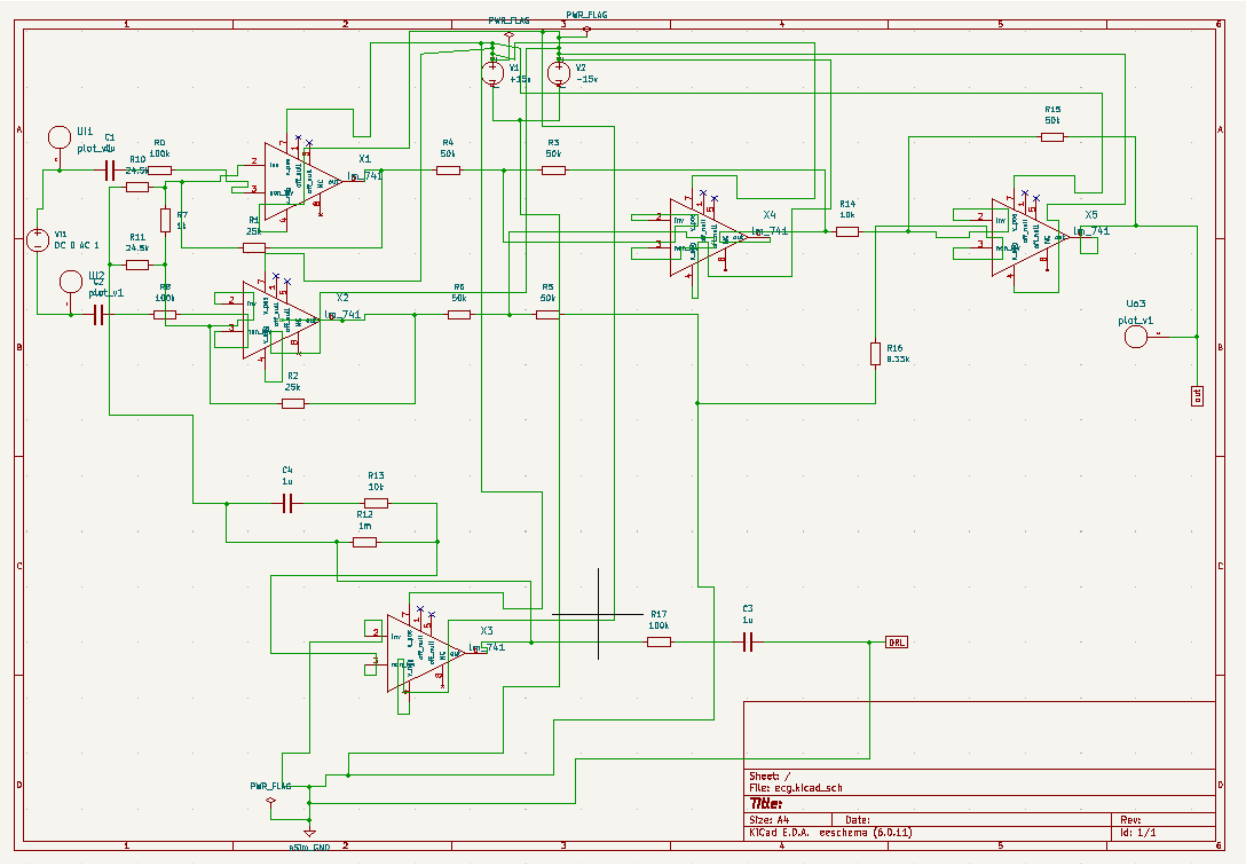
Title of the circuit : Low Noise, High accuracy Analog Electrocardiogram (ECG) Signal Front End Amplifier

