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Title
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Journal
Proceedings of the Annual Meeting of the Cognitive Science Society, 10(0)

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Publication Date
1988

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Causal Reasoning about Complex Physiological Mechanisms by Novices

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Acquiring expertise in medicine involves the mastery of a wide repertoire of cognitive skills and and the accumulation and integration of a vast store of knowledge. It is generally agreed that in medicine, there are two distinct domains of knowledge; clinical knowledge and basic science knowledge. Clinical knowledge is primarily categorical and includes a classificatory scheme for disease entities and associated clinical findings. Basic science knowledge in medicine involves the organization of biomedical models at different levels of abstraction, (e.g., from the biochemical level to the organ systems level). Much of our research has addressed the issue of the of basic science in a clinical problem-solving context. We have found certain anomalies, namely that basic science knowledge does not necessarily support the diagnostic reasoning process (studies summarized in Patel, Evans & Groen, in press). Students use of basic science concepts frequently resulted in inconsistent clinical inferences that were actually counter-productive. In fact, the use of basic science in the causal explanations of expert physicians did not seem to improve the accuracy of diagnostic performance. This motivated us to examine in greater detail the process of how these basic science concepts are acquired and utilized. In this paper we investigate the application of complex concepts in the domain of pulmonary physiology.

Recently, a great deal of research has focused on the acquisition of scientific concepts (e.g., Pines & West, 1986). Many of the findings suggest that scientific concepts are inherently difficult to master because they typically require rather abstract formal representations that are not easily amenable for application in a particular domain (White & Frederiksen, 1987). The quantitative formal instantiation of these concepts is not generally consistent with students' naive intuitions about the phenomena that they purport to explain (diSessa, 1983). The evidence from many investigations clearly suggest that many students who have completed science courses acquire a knowledge of scientific concepts that cannot be used flexibly to interpret causal events and result in the acquisition of significant scientific misconceptions (Reif, 1987).

Feltovich et al. (1988) investigated medical students understanding of complex concepts in the domain of cardiac physiology. These studies document widespread misconceptions in the structure and function of a diseased heart. These misconceptions are developed early on in the learning process and become stabilized and entrenched in a student's diagnostic thinking. The authors argue that difficulty in attaining a deep level of understanding is often a result of reductionistic approaches to imparting the knowledge to the learner. Complexity is progressively introduced to early "scaffold" models, in order to facilitate an orderly acquisition of simplified models of complex biomedical phenomena. This oversimplification results in an in an inability to apply these concepts in "real-world" phenomena.

In this experiment, we continue on this trend towards investigating the nature of reasoning in biomedicine. We deviate slightly from our previous work in that we look at a problem which, although couched in a clinical setting, remains a problem of causal reasoning about

a mechanism and therefore fairly closed within the basic scientific sub-domain of physiology. As a result we propose that reasoning in the domain of physiology will have a certain correspondence to reasoning in *physical systems* domains such as physics (Mcloskey, 1983) and electronics (White and Frederiksen,1987), in the sense that there is a need for a qualitative causal understanding to enable the problem-solver to characterize a process and predict future states of a system. One important difference is that, like all biomedical sciences, pulmonary physiology is not a formal domain, in the sense that it is not possible to unambiguously state a set of principles that are sufficient to solve all clinically related problems (Larkin, 1981). The application of these concepts to clinical problems are therefore by abstraction and analogy (Patel, Evans & Groen, in press).

The Instructional Setting

The topic of pulmonary physiology is typically introduced to second-year medical students. The structural components that constitute the gas-exchange system are--air spaces- upper airway, bronchi, bronchiole, alveolar units, etc. The focus then shifts to the topic of pulmonary mechanics, in which the system is reduced to a *physical device* , and the behavior is explained in terms of the scientific laws governing fluid and gas mechanics-- *A difference in pressures drives a gas through a membrane.* This progresses to identifying the constraints of gas-exchange through a membrane, gas exchange through a lung unit, and exchange through the lung as a whole. The precise interrelationships are explicated through identifying the algebraic relations that hold. This is often without an alternate qualitative perspective. Class-lectures include an introduction to clinical cases; specifically, the measurable symptoms and the respective causal agents (the influence), and an identification of exactly what is happening at a a deep level by characterization in some physiological interpretation. Toward this end, many abstract concepts are introduced. For example, understanding the pathophysiological state of right heart failure may require an understanding of the relationship between pressure and flow and their relation to resistance in a compliant set of vessels.

