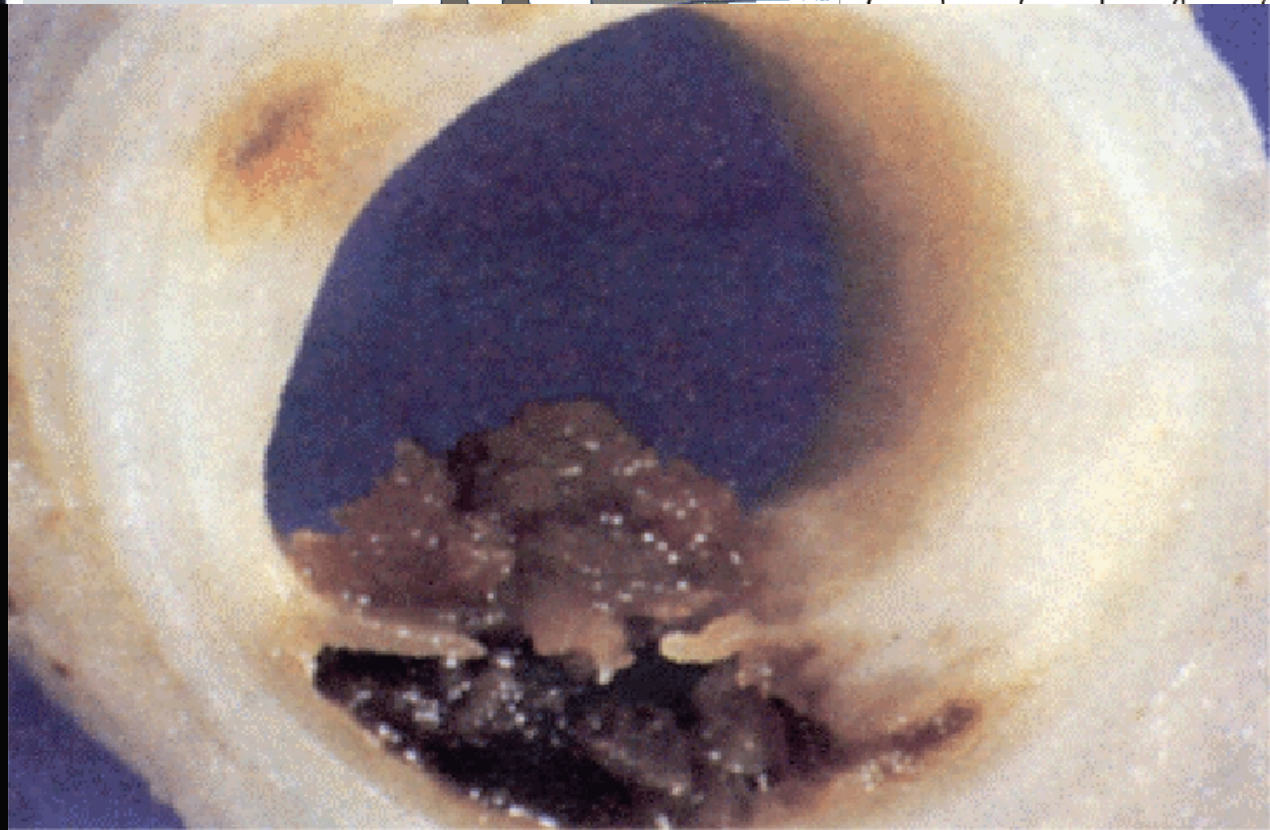



Hypertension
oh oh!


Cholesterol
MILK



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The ABC of T2DM care

Glycemic control is important.
Non glycemic factors are even more important towards outcome

- Glycemic control
- HbA1c/HBGM
- Hypos / illness
- Work / leisure
- Special events

Glucose **A**

- Kidneys
- Eyes
- Feet
- Nerves
- Erectile Dysfunction

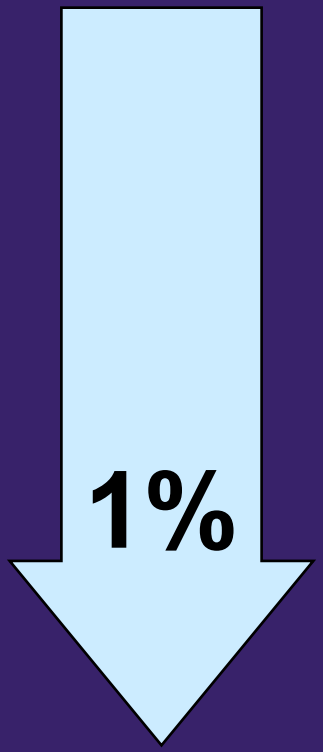
Micro vascular **B**

- BP
- Lipids
- Aspirin
- Smoking cessation
- Diet / Exercise

Macro vascular **C**

Lessons from UKPDS: Better control means fewer complications

EVERY 1% reduction in HBA_{1C}

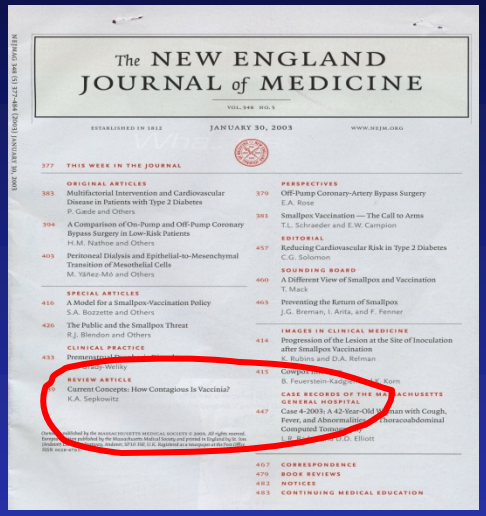


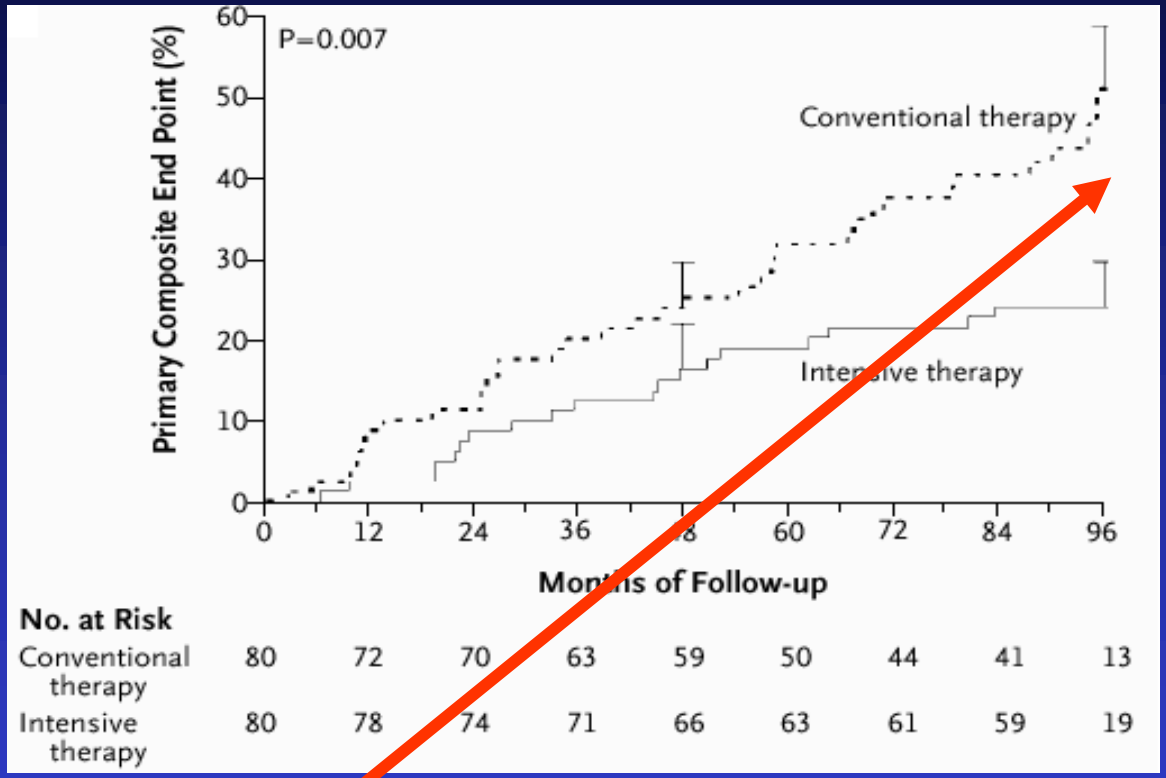
	REDUCED RISK*
Deaths from diabetes	-21%
Heart attacks	-14%
Microvascular complications	-37%
Peripheral vascular disorders	-43%

