CM Vitals

CARDIOMETABOLIC SOLUTIONS

In-Practice Guide

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Video: Introduction to the CM Vitals In-Practice Guide

Over the past century, society has seen dramatic advances in medical care that have helped eradicate numerous infectious diseases, improve quality of life and increase life expectancy. Cardiovascular disease (CVD) is a prime example, as the death rate for this disease continues to decrease each decade because of advances in technology and pharmacological interventions. However, CVD is still the leading cause of death for both men and women in the United States, and adds a major burden to public health costs. The major reasons for this dichotomy are the continued decline in lifestyle prevention activities considered to be the root cause of over 70% of cardiometabolic risk and the overwhelming use of polypharmacy as the primary preventative strategy for CVD in clinical practice. This has left a large portion of the population with a growing vulnerability for a host of cardiometabolic and lifestyle-related disease outcomes (type 2 diabetes, fatty liver disease, Alzheimer's disease, etc.) and a false sense of security in pharmaceutical interventions nullifying those risks.

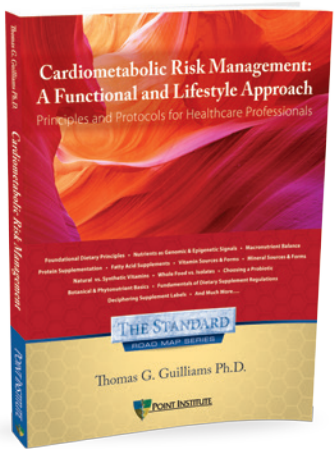
The CM Vitals Program is a different approach, designed to attack the underlying causes of cardiometabolic disease by using lifestyle medicine to reduce risk. Activities such as stress reduction, diet, avoiding environmental toxins and daily physical activity are all part of the CM Vitals Program. This model can help eliminate the false sense of security brought on by the polypharmacy approach and help your patients regain control of their cardiometabolic risk.

This In-Practice Guide will help you understand the underlying mechanisms of cardiometabolic disease and how to reduce risk through the lens of case studies and protocols. Included in this guide are protocols and treatment plans for peripheral vascular disease (PVD), dyslipidemia, hypertension, uncontrolled type 2 diabetes (T2D), cardiogenomics and homocysteine and arterial calcification.

The clinical case studies in this guide include patient overviews, first and second office visits, sample tests, treatment protocols and therapeutic lifestyle applications for patients with cardiometabolic disease.

Additional Educational Resources

The CM Vitals In-Practice Guide provides an overview of cardiometabolic dysfunction, clinical case studies and protocols. *Cardiometabolic Risk Management: A Functional and Lifestyle Approach*, written by Thomas G. Guilliams, PhD, provides a comprehensive review of cardiometabolic dysfunction and the latest research on this topic. As such, it serves as an excellent companion to this guide, and references are found throughout.

