Table 2
Attending perceptions of the impact of the RAT on ACGME competencies.

Competency	Strongly negative	Negative	Neutral	Positive	Strongly positive
	n (%)	n (%)	n (%)	n (%)	n (%)
Recognition of critically ill patients	0 (0)	1 (4)	20 (80)	3 (12)	1 (4)
Performance of focused history and physical	0 (0)	5 (20)	18 (72)	2 (8)	0 (0)
Interpretation of diagnostic studies	1 (4)	2 (8)	18 (72)	4 (16)	0 (0)
Creation of differential diagnosis	1 (4)	8 (32)	13 (52)	3 (12)	0 (0)
Implementation of appropriate pharmacotherapy	1 (4)	6 (24)	16 (64)	2 (8)	0 (0)
Observation and reassessment of patients	2 (8)	2 (8)	16 (64)	4 (16)	1 (4)
Disposition making	1 (4)	1 (4)	12 (48)	10 (40)	1 (4)
Ability to multi task	0 (0)	4 (16)	15 (60)	5 (20)	1 (4)
Medical knowledge	0 (0)	2 (8)	18 (72)	5 (20)	0 (0)
Patient safety	1 (4)	2 (8)	10 (40)	10 (40)	2 (8)

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Practice behavior of emergency department physicians caring for patients with chest pain

Chest pain is the most common medical diagnosis among adult patients admitted to emergency departments (EDs) around the world [1,2]. Several risk stratification tools have been devised to aid physicians in decision making. Three well-known risk scores are the TIMI (Thrombolysis in Myocardial Infarction) [3], GRACE (Global Registry of Acute Coronary Events) score [4], and HEART (History, ECG, Age, Risk factors and Troponin) score [5]. These scores utilize clinical, electrocardiogram (ECG), and laboratory data to identify patients in the ED who are at the highest risk for short term acute coronary syndrome (ACS) or major adverse cardiac events (MACE), and who may benefit most from aggressive therapies. The current study investigated the practice behavior of emergency physicians in caring for patients with chest pain and addressed the following; a) whether their decisions are based on validated risk-stratification scoring systems, b) whether their assessments were compatible with those of physicians' in internal medicine departments regarding the short term risk for major cardiac events and c) the management and outcomes of these patients.

The study entailed a prospective non-interventional design in which data were collected from admission notes at the ED and departments of internal medicine. We included adult patients who presented to the ED with a chief complaint of non-traumatic chest pain for which no definitive non-ischemic cause was found.

A total of 4589 patients comprised the study cohort. Of these, 763 (17%) were hospitalized for further investigation and workup. Nearly 74% of ED admission notes (for admitted and discharged patients) lacked any formal risk assessment. The rest (26%) used medical calculators available on smartphone applications. Table 1 shows reasons for hospitalization based on notes made by ED admitting physicians. The reason relevant to the largest proportion of patients was atypical chest pain and abnormal hs-cTnT (32.8%). For 16.7% of patients, hospital admissions were due to social and medicolegal concerns. Nearly one-third of hospitalized patients had a HEART score of 1–3, the rest (68%) had scores ≥4. Patients who were discharged from the ED were mostly younger males with low HEART scores (Table 2). Rates of MACE within 30 days of discharge from the ED or of hospitalization were 1.4% and 0.08% for hospitalized and discharged patients, respectively. Altogether, 10% (n = 464) of the patients in this cohort eventually underwent coronary angiography; significant coronary disease was found in 58%.

Table 1
Reasons for hospitalization of patients with chest pain based on ED[a] physicians' notes.

Summary of emergency physicians notes	No. of hospital admissions (%) n = 763
Unstable angina/non ST-elevation MI[a] (typical chest pain with new ECG changes and/or elevated hs-cTnT)[b]	136 (17.8)
Typical chest pain with multiple[c] cardiovascular risk factors (normal hs-cTnT, without new ECG changes)	75 (9.8)
Atypical chest pain with multiple[c] cardiovascular risk factors (normal hs-cTnT, without new ECG changes)	174 (22.8)
Atypical chest pain with abnormal hs-cTnT (with or without cardiovascular risk factors, and without new ECG changes)	251 (32.9)
Other[d] (normal hs-cTnT, without new ECG changes)	127 (16.7)

[a] ED; emergency department, MI; myocardial infarction.
[b] hs-cTnT: high sensitivity cardiac troponin-T.
[c] Multiple; ≥ 3 risk factors.
[d] Patient's or spouse's concern for adverse outcomes, patients' refusal for outpatient workup, and ≥2 ED visits due to chest pain within 1 week.