*p<0.0001

UKPDS 35. BMJ 2000; 321: 405-12

Benefits of intervention to reduce multiple risk factors – Danish Steno 2 Study





53% Risk Reduction with Intensive Therapy

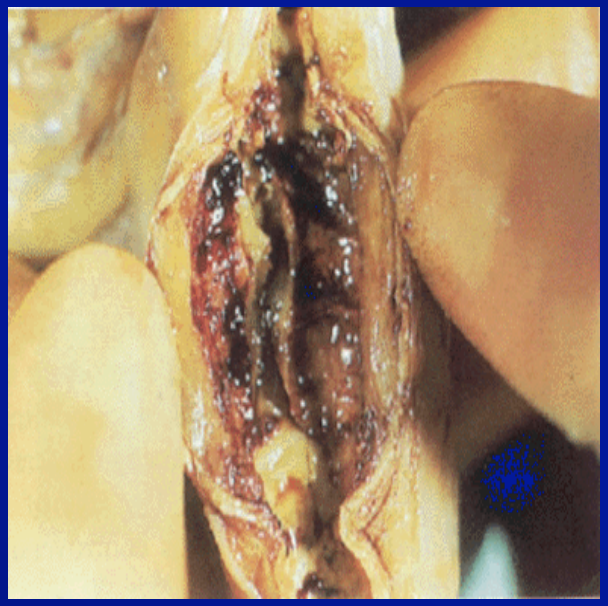
Gaede P et al. NEJM 2003; 348: 383-393
Gaede P et al. NEJM 2008; 358: 580-591

UKPDS: Clinical Outcomes

Diabetes Deaths

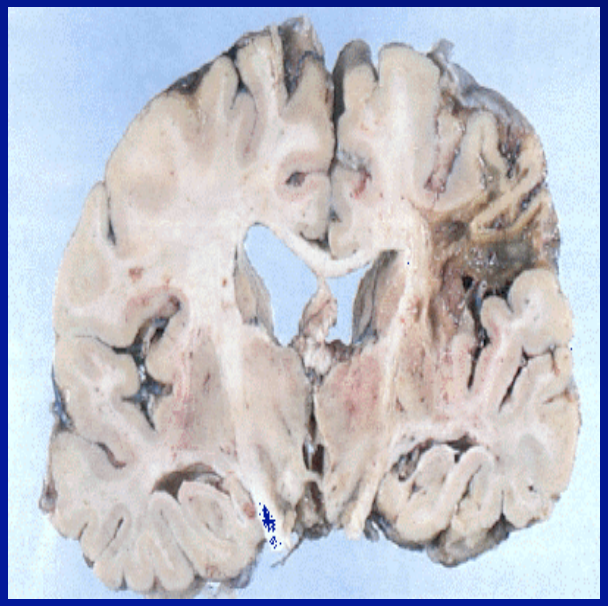
↓ 42%
Reduction

Heart Attack



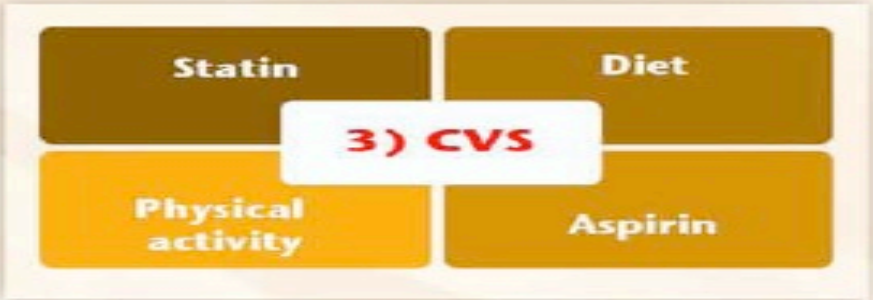
↓ 39%
Reduction

Stroke



↓ 41%
Reduction

T2DM: 2nd FU OPD visit
Consolidation of therapy



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ASPIRIN
GET WELL!
GREAT BIG STUFF


Cholesterol
MIL

Lessons from major trials

1. **DM complications are present at diagnosis**
2. **DM complications progress with time**
3. **DM control predicts rate and state of complications**
4. **Early and sustained control limits complications**
5. **Management is multifaceted and complex**
6. **Majority of patients are NOT at target**

Guidelines need to address all concerns

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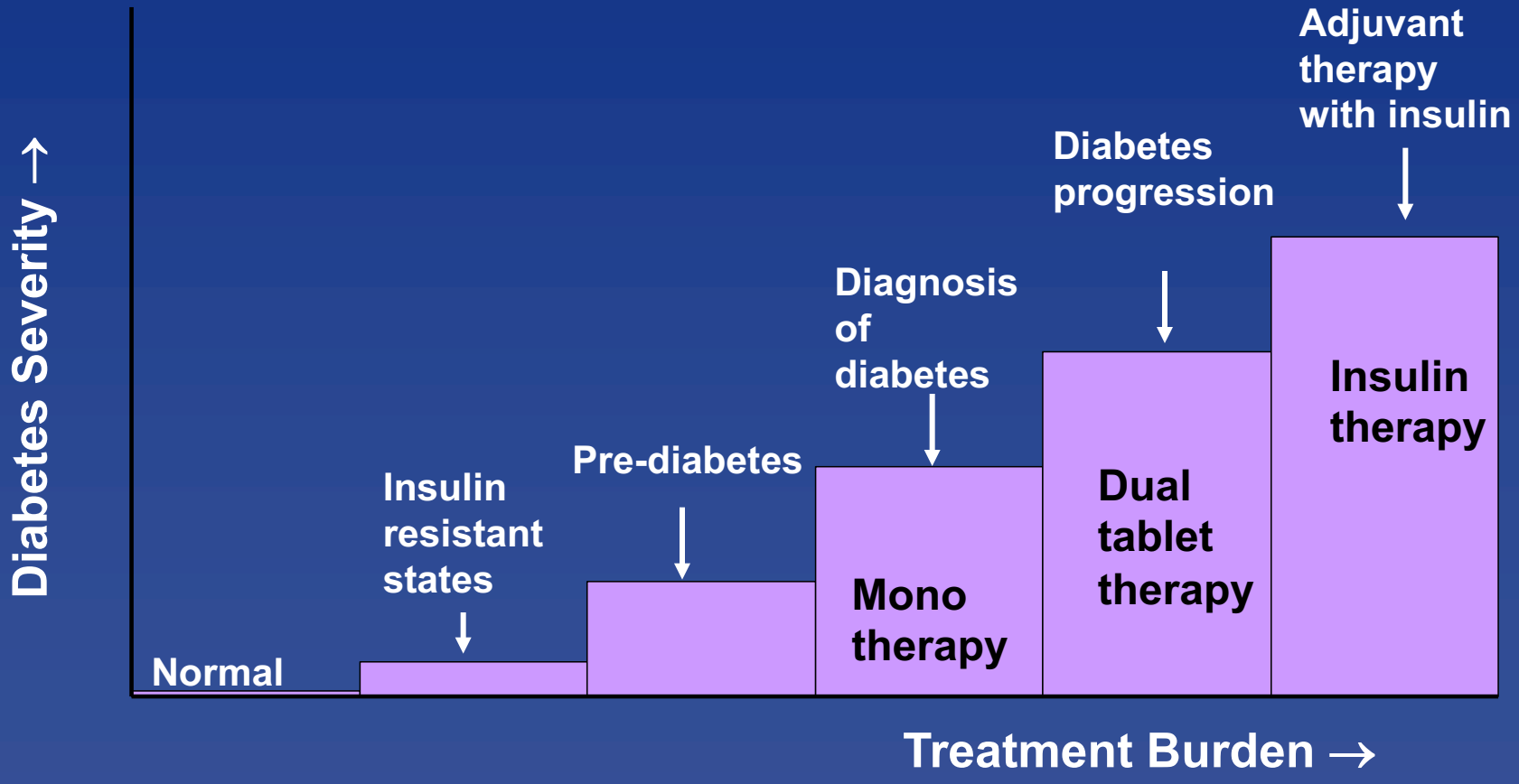
Targets