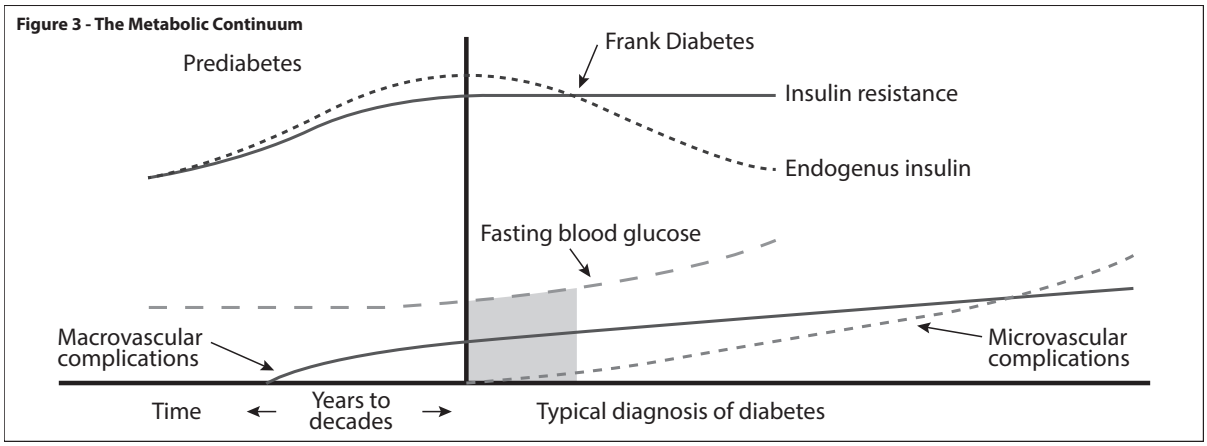


Cardiometabolic Reserve and Metabolic Continuum

Lifestyle enhances risk for cardiometabolic disease. This is best viewed through the lens of cardiometabolic reserve, which is defined as the long-term capacity of tissues to withstand chronic changes to physiological needs. As with any reserve, its capacity is vulnerable to depletion, but also capable of being resupplied and strengthened. A great example of this can be seen when examining the metabolic continuum and the progression of cardiometabolic disease.

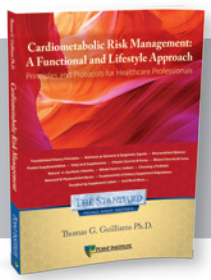
This progression often goes unnoticed for decades, often until a frank disease condition can be identified. Especially in the case of T2D, the diagnostic criteria (fasting blood glucose (FBG) of 126 mg/dl) is preceded by years (and often decades) of a gradual increase in insulin resistance and inflammatory signaling. This process has been termed "The Metabolic Continuum" (Figure 3).

Figure 3. The Metabolic Continuum



The speed of this metabolic continuum is determined by genetics, diet and other lifestyle factors that affect cardiometabolic reserve. Viewing cardiometabolic risk as a progressive metabolic continuum provides the opportunity to determine which patients are at increased risk long before those risks drive macrovascular and microvascular disease outcomes.

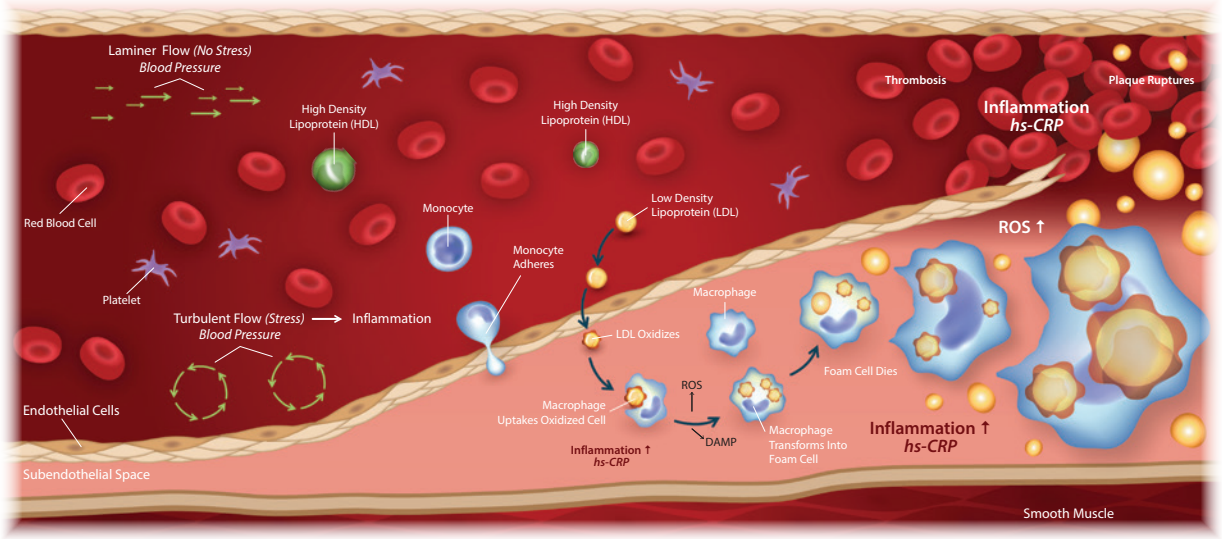
For additional information on cardiometabolic reserve and the metabolic continuum, reference *Cardiometabolic Risk Management: A Functional and Lifestyle Approach*, available at LifestyleMatrix.com.



