TİP 2 DİYABETES MELLİTUS'TA AGRESİF TEDAVİ GEREKLİ DEĞİLDİR

24.04.2014
ANTALYA

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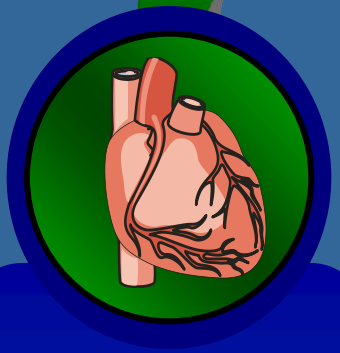
Karadeniz Teknik Üniversitesi

Tıp Fakültesi

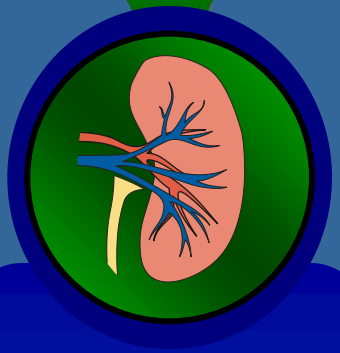
Trabzon

Diyabetes Mellitusun Klinik Önemi

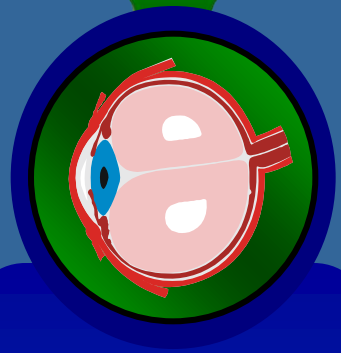
Diabet



Kardiyo Vasküler Mortalitede 2-4 Kat Artış

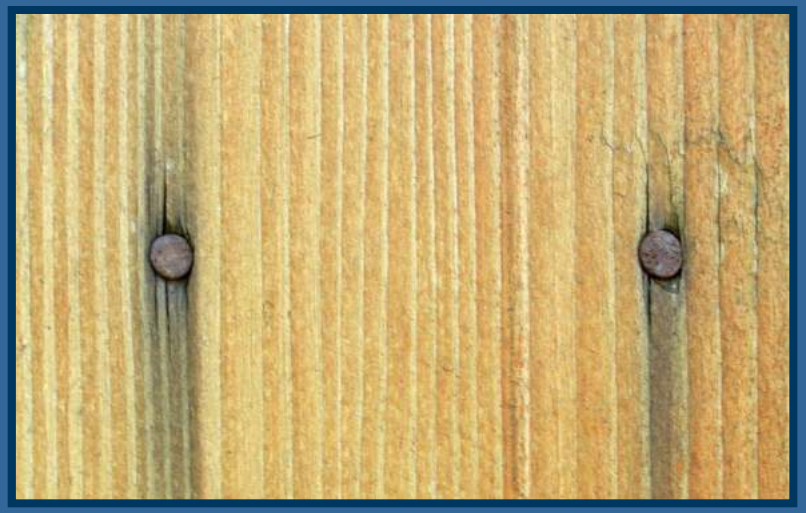


Son Dönem Böbrek Yetmezliğinin En Önemli Nedeni



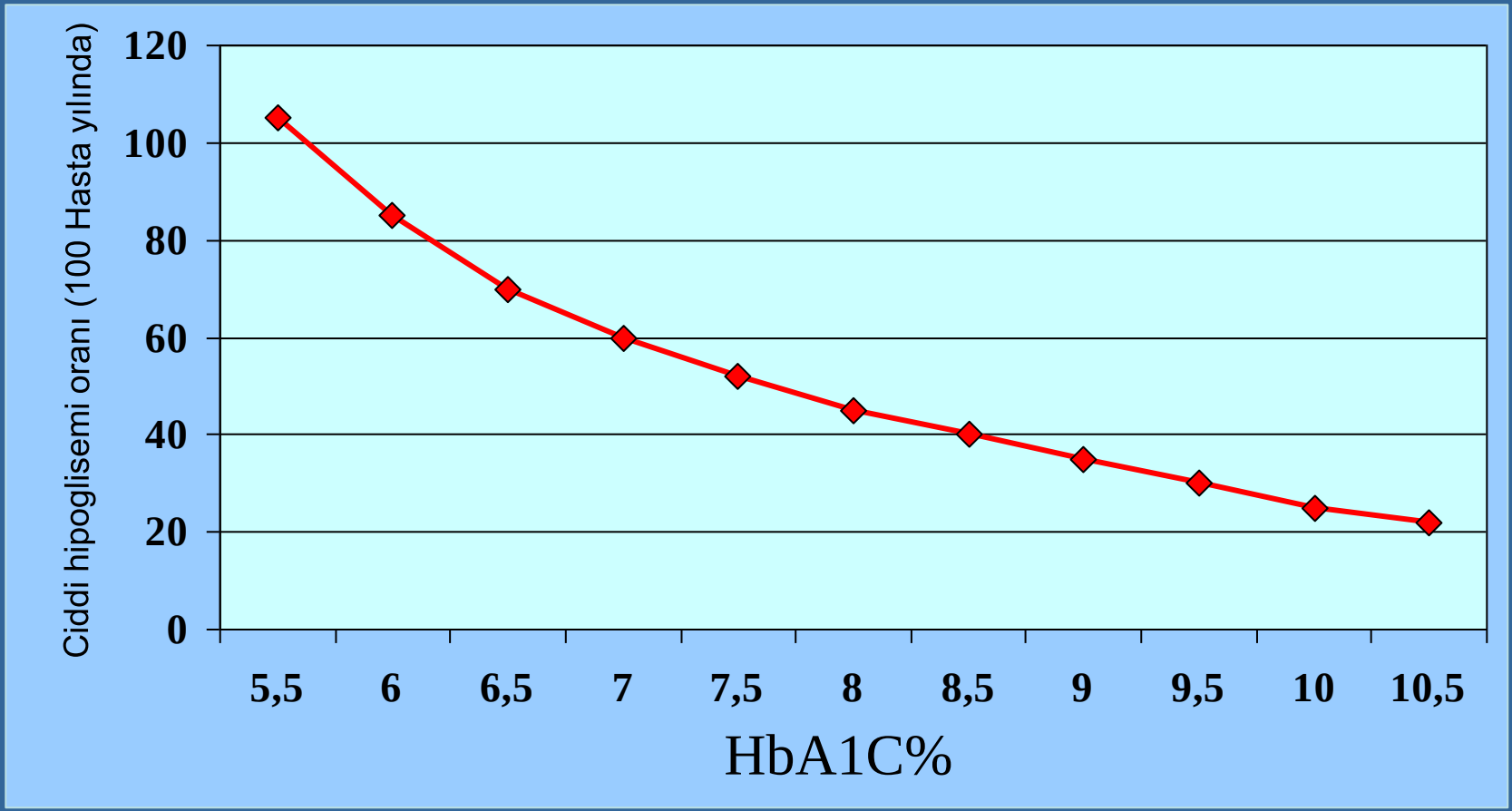
Erişkinlerde Görme Kaybının Bir Numaralı Nedeni

Nontravmatik Bacak Amputasyon-larının En Sık Nedeni




HbA1c %
14
12
10
8
7
6.5
6

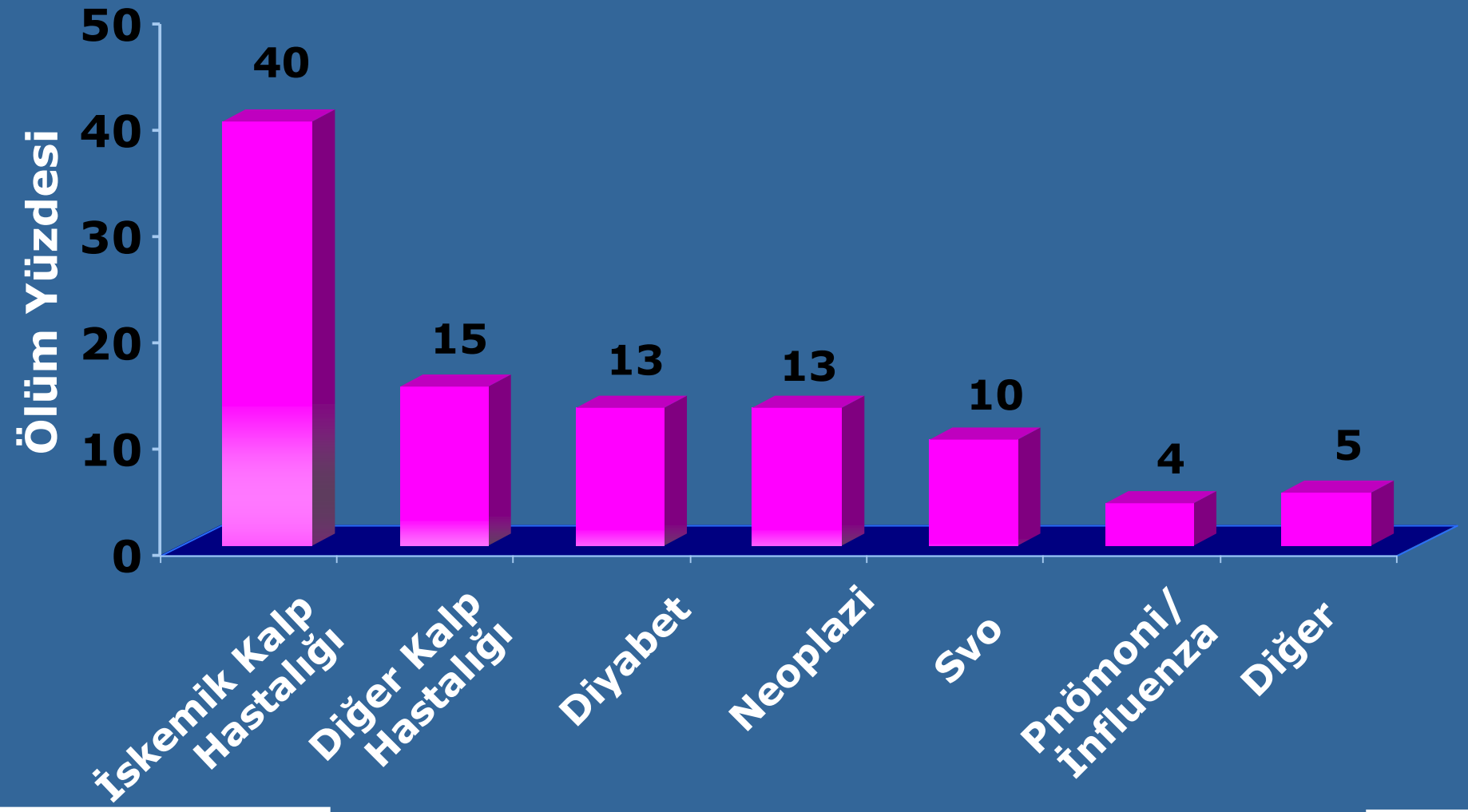
Yoğun İnsülin Tedavisi Alan Hastalarda Ciddi Hipoglisemi Oranı



DCCT çalışmasında, yoğun insülin tedavisini takiben HbA1c düzeylerinde anlamlı azalmayla birlikte ciddi hipoglisemi ataklarında önemli bir artış olmuştur.

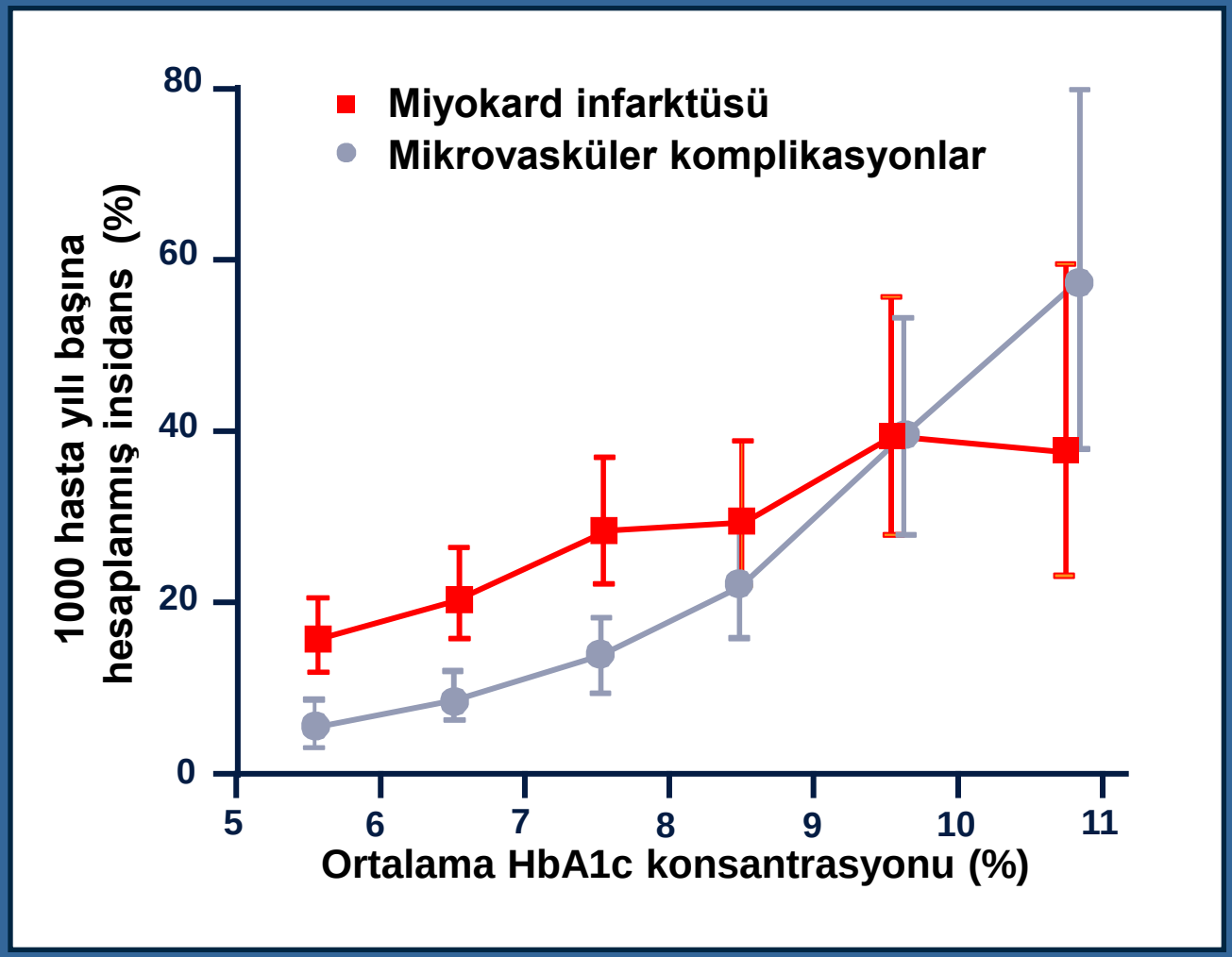
DCCT Research Group. New England Journal of Medicine 1993

Diyabetik Hastalarda Ölüm Nedenleri



Geiss LS, Diabetes in America, National Institutes of Health,1995

UKPDS: Mikro ve Makrovasküler Komplikasyonlar ve HbA1c İlişkisi



Stratton IM, BMJ, 2000

Komplikasyon riski: IDF Rehberi

	Makrovasküler risk	Makro ve Mikro vasküler risk
HbA_{1c} (%)	> 6.5	> 7.5
Açlık plazma glukozu (mg/dl)	> 100	≥ 126
Post-prandiyal kan glukozu (mg/dl)	≥ 135	> 160

European Diabetes Policy Group, Diabet Med, 1999

Diabetes Mellitus'ta Metabolik Kontrol Hedefleri

TABLO 4.1: **Glisemik kontrol hedefleri**

	Hedef[*]	Gebelikte
A1C	≤%6.5 (≤48 mmol/mol)	≤%6.5 (tercihen ≤%6; ≤42 mmol/mol)
APG ve öğün öncesi PG	70-120 mg/dl	60-95 mg/dl
Öğün sonrası 1.stPG	-	<140 mg/dl[**] (tercihen <120 mg/dl)
Öğün sonrası 2.stPG	<140 mg/dl	120 mg/dl

[*]Glisemik hedefler bireyselleştirilmelidir. Hastanın yaşam beklenti süresi, diyabet yaşı, hipoglisemi riski, diyabet komplikasyonları ve eşlik eden diğer hastalıklarına göre belirlenmeli, gerekirse daha esnek glisemik kontrol hedeflenmelidir.
[**]Gebelerde öğün sonrası 1.st PG takip edilmelidir.

"A1C: HbA1c, APG: Açlık plazma glukoz, 1.stPG ve 2.stPG: 1.st ve 2.st plazma glukoz."

Diyabetes Mellitus'ta Metabolik Kontrol Hedefleri

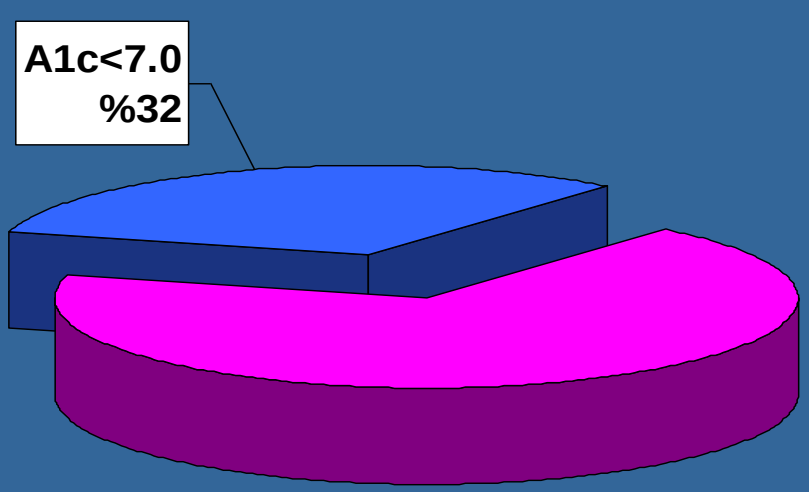
Glisemik Kontrol

- **A1c**	**<%7.0 ****
- **Preprandiyal glukoz***	**80-130 mg/dl**
- **Pik postprandiyal glukoz***	**<180 mg/dl**
Kan Basıncı	**<140/90mmHg**

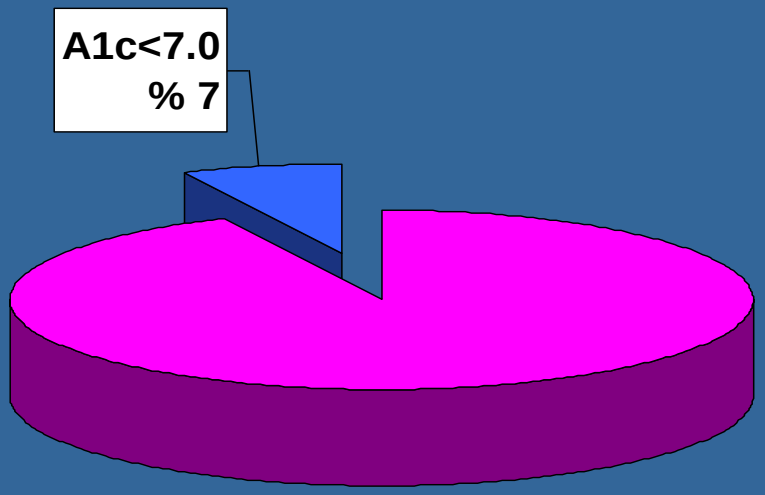
* Plazma glukoz düzeyi

** Eşlik eden KVH, hipoglisemi riski, yaşam süresi beklentisi

Diyabetli Hastaların Tedavisinde Ne Kadar Hedefteyiz?



