A Patient Simulation Model Based on Decision Tables for Emergency Shocks

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Abstract. Physicians in Intensive Care Units (ICU) have to deal with shocked patients very often. These are critical emergencies that require rapid and precise clinical reactions in order to avoid fatal organ decline and patient's death. New treatments cannot be checked directly on real patients because of the risks this could imply. For this reason several simulators have been published. These simulators use to be implemented as complex mathematical models and they are oriented to concrete types of shocks or focused on fluid resuscitation. Here, we present a new simulator which is based on decision tables. It is able to simulate the evolution of seven different sorts of shocks when treated with fluid resuscitation and vasoactive agents.

Keywords: Simulation · Shock · Decision tables · Knowledge representation

1 Introduction

Shock is a common condition in critical care, affecting about one third of patients in the intensive care units (ICU). It is described as the clinical expression of circulatory failure that results in inadequate cellular oxygen utilization [1].

Some of the most common shocks are cardiogenic shock, anaphylactoid shock, cardiac tamponade, hemorrhagic shock, neurogenic shock, shock due to acute pulmonary embolism, and septic shock.

Clinical reaction to shocks in ICU must be fast and precise because of the vital consequences on the patient and to prevent worsening organ dysfunction and failure. These reactions entail the combined application of ventilatory support, fluid resuscitation, and vasoactive agents [1].

All these actions have a direct and sometimes immediate consequence in some internal hemodynamic parameters. These parameters combine under the name of cardiac output, and they are: volemia (or the amount of fluids), heart rate, contractility (or heart strength), and vasoconstriction (or the weight of the vessels).

Since many of these parameters are not directly observable by the physician who is attending the patient, medical decisions must be taken in terms of some

D. Riaño et al. (Eds.): KR4HC/ProHealth 2015, LNAI 9485, pp. 21–33, 2015.
DOI: 10.1007/978-3-319-26585-8_2

observable vital signs such as: heart rate, central venous pressure, arterial blood pressure, systolic blood pressure, diastolic blood pressure, finding of hematocrit, and superior vena cava oxygen saturation.

The capacity to foresee the consequences of medical interventions in patients with shock can reduce the risks associated to this medical problem. In this sense, several simulators have been implemented for shocks. So, Arturson et al. [2] describe a mathematical model based in 19 differential equations, 147 algebraic equations, and about 150 variables. Roa et al. [3,4] propose a non-linear macroscopic mathematical model for patient fluid distribution during the first 48 h after injury is presented. Dagan et al. [5] incorporate the Sheffer's model [6] and baroreflex [7] in a multi-layer system, providing a mathematical model for hemodynamic, oxygen balance, and control mechanism.

All these simulators are oriented to burned or bleeding patients, that are two cases of the hypovolemic shock and they are focused on fluid resuscitation exclusively. This implies two major limitations: on the one hand, the existing models are single-shock oriented, therefore several independent models are needed if we want to deal with multiple shocks, but integrated models are preferred in ICU's where a response to all sort of shocks must be given. On the other hand, as far as we are aware, existing models are centered in fluid resuscitation for shock, which represents only one part of the real treatment provided at ICU's.

In order to overcome these two limitations, we constructed a simulator able to represent not only hypovolemic shock, but also distributive, cardiogenic and obstructive shocks in an ICU. Our model will consider both fluid resuscitation and vasoactive agents and it will represent the knowledge about shock hemodynamics as a variation of decision tables [8,9].

Our simulator relies on initial emergency treatment of patients with shock arriving to an ICU, accordingly to the signs and clinical actions mentioned in clinical practice guidelines [1,10–18].

Decision tables are knowledge structures in which columns represent rules, and rows represent either conditions (antecedents of the rules) or actions (consequents of the rules). They have been qualified as intuitive, simple, fast, flexible, clear, and powerful structures [19,20]. These features make decision tables very suitable to represent knowledge coming from medical experts and its subsequent validation [21,22].

