Interviewed on KARE 11: Dr. Paul Sorajja
Small device shows big promise for heart patients
COAPT study using MitraClip

Interviewed on KDUZ Radio: Dr. Jay Traverse
CONCERT and SENECA studies for stem cell therapy in HF

CONGRATULATIONS to MHIF Research Fellow Dr. Peter Tajti!
for winning the TCT Fellow Case Competition

FEATURING MHIF STUDIES
Open for Enrollment and Referrals!

TRANSCEND for peripheral artery disease
CONTACT: JoAnne Goldman, 612-863-3973

ASAP-SVG for coronary artery disease
CONTACT: Pamela Morley, 612-863-6066

MINT for myocardial ischemia & transfusion
CONTACT: Rose Peterson, 612-863-6051

First Recipient of The Jon DeHaan Foundation Award for Innovation in Cardiology

Dr. Kelly Han is recognized for her outstanding contributions to improving both safety and quality of imaging Congenital Heart Disease in adults, children and infants.

DISSEMINATING RESEARCH

Call out to Dr. John Lesser and the IIR team, including Sue Casey, for work on the PROTECTION VI study

LateBreaker at ESC Congress in August

Published in the European Heart Journal

Study assessed the use of strategies for dose reduction during CCTA as part of a multi-center, international registry
2018 Kevin Graham Prevention Lecture
Precision Cardiovascular Prevention: Past, Present and Future

Speaker: Philip Greenland, MD
- Harry W. Dingman Professor of Cardiology and Professor of Preventive Medicine at Northwestern University’s Feinberg School of Medicine
- Senior Editor for Journal of the American Medical Association | Chicago, IL

Learning Objectives
At the completion of this activity, the participants should be able to:
- Demonstrate the progress in CVD prevention
- Define blood pressure and cholesterol goals
- Identify new strategies for CVR risk assessment and treatment

Minneapolis Heart Institute Foundation Cardiovascular Grand Rounds
Date: October 22, 2018 | Time: 7 – 8 A.M. | Location: Abbott Northwestern Hospital Education Building, Auditorium A/B

Webinar: If you cannot attend grand rounds in person, attend via webcast (you can join the webinar up to 15 minutes before the presentation starts at 7:00 a.m.).

Link to attend webinar: mhif.adobeconnect.com/gr/ Please enter as a guest (first and last name), not a registered user.

About: Dr. Kevin Graham
Dr. Kevin J. Graham has been a tireless and innovative thought leader in cardiology. His bold vision spearheaded Hearts Beat Back: The Heart of New Ulm Project — an initiative to reduce heart attacks and improve modifiable cardiovascular disease risk factors in a southwestern Minnesota community. This award-winning heart disease prevention initiative, offered in partnership with the Minneapolis Heart Institute Foundation®, Allina Health and the city of New Ulm, moved beyond the walls of health care establishments and into the community to make health and well-being the easy choice where people live, learn, work, worship and play.

He has been actively involved in the development of cardiology practice guidelines and computerized interfaces for cardiovascular disease management in both specialty and primary care settings. He has authored numerous articles in both preventive cardiology and managed care, especially with regard to quality measures.

Dr. Graham served as the President of the Minneapolis Heart Institute® and the Cardiovascular Services Division of Abbott Northwestern Hospital from 2007 to 2012. He established and led the Preventive Cardiology practice and was Director of Outpatient Clinical Laboratory at the Minneapolis Heart Institute®. Dr. Graham was also an Assistant Professor of Clinical Medicine at the University of Minnesota Medical School. He completed his residency in Internal Medicine at Hennepin County Medical Center and his fellowship in cardiovascular disease at the University of Minnesota.
ACCREDITATION

**Physician:** Allina Health is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. Allina Health designates this live activity for a maximum of 1.0 AMA PRA Category 1 Credit(s)™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

**Nurse:** This activity has been designed to meet the Minnesota Board of Nursing continuing education requirements for 1.0 hours of credit. However, the nurse is responsible for determining whether this activity meets the requirements for acceptable continuing education.