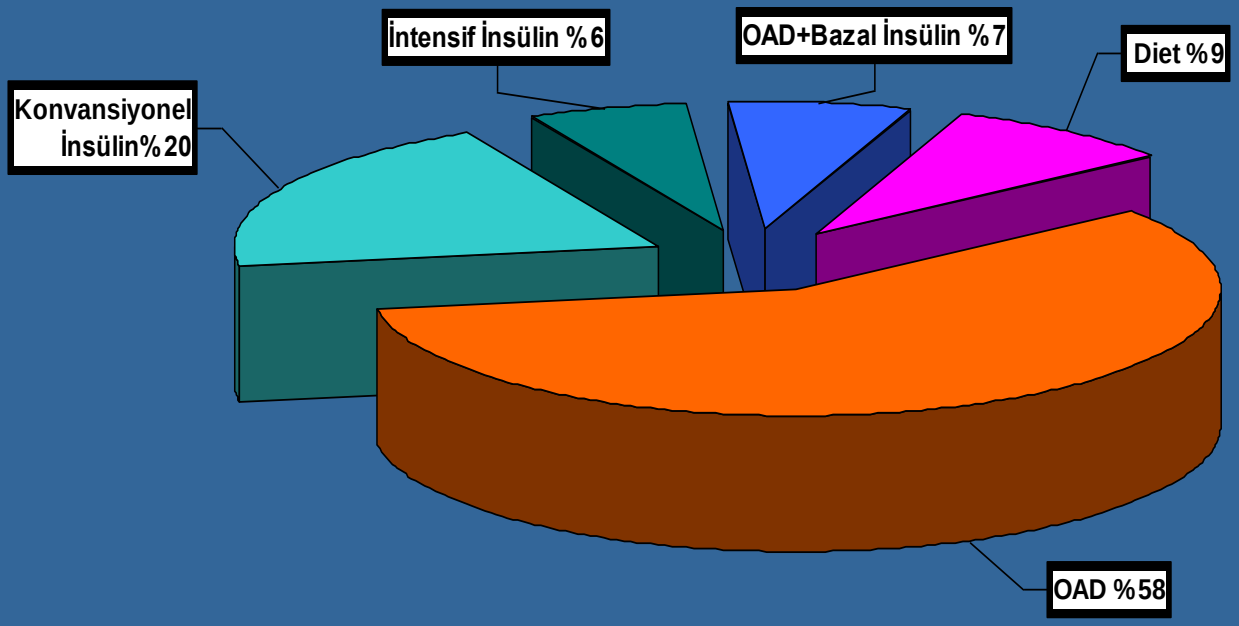
Tip 1 DM Hastaları



Tip 2 DM Hastaları

Ükinç K, Ersöz HÖ, Erem C et al TJEM 2004

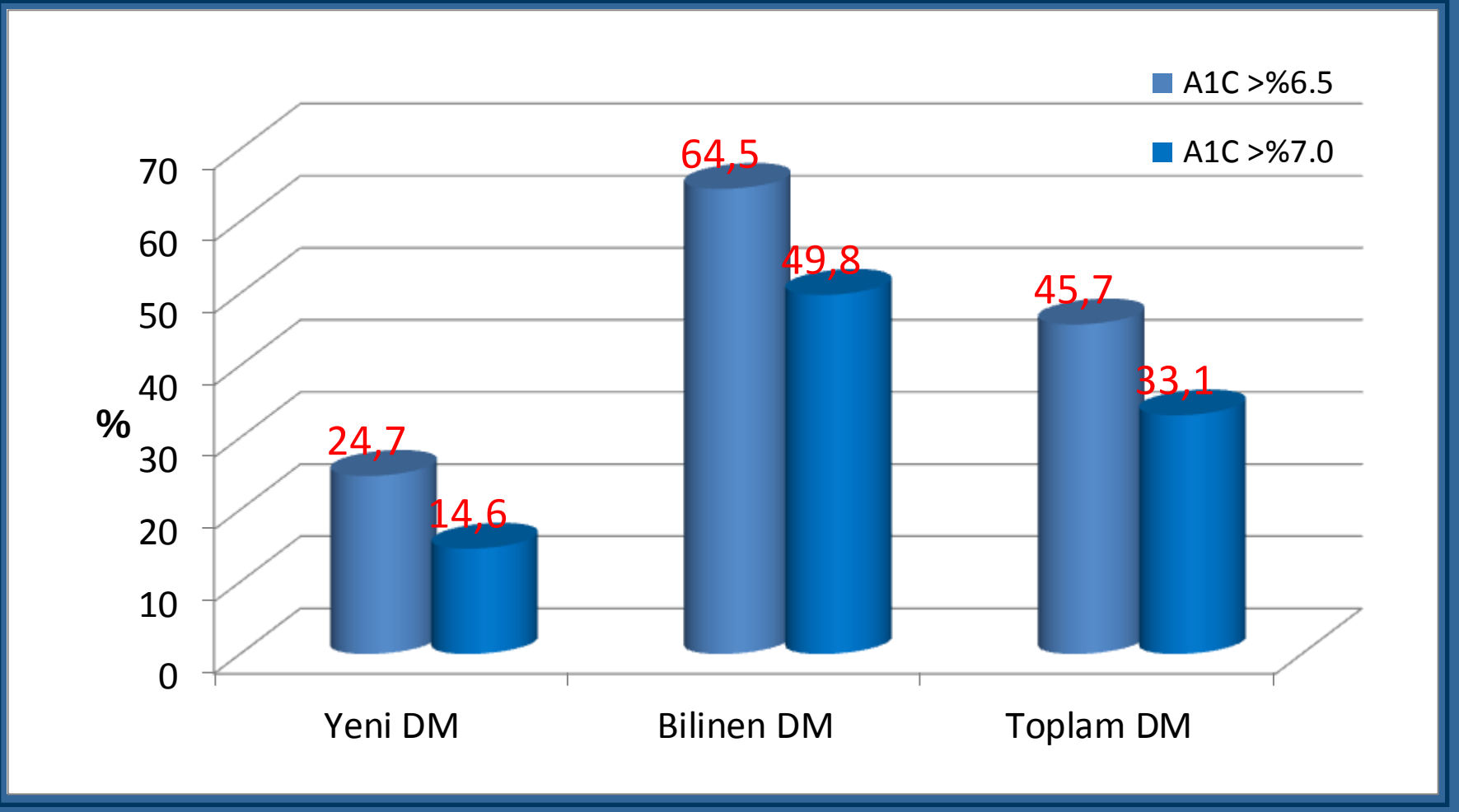
Diyabetli Hastaların Tedavisinde Ne Kadar Hedefteyiz?



Tip 2 DM Hastaları

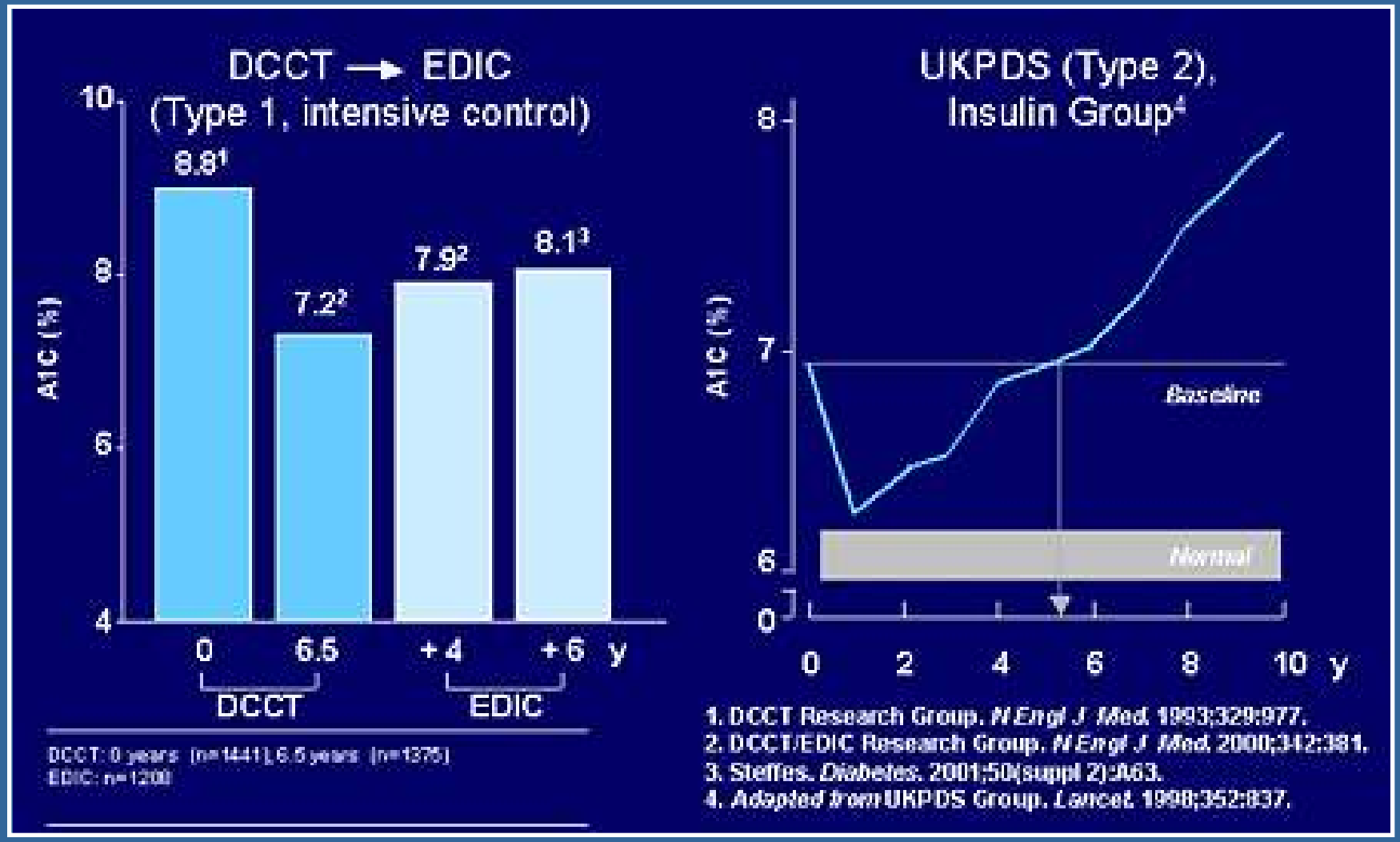
Ükinç K, Ersöz HÖ, Erem C et al TJEM 2004

TURDEP-II: Diyabette Yetersiz Glisemik Kontrol

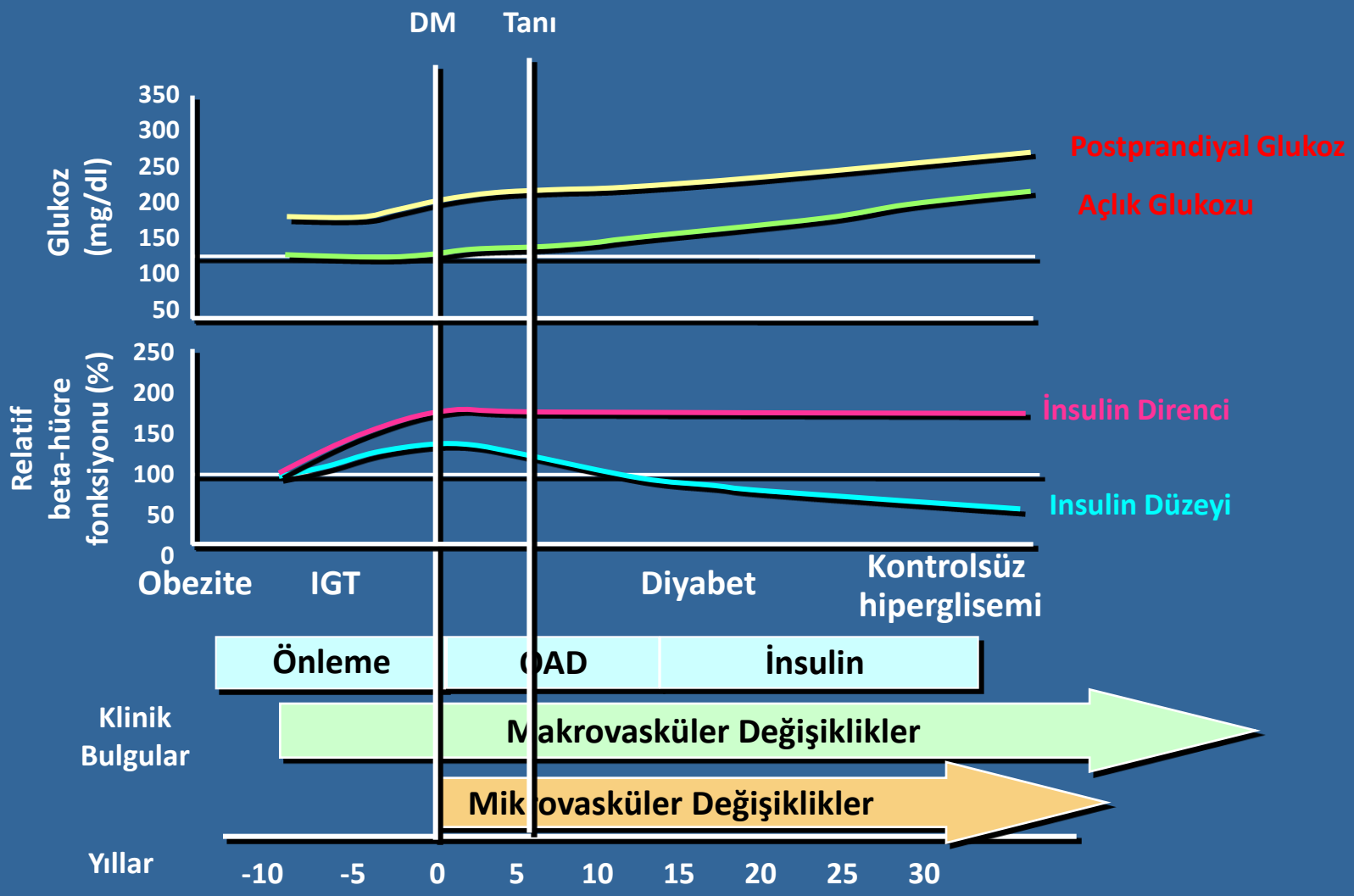


Satman İ ve TURDEP-II Çalışma Grubu. Eur J Epidemiol 2013

Diyabette Glisemik Kontrolü Sürdürmek Uzun Dönemde Kolay Değildir



Tip 2 Diyabetin Doğal Seyri



Adapted from Type 2 Diabetes BASICS. Minneapolis, MN: International Diabetes Center; 2000.

HbA1c'yi %1 Düşürmenin Etkileri

Tip 1 diyabet (DCCT)

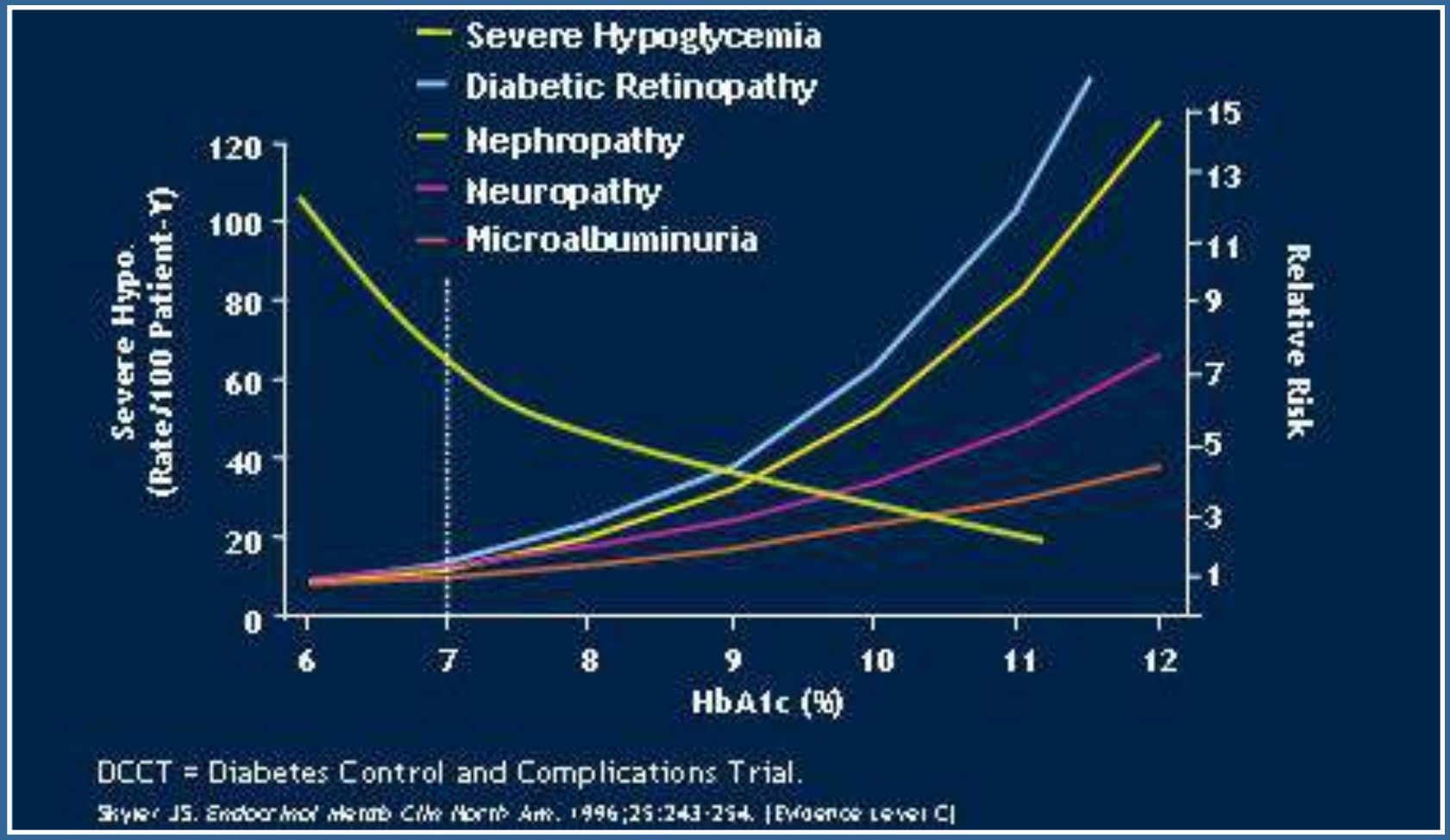
- Retinopati riski %35 ↓
- Nefropati riski %24-44 ↓
- Nöropati riski %30 ↓