The rest of the paper is organized as it follows: in Sect. 2 we formalize the basic information and knowledge structures that define a treatment of the seven sorts of shock considered. In Sect. 3, we describe the simulator in terms of the structural design. The knowledge contained inside the simulator is explained in Sect. 4. Then in Sect. 5 we discuss the model and provide the conclusions of this work.

2 Formalization of the Treatment of Shock

The treatment of shock is based on three main aspects: ventilate (i.e., oxygen administration), infuse (i.e., fluid resuscitation), and pump (i.e., administration of vasoactive agents).

These are three sorts of treatment actions that, combined together, aim to control a set of non-observable hemodynamic parameters (cardiac output) that manifest in terms of some observable vital signs. Formally speaking, we have a set of signs and symptoms (S) that describe the patient condition with regard to shock, and a set of clinical actions (A) which can be continuous or discrete depending on the duration of their effects on the hemodynamic parameters. Continuous actions have effect while they are applied, but the effect disappears when the action is interrupted. On the contrary, discrete actions have persisting effects over time. From a medical point of view, in an emergency context (few hours), we can assume that the effect of discrete actions persists along the whole patient simulation time at the ICU.

During the treatment, the patient may evolve along a sequence of states (P_i), being P_0 the condition of the patient when admitted, and P_m the condition of the patient at discharge time. The evolution of a patient is then seen as a sequence $<P_0, P_1, ..., P_m>$, where each P_i defines the values observed for the signs and symptoms in S at the i-th stage of the patient evolution. Each P_i is a subset $\{(S_j^i, U_j^i)\}_j$, where S_j^i is a sign in S, and U_j^i the value of S_j^i for the patient in state P_i.

Simultaneously, the treatment can be adjusted as the patient evolves. The complete treatment T on a concrete patient is then a temporal sequence $<T_1, T_2, ..., T_m>$, where each T_i defines the clinical actions performed when the patient was in condition P_{i-1}. Each T_i is a subset $\{(A_j^i,V_j^i)\}_j$ where A_j^i is one of the actions in A, and V_j^i can be "take", "do not take", or a dosage, with regard to the clinical action A_j^i. Moreover, a time δ_i exists between the application of T_i and T_{i+1}. For example, when a patient arrives with an anaphylactoid shock, with Systolic and Diastolic blood pressure 72/39 mmHg P_0 is $\{SBP = 72, DBP = 39\}$), then the physician may decide to give epinephrine 1mg IV bolus (i.e., $T_1 = \{(EB,1\,mg)\}$), which causes the patient to evolve to a new state with a blood pressure 95/58 mmHg ($P_1 = \{SBP = 95, DBP = 58\}$), after $\delta_1 = 10$ m.

In order to construct a patient simulator for cardiogenic shock, anaphylactoid shock, cardiac tamponade, hemorrhagic shock, neurogenic shock, shock due to acute pulmonary embolism, and septic shock, we have used the clinical guidelines [1,10–18] to identify a set of signs and symptoms S containing seven vital signs: heart rate, central venous pressure, arterial blood pressure, systolic blood pressure (SBP), diastolic blood pressure (DBP), finding of hematocrit, and superior vena cava oxygen saturation. In addition, our simulator will be sensitive to a set of clinical actions A containing 17 different actions: antihistamine, hydrocortisone, epinephrine bolus, atropine, diuretic, fluid infusion, plasma transfusion, red blood cell packed, dopamine infusion, dobutamine infusion, norepinephrine infusion, epinephrine infusion, vasodilators, thrombolytic therapy, reperfusion (KT), pericardiocentesis, and insertion of intra-aortic balloon counterpulsation[1].

[1] Clinical actions of the sort infusion are considered continuous. The rest are discrete.

3 The Patient Simulation Model

The evolution of patients affected of some of our seven targeted shocks when they are subject to fluid resuscitation and vasoactive agents is reproduced with a Patient Simulation Model that uses the formalization introduced in the previous section. This Patient Simulation Model (PSM) has two different modules. The first one is the Action Module (AM) which deals with calculating the changes that would be produced in a theoretical standard patient if a set of treatment actions were applied. AM is where simulation holds. The second module, called the Patient Module (PM), is able to define patient cases with different features in order to interact with the simulator. Both modules are described in the next subsections.

