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I hope you will find *Practical Pointers* interesting and helpful.

Richard T. James Jr. M.D.

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5-1 BLOOD PRESSURE AND INCIDENCE OF TWELVE CARDIOVASCULAR DISEASES (CVD): Lifetime Risks, Healthy Life-years Lost, and Age-specific Associations in 1.25 Million People

High blood pressure was the leading risk factor for the overall burden of disease in 2010. The recent decline in cardiovascular mortality in high income countries has been associated with wider use of preventive drugs and a rise in the numbers of patients living with CVD.

This study was based on a large number of linked electronic health records with high prevalence of BP-lowering treatments, and with BP measurements done as part of usual clinical practice.

STUDY

1. Used electronic health records from 1997 to 2010 to assemble a cohort of 1.25 million primary care patients age 30 and over. All were initially free of CVD. One fifth received BP lowering treatments.
2. Studied the age-specific associations of clinically measured BP with 12 chronic CVDs in 225 primary care practices, and estimated the lifetime risks (up to age 95) and healthy years of life lost due to hypertension-related CVD, adjusted for other risk factors, at index ages 30, 60, and 80 years.
3. Baseline BP and other CVD risk factors were recorded. Baseline BP was determined by readings taken within 2 years of the index date, averaged across repeated measurements.
4. Patients were classified as having hypertension if their baseline BP was 140/90 or higher, or they had a diagnosis of hypertension, or received prescriptions for BP-lowering drugs.
5. Endpoints were the initial presentations of CVD as any of 12 CVDs diagnosed.
6. The 12 CVDs:
Stable angina; unstable angina, myocardial infarction, unheralded coronary heart disease death, heart failure, cardiac arrest/sudden cardiac death, transient ischemic attack, ischemic stroke, subarachnoid hemorrhage, intracranial hemorrhage, peripheral arterial disease, abdominal aortic aneurysm.
7. Primary analysis: Associations of each CVD with every 20/10 mm Hg increase in BP in 3 different age groups (30-59, 60-79, and 80 and older). Secondary analysis: Associations of BP with different outcomes after adjustment for smoking, diabetes, total cholesterol, high-density lipoprotein cholesterol, body mass index, and baseline treatment with BP-lowering drugs.

RESULTS

1. The original cohort contained 1 937 360 individuals. After exclusions, 1 258 006 remained for analysis (58% women). There were large variations in terms of patients' ethnic backgrounds, socio-economic status, and uptake of BP-lowering treatments.
2. A total of 83 098 had first cardiovascular events during a median follow-up of 5.2 years.
3. Effect of increasing systolic BP on 12 CVDs at different age:.

For all 12 CVDs, risk increased in a progressive manner as systolic BP increased.

For example, hazard ratios (HR; compared to systolic 115) for ischemic stroke:

	Age 30-59	Age 60-79	Age 80+ (Age at entry into the study)
Systolic	HR	HR	HR
90-114	0.84	0.89	1.01
115-129	1.09	1.05	1.00
130-139	1.41	1.17	1.00
140-159	2.09	1.41	1.04
160-179	3.48	2.00	1.22
180+	5.82	2.88	1.48

As systolic BP increased, risk of ischemic stroke increased.

As age of entry into the study increased, risk also increased, but at a much slower rate in the elderly.

4. Similar patterns were observed for diastolic BP, although at a slower progression rate than for systolic.

For all 12 CVDs, rate of incidence increased in a progressive manner as diastolic BP increased.

For example, hazard ratios (HR compared to diastolic 70) for ischemic stroke:

	Age 30-59	Age 60-79	Age 80+
Diastolic	HR	HR	HR
60-74	0.83	0.85	0.93
75-84	1.11	1.08	1.03
85-89	1.49	1.33	1.13
90-94	1.85	1.57	1.22
95-99	2.30	1.86	1.30
100+	3.20	2.41	1.44

As diastolic BP increased, risk of ischemic stroke increased.

As age of entry into the study increased, risks also increased, but at a much slower rate in the elderly.

5. There was considerable heterogeneity in the relations between different CVDs and HRs at different BP levels. For example, for myocardial infarction, HR for systolic 140-159 was 1.89 at entry age 30-59; 1.69 at age 60-79; and 1.44 at age 80+.

6. For all 12 CVDs, lifetime risk rose gradually with age beginning at age 30, for both those with and those without hypertension, but at a steeper rate for those with hypertension.