Tip 2 Diyabet (UKPDS)

- Diyabete bağlı ölüm %25 ↓
- Tüm nedenlere bağlı mortalite %7 ↓
- MI riski %18 ↓
- Mikrovasküler komplikasyon riski %35 ↓

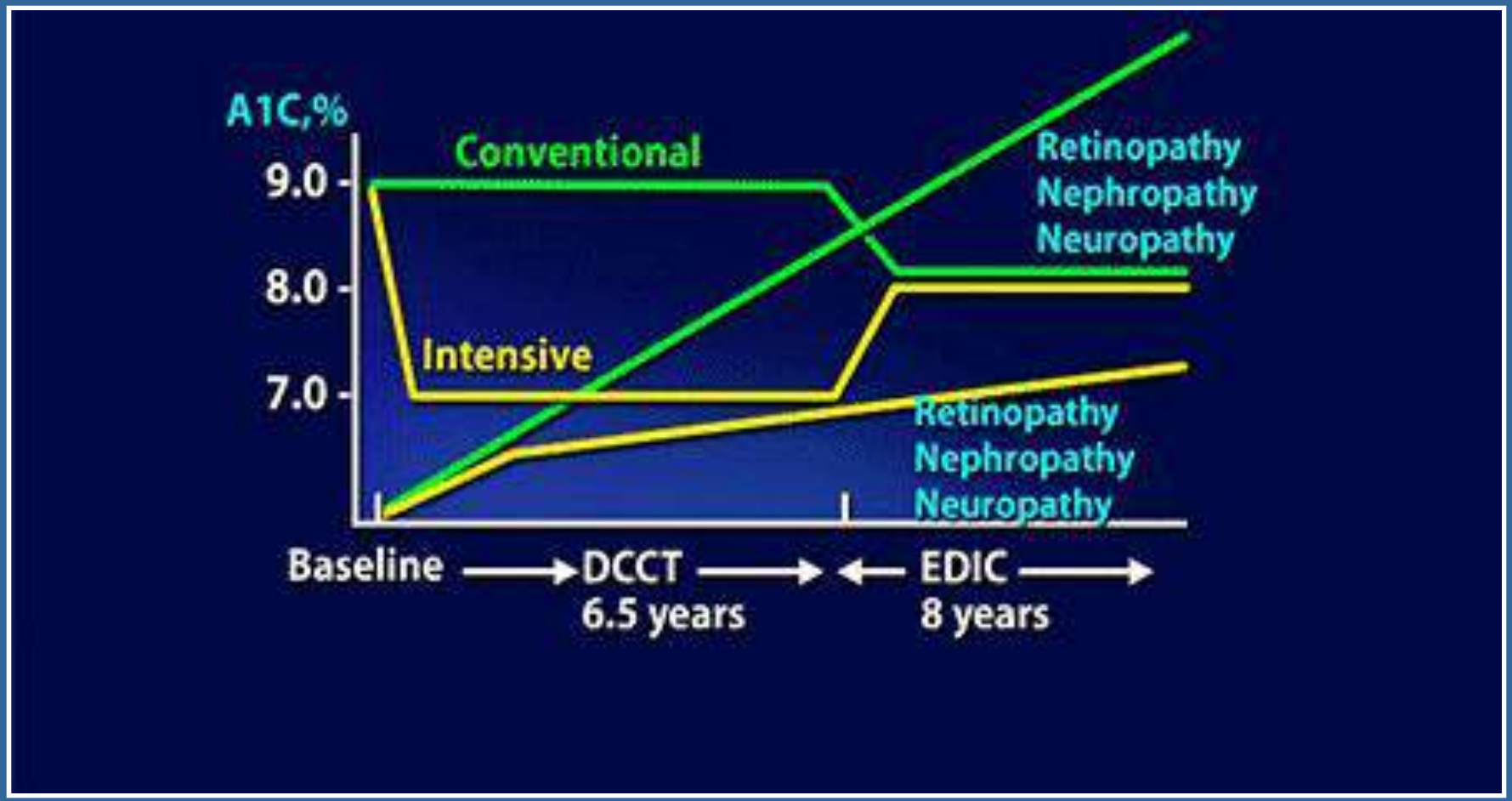
DCCT Research Group. New England Journal of Medicine 1993
UKPDS Group. Lancet 1998

Tip 1 Diyabetli Hastalarda Kan Şekerinin Agresif Kontrolü



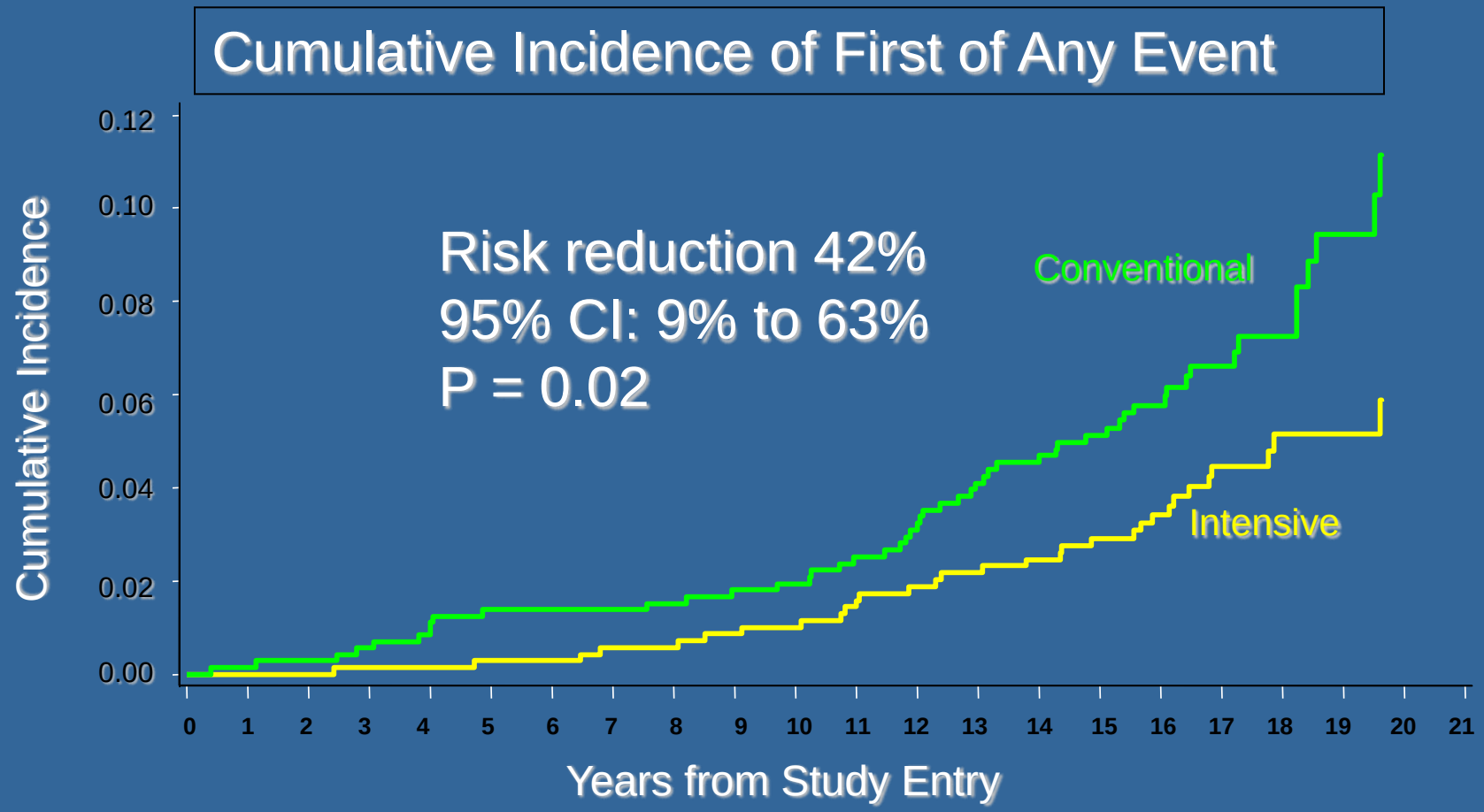
DCCT Research Group. New England Journal of Medicine 1993

Tip 1 Diyabetli Hastalarda Kan Şekerinin Agresif Kontrolü



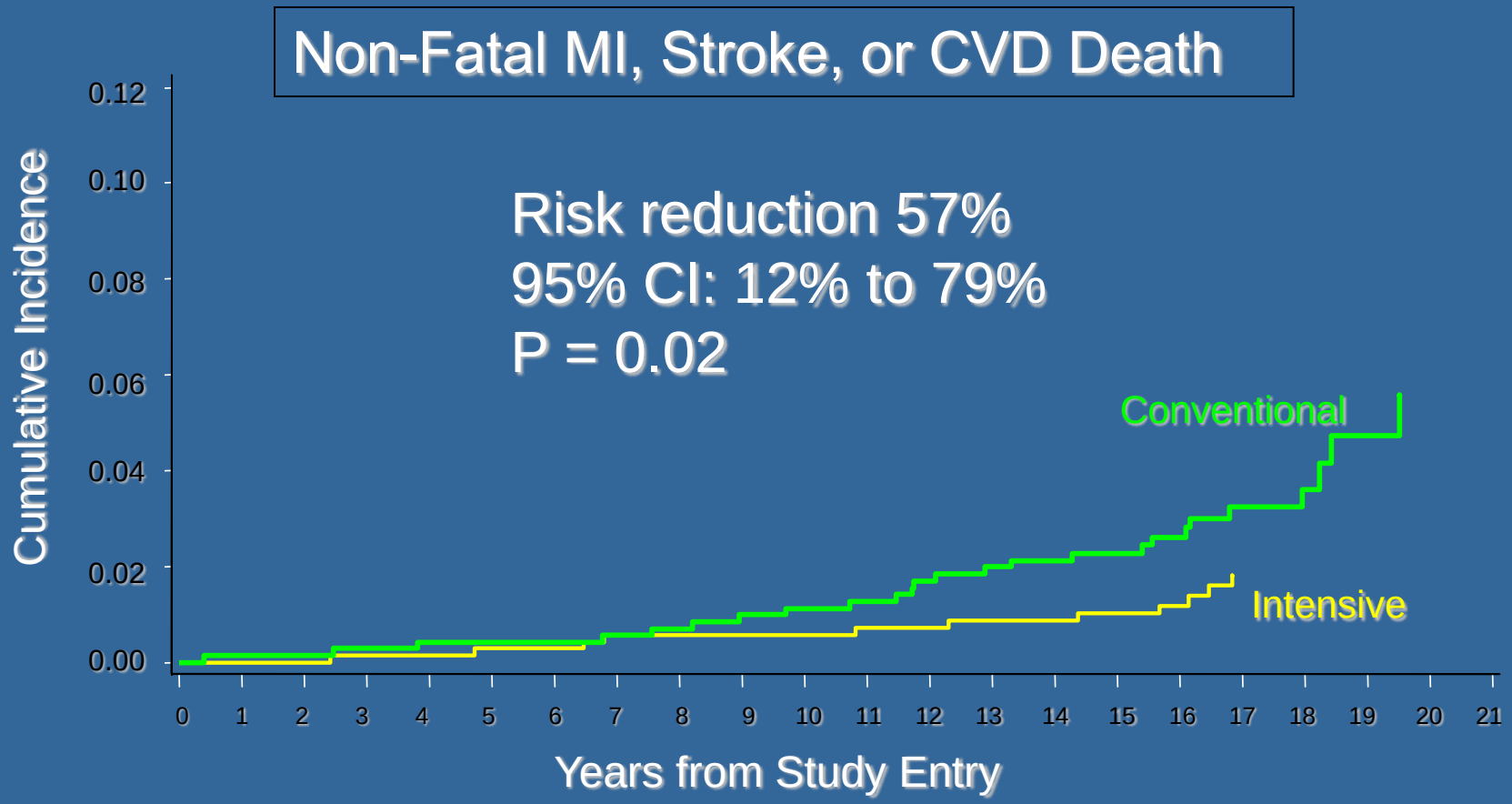
DCCT Research Group. New England Journal of Medicine 1993

EDIC Bulguları: Kardiyovasküler Olaylar



DCCT/EDIC N Engl J Med 2005: 353:2643-2653.

EDIC Bulguları: Kardiyovasküler Olaylar



DCCT/EDIC N Engl J Med 2005: 353:2643-2653.

HbA1c ve Komplikasyonlar

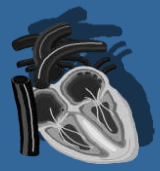
HbA1c DÜZEYİNDEKİ AZALMA

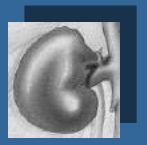
%1

RİSK AZALMASI

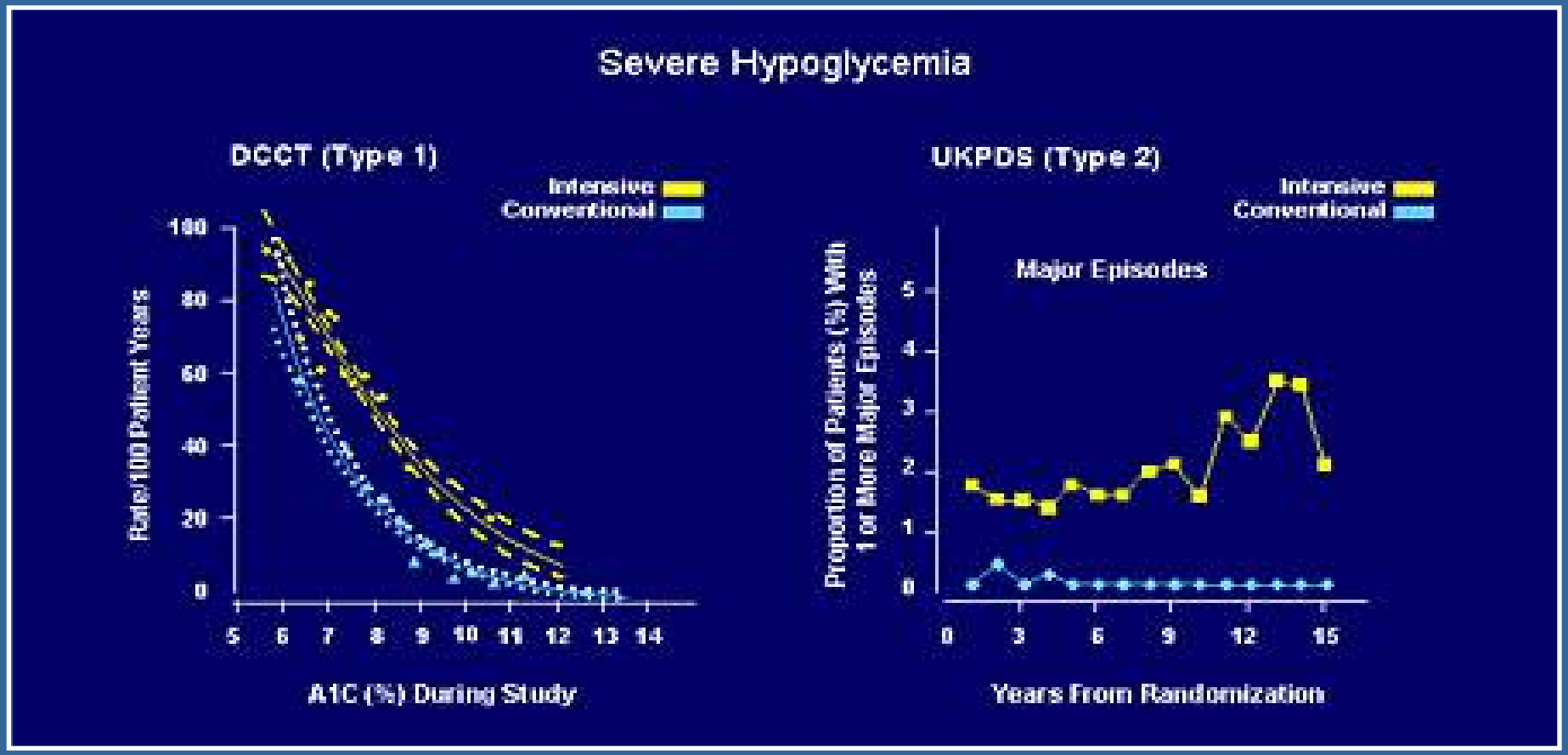
*p<0.05

Diyabete bağlı olay	*	–% 21
Miyokard infarktüsü	*	–% 18
Mikrovasküler komplikasyonlar	*	–% 35
Periferik damar hastalığı	*	–% 43





Sıkı Tedavi İşe Yarıyor Ancak Bir Bedeli Var!



DCCT Research Group. New England Journal of Medicine 1993
UKPDS Group. Lancet 1998

UKPDS Post-Treatment Analizleri

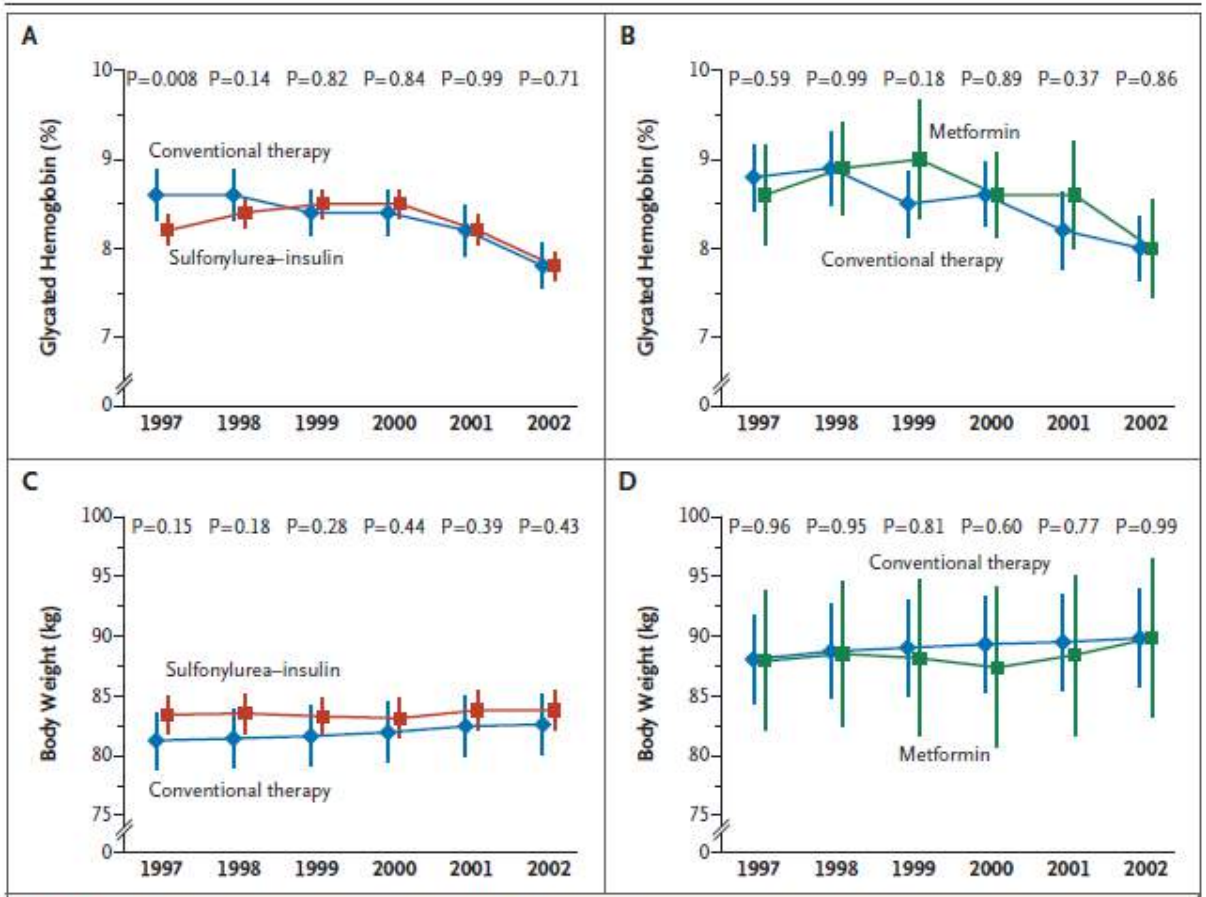


Figure 2. Mean Glycated Hemoglobin Levels and Body Weight.