3.1 The Action Module

When a patient receives several treatment actions, her hemodynamic parameters (cardiac output) may be affected, and this can provoke some of her vital signs to suffer a modification. The primary task of AM is to calculate the effect of one or more treatment actions in the patient vital signs. Every single action may affect one or more vital signs, and the combination of several actions may affect the same vital sign in many different ways.

In order to calculate how the treatment actions modify the vital signs of the patient, the AM module uses a variation of decision tables [21]. Regular decision tables are knowledge structures representing rules as columns. The premises of the rules appear as rows at the top of the table, while the conclusions appear as rows at the bottom of the table. Conditions in the premises appear in the table cells intersecting the column of the rule and the row of the corresponding premise. Conclusions in the rule are marked with an X in the corresponding conclusion row of the column representing the rule.

We proposed a modification of the structure of regular decision tables so that the new structure could contain the main simulation rules required to implement AM. See Table 1. These new tables have as inputs the current vital signs of the patient (rows $vitalSign_i$, for all $vitalSign_i$ in S), and the treatment actions performed (rows $treatmentAction_i$, for all $treatmentAction_i$ in A). The output of these tables are the increment or decrement expected for each vital sign (rows $vitalSign_i$ at the bottom).

Table 1 shows a generic example of such sort of decision tables. One of such tables exists for each shock in the simulator.

In the table, the first inputs represent vital signs. We can represent Yes/No, enumerated, and numeric signs. Yes/no signs such as $VitalSign_1$ can contain values of the sort Yes or No. Enumerated signs such as $VitalSign_2$ can contain labels representing numeric intervals (e.g., very high, high, medium, low, or very low) and numeric signs such as $vitalSign_s$ can contain explicit numeric intervals (e.g., $>= 90$, < 120, or $90 - 120$). Tables can also contain unknown values ($-$) as in $Rule_n$.

Table 1. Generic example of decision table in the Action Module

	$Rule_1$	$Rule_2$		$Rule_n$
$VitalSign_1$	Yes	No	...	–
$VitalSign_2$	*medium*	*low*	...	*high*
...				
$VitalSign_s$	$>= 90$	< 120	...	$90 - 120$
$TreatmentAction_1$	Yes	No	...	Yes
$TreatmentAction_2$	high dosage	low dosage		medium dosage
...				
$TreatmentAction_t$	No	No	...	Yes
$VitalSign_1$	$+15$	$+5$	...	
$VitalSign_2$	-6	-10	...	$+10$
...				
$VitalSign_s$		$+0.4$	...	-0.1

The last inputs in the decision table are the treatment actions. They can contain Yes/No values (e.g. $TreatmentAction_1$), used to indicate whether the treatment follows a clinical procedure or not, but also enumerated values (e.g. $TreatmentAction_2$), used to indicate dosages as high dosage, low dosage, or medium dosage. Other dosage granularities are also possible.

The output of decision tables represents the modifications of the vital signs caused by treatment actions or by combinations of treatment actions. The modification can represent an increment (+), a decrement (−) or a null effect (empty cell) for each vital sign. Increments and decrements are associated a relative magnitude of the change (e.g., +5 or −0.1), +15 meaning that it has triple incidence in the vital sign than +5.

During the simulation process, one or more rules of the table can be activated simultaneously. In this case, all the values are added to the corresponding vital sign. For example, if two rules activate with respective incidences +15 and −5 on a vital sign S_j^i with a current value U_j^i, the new value for that sign after δ_{i+1} time will be U_j^{i+1}=$U_j^i + 15 - 5$.

3.2 The Patient Module

Under the same health condition, different patients can have different normality parameters for their vital signs, and their response to a same treatment can vary. For example, a SBP = 100 could be considered normal for a certain patient, but very low for a patient with hypertension because the normality parameter of these two patients for SBP are different. Also, some patients may present resistance to certain drugs or hypersensitivity to some treatments. Sometimes, the general health condition of a patient or her risk factors (which are not necessarily

related to the shock under consideration) can make certain clinical actions not to be recommended or even counter-indicated. Additionally, the same dosages may have different effects depending on each clinical condition.