1. HgbA1c: 7% Early Young No AT
2. HgbA1c: 8% Late Old W AT

Progressive nature of diabetes means stepwise increase in therapies to achieve optimal outcomes in management



Glycemic Control Algorithm

LIFESTYLE MODIFICATION
(Including Medically Assisted Weight Loss)

ENTRY A1c < 7.5%

MONOTHERAPY*
- Metformin
- GLP-1 RA
- DPP4-i
- AG-i
- SGLT-2 **
- TZD
- SU/GLN

If A1c > 6.5% in 3 months add second drug (Dual Therapy)

ENTRY A1c ≥ 7.5%

DUAL THERAPY*

MET or other first-line agent +
- GLP-1 RA
- DPP4-i
- TZD
- ** SGLT-2
- Basal insulin
- Colesevelam
- Bromocriptine QR
- AG-i
- SU/GLN

If not at goal in 3 months proceed to triple therapy

TRIPLE THERAPY*

MET or other first-line agent + 2ND LINE AGENT
- GLP-1 RA
- TZD
- ** SGLT-2
- Basal insulin
- DPP4-i
- Colesevelam
- Bromocriptine QR
- AG-i
- SU/GLN

If not at goal in 3 months proceed to or intensify insulin therapy

ENTRY A1c > 9.0%

NO SYMPTOMS
- DUAL THERAPY
- OR
- TRIPLE THERAPY

SYMPTOMS
- INSULIN ± OTHER AGENTS

ADD OR INTENSIFY INSULIN

LEGEND
- = Few adverse events or possible benefits
- = Use with caution

* Order of medications listed are a suggested hierarchy of usage

** Based upon phase 3 clinical trials data

PROGRESSION OF DISEASE

PATHOPHYSIOLOGIC-BASED (DEFRONZO) ALGORITHM

Lifestyle +
TRIPLE COMBINATION:

Metformin + PIO + GLP-1 Analogue

↓

$HbA_{1c} < 6.0\%$

CONSENSUS

Saudi Arabia, Kuwait, Egypt, Lebanon, UAE, Jordan

Optimising the medical management of hyperglycaemia in type 2 diabetes in the Middle East: pivotal role of metformin

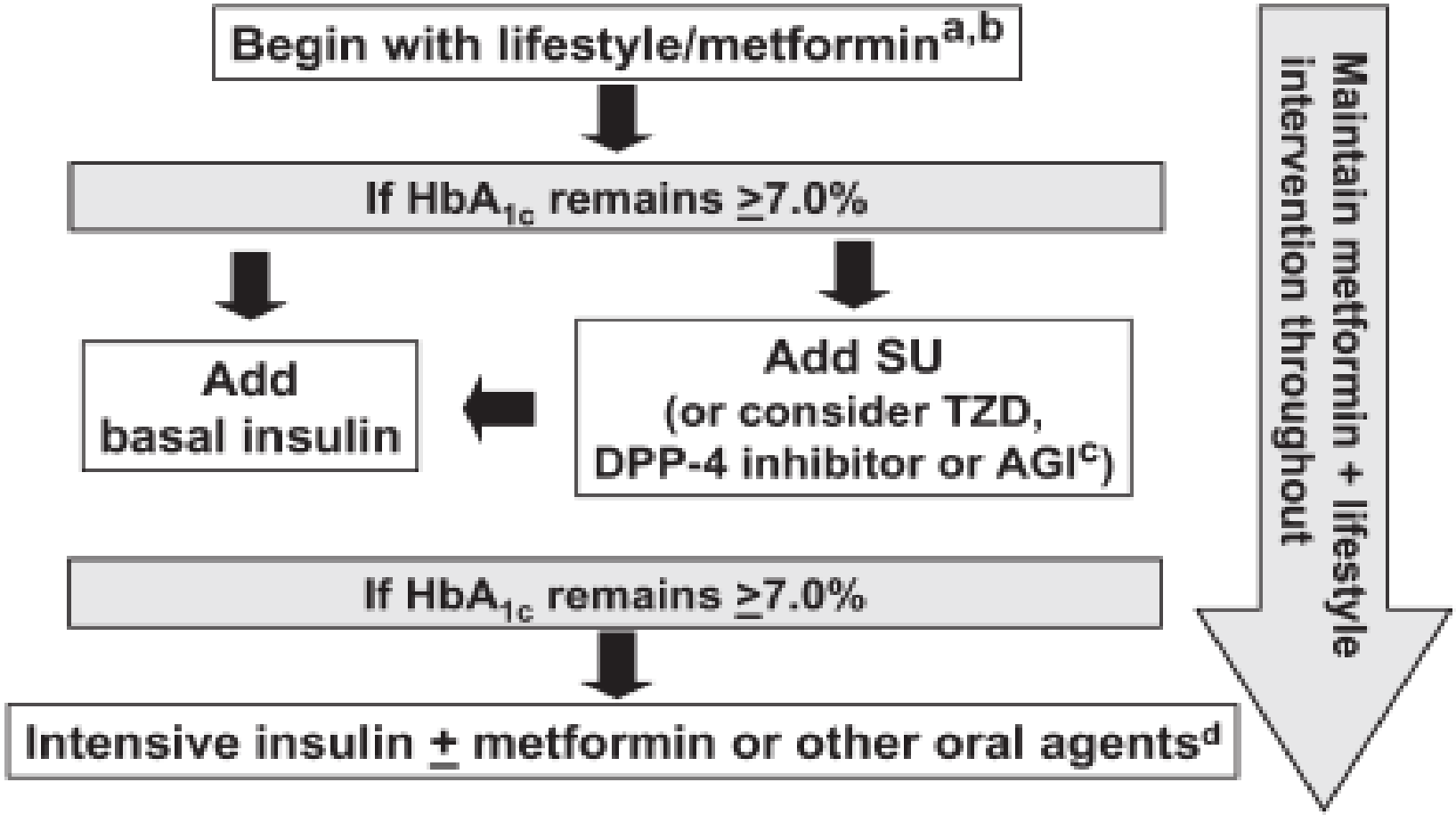
M. Al-Maatouq,[1] M. Al-Arouj,[2] S. H. Assaad,[3] S. N. Assaad,[3] S. T. Azar,[4] A. A. K. Hassoun,[5] N. Jarrah,[6] S. Zatari,[7] K. G. M. M. Alberti[8]

