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PROVA SCRITTA N. 1

- Diagnosi e trattamento dello scompenso cardiaco a funzione sistolica preservata

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PROVA SCRITTA N. 2

- Valore prognostico e trattamento a lungo termine della fibrillazione atriale post cardiochirurgica

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PROVA SCRITTA N. 3

- Diagnosi e trattamento della stenosi aortica asintomatica

ASST CENTRO SPECIALISTICO ORTOPEDICO TRAUMATOLOGICO GAETANO PINI / CTO
MILANO

ID:
Nato/a:
anni,

[redacted]

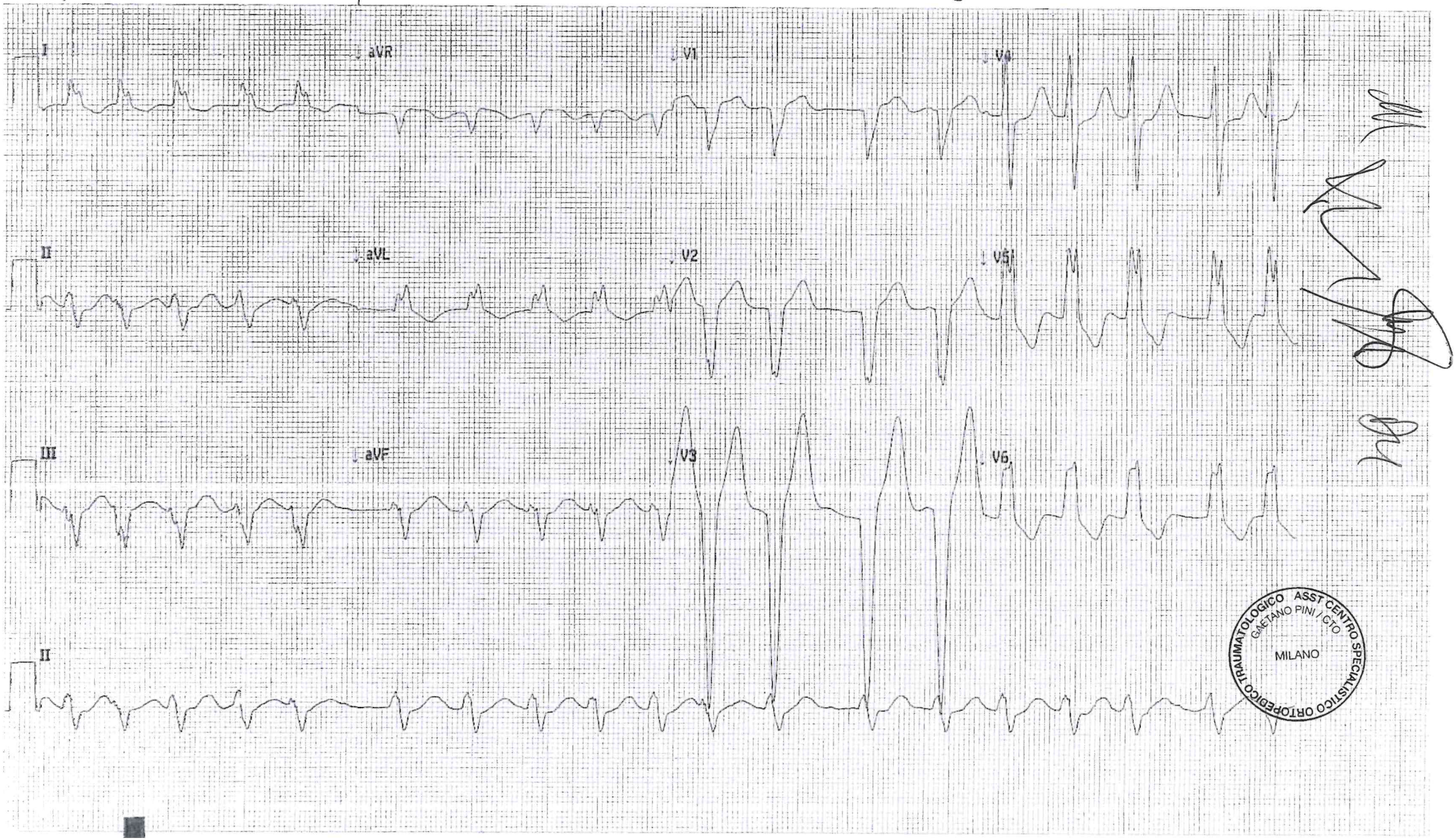
M-83
28/07/42

17-Set-2025 2:55:37

Freq Vent:	112 BPM
Int PR:	0 ms
Dur QRS:	143 ms
QT/QTc:	386/452 ms
Assi P-R-T:	999 -47 117

[redacted]