Glycated hemoglobin levels for patients who were originally assigned to receive either sulfonylurea–insulin or conventional therapy (Panel A) or metformin or conventional therapy (Panel B) are shown. Panels C and D show the corresponding mean body weights in the two groups. Clinical data were not available in years 6 through 10, when questionnaires were used. The vertical bars represent 95% confidence intervals.

Holman RR et al NEJM 2008;359:1577-89

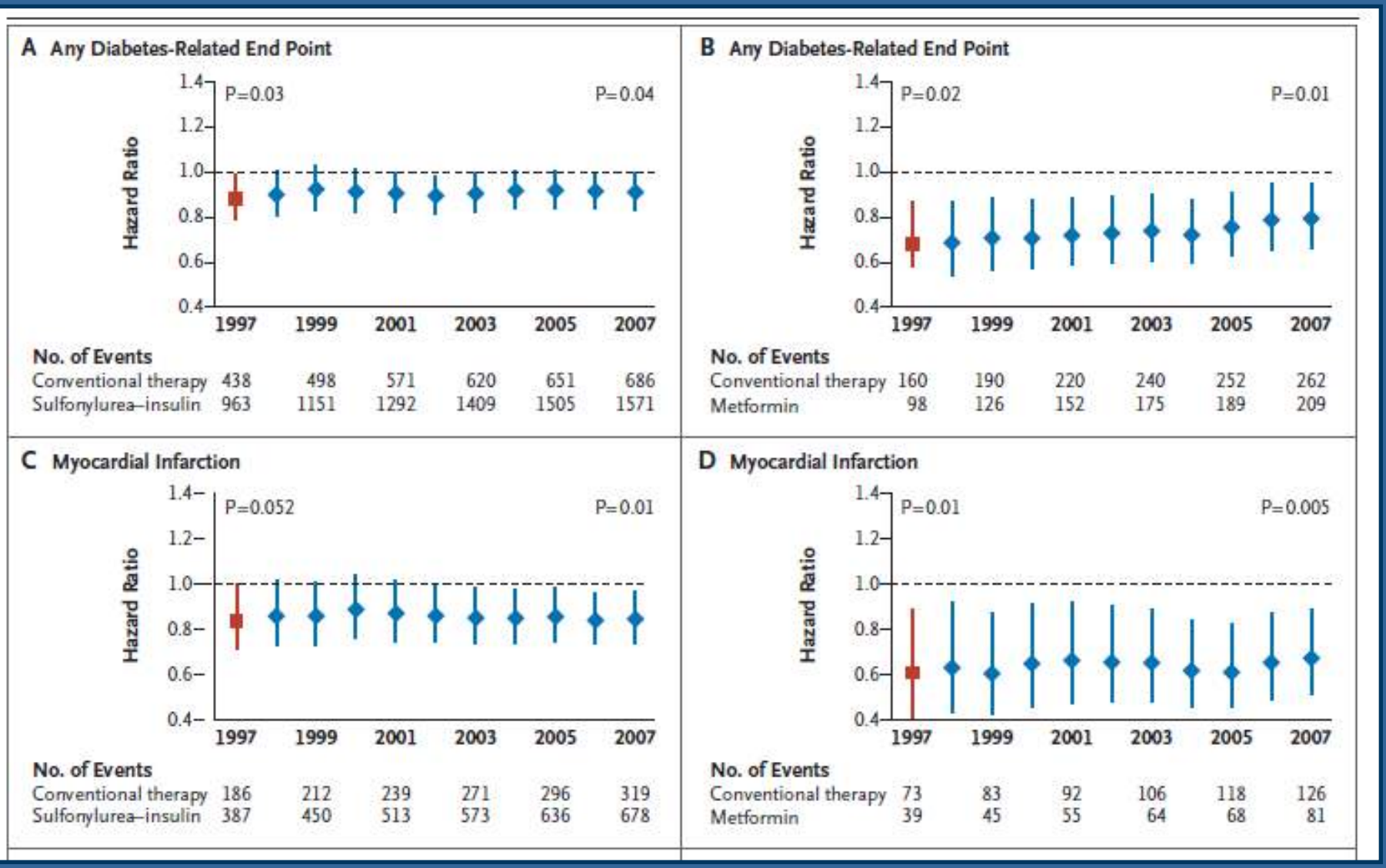
UKPDS Post-Treatment Analizleri

Table 2. Aggregate Outcomes for Patients during Follow-up.*

Aggregate Outcome	Patients with Clinical Outcome		Absolute Risk†		P Value‡	Risk Ratio for Intensive-Therapy Regimen (95% CI)
	Intensive Therapy	Conventional Therapy	Intensive Therapy	Conventional Therapy		
	no. of patients					
Sulfonylurea–insulin group	2729	1138				
Any diabetes-related end point	1571	686	48.1	52.2	0.04	0.91 (0.83–0.99)
Diabetes-related death	618	297	14.5	17.0	0.01	0.83 (0.73–0.96)
Death from any cause	1162	537	26.8	30.3	0.007	0.87 (0.79–0.96)
Myocardial infarction	678	319	16.8	19.6	0.01	0.85 (0.74–0.97)
Stroke	260	116	6.3	6.9	0.39	0.91 (0.73–1.13)
Peripheral vascular disease	83	40	2.0	2.4	0.29	0.82 (0.56–1.19)
Microvascular disease	429	222	11.0	14.2	0.001	0.76 (0.64–0.89)
Metformin group	342	411				
Any diabetes-related end point	209	262	45.7	53.9	0.01	0.79 (0.66–0.95)
Diabetes-related death	81	120	14.0	18.7	0.01	0.70 (0.53–0.92)
Death from any cause	152	217	25.9	33.1	0.002	0.73 (0.59–0.89)
Myocardial infarction	81	126	14.8	21.1	0.005	0.67 (0.51–0.89)
Stroke	34	42	6.0	6.8	0.35	0.80 (0.50–1.27)
Peripheral vascular disease	13	21	2.3	3.4	0.19	0.63 (0.32–1.27)
Microvascular disease	66	78	12.4	13.4	0.31	0.84 (0.60–1.17)

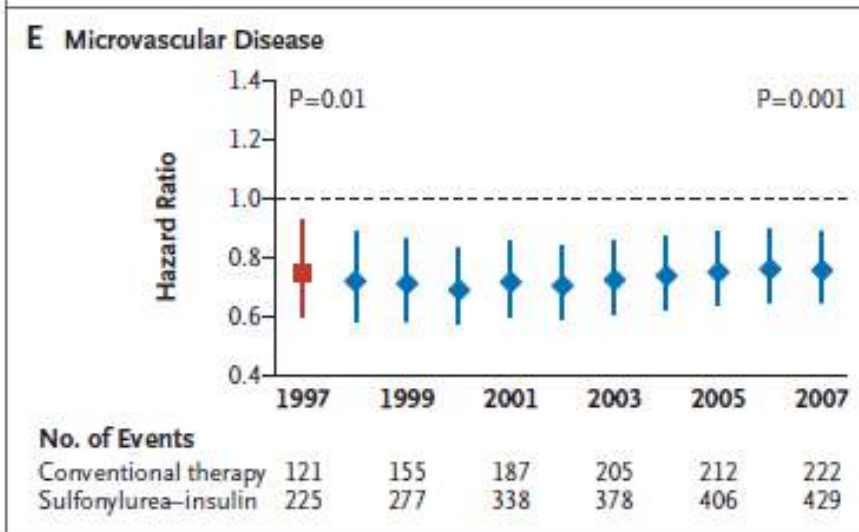
Holman RR et al NEJM 2008;359:1577-89

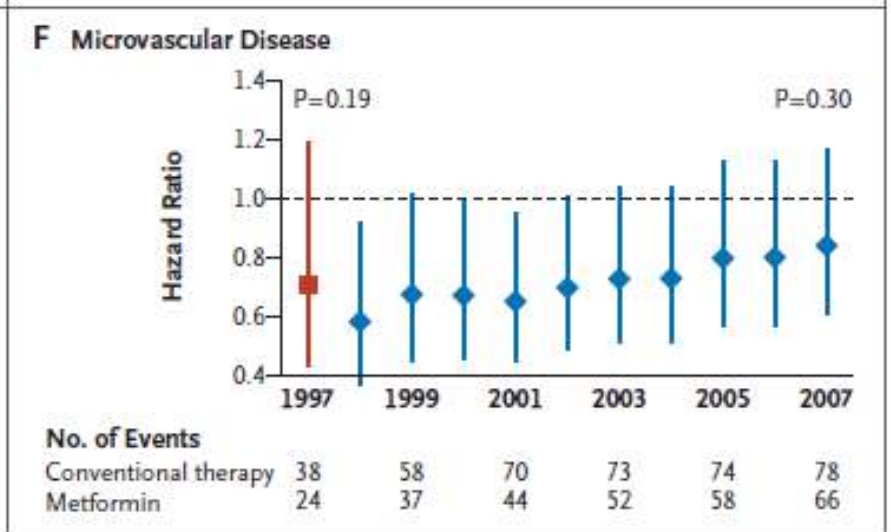
UKPDS Post-Treatment Analizleri

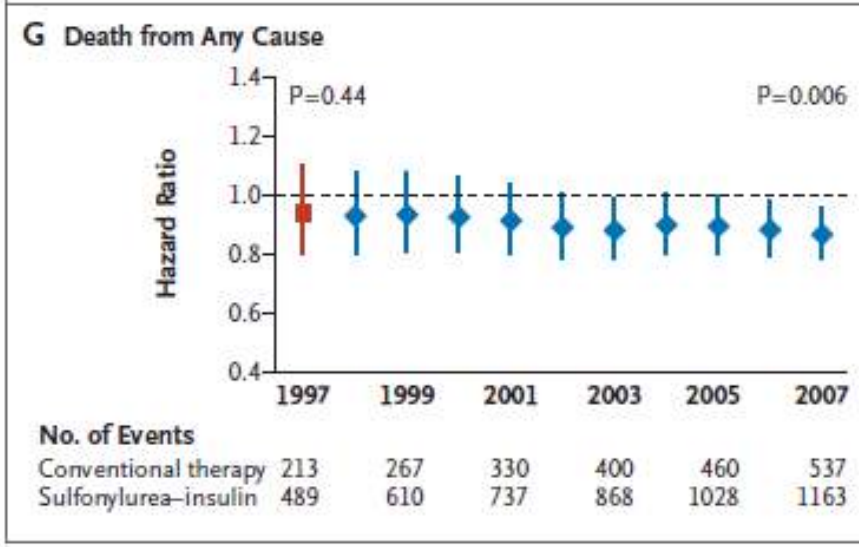


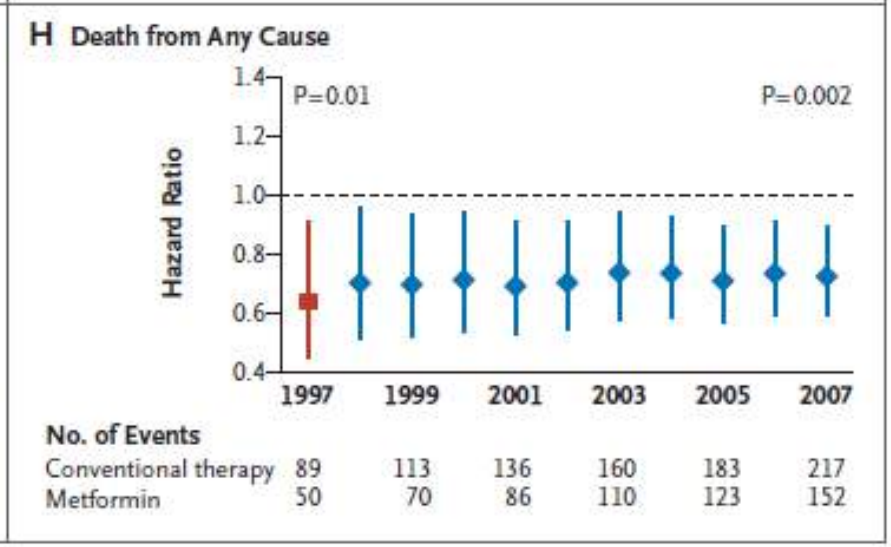
Holman RR et al NEJM 2008;359:1577-89

UKPDS Post-Treatment Analizleri









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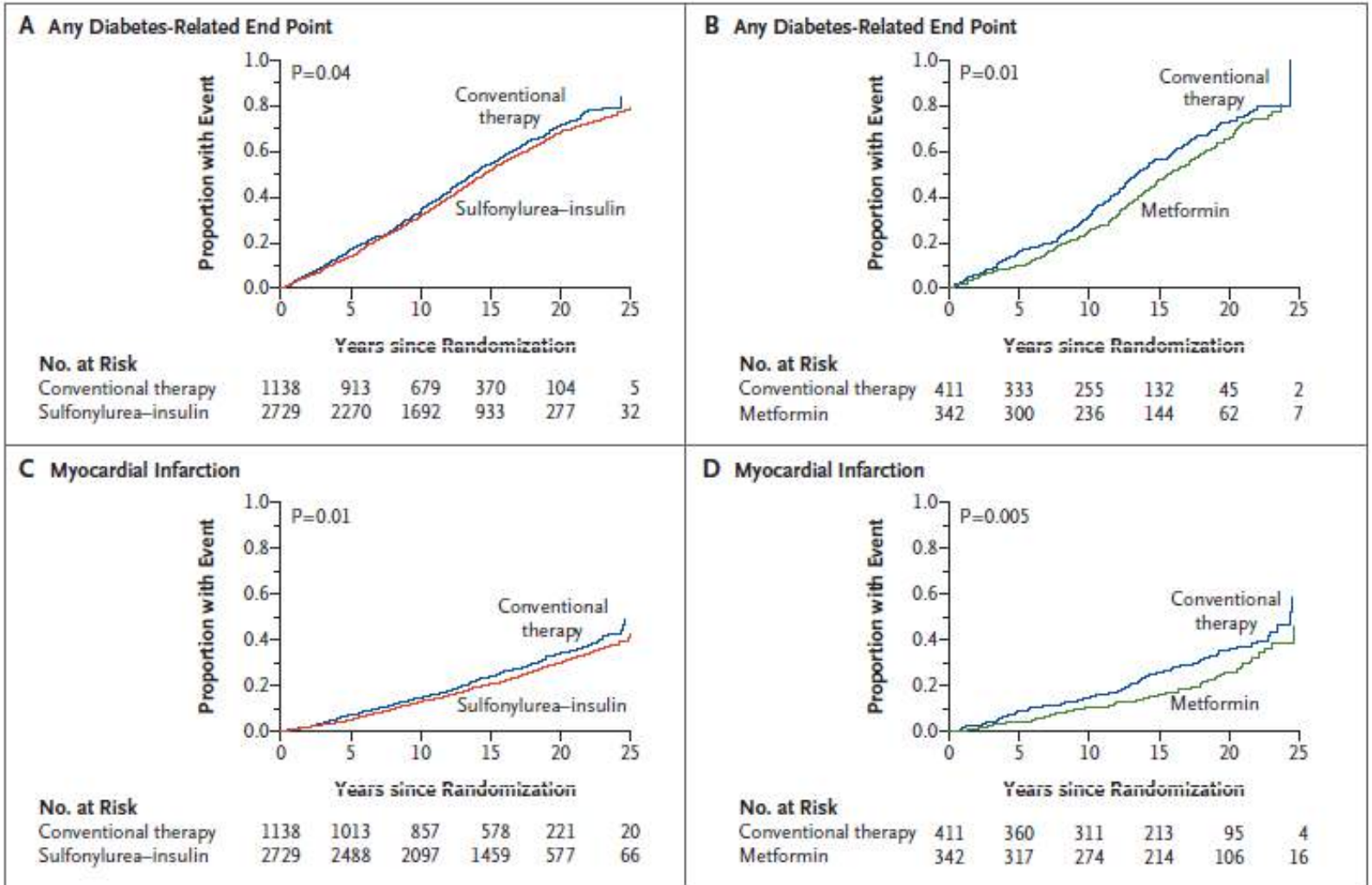