Inflammation, Insulin Resistance and Atherosclerosis

Much of the modifiable cardiometabolic risk is driven by abdominal obesity and insulin resistance. It is difficult to overestimate the impact of these two related factors. Over the past several decades, it has become clear that insulin resistance drives cardiometabolic events. Insulin-resistant adipose tissue will readily convert excess serum glucose into free fatty acids (FFA), where they are either stored in adipose tissue or released into circulation. The abundance of serum FFA will cause the liver to produce triglycerides (TG)-rich VLDL particles, which in turn will shift cholesterol stores away from HDL particles (reducing HDL-C levels) and drive a shift in both LDL-C and HDL-C particles toward smaller, more atherogenic particles.

Figure 4. Atherosclerosis and Chronic Inflammation



Insulin-resistant adipocytes produce many cytokines (adipokines) that drive immune/inflammatory processes (TNF-α, IL-6, IL-1β, MCP-1, etc.) and hormones that drive metabolic processes (leptin, adiponectin). Since the atherogenic process is initiated and mediated by immune cell and tissue inflammatory signaling, peripheral insulin resistance in adipocytes (and myocytes) has a strong, indirect impact on cardiovascular events. The acute-phase reactant C-reactive protein (CRP) is produced by the liver in response to these inflammatory signals and has now become an important independent marker to predict cardiometabolic risk. The relationship between CRP and the metabolic syndrome is striking and increases with each added component. This global inflammatory signaling is also at the heart of most other chronic diseases, many linked to obesity and cardiometabolic risk. In addition to classic inflammatory signals, other acute-phase reactants related to inflammation can drive thrombosis by triggering the production of fibrinogen and plasminogen activator inhibitor-1 (PAI-1), both independent risk factors for cardiovascular events.

Lastly, the burden of insulin resistance puts continued pressure on the insulin-producing capacity of pancreatic β-cells. This can produce a progressive rise in post-prandial insulin and glucose, and a gradual increase in fasting glucose. The increase in post-prandial glucose leads to elevated levels of protein glycation, which in turn leads to increased circulation of AGEs. These modified proteins are strong inducers of inflammation (via toll-like receptors) and cellular aging and play an important role in mediating the cardiometabolic risk derived from insulin resistance and obesity (Figure 5). Combined, these insulin-resistant drivers place a heavy burden on the cardiovascular tissues, thereby driving atherosclerosis.

Measuring Insulin Resistance

Insulin resistance often increases the likelihood that a patient will display certain characteristic signs on a physical exam. The most obvious is an increased waist circumference or waist-to-hip ratio. However, other noticeable physical characteristics of insulin resistance and cardiometabolic risk include skin tags, acanthos nigricans, androgenic alopecia, and hirsutism. Hirsutism is considered to be a physical sign of androgen-mediated insulin resistance in women, particularly those with polycystic ovarian syndrome (PCOS). However, while hirsutism is very common in PCOS, some studies report that hirsutism is not more prevalent in insulin-resistant women without measurably elevated androgen levels or PCOS.

Clinical Pearl

Understanding Patient Patterns with Cardiometabolic Risk

Insulin-resistant patterns:

- Elevated TG
- Elevated VLDL
- Increased small dense LDL
- Generally more LDL-P
- Low HDL

Non-insulin resistant patterns:

- High LDL-C
- High Lp(a)
- Low HDL-C in the absence of high TG

Direct measurement of an individual's insulin resistance is very difficult, time-consuming and expensive; therefore, most clinical and laboratory measures of insulin resistance (or sensitivity) are surrogate markers of the "gold standard" method, the euglycemic clamp. Since this technique is mostly used in research and not clinical settings, it will not be discussed further. This guide will delve into dynamic and fasting biomarkers of insulin sensitivity, such as the oral glucose tolerance test (OGTT), fasting insulin, blood glucose, HbA1c and TG.