PROVA PRATICA
N. 1



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MILANO

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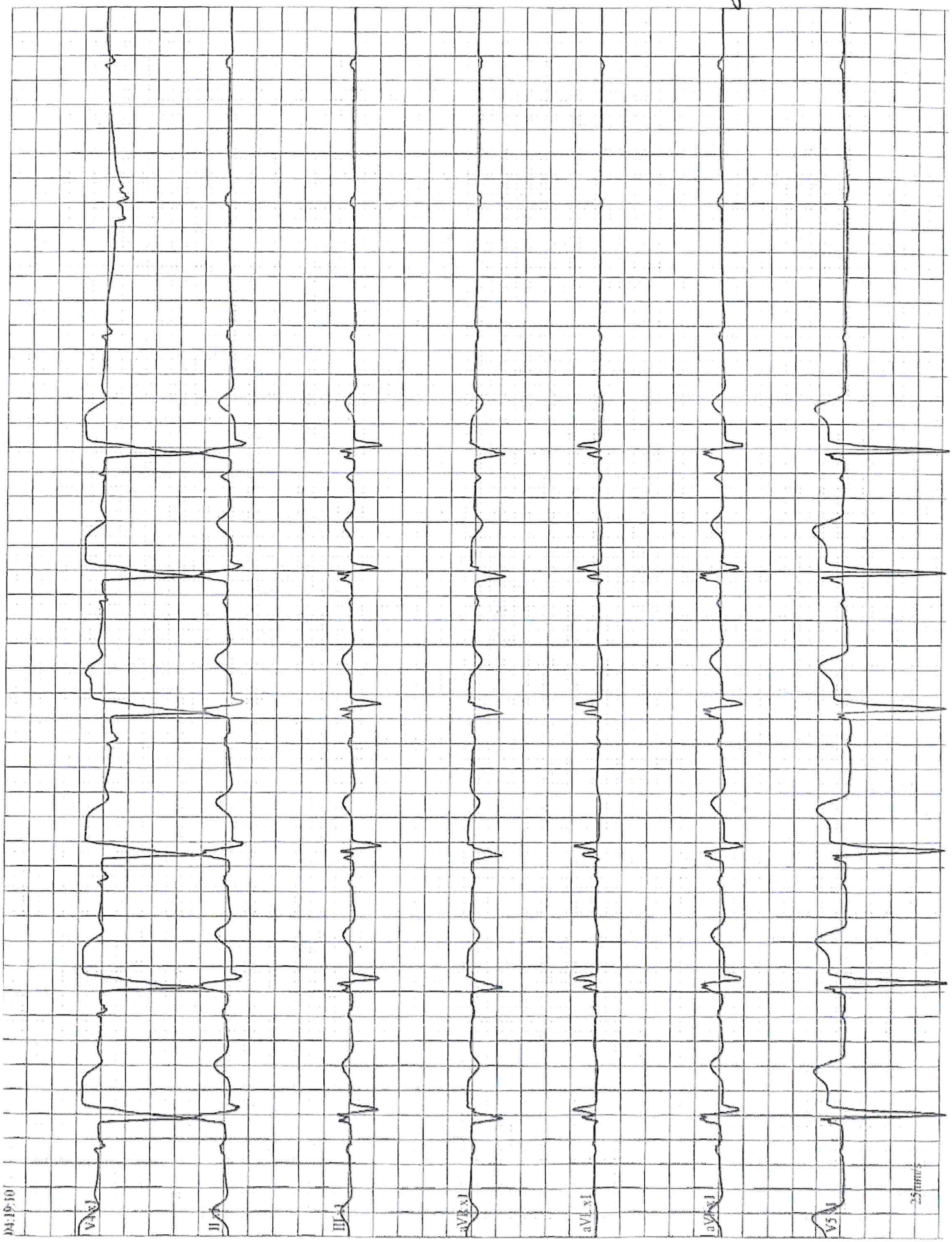
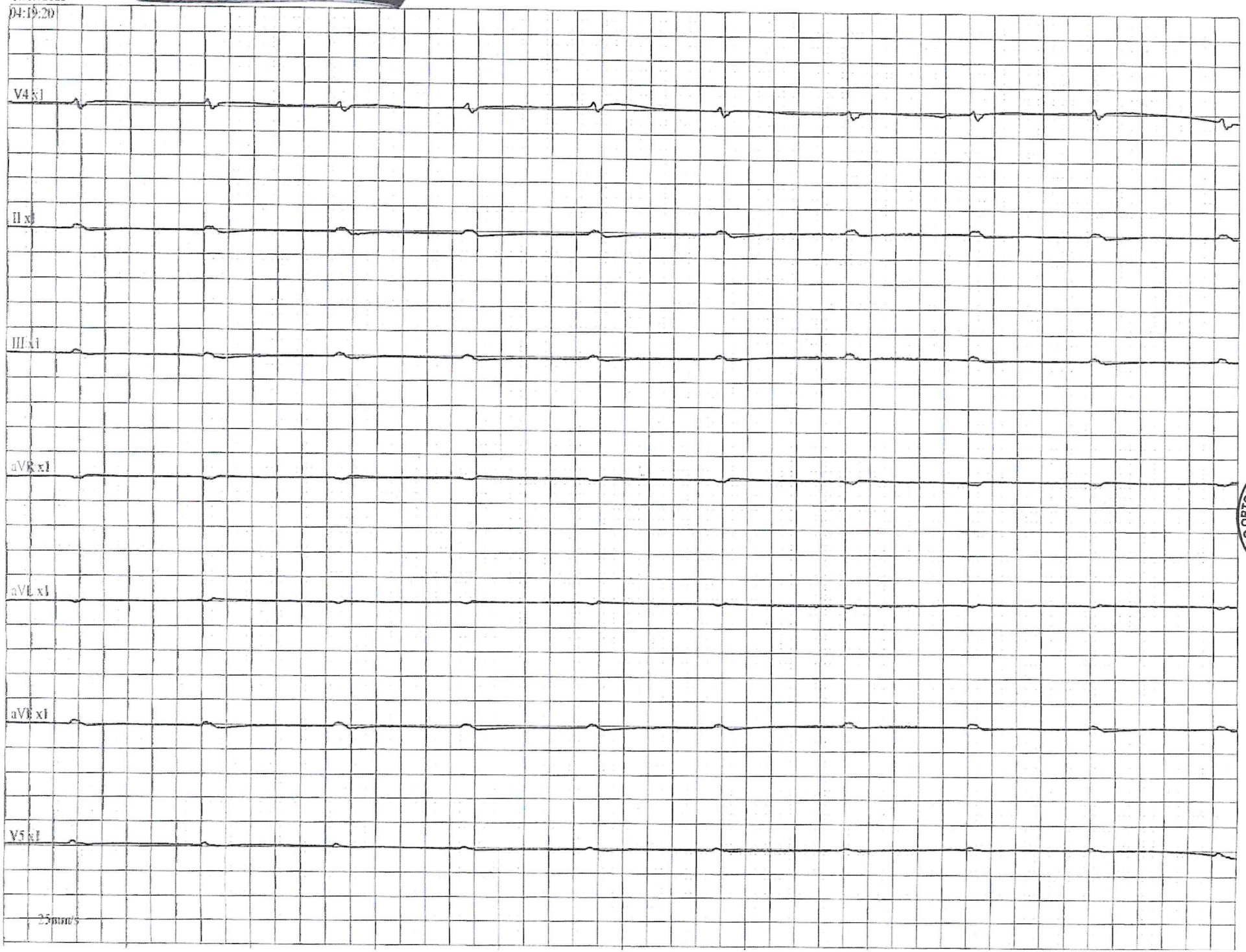
PROVA PRATICA
N. 2