The burden of diabetes is high in the Middle East

The Middle East has largely been overlooked by guideline writers

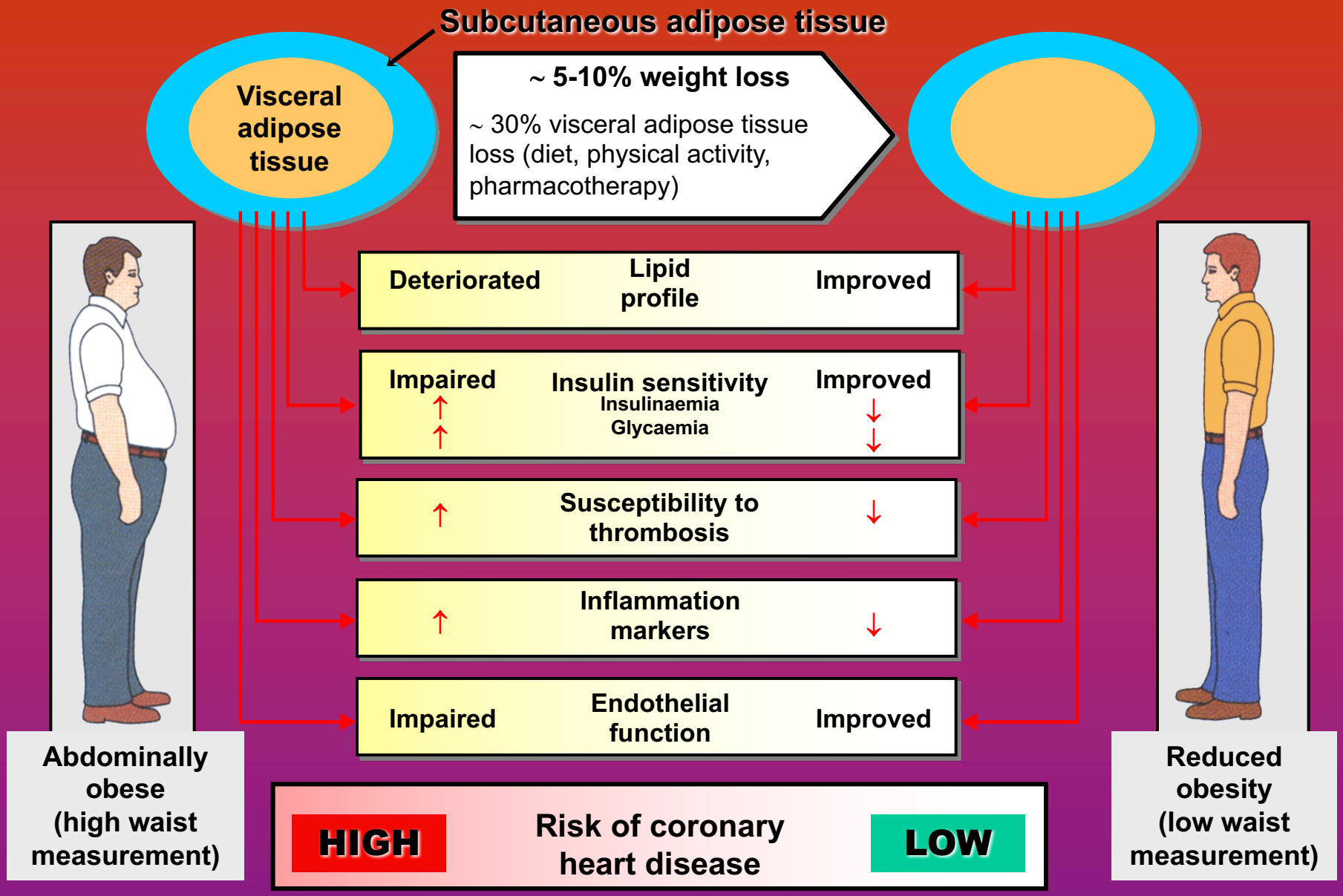
Al-Maatouq M. Int J Clin Pract 2010; 64: 149-159

Recommended treatment algorithm for the Middle East



AlMaatouq MA. Int J Clin Practice 2010; 64: 149-159

Potential benefits of moderate (5-10%) weight loss in high risk patients with the metabolic syndrome



J P Després. BMJ 2001; 322:716-720

Practical dietary advice

1. Salad: 1hour BEFORE the meal

2. ¼ - ½ what you are used to. No cheating.

3. 1 Fruit per meal (juice is fruit)

4. 2 DATES BID (1 extra date BID)

5. No Communal eating

6. Avoid what you can live without.

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next

Make Your Diabetic Patients Walk

Long-term impact of different amounts of physical activity on type 2 diabetes

Chiara Di Loreto, MD
Carmine Fanelli, MD
Paola Lucidi, MD
Giuseppe Murdolo, MD
Arianna De Cicco, MD
Natascia Parlanti, MD
Anna Ranchelli, MD
Cristina Fatone, MD
Chiara Taglioni, MD
Fausto Santeusanio, MD
Pierpaolo De Feo, MD

Western and developing countries face two serious health problems: the rising prevalence of obesity and diabetes and the fact that people no longer need to be physically active in their daily lives (1–4). Many studies

- T2D
- n = 182
- 2 year follow up
- HbA1c baseline: 7.6%
- Diabetes duration: 7.6 years
- Advice for physical activity: moderate, aerobic endurance training (30-60% of max. HF), aim: > 10 MET/ h /wk)
- 7 visits, total of ca 2 h counseling, 1 visit every 3 Month

Di Loreto et al , Diabetes Care, 2005

Effects of physical activity in T2DM

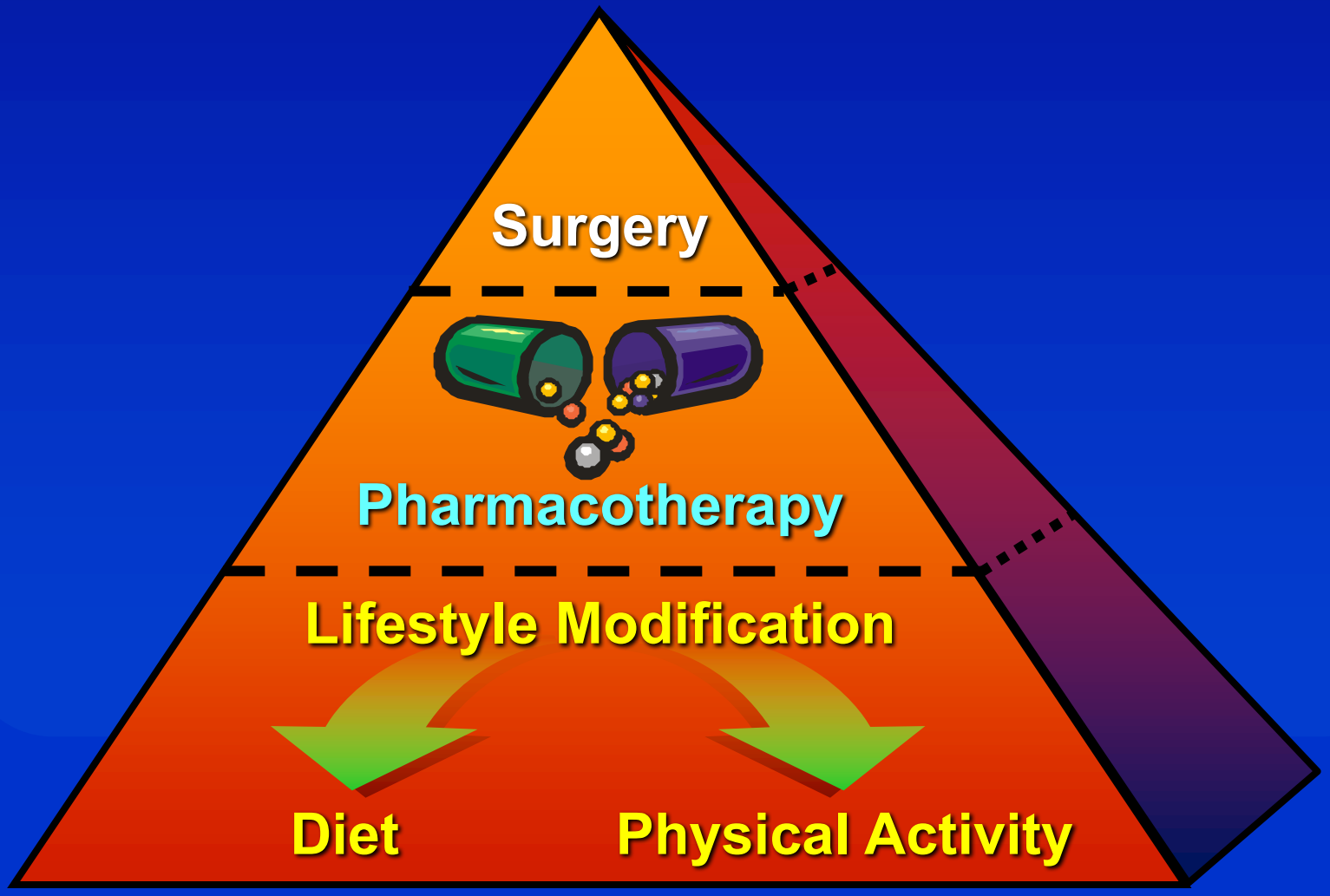
	Walking / Hours / Week*					
	0	1,5	4	5,5*	7,5	12
Weight (kg)	+ 0,8	+ 0,6	+ 0,1	- 2,2	-3,0	-3,2
Waist (cm)	+ 1,0	+ 1,0	- 0,9	- 3,8	- 5,5	- 7,1
HbA_{1c} (%)	+ 0,03	- 0,06	- 0,44	- 0,8	- 1,11	- 1,19
BD syst. (mmHg)	- 1,8	- 1,5	- 6,4	- 5,5	- 6,6	- 9,2
BD diast. (mmHg)	- 4,6	- 2,4	- 2,9	- 4,8	- 5,3	- 7,1
Chol. (mg/dl)	- 3,8	- 5,6	- 10,2	- 10,7	- 7,4	- 10,9
LDL-Chol. (mg/dl)	- 4,5	- 7,1	- 3,4	- 5,3	- 6,3	- 7,7