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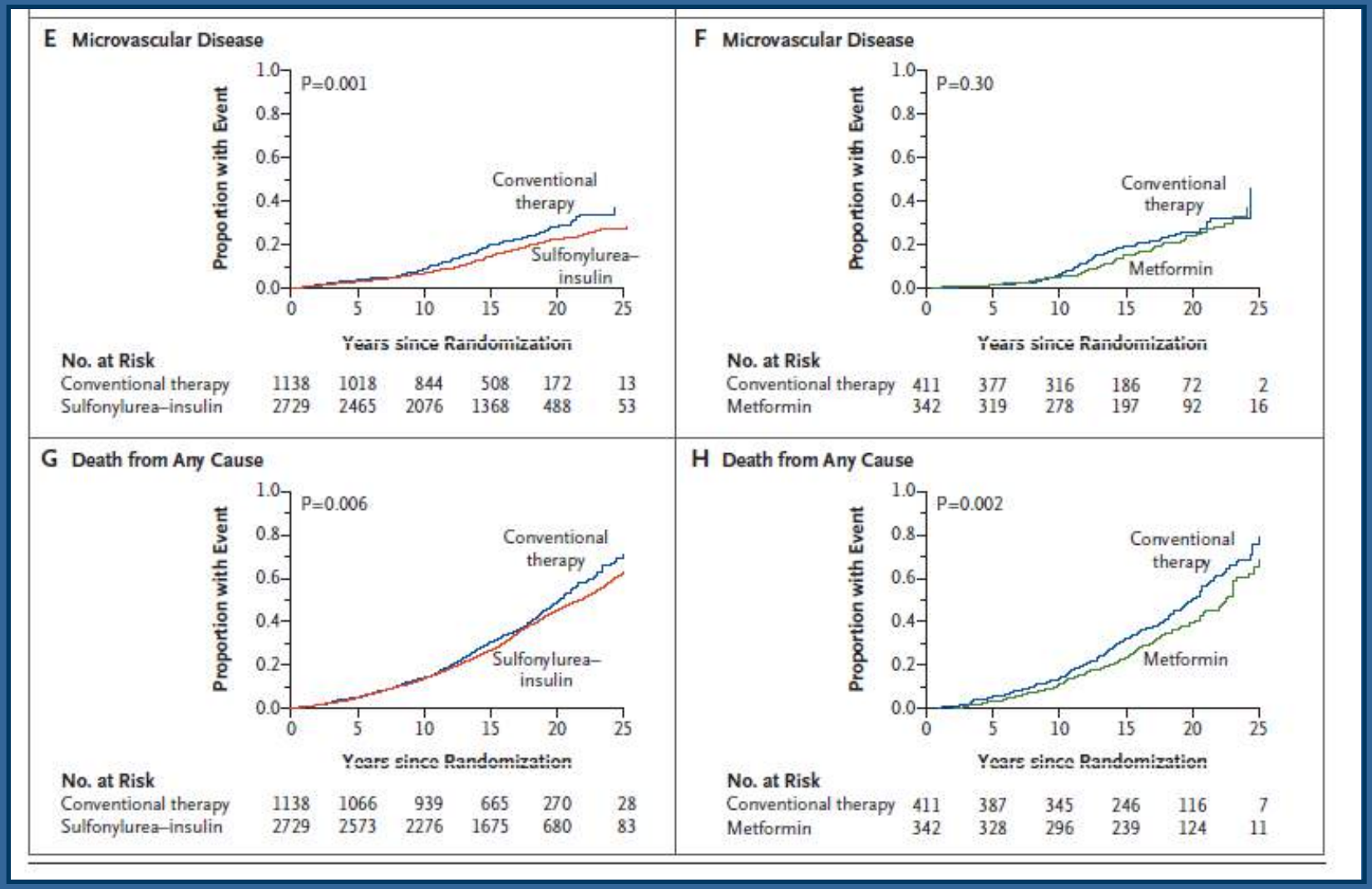
UKPDS Post-Treatment Analizleri



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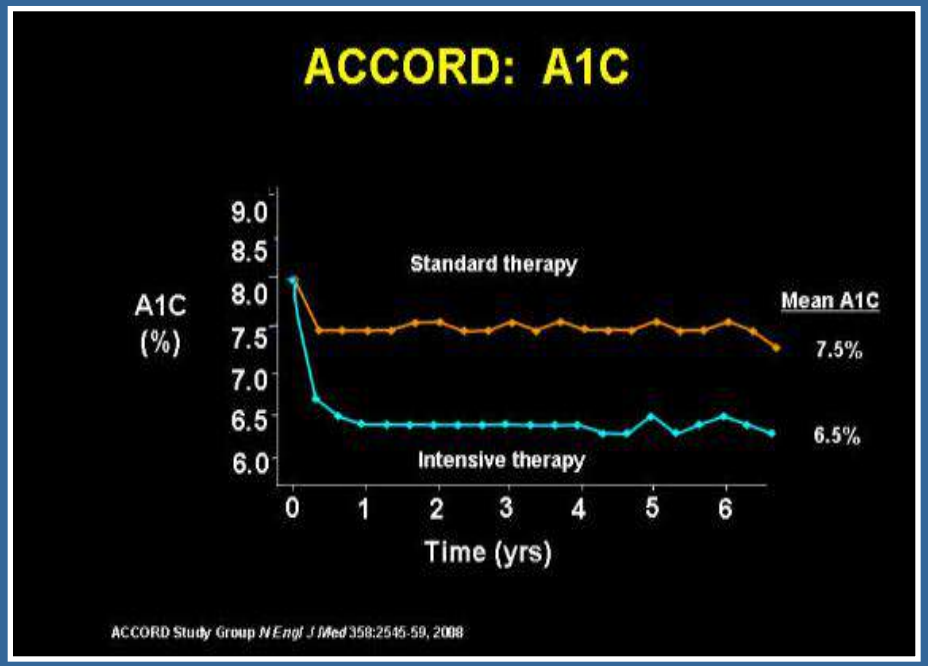
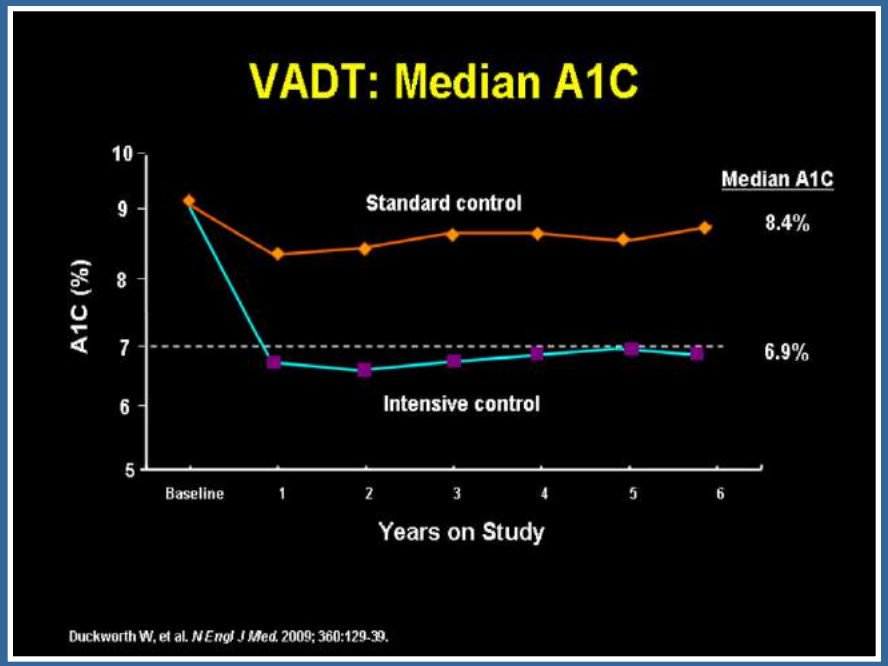
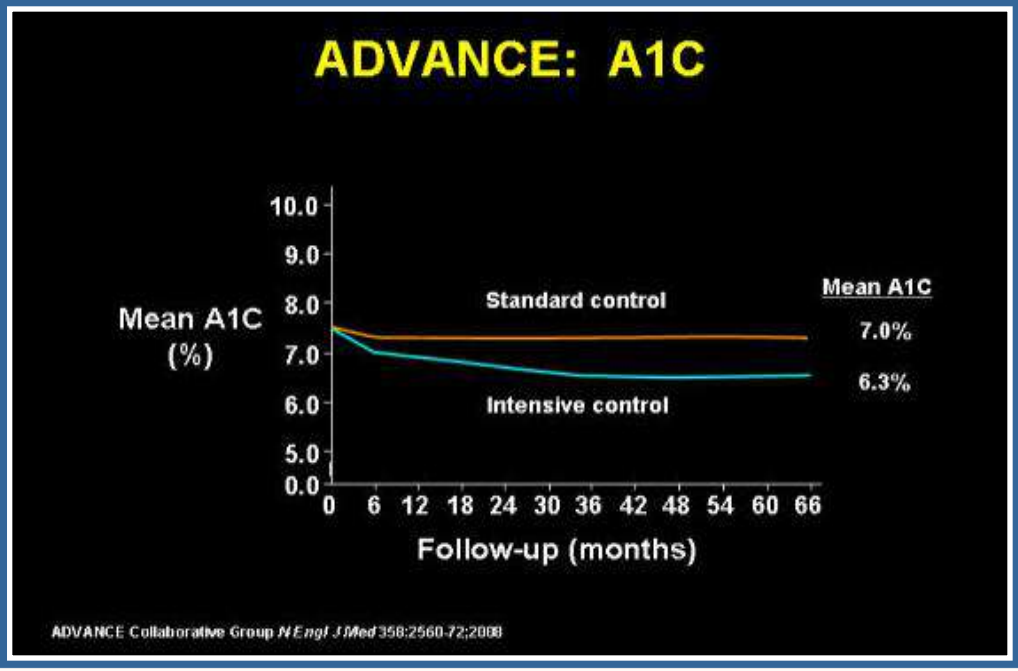

ACCORD: A1C
A1C (%)
9.0
8.5
8.0
7.5
7.0
6.5
6.0
Standard therapy
Intensive therapy
Mean A1C
7.5%
6.5%
0 1 2 3 4 5 6
Time (yrs)
ACCORD Study Group N Engl J Med 358:2545-59, 2008


VADT: Median A1C
A1C (%)
10
9
8
7
6
5
Standard control
Intensive control
Median A1C
8.4%
6.9%
Baseline 1 2 3 4 5 6
Years on Study
Duckworth W, et al. N Engl J Med. 2009; 360:129-39.


ADVANCE: A1C
Mean A1C (%)
10.0
9.0
8.0
7.0
6.0
5.0
0.0
Standard control
Intensive control
Mean A1C
7.0%
6.3%
0 6 12 18 24 30 36 42 48 54 60 66
Follow-up (months)
ADVANCE Collaborative Group N Engl J Med 358:2560-72;2008

Tip 2 Diyabetli Hastalarda Kan Şekerinin Daha Agresif Kontrolü

Table 1 Summary of the main features and results of the most recent intervention trials in type 2 diabetic patients

Variable	VADT (n=1,700)	ACCORD (n=10,250)	ADVANCE (n=11,140)
HbA_{1c} (%)[a]	8.4 vs 6.9	7.5 vs 6.4	7.3 vs 6.5
Primary outcome	MI, stroke, death from CV causes, new or worsening CHF, revascularisation[b] and inoperable CAD, amputation for ischaemic gangrene	Non-fatal MI, non-fatal stroke, CVD death	Non-fatal MI, non-fatal stroke, CVD death
HR (95% CI) for primary outcome	0.87 (0.730–1.04)	0.90 (0.78–1.04)	0.94 (0.84–1.06)
HR (95% CI) for mortality	1.065 (0.801–1.416)	1.22 (1.01–1.46)[b]	0.93 (0.83–1.06)

[a]Conventional vs intensive

[b]p=0.04

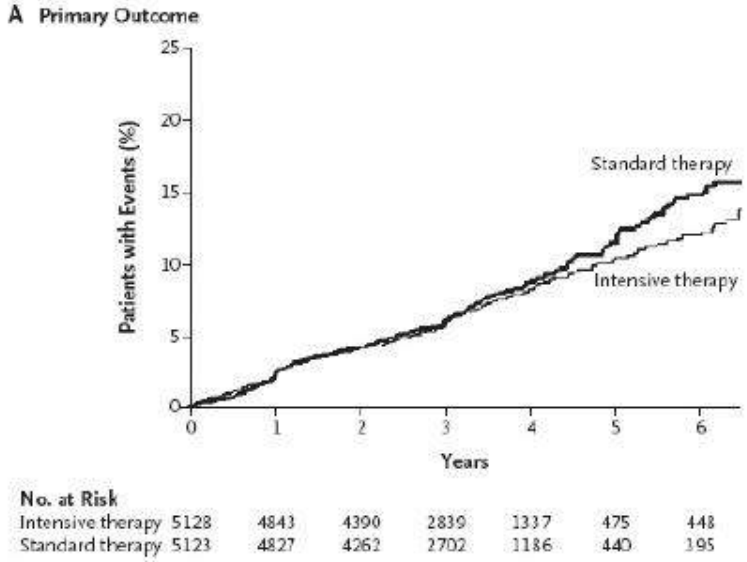
CAD, coronary artery disease; CHF, congestive heart failure; CVD, cardiovascular disease; MI, myocardial infarction

The Action to Control Cardiovascular Risk in Diabetes Study Group (2008) Effects of intensive glucose lowering in type 2 diabetes. N Engl J Med 358:2545–2559

The ADVANCE Collaborative Group (2008) Intensive blood glucose control and vascular outcomes in patients with type 2 diabetes. N Engl J Med 358:2560–2572

Duckworth W, Abraira C, Moritz T et al (2009) Glucose control and vascular complications in veterans with type 2 diabetes. N Engl J Med 360:129–139

Accord:Primer Sonlanım Noktası ve Tüm Nedenlere Bağlı Ölüm

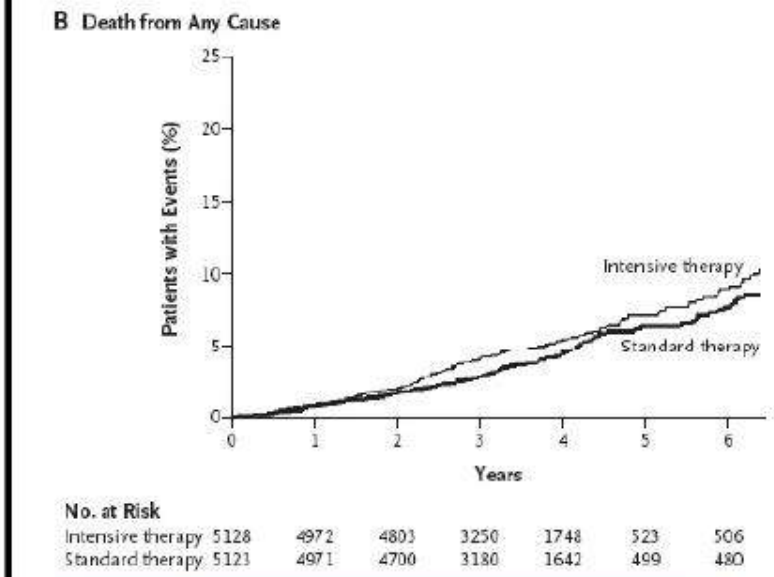


Composite primary outcome

Nonfatal MI + nonfatal stroke + death from CV causes

(6.9% in Intensive vs. 7.2% in std therapy group
HR 0.90 CI 0.78-1.04, p: 0.16)

Not significant



Death from any cause

Intensive vs. Std
257 vs. 203
5 % vs. 4 % , HR 1.22 95 %
CI : 1.01-1.46, p=0.04)

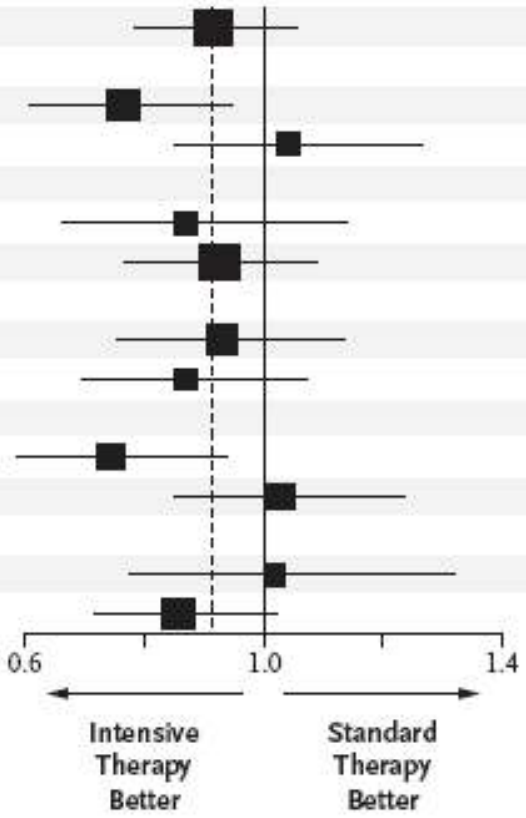
The Action to Control Cardiovascular Risk in Diabetes (ACCORD), New Engl J Med 2008

ACCORD Altgrup Analizi

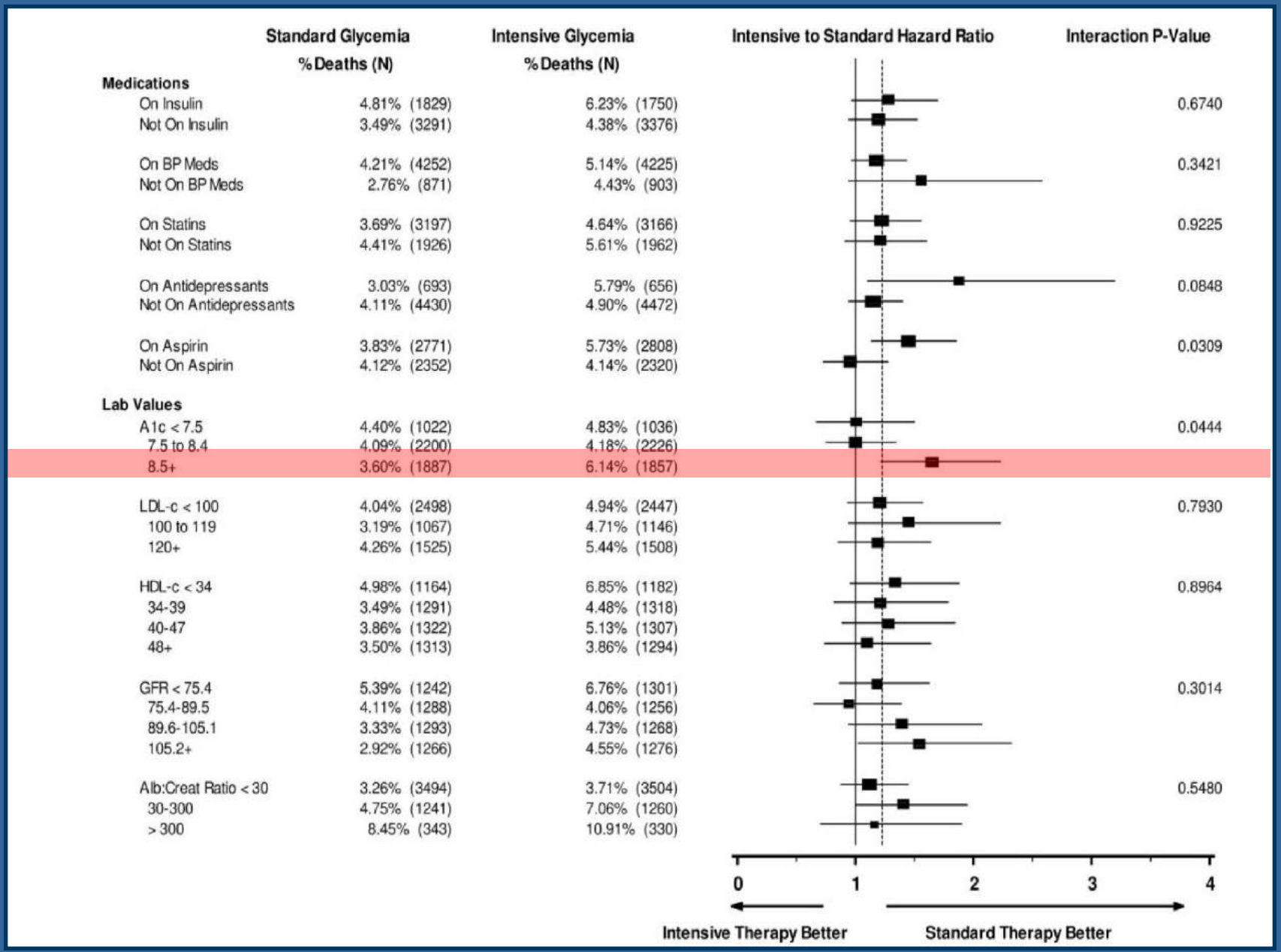
Daha önce KVH olmayan ve başlangıçtaki A1C ≤%8.0 olan hastalarda intensif glisemik kontrol KV olayları azaltmıştır.