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**Moderator(s)/Speaker(s):** Dr. Philip Greenland has disclosed that he DOES NOT have any real or apparent conflicts with any commercial interest as it relates to presenting his content in this activity/course.

**Planning Committee:** Dr. Alex Campbell, Jake Cohen, Jane Fox, Dr. Mario Goessl, Dr. Kevin Harris, Dr. Kasia Hryniewicz, Rebecca Lindberg, Amy McMeans, Dr. Michael Miedema, Dr. JoEllyn Moore, Pamela Morley, Laura Onstot, Dr. Scott Sharkey, and Jolene Bell Makowesky have declared that they do not have any conflicts of interest associated with the planning of this activity. Dr. David Hurrell declares the following relationship – Boston Scientific: Chair, Clinical Events Committee.

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Maintaining these details are the responsibility of the individual.

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Signature:

My signature verifies that I have attended the above stated number of hours of the CME activity.

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2925 Chicago Ave - MR 10701 - Minneapolis MN 55407

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Together, we can create a world without heart and vascular disease.
Disclosures

• Grants from American Heart Association and NIH

• No relationships with Industry
Evolution in personalized and precision medicine

<table>
<thead>
<tr>
<th>Individualized medicine</th>
<th>Population medicine</th>
<th>Precision Medicine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dominated till ~ 1980’s</td>
<td>1980’s and onward</td>
<td>2000’s and future?</td>
</tr>
</tbody>
</table>
Cardiovascular disease prevention – and risk factors for CHD -- were known before 1950 – but largely ignored in clinical medicine

History of CHD – Henry Blackburn, MD

• “In hindsight, discussion of either the pathogenesis or treatment of coronary heart disease now seems conspicuously absent from the great classic texts on cardiology until Paul White’s in 1941. And even White, as late as his third edition in 1948, makes no mention of prevention.”
• “A heart attack after age 80 is an act of God; before 80 a failure of medicine.” “A vigorous five-mile walk will do more good for an unhappy but otherwise healthy adult than all the medicine and psychology in the world.”
Some critical observations before 1970’s

- Experimental animal work going back to 1920’s on cholesterol and atherosclerosis
- Smoking data – well known to life insurance industry as early as 1940’s
- Framingham – started 1948 – first papers appeared 1960-61

From: What is a normal blood pressure?
Eur Heart J. 2018;39(24):2233-2240

Personalized hypertension treatment

From: What is a normal blood pressure?
Eur Heart J. 2018;39(24):2233-2240
1984: The beginning of the era of CHD prevention and continued efforts to personalize CVD prevention.


1988

Table 1.—Initial Classification and Recommended Followup Based on Total Cholesterol

<table>
<thead>
<tr>
<th>Classification, mg/dL</th>
<th>Desirable blood cholesterol</th>
<th>Borderline-high blood cholesterol</th>
<th>High blood cholesterol</th>
<th>Repeat within five years</th>
<th>Dietary information and recheck annually</th>
<th>Lipoprotein analysis; further action based on LDL-cholesterol level</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;200</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>200 to 239</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥240</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recommended followup</td>
<td>Total cholesterol, &lt;200 mg/dL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol, 200-239 mg/dL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without definite CHD or two other CHD risk factors (one of which can be male sex)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With definite CHD or two other CHD risk factors (one of which can be male sex)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol ≥240 mg/dL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2.—Classification and Treatment Decisions Based on LDL-Cholesterol

<table>
<thead>
<tr>
<th>Classification, mg/dL</th>
<th>Desirable LDL-cholesterol</th>
<th>Borderline-high-risk LDL-cholesterol</th>
<th>High-risk LDL-cholesterol</th>
<th>Initiation Level, mg/dL</th>
<th>Minimal Goal, mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;130</td>
<td>≥160</td>
<td>&lt;160‡</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>130 to 159</td>
<td>≥130</td>
<td>&lt;130§</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥160</td>
<td>≥190</td>
<td>&lt;160</td>
<td></td>
<td></td>
<td>≥160</td>
</tr>
</tbody>
</table>

