

Upcoding of clinical information to meet Appropriate Use Criteria for PCI

Short title: Upcoding of PCI following AUC.

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1 A reason to disregard ORBITA¹ in the USA, according to some key opinion leaders is
2 the startlingly high proportion of PCI that is performed for unstable coronary disease
3 in the USA by comparison to peers. In the USA, 82% of PCIs are reported to be
4 performed in emergency or unstable situations² in comparison to approximately 65%
5 in the United Kingdom³ and, closer to home, 59% in the province of Ontario, Canada⁴.
6 In this article we look for evidence of so-called “gaming” or “upcoding” in which
7 coronary disease becomes rated as more acute.

8
9 Appropriate Use Criteria (AUC) for coronary revascularisation seek to inhibit
10 unnecessary procedures⁵, but there are in fact two ways a physician can become
11 compliant. One is to do fewer unnecessary procedures, and the other is to make
12 unnecessary procedures seem necessary. This is much easier to do than many non-
13 physicians appreciate.

14 15 **Instability is in the eye of the beholder**

16
17 Appropriate Use Criteria make a sharp distinction between stable angina which arises
18 predictably on exertion and unstable angina which is rapidly escalating in intensity, or
19 occurring at rest.

20
21 In medical school, these appear to be easy to distinguish. On the one hand is a patient
22 attending an outpatient clinic because of angina on walking up hills. On the other is a
23 patient confined to the hospital bed because of episodes of rest pain, perhaps with
24 dynamic ECG changes during the pain. We know the latter group stand to gain from
25 early PCI.

26
27 However, unlike many cardiological assessments we make, there is no diagnostic test
28 to confirm or refute unstable angina. As a result the clinician is free to judge whether
29 a patient’s angina is unstable or not.

30 31 **Infectious instability**

32
33 Patients, too, have to make decisions, for example whether sensations do indeed fall
34 into the category of “pain”. They already know that pain is more concerning in the
35 chest than elsewhere. Seeing a cardiologist can have a transformative effect on the
36 symptom experienced and its instability. This impact is at three levels: knowledge,
37 attitude and behaviour.⁶

38
39 At the simple *knowledge* level, the doctor reassures the patient that medication is the
40 key to preventing stable coronary disease from turning into a serious event. However,
41 the explicitly verbalised facts are only a small part of what is communicated.

42
43 More importantly, we simultaneously convey an *attitude* of concern, by asking detailed
44 questions about the symptom which a blinded third party might rate as atypical but in
45 light of ischaemia tests gradually crystallises around textbook patterns of angina.

46
47 Finally, our consultation ends with the *behaviour* of arranging an angiogram as soon
48 as possible. No wonder patients feel that PCI for stable angina is necessary to save
49 their lives⁷, even though no cardiologist has said this to them at the *knowledge* level.

The 2010 experiment of nature

This complex process of judgement has existed since time immemorial. What has changed recently is the external influence on the process.

Although there have been guidelines for some years, it was in 2010 that PCI appropriateness assessments began in hospitals in the USA; assessments made automatically based on data provided by the individual physician operators⁸. Some states have sought to reduce misuse, control costs, and improve compliance with Appropriate Use Criteria (AUC)^{8,9}. For two states, New York⁹ and Washington⁸, annual data around this time are in the public domain, broken down by physician reported category (stable or unstable).

New York state has gone so far as to consider linking Medicare reimbursement to AUC adherence⁹. Ontario, the Canadian province geographically adjacent to New York but not exposed to the regulatory effect of AUC, has also provided equivalent data⁴.

We used these data sources to assess the changing indications for PCI following publication of AUC. A description of each data source is supplied in Appendix 1.

Acute PCI growing in dominance, in the USA

With each passing year following the introduction of AUC in 2009, both New York and Washington saw a progressive annual increase in the proportion of all PCI that was performed for acute coronary syndromes (Figure 1)^{8,9}.

In the adjacent province of Ontario, there was no such secular increase in the proportion of PCI that was performed for ACS⁴. In fact, over the same time interval, this proportion even saw a modest fall.

4 to 10-fold larger increase in acute PCI

Before implementation of AUC feedback, the per capita rate of acute PCI in New York was 68.2% higher than in Ontario. In Washington it was 33.1% higher than in Ontario. Over the next four years, New York rose to 84% above Ontario, and Washington to 38.1% above Ontario (Figure 2).

Absolute rates of acute PCI procedures had escalated by 197 per million in New York and 78 per million in Washington, respectively ten and 4 times larger than the absolute increase in Ontario, 19 per million.