A Primary Outcome

Subgroup	No. of Patients	No. of Events	P Value
Total	10,251	723	
Previous cardiovascular event			0.04
No	6,643	330	
Yes	3,608	393	
Sex			0.74
Female	3,952	212	
Male	6,299	511	
Age at baseline			0.65
<65 yr	6,779	383	
≥65 yr	3,472	340	
Glycated hemoglobin at baseline			0.03
≤8.0%	4,868	284	
>8.0%	5,360	438	
Race			0.29
Nonwhite	3,647	222	
White	6,604	501	

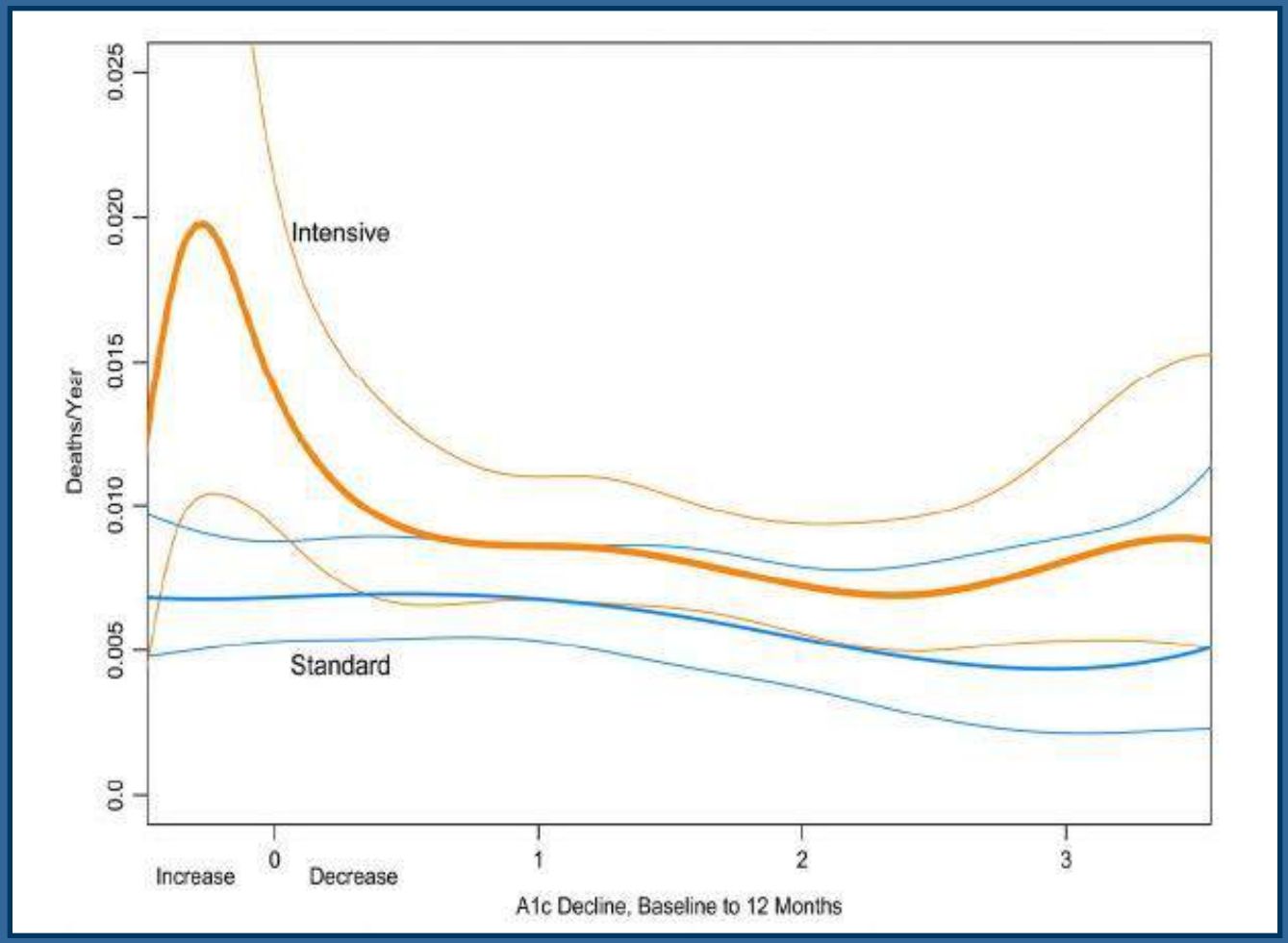


The Action to Control Cardiovascular Risk in Diabetes (ACCORD), New Engl J Med 2008



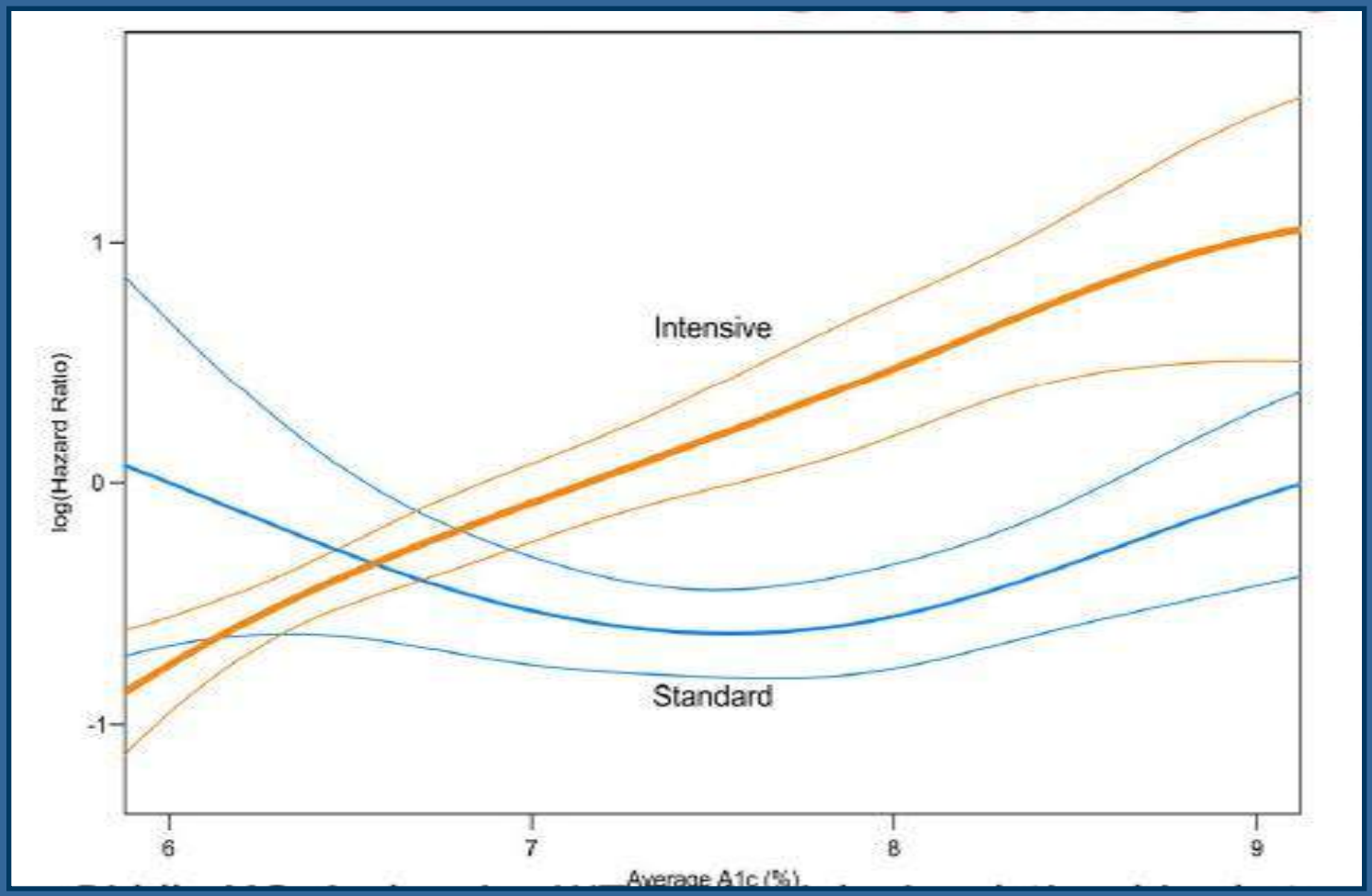
The Action to Control Cardiovascular Risk in Diabetes (ACCORD) Diabetes Care 2010

Accord: Tedavi Stratejisine Göre Mortalite Oranları



Riddle Mc, Diabetes Care 2010

Accord: Tedavi Stratejisine Göre log HR Oranları



Riddle Mc, Diabetes Care 2010

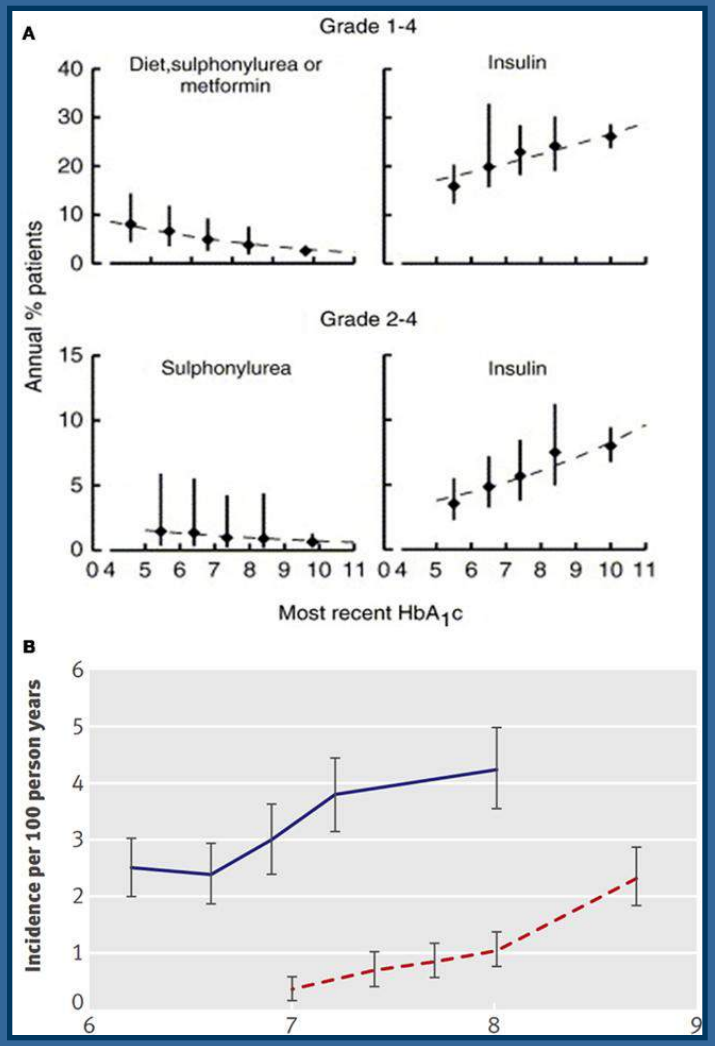
Accord: Hipoglisemi Sıklığına Göre Mortalite Oranları

	Mortality (% per year)[1]
≥1 severe hypoglycemia (n = 705)	3.1
No hypoglycemia (n = 9,546)	1.2

[a] Defined by requirement for medical or paramedical intervention, with documented glucose <50 mg/dL and relief by parenteral or oral glucose or by glucagon.