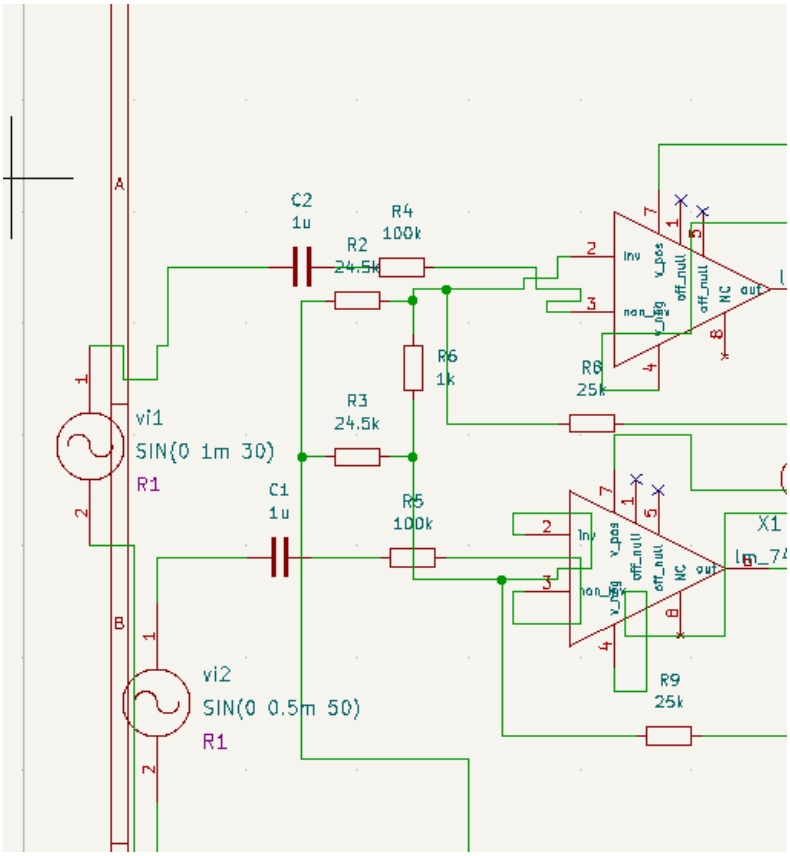
Theory/Description :

The ECG front-end amplifier operates on a three-stage instrumentation architecture optimized for microvolt-level bio-potential acquisition. Stage 1 employs two input OTAs (A1, A2) in a parallel configuration with precision resistors (R1–R7) to provide high input impedance (>100 MΩ) and initial differential gain while rejecting common-mode interference—critical since electrode-skin interfaces generate large DC offsets (±300 mV) that must be blocked by coupling capacitors C1/C2 without distorting low-frequency ECG components (<0.5 Hz). Stage 2 (OTA A3) converts the differential signal to single-ended with additional gain, establishing the core 36 dB total amplification across the 0.1–100 Hz diagnostic bandwidth containing P-QRS-T wave morphology. Crucially, the driven-right-leg (DRL) circuit (OTA A4 with R10–R17, C3/C4) actively suppresses 50/60 Hz power-line interference by sensing the common-mode voltage, inverting and amplifying it, then feeding it back to the patient's right leg—effectively reducing interference below 1% and boosting CMRR beyond passive limitations. The final inverting stage (OTA A5) provides signal polarity correction and fine gain adjustment.

