patient study both as an encouragement and reinforcement that her healthy lifestyle changes resulted in measurable physiologic improvement.

Algorithms using AI-ECG have been validated as promising screening tools for specific cardiac pathologies, and the clinical application of AI continues to evolve as this area of research grows exponentially. It can be expected that AI tools will become a routine part of patient care. Here we offer an example of how AI-ECG results may be introduced in the physician-patient encounter both as a quantifiable marker of physical well-being and as a reinforcement of positive lifestyle changes.

Previously, we found that patients with an AI-ECG age greater than chronological age (a positive age gap) more frequently had pre-existing comorbidities including hypertension, coronary disease, and low ejection fraction frequently had pre-existing comorbidities. Patients with an AI-ECG age greater than chronological age had significantly less comorbidities than their “older” AI-ECG counterparts. In the present case, a young woman with obesity but no other cardiovascular comorbidities exhibited a decreasing AI-ECG age over 6-months’ time correlating with improved physical fitness and weight loss stemming from alterations in diet and exercise. The AI-ECG results were discussed with the patient to demonstrate that her lifestyle changes had a measurable, physiologic impact recorded by her heart, beyond simple weight change identified by a scale. Although significant physiologic changes may be represented by variation in AI-ECG age, further prospective study is needed to validate that “fitness” interventions result in a reduction of physiologic age.

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To the Editor: Arteriovenous fistulas are a common form of autogenous access in patients requiring renal replacement therapy. Aneurysmal degeneration of segments of the outflow vein and outflow stenosis are frequently identified. Albeit rare, arteriovenous fistulas bleeding can be a devastating and fatal dialysis access complication. Skin thinning and ulceration are signs of increased risk for bleeding or impending rupture. Immediate investigation of the fistula for any concerning signs of bleeding is imperative and any issues should prompt swift referral. Herein we present a case of impending fistula rupture.

The patient is a man in his mid-60s with a history of end-stage renal disease on hemodialysis secondary to hypertensive glomerulosclerosis. Access for hemodialysis was performed through a right brachiocephalic fistula created nearly 12 years before presentation. The patient had previous history of central outflow vein stenosis, with placement of a cephalic vein stent and several previous balloon venoplasty procedures. At the time of presentation, the patient was having no issues with hemodialysis. During dialysis session, the patient had small volume bleeding from...
the fistula; physical examination revealed significant skin thinning, a new ulceration, and a punctate area of bleeding (Supplementary Video, available online at http://www.mayoclinicproceedings.org). These findings led to urgent emergency department referral for evaluation and vascular surgery consultation.

On evaluation, the patient was hemodynamically stable with resolution of the bleeding with compression wrap placed for transfer. There were no stigmata of infection; however, there were signs of skin breakdown with impending hemorrhage, and blood could be seen “swirling” at the base of the wound. The patient was therefore admitted for further fistula revision. Ultrasound evaluation of the fistula showed only mild cephalic vein stenosis, normal flow volumes (1172 mL/min), as well as two aneurysmal segments each measuring 1.7 cm in diameter with the wound originating over the more central aneurysm. Before proceeding to the operating room, a tunneled dialysis catheter was placed, and the patient had a hemodialysis session.

The cephalic vein was mapped with ultrasound (Figure 1). Two incisions were made before exploring the ulcerated fistula, one central and the other peripheral to the aneurysmal segments. The cephalic vein was isolated and dissected free circumferentially in both exposed segments for vascular control before exposure of the aneurysm. At this point, the incisions were connected and the aneurysmal segments of vein were dissected free (Figure 2). The patient was heparinized, and vascular clamps were applied proximally and distally. The aneurysmal segments were explored. There was a large defect identified in the more central aneurysm with
thrombus preventing frank rupture (Figure 3).

Both aneurysmal segments of the cephalic vein were then opened, plicated, and the excess aneurysmal tissue was resected. A limited endovenectomy was performed to allow for sewing to be performed. The venotomy was then closed in two layers with a running polypropylene suture to a diameter of approximately 6 to 8 mm. After appropriate fore- and back-bleeding, the anastomosis was completed. Flow was restored through the fistula (Figure 4). There was a strong thrill over the fistula and preserved palpable radial pulse distally. The excess soft tissue and thin ulcerated skin were excised. The soft tissue and skin were then closed in multiple layers. Postoperative course was unremarkable, and the patient was discharged home on the second postoperative day. On follow-up, the patient was able to resume dialysis through the reconstructed AVF 2 months postoperatively.