These are some of the reasons why the application of AM alone is incomplete to implement a correct simulator. So, we complemented our simulator with a Patient Module (PM) that adapts the results provided by AM to the patient under consideration.

While the AM can be seen as running the simulation for a standard patient and calculating a standard response, the PM works to simulate a customization of the results in accordance to the features of each single patient.

In the PM we are allowed to determine the special behavior of the patient for each treatment action in A by defining her sensitivity/resistance with a percentage: 0 % representing full resistance, values between 0 % and 100 % partial resistances, 100 % the standard effect, and values above 100 % crescent sensitivities. See an example in Table 2 with patient sensitivities in the section `ACTION SENSITIVITIES`.

Furthermore, comorbidities and physical conditions may vary vital signs normal references and their limits, in every single patient. Clinicians use this sort of variations to determine the treatment goals. In the PM we are allowed to specify normality parameters of cases by means of ranges that will be used not only to assess the effects of clinical actions over tolerable limits but also to decide on ICU discharges. See these ranges for a case example under `VITAL SIGN RANGES` in Table 2.

In PM, cases are allowed to contain sensitivity/resistance percentages for all the 17 clinical actions related to the shocks, and vital sign ranges for all the 7 vital signs relevant to the shocks under consideration. Table 2 describes a case of a 72-year female admitted with a septic shock with risk factor hypertension and other secondary diseases. Her normality parameters are defined under the section `VITAL SIGN RANGES`, with boundaries `MIN`, `LOW`, `HIGH` and `MAX`. These values define the ranges for unacceptably low (below `MIN`) and unacceptably high (above `MAX`) causing the simulation to stop, at risk (between `MIN` and `LOW` or between `HIGH` and `MAX`) requiring urgent intervention at the UCI, and normal (between `LOW` and `HIGH`) to consider ICU discharge. The patient also shows 130 % hypersensitivity to resuscitation using intravenous fluid and 80 % sensitivity (i.e., 20 % resistance) to norepinephrine effects. The section `INITIAL SIGNS` describe the values for all the sings of the case at the time of admission in the ICU.

3.3 The Iteration System

The combined action of AM and PM provides simulation of a case under some particular circumstances. This simulation is punctual in time, but shock management uses to be a process consisting of several steps. In order to provide "long term"[2] simulations representing patient evolution, we propose an iteration system.

[2] The concept long term must be understood in the context of an ICU (few hours).

Table 2. Example of patient definition with vital sign ranges and clinical action sensitivities

```
Patient ID : 12
AGE : 72    SEX : female    WEIGHT : 65.0 Kg   HEIGHT : 1.57 m
SHOCK : Septic shock

DESCRIPTION:
Medical history: Arterial hypertension. No previous medications.
Diagnostics: biliary septic shock due to cholecystitis
             acute renal failure

VITAL SIGN RANGES:          MIN     LOW   HIGH    MAX    UNITS
Systolic blood pressure    40.0    85.0  140.0   280.0    mmHg
Diastolic blood pressure   20.0    50.0   90.0   130.0    mmHg
Arterial blood pressure    26.6    61.6  106.7   180.0    mmHg
Heart rate                 25.0    60.0  100.0   148.0    bpm
Central venous pressure     0.0     3.0    8.0    20.0    cmH2O
Finding of hematocrit      15.0    35.0   45.0    60.0      %
Sup vena cava oxygen sat   50.0    65.0   85.0    88.0      %

ACTION SENSITIVITIES:
Resuscitation using intravenous fluid  ==>  130 %
Norepinephrine                         ==>   80 %

INITIAL SIGNS:
  <Systolic blood pressure = 62.0>
  <Diastolic blood pressure = 38.0>
  <Arterial blood pressure = 46.0>
  <Heart rate = 118.0>
  <Central venous pressure = 0.1>
  <Finding of hematocrit = 47.0>
  <Sup vena cava oxygen sat = 64.0>
```

The external appearance of the iteration process starts with the initial set of vital signs values for the patient (i.e., P_0 or the values of `INITIAL SIGNS` in Table 2) and the definition of the sensitivity/resistance percentages for that case (section `ACTION SENSITIVITIES`). Then, a set of clinical actions (T_1) is applied, this causing some modifications in the state of the patient (P_1) after a time δ_1 and according to both the rules in the AM decision table of the shock under consideration and the sensitivity/resistance values of the case in PM. This is repeated till a discharge state (P_m) is reached.