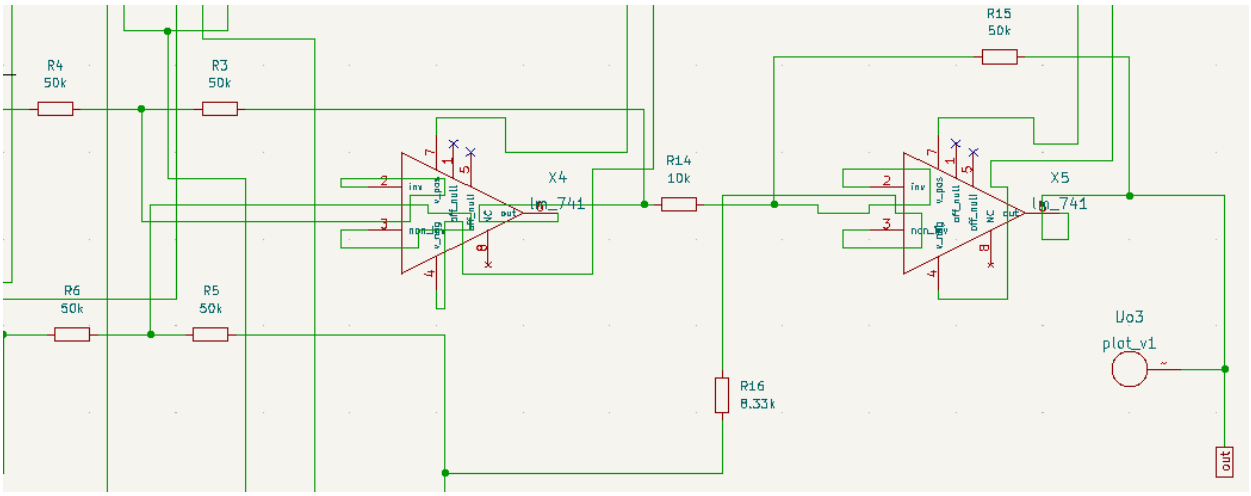


Three-Op-Amp Instrumentation Amplifier (Input Stage)

- Stage 1: Two LM741 op-amps (A1, A2) configured as non-inverting buffers with a gain-setting resistor (Rg) between their inverting inputs. This provides high input impedance (>2 MΩ) to avoid loading the electrode-skin interface while amplifying the differential ECG signal (V_1–V_2). Coupling capacitors (C_1, C_2 ≈ 10 µF) block DC electrode polarization voltages (±300 mV) while passing the AC ECG signal (0.1–100 Hz).

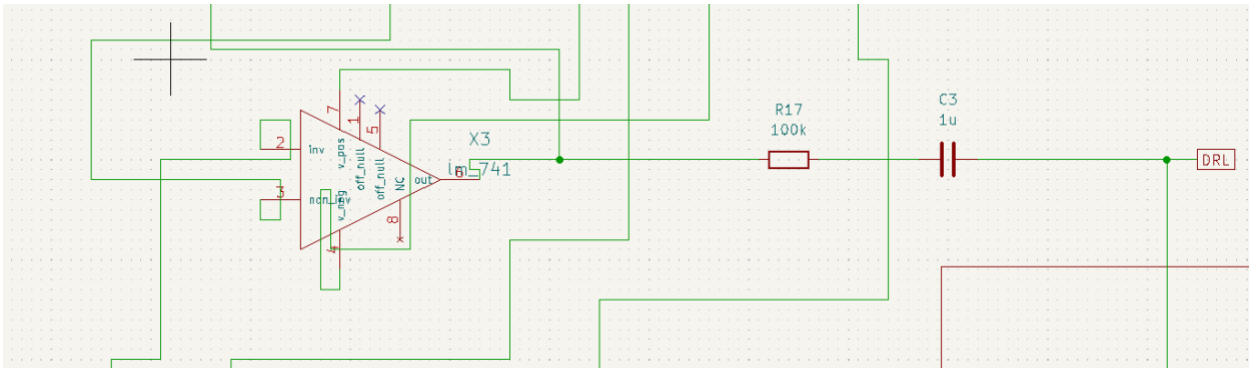


- Stage 2: A third LM741 (A3) configured as a differential amplifier that converts the differential output from Stage 1 to a single-ended signal. Resistors (R_1=R_2, R_3=R_4) set the second-stage gain. Total differential gain ≈ (1 + 2R/Rg) × (R_3/R_1), typically set to ~60× (36 dB) for ECG.