Table 2
Characteristics of patients included in the study, chest pain workup, and their outcome.

	Hospitalized (n = 763)	Discharged (n = 3826)	p value
Mean age (range)	61.6 (33–94)	47 (27–93)	p < 0.001
Male (%)	68%	62.5%	p < 0.001
Level of training of ED admitting physician			
Resident	81%	72%	p < 0.001
Board-certified in internal medicine	17%	23%	p = 0.003
Board-certified in emergency medicine	2%	5%	p = 0.004
Mean HEART score			
1–3	32%	72%	p < 0.001
4–5	24%	17%	p < 0.001
6–7	29%	9%	p < 0.001
8–10	15%	2%	p < 0.001
Chest pain workup			
Treadmill stress test	204 (26.7)	312 (18.5)	p < 0.001
Stress test with nuclear imaging or echocardiography	62 (8.1)	176 (4.6)	p < 0.001
CCTA[a]	11(1.4)	68 (1.8)	NS
Coronary angiography	218 (28.6)	246 (6.4)	p < 0.001
None	383 (50.2)	2953 (77.2)	p < 0.001
MACE[b] within 30 days (no. of patients)	1.4% (11)	0.08% (3)	p < 0.001

[a] CCTA; coronary computerized tomography angiography.
[b] MACE; major adverse cardiac events.

This study showed that the practice of emergency physicians in this teaching hospital lacked formal risk assessment. When we applied the HEART score, nearly one-third of the patients who were hospitalized due to a risk of ACS, had a low score. This is despite sufficient data suggesting that patients with low HEART scores could be safely discharged from the ED for ambulatory workup [6]. In general, emergency physicians overestimated the risk of coronary heart disease, without substantiating their assessment. Resident physicians more often decided to hospitalize and board-certified physicians more often decided to discharge patients. Another study stated possible reasons for overestimating risk for ACS as fewer years of clinical experience of emergency physicians, patients' belief about their own risk of cardiovascular disease, and perhaps poor communication between emergency physicians and patients [7].

Reasons for hospitalization of patients with chest pain were variable; the most common reason was atypical chest pain and abnormal hs-cTnT (32.6%), regardless of the presence of cardiovascular risk factors. We believe that the relatively large proportion of patients fitting this profile is directly related to the use of hs-TnT at EDs. This concurs with a recent study from Italy that reported a significant increase in the absolute number of hospitalized patients with positive troponin, following the introduction of hs-cTnT in clinical use, without an increase in the rate of admissions to intensive cardiac care units [8]. The current study suggests that decisions by less experienced emergency physicians may also have contributed to the increased rate of hospital admissions of patients with chest pain. Further, the relatively high rate of hospital admissions (16.7%) due to medicolegal concerns was surprising. It seems that the associated uncertainty of missed ACS diagnosis heavily impacted decision-making by emergency physicians. Accordingly, a US study showed that ED medicolegal and professional concerns have substantially increased hospital admissions for possible ACS [9]. Additionally, though not examined in the study, it is possible that decision-making of emergency physicians may have been affected by patients' own beliefs about their risk of cardiovascular disease. The proportion of patients with non-obstructive coronary angiogram (lesion causing a stenosis of <50% on the CCTA or angiogram) in the current cohort was 42%. This relatively high rate of normal angiograms suggests a low threshold for selecting patients to undergo angiography.

In summary, emergency physicians tended to overestimate the risk of ACS without using formal risk assessment tools. Their decision to hospitalize patients presenting with chest pain may be influenced by their years of clinical experience, medicolegal concerns, the uncertainty involved in the presence of abnormal cardiac troponin, and perhaps patients' own beliefs about their risk of cardiovascular disease.

Declarations of interest

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Invasive bacterial infections in childhood

We have greatly enjoyed reading the recently published article by Niloufar Paydar-Darian and colleagues [1]. In this retrospective study, the authors included 1460 previously healthy febrile children <21 years of age, who presented to an emergency department (ED) for evaluation of fever of <14 days' duration, who had both C-reactive protein and erythrocyte sedimentation rate obtained. Of the 1460 eligible ED encounters, the median patient age was 5.3 years and 762 (50.4%) were hospitalized. The authors showed that none of the children with an invasive bacterial infection had both a normal C-reactive protein and erythrocyte sedimentation rate result, suggesting these tests could be used to reliably exclude invasive infection before results of bacterial culture become available. However, we think that the hospitalization rate of 50.4% is quite high for previously healthy febrile children most of whom have viral infections in real-life practice. Therefore, we think that the authors should detail how did they avoided selection bias which is an error in not ensuring random sampling.