Changes in documented angina score in patients undergoing PCI for stable coronary disease following AUC

Even within stable angina, physician interpretation is required to judge the severity of symptoms. During these transition years, there was a dramatic increase in the intensity of stable angina reported by physicians (Figure 3A). This can be viewed from data across the whole of the USA, between 2009-2014, covering 397,737 procedures².

This pan-USA data demonstrates a doubling of the rate of CCS 3 (angina on mild effort) and CCS 4 (angina at rest) reported in those undergoing PCI for stable coronary disease following the publication of AUC. In 2009, the proportion of patients with CCS 3 or 4 angina was just 15.8%. By 2014, this proportion had more than doubled to 38.4%. The excess number of patients experiencing CCS 3 or 4 angina in 2014 by comparison to 2009 was 9,837 patients per year.

Such dramatic changes in reported symptomatology over such a short timespan render a biological cause improbable. Certainly, this was not caused by a worsening pattern of coronary disease over time. The chart of number of diseased vessels shows only a miniscule trend to worsening; with the proportion of patients having 2 or 3 vessel disease rising only from 42.7% to 47.5% (Figure 3B).

Is this upcoding?

There was a four to ten-fold greater increase in population rates of PCI for ACS in the AUC states following the onset of appropriateness assessments. Simultaneously, patients with stable disease showed a dramatic rise of high grade angina symptoms, disproportionate to the minimal secular trend in coronary anatomy. These two findings are difficult to explain by biological processes alone.

“Upcoding” encompasses a wide spectrum of behaviours ranging from fraud, claiming payments for untruthful diagnoses or treatments, to a subtle bias towards one diagnostic interpretation rather than another. The benign end of the spectrum exists because the boundary between diagnostic categories is blurred, with many patients fitting two or more categories.

A similar analysis has identified a dramatic rise in the volume of outpatient PCI that was categorised as ‘acute’ following AUC¹⁰. The National Cardiovascular Data Registry provides an undemanding definition of ‘unstable angina’ which is simple to fulfil¹¹. But why procedures for unstable angina continued to increase, year-on-year following AUC, in the face of high sensitivity troponin assays, remains unexplained. Our analysis adds to this data, demonstrating a concordant and progressive rise in high grade angina in those undergoing PCI for stable disease.

Formal confirmation of the magnitude of upcoding could be obtained by a randomised controlled trial in which at sometimes physicians were incentivised to make one diagnosis and at other times, another. No such trial has been carried out. Moreover, since the physicians being trialled would have to give informed consent, they would know what was being tested and could easily influence the result. For this reason, large scale observational data may be the current best evidence on this question.

Major effort has recently been put into standardised documentation of symptoms (sometimes called Patient Reported Outcome Measures, PROMs) using the Seattle Angina Questionnaire and the Rose Dyspnoea Scale. However, these are applied after contact with a cardiologist who has recommended PCI. During such a consultation, it is inevitable that emphasis will be placed on symptoms, because the doctor and patient expect these to be improved by the procedure. Therefore, although

the documentation may become more standardised, this may simply standardise the inclusion of bias rather than remove it.

Nor does the substantial effort put into the assessment of post procedural symptoms eliminate the placebo effect. Patients filling out the questionnaires have invested personal risk which, through the principal of cognitive dissonance increases the likelihood of reporting benefit.

Upcoding or consensus building?

The upcoding we observe is fundamentally not a malicious process. It represents doctors and patients sharing the simple and common-sense idea that tight coronary lesions are undesirable and should be treated with stents. Starting from that premise, the doctor and patient build a consensus so that care can go forwards rather than moving in circles of doubt. This consensus building process naturally includes discussing advantages of the procedure for symptom relief, even though the true reason many patients undergo PCI for stable coronary artery disease is that they and their doctors do not like the idea of leaving those lesions alone.

Our community and our patients would benefit if we accepted this fact and discussed it openly. The only way to truthfully understand why procedures are done is to not restrict their performance to patients with specific conditions.

Limitations

Individual state data separating stable from acute PCI is only publicly available for a few states. There is nationwide data², but this is the sum of many states, each of which can have a different timing of enforcement of AUC, precluding any possibility of detecting a temporal match in the aggregate data.

Upcoding is only one component of the change in proportions of acute versus stable PCI in the USA. Much of this shift is likely to be due to genuinely more conservative handling of stable coronary artery disease since AUC. Indeed, there has been a definite nationwide fall in PCI overall of 15% from 2010 to 2014, which represents more judicious care overall and not upcoding².