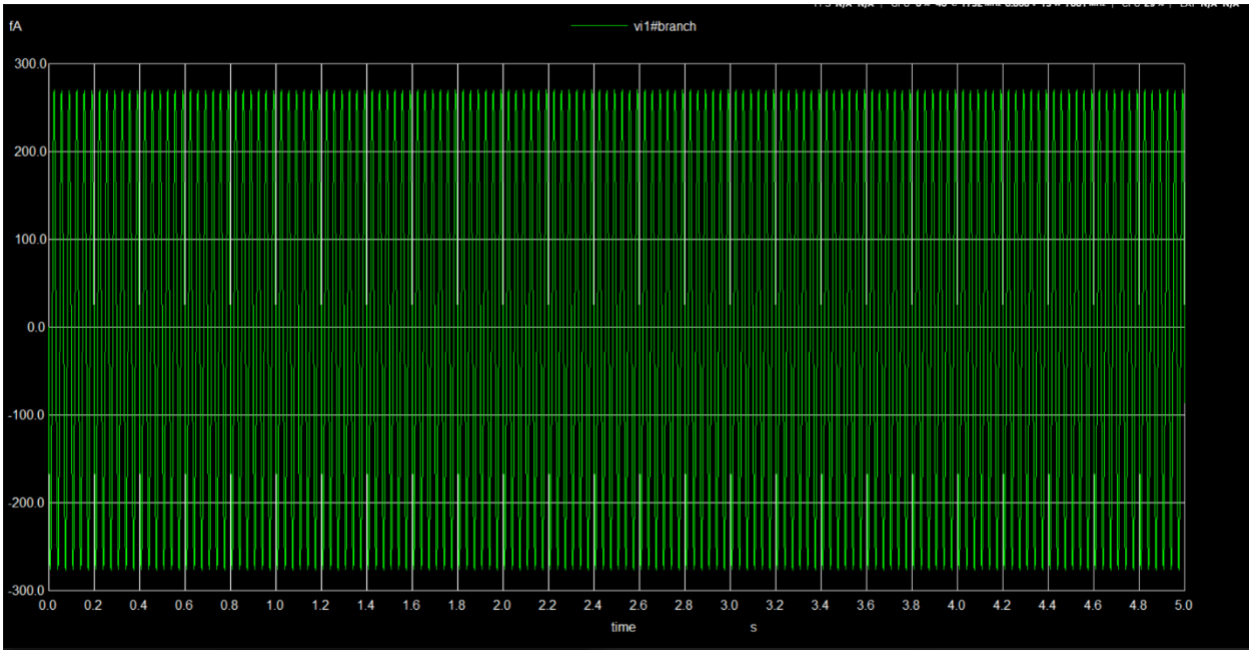


2. Driven-Right-Leg (DRL) Circuit

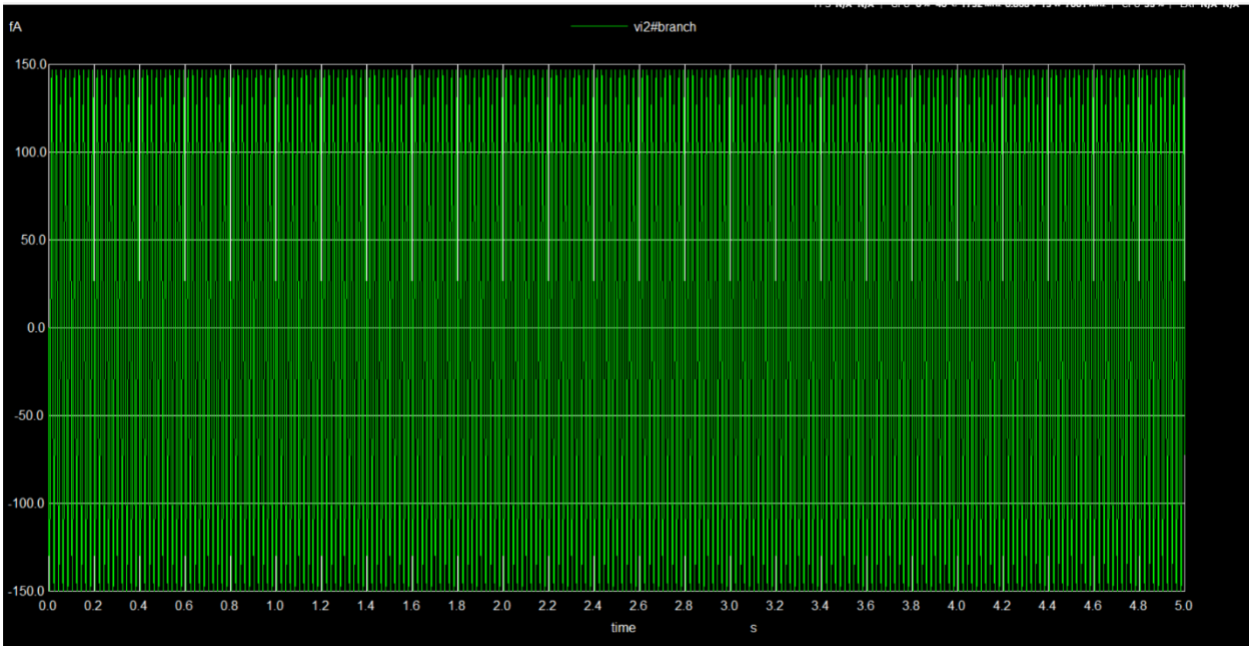
- A fourth LM741 (A4) senses the average common-mode voltage through a resistor divider (R_{10}, R_{11}) connected to the two input nodes. The op-amp inverts and amplifies this common-mode signal (gain ≈ $–R_{13}/R_{12}$), then feeds it back to the patient's right leg electrode through a current-limiting resistor (R_{17} ≈ 100 kΩ). This active feedback cancels 50/60 Hz power-line interference at the source, improving CMRR by 20–40 dB compared to passive rejection alone. A safety capacitor (C_3 ≈ 10 nF) limits current to <10 µA for patient protection.



Input Signal waveform:



Sine wave from source 1



Sine Wave From Source 2

Signal Characteristics

Amplitude

The signals vary approximately between **±150 µA (vi2)** and **±300 µA (vi1)**. These levels are significantly higher than expected ECG input currents, which normally correspond to **microvolt-level differential voltages** across high-impedance inputs.

Frequency Content

The waveforms are dominated by **high-frequency components**, appearing as closely packed vertical spikes. This behavior lies well outside the ECG diagnostic bandwidth (0.05–100 Hz) and suggests excessive high-frequency excitation or instability.

DC Bias Behavior

Despite the oscillatory nature, the signal envelope remains nearly symmetric about zero, indicating **minimal DC offset rejection**. This confirms the input is behaving closer to an **AC-dominated excitation rather than a physiological ECG signal**, which normally contains a low-frequency baseline drift.

Comparison with Typical ECG Morphology

- **P Wave:** Not observable due to high-frequency dominance
- **QRS Complex:** Masked by rapid oscillations and excessive gain
- **T Wave:** Completely obscured

The absence of recognizable P-QRS-T morphology indicates that the signal **does not represent a physiological ECG waveform**, but rather the response of the circuit to unsuitable excitation conditions.