* e.g. 21-30 MET: 45 min walking (4 mph) /day, ca. 5 km/day)

p <0,05

Di Loreto C. et al. Diabetes Care (2005)28:1295-1302

Obesity Treatment Pyramid



Complications of Bariatric Surgery

All procedures:

- Atelectasis and pneumonia
- Deep vein thrombosis
- Pulmonary embolism
- Wound infection
- Gastrointestinal bleeding
- Gallstones
- Failure to lose weight
- Intractable vomiting/kwashiorkor (B1)
- Mortality (0.1%–2%)

Gastric bypass:

- Anastomotic leak with peritonitis
- Stomal stenosis
- Marginal ulcers
- Staple line disruption
- Nutrient deficiencies (iron, calcium, folic acid, vitamin B12)
- Dumping syndrome
- Small bowel obstruction
  - Internal hernia
  - Adhesions

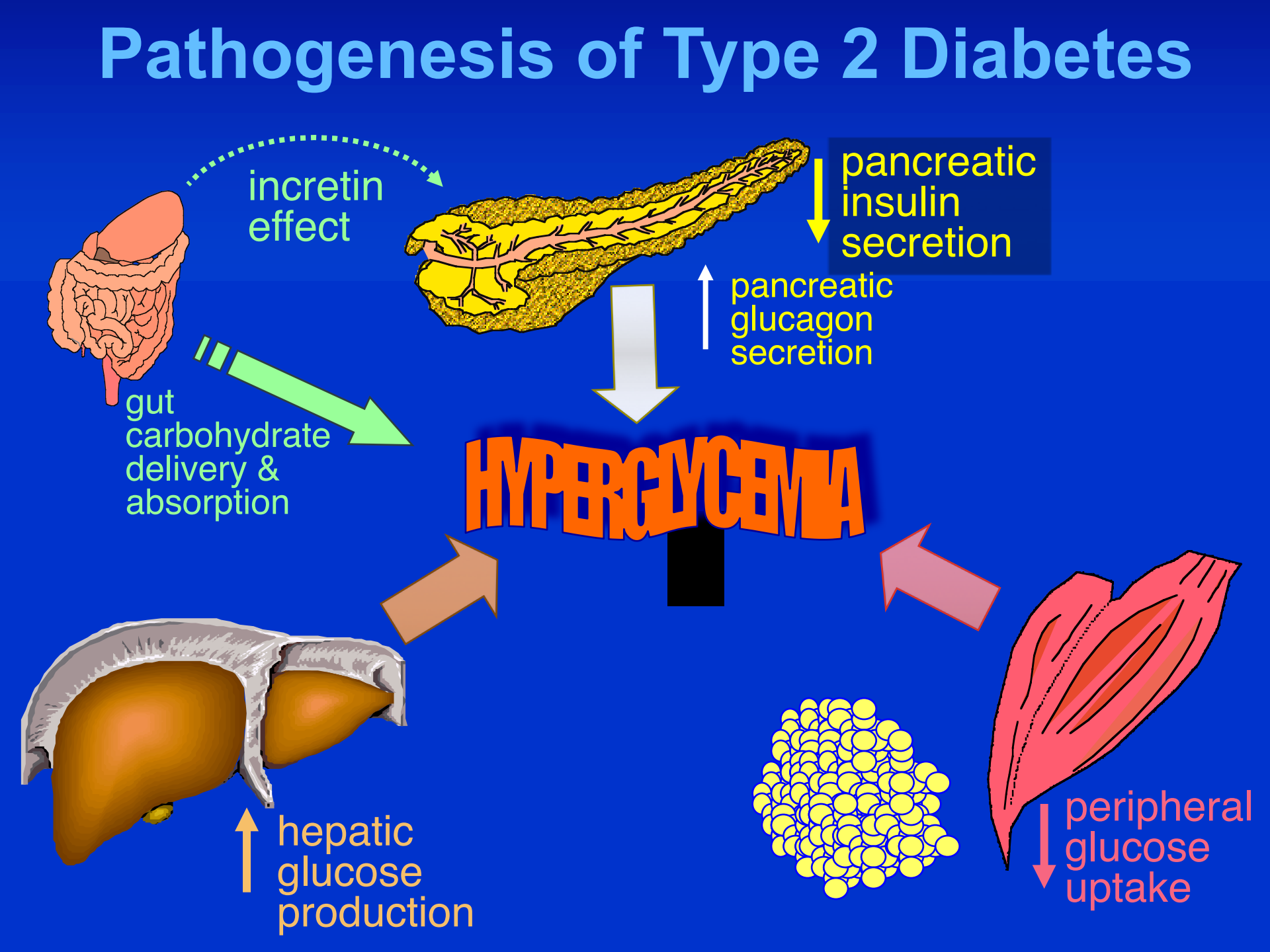
Gastric banding procedure:

- Band slippage
- Band erosion
- Esophageal dilatation
- Band or port infections
- Port disconnection
- Port displacement

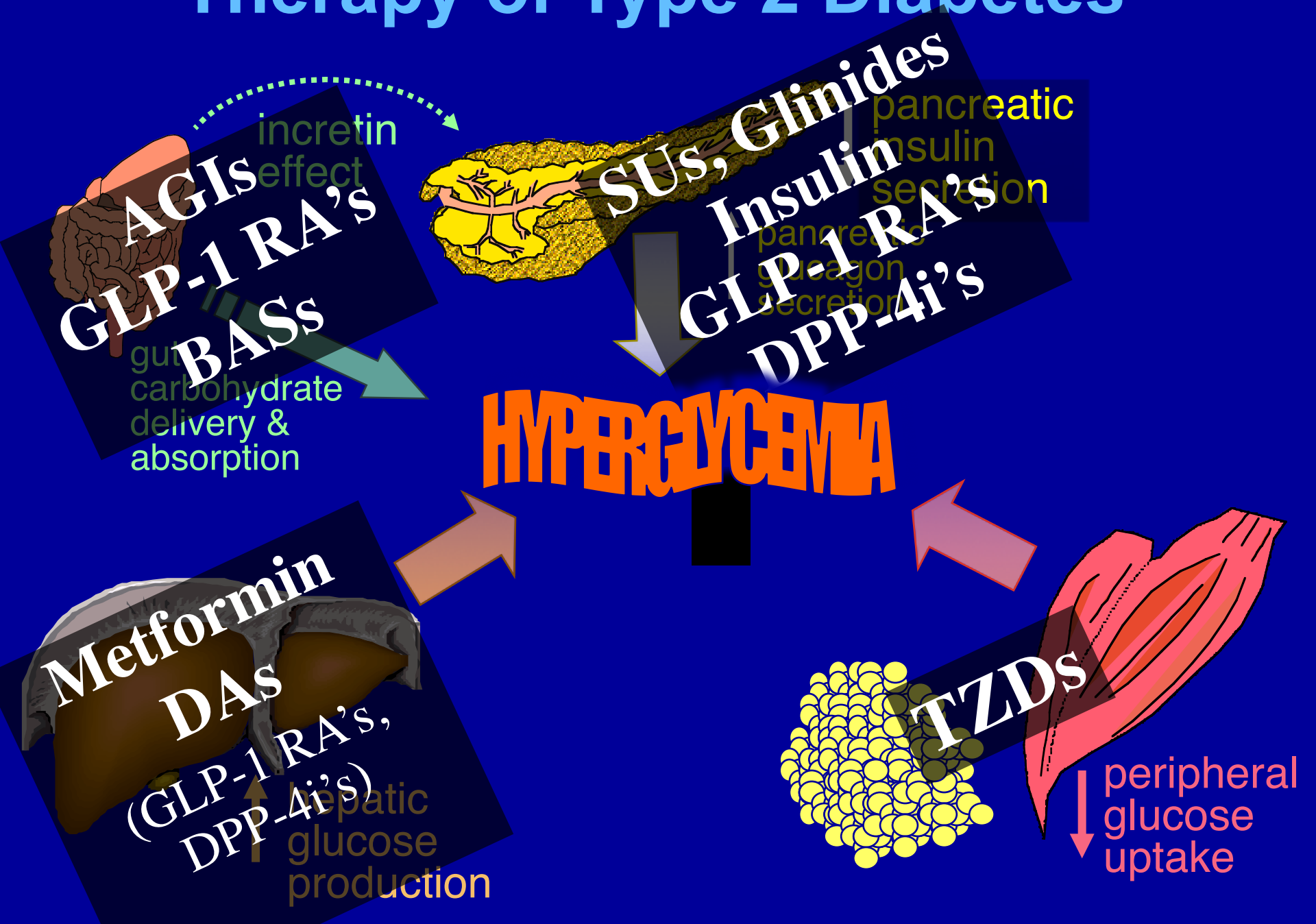
Biliopancreatic diversion:

- Anastomotic leak with peritonitis
- Protein-calorie malnutrition
- Calcium, iron, folic acid, fat soluble vitamin (A,D,E,K) deficiencies
- Dehydration
- Steatorrhea
- Small bowel obstruction
  - Internal hernia
  - Adhesions

Pathogenesis of Type 2 Diabetes

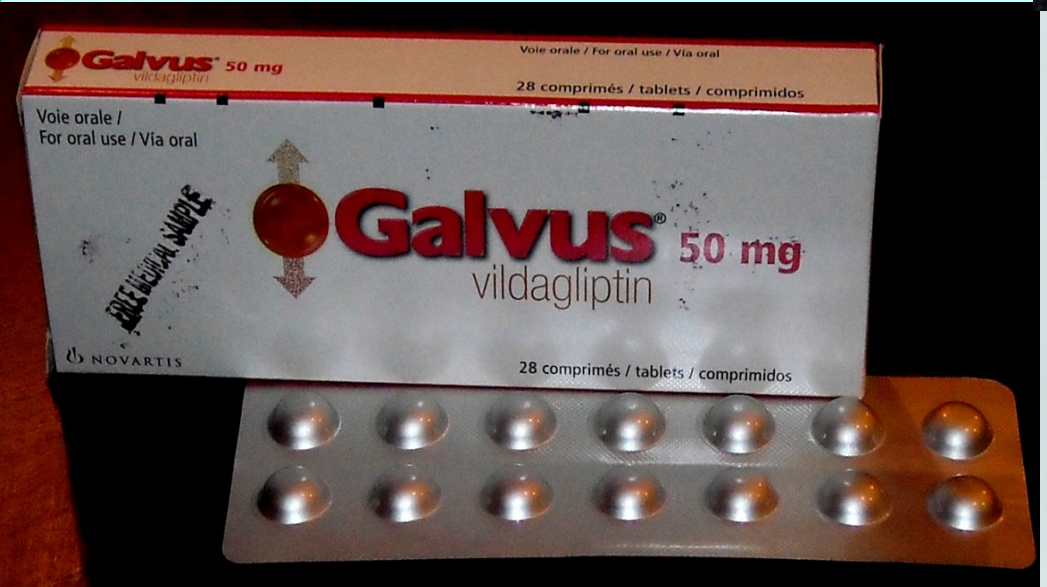


Therapy of Type 2 Diabetes



Dipeptidyl Peptidase 4 inhibitors

- Sitgliptin: Januvia
- Vildagliptin: Galvus
- Saxagliptin: Onglyza



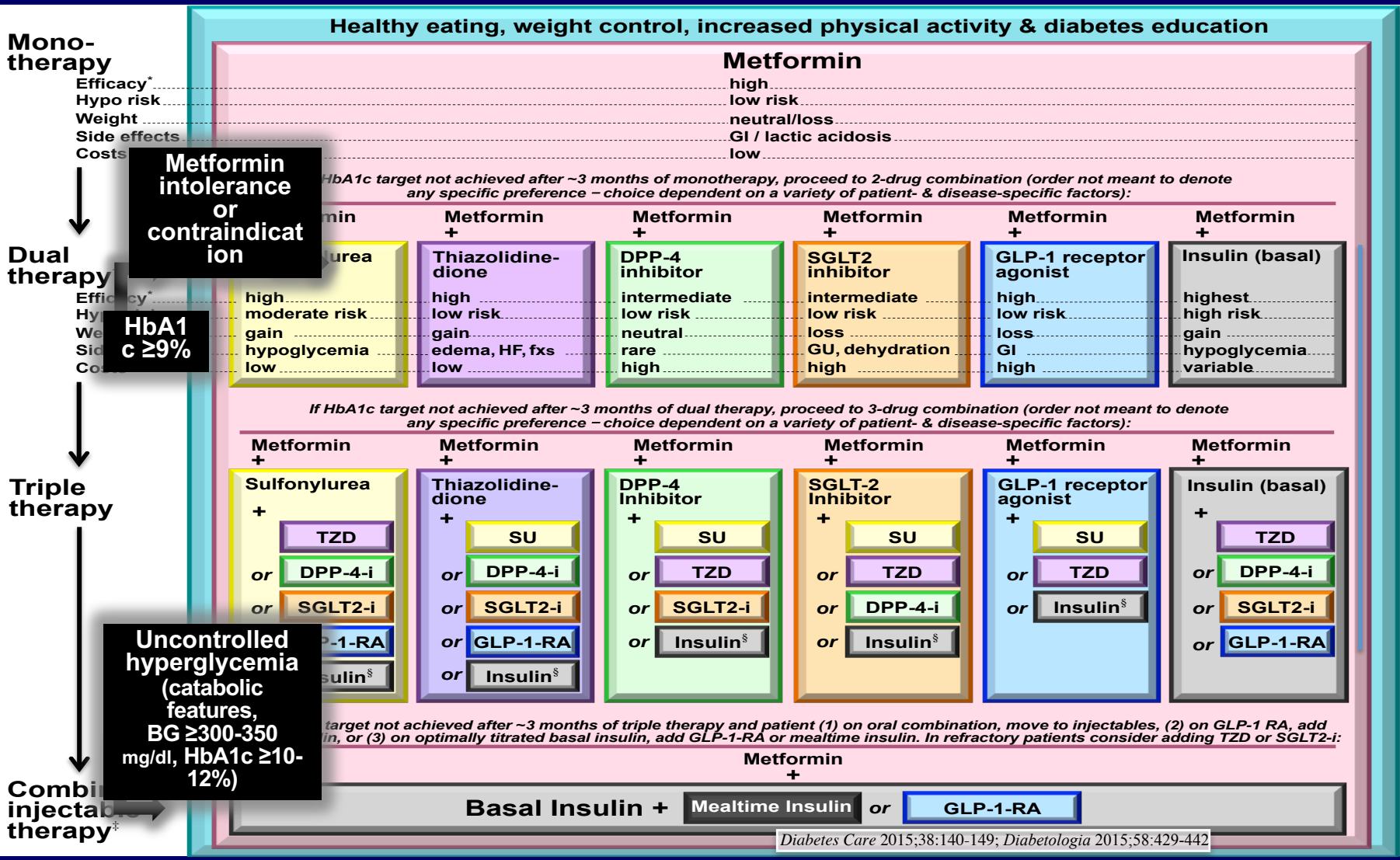
Incretin Mimetics = GLP-1 Analougues

Exenatide:

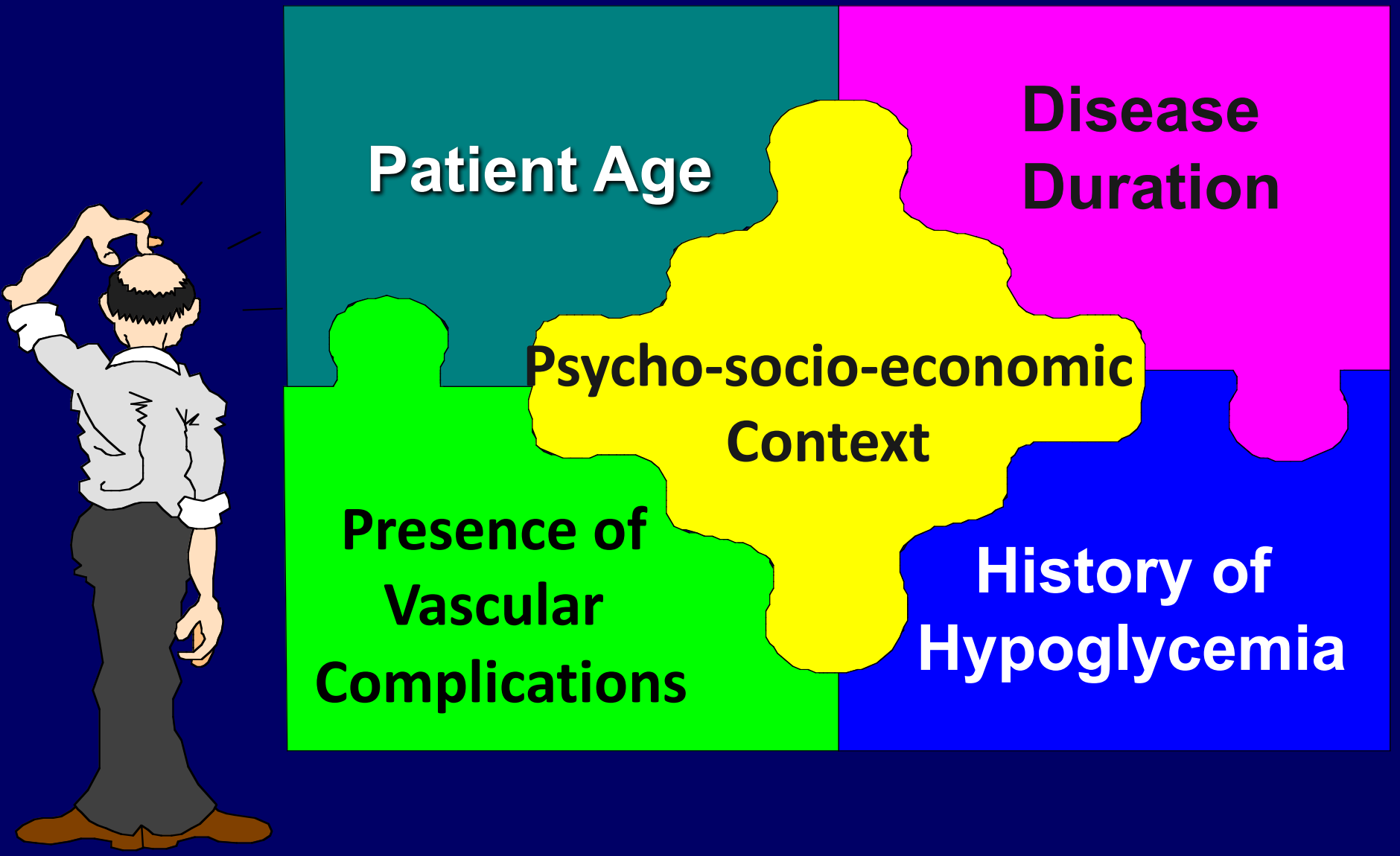
- Twice daily
- 2 doses: 5 mcg -10 mcg
- Weight reduction

Liraglutide:

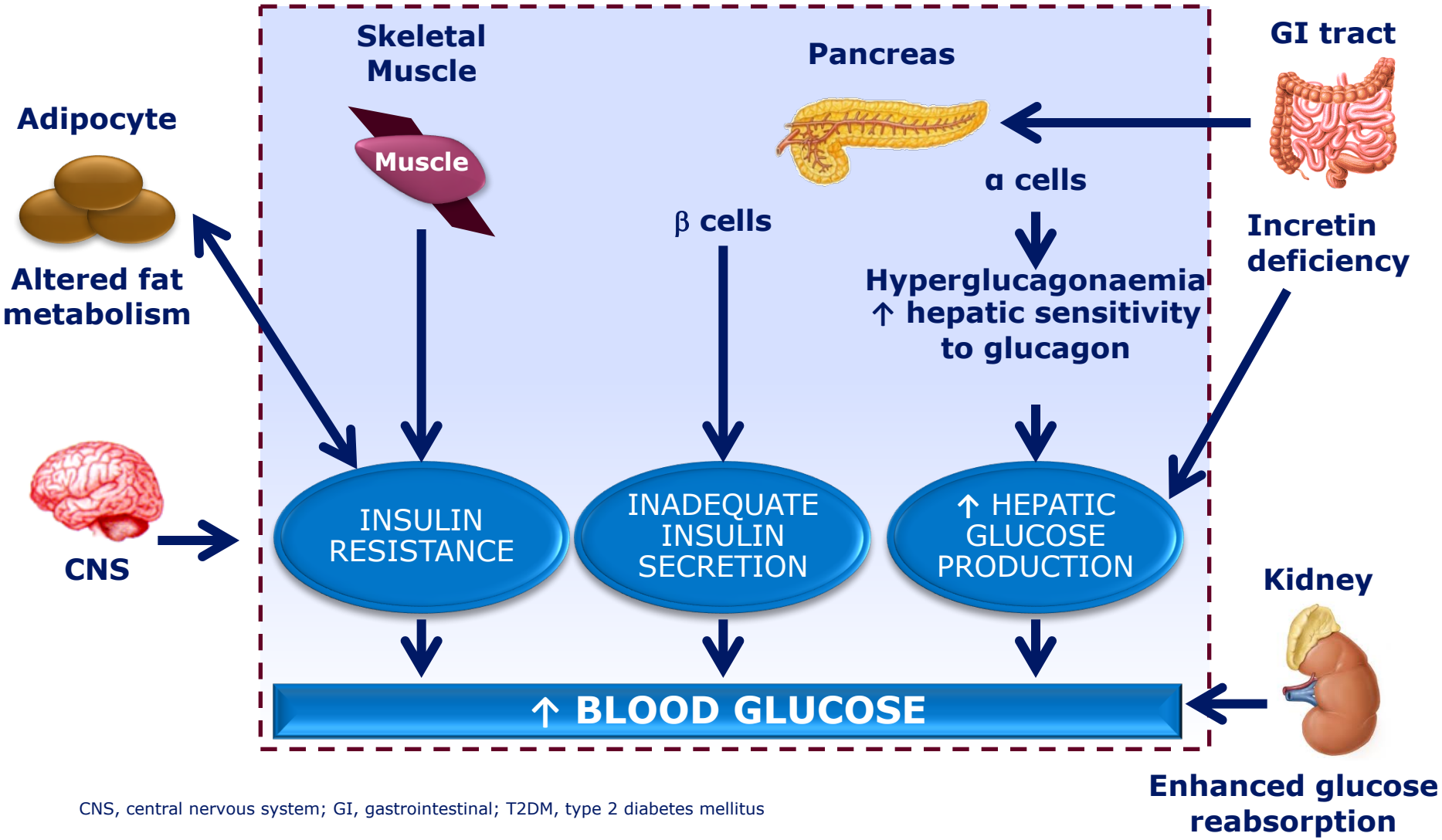
- is a once daily
- 3 doses: 0.6, 1.2, 1.8 mg
- HbA1c 0.8-1.8
- Weight reduction, Less nausea