Bloomgarden ZT, Diabetes Care 2008

UKPDS&Accord: Ulaşılan HbA1c ve Hipoglisemi Sıklığı

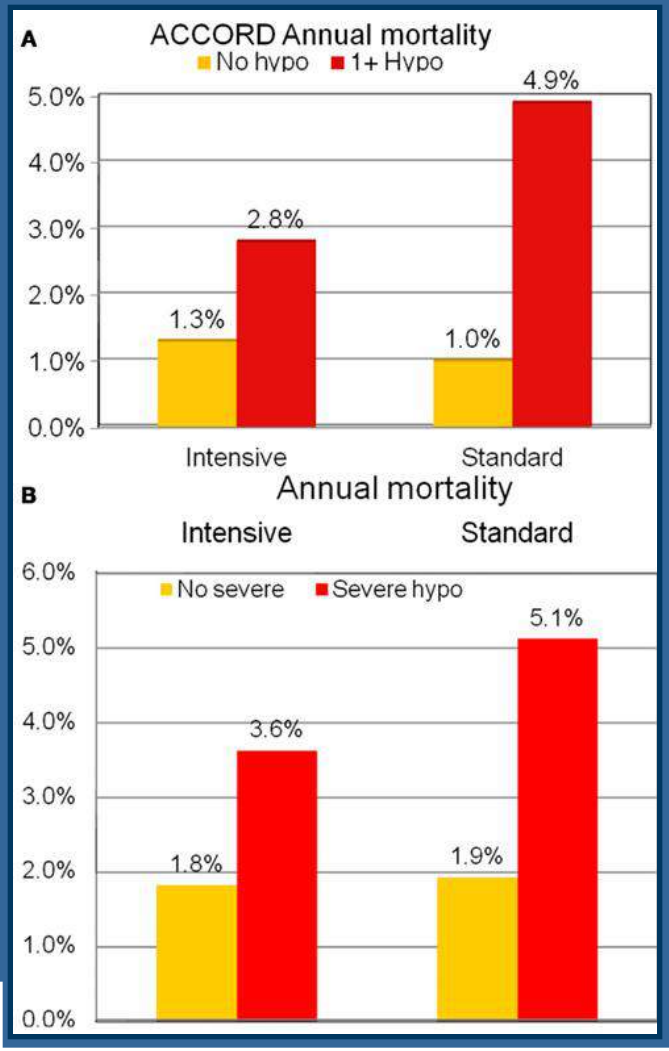


A Paneli: UKPDS Hemmingsen 2011

B Paneli: ACCORD Bonds 2010

Zachary T, Frontiers in Endocrinology, 2012

Accord&Advance: Hipoglisemi Sıklığına Göre Mortalite Oranları

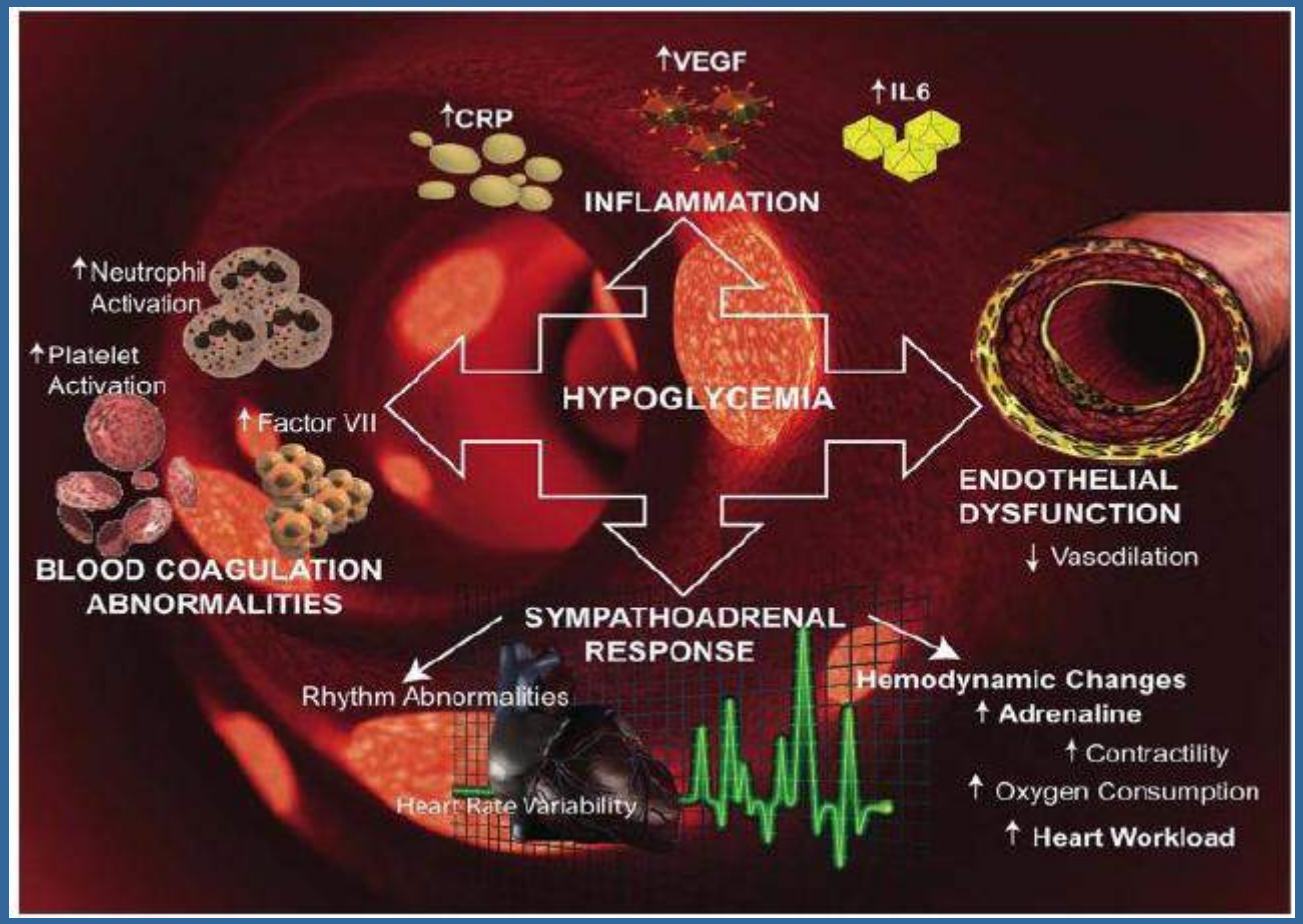


A Paneli:
ACCORD
Bonds 2010

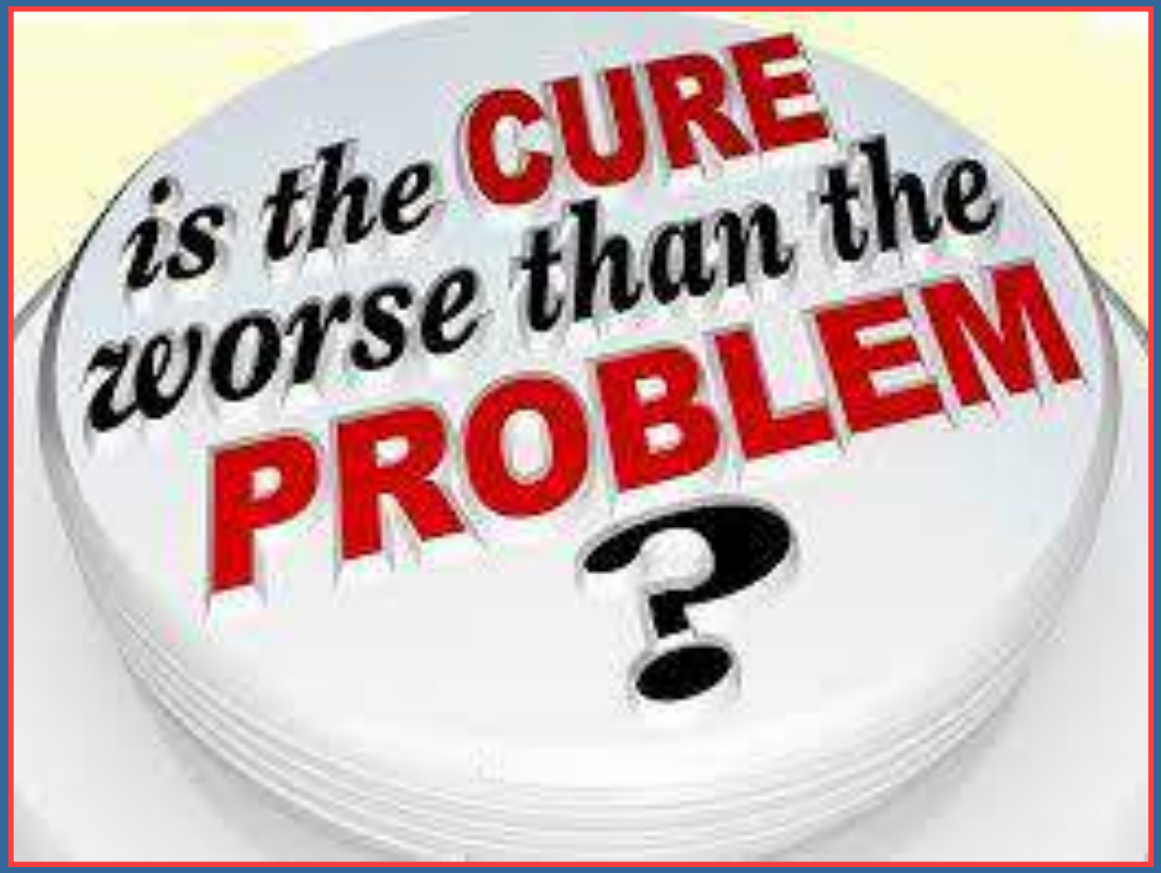
B Paneli:
ADVANCE
Holman 2009

Zachary T, Frontiers in Endocrinology, 2012

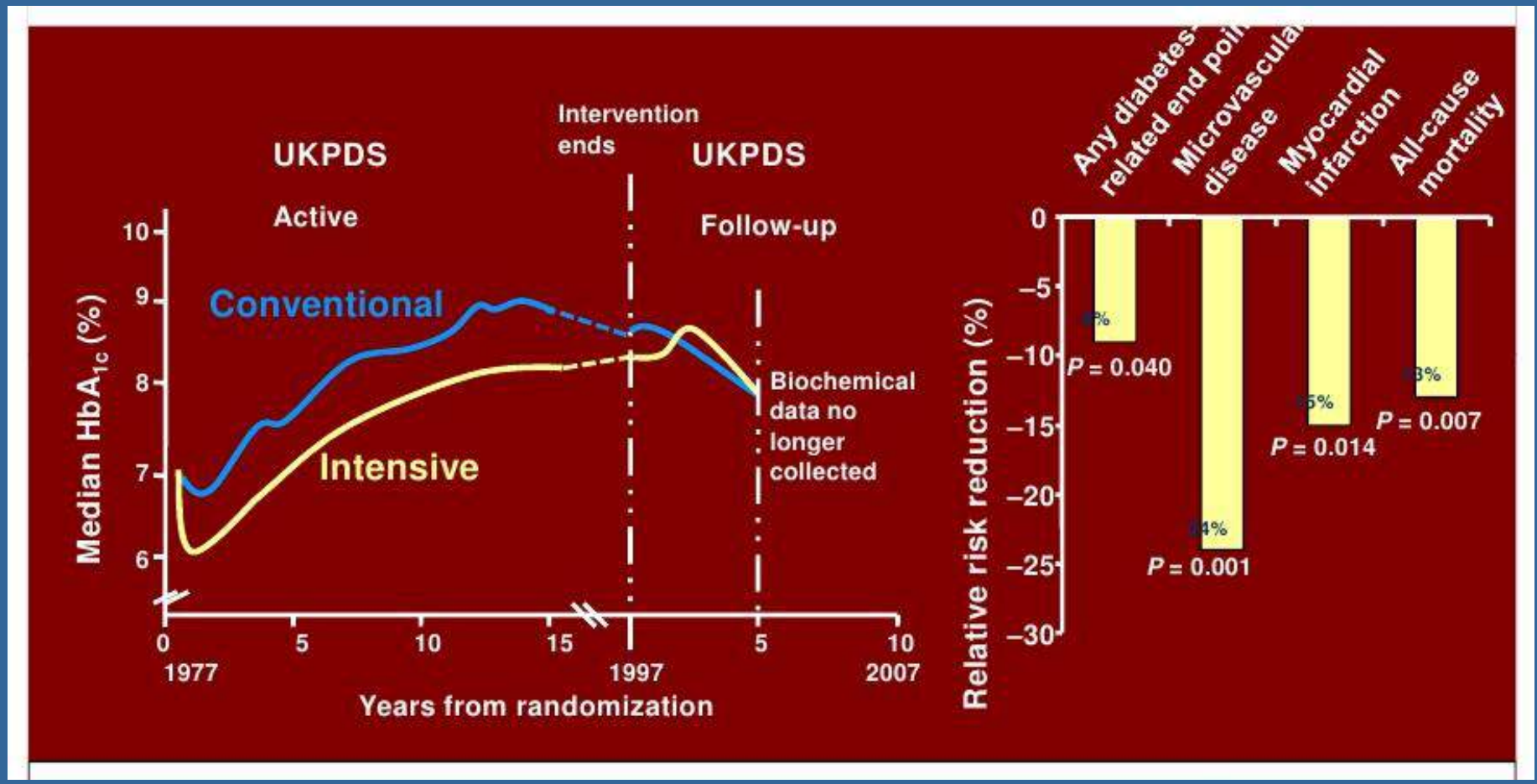
Hipoglisemi ve Kardiyovasküler Olaylar



Souza CV, Diabetes Care, 2010


is the CURE
worse than the
PROBLEM
?

UKPDS Post-Treatment Analizleri: Miras Etkisi



Holman RR et al NEJM 2008;359:1577-89

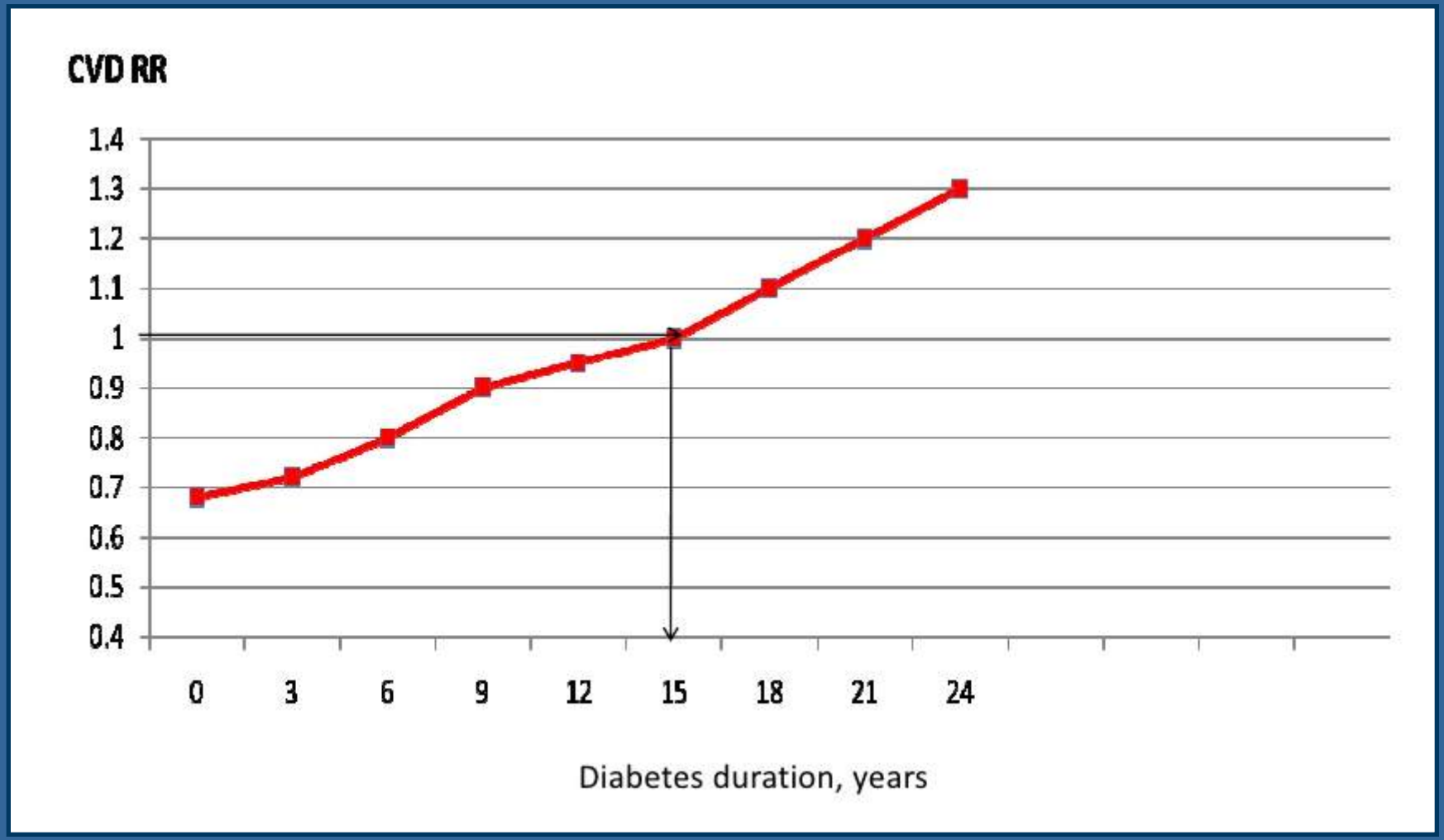
UKPDS Post-Treatment Analizleri: Miras Etkisi

After median 8.5 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:	0.0099	**0.001**
Myocardial infarction	*RRR:*	16%	**15%**
	P:	0.052	**0.014**
All-cause mortality	*RRR:*	6%	**13%**
	P:	0.44	**0.007**

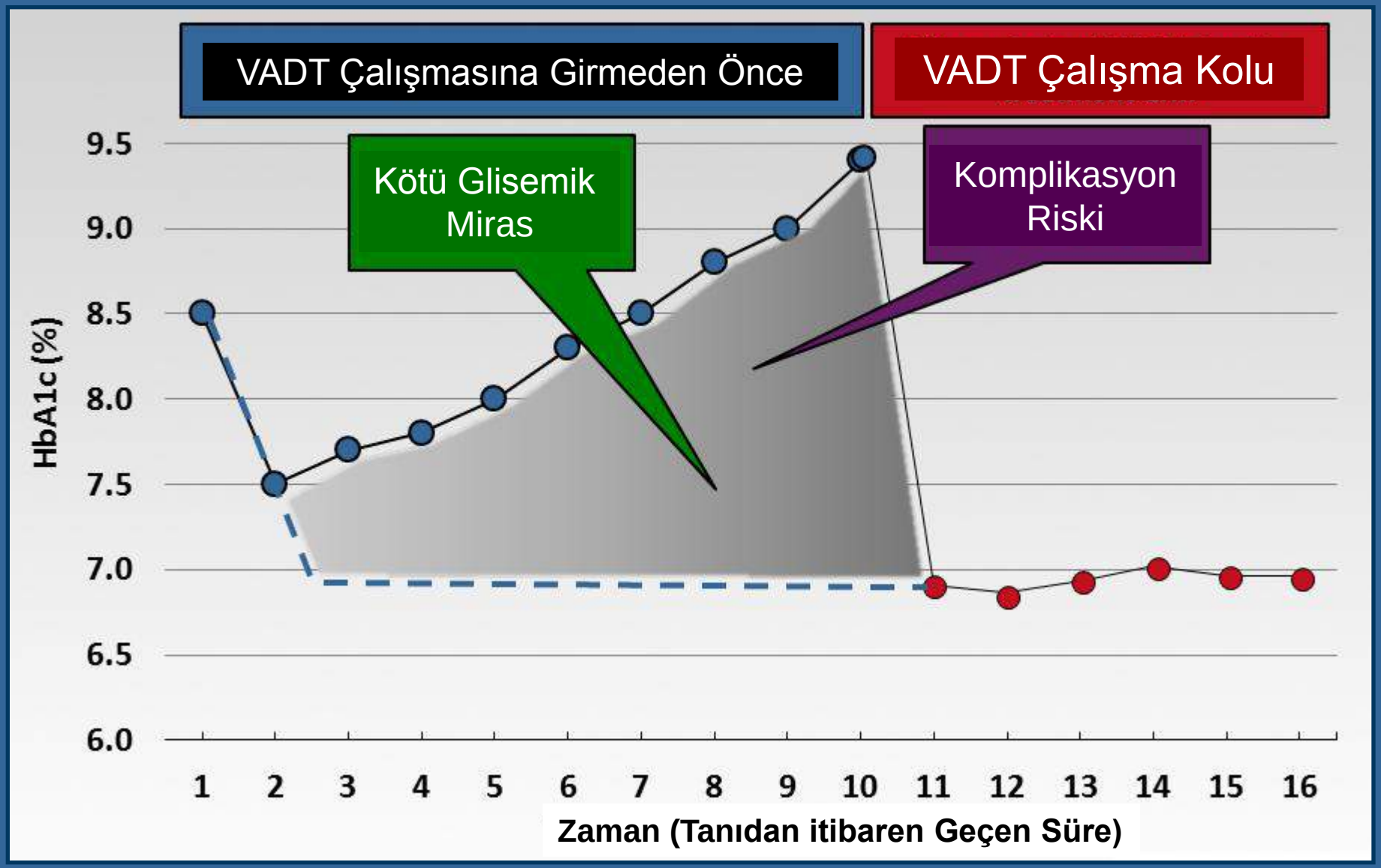
Holman RR et al NEJM 2008;359:1577-89

Gecikmiş Müdahalenin Sonuçları: VADT Çalışması: Diyabet Süresi ve Yoğun Tedavinin KV Yararları



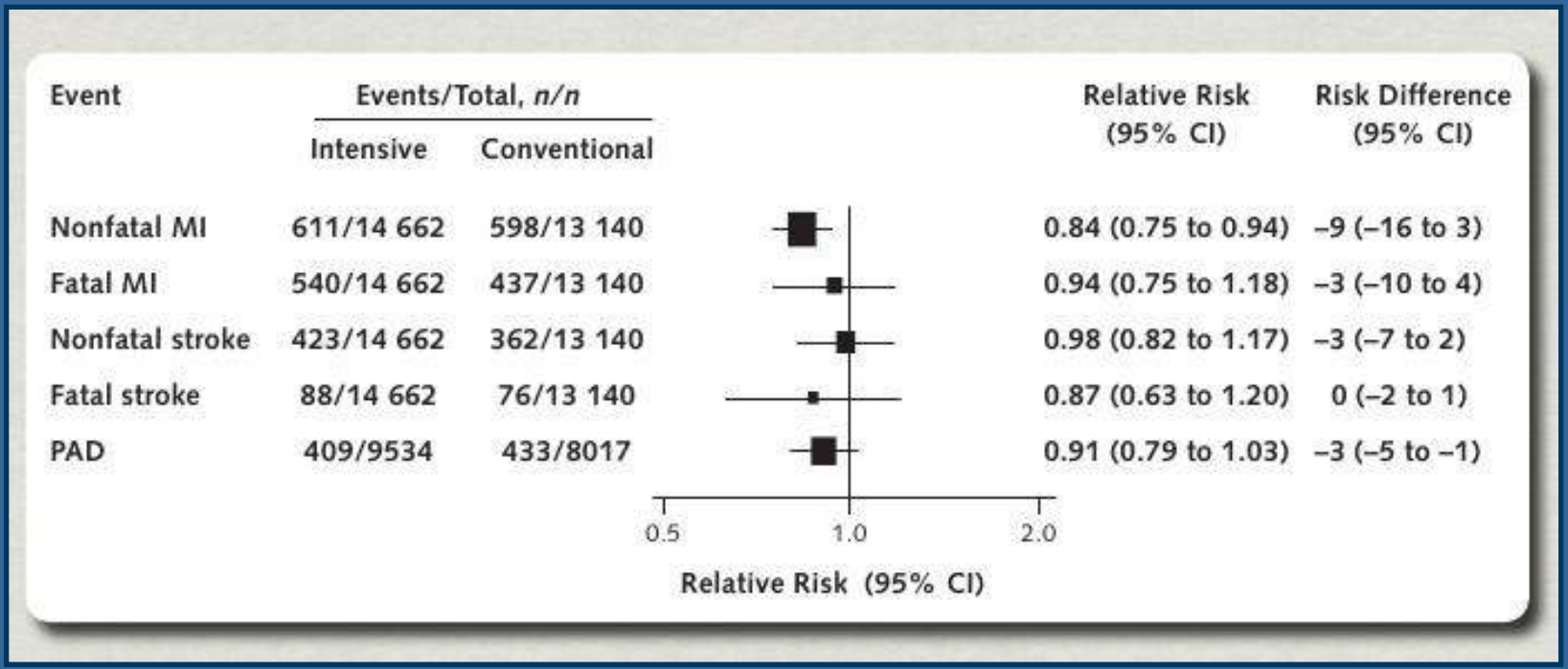
Duckworth W, N Engl J Med, 2009

Gecikmiş Müdahalenin Sonuçları: VADT Çalışması



Duckworth W, N Engl J Med, 2009

Agresif Tedavi ve Kardiyovasküler Sonlanım Noktaları



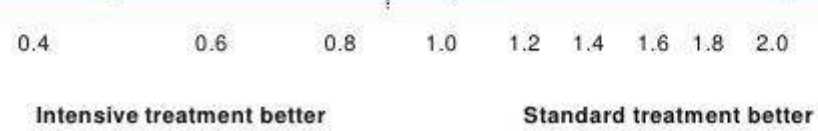
Event	Events/Total, *n/n*		Relative Risk (95% CI)	Risk Difference (95% CI)
	Intensive	Conventional		
Nonfatal MI	611/14 662	598/13 140	0.84 (0.75 to 0.94)	−9 (−16 to 3)
Fatal MI	540/14 662	437/13 140	0.94 (0.75 to 1.18)	−3 (−10 to 4)
Nonfatal stroke	423/14 662	362/13 140	0.98 (0.82 to 1.17)	−3 (−7 to 2)
Fatal stroke	88/14 662	76/13 140	0.87 (0.63 to 1.20)	0 (−2 to 1)
PAD	409/9534	433/8017	0.91 (0.79 to 1.03)	−3 (−5 to −1)

Yoğun glukoz kontrolü KV ölüm, ve tüm nedenlere bağlı ölüm oranlarını azaltmamış ancak hipoglisemi riskinde ciddi bir artışa neden olmuştur

Kelly, Ann Intern Med, 2009

Agresif Tedavi ve Koroner Arter Hastalığı

	Intensive treatment/standard treatment		Odds ratio (95% CI)	Odds ratio (95% CI)
	Participants	Events		
UKPDS	3,071/1549	426/259		0.75 (0.54–1.04)
PROactive	2,605/2633	164/202		0.81 (0.65–1.00)
ADVANCE	5,571/5,569	310/337		0.92 (0.78–1.07)
VADT	892/899	77/90		0.85 (0.62–1.17)
ACCORD	5,128/5123	205/248		0.82 (0.68–0.99)
Overall	**17,267/15,773**	**1,182/1,136**		**0.85 (0.77–0.93)**



*Included non-fatal myocardial infarction and death from all cardiac mortality.