7. Years of life-lost due to total CVD associated with hypertension (140/90 and above) age 30-95:

Isolated systolic hypertension	1 year
Systolic and diastolic hypertension	3.5 years

8. There was no evidence of a “J-shaped” increase in risk at lower BPs.

9.. After age 30, people with hypertension (140/90 and above) or those receiving BP-lowering treatment, had a 63% lifetime risk of CVD compared with 46% for those with normal BP, and they developed CVD 5 years earlier.

10. Stable and unstable angina accounted for most (43%) of the years of life living with hypertension-associated CVD from index age 30. Heart failure and stable angina accounted for the largest proportion from index age 80.

DISCUSSION

1. In this study of 1.25 million patients across 83 000 events of 12 different CVDs during a median follow-up of 5.2 years, the lifetime burden of hypertension remained substantial despite modern therapy.
2. Across different diseases and different ages there was substantial heterogeneity of CVDs in associations with BP.
3. For nearly all CVDs, there was a relative decrease in risks as age at entry into the study increased.
4. There were significant and sustained risk reductions for CVD endpoints with lowering of BP.
5. Substantial debate has surrounded the benefit of treatment of mild (stage 1) hypertension in young people, especially those without evidence of target organ damage or those calculated to have a low 10-year risk of CVD. In the absence of long-term randomized primary prevention trials, the estimates of lifetime risk and years living with CVD provide epidemiological evidence of a substantial morbidity associated with raised BP, irrespective of the baseline age.
6. Isolated systolic and combined systole-diastolic hypertension accounted for the largest proportion of years lived with CVD, even for those age 30-59 in whom the presence of isolated diastolic hypertension was the highest. Thus, the estimates of this study support the shift in guidelines in recent years from the importance of diastolic toward the greater importance of systolic in people age 60 and older.
7. This study has important clinical implications:

The primary purpose of assessment of CVD-risk is to provide the basis of a risk discussion with the patient. The present estimates of lifetime risks and years living with CVD can be used to extend counseling of patients and decision-making, rather than estimates based on heart attacks or stroke alone. This study shows the importance of other CVDs that might be more common. Of the 5 years of life lived with hypertension-associated CVD, nearly half were attributable to angina. In the 80+ age group, heart failure accented for nearly 1/5 of the years.

The study also emphasizes an unmet need of existing BP lowering strategies as shown by the width of separation of the lifetime risk curves in people with and without hypertension. Better mitigation of excess risks could come from better implementation of existing BP-lowering treatments and through better management of other CVD risk factors.
8. An important need also exists for new interventions. Heart failure and peripheral arterial disease are among the most common initial presentations of CVD, but are included less frequently in the primary outcomes of BP-lowering trials. The need for vigorous treatment as part of a package of risk reduction remains undiminished.

CONCLUSION

Systolic and diastolic BP show heterogeneous associations across a wide range of acute and chronic CVDs and at different ages and BP levels. BP has varying associations with different CVDs.

Diastolic and systolic are not concordant in predicting risk.

Despite modern treatment, the lifetime burden of hypertension is substantial. New BP-lowering strategies are needed.

These findings have implications for the design of new trials and preventive strategies to address the substantial contemporary unmitigated lifetime burden of hypertension.

Lancet May 30, 2014;383:1899-1911 Original investigation, first author Eleni Rapsomaniki Institute of Health Information Research, London, UK

Funded by the (UK) Medical Research Council and others.

Key points:

1. Risks for 12 different CVDs increased progressively as systolic BP rose from 90 to 180+.
2. Risks also rose progressively as diastolic rose from 60 to 100+, but at a slower rate.
3. Systolic is more highly related to development of CVD than diastolic.
4. As age increased, risks of hypertension-related CVDs were actually lower than for younger persons.
5. There was no evidence that lowering systolic to 90 was associated with increased risk.
6. Years of life lost, and years of living with CVD due to hypertension-associated CVD remain high despite present day therapy. We need better.
7. Persons with hypertension (140/90 and above) had a lifetime risk of CVD of about 63% vs 46% in those without hypertension
8. The lifetime burden of hypertension-related CVD remains high despite modern treatment.
9. Even mild increases in BP in younger persons carry a high risk of CVD.
10. The present treatment goals for hypertension (systolic 140; 150 in elders) are arbitrary. Benefits may be increased at lower BPs.

These investigators must have had unending patience while wading through this mountain of data.

The years of life lost to hypertension-related CVD (up to 3.5 years) remains a major challenge, as does the lifetime risk of CVD in people with hypertension after age 30 (63% vs 46% without hypertension). This is a good argument for starting treatment of high BP (even slightly elevated BP) at an earlier age.