Full Disclosure Zoom - [redacted]
17/09/2025



PROVA PRATICA
N° 2

CENTRO SPECIALISTICO ORTOPEDICO TRAUMATOLOGICO
ASST GAETANO PINI/CTO
MILANO

Disclosure Zoom --

7/09/2025

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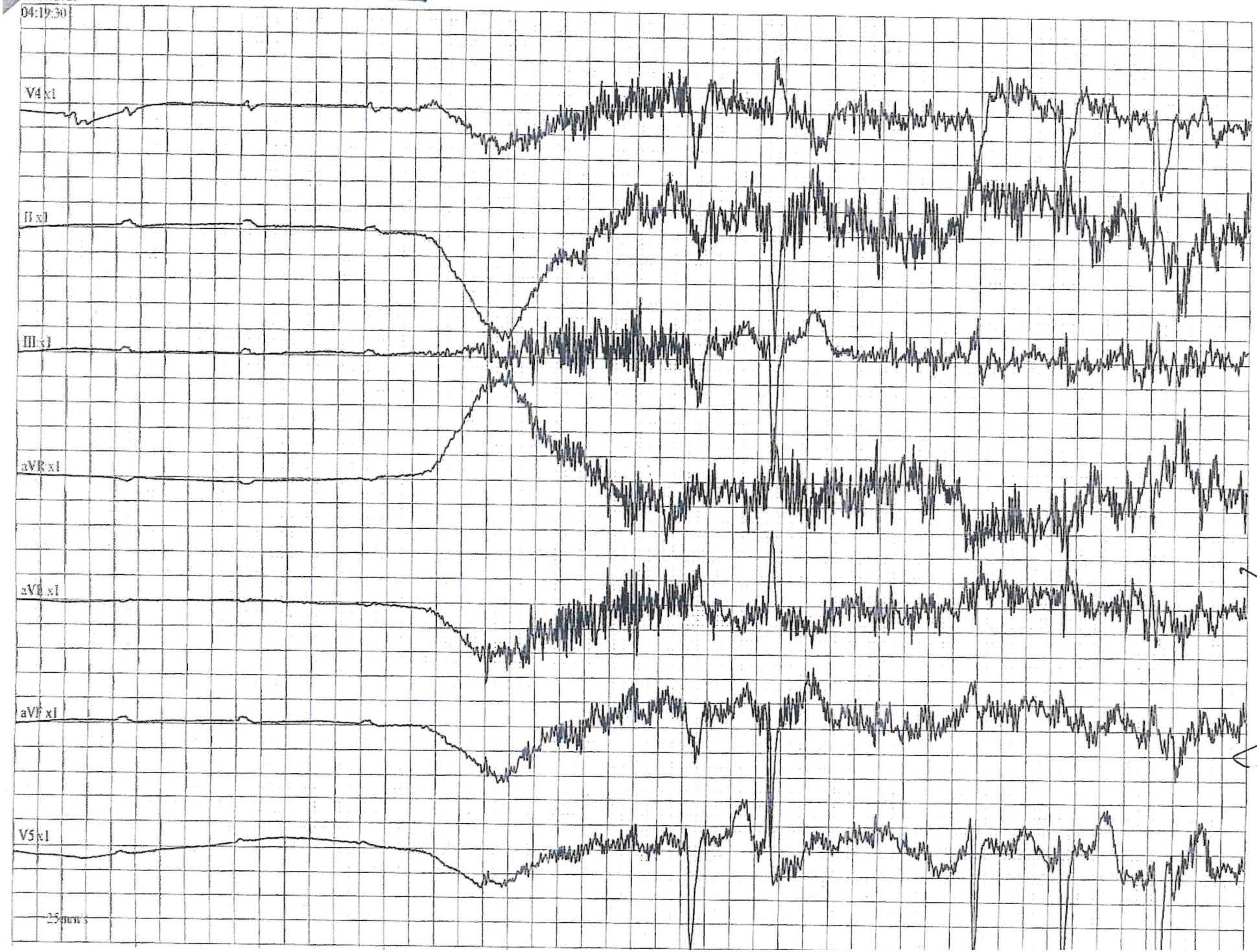