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Low ventilation associated with chest compression, an old observation that requires new physiological interpretation

We read with great interest the paper of Robyn McDannold et al. about the quantification of ventilation volumes produced by chest compressions (CCs) in a convenience sample of 21 out-of-hospital cardiac arrest (OHCA) intubated patients. They assured CCs quality (accelerometer-based technology) and took a sample of delivered ventilations derived from flow sensor placed at the endotracheal tube [1].

The research question addressed in this work is whether CCs alone provide meaningful passive ventilation, which could support the low requirement for additional ventilation during the early phase of resuscitation. Median delivered tidal volume was 7.5 mL, with a maximal at 45.8 mL, remaining significantly below the average of previously estimated anatomical dead space (<120 mL), in cardiac arrest patients [2]. Interestingly, authors hypothesized that the specific anatomical features of the human thorax (i.e.; small sterno-vertebral axis) may explain the low amount of ventilation passively delivered by CCs, which is significantly different from what was quantified in animals that present a different thorax anatomy (large sterno-vertebral axis).

The presented data are consistent with the initial description by Safar et al., who reported that CCs produce an average tidal volume of 156 mL in paralyzed healthy subjects with intact circulation, but no ventilation in patients with cardiac arrest [3]. At that time, this was ascribed to airway resistance after aspiration, lung volumes loss or lung edema, leading to decreased lung compliance [4].

The concept of "intrathoracic airway closure phenomenon", which is associated to lung volume reduction during CCs, was observed in a series of OHCA patients and may alternatively explain these observations [5]. In case of airway closure, the negative alveolar pressure generated by chest release is not transmitted to airway opening and no inspiratory flow is generated. Based on a human cadaver model, the same group showed that the application of positive pressure at the airway opening, set just above the closing pressure of the airways, increases alveolar ventilation generated during CCs [6]. More recently, the systematic analysis of CCs related oscillations magnitude observed in capnogram, obtained immediately after intubation of OHCA patients, permitted to detect intrathoracic airway closure and indirectly reflect the amount of alveolar ventilation [7]. The derived airway opening index (AOI) from oscillating CO_2 varied in that series from 0% (no oscillation indicating closure) to 100% (full oscillation indicating airway patency), with a median value around 50%. These observations are consistent with the proportion of 58% non-oscillating capnograms reported in a recent series of OHCA [8]. In accompanying bench and cadaver models experiments, high AOI reflecting airways patency was associated with a reduction in CO_2 due to the sole effect of CCs, suggesting an effective contribution of fresh gas on alveolar ventilation despite low CCs related volumes; on the opposite, no alveolar ventilation was produced during CCs if AOI was below 25%, which occurred in 20% of OHCA patients [7].

The presented results by McDannold et al. support the hypothesis that the geometry of human thorax is likely associated with lung volume reduction and intrathoracic airway closure during CCs; it may explain the low amount of ventilation generated in a substantial proportion of OHCA patients after the initial phase of resuscitation [1]. Looking at the Fig. 1, we noticed that CCs produce very minimal inspiratory flow during the decompression phase (upper part) when compression phases (lower part) reach higher peak flows. The flow tracing obtained in a cadaver model during manual CCs exhibits very similar airflow limitation during the decompression phase, despite a complete release of the positive intrathoracic pressure generated by CCs, which is prevented by adding some positive pressure above the closing pressure (Figure) [6]. It strongly suggests that the intrathoracic airway closure is the main determinant of flow limitation during CCs.

In addition, it is worth to note that in the set of measurements reported by McDannold, volume could reach 50 mL, which correspond to observations in case of high AOI and fully patent airways. Hence this series probably mixed patients with low and high AOI, meaning different levels of closure at different phase of resuscitation. As suggested by Idris, in a swine model, the decline of ventilation during CCs may be time-dependent [9].

In conclusion, the low tidal volumes generated by CCs reported after initial resuscitation, in different series of OHCA patients, interestingly comfort previous observations; new physiological data show that it is related to intrathoracic airway closure, likely occurring when the respiratory system (thorax/lung) compliance and volume have been