Present guidelines recommend a target systolic lower than 140 for patients with hypertension (150 for elders). The study points out how arbitrary these cut-points are. The study would

recommend lower levels if they can be obtained without adverse treatment effects. It also would suggest starting treatment at an earlier age. At all ages above 30, lower systolic was associated with lower risk of CVD.

Of course, all other risk factors for CVD should be assessed and treated.

5-2. SO MUCH INSULIN. SO MUCH HYPOGLYCEMIA: Commentary

Hypoglycemia is the most common and serious adverse event caused by insulin. Besides causing injury, coma, and even death, there is evidence that hypoglycemia may increase the risk of dementia many years later.

A study in this issue of *JAMA Internal Medicine* ¹ estimated the number of emergency department (ED) visits for 1000 persons taking insulin, stratifying the results by age and concurrent use of oral glucose-lowering medications.

Across all age groups, patients taking insulin alone were several times more likely to have an ED visit for hypoglycemia than were patients taking oral glucose-lowering medications with insulin.

Persons over age 80 had nearly twice the risk of hypoglycemia as younger adults.

Insulin-related hypoglycemia is remarkably common.

There are several possible explanations for the finding that using insulin alone leads to higher rates of hypoglycemia than does insulin taken with oral glucose-lowering medications:

Patients with type 1 diabetes are probably highly overrepresented in the insulin-only group and are known to be at 3 to 4 times higher risk of hypoglycemia than insulin-treated patients with type 2 diabetes. However, because less than 10% of all patients with diabetes have type 1, this segregation of patients by diabetes type is unlikely to fully account for the result.

Previous studies suggest that continuing oral medications when starting insulin leads to lower insulin requirements and less risk of hypoglycemia.

The finding that older patients are at especially high risk for hypoglycemia is not new. In the 1980s a study of patients age 65 and older reported 28 episodes of hospitalization, ED use, or death per 1000 person-years of insulin use, and for those older than 80 compared with 65-70, risk of hypoglycemia increased by 80%.

There are nearly 100 000 ED visits for insulin-related hypoglycemia annually compared to 715 000 for myocardial infarction. Unlike MI, the vast majority of hypoglycemic episodes are caused by the health care system.

The pharmaceutical industry has shaped the current belief in tight glycemic control. This has led to aggressive prescribing of glucose-lowering agents including insulin. The American Diabetes Society (ADA) endorses more intensive glycemic treatment. Achieve an “A1c <7% by 2007” encouraged patients and providers to push for intensive control.

In 2008, the ACCORD study showed that intensive glycemic control increased mortality. However, the ADA has continued to recommend an HbA1c goal of less than 7% for most patients, contributing to the belief that tight control is always better. The 50% increase in insulin use in the past decade and the resultant epidemic of insulin-related hypoglycemia (IRH) is due in part to the all-too-effective efforts to intensify glycemic treatment.

The commentator proposes 3 changes to current practice and guidelines:

1) HbA1c recommendations should be a range rather than “less than 7%”. By recommending less than 7, proponents are sending a subtle but powerful message that lower is better. A target less than 7% suggests that 6% is better, and 5% better still. However, when we treat to an overly aggressive HbA1c, (target 6.4%) increased mortality may result. Recommending a target range (eg, 6.5% to 7%) rather than “less than” would send a message that too low can be dangerous.

2) Quality indicators for glycemic overtreatment must be developed. Currently, nearly all quality indicators for diabetes care encourage healthcare providers to do more. The Healthcare Effectiveness Data and Information Set measures for 2013 encourage providers to achieve HbA1c levels less than 8%, BP less than 140/80 and LDL-cholesterol less than 100. Thus a provider who aggressively treats to these targets is a “good doctor” even if the patient develops hypoglycemia or orthostatic hypotension with syncope or severe myalgia. Quality indicators that highlight overtreatment are needed.

3) Insulin treatment should be avoided for most nonhospitalized patients older than 80. Most in this age group have significant co-morbidities, functional limitations, and limited lifetime expectancy. Because the benefits of tight glycemic control are not seen for many years, patients with limited life expectancy are exposed to immediate hypoglycemia with little chance of benefits. Although some patients in their 80s are unusually healthy and may benefit from insulin, most are likely to be harmed. Thus, the default decision should be to elect control of blood glucose levels less tightly by using oral medications.

Occasional episodes of hypoglycemia have been accepted as the price of good control. However, IRH is too common to be an acceptable price for treatment.