METHOD

The subject pool consisted of 160 second year medical students at McGill University ,who were attending a series of lectures on cardio-pulmonary physiology. Following the lectures, the students were examined using a conventional multiple choice format. The lectures were monitored by our researchers and an additional essay question for the examination was devised in consultation with the instructor. The question was designed to assess the students' ability to comprehend selected concepts and use them in applied context. 40 of the students' essay questions were randomly sampled for our analysis. The stimulus text and question are presented below.

Table 1: Stimulus Text and Question Presented to Students.

A 30 year-old previously well non-smoker has a large pulmonary embolus which blocks the right pulmonary artery and half the vasculature of the left pulmonary artery. When he presents to the emergency department, he is very short of breath. His pO2 is 48 torr (N=100), and his pCO2 is 30 torr (N = 40 torr). A catheter was placed in his pulmonary artery and the pressure was 50/15 mmHg (N=25/8). The pressure in the right atrium was 12 mmHg (N < 8) and the cardiac output was 3.5 L/min. (N = 5-6 L/min.)
Discuss the factors which could have produced the hypoxia by predicting what would happen to dead space and ventilation-perfusion matching.

Cognitive Task Analysis

The task requires that subjects initially form a situational representation of the problem statement. The givens consist of a quasi-anatomical description of the patient's state and a set of values which represent deviations from the normal values (in brackets), reflecting the underlying pathophysiological perturbations. The first part of the task requires that subjects identify the process by which hypoxia (low concentration of Oxygen in the blood) results. They are primed to interpret the scenario through the concepts of dead space, which characterizes the state and ventilation-perfusion matching which, additionally, identifies a process of inadequate oxygenation of blood. The task is essentially, to build a causal chain from the given representation to the end state of hypoxia. These latter two concepts look at the relationship between ventilation and perfusion and therefore can be applied to various representations of the pulmonary unit for example the lung as a whole, the left and right lungs respectively, specific lung units, etc.

Data Analysis

A reference model was devised in consultation with three expert physicians and textual materials on the subject matter. The subjects' protocols were compared to a reference model by identifying; 1) the various concepts in the respective set of protocols; 2) the representation on which these are applied, by identifying locational cues in the text--" On the *left* side you have decreased perfusion, and normal ventilation..." which contrasts with a description such as-- "perfusion in the *entire lung* has decreased..."; and 3) concepts with a quantitative attribute, the specific value or direction of change-- " The V/Q ratio in the left lung has increased..."

The following represents a list of concepts that they may choose to bring to bear to the problem:

diffusion of gases through a membrane
shunt
oxyhemoglobin curve
O2-CO2 relationship
(inverse) relationship between CO2 and ventilation
compartmental model
dead space
relationship between ventilation and perfusion--V/Q
V/Q distribution
anatomical features of the respiratory system.

Table 2: Reference Model

There are four factors which produce hypoxia:

1)hypoventilation
2)shunt
3)ventilation-perfusion mismatching
4)diffusion problems

Hypoxia is present here as reflected in the low po2 value which is indicative of the concentration of oxygen in the blood as being low (not meeting demands of the tissues).

An embolus blocks all the flow to the right lung and half the left. This blood must then be accommodated by the 1/2 functioning left lung. That is, it gets forced over here. So in other words 1/4 of

the entire lung is receiving all the blood. The 3/4 of the lung which is now not receiving blood but is still being ventilated therefore represents physiologic dead space.

Normally the blood received in the still functioning lung is one quarter the cardiac output--1.5 L/min(6 L/min. divided 4 since it is one fourth the lung). In this case it is 3.5 L/min. so there is definitely increased perfusion. He is blowing off more CO2 as well which is reflected in his CO2 being below normal. So he is blowing it off faster than it is being delivered. And since it is only in the working region of the lung through which the gases can diffuse in or out (since this is the only place where air and blood meet), it seems that ventilation has increased as well,since ventilation is linearly related to co2. So v/q is normally 1-- same flow of blood as air in that region for which this v/q is applied; and in this case it is near that. So the v/q of this working lung is ok in the sense of measuring flow of air and blood through this region at a *gross* level.

Diffusion problems: 1)Blood may be going through at such a fast velocity now that by the time it reaches the end-capillary (there is a certain distance through which the blood and gas meet) the hemoglobin in the blood doesn't have time to pick up the oxygen diffusing through the membrane? That is, there is not full equilibration. This may be possible in cases where there is a massive amount of overperfusion. 2) Another secondary factor may be that the increased pulmonary pressure may cause edema leading to impaired diffusion. Shunt: If the increased volume of blood OPENED UP some blood paths which allow blood to flow from the venous side to the arterial side without it coming in contact with air (in alveoli) then this blood would not be oxygenated. This would be physiologic shunt. V/Q mismatch: There is a distribution of lung units with respect to the ventilation perfusion ratios i.e.,. Take any lung unit and see ventilation /perfusion. Most units in a normal lung have a ratio of one. In our case, there is a redistribution such that a lot more have a v/q ratio of < 1 with the increased perfusion and a lot less have a v/q of > 1 and of course there are less with v/q = 1 which is optimal for gas exchange. So the distribution is such that CO2 can still escape enough but not such that O2 can come in. CO2 and O2 behave differently from each other because hemoglobin hold oxygen and not CO2. There are different affinities for both of these by the blood as illustrated by the oxyhemoglobin curve.

