**SUPPLEMENTAL DATA**

**Fig. S1.** Formation of a new bridging segment between two HUVEC vessel analogues. HUVEC-dsRed (top channel) and HUVEC-GFP (bottom channel) cells stimulated with 50 ng/ml VEGF invade the collagen gel region from opposite sides and connect to form a perfusable bridging vessel. Scale bar is 100 µm.
Fig. S2. Lateral anastomosis between adjacent HUVEC sprouts leads to vessel network formation. (A) Upon VEGF stimulation, adjacent HUVEC sprouts stimulated with VEGF can connect to form lateral bridging vessels (arrow). (B) Following 3 days of VEGF treatment, sprouts originating from the same aperture can connect laterally to initiate anastomosis (arrow). (C) 11 days after VEGF stimulation, two bridging vessels that had formed between the two pre-existing vessel analogues subsequently form lateral bridging segments leading to a branched, perfused vessel network. Images in (A) were acquired using epi-fluorescence microscopy. Images in (B) and (C) were projections of confocal images. Scale bars are 100 μm.
**Fig. S3.** Endothelial sprout formation is affected by the collagen gel concentration. HUVECs stimulated with VEGF for 2-3 days were induced to sprout into (A) 1.5 mg/ml or (B) 3 mg/ml collagen gel. The HUVECs in the 1.5 mg/ml collagen invaded as a discontinuous sprout (arrow). As shown, sprouts are extending from the bottom channel. Scale bars are 100 μm.
Fig. S4. Sprouting into the collagen gel is not restricted by aperture width. Upon stimulation with VEGF, HUVECs lining 50 µm wide apertures sprout into the bulk of the collagen gel.
Fig. S5. Anastomosis is enhanced by VEGF and inhibited by bevacizumab. HUVECs under static conditions exhibited more anastomosis events with increasing VEGF concentration. (A) 0, (B) 5 ng/ml, and (C) 50 ng/ml of VEGF was added to both HUVEC channels. (D) Addition of 500 ng/ml of bevacizumab to both channels neutralized VEGF (50 ng/ml) and inhibited anastomosis. Scale bars are 100 μm.