Dietary treatment

Without CHD or other risk factors

With CHD or other risk factors

Drug treatment

Without CHD or other risk factors

With CHD or other risk factors
2013 ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults
Statin Benefit Groups

- Clinical ASCVD (1A)
- LDL-C ≥190 mg/dL without secondary cause (1A)
- Primary prevention – Diabetes(DM) – Age 40-75 years – LDL-C 70-189 mg/dL (1A)
- Primary prevention – No DM – Age 40-75 years – LDL-C 70-189 mg/dL; ASCVD risk ≥ 7.5%* (1A)

Original Investigation

Validation of the Atherosclerotic Cardiovascular Disease Pooled Cohort Risk Equations

Paul Muntner, PhD; Lisandro D. Colantonio, MD; Mary Cushman, MD; David C. Goff, Jr, MD, PhD; George Howard, DrPH; Virginia J. Howard, PhD; Brett Kissela, MD, MS; Emily B. Levitan, ScD; Donald M. Lloyd-Jones, MD, ScM; Monika M. Safford, MD

DESIGN, SETTING, AND PARTICIPANTS Adults aged 45 to 79 years enrolled in the Reasons for Geographic and Racial Differences in Stroke (REGARDS) study between January 2003 and October 2007 and followed up through December 2010. We studied participants for whom atherosclerotic CVD risk may trigger a discussion of statin initiation (those without clinical atherosclerotic CVD or diabetes, low-density lipoprotein cholesterol level between 70 and 189 mg/dL, and not taking statins; n = 10,997).

“With more complete ascertainment of events in this subgroup (with Medicare data on events), there tended to be modest under-prediction of event rates by the Pooled Cohort equations.”
Coronary Calcium as a Predictor of Coronary Events in Four Racial or Ethnic Groups

Robert Detrano, M.D., Ph.D., Alan D. Guerci, M.D., J. Jeffrey Carr, M.D., M.S.C.E., Diane E. Bild, M.D., M.P.H., Gregory Burke, M.D., Ph.D., Aaron R. Folsom, M.D., Kiang Liu, Ph.D., Steven Shea, M.D., Moyes Szklo, M.D., Dr.P.H., David A. Bluemke, M.D., Ph.D., Daniel H. O’Leary, M.D., Russell Tracy, Ph.D., Karyl Watson, M.D., Ph.D., Nathan D. Wong, Ph.D., and Richard A. Kronmal, Ph.D.

NEJM 2008, Cited Over 650 times
A Citation Classic by Thomson Reuters
### Table 3. Risk of Coronary Events Associated with Increasing Coronary-Artery Calcium Score after Adjustment for Standard Risk Factors.

<table>
<thead>
<tr>
<th>Coronary-Artery Calcium Score</th>
<th>Major Coronary Event</th>
<th>Any Coronary Event</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No./No. at Risk</td>
<td>Hazard Ratio (95% CI)</td>
</tr>
<tr>
<td>0</td>
<td>8/3409</td>
<td>1.00</td>
</tr>
<tr>
<td>1–100</td>
<td>25/1728</td>
<td>3.89 (1.72–8.79)</td>
</tr>
<tr>
<td>101–300</td>
<td>24/752</td>
<td>7.08 (1.05–46.47)</td>
</tr>
<tr>
<td>&gt;300</td>
<td>32/833</td>
<td><strong>6.84 (2.93–15.99)</strong></td>
</tr>
<tr>
<td>Log10((CAC+1))§</td>
<td>1.20 (1.12–1.29)</td>
<td>&lt;-0.001</td>
</tr>
</tbody>
</table>

---

### Figure 1

Distribution of coronary artery calcium by risk factor burden.
Figure 4: Total (A) and hard (B) coronary heart disease event rates (per 1000 person-years) with increasing coronary artery calcium scores according to Framingham risk score category.