**All biomarkers are based on standardized reference ranges*

Oral Glucose Tolerance Test (OGTT)

The OGTT is a functional test of glucose tolerance. It is a simple test that measures the excursions of serum glucose and insulin following an oral glucose load (usually 75 g) or standard meal for at least two hours. It is designed to approximate the physiological impact of insulin resistance on postprandial glucose levels.

	Normal	Criteria for T2D	Criteria for Prediabetes
Fasting	<100 mg/dl	<126 mg/dl	100 to 126 mg/dl
1 hour (after glucose administration)	<160 mg/dl	None	None
2 hours (after glucose administration)	<140 mg/dl	>200 mg/dl	140 to 199 mg/dl

Measuring Additional Cardiometabolic Risk

In recent years, biomarkers such as homocysteine, myeloperoxidase (MPO), Lp-PLA2 and CRP as well as genetic markers like methylene tetrahydrofolate reductase (MTHFR) and APO-E have increased in popularity because of their association with cardiometabolic risk.

Homocysteine

Hyperhomocysteinemia is a significant risk factor for CVD. It is strongly associated with an increased risk for endothelial damage, inflammation and increases in ROS production.

Homocysteine Level*	Cardiac Risk Level
<6.3	<1x greater risk
6.3	1x greater risk
10	2x greater risk
15	4x greater risk
20	9x greater risk

C-Reactive Protein (CRP)

CRP is a biomarker that measures inflammation in the body. It is produced in the liver and its level is measured by testing the blood. CRP is classified as an acute phase reactant, which means that its levels will rise in response to inflammation. Wounds, injuries, infection, obesity, stress and damaging physical activity can all elevate CRP levels.

Myeloperoxidase (MPO)

Much like CRP, MPO is a biomarker that measures inflammation in the body. It is a peroxidase enzyme that is produced by neutrophils when they are activated. It is classified as an acute phase reactant and will respond to wounds, injuries, infections, obesity, stress and damaging physical activity.

Lipoprotein-associated phospholipase A2 (Lp-PLA2)

Lp-PLA2 is an enzyme that plays a role in the inflammatory process. Studies indicate that, along with hs-CRP, it is strongly associated with an increased risk for CVD.

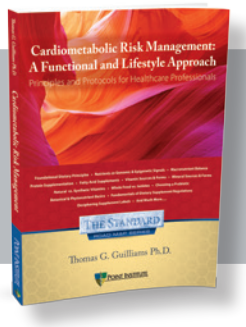
	Optimal	Borderline	High Risk
hs-CRP		2.2	
Range	<1.0	1.0-3.0	>3.0 mg/L
MPO			694
Range	<350	350-633	>633 pmol/L
$LpPLA_2$			244
Range	<200	200-235	>235 ng/mL

Methylenetetrahydrofolate Reductase (MTHFR)

MTHFR is a rate limiting enzyme in the methylation cycle that is gaining popularity because of the various allele's that have been discovered. At position 677, this gene has two possibilities: cytosine (C) (normal) or thymine (T) (abnormal). A single nucleotide polymorphism (SNP) of 677TT is strongly associated with an increased risk for numerous chronic disease, including CVD because of a lower MTHFR activity. Low folate intake affects individuals with the 677TT genotype to a greater extent than those with 677CC or 677CT. While this genetic test is relevant, as a clinician it is important to always treat phenotypes, not genotypes.

Apolipoprotein Subclass Particle E (APO-E)

APO-E is well-known to have anti-atherosclerotic activity. This beneficial effect of APO-E is due primarily to its role in the process of receptor-mediated uptake of LDL by the liver. In addition, different APO-E alleles have demonstrated anti-inflammatory benefits that make it a protective agent against CVD.