Healthy eating, weight control, increased physical activity & diabetes education
Mono-therapy
Metformin
Efficacy*
high
Hypo risk
low risk
Weight
neutral/loss
Side effects
GI / lactic acidosis
Costs
low
Metformin intolerance or contraindication
HbA1c target not achieved after ~3 months of monotherapy, proceed to 2-drug combination (order not meant to denote any specific preference – choice dependent on a variety of patient- & disease-specific factors):
Dual therapy
HbA1c ≥9%
Metformin +
Sulfonylurea
high
moderate risk
gain
hypoglycemia
low
Thiazolidine-dione
high
low risk
gain
edema, HF, fxs
low
DPP-4 inhibitor
intermediate
low risk
neutral
rare
high
SGLT2 inhibitor
intermediate
low risk
loss
GU, dehydration
high
GLP-1 receptor agonist
high
low risk
loss
GI
high
Insulin (basal)
highest
high risk
gain
hypoglycemia
variable
If HbA1c target not achieved after ~3 months of dual therapy, proceed to 3-drug combination (order not meant to denote any specific preference – choice dependent on a variety of patient- & disease-specific factors):
Triple therapy
Metformin +
Sulfonylurea + TZD or DPP-4-i or SGLT2-i or GLP-1-RA or Insulin§
Thiazolidine-dione + SU or DPP-4-i or SGLT2-i or GLP-1-RA or Insulin§
DPP-4 Inhibitor + SU or TZD or SGLT2-i or Insulin§
SGLT-2 Inhibitor + SU or TZD or DPP-4-i or Insulin§
GLP-1 receptor agonist + SU or TZD or Insulin§
Insulin (basal) + TZD or DPP-4-i or SGLT2-i or GLP-1-RA
Uncontrolled hyperglycemia (catabolic features, BG ≥300-350 mg/dl, HbA1c ≥10-12%)
target not achieved after ~3 months of triple therapy and patient (1) on oral combination, move to injectables, (2) on GLP-1 RA, add in, or (3) on optimally titrated basal insulin, add GLP-1-RA or mealtime insulin. In refractory patients consider adding TZD or SGLT2-i:
Combi injectable therapy‡
Metformin +
Basal Insulin + Mealtime Insulin or GLP-1-RA
Diabetes Care 2015;38:140-149; Diabetologia 2015;58:429-442

The ‘Puzzle’ of Setting Glycemic Goals in T2DM



Pathophysiology of type 2 diabetes



CNS, central nervous system; GI, gastrointestinal; T2DM, type 2 diabetes mellitus

Cernea S & Raz I. *Diabetes Care* 2011;34(suppl 2):S264–S271

6

OBJECTIVES:

1. SCOPE OF DIABETES
2. MAKING THE DIAGNOSIS
3. PATHOPYSIOLOGY
4. DISEASE CONSEQUENCES
5. MANAGEMENT
6. CONCLUSION

The ABC of T2DM care

Glycemic control is important.
Non glycemic factors are even more important towards outcome

- Glycemic control
- HbA1c/HBGM
- Hypos / illness
- Work / leisure
- Special events

Glucose **A**

- Kidneys
- Eyes
- Feet
- Nerves
- Erectile Dysfunction

Micro vascular **B**

- BP
- Lipids
- Aspirin
- Smoking cessation
- Diet / Exercise

Macro vascular **C**

Intensive Day Course in Diabetes Practice

1	0800-0845	PRE-TEST & Course Introduction Hand-in Your Own Questions (Max 5 each)	
2	0900-0930	T2DM at a glance	
3	0935-1000	Newly diagnosed Hyperglycemia: What to do?	
4	1005-1035	Practical Life Style Modification	
5	1035-1100	Morning Break	
6	1100-1130	Oral Antidiabetic Therapy in Practice I	
7	1130-1200	Oral Antidiabetic Therapy in Practice II	
8	1200-1300	Prayer and Lunch	
9	1300-1400	Insulin therapy in T2DM	
10	1430-1500	Beyond oral agents: what next? The new injectables	
11	1505-1530	Break and Prayer	
12	1535-1600	Micro-vascular Care in T2DM	
13	1605-1630	Macro-vascular Care in T2DM	
14	1635-1700	Achieving Goals & Targets in T2DM: Putting it together	
15	1705-1730	1. Q & A 2. Post-Test	

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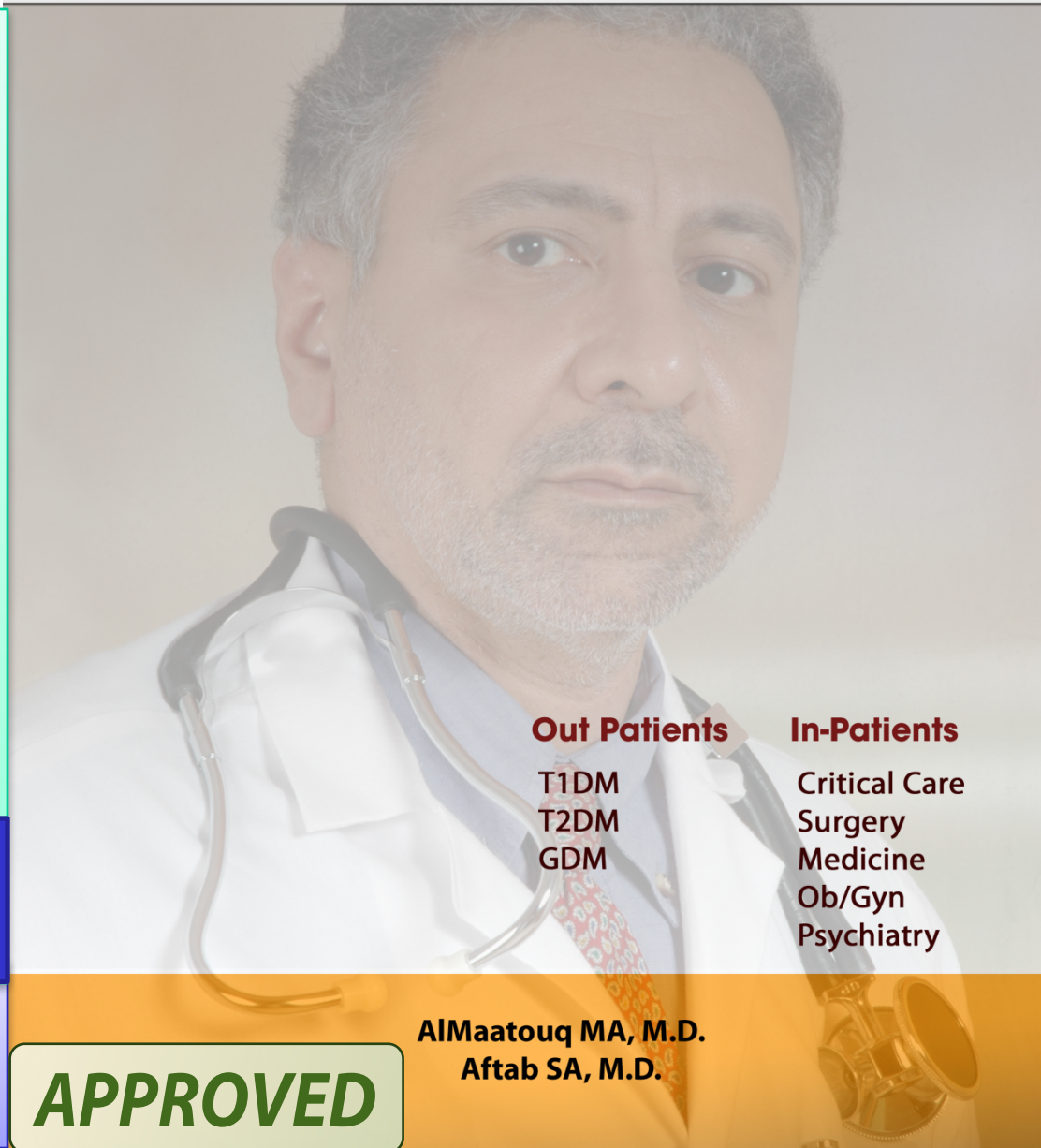
GO TO IT

AlMaatouq manual of

DIABETES PRACTICE

Goals, Targets, Tools & Outcome monitoring

Adapting the guidelines to individualization of care



Out Patients	In-Patients
T1DM	Critical Care
T2DM	Surgery
GDM	Medicine
	Ob/Gyn
	Psychiatry

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APPROVED

Riyadh 2021