A systematic approach to clinical reasoning in psychiatry
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What is This?
Clinical reasoning is a complex process that involves identifying and prioritising the key clinical issues, ruling in and ruling out diagnostic possibilities, and synthesising a case formulation that justifies the proposed treatment plan. It requires highly developed clinical skills to integrate and filter a vast amount of diverse clinical information in a time-efficient manner. Compared with other branches of medicine that often deal with objective clinical data, clinical reasoning in psychiatry can be more challenging. The highly subjective and ambiguous nature of psychiatry tends to obscure the clarity of the clinical reasoning process. Therefore, clinicians and trainees can find it difficult to learn. In this context, a systematic method that employs explicit reasoning strategies can be a useful educational tool. Unfortunately, educational material on such a method is lacking. Clinical reasoning is learnt largely through an implicit process involving years of extensive clinical training.

This paper introduces a paradigm for teaching diagnostic reasoning and case formulation in psychiatry, using an explicit approach to guide clinicians’ thinking.

The proposed approach has been developed by adopting a theory known as epistemological framework, which is founded on the logical inferences known as abduction, deduction and induction. This paper complements the previous paper by the authors on psychiatric case formulation, by providing a complete theoretical foundation for diagnostic reasoning and clinical reasoning in psychiatry. The first part of the paper introduces a model for conceptualising clinical knowledge as a network of interconnected components that can be represented visually. Next, it introduces the clinical reasoning model as an iterative process involving four stages: abstraction, abduction, deduction and induction. These stages are elaborated using examples of symptoms, clinical phenomena, differential diagnoses, and aetiological formulations related to depression.

Clinical knowledge

Clinical reasoning in psychiatry incorporates three broader domains of knowledge, including: diagnostic knowledge (e.g. clinical symptoms and signs, syndromes,
diagnostic criteria); aetiological knowledge (e.g. various biological, psychological and social theories); and treatment knowledge. In order to reason effectively, it is crucial to conceptualise the vast body of clinical knowledge as a well-organised structure.

In order to enable effective clinical reasoning, we suggest that it is important to organise clinical knowledge as a hierarchically organised set of entities involving: (1) clinical phenomena incorporating symptoms and symptom attributes; (2) explanatory models comprising model concepts; and (3) treatments comprising treatment components (Figure 1).

The term ‘clinical phenomena’ was introduced in our previous paper, and we generally use it to describe a small group of related symptoms. It can also be expanded to include important recurrent themes in clinical scenarios, certain characteristics of the patient (e.g. disengagement and noncompliance), and even system or service issues. Clinicians can freely conceptualise clinical phenomena by dissecting the whole set of symptoms that are observed in a patient into building blocks of smaller sets, in relation to explanatory models that can be used to formulate them. Whilst the more familiar term ‘syndrome’ also refers to a set of related symptoms, most of the psychiatric disorders can be viewed as syndromes, and therefore can often be dissected into a collection of individual clinical phenomena. Each of the symptoms can be described using a number of symptom attributes such as the duration, onset, course of the symptom, precipitants, and severity. For the process of clinical reasoning, for example, when considering the diagnosis of major depression, the group of related symptoms of feelings of guilt, self-blame, shame, and low self-esteem could be considered as a single clinical phenomenon. We have termed it ‘guilt, self-blame & shame’, and will progress the development of our concept using this terminology.

Aetiological knowledge is drawn from a broad array of theories and models that can be used to understand individual clinical phenomena. For example, a model based on cognitive schema theory or object-relation theory are two possible explanatory models that can be used to understand the above-stated clinical phenomenon, guilt, self-blame and shame, as described later in this paper (Figure 7). This model-based aetiological knowledge can be structured as templates, which link each clinical phenomenon to a particular component of a treatment regimen as described in our previous paper.

Treatment knowledge comprises pharmacological, physical (e.g. electroconvulsive therapy (ECT) and regular exercise), psychological and social interventions. Each treatment intervention can be viewed as a set of components. For example, cognitive behaviour therapy (CBT) can be considered to comprise patient engagement, psychoeducation, cognitive restructuring and behaviour modification. Similarly, antidepressant treatment may consist of patient engagement, psycho-education, strategies to improve compliance, and strategies to deal with side effects, in addition to the medication-dosing schedule. The relative importance of each treatment component may vary according to an individual patient’s situation. For example, the main problem with one patient may be non-engagement, whereas intolerability due to side effects may be the key issue with another patient. Viewing each treatment as a set of subcomponents has greater utility, since it enables the individualisation of treatment for each patient by emphasising the most relevant sub components of the treatment that are applicable to a given patient situation.