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16/09/2025

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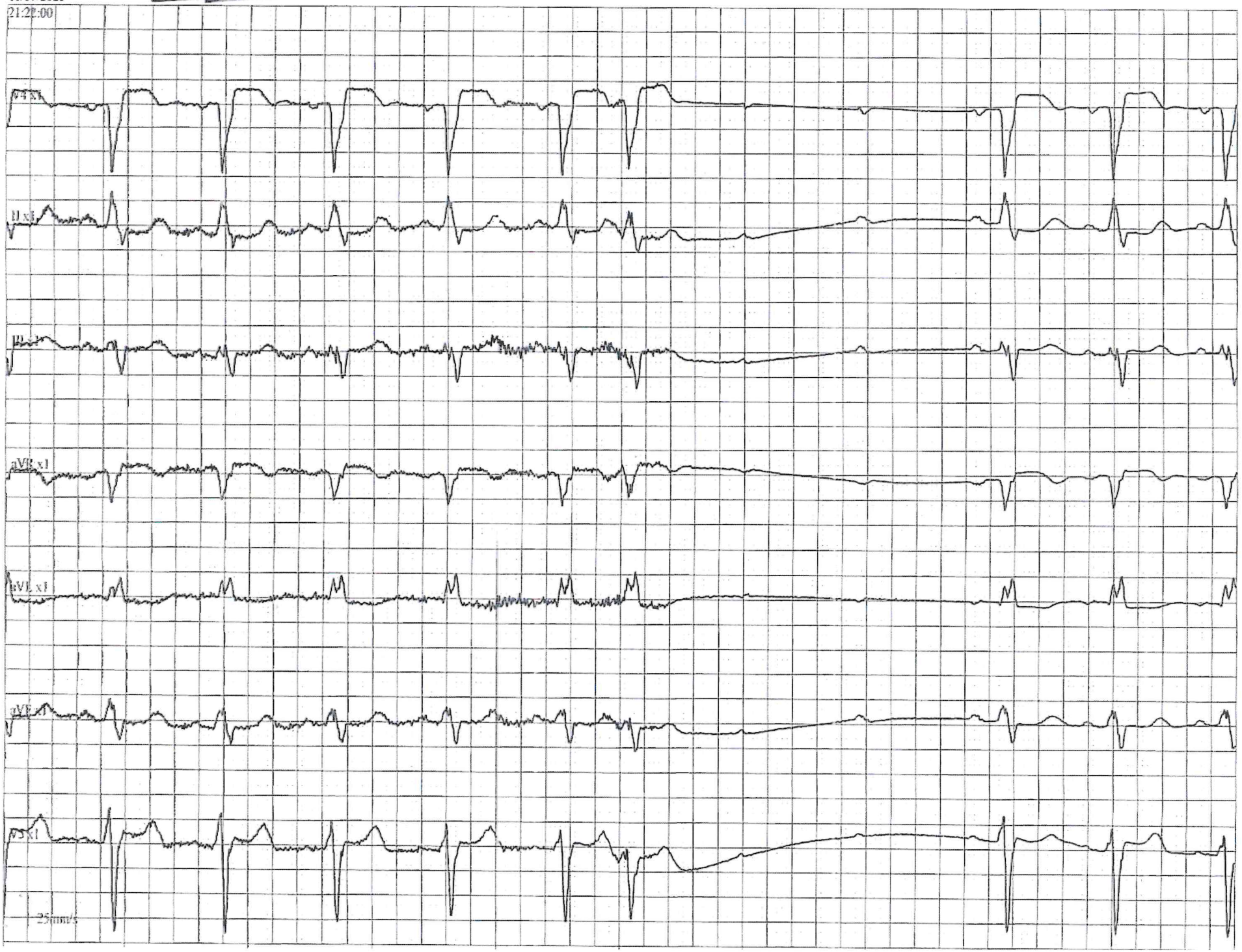
17/09/2025 11:07 (1)