Central Illustration: Proposed Decision-Making Approach to Selective Use of Coronary Artery Calcium Measurement for Risk Prediction

Using 10-year ASCVD risk estimate plus coronary artery calcium (CAC) score to guide statin therapy:

<table>
<thead>
<tr>
<th>Patient's 10-year atherosclerotic cardiovascular disease (ASCVD) risk estimate:</th>
<th>&lt;5%</th>
<th>5-7.5%</th>
<th>&gt;7.5-20%</th>
<th>&gt;20%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consulting ASCVD risk estimate alone</td>
<td>Statin not recommended</td>
<td>Consider for statin</td>
<td>Recommend statin</td>
<td>Recommend statin</td>
</tr>
<tr>
<td>Consulting ASCVD risk estimate + CAC</td>
<td>Statin not recommended</td>
<td>Statin not recommended</td>
<td>Statin not recommended</td>
<td>Recommend statin</td>
</tr>
<tr>
<td>If CAC score = 0</td>
<td>Statin not recommended</td>
<td>Consider for statin</td>
<td>Recommend statin</td>
<td>Recommend statin</td>
</tr>
<tr>
<td>If CAC score &gt; 0</td>
<td>Statin not recommended</td>
<td>Statin not recommended</td>
<td>Statin not recommended</td>
<td>Recommend statin</td>
</tr>
<tr>
<td>Does CAC score modify treatment plan?</td>
<td>CAC not effective for this population</td>
<td>CAC can reclassify risk up or down</td>
<td>CAC can reclassify risk up or down</td>
<td>CAC not effective for this population</td>
</tr>
</tbody>
</table>

What is the future of preventive cardiovascular medicine?

“Precision medicine is poised to become the next great revolution in the practice of medicine, as well as in the maintenance of cardiovascular health and the prevention and cure of cardiovascular disease. Precision medicine disrupts standard practice and draws from clinical testing, electronic health records, pan-omics profiling, big data sets, and novel analytical methods, such as systems biology and network science, to create a person-specific phenotype that can then be used to identify an optimal intervention with minimal risk.”

“The obvious benefits of this approach to patients, clinicians, and researchers are numerous and include individual phenotype specificity, identification of individuals with a similar molecular phenotype, selection of best drugs or therapies with maximal efficacy and no or limited adverse reactions, efficient selection and enrichment of clinical trial participants, potential to improve adherence and reduce costs, and creating a paradigm shift in how cardiovascular care is delivered.”
FORMULA FOR SUCCESS: UNDER PROMISE AND OVER DELIVER

TOM PETERS

PICTUREQUOTES.COM

Treatment Approaches

Standard-of-care

Clinical Evaluation

Standard Algorithm

Generalized Recommendation

Precision Cardiology

Therapeutic Space

Multi-Oncic Information

Machine Learning

Data-Driven Recommendation

Clinician Review and Decision

Clinical Evaluation

Standard Algorithm

Generalized Recommendation

Multi-Oncic Information

Machine Learning

Data-Driven Recommendation

Clinician Review and Decision
• OR estimated at 4.4
• Lowest risk group is present in ~50%; Intermediate ~40%; Highest risk ~10%
Assessing Risk Factors as Potential Screening Tests

A Simple Assessment Tool

Nicholas J. Wald, FRS, FRCPI, Joan K. Morris, PhD

Many risk factors for disease are suggested as screening tests when there is little prospect that they could be useful in predicting disease. To avoid this, it is useful to know the relationship between the relative risk of a disease or disorder in people with high and low values of a risk factor, and the equivalent screening performance in terms of the detection rate (sensitivity) for a specified false-positive rate. We describe an interactive Risk-Screening Converter, accessible from the Internet (http://www.wolfson.qmul.ac.uk/rsc/), that transforms an odds ratio into the equivalent estimates of detection and false-positive rates. The converter is intended for general clinicians, for people engaged in research into risk factors and disease, and for those who give advice on applying such research findings into medical practice. It should help to distinguish effective screening methods from ineffective ones, and so improve clinical guidelines relating to screening and the prediction and prevention of disease. Arch Intern Med. 2011;171(4):286-291.