It is possible that prompting by modern Electronic Health Record (EHR) systems ensures that key symptom information suggesting instability is collected more systematically now than previously, which would cause a tilt towards categorisation as acute. However, this would have to be an EHR trend only on the South side of the border with Canada.

Our interpretation of the data has important limitations. First, we have not attempted to adjust for differences between the countries in demographic trends that may cause plaque instability. However, the size of such divergence is limited by the geographical proximity of the neighbouring regions. Second, the Canadian hub-and-spoke model may be more restrictive of the provision of PCI than the fee-for-service model employed in the United States.

Third, we have addressed these two US states because they have implemented policies and published their data. Action chosen by different states may have different

effect sizes and different time courses. Therefore, even if the effects are in the same direction, the national total will experience an effect which is not sharply localised in time but smeared out over years. On a background of generally falling age adjusted rates of atherosclerosis¹², this would manifest as an enhancement of the downtrend in stable PCI and an attenuation of a downtrend in acute PCI. If the strength and progressiveness of the upcoding effect was just right the rates of acute PCI could hold almost constant while the stable PCI rate plummets.

Perhaps by chance this is the pattern seen across the USA as a whole². Whether this is the mechanism is a testable hypothesis since such a mechanism would last only as long as rates of upcoding can continue to increase. Once reported stable PCI bottoms out, we hypothesise that reported acute PCI, now including everyone that can be upcoded, will return to tracking the underlying downtrend.

Significance for policy makers, patients and researchers

The lesson from upcoding is that if trying to restrain free enterprise in a fee-for-service system, it is insufficient to impose a gatekeeper function depending on clinical judgement of the same physicians in whom one is trying to curtail unnecessary activity. An elaborate solution might be a system of independent review of the patient before the patient has had contact from a cardiologist who may bias their description of their symptoms. Simpler might be to remove the financial incentive to perform more procedures.

Upcoding can harm patients by facilitating unnecessary PCI, but also by misrepresenting to the patient the nature of their cardiac diagnosis. Moreover, researchers analysing registries of data in which upcoding is embedded will unintentionally produce a distorted picture in their publications.

Conclusion

Observations of geographical differences in trends of procedure rates, and the discrepancy between the time trends of symptoms and extent of coronary disease, are consistent with (but not proof of) a rise in upcoding around the time of introduction of reporting of adherence to AUC. There may never be a better test of the upcoding response to regulatory attempts to curb free enterprise in healthcare. We should face up to the fact that as a community, we find it difficult to accept that stable coronary stenoses need not be stented. Evidence of this difficulty is that we cannot resist the temptation to hunt these coronary stenoses down.

If we ever doubt the fundamental problem of which upcoding is a symptom, we only need to ask ourselves why there is not a corresponding problem of “downcoding”.

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Nil

Figure Legends

Figure 1

The annual proportion of PCI performed for stable coronary artery disease and acute coronary syndromes in New York and Washington states and the province of Ontario, Canada since the application of AUC in the USA in 2009.

Figure 2

Annual numbers of PCI performed for acute coronary syndromes /million population in New York and Washington States as well as Ontario, Canada. The shaded areas indicate the unexpected increase in numbers of PCI for ACS performed in New York and Washington following PCI appropriateness assessments initiated in 2010.

Figure 3

Documented patient reported symptoms (A), and angiographic disease (B), in patients undergoing elective PCI for stable coronary artery disease across the USA. CCS = Canadian Cardiovascular Society.

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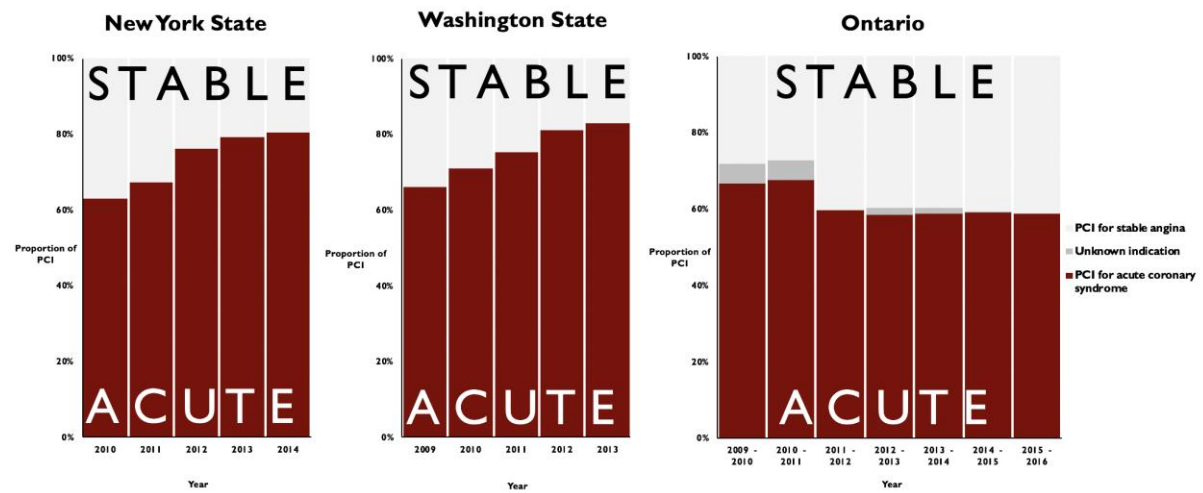