We should not accept the current rates of hypoglycemia as inevitable. We should begin using a multi-pronged approach to decrease the overuse of insulin and minimize the risks of hypoglycemia.

JAMA Internal Medicine May 2014; 175:686-87 Commentary by Sei J Lee, University of California, San Francisco

1 NATIONAL ESTIMATES OF INSULIN-RELATED HYPOGLYCEMIA AND ERRORS LEADING TO EMERGENCY DEPARTMENT VISITS AND HOSPITALIZATIONNS:

Report of the National Health Interview Survey (2007-2011)

First author Andrew I Geller, Centers for Disease Control and Prevention, Atlanta, GA.

1. An estimated 97 648 ED visits for insulin-related hypoglycemia (IRH) occurred annually.
2. Twenty nine % resulted in hospitalization.
3. Severe neurological sequelae were documented in an estimated 60% of ED visits for IRH.
4. Blood glucose levels of 50 mg/dL or less were recorded in more than half of cases. (53%) .
- 5 Insulin-treated patients 80 years or older were more than twice as likely to visit the ED and nearly 5 times as likely to be subsequently hospitalized for IRH than those age 45 to 64.
6. The most commonly identified IRH precipitants were reduced food intake, and administration of the wrong insulin products.
7. Number of cases and estimates of ED visits for IRH by diabetes therapy and patient age. Annual National Estimates:

Age and therapy	Insulin treatment with and without oral agents	ED visits per 1000
18-44		
Insulin only	579 860	35

Insulin + orals	291 290	3
45-64		
Insulin only	1 042 828	27
Insulin + orals	1 449 876	4
65-79		
Insulin only	688 371	27
Insulin + oral	826 706	7
80 and over		
Insulin only	242 003	50
Insulin + oral	201 493	16

This is convincing. If the patient requires insulin, primary care physicians should prescribe oral medications in addition, or start treatment with oral agents and add insulin later if needed.

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5-3. POWER IMBALANCE PREVENTS SHARED DECISION MAKING (SDM): Analysis

Adoption of SDM into routine medical practice has been slow.

SDM is unlikely to become the norm if we do not deal with the barriers that patients perceive.

Deep rooted attitudes need to be changed in order to prepare patients for a new type of clinical encounter.

Patients find it hard to speak up:

Attitudinal barriers are hindering progress in implementing SDM. Even when patients are well educated and well informed, many still find it difficult to use their knowledge to participate meaningfully in decisions about their health care. They often feel prohibited from speaking up, even when they are extremely concerned about safety and quality of care they are receiving.

Doctors are not immune to the power imbalance when they become patients, feeling they represent a disease rather than that they are an individual.

How then can we expect patients to express their preferences about treatment options—especially when they often observe doctors displaying unquestioned confidence in being able to make the best decisions on the patient’s behalf?

Knowledge is not enough:

Many patients currently feel they cannot participate in SDM, rather than that they do not want to. Having information needs met in an appropriate way is a key factor for many patients. If patients do not know about their condition, and they do not understand their available options, they cannot take part in decision making.

Many patients also do not recognize the unique expertise they bring to the clinical encounter—the knowledge about their personal preferences. Until patients believe that they are capable of understanding the information provided them and believe that personal expertise and medical expertise are equally important, they are unlikely to become actively engaged.

Some patients develop covert contracts with clinicians with whom they feel compelled to adopt the role of a “good patient”—characterized by passivity and compliance.

Many patients believe that they cannot or should not be involved in decisions, often out of fear of annoying the clinician and being labeled as a difficult patient.

How to overcome passivity;

Patient passivity has been neglected. The focus has been on supporting the process if and when a patient becomes engaged, rather than working out how to engage the patient. A SDM encounter is different from what patients are used to. We cannot expect patients to change their long established behaviors just because they are given the opportunity to participate in SDM.

Efforts have been made to increase patient participation by encouraging patients to ask:

What are my treatment options?

What are the benefits and harms?

How likely are they to happen to me?

Better preparation:

Patients perceive that their medical knowledge is inferior to that of their physician. They desire to act like a good patient out of fear that otherwise they will receive worse care.

In primary care:

Patients should be informed about SDM—what it is, what to expect, and what is appropriate.

Explain that there are two experts in the clinical encounter with different but complementary knowledge.

Challenge attitudes that there are right and wrong decisions.

Redefine perceptions of what a good patient is and reassure that participation will not result in retribution.

Promote the social acceptability of the role—confirm that clinicians want patient participation.

Build patients' belief in their ability to take part.