Ray KK, Lancet, 2009

Tip 2 Diyabette Agresif Tedavi

SUMMARY OF FINDINGS FOR THE MAIN COMPARISON *[Explanation]*

Intensive glycaemic control versus conventional glycaemic control for type 2 diabetes mellitus

Patient or population: participants with type 2 diabetes mellitus
Settings: mostly outpatients
Intervention: intensive glycaemic control versus conventional glycaemic control

Outcomes	Illustrative comparative risks* (95% CI)		Relative effect (95% CI)	No of participants (studies)	Quality of the evidence (GRADE)	Comments
	Assumed risk	Corresponding risk				
	Control	Intensive glycaemic control versus conventional glycaemic control				
All-cause mortality Follow-up: median 24 months	95 per 1000	95 per 1000 (87 to 103)	RR 1 (0.92 to 1.08)	34325 (24)	⊕⊕⊕◯ moderate[a]	-
Cardiovascular mortality Follow-up: median 27 months	45 per 1000	48 per 1000 (42 to 55)	RR 1.06 (0.94 to 1.21)	34177 (22)	⊕⊕⊕◯ moderate[a]	-
Non-fatal myocardial infarction Follow-up: median 60 months	48 per 1000	41 per 1000 (37 to 47)	RR 0.87 (0.77 to 0.98)	30417 (14)	⊕⊕⊕◯ moderate[a]	-
Non-fatal stroke Follow-up: median 54.6 months	29 per 1000	29 per 1000 (25 to 35)	RR 1 (0.84 to 1.19)	30003 (13)	⊕⊕⊕◯ moderate[b]	-

Hemmingsen B, Cochrane Library, 2013

Tip 2 Diyabette Agresif Tedavi

Amputation of lower extremity Follow-up: median 65.1 months	**13 per 1000**	**9 per 1000** (6 to 12)	**RR 0.65** (0.45 to 0.94)	11200 (11)	⊕⊕◯◯ **low**[c]	-
End-stage renal disease Follow-up: median 93.6 months	**16 per 1000**	**14 per 1000** (11 to 17)	**RR 0.87** (0.71 to 1.06)	28145 (8)	⊕⊕⊕◯ **moderate**[b]	-
Hypoglycaemia - Severe hypoglycaemia Follow-up: median 12 months	**29 per 1000**	**64 per 1000** (45 to 91)	**RR 2.18** (1.53 to 3.11)	28794 (17)	⊕⊕⊕⊕ **high**	Trial sequential analysis showed firm evidence for a 30% relative risk increase with intensive glycaemic control

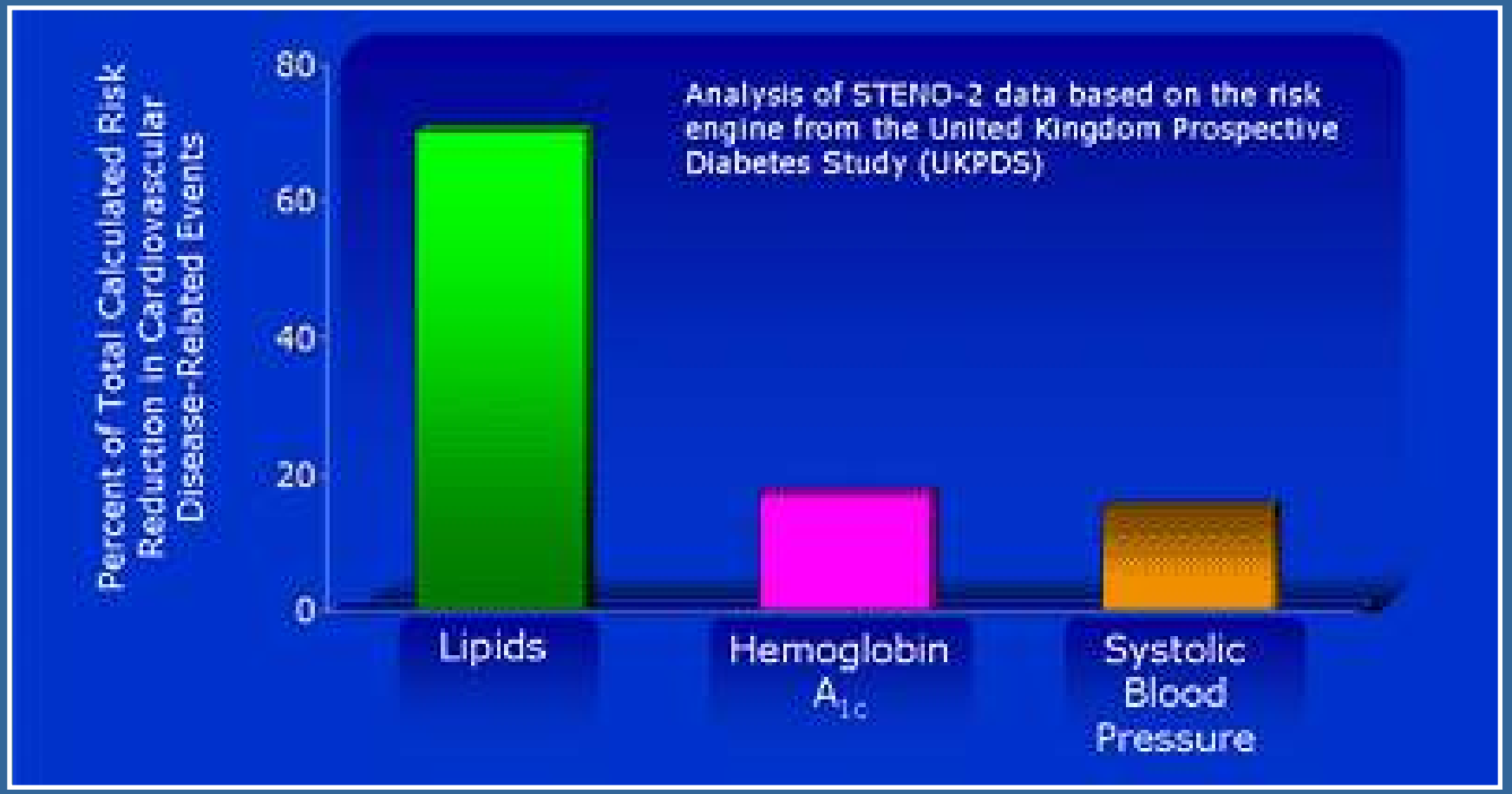
*The basis for the **assumed risk** (e.g. the median control group risk across studies) is provided in footnotes. The **corresponding risk** (and its 95% confidence interval) is based on the assumed risk in the comparison group and the **relative effect** of the intervention (and its 95% CI).
CI: Confidence interval; **RR:** Risk ratio

GRADE Working Group grades of evidence
High quality: Further research is very unlikely to change our confidence in the estimate of effect.
Moderate quality: Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.
Low quality: Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate.
Very low quality: We are very uncertain about the estimate.

[a] Downgraded by one due to trial sequential analysis showing that more data are needed; funnel plot indicates small trial bias
[b] Downgraded by one due to a relatively few number of participants reporting non-fatal stroke or end-stage renal disease
[c] Downgraded by two due to trial sequential analysis showing that more data are needed, a relatively few number of participants reporting amputation of lower extremity and most of the events stemming from one trial only

Hemmingsen B, Cochrane Library, 2013

STENO-2: Tip 2 Diyabetik Hastalarda Yoğun Tedavi ile KV Risk Azaltımında Temel Etki Gösteren Lipid Düşürücü Tedavidir



Goede P, Diabetes, 2004

Yeni Tanı Alan Hasta

- ✓ Agresif glisemik kontrol
- ✓ Düşük hipoglisemi riski olan ajanlar seçilmeli
- ✓ HbA1c hedefi < 6.5-7 %
- ✓ Beta hücre koruyucu tedavi
- ✓ Kardiyovasküler risk faktörlerinin kontrolü
- ✓ HbA1c>9% ise insülin düşünülebilir

UKPDS

Miras Etkisi

Del Prato S, Int J Clin Pract, 2010

Kötü Glisemik Kontrolü Olan Ancak Ko-Morbiditesi Olmayan Hasta

- ✓ Kötü metabolik miras
- ✓ Bir veya daha fazla mikrovasküler komplikasyon

- ✓ Agresif glisemik kontrol
- ✓ HbA1c kademeli düşürülmeli
- ✓ Diyabet eğitimi
- ✓ Hipoglisemi riski değerlendirilmeli

UKPDS

Miras Etkisi

Del Prato S, Int J Clin Pract, 2010

Koroner Arter Hastalığı Olan Hasta

- ✓ Uzun süreli diyabet öyküsü
- ✓ Kötü glisemik kontrol
- ✓ Aşırı ilaç yükü

- ✓ İyi glisemik kontrolün faydaları ve hipoglisemi riski karşılaştırılmalı
- ✓ HbA1c kademeli düşürülmeli
- ✓ Tedavi ajanlarının kontraendikasyonları
- ✓ Hipoglisemi riski değerlendirilmeli

ACCORD

VADT

ADVANCE

Del Prato S, Int J Clin Pract, 2010

Hipoglisemi Riski Olan Hasta

- ✓ Uzun süreli diyabet öyküsü
- ✓ Daha önce hipoglisemi öyküsü
- ✓ Kreatinin klirensi azalmış
- ✓ Düzensiz beslenme/yaşam alışkanlıkları

- ✓ Daha gevşek HbA1c hedefleri
- ✓ HbA1c kademeli düşürülmeli
- ✓ Hipoglisemi riski düşük ajanlar seçilmeli
- ✓ Hipoglisemi riski değerlendirilmeli

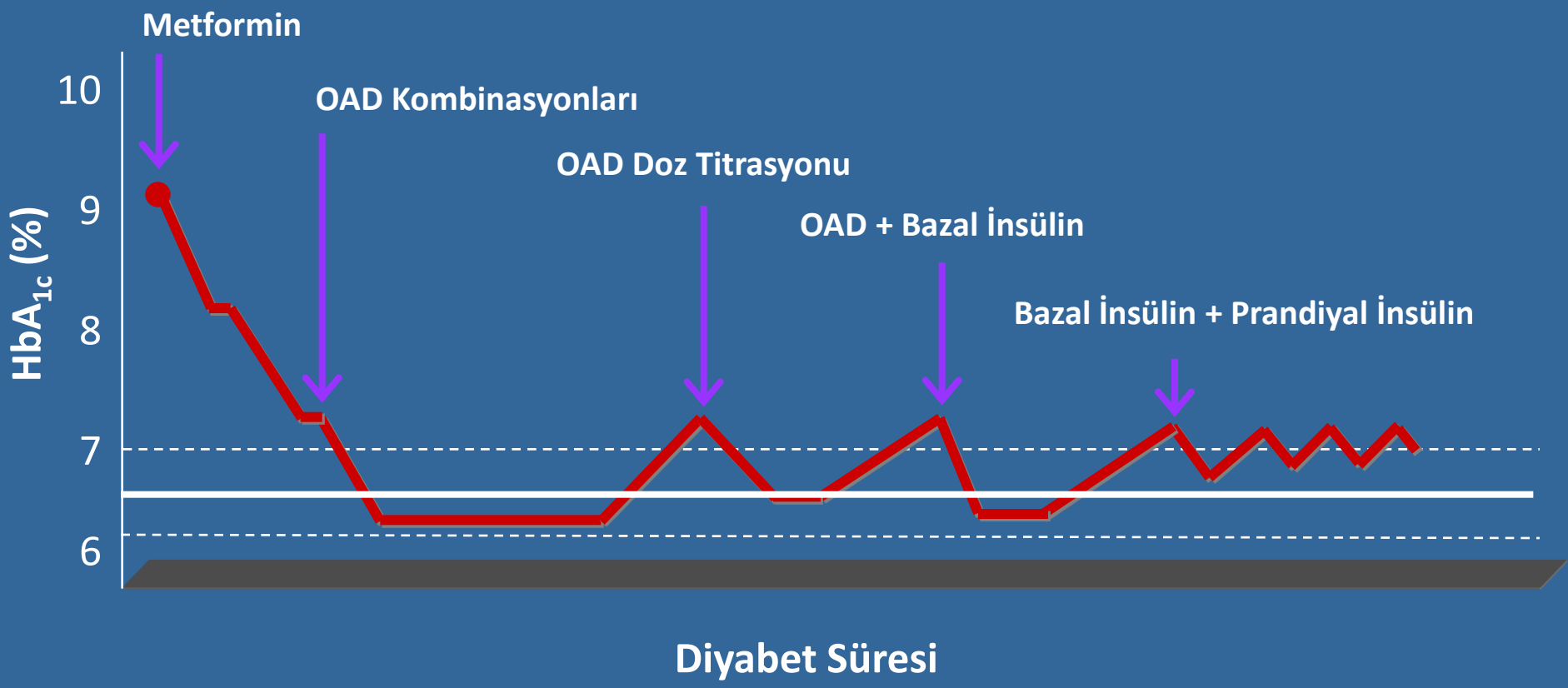
ACCORD

VADT

ADVANCE

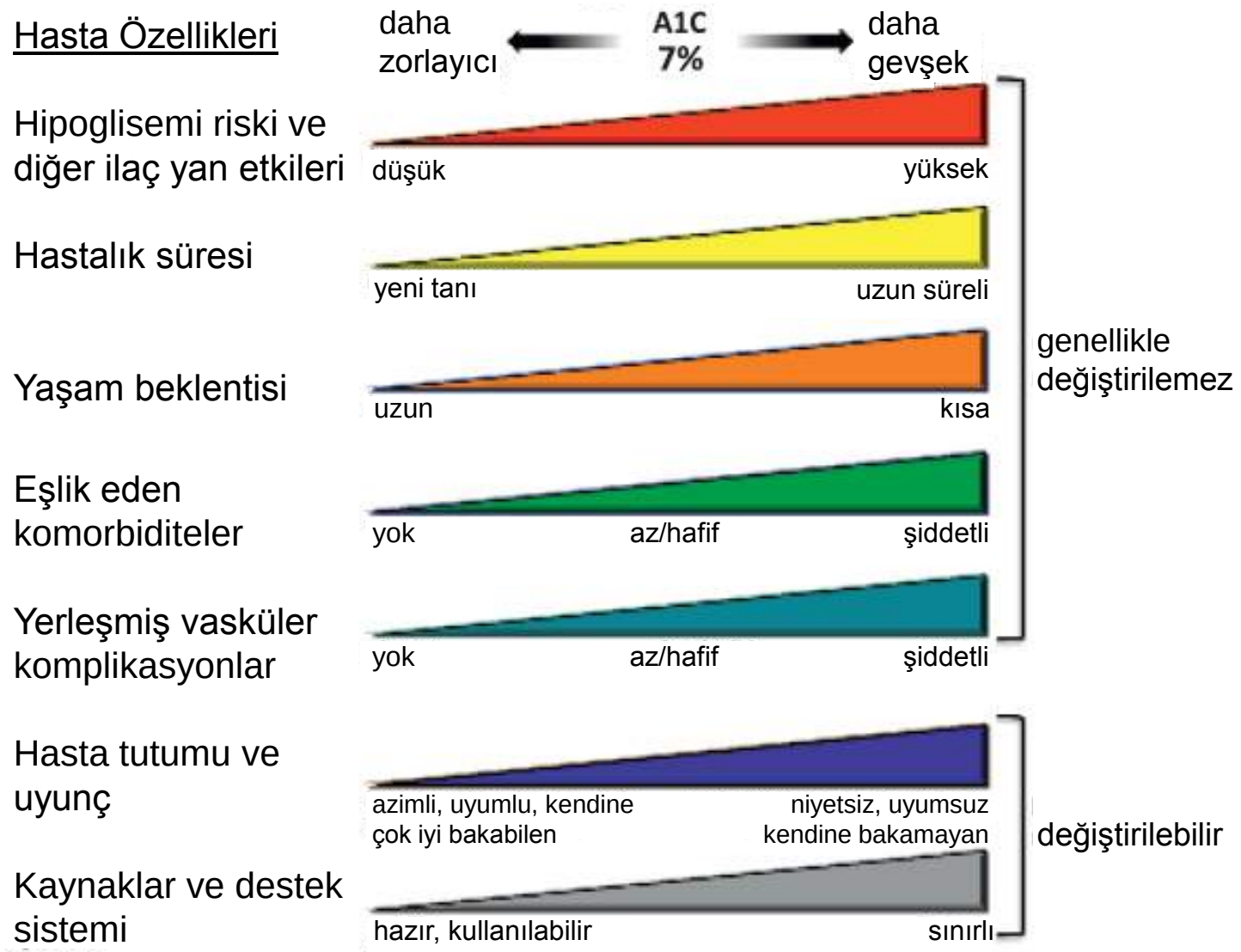
Del Prato S, Int J Clin Pract, 2010

Glukoz Kontrol Süreci



Del Prato S, Int J Clin Pract, 2005

Hiperglisemi Yönetimi



A1C ve Ortalama Glisemi

Table 6.1—Mean glucose levels for specified A1C levels (21,25)

A1C (%)	Mean plasma glucose* mg/dL	Mean plasma glucose* mmol/L	Mean fasting glucose mg/dL	Mean premeal glucose mg/dL	Mean postmeal glucose mg/dL	Mean bedtime glucose mg/dL
6	126	7.0				
<6.5			122	118	144	136
6.5–6.99			142	139	164	153
7	154	8.6				
7.0–7.49			152	152	176	177
7.5–7.99			167	155	189	175
8	183	10.2				
8–8.5			178	179	206	222
9	212	11.8				
10	240	13.4				
11	269	14.9				
12	298	16.5				

A calculator for converting A1C results into eAG, in either mg/dL or mmol/L, is available at http://professional.diabetes.org/eAG.
*These estimates are based on ADAG data of ~2,700 glucose measurements over 3 months per A1C measurement in 507 adults with type 1, type 2, and no diabetes. The correlation between A1C and average glucose was 0.92 (25).

“intensive diabetes management is a remedy primarily for the wise and not for the foolish, whether they be patients or doctors.” Joslin, 1928