Figure 1.

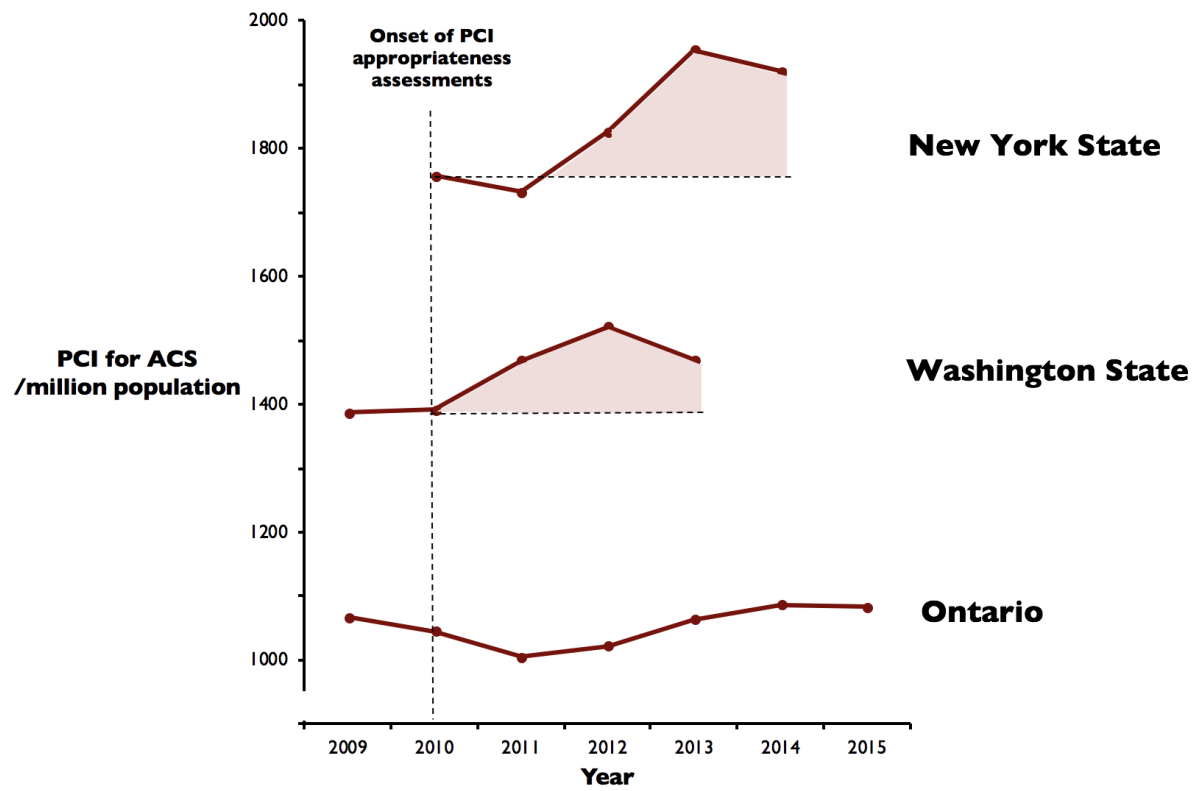
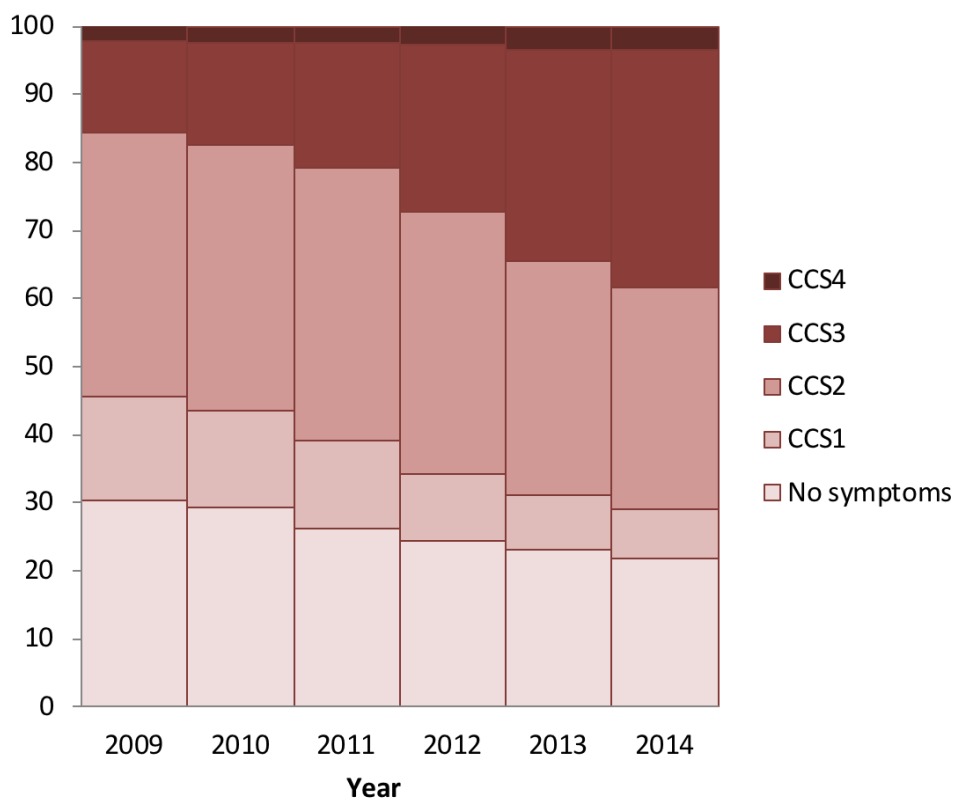