In order to assist the clinician in understanding our model, we have represented the above visually (Figure 2) in a knowledge network. The knowledge entities (i.e. symptom attributes, clinical phenomena, diagnoses, aetiological explanatory models, treatments, and treatment components) related to the symptom ‘depressed mood’, are represented. The authors assert that a coherent and logical structure for organising clinical knowledge is vital for good quality formulation, because the process of clinical reasoning can be considered as a deterministic process of navigating this vast network of clinical knowledge.

Clinical reasoning

Whilst clinical reasoning is a mostly implicit procedural skill developed by clinicians over years of clinical experience and training, there are also a number of explicit reasoning strategies employed by expert clinicians. These include hypothetico-deductive reasoning, scheme-inductive reasoning, pattern recognition, and forward and backward reasoning. Expert clinicians may use more than one of these reasoning

Figure 1. A simple model for conceptualising different entities in the three knowledge domains as hierarchically organised categories.
strategies, depending on the complexity of the clinical problem.\textsuperscript{10} The epistemological framework, also known as the Select and Test (ST) model,\textsuperscript{1} is of particular importance for two reasons. Firstly, it is based on the three main types of logical inferences (abduction, deduction and induction) that were described by the pioneer in mathematical logic, Charles Peirce.\textsuperscript{11} Secondly, the ST model is not an exclusive one. In other words, it is able to accommodate most of the other clinical reasoning strategies within itself.

For the purposes of this paper, we will focus on diagnostic reasoning and aetiological reasoning, leaving the large domain of arriving at treatment decisions for a subsequent paper. In diagnostic reasoning, symptom attributes, symptoms, clinical phenomena, and diagnoses are elicited in an iterative process in a systematic manner. In aetiological reasoning, case formulation is constructed by linking clinical phenomena to relevant explanatory aetiological models. Below we apply the ST model to derive the four stages – abstraction, abduction, deduction and induction – as described in Figure 3, to arrive at a formulation.

Abstraction

Abstraction involves eliciting individual symptoms, clinical signs, and phenomena during the clinical examination. Clinical symptoms are abstract concepts defined in the clinician’s mind, noting that patients may not express their symptoms and clinical phenomena in a direct way. Instead, they may convey cues to the clinician using their own language and behaviour. For example, a patient may use metaphorical terms such as ‘a dark cloud’ to indicate the symptom of depressed mood. Therefore, abstraction can be thought as the process of translating a set of data, gathered by the clinician during clinical interview and mental state examination, into abstract concepts (i.e. entities in the diagnostic and aetiological knowledge domains). It also involves substantiating the actual presence of the indicated clinical symptoms by eliciting their symptom attributes. For example, if the patient indicates the presence of depressed mood, abstraction requires eliciting its attributes, which may include duration, progress, presence of fluctuations, and severity in a scale from zero to ten, as described in Figure 4.

Starting from the initial clinical findings (e.g. patient’s presenting complaint) abstracted, clinical reasoning...
continues as an iterative and dynamic process using the logical inferences described in the following sections.

Abduction

The abduction stage requires that for each symptom two parallel processes commence, which involve generating diagnostic hypotheses (differential diagnosis), and aetiological hypotheses by tracing the related diagnoses and clinical phenomena. For example, in relation to the abstracted symptom, ‘depressed mood’, abduction will generate a set of diagnostic hypotheses that may include major depression, bipolar affective disorder, adjustment disorder, borderline personality disorder, organic mood disorder and so on. Similarly, as described in Figure 5, it may also indicate possible clinical phenomena that could include guilt, self-blame and shame, interpersonal difficulties, grief and loss, victimisation, and/or existential crisis.

Each symptom can be considered to carry a different level of diagnostic likelihood in relation to any given diagnosis. It is useful to treat symptoms as diagnostic tests, because this enables the clinician to use symptoms effectively to rule in and rule out diagnostic hypotheses. For example, the symptom, ‘depressed mood’ can be considered to carry a higher diagnostic likelihood towards major depression compared with the symptom ‘loss of weight’, which may carry a higher diagnostic likelihood towards medical conditions causing weight loss.

Even though it is not the usual practice, the statistical concepts of sensitivity and specificity, which have been described in relation to diagnostic instruments, can still be applied for individual symptoms and clinical phenomena in order to gauge their diagnostic likelihood in relation to each diagnosis.12 In other words, the presence or absence of a given symptom can be thought of as a positive or negative test result carrying a certain degree of diagnostic sensitivity and specificity towards a particular diagnosis. Using their clinical expertise and judgement, clinicians may assign subjective estimates for sensitivity and specificity for each symptom–diagnosis pair.
Each diagnostic hypothesis indicated by a given symptom can be considered to carry a certain degree of criticality that can be determined considering the potential consequences caused by the failure of making the diagnosis. For example, whilst the symptom, ‘chest discomfort’ may indicate diagnostic hypotheses, ischemic heart disease and panic attack, ischemic heart disease can be considered to have a higher degree of criticality considering its associated mortality risk.