Increased perfusion causes low pO2 i.e.,.--with normal perfusion, it happens to equilibrate at the point of 100 torr (mmHg). As you increase the perfusion you decrease the point of equilibration.

RESULTS and DISCUSSION

Out of 40 students, nineteen made the incorrect inference of 1/2 blockage in the left lung leading to decreased perfusion in the perfused portion of the left lung. Of related concepts, i.e., concepts apart from v/q mismatch and dead space, for which the students were primed for, 12 of 40 discussed the relationship between CO2 and ventilation, 5 of 40 discussed v/q distribution, 11 of 40 discussed the relationship between O2 and CO2 (often implicitly through mentioning the relationship between o2 and hemoglobin which makes it act differently than CO2).

With respect to other mechanisms being offered as an explanation to hypoxia, 17 of 40 attributed the hypoxia to edema resulting from increased pulmonary pressure; 2 of 40 mentioned the increased transit time (failure of complete equilibration-a diffusion problem), and 7 of 40 mentioned shunt as the significant factor.

Specific to the application of V/Q, some students chose to apply V/Q to the right and left lung respectively. That is, looking at the amount of ventilation in the right and the amount of perfusion. Since there is no mention in the stimulus text of a change in ventilation but the blockage infers zero perfusion, the two are combined to give a V/Q of infinity for this lung (some value / 0). Then looking at the left lung, some chose to apply it to the whole left lung while others partitioned this lung into the perfused and nonperfused portions. Others applied the concept at a global level to give a V/Q > 1-- reduced perfusion to ventilation.--

"The obstruction greatly decreases the blood perfusion, therefore resulting in mismatching of ventilation-perfusion ratio.." (S78)

The use of dead space was similar although many students limited the dead space to the right lung, failing to consider the nonperfused half of the left lung. Some students were global in their application by merely stating that overall dead space had increased.

In identifying the process of Ventilation-perfusion mismatch as the causal factor, it is essential that the reasoner determine that there are regions of overperfusion . We postulate that incorrect inference-- *partial blockage in the left lung causes decreased perfusion* arises for two distinct reasons:

1) There is a failure to consider that the blood normally going to the right lung must be accommodated by the unblocked left lung, illustrated by the following student protocols :

"The pressure in the pulmonary artery is increased since normal blood flow is impeded: blood is stuck there..."(S53).

"The embolus in the artery means that less blood is getting to the lung where it is needed...This [compensatory mechanism of hyperventilation] does nothing for the blood that is not reaching alveoli, since in this case most of the flow is in this position (i.e., not reaching air fluid spaces)..." (S82)

These excerpts illustrate that students often are incapable of maintaining the concept of circulation as a closed system, that is there is in some sense, no choice but for the entire blood flow to be routed to the unblocked pulmonary vessels. All the necessary information is not represented (carried over) when the focus of attention shifts to the left lung. In fact, this is largely present in protocols that partitioned the initial problem into a right/left scenario as opposed to a functional/non-functional distinction. The problem then, results from the *static interpretation* of concepts and the problematic use of *oversimplified representations* identified by Feltovich et. al. (1988)

2) If there are features in a sub-problem that have similar components to a previously solved sub-problem, then heuristics are used to infer relationships based on the respective change in values:

"The pulmonary embolus blocking the R.(right) pulmonary artery would cause an increase in physiological dead space and a ventilation/perfusion imbalance where essentially V/Q = infinity. This lung becomes non-functional in its capacity of eventual oxygenation of tissues. On the left side, there is also an increase in physiological dead space and an imbalanced V/Q ratio. There is less perfusion due to the embolus and so the V/Q ratio would increase. Assuming that you get half the perfusion expected initially, this would give a V/Q ratio of 2."(S97)

A *surface similarity mapping* is used in examination of different representations with overlapping sets of variables--In the right lung there is total blockage and zero perfusion, therefore since there is half blockage in the left lung, there is half perfusion.