Importantly, an invitation to participate in SDM should be given by the clinician—"I want to know what is important to you". This indicates that their doctor is giving the patient permission to participate.

Achieving SDM in routine practice will require interventions targeted to both clinicians and patients. Clinicians will not be able to change the experience of every patient, but should try to make it easier and safer for them to feel included and respected.

Patients need to believe that they can and should be involved.

Clinicians need to make efforts to understand what matters most to patients.

BMJ May 31 2014;348:17-18 BMJ2014;348:g3178<http://>

"Analysis" first author Natallie Jopseph-Willimas, Cardiff University School of Medicine, Cardiff, Wales.

I believe SDM is a worthy approach to practice—difficult to achieve completely.

I also believe than many doctors are not enthusiastic about SDM.

The doctor-patient relationship is a contract between 2 people. Medical encounters always lead to decision-making. The doctor makes some decisions; the patient makes some. Ideally they are congruent. Often they are not.

As with business contracts, both parties must negotiate a full and transparent agreement, understanding all components. Medical decision-making is often not shared. Patients may not fully understand and may not be willing or able to comply with the entire program, but they remain silent.

We should end with a process the patient fully understands and accepts. We have a long way to go before this relationship is perfected.

Time is a barrier.

The doctor offers the best advice, and the reasoning for the advice. He explains all harms, benefits, and costs of interventions. He makes sure the patient understands it. He asks the patient if she has any questions, exceptions, reservations, or objections to the outlined testing-treatment program.

Doctors must know the patient's ethnic/racial background, and educational status. This makes a difference in the patient's understanding and the doctor's explanation.

Doctors should not expect to accomplish complete mutual understanding in one visit.

The patient states that she understands and accepts the advice, and she understands and accepts all possible harms, benefits, and costs. Or, she states her exceptions to the advice and the reasons for the exception.

If the patient voices objections, a process of re-negotiation begins.

Decisions are subject to change. The doctor must immediately inform the patient of any change. The patient often does not notify the doctor.

The "if" or "delayed" prescriptions is an example of SDM. The patient attends for sore throat and bronchitis. The doctor is not sure whether it is viral or bacterial. He gives the patient a prescription for an antibiotic with the admonition not to have it filled for a day or two while the patient observes the course of symptoms. If he gets worse, or does not improve, he should fill the prescription; if symptoms improve the prescription is not filled. Many patients never fill the prescription.

Surprisingly, many patients do not follow doctors' advice. Some never get the first prescription filled. Many do not continue taking long-term medications.

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5-4. USING HEMOGLOBIN A1c TO DIAGNOSE TYPE-2 DIABETES OR TO IDENTIFY PEOPLE AT HIGH RISK FOR DIABETES: "Practice" Rational Testing

Since the early 20th century, the diagnosis of diabetes has been based on the measurement of glucose concentrations in the blood. This usually takes the form of laboratory measured fasting plasma glucose, and when indicated, a glucose concentration 2 hours after an oral glucose load.

A "random" (post-prandial) measurement can suffice if it is unequivocally raised, especially in patients with symptoms. The diagnostic threshold for glucose used by the WHO is defined as the level above which it is known that a person will be at high risk of developing the microvascular complications of diabetes, especially retinopathy. In non-pregnant adults, the main indication for a glucose tolerance test lies between normal values and those of overt diabetes—the impaired fasting glucose range of 6.1-6.9 mmol/L (110-124 mg/dL). The 2-hour post-load glucose measurement can help to distinguish patients who have solely impaired fasting glucose from those who have both impaired fasting glucose and impaired glucose tolerance (plasma glucose 7.8 to < 11.1 mmol/L (140-199) and from those who can be diagnosed as having diabetes purely on the basis of their 2-hour glucose result being 11.1% (200 mg/dL) or above.

But measuring glucose in blood to diagnose diabetes can become inconvenient for patients, as they are usually required to fast overnight. If an oral glucose tolerance is needed, the process is laborious, time consuming, and costly.

For these reasons, in recent years, more consideration has been given to whether measurement of HbA1c might be an alternative to glucose as a diagnostic test for diabetes, although the concept has led to controversy.

Using HbA1c to diagnose type-2 diabetes:

HbA1c gives an indication of glycemia over several preceding weeks rather than at a single time point. As a consequence, day to day variations in HbA1c are much less than variations in blood glucose.

Advances in the global standardization of HbA1c measurement culminated when WHO published advice in 2011 recommending an HbA1c threshold of 48 mmol/mol (6.5%) or above for diagnosis of type-2 diabetes, but WHO did not give specific guidance below this single value.