AVF bleeding is associated with central venous stenosis, large aneurysms/pseudoaneurysms, infection, and skin ulceration. Fatal vascular access bleeding contributes to 0.4% to 1.6% of deaths in hemodialysis patients, although both fatal and nonfatal bleeding events are believed to be under-reported. Up to 40% of fatal vascular access bleeding events are preceded by a herald bleeding event or infection. Clinicians should be able to quickly recognize this entity for appropriate management. Treatment should be expeditious and is usually performed with endoaneurysmorrhaphy and reconstruction, and often patients require additional treatment for central venous stenosis. Our case shows an impending rupture of an arteriovenous fistula that, if left untreated, could have caused catastrophic

**FIGURE 3.** Full thickness disruption of venous wall of the cephalic vein aneurysm.

**FIGURE 4.** Endoaneurysmorrhaphy of cephalic vein.
hemorrhage. Endoaneurysmorhaphy and plication remain viable treat-
ment modalities to preserve a functional arteriovenous fistula, although this does require a period of temporary dialysis catheter use. We favor this approach over ligation in the appropriate clinical setting.

SUPPLEMENTAL ONLINE
MATERIAL
Supplemental material can be found online at http://www.
mayoclinicproceedings.org. Supplemental material attached to journal articles has not been edited, and the authors take responsibility for the accuracy of all data.

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Complement C5 Inhibition as a Novel Therapeutic Approach in Severe Pre-eclampsia

To the Editor: Determining the clinical diagnosis underlying thrombotic microangiopathy (TMA), a pathologic entity, may be challenging, as is its effective management. This is especially true when TMA occurs in pregnancy. We report a case of a woman presenting in mid-pregnancy with severe pre-eclampsia and acute TMA who was successfully managed with eculizumab.

The patient is a woman in her late 20s with gravidity G2P0010, prior end-stage kidney disease due to autosomal dominant tubulointerstitial disease, and a mutation in the uromodulin gene (UMOD). She received a living-related-donor kidney transplant 6 years previously. Her post-transplantation course was notable for urinary tract infections; however, there were no rejection episodes and no donor-specific antibodies. She was immunosuppressed with tacrolimus, predni-
sone, and azathioprine, because of her desire to become pregnant 5 years post-transplantation; graft function was stable (iothalamate glomerular filtration rate of 68 mL/min per 1.73 m²). She successfully conceived, but during an episode of urosepsis, she miscarried.

Her second pregnancy was initially uneventful, with a baseline serum creatinine (sCr) of 1.0 mg/dL, urinary protein 76 mg/24 h, and a normal blood pressure. At 13 weeks of gestation, she tested positive for coronavirus disease 2019, with mild upper respiratory symptoms. At 17 weeks of gestation, she presented with an acutely elevated sCr to 1.9 mg/dL, increased 24-hour urine protein (247 mg/24 h), and hypertension (139/91 mm Hg). One day later, she was admitted because of an additional rise in sCr (2.4 mg/dL); she was without systemic symptoms. Workup for acute allograft dysfunction was pursued (Table). ADAMTS13 activity was normal, her tacrolimus level was in the therapeutic range, and no new donor-specific antibodies were identified. The only positive serologic finding was a mildly elevated anti-phospholipid immunoglobulin M (IgM). A kidney biopsy was performed at 17 weeks and 6 days of gestation, which was diagnostic for TMA (Figure 1). Immunofluorescence studies revealed 1-2+ mesangial C1q positivity (not considered clinically relevant), arteriolar C3 positivity, and C4d negativity in peritubular capillaries, with trace to 1+ in the mesangium and arterioles.

In establishing a diagnosis, the following considerations were germane. Soluble membrane attack complex (sMAC) was mildly elevated, but not accompanied by abnormalities in the alternative complement pathway. There were no genetic mutations associated with atypical hemolytic-uremic syndrome and TMA. Tacrolimus-related kidney injury was not present on prior protocol biopsy specimens; however, tacrolimus was