For additional information on cardiometabolic laboratory testing reference, *Cardiometabolic Risk Management: A Functional and Lifestyle Approach,* available at LifestyleMatrix.com.

In-Practice Template

First Appointment

Before you meet in the exam room:

Step 1:

A. During the first appointment, the patient completes the CM Vitals Advanced Cardiometabolic Analysis before you meet.

B. While the patient is waiting to see you, the patient watches the CM Vitals Patient Education Video as an introduction into how lifestyle impacts cardiometabolic health.

In the exam room:

Step 2:

A. Review the questionnaire and determine the driver of insulin resistance and cardiometabolic risk.

B. Using the CM Vitals Inventory sheet, discuss the role of the heart, how CVD develops and the difference between traditional medicine and the CM Vitals Program. Make sure to highlight how the patient is in control of their cardiometabolic risk.

C. Use the Atherosclerosis and Chronic Inflammation and Insulin Resistance Presentation Pads to help the patient understand their biomarkers and lab results.

D. Begin the patient on a lifestyle medicine program that includes stress reduction, the Mediterranean Diet, daily physical activity and limiting environmental exposure.

E. Patient receives the CM Vitals Program Patient Handbook. Instruct the patient to read the first 12 pages and the chapter corresponding to their driver of insulin resistance. The patient should implement the applicable lifestyle and nutritional recommendations and bring questions to the next appointment.

At checkout:

Step 3:

A. The patient goes home with targeted nutrients to help control cardiometabolic risk.

B. Schedule follow-up visit to increase compliance.

First Follow-Up Visit (3-4 weeks)

In the exam room:

Step 4:

A. Review steps patient has taken to improve nutrition and implement lifestyle change along with any questions the patient has on integrating these steps based on information read in the patient handbook.

B. Review test results along with patient history to identify the patient's specific dietary needs and supplement protocols.

C. Although the nutritional supplementation protocol will likely improve patient symptomology, it is important to set patient expectations by reinforcing that if no lifestyle change is implemented, improvement in cardiometabolic dysfunction will be hindered.

At checkout:

Step 5:

A. Provide clear recommendations on duration of each therapy.

B. Schedule follow-up based on patient's need for coaching and accountability.

Subsequent Follow-Up Visits

Step 6:

Within four to six months of implementing the initial protocol, the patient should be offered retesting, provided there has been some symptom change, as well as lifestyle change. During that time, schedule a group medical appointment to follow up on 10 to 16 patients in a 90-minute time frame. Prescribe this as a required follow-up. Reinforcing the proper changes is crucial to improvement upon retesting. Consider waiting to retest until these steps have been properly implemented by the patient.

Implementation Tip

When implementing the CM Vitals Program in your practice, start with small lifestyle changes to avoid overwhelming the patient on the first office visit.

Initial In-Office Visit

STEP 1

Patient completes the CM Vitals Advanced Cardiometabolic Analysis

STEP 2

Clinician uses the CM Vitals Inventory Sheet to discuss the basics of heart health and the importance of lifestyle medicine with the patient

STEP 3

A. Clinician and patient identify the area(s) of dysfunction

B. Clinician uses Atherosclerosis and Inflammation or Insulin Resistance Presentation Pads to illustrate factors affecting cardiometabolic health

C. Patient begins initial cardiometabolic health supplements to address dysfunction

D. Patient watches the CM Vitals Patient Education Video

The CM Vitals Patient Education Video can be used as an outreach tool for both new and existing patients. This video can be used to introduce key concepts to new patients or as a follow-up to the first visit to review important discussion points.

STEP 4

A. Patient receives CM Vitals Patient Handbook

It is recommended that every patient reviews the first chapter of the handbook and the chapter correlating with their area(s) of dysfunction.