In consideration of the variability in descriptions of causal relations that were identified, we postulate that explanation takes place by characterizing the problem state in terms of modifications to quantitative parameters in a piecemeal manner. These modifications are incorporated into discrete algebraic constraints with some apparent gaps between specific relationships in attempting to construct a coherent explanation. The solution is to use a heuristic to infer weak relations between these gaps, by observing the mutual deviations from the assumed "ideal" values among parameters in a system, in a *normally* functioning

state. These relations that are drawn support causal attribution, but fail to truly identify the pathophysiological process at the level of mechanism. This is a necessary condition for discriminating between different possible pathophysiological outcomes which is critical for practical implications such as therapy. Such modes of reasoning supercede more basic methods of envisioning a process, a form that better characterizes expert reasoning in such physical system domains (Kuipers, 1985; DeKleer and Brown, 1985).

In physiology, concepts such as ventilation-perfusion matching and dead space act primarily on *anatomically* based representations. For example, any attempt to determine dead space ventilation requires that one first identify the location of the lung being perfused and ventilated. This, we feel, is anatomical because it holds in the most efficient manner, the required information to reason about function and behaviour. Schematic representations serve a purpose in this respect (Larkin and Simon, 1987). There is, however, more than one such representation that must be invoked to understand a typical physiological phenomenon. How abstract concepts such as V/Q act on these represent the procedural component of the knowledge required to reason about a system. What is then needed is an explicit identification of how they relate to such representations apart from the models of dysfunction.

CONCLUSION

We have attempted to investigate reasoning about complex concepts in the domain of pulmonary physiology. With respect to instructional implications, our findings suggest that what is needed is an explicit identification of how a piece of knowledge contributes to comprehending the underlying pathophysiological state, as well as a specification of the cognitive nature of the representations that are used in reasoning about a problem. We argue that without a proper scheme for imparting this knowledge, reasoning proceeds in a non-optimal manner. Causal reasoning that employs models of *mechanism* is displaced by strategies that link knowledge components through more general heuristics in the process of constructing an explanation.

An effort must be made to identify the principles/attributes around which the concepts are most effectively organized in this domain. We speculate that most can be anchored in an *ideal* anatomical representation. Such a representation is more concrete and at the same time makes the relevant constraints explicit (for a large subset of problems) such that operations that require less cognitive effort, but concede to varying interpretations are avoided. They also serve as anchoring for other related concepts, which may rely on different anatomical representations but because it is the anatomical relationships that are linked, more abstract relationships are much more accessible and easily maintained.

References

DeKleer, J. & Brown, J.S. (1985). A qualitative physics based upon confluences. In D.G. Bobrow, (Ed.) *Qualitative rteasoning about physical sstems*. Cambridge, MA: MIT Press

diSessa, A.A. (1983). Phenomenology and the evolution of intuition. In Gentner, D. and Stevens, A.L. (Eds.) *Mental models*. Hillsdale, NJ: LEA.

Feltovich, P.J., Spiro, R., & Coulson, R.L. (in press). The nature of conceptual understanding in biomedicine: The deep structure of complex ideas and the development of misconceptions. In V.L. Patel & D.A. Evans (Eds.) *Cognitive science in medicine*. Cambridge, MA: MIT Press.

Kuipers, B.J. (1985). Qualitative Simulation. In D.G. Bobrow (Ed.) *Qualitative reasoning about physical systems*. Cambridge, MA: MIT Press

Larkin. J.H. (1981). Enriching formal knowledge; A model for learning to solve problems in physics. In J.R. Anderson, (Ed.), *Cognitive skills and their acquisition.* Hillsdale, NJ: LEA.

Larkin, J.H. & Simon, H.A. (1987). Why a diagram is (sometimes) worth ten thousand words. *Cognitive Science, 11*, 65-99.

McLoskey, M. (1983). Naive theories of motion. In Gentner, D. and Stevens, A.L. (Eds.) *Mental models*. Hillsdale, NJ: LEA.

Patel, V.L., Evans, D.A. & Groen, G. (in press). Biomedical knowledge and clinical reasoning. In V.L. Patel & D.A. Evans (Eds.) *Cognitive Science in medicine.* Cambridge, MA: MIT Press.

Pines, A.L. & West, L.H.T. Conceptual understanding and science learning: An interpretation of research within a sources-of-knowledge framework. *Science Education 70*, 583-604.

Reif, F. (1987). Interpretation of scientific or mathematical concepts: Cognitive issues and instructional implications. *Cognitive Science, 11*, 395-416.

White, B.Y. & Frederiksen, J.R. (1987). Qualitaive models and intelligent learning environments. In R.W. Lawler & M. Yazdani (Eds.) *Artificial Intelligence and education volume one.*. Norwood, NJ.: Ablex Publishing