Delta times (δ_i) simulate the times that physicians have to wait in order to the changes in the patient states become effective. These times may vary between 15–20 m in emergency treatments and weeks in other clinical treatments.

These delta times are narrowly related to the response times associated to the clinical actions in A. The response time of an action is defined as the time for the

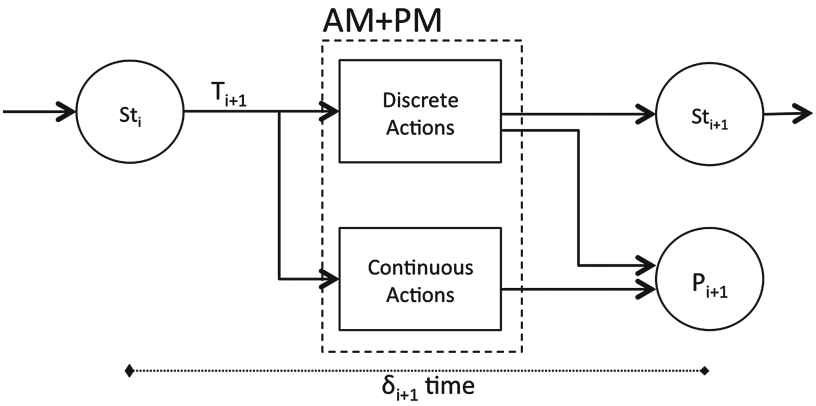

Fig. 1. Iterations with AM and PM

effects of the action to be observed. All the clinical actions in our simulator have a response time that ranges between immediate response (e.g. pericardiocentesis) and 30 m (e.g. thrombolytic therapy).

This external appearance of the iteration process has an internal implementation in the simulator that Fig. 1 depicts.

Here, the state of the patient at admission (P_0=St_0) is treated with the set of clinical actions T_1. This set may contain both, continuous actions and discrete actions. Continuous actions have an effect in the immediate next state of the patient. On the contrary, discrete actions have an effect that persists for the whole ICU treatment, as for example the administration of some drugs like epinephrine bolus or the application of a medical procedure like the insertion of intra-aortic balloon counterpulsation. Some procedures can only be applied once along the treatment.

In our simulation of shocks, continuous clinical actions are dopamine infusion, dobutamine infusion, norepinephrine infusion, and epinephrine infusion. The rest of actions (antihistamine, hydrocortisone, epinephrine bolus, atropine, diuretic, fluid infusion, plasma transfusion, red blood cell packed, vasodilators, thrombolytic therapy, reperfusion (KT), pericardiocentesis, and insertion of intra-aortic balloon counterpulsation) are discrete, being the last four procedures.

The application of both continuous and discrete actions in T_{i+1} to the patient transforms (some of) her vital signs obtaining a new patient state (P_{i+1}) which is observable by the users of the simulator. This process is the result of applying AM and PM to the pair (St_i, T_{i+1}). However, another internal state (St_{i+1}) of the patient is calculated by the simulator. This new state describes the vital signs of the patient from a global perspective required by the simulator to continue with a new iteration of the simulation process, after a time δ_{i+1}. This internal state is the result of applying AM and PM to the pair (St_i, D_{i+1}), with D_k=$\{A_j^k \in T_k$: A_j^k is a discrete action$\}$.

In order to calculate δ_i we take the largest response time of the clinical actions in T_i.