Chapter 1: Understanding Cardiometabolic Risk
Chapter 2: Stress and Cardiometabolic Disease
Chapter 3: Diet and Cardiometabolic Disease
Chapter 4: Environmental Factors and Cardiometabolic Disease
Chapter 5: Exercise and Cardiometabolic Disease
Chapter 6: Targeted Nutrients for Cardiometabolic Disease

STEP 5

Patient receives CM Vitals educational exercise and diet videos

A. Patient watches one exercise video each week for eight weeks in succession

B. Patient watches two diet videos each week for two weeks in succession

Follow-Up Visit (2–3 Weeks)

STEP 6

Clinician and patient review pertinent test results and diagnosis. Clinician initiates targeted therapies based on results. For example, if blood test reveals insulin resistance and inflammation, clinician initiates anti-inflammatory and insulin sensitivity protocol with regular exercise and a plant-based Mediterranean diet.

STEP 7

At this time, patients enroll in the **Advanced Prevention for Cardiovascular Disease** Group Visit

SEE THE FOLLOWING PAGE FOR MORE DETAILS

Follow-Up Visit (12+ Weeks)

STEP 8

In three to four months, clinician assesses the patient to track progress and adjust treatment plan as necessary. Treatment focus may transition to another foundational area of cardiometabolic health at this time.

GVT GROUP VISIT TOOLKIT
BUILDING HEALTHIER PRACTICES +

TRANSFORMING MEDICINE WITH GROUP VISITS

During the initial phase of care, implementing lasting lifestyle changes is essential for improvement of patient health outcomes. Group Visits are a great way to reinforce healthy lifestyle habits and keep patients motivated and accountable prior to one-on-one reassessments. In addition, Group Visits are an effective tool to create better practice efficiencies both financially and clinically. We recommend using the *Advanced Prevention of Cardiovascular Disease* Group Visit Toolkit. You can find this and other Group Visit Toolkit resources at **LifestyleMatrix.com**

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Lifestyle modification protocols should be used for both primary and secondary prevention of CVD.

Protocols for Reversing and Preventing Cardiometabolic Risk

As previously discussed, daily lifestyle choices significantly affect risk for cardiometabolic disease. The idea that these are daily choices must be stressed to your patients. This understanding will help the patient feel in control of their cardiometabolic risk.

Stress Reduction

Stress is a major underlying cause of cardiometabolic risk for many patients. It is the responsibility of the clinician to determine which patients are becoming overwhelmed by the effects of stress as well as develop individualized support protocols to help patients regain their wellbeing.

While a full review of stress and its role in HPA and HPT axis dysfunction will not be covered in this guide, it will provide a brief overview of DHEA, cortisol and their relationship to cardiometabolic risk. For an extensive review of HPA axis dysfunction, including clinical case studies, please reference the ARK Stress Recovery Program In-Practice Guide and *The Role of Stress and the HPA Axis in Chronic Disease Management*.

Cortisol and DHEA

Cortisol, a glucocorticoid hormone, is a pleiotropic modulator of cellular activity through intracellular glucocorticoid receptors (GR) found in most tissues. Like the stress response in general, cortisol is intended to shunt cellular resources away from long-term metabolic processes and toward those that function primarily on immediate survival and homeostasis. Cortisol mobilizes protein stores in all tissues except the liver. It mobilizes fatty acids from adipose, is the precursor of cortisone, acts as the body's main anti-inflammatory agent, and is the primary hormone directing immune function in elderly patients.

DHEA, a glucocorticoid antagonist, serves not only to prevent excessive systemic inflammation but also to protect the neurologic machinery, particularly the hippocampus, from the damaging effects of cortisol, a phenomenon that may also be true of its neurosteroid precursor pregnenolone. Exposure to chronic stress leads to a substantial reduction in circulating levels of DHEA-S and DHEA, which can further damage underlying metabolic processes caused by the effects of cortisol, leading to accelerated aging and inflammation. Sources of acute stress are obvious, but helping patients identify their unique source(s) of chronic stress is vital to treating any chronic health condition, especially those related to cardiometabolic risk.