Graph of detection rate according to false positive rate for a specified odds ratio

Enter proportion of population in lowest group
Enter proportion of population in highest group
Enter odds ratio comparing highest group with lowest group

Unaffected

Affected

Specify the detection rate as a percentage

90.0%

False positive rate is 27.8% for a 90% detection rate

OR = 4.4

Comparing lowest 50% with highest 10%

Useless test

This Risk-Screening converter is available from www.wolfson.qmul.ac.uk/rsc/
Table 3 | Prevalence and clinical impact of a high GPS

<table>
<thead>
<tr>
<th>High GPS definition</th>
<th>Reference group</th>
<th>Odds ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Top 20% of distribution</td>
<td>Remaining 80%</td>
<td>2.55</td>
<td>2.43-2.67</td>
</tr>
<tr>
<td>Top 10% of distribution</td>
<td>Remaining 90%</td>
<td>2.89</td>
<td>2.74-3.05</td>
</tr>
<tr>
<td>Top 5% of distribution</td>
<td>Remaining 95%</td>
<td>3.34</td>
<td>3.12-3.58</td>
</tr>
<tr>
<td>Top 1% of distribution</td>
<td>Remaining 99%</td>
<td>4.83</td>
<td>4.25-5.46</td>
</tr>
<tr>
<td>Top 0.5% of distribution</td>
<td>Remaining 99.5%</td>
<td>5.17</td>
<td>4.34-6.12</td>
</tr>
</tbody>
</table>

Graph of detection rate according to false positive rate for a specified odds ratio

- Enter proportion of population in lowest group
- Enter proportion of population in highest group
- Enter odds ratio comparing highest group with lowest group

To obtain the detection rate (DR) for a specified false positive rate (FPR) enter "FPR" in the box below.
To obtain the false positive rate (FPR) for a specified detection rate (DR) enter "DR" in the box below.

Odds ratio per 1 sd increase in value of risk factor is 1.75
From the Abstract:

An elastic net Cox regression based with 586 unimputed variables with continuous values discretised achieved a C-index of 0.801 (bootstrapped 95% CI 0.799 to 0.802), compared to 0.793 (0.791 to 0.794) for a traditional Cox model comprising 27 expert-selected variables with imputation for missing values. We also found that data-driven models allow identification of novel prognostic variables; that the absence of values for particular variables carries meaning, and can have significant implications for prognosis; and that variables often have a nonlinear association with mortality, which discretised Cox models and random forests can elucidate. This demonstrates that machine-learning approaches applied to raw EHR data can be used to build models for use in research and clinical practice, and identify novel predictive variables and their effects to inform future research.
Comparison of modelling methods

We found that random forests did not outperform Cox models despite their inherent ability to accommodate nonlinearities and interactions [48, 49]. Random forests have a number of shortcomings which may explain this. First, only a random subset of variables \( m_{\text{sub}} \) are tried at each split, so datasets that contain a large proportion of uninformative ‘noise’ variables may cause informative variables to be overlooked by chance at many splits. Increasing \( m_{\text{sub}} \) can improve performance, but often at a large cost in computation time. Second, when random forests are used for prediction, the predictions are a weighted average of a subset of the data, and are biased away from the extremes [50]. This may partly explain their poor calibration.

We did not find that discretisation of continuous variables improved model performance, probably because the majority of these variables had associations with prognosis that were close to linear, and the small improvement in fit was offset by the large increase in the number of model parameters.

Disadvantages of data-driven approaches

Conventional statistical modelling techniques retain advantages and disadvantages which are the converse of these: models are more readily interpretable, and may generalise better, but at the expense of requiring significant expert input to construct, potentially not making use of the richness of available data, and only being applicable to complete data.

ORIGINAL INVESTIGATIONS

Genomic Risk Prediction of Coronary Artery Disease in 480,000 Adults

Implications for Primary Prevention
CONCLUSIONS

“The genomic score developed and evaluated in the present study strengthens the concept of using genomic information to stratify individuals for CAD risk in general populations and demonstrates the potential for genomic screening in early life to complement conventional risk prediction.”