4 Application of the Patient Simulation Model

The above model has been applied to the construction of a simulator for patients affected of one of the following shocks: cardiogenic shock, anaphylactoid shock, cardiac tamponade, hemorrhagic shock, neurogenic shock, shock due to acute pulmonary embolism, and septic shock.

The construction of the AM decision tables was carried out in cooperation with senior physicians of the Emergency Department of the Hospital Clínic de Barcelona. The implications of each action in the vital signs were studied for each one of the shocks. The medical experts were consulted through examples about the expected effects that each one of the actions should have (e.g. *If a patient has anaphylactoid shock, a heart rate of 90 bpm, and she takes antihistamine, then what the new expected value for the heart rate is?*). More than 1000 examples of clinical conditions were analyzed for each shock. The results were analyzed to define the rules that were included in the decision tables. For each shock a decision table was constructed with an average of 50 rules (i.e., columns) per table.

Table 3. Extract of Decision table for Septic Shock

	rule 1	rule 2	rule 3	rule 4	rule 5	rule 6
SBP	< 85			< 85		
DBP		< 50			< 50	
HR			> 100			$60 - 100$
Fluid infusion	Yes	Yes	Yes			
Norepinephrine infusion				$0.12 - 0.6$	$0.12 - 0.6$	$0.12 - 0.6$
SBP	$+5$			$+25$		
DBP		$+1$			$+10$	
HR			-10			$+15$

Table 3 shows an extract of the decision table for the septic shock. We use this table to illustrate the next case example that correspond to the patient described in Table 2: A 72-year, 65 Kg female arrives to the ICU with a biliary septic shock (i.e. gallbladder infection) and initial vital signs: systolic blood pressure (SBP) 62 mmHg, diastolic blood pressure (DBP) 38 mmHg, and heart rate (HR) 118 bpm, among others (see `INITIAL SIGNS` in Table 2).

The physician decides to deliver fluids (500 ml saline solution). The simulator (AM) activates rules 1 to 3 and calculates that, after 20 m, the new vital signs will be: SBP 67 mmHg, DBP 39 mmHg, and HR 108 bpm. These 20 m are calculated as the response time of the fluid infusion applied. Since the case shows default sensitivity (100 %) to fluid infusion (note that no special sensitivity/resistance is indicated for fluid infusion in Table 2), the calculated vital sign values are not modified by the PM.

With this new information on the state of the patient, the physician concludes that more fluids are required, and continues with other saline solution. After a new delay of 20 m, the new vital signs are calculated to be: SBP 72 mmHg, DBP 40 mmHg, and HR 98 bpm. This is the result of applying the same rules 1 to 3 in the decision table.

In front of this new state, the physician decides to give norepinephrine infusion at 0.2 mcg/Kg/min. Now rules 4, 5 and 6 in the Table 3 conclude that after 15 m (response time for norepinephrine infusion), the patient's vital signs should evolve to SBP 97 mmHg, DBP 50 mmHg, HR 113 bpm. But, since the patient is 80 % sensitive to norepinephrine (see Table 2), the simulated evolution of the patient with AM+PM will be SBP 92 mmHg, DBP 48 mmHg, HR 110 bpm. Notice that these values are the result of calculating 80 % of the values +25, +10, and +15 (i.e., +20, +8, and +12, respectively) and modify previous DBP, SBP, and HR values with these increments.

Since none of the vital signs is at a normal level for the patient (see `VITAL SIGN RANGES` in Table 2), the shock intervention at the ICU should continue.

5 Discussion and Conclusions

Simulation is a technique to "replace or amplify real experiences with guided experiences that evoke or replicate aspects of the real world" [23]. The term "simulator" used in health care usually refers to a device that presents a simulated patient and interacts appropriately with the actions taken by the simulation participant [24]. Barjis et al. [25] have classified health care simulation in four areas: Clinical Simulation, Operational Simulation, Managerial Simulation, and Educational Simulation.

Clinical Simulation is used to study, analyze and replicate the behavior of diseases and biological processes in human body. *Operational Simulation* is used for capturing, analyzing and studying health care operations, service delivery, scheduling, health care business processes, and patient flow. *Managerial Simulation* is used as a tool for managerial purposes, decision making, policy implementation, and strategic planning. *Educational Simulation* is used for training and educational purposes.