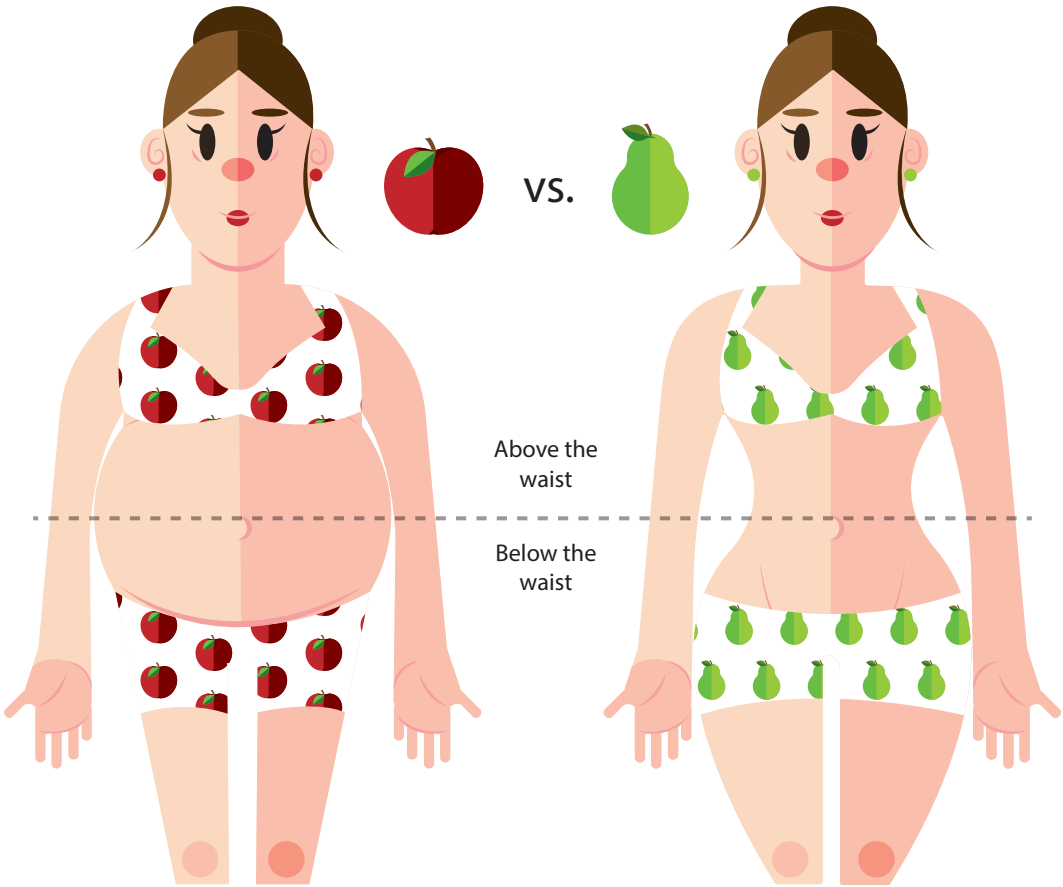
There are four categories of chronic HPA axis stress that clinicians should evaluate in each patient: mental/emotional stress, sleep disorders, metabolic/glycemic dysregulation, and chronic inflammation.

Altered Body Composition: Prediabetes

A 58-year-old Caribbean Indian female presents as a new patient with a recent diagnosis of hyperlipidemia and refusal to initiate statin therapy. She is a high-performance executive with a successful family business she has been running with her husband for the last 23 years. With her increased work demands, she has gradually gained weight in her midsection and is reporting increasing fatigue with occasional mild-moderate bloating/constipation as her only other symptoms of concern. Social history reveals a glass of wine with dinner most nights to relax after a 12- to 14-hour workday.