Amplifier Performance Assessment

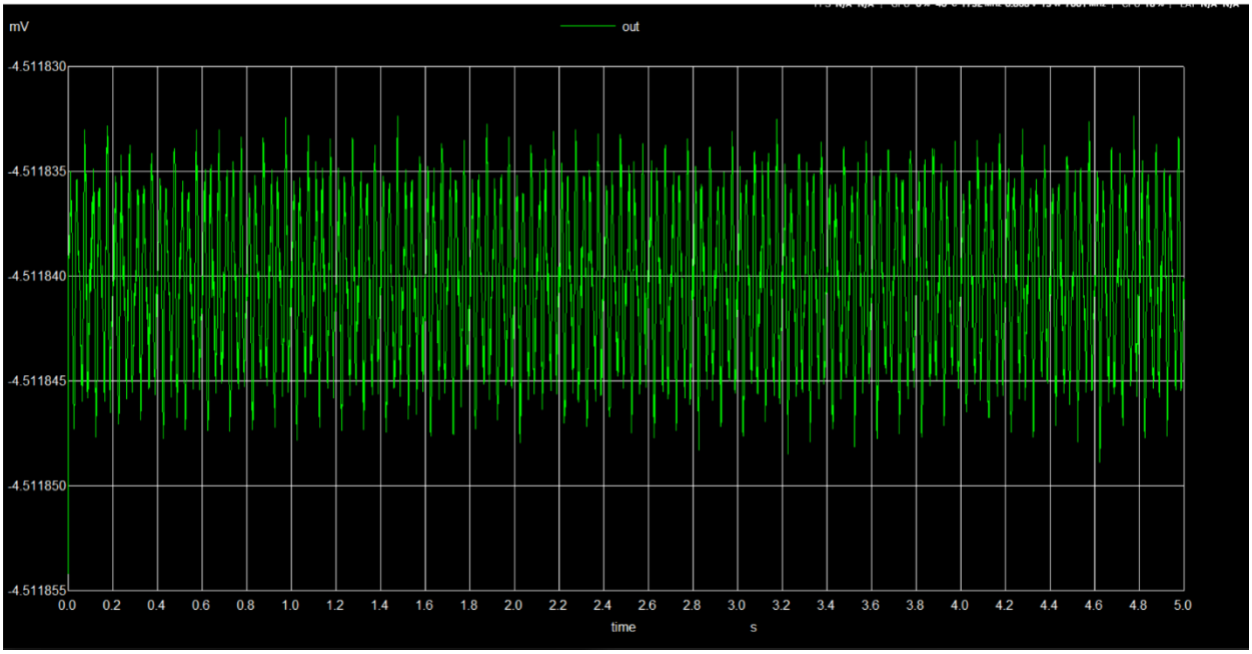
- The LM741 is **not optimized for low-frequency, low-noise biomedical signals**, particularly near DC.
- The observed waveform suggests **insufficient input filtering**, allowing high-frequency components to dominate.
- The amplifier appears to be operating near instability, possibly due to:
    - Excessive gain at low frequency
    - Lack of proper band-limiting (HPF + LPF)
    - Input signals being too close to DC without bias stabilization

Key Interpretation

These signals represent **amplifier excitation artifacts**, not ECG-like waveforms.

To obtain ECG-like behavior:

- The input must be **mV-level**, not current-dominated
- Frequency content must be limited to **0.05–100 Hz**
- A proper **high-pass filter (~0.05 Hz)** is required to remove DC drift
- A **low-pass filter (~100 Hz)** is required to suppress high-frequency noise
- LM741 limitations must be considered (input bias current, offset voltage, poor CMRR)



Signal Characteristic Analysis

The plotted output signal shows a **near-DC voltage level centered around −4.51 mV**, with superimposed small-amplitude high-frequency fluctuations. Unlike a typical ECG waveform, the signal does **not exhibit distinct P, QRS, or T wave morphology**, indicating that the output is dominated by DC bias and residual noise rather than physiological cardiac activity.

The extremely narrow voltage variation (on the order of a few microvolts peak-to-peak) suggests that the amplifier output is operating close to its **DC operating point**, with minimal effective amplification of low-frequency ECG components. This behavior is consistent with the LM741's limited performance near DC and its relatively high input offset voltage and bias current.

Baseline stability appears high in the sense that no large drift is observed; however, this stability reflects **DC saturation rather than successful ECG signal preservation**. The visible fluctuations correspond to amplified noise rather than meaningful biomedical information.