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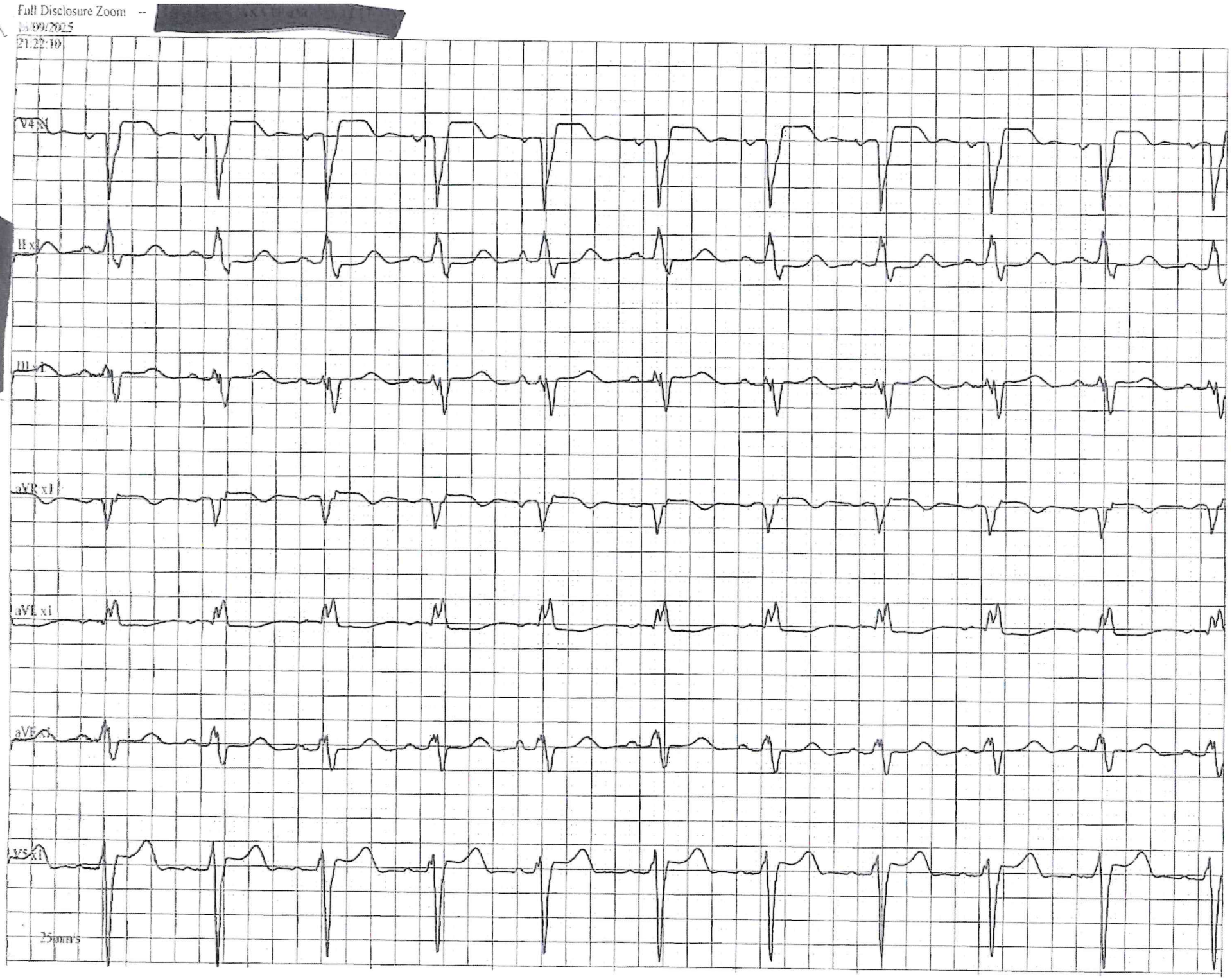
PROVA PRATICA n° 2



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PROVA ORALE N. 1

Domanda:

Trattamento conservativo della cardiopatia ischemica

Accertamento lingua inglese:

Si veda allegato

Accertamento conoscenze informatiche:

Che cosa è un PACS?