Figure 2

A. Symptoms

% of patients undergoing elective PCI



B. Angiographic disease

% of patients undergoing elective PCI

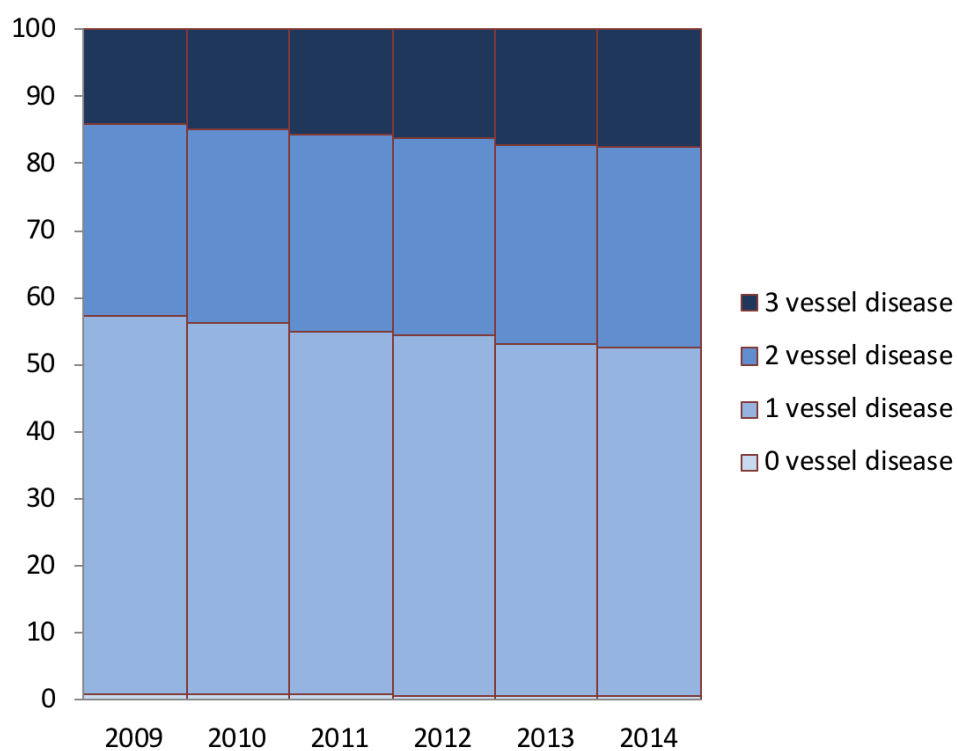


Figure 3