First Office Visit

The patient receives comprehensive anthropometric measurement evaluation including BMI, waist circumference, hip circumference, and bio-impedance analysis. Analysis reveals a BMI within healthy range; visceral adiposity (by ethnicity specific waist circumference cut-off and waist:hip ratio); and total body % fat of 32%. This clinical vignette is also known as "skinny fat" with apple obesity distribution. Review of her past medical records reveals she does not meet ATP III diagnostic guidelines for metabolic syndrome.



Appendix I: CM Vitals Program Revenue Generator

The following tables provide a general model for revenue generation based on each patient that is run through the CM Vitals Program for six months. This includes a first and second office visit, baseline and follow up testing, a group visit, as well as a six-month supply of supplements for chronic cardiometabolic dysfunction. The revenue generation models below include an insurance-based model and a cash practice model.

Insurance-Based Revenue Generation Model

First Office Visit	$150	$220 bill sent to insurance for new patient E&M code #99203 or established patient code #99214 billed on time, with roughly 70% gross collection
Approximate initial supplement sales profit	$160	Products may include but not limited to: insulin sensitizing agents, omega 3-fatty acids, lipid control
Baseline Testing	variable	Baseline testing assumes a CBC, blood chemistry, and A1C are already performed. Therefore, "baseline" refers to a comprehensive cardiometabolic tests such as advanced particle testing, CIMT and inflammatory markers
Second Office Visit	$150	$220 bill sent to insurance for E&M code #99214 billed on time with roughly 70% gross collection
Group Visit	$60	Bill a level 3, 99213 CPT code. Billing is based on complexity, not time in a Group Visit. See figure for additional information regarding group visits
Supplement sales profit for 5-month refills	$790	Products may include but not limited to: insulin sensitizing agents, omega 3-fatty acids, lipid control
Testing	variable	
Total Profit from 6 months of CM Vitals Program per patient	$1,310 (w/o testing profits)	

Based on the insurance-based model, if, each week, the practice has one patient starting and adhering to a six-month CM Vitals Program, the total revenue generated will be in $67,120 in 12 months.

Cash-Based Revenue Generation Model

First Office Visit	$220	Based on 30-minute office visit
Group Visit	$75	
Approximate initial supplement sales profit	$160	Products may include but not limited to: insulin sensitizing agents, omega 3-fatty acids, lipid control
Baseline Testing	variable	Baseline testing assumes a CBC, blood chemistry, and A1C are already performed. Therefore, "baseline" refers to a comprehensive cardiometabolic tests such as advanced particle testing, CIMT and inflammatory markers
Second Office Visit	$220	Based on 30-minute office visit
Supplement sales profit for 5-month refills	$790	Products may include but not limited to: insulin sensitizing agents, omega 3-fatty acids, lipid control
Testing	variable	
Total Profit from 6-month CM Vitals Program per patient	$1,465 (w/o testing profits)	

Based on the cash-based model, if, each week, the practice has one patient starting and adhering to a six-month CM Vitals Health Program, the total revenue generated will be in $76,180 in 12 months.

Advanced Prevention for Cardiovascular Disease Group Visit Toolkit

Following the second office visit, it is recommended to have the patient set up their next appointment approximately one month later in a Group Visit. Group visits help to maximize time with patients who need lifestyle education. The Advanced Prevention for Cardiovascular Disease Group Visit Toolkit provide the necessary resources to implement and conduct a successful Group Visit Model in your practice. This relieves practitioners from the task of creating group classes on their own. The tools include the SOAP note, patient handouts, promotional flyers, and PowerPoint slides at a quality level that patients enjoy and understand.

For insurance-based practices: Each patient will have a face-to-face E/M with you, the provider, while the presentation segment of the Group Visit is being conducted. Each Group Visit lasts 90 minutes from the time patients check in to conclusion and can be conducted at the end of a regularly scheduled business day (4:30-6 p.m.).

To learn more about how Group Visits can help your practice grow and improve patient care, please visit LifestyleMatrix.com.

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