Since then, an expert committee in the UK came to a consensus recommending that a diagnosis of diabetes should be made only after a confirmed raised HbA1c value.

The committee also introduced a new category of patients who are judged as being at high risk of developing diabetes solely on the basis of an HbA1c of 42-47 mmol/mol (6.0% to 6.4%).

When not to use HbA1c to diagnose diabetes:

One of the advantages of HbA1c is that it gives an indication of previous glycemia. This is a disadvantage when hyperglycemia may have developed rapidly, as a rise in HbA1c will lag behind rises of glucose.

Examples:

Suspected type 1 diabetes

All children and young people

Pregnancy (current or within 2 months)

Short duration of symptoms (< 2 months)

Patients at high risk of diabetes who are acutely ill.

Patients taking drugs that may cause rapid rise in glucose (eg, corticosteroids,, antipsychotics)

Acute pancreatic injury or pancreatic surgery

Patients being treated for HIV

Patients who have or may have abnormal hemoglobin

Patients with anemia (any cause)

Patients likely to have altered red cell lifespan (eg, post-splenectomy)

Patients who have had recent blood transfusion

Most laboratories are able to analyze glucose more rapidly than HbA1c, so requesting HbA1c could introduce delay in acute situations.

Other cautions:

In kidney failure the situation is complicated by patients often having a combination of hemolytic anemia, iron deficiency, and chronic inflammatory anemia as well as forming urea-derived carbamylated HbA1c, which can affect some HbA1c analyses.

HbA1c increases with age beyond what can be explained by any changes in glucose levels.

People with African-Caribbean or Asian heritage have higher HbA1c values than those of European descent, which cannot be accounted for by differences in glucose tolerance tests.

However, the relevance of these observations to the use of HbA1c as a diagnostic test remains uncertain.

Glucose or HbA1c for diagnosis?

Diagnoses made by the two tests are not completely concordant. They will not identify an identical population of people. For this reason, UK recommends that only one or the other test be used to follow the same patient and not a mixture of the two.

If HbA1c shows the patient to be at high risk of diabetes, follow-up should be made with the same test, unless the HbA1c test is identified as being inappropriate as noted above. If so, a change to glucose is warranted.

Outcome:

A patient has a HbA1c of 6.2% (44 mmol/mol)—at high risk for diabetes. He is given instructions about lifestyle and diet and assessment of other CVD risk factors. He should report any symptoms of diabetes and be rechecked periodically.

BMJ May 24,2014;348:39 “Rational Testing” first author Eric S Kilpatrick, Hull York Medical School, Hull UK

A welcome review. I can see advantages to both

I believe elder clinicians will resist changing over to HbA1c.

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5-5 SMALL FALLS IN WEIGHT CAN IMPROVE HEALTH, SAYS NICE

Weight loss programs that focus on diet, activity, and lifestyle are cost effective even if the weight loss is small, so long as the improvement is maintained for the rest of life says the National Institute for Health and Care Excellence (NICE; UK).

In new guidelines on lifestyle and weight management programs, NICE says that 100 pounds spent on putting an individual through a 12 week course is cost effective if at least 1 kg weight is lost and if this achieves a permanent change in the person’s weight trajectory—that is for the rest of a person’s life. His or her weight is kept less than it otherwise would have been. For programs costing 500 pounds per head to be cost effective, the weight loss must be 2 kg; for 1000 pounds, 3 kg.

But the programs are not likely to be cost-effective if the weight is regained within 2 to 3 years or less.

However, long-term evidence is limited. None of 29 RCTs reported outcomes beyond 5 years.

Such evidence as does exist suggests that commercial programs (eg, Weight Watchers) might be more effective than primary care services, but it is not clear why. The NHS might target poorer people who are less motivated.

The guideline urges all involved not to adopt a censorious or lecturing tone when dealing with those who need or want to lose weight because many avoid treatment for fear of being stigmatized.

The average weight loss achieved by the lifestyle programs the group considered was 3% of body mass, although the target was usually 5%. But this should not put anybody off. Even at 3%, there will be health benefits. If you weigh 18 stones (a stone = 14 pounds) you may wish to lose 6 stones, but this is very difficult to do and very difficult to maintain. NICE is talking about things that work. If the goal is too high, people will give up.

This guideline is not about quick fixes. There is no magic bullet.

BMJ May 3, 2014;348:1 BMJ2014;348:g3558

“News” by Nigel Hawken, London