A given symptom can potentially be related to a large number of diagnoses, and it may be practically impossible to complete an exhaustive exploration of all diagnostic possibilities within a given time frame and resource constraint. For example the symptom ‘chest discomfort’ can be related to a large number of both medical (e.g. cardiac, respiratory, musculoskeletal, infection and so on) and psychiatric diagnostic hypotheses (anxiety disorders, somatisation disorders, depression, substance use, etc). The clinician’s knowledge of relative diagnostic likelihood and criticality of a given symptom in relation to any diagnosis has a greater utility in this situation, since it helps the clinician to narrow down the search, but still not to miss any critical diagnostic possibilities.

**Deduction**

Deduction involves continuing the two parallel reasoning processes of generating diagnostic hypotheses and aetiological hypotheses that were initiated during the abduction stage. In relation to diagnostic hypotheses, deduction involves exploring the other symptoms expected in relation to each diagnostic hypothesis. In comparison with abduction, which involves tracing the likely diagnoses related to a given symptom, deduction involves tracing all expected symptoms in relation to a given diagnosis. Therefore, abduction and deduction can be considered to have opposite directions in their inference (i.e. from a symptom to diagnoses versus from a diagnosis to symptoms). In relation to aetiological hypotheses, deduction involves exploring likely aetiological explanatory models related to each clinical phenomenon.

For example, after raising major depressive disorder as one of the diagnostic possibilities that are related to the abstracted symptom ‘depressed mood’, deduction involves tracing the other clinical symptoms that are expected in major depression as described in Figure 6.

In order to explain the deductive inference in relation to aetiological reasoning using an example, consider two clinical phenomena related to the symptom, ‘depressed mood’: guilt, self-blame and shame; and interpersonal difficulties. Two possible explanatory models that may explain the clinical phenomenon, ‘guilt, self-blame and shame’ could be a model based on CBT, and a second model based on object-relation theory (Figure 7).

Similarly, three possible explanatory models that may explain the clinical phenomenon ‘interpersonal difficulties’, would be an interpersonal psychotherapy (IPT) model, a CBT model, and a self-psychology model (Figure 8). For more detail on how better to understand the process of deriving aetiological explanatory models, the reader is referred to our previous paper.2

The next step involves repetition of abstraction, in which the symptoms indicated by each diagnostic hypothesis, clinical phenomenon and their explanatory models, are elicited as described previously. For example, deductive inference in relation to the diagnostic hypothesis of major depression will indicate a number of expected symptoms, including anhedonia, decreased appetite, and suicidal behaviour and thoughts as described in Figure 6. Each of these symptoms is elicited as previously described in the section on abstraction.
The presence of new symptoms and clinical phenomena elicited during the abstraction will indicate new diagnostic and aetiological hypotheses, which will then be generated, and explored during abduction, and deduction stages respectively. Thus, an iterative process is formed that will continue until it is deemed that all likely diagnostic and aetiological possibilities have been adequately explored.

**Induction**

Once the iterative process involving the above described stages is completed, the last inference, induction, involves evaluating each diagnostic hypothesis. Induction requires matching the relevant symptoms and clinical phenomena that have been elicited from the patient, with those expected in relation to each diagnostic hypothesis. In doing so, the clinician may compare the elicited symptoms and clinical phenomena against the standard diagnostic criterion given in DSM-IV or ICD-10. A diagnostic hypothesis is accepted if the elicited symptoms and clinical phenomena satisfy the relevant diagnostic criteria; otherwise, the diagnostic hypothesis is rejected.

Similarly, in relation to the aetiological hypotheses, the elicited clinical phenomena related to each aetiological hypothesis, and the relevant information available, need to be matched against what is expected in each relevant explanatory model. In doing so, the clinician will then choose the best fitting explanatory model. The accepted explanatory model may then indicate an appropriate treatment strategy. For example, testing an aetiological hypotheses in relation to the clinical phenomenon ‘interpersonal difficulties’ (Figure 8) involves choosing the explanatory model that best describes the patient’s situation, which may be the IPT model as the appropriate intervention.

**Conclusion**

This paper introduces a systematic approach for clinical reasoning with the aim of teaching diagnostic reasoning and case formulation in psychiatry. The underpinning logical inferences in our approach will help to crystallise the clinical reasoning process, reduce ambiguity and assist with more rapid conceptualisation of case formulation by the trainee. The authors also have described elsewhere a formal version of this clinical reasoning model that can be used to develop clinical decision support tools, and expect to introduce a learning tool by converting the theoretical framework introduced in this paper into a software program. Importantly, this paper complements the previous paper on psychiatric case formulation, and we hope that these two papers will together serve as a useful resource for teaching and learning diagnostic reasoning and case formulation in psychiatry.

**Disclosure**

The authors report no conflict of interest. The authors alone are responsible for the content and writing of the paper.
References