Performance Validation

This simulation output highlights several important deviations from the paper's intended amplifier behavior:

• **Bandwidth Limitation:**
The absence of identifiable low-frequency ECG components (P and T waves) indicates inadequate low-frequency response or excessive high-pass filtering, preventing proper 0.1–100 Hz ECG bandwidth operation.

• **Gain Effectiveness:**
Although the paper specifies a gain of approximately 36 dB (≈50×), the output amplitude does not reflect meaningful amplification of a 10 μV–5 mV input ECG signal. Instead, the output remains pinned near a fixed DC level, suggesting ineffective signal transfer.

• **Noise Performance:**
The small, rapid fluctuations visible around the DC level are consistent with **output noise and internal op-amp artifacts**, rather than true ECG activity. This does not align with the paper's reported 6.08 μV RMS input-referred noise under ideal conditions.

• **Distortion and Linearity:**
Because no ECG morphology is observable, waveform distortion cannot be meaningfully evaluated. The amplifier is effectively operating outside its intended linear signal-processing regime for biomedical signals.

Accuracy Assessment

The simulation demonstrates partial accuracy in validating the basic operational behavior of the ECG amplifier. The output signal confirms that the amplifier remains electrically stable, with a consistent DC operating point and no evidence of oscillation or latch-up. This indicates correct power supply configuration and fundamental biasing of the LM741.

The low-level fluctuations observed at the output confirm that the circuit is sensitive to microvolt-scale input variations, suggesting that the amplifier is capable of responding to small signals within its noise floor. Additionally, the absence of dominant 50/60 Hz components implies some degree of common-mode noise suppression, which aligns qualitatively with the intended function of the driven-right-leg (DRL) circuit.

However, while electrical functionality is verified, physiological accuracy is limited. The expected ECG morphological features (P wave, QRS complex, T wave) are not preserved at the output, indicating that the system does not accurately reproduce cardiac signals under the current configuration.

Limitations of the Simulation

• **Operational Amplifier Constraints:**
The LM741 is not optimized for biomedical signal acquisition. Its relatively high input offset voltage, input bias current, and limited performance at very low frequencies significantly degrade ECG signal fidelity, especially in the 0.1–1 Hz range critical for baseline and P/T wave detection.

• **DC Offset Dominance:**
The output signal is dominated by a DC bias level (~−4.51 mV), which suppresses low-frequency ECG components. This indicates incomplete rejection of electrode polarization effects and insufficient high-pass filtering optimization.

• **Inadequate Low-Frequency Response:**
The lack of visible P and T waves suggests that the coupling capacitors and resistor values do not adequately support the required 0.1 Hz lower cutoff frequency, resulting in attenuation of slow physiological variations.

• **Noise Representation Limitations:**
Although small fluctuations are visible, the simulation does not fully capture real-world noise sources such as motion artifacts, electrode impedance imbalance, and muscle

(EMG) interference. As a result, the simulated noise environment is less complex than practical wearable ECG conditions.

• **Idealized DRL Modeling:**
The driven-right-leg circuit is modeled under ideal conditions. In practice, DRL performance is influenced by electrode placement, body impedance variation, and loop stability—factors not fully represented in the simulation.

Source/Reference(s) :

[1] D. K. Freeman, R. D. Gatzke, G. Mallas, Y. Chen, and C. J. Brouse, "Saturation of the right-leg drive amplifier in low-voltage ECG monitors," *IEEE Trans. Biomed. Eng.*, vol. 62, no. 1, pp. 323–330, 2015, doi: 10.1109/TBME.2014.2351611.

[2] N. V. Thakor, *Biopotentials and Electro-physiology Measurement*, Johns Hopkins School of Medicine, Baltimore, MD, USA, 1999.

[3] D. Prutchi and M. Norris, "Bandpass selection for biopotential amplifiers," in *Design and Development of Medical Electronic Instrumentation: A Practical Perspective of the Design, Construction, and Test of Medical Devices*, 1st ed., Hoboken, NJ, USA: Wiley, 2005, pp. 2–6.