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PROVA ORALE N. 2

Domanda:

La disfunzione del microcircolo

Accertamento lingua inglese:

Si veda allegato

Accertamento conoscenze informatiche:

Che cosa è un PACS?

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PROVA ORALE N. 3

Domanda:

La sindrome delle apnee ostruttive in cardiologia

Accertamento lingua inglese:

Si veda allegato

Accertamento conoscenze informatiche:

Che cosa è un PACS?

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Testo di inglese:

6. Lipoprotein(a)

Epidemiologic and genetic studies strongly support a likely causal and direct continuous association between high plasma levels of Lp(a) and higher risk of ASCVD and aortic valve stenosis (AVS). Emerging data are suggesting that high Lp(a) may confer a higher risk of ASCVD and AVS per particle or cholesterol content than LDL-C. The mechanisms by which Lp(a) and LDL lead to these increased risks therefore likely differ. Based on studies from the United Kingdom, Denmark, and the United States of America, the highest risks were for MI and AVS, less in peripheral artery disease and heart failure, and the lowest risks were for ischaemic stroke and CV and all-cause mortality. Risk from high Lp(a) increases slightly at levels of 30 mg/dL (62 nmol/L) to 50 mg/dL (105 nmol/L) and becomes clinically relevant above 50 mg/dL (105 nmol/L), with higher levels associated with a greater increase in CV risk. Lp(a) concentration is predominantly determined by genetics (>90%), more than any other lipoprotein, and levels vary with ethnicity. There is an incremental increase in risk caused by higher Lp(a) levels, and if Lp(a) level is not considered, risk might be substantially underestimated. Lp(a) measurement should be considered at least once in every adult's lifetime,1 either at the first lipid profile or at the next one if lipid profiles have previously been performed. Screening is particularly relevant in younger patients with FH or premature ASCVD and no other identifiable risk factors, or a family history of premature ASCVD or high Lp(a) levels, or in individuals at moderate risk or around treatment decision thresholds to improve risk classification. Lp(a) levels may increase after menopause and a second measurement is reasonable, particularly if the pre-menopausal levels were borderline. For Lp(a) measurement, there is substantial variability among different assays, in part relating to apo(a) structure and variability in Kringle-IV repeats, potentially under- or overestimating Lp(a) levels. Although measurement in molar units (nmol/L) is preferred, mass units (mg/dL) can be used for clinical purposes.

Although Lp(a) has not yet been shown to improve risk prediction on top of currently recommended scores (SCORE2, SCORE2-OP), another study testing whether elevated Lp(a) could improve MI risk prediction beyond conventional risk factors showed that Lp(a) concentrations >47 mg/dL reclassified 23% of first MI events correctly, while no events were reclassified incorrectly, in a cohort of 8720 individuals.

Whether lowering Lp(a) reduces the risk of ASCVD and AVS progression has yet to be shown; the extent of Lp(a) lowering required for clinical benefit is also not known. In the absence of specific Lp(a)-lowering therapies, early risk factor management and more intensive LDL-C lowering is reasonable, considering both absolute CV risk and Lp(a) levels. An online Lp(a) risk and benefit algorithm is available at http://www.eas-society.org/LPA_risk_and_benefit_algorithm. Although small studies have suggested that statins may have a slight Lp(a) increasing effect, individual-level data on participants in seven randomized, placebo-controlled, statin outcome trials found that statins had no effect on Lp(a) concentrations. Therefore, clinical decision-making should be influenced by the degree of Lp(a) elevation and the patient's other risk factors, and patients with high levels of Lp(a) should be strongly encouraged to take or continue to take high-intensity statins if their risk is sufficiently high.

Currently, there are specific Lp(a)-lowering medications being tested in randomized clinical trials. The injectable RNA (either antisense oligonucleotide or small interfering RNA)-based therapies that target apolipoprotein(a) production in the hepatocyte lower Lp(a) concentration by 80%–98%. An oral small molecule inhibitor and a small interfering RNA that can lower Lp(a) significantly are currently under investigation.