A closer look

• METHODS: Using a meta-analytic approach to combine large-scale, genome-wide, and targeted genetic association data, we developed a new genomic risk score for CAD (metaGRS) consisting of 1.7 million genetic variants. We externally tested metaGRS, both by itself and in combination with available data on conventional risk factors, in 22,242 CAD cases and 460,387 noncases from the UK Biobank.

• RESULTS: The hazard ratio (HR) for CAD was 1.71 (95% confidence interval [CI]: 1.68 to 1.73) per SD increase in metaGRS, an association larger than any other externally tested genetic risk score previously published.

• The metaGRS stratified individuals into significantly different life course trajectories of CAD risk, with those in the top 20% of metaGRS distribution having an HR of 4.17 (95% CI: 3.97 to 4.38) compared with those in the bottom 20%.

• The metaGRS had a higher C-index (C = 0.623; 95% CI: 0.615 to 0.631) for incident CAD than any of 6 conventional factors (smoking, diabetes, hypertension, body mass index, self-reported high cholesterol, and family history).

J Am Coll Cardiol 2018;72:1883–93
Graph of detection rate according to false positive rate for a specified odds ratio

- Enter proportion of population in lowest group
- Enter proportion of population in highest group
- Enter odds ratio comparing highest group with lowest group

False positive rate is 30.6% for a 50% detection rate

Odds of being affected given a positive result (OAPR) is 1 : 26
The numbers for CAC: Highest 11%; Lowest (0) 50%; OR 10

Some additional concerns about the approach
Getting to “Precision” -- *Against the Odds*

Detection rates for a 10% FPR by OR comparing lowest 20% to highest 20%

Assessing Risk Factors as Potential Screening Tests: A Simple Assessment Tool
Nicholas J. Wald, FRS, FRCP; Joan K. Morris, PhD.

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**JAMA Internal Medicine | Special Communication**

**Potential Biases in Machine Learning Algorithms Using Electronic Health Record Data**

Milena A. Gianfrancesco, PhD, MPH; Suzanne Tamang, PhD, MS; Jinoos Yazdany, MD, MPH; Gabriela Schmajuk, MD, MS

JAMA Internal Medicine August 2018
Summary

- Current approaches to “precision cardiovascular prevention” are admittedly imperfect.
- But – current approaches allow considerable personalization and customization. NOT “one-size” fits all.
- Much of what is now being called “precision medicine” is not appreciably better than what already exists.
- A healthy skepticism, along with greater acceptance of, and confidence in, what we currently can do, is justified.
- George Box:

> All models are wrong, but some are useful.

— George E. P. Box —

---

<p>| Table. Sources of Bias in EHR Data and Their Potential to Contribute to Health Care Disparities |
|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|</p>
<table>
<thead>
<tr>
<th>Sources of Bias Entering EHR Systems</th>
<th>Potential to Differentially Affect Vulnerable Populations</th>
<th>Example of Biases With Respect to Clinical Decision Support Output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Missing data</td>
<td>Certain patients may have more fractured care and/or be seen at multiple institutions; patients with lower health literacy may not be able to access online patient portals and document patient-reported outcomes</td>
<td>The EHR may only contain more severe cases for certain patient populations and make erroneous inferences about the risk for such cases; conditioning on complete data may eliminate large portions of the population and result in inaccurate predictions for certain groups</td>
</tr>
<tr>
<td>Sample size</td>
<td>Certain subgroups of patients may not exist in sufficient numbers for a predictive analytic algorithm</td>
<td>Underestimation may lead to estimates of mean trends to avoid overfitting, leading to uninformative predictions for subgroups of patients; clinical decision support may be restricted to only the largest groups, sparing improvements in certain patient populations without similar support for others</td>
</tr>
<tr>
<td>Misclassification or measurement error</td>
<td>Patients of low socioeconomic status may be more likely to be seen in teaching clinics, where data input or clinical reasoning may be less accurate or systematically different than that from patients of higher socioeconomic status; implicit bias by health care practitioners leads to disparities in care</td>
<td>Algorithm inaccurately learns to treat patients of low socioeconomic status according to less than optimal care and/or according to implicit biases</td>
</tr>
</tbody>
</table>