Operational Simulation and Managerial Simulation are closely interrelated and correspond to the components for health care process management. Conversely, Clinical Simulation and Educational Simulation are more related with the patient care, and the sort of simulator that we have described in this paper.

Shock treatment has two important features that make it a special case in ICUs. These are, the need of a rapid intervention and the vital risks of the clinical decisions. Simulators of shocked patients are tools that can allow physicians to have more calm, reflexive, and risk-free performances [26]. All such simulators that we are aware of have complex internal mathematical models whose adaptation to new evidences and to the changes in clinical guidelines is complicated and it puts some difficulties to their evolution as new computer versions. It is also worth to mention that the knowledge behind these simulators use to be

hidden behind complex mathematical formalisms or distribution privacy rules, that make the physicians using these simulators unable to fully understand the reasoning of the simulator and also prevent them from participating in the modification or extension of that knowledge.

In addition, these sort of simulators are single-shock oriented and therefore difficult to use in a combined way to manage patients with more than one shock, for example cardiogenic shock whose treatment causes an anaphylactoid shock.

In spite of the complexity of the shock simulators that we are aware of, they use to be focused on partial treatments as the use of fluid resuscitation. A need of simulators based on flexible, incremental, upgradable and user understandable technologies is detected. For clinical and educational simulation in ICU's, these tools should also allow the simulation of the most prevalent shocks.

Decision tables are computer knowledge-based structures that could satisfy all these requirements. Our experience in the construction of a simulator for seven different shocks involving fluid resuscitation and vasoactive agent treatments concludes that the structure of the internal modified decision tables facilitates the knowledge engineer to identify the right questions for the physicians (or domain experts) during the knowledge acquisition process. Our proposed decision table structure also allows an easy representation of the acquired knowledge and the incremental versioning of knowledge about shocks. Extending the simulator with other shocks or diseases is also possible with the addition of new tables. Incorporating new clinical actions for a shock is also possible.

During this work, we observed that our simulation model with decision tables had two issues that required further consideration: On the one hand, decision tables provide similar response to patients who are in a similar state and receive the same treatment. In order to adjust this behavior to real patients arriving to an ICU who may evolve differently even if the same treatment is applied, we extended the simulator with a patient module (PM). Modeling patient's sensitivities/resistances to clinical actions, and patient's vital signs normality, was a complex process that concluded with an intuitive way to customize standard evolutions. The patient models are used by the PM in order to allow that different patients could evolve differently even if the same treatment is provided.

On the other hand, we consider that the patient's age, weight, and sex are closely related to the patient resistance to drugs and also to the response time of these drugs. In this work we have not included these relationships in the current simulator but they will be considered in future versions.

According to Gaba et al. [23], the purposes of simulation in health care can be classified in: *Education*, *Training*, *Performance assessment*, *Clinical Rehearsals*, and *Research*.

These are interesting areas of application of our Patient Simulation Model. In the immediate future we are addressing a couple of actions. On the one hand, we are defining with the Hospital Clínic de Barcelona a validation study of the model. This study will integrate several senior physicians of the ICU of this hospital that will interact with the model in order to provide feed-back on the correct and incorrect simulation allowing us to refine the model with new

versions of the decision tables contained. On the other hand, we will start a training program of the residents in the Emergency Unit of that same hospital. We are currently working in a on-line tool for residents to train their treatment recommendations for shocks with patient cases whose evolution will be calculated with our simulator as the users decide new clinical actions.

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http://www.springer.com/978-3-319-26584-1

Knowledge Representation for Health Care
AIME 2015 International Joint Workshop,
KR4HC/ProHealth 2015, Pavia, Italy, June 20, 2015,
Revised Selected Papers
Riaño, D.; Lenz, R.; Miksch, S.; Peleg, M.; Reichert, M.;
ten Teije, A. (Eds.)
2015, XIV, 159 p. 36 illus. in color., Softcover
ISBN: 